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CONTEMPORARY DEBATES IN

PHILOSOPHY OF BIOLOGY

EDITED BY FRANCISCO J. AYALA
AND ROBERT ARP

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Contemporary Debates in Philosophy of Biology

Edited by

Francisco J. Ayala and Robert Arp

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The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

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Notes on Contributors

Robert Arp has specializations in philosophy of biology and ontology in the informatics sense of the term. With Alex Rosenberg, he is co-editor of *Philosophy of Biology: An Anthology* (Wiley-Blackwell, 2009), with George Terzis, he is co-editor of *Information and Living Systems: Philosophical and Scientific Perspectives* (forthcoming), and his latest book dealing with issues in evolutionary psychology is titled *Scenario Visualization: An Evolutionary Account of Creative Problem Solving* (2008). Currently, he works for The Analysis Group (Falls Church, VA) as an ontologist and is completing a book with Barry Smith and Andrew Spear that deals with the construction of ontologies.

Francisco J. Ayala is University Professor and Donald Bren Professor of Biological Sciences at the University of California, Irvine. He has published over 900 articles and is author or editor of 31 books. He is a member of the U.S. National Academy of Sciences and the American Philosophical Society, and received the 2001 U.S. National Medal of Science. *The New York Times* named Ayala the "Renaissance Man of Evolutionary Biology."

Susan Blackmore is a freelance writer and lecturer, previously Reader in Psychology at the University of the West of England, Bristol. Her research on consciousness, memes, and anomalous experiences has been published in over 60 academic papers, as well as book chapters, reviews, and popular articles. She writes a regular blog for *The Guardian*, and often appears on radio and television. Her books include *The Meme Machine* (translated into 13 other languages, 2000), *Consciousness: An Introduction* (2005), and *Conversations on Consciousness* (2007). She has been practicing Zen for 20 years, and has recently published *Ten Zen Questions* (One World Press, 2009).

Richard M. Burian, Ph.D. University of Pittsburgh, is Professor Emeritus of Philosophy and Science Studies at Virginia Tech. He is a historian and philosopher of biology, with research focusing on history and philosophy of genetics, exploratory experimentation in biology, interactions among developmental biology, evolutionary

biology, and genetics, and philosophical accounts of conceptual change in science. He was one of the co-founders of the International Society for History, Philosophy, and Social Studies of Biology, of which he was President, 1999–2001.

Michael F. Claridge is Emeritus Professor of Entomology at Cardiff University. After graduating M.A. and D.Phil. in zoology and entomology at Oxford in 1959, he has since worked continuously from a base in Wales. He has served as Presidents of the Linnean Society of London (1988–1991), the Systematics Association (1991–1994), and the Royal Entomological Society (2000–2002). He has published in areas such as insect systematics, ecology, behavior, and evolutionary biology and has always had a particular interest in the nature of species and species concepts.

Robert Cummins is Professor and Chair of the Department of Philosophy at the University of Illinois, Urbana-Champaign. He is the author of *The Nature of Psychological Explanation, Meaning and Mental Representation* (1989) and *Representations, Targets and Attitudes* (1996), as well as articles in the philosophy of mind, science, and biology and the history of early modern philosophy. His current research focuses on representation as well as the interaction development, learning, and selection that pertain to informing the mind.

Michael R. Dietrich is a historian and philosopher of biology in the Department of Biological Sciences at Dartmouth College. His work ranges from historical and conceptual issues in molecular evolution to the gendered division of labor in *Drosophila* genetics. He is currently completing a biography of Richard Goldschmidt.

Stephen M. Downes is Professor of Philosophy at the University of Utah. His main areas of specialization are philosophy of science and philosophy of biology. He has recently published on evolutionary psychology, the levels of selection debates, and modeling in science.

John Dupré is Professor of Philosophy of Science, and Director of the ESRC Centre for Genomics in Society (Egenis) at the University of Exeter. He has held posts at Oxford, Stanford, and London, and in 2006 was the Spinoza Professor of Philosophy at the University of Amsterdam. He has published widely in the philosophy of biology. His most recent book, co-authored with the sociologist Barry Barnes, is *Genomes and What to Make of Them* (2008).

Douglas H. Erwin is a Senior Scientist and Curator of Paleozoic Invertebrates in the Department of Paleobiology, National Museum of Natural History, Smithsonian Institution and Professor at the Santa Fe Institute. He has a Ph.D. in Geology from UC Santa Barbara, and his research concerns major evolutionary innovations, the end-Permian mass extinction, and related issues.

Evelyn Fox Keller is Professor Emerita of History and Philosophy of Science at MIT. With a Ph.D. in theoretical physics from Harvard University, she is the author of publications extending from technical contributions to physics and biology to historical and philosophical analyses of scientific practices. She is the recipient of

many awards and honorary degrees, a member of the American Philosophical Society, the American Academy of Arts and Sciences, a MacArthur Fellow, and, most recently, holder of the Chaire Blaise Pascal in Paris. Her most recent book is *The Mirage of a Space between Nature and Nurture* (2009).

Manfred D. Laubichler is Professor of Theoretical Biology and History of Biology and Affiliated Professor of Philosophy at the School of Life Sciences and Centers for Biology and Society and Social Dynamics and Complexity at Arizona State University. He is the co-editor of *From Embryology to Evo-Devo* (2007), *Modeling Biology* (2007), *Der Hochsitz des Wissens* (2006), *Form and Function in Developmental Evolution* (2009), and an associate editor of *Endothelial Biomedicine* (2007). He is also an associate editor of the *Journal of Experimental Zoology, Part B Molecular and Developmental Evolution* and of *Biological Theory.*

Alessandro Minelli is a Professor of Zoology at the University of Padova. His research interests include animal phylogeny, the principles of systematics, and especially evo-devo, with a focus on the evolution of modular body parts. He is the author of *Biological Systematics* (1993), *The Development of Animal Form* (2003), *Perspectives in Animal Phylogeny and Evolution* (2009), and *Forms of Becoming* (2009).

Brent D. Mishler is Director of the University and Jepson Herbaria at University of California-Berkeley, as well as Professor in the Department of Integrative Biology, where he teaches phylogenetic systematics and plant diversity. He received his Ph.D. from Harvard University in 1984, and then was on the faculty at Duke University before moving to UC Berkeley in 1993. His research interests are in the systematics, evolution, and ecology of bryophytes, especially the diverse moss genus *Tortula*, as well as in the phylogeny of green plants and the theory of systematics. He applies methods ranging from microscopy through growth experiments, DNA sequencing, and genomics.

Mark Perlman is Professor of Philosophy at Western Oregon University. He received his Ph.D. from the University of Arizona in 1994, working with Robert Cummins. He is the author of *Conceptual Flux: Mental Representation, Misrepresentation, and Concept Change* (2000), and is co-editor, with André Ariew and Robert Cummins, of *Functions: New Essays in Philosophy of Psychology and Biology* (2002), as well as other papers on functions, philosophy of law, conceptual role semantics, and philosophy of music. He is also a musician, both orchestral conductor and string bassist.

Del Ratzsch (Ph.D., University of Massachusetts-Amherst) is Professor and Chair of the Philosophy Department of Calvin College in Grand Rapids, Michigan. His primary areas of research are philosophy of science, and interactions between science and religion. His published work in those areas includes *Nature, Design and Science* (2001) and *Philosophy of Science: The Natural Sciences in Christian Perspective* (1986).

Martin Roth is an Assistant Professor of Philosophy at Drake University. A Ph.D. in philosophy from UC-Davis, he has published in the areas of philosophy of mind, cognitive science, and epistemology.

Michael Ruse is Lucyle T. Werkmeister Professor of Philosophy at Florida State University and Director of the Program in the History and Philosophy of Science. He is the author of many books, most recently *Charles Darwin* (Wiley-Blackwell, 2008), a work which looks at Darwin's importance for philosophy, and he is the co-editor of the *Cambridge Companion to the Origin of Species* (2008).

Carmen Sapienza is Professor of Pathology and Laboratory Medicine at the Fels Institute for Cancer Research at Temple University. His laboratory was among the first to discover DNA methylation differences between maternally and paternally inherited genes. The Sapienza laboratory is studying natural variation in epigenetic marking, the selective forces at play, and whether environmental factors have an effect on the stability of epigenetic marks, leading to disease.

Todd K. Shackelford received his Ph.D. in psychology from the University of Texas at Austin in 1997. He is Professor of Psychology at Florida Atlantic University, and chair of the Evolutionary Psychology Area. His current research interests include conflict between the sexes, particularly with regard to sexual behavior (see www.ToddKShackelford.com).

Valerie G. Starratt received her Ph.D. in psychology from Florida Atlantic University in 2008 while working with Todd Shackelford. Currently, she is a visiting Assistant Professor of Psychology at Nova Southeastern University. Her research interests include intimate relationships and human sexuality from an evolutionary psychological perspective.

William C. Wimsatt, Peter B. Ritzma Professor, teaches philosophy, history of science, and evolutionary biology at the University of Chicago. He studied engineering physics and philosophy at Cornell, and then went from philosophy at Pitt in 1969 to a post-doc with Richard Lewontin at Chicago. He has written on functional organization, reductionism, and reductionistic research strategies, levels of organization and mechanistic explanation, units of selection, heuristics, emergence, modeling, robustness, satisficing, generative entrenchment, and methods and problems in studying complex systems. His book *Re-Engineering Philosophy for Limited Beings* (2007) integrates many of these themes. He currently works on issues surrounding cultural evolution and on the role of generative entrenchment in evolutionary processes.

General Introduction

Who Is This Book For?

This book features current research by scholars doing work in the central areas of philosophy of biology. Further, the papers are presented in a debate style with *yes* and *no* responses—often qualified—to basic questions posed in this continually developing sub-discipline of philosophy. This being the case, this book is ideal as (1) a stimulus for students in philosophy of biology and biology classrooms, as well as (2) a reference work for scholars who are working in this exciting field.

What Is the Philosophy of Biology?

The word "philosophy" comes from two Greek words: *philos*, meaning "love," and *sophos*, meaning "wisdom." Love here means something like an intense desire for something, while wisdom is arguably a kind of knowledge gained from experience, whether this be practical experience (gained from living life with all of its ups and downs) or theoretical experience (gained from understanding, evaluating, critiquing, and synthesizing ideas, positions, and concepts). Ever the theoretician, the philosopher has always been the person who not only desires to look deeper into some claim, idea, argument, event, or state of affairs by questioning assumptions and challenging status quo thinking, but also attempts to broadly explain and systematize aspects of reality (also see Craig, 2002; Pojman, 2007). In Bertrand Russell's (1912/1999) words, which are appropriate given the nature of this book: "Philosophy, like all other studies, aims primarily at knowledge. The knowledge it aims at is the kind of knowledge which gives unity and system to the body of the sciences, and the kind which results from a critical examination of the grounds of our convictions, prejudices, and beliefs" (p. 9).

The word "biology" comes from two Greek words as well: *bios*, meaning "life," and *logos*, meaning "word," "rational account," or "science." Thus, biology is the kind or type of science that studies life, which most of us already know. Whereas *biology*

can be characterized as a set of sub-disciplines (the biological or life sciences) under science, the concern of which includes the description, classification, analysis, explanation, prediction, and ultimately control of living things (Audesirk, Audesirk, & Byers, 2008; Campbell & Reece, 2007), *philosophy of biology* can be characterized as a sub-discipline of philosophy—complete with topical subject-matter to be discussed momentarily—the concern of which is the meta-leveled attempt on the part of philosophers, biologists, and other thinkers to understand, evaluate, and critique the methods, foundations, history, and logical structure of biology in relation to other sciences, disciplines, and life endeavors so as to better clarify the nature and purpose of biological science and its practices (see Hull & Ruse, 2007; Rosenberg & Arp, 2009; Rosenberg & McShea, 2007; Ruse, 2008; Sarkar & Plutynski, 2008).

The Classification of Biology and Philosophy of Biology

Concerning the classification of biology within the general discipline of science, it is usually envisioned as a natural, empirical, pure science, as we illustrate in Figure 0.1 (also see Sadava, Heller, Orians, Purvis, & Hillis, 2008; Silberberg, 2008; Tippens, 2007). We are aware that what is represented in the figure is a partial taxonomy, and that there may be other ways to classify the sciences.

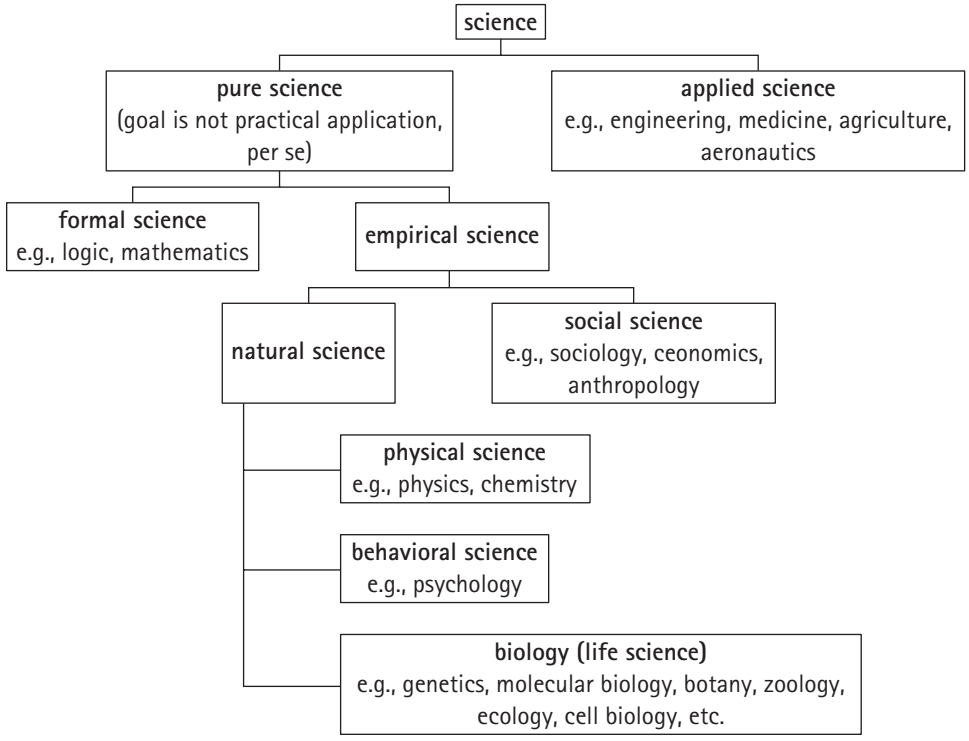

Figure 0.1: A basic classification of biology as a science

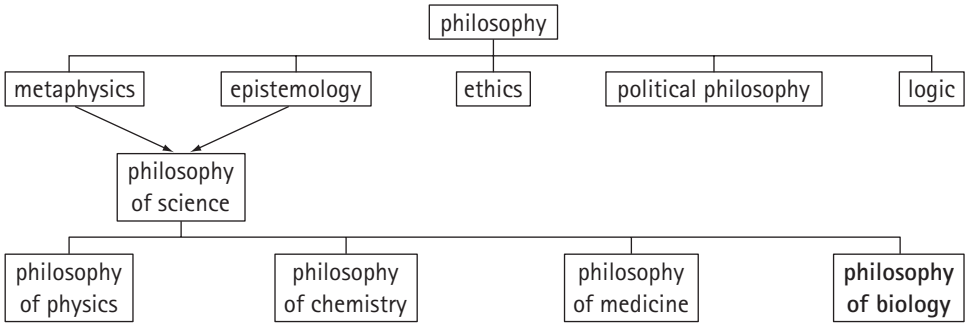

Figure 0.2: Philosophy of biology classified

Concerning the classification of philosophy of biology within the general discipline of philosophy, it is usually envisioned as a sub-discipline of philosophy of science, along with others like philosophy of physics, philosophy of chemistry, and philosophy of medicine. Because it concerns not only what kinds of things exist (*metaphysical* parts, processes, principles) as well as how we can know these things (*epistemological* perceptions, models, beliefs, justifications), the classification of philosophy of science itself can be considered a hybrid under metaphysics and epistemology; although, of course, this is debatable (Godfrey-Smith, 2003; Newton-Smith, 2001). Figure 0.2 represents a partial taxonomic classification of philosophy of biology, and we are aware that there are many other philosophical disciplines and sub-disciplines not shown, as well as that it is possible to classify the discipline of philosophy by historical time-periods or major movements (Copleston, 1994; Jones, 1997; Solomon, 2005).

The Relationship between the Biologist and the Philosopher

There are many biologists who think philosophically, and there are many philosophers who think like biologists, and this has always been the case in Western history since these two disciplines began coexisting with one another. In fact, researchers in these two disciplines have been able to assist one another in advancing ideas, putting issues to rest once and for all, and overthrowing faulty paradigms, as well as furthering technological comforts, establishing moral codes, and alleviating pain and disease (National Research Council, 1996, 2000; Watson & Arp, 2008).

A universally known example of this relationship between biology and philosophy is Charles Darwin (1809–1882), the field biologist and scientific naturalist, thinking like a philosopher of biology by mounting his self-proclaimed "one long argument" for natural selection in his famous work titled *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life* (1859/1999). In line with our descriptions of philosophy and philosophy of biology put forward above, Darwin definitely challenged status quo thinking with natural selection and offered a meta-level analysis, explanation, and systematization of the biosphere. Darwin's contemporaries even referred to him as a philosopher (Schad,

2004, p. 9). There is a famous paper by Theodosius Dobzhansky (1900–1975) titled "Nothing in Biology Makes Sense Except in the Light of Evolution" (1973) and it is obvious that, were it not for Darwin's philosophy of biology-like thinking and theorizing concerning natural selection and evolution, the biological sciences would be foundationless today.

Just as many biological conundrums have been aided by philosophical thinking, so, too, many philosophical problems have been either solved or enlightened with the help of the biological sciences. Let us focus on one example. In philosophy of mind, *substance dualism* is the belief that a person is made up of two fundamental things—a material or physical body and an immaterial or non-physical mind/soul/spirit—that can exist apart from one another. Those who believe in the immortality (or reincarnation) of the soul are substance dualists because they think that the death of the body does not mean the death of the soul (for example, *Catechism of the Catholic Church*, 1994; also Baker & Morris, 1996). The soul lives on as a separate substantial thing after the death of the body, which is another, distinct, separate substantial thing. A lot of people on the planet are substance dualists of one sort or another, probably because of their religious upbringing (Morgan & Laungani, 2005). Think of the cartoons where a character gets killed and the body stays flat on the ground while the soul/mind/spirit/immaterial substantial part leaves the body and ascends into a heavenly world—this is straight-forward mind–body substance dualism.

Contemporary discussions of religious and non-religious forms of substance dualism in Western history usually trace their roots back to the famous Modern philosopher, René Descartes (1596–1650) (Descartes, 1998; see also Baker & Morris, 1996), but forms of substance dualism can be found in the history of Western philosophy in the twentieth century and back through Aquinas (1225–1274) to Augustine (354–430), Plotinus (ca. 204–270), Aristotle (384–322 BCE), and Plato (ca. 428–348 BCE) (Foster, 1991; Aquinas, 1949; Augustine, 1991; Plotinus, 1992; Aristotle, 1995; Plato, 1997). In fact, the cartoon character rendition of the soul leaving the body is very close to what people actually believed in most Western societies throughout the history of Western civilization. The histories of Eastern and Middle Eastern philosophy are also peppered with beliefs in various forms of substance dualism (Abramson & Kilpatrick, 1995; Hook, 1963; Knapp, 1992).

Now, here is where *neurobiology* has made important contributions to the philosophy of mind, and our thinking concerning substance dualism. First, it seems that the mind is, at best, an emergent or supervenient *property* that is the result of brain states; it may not be reducible to brain states, but it is certainly dependent upon brain state processes (Baars & Newman, 2001; Bisiach, 1999; Gold & Roskies, 2008; Hardcastle, 2007; Kim, 2000, 1999, 1995). If there is any doubt about this, one need only peruse any textbook or journal devoted to the human brain's workings and read about the effects of brain damage upon the psychology of a person (see Bear, Connors, & Paradiso, 2006; Kandel, Schwartz, & Jessell, 2000). For example, without the normal functioning of the prefrontal cortex, individuals are not able to make plans, nor are they able to carry out the behavior necessary to fulfill those plans (Fuster, 1997; Passingham, 1993). Also, as Finke (1980) demonstrated many years ago, damage to the prefrontal cortex causes a person to be unable to store short-term memories. Further, damage to the limbic system can cause certain autisms and other emotional dysfunctions (Bauman & Kemper, 1994).

Given the influence and preponderance of neurobiological data, and the fact that no one has ever witnessed a soul leaving a body or existing in some other "state"—both indicating the fact that, no brain, no mind—many philosophers and other thinkers who still think that there is something special about the mind and mental capacities have opted for forms of *property dualism* in place of substance dualism. According to property dualism, a person is *one substance* that is made up of two wholly distinct features, characteristics, or properties: an immaterial mental property (the mind and mental states) and a material bodily property (the brain and neurobiological states). On this view, the mind and brain are distinct properties of some one person, similar to the way *roundness* and *blackness* are distinct properties found in the one period at the end of this sentence. Just as we can distinguish the property of roundness from the property of blackness in some one period, so, too, we can distinguish an immaterial mental property from a material bodily property in some one person.

However, just as the roundness and blackness of that particular period can exist only while that particular period exists, so, too, according to property dualists, the mental and bodily properties of a person can exist only while that person is alive. So when we delete the period, the properties of roundness and blackness in that particular period cease to exist along with the period. Likewise, when a person dies, both that person's body and mind cease to exist (no brain, no mind). Such a view of mind in relation to body seems to be consistent with neurobiological and other scientific data, and is appealing to those who do not believe in the immortality or reincarnation of the soul.

There is another possibility, namely, that the mind and mental states are completely illusory notions and all that really takes place when one thinks, decides, calculates, feels, believes, and the like, consists solely of neurobiological parts, processes, and principles. Thus, there is neither mental substance nor mental property, just brain and various brain functions. Given the influence and success of neurobiology—as well as the influence and success of physics, cognitive science, and artificial intelligence—many famous living philosophers, such as Paul Churchland (1989), Daniel Dennett (1990), and Jerry Fodor (2001), hold to this materialistic or physicalistic view of mind/brain. There are other positions concerning the nature and existence of mind that have come about as a result of the interaction of philosophy with the various life and behavioral sciences (see Heil, 1998; Lowe, 2000).

There are countless other ways in which the biologist and the philosopher have been helpful to one another, and this will become all the more evident to the reader after having gone through this book. Also, the reader is encouraged to investigate the material in the philosophy of biology, philosophy of science, and the history of biology and science that is referenced at the end of this introduction. It is through the fruitful interactions of the biologist and the philosopher that the subject-matter of philosophy of biology has come to be the way that it is in its present state today.

The Subject-Matter of Philosophy of Biology

Every body of knowledge—science, discipline, study, domain—has a subject-matter and specific questions that give a limit, form, and function to that body. So, for example, biology studies parts, processes, and principles associated with living things primarily as its subject-matter, and not stamp-collecting, business ethics, or World War II.

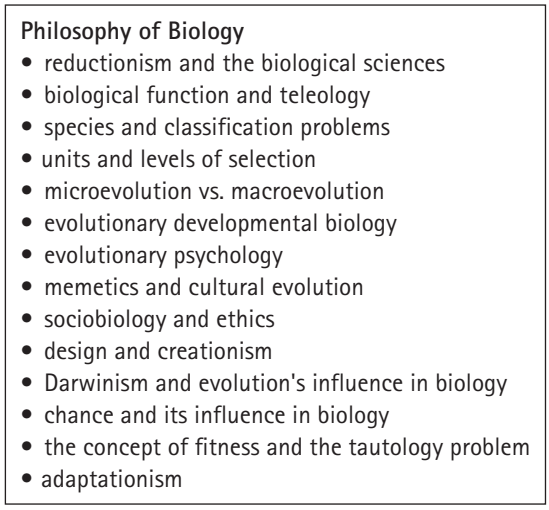

Figure 0.3: Topics that comprise much of the subject-matter of philosophy of biology

Philosophy of biology, too, has a subject-matter and specific questions which it has developed near the end of the twentieth century, and we have included material in this book that reflects much of the current discipline. Figure 0.3 offers some of the central topics that comprise the subject-matter of philosophy of biology (there are others). We have included most of these topics in this book, and describe and explain the basics of the topics in the introductions to each section, complete with further reading material.

There are other topics that, depending upon whom you talk to, can be considered either central or peripheral to philosophy of biology. For example, the philosopher of biology and the ethicist converge (along with other thinkers) in *bioethics*, a sub-discipline of ethics or moral philosophy that deals with issues such as abortion, personhood, contraception, euthanasia, advance directives, informed consent, human and animal experimentation, cloning, prenatal screening, gene therapy, and others (Beauchamp & Childress, 2001; Kuhse & Singer, 2001). The philosopher of biology, the epistemologist, the neurobiologist, and other thinkers converge in *evolutionary epistemology*, a sub-discipline of epistemology that investigates (among other things) the extent to which human perception, cognition, and theorizing are reliable because of natural selection and other evolutionary factors (Harms, 2004; Radnitzky & Bartley, 1993). Philosophers of biology, metaphysical ontologists, biologists, practitioners of medicine, bioinformaticians, and others converge in the burgeoning field of *biomedical ontology*, an area concerned with building domain and formal ontologies (here, understood as standardized, structured, taxonomic classification systems) so as to assist biomedical researchers in classifying, categorizing, and coding their data and information so that it may become optimally interoperable, re-usable, and shareable with the assistance of computational systems and the World Wide Web (Arp, 2007; Arp, Romagnoli, Chhem, & Overton, 2008; Smith, 2003, 2004).

Several of the topics in philosophy of biology exist because of Charles Darwin's ideas concerning evolutionary biology, many of which can be found in the *The Origin*

of Species. Also, every topic in philosophy of biology touches upon Darwin's principles in some way, and vice versa. In the Introduction to the *Origin*, Darwin (1859/1999) notes:

> In considering the Origin of Species, it is quite conceivable that a naturalist, reflecting on the mutual affinities of organic beings, on their embryological relations, their geographical distribution, geological succession, and other such facts, might come to the conclusion that each species had not been independently created, but had descended, like varieties, from other species. Nevertheless, such a conclusion, even if well founded, would be unsatisfactory, until it could be shown how the innumerable species inhabiting this world have been modified so as to acquire that perfection of structure and co-adaptation which most justly excites our admiration. . . . It is, therefore, of the highest importance to gain a clear insight into the *means* [italics added] of modification and coadaptation. (pp. 4–5)

Chapters 3 and 4 of the *Origin* describe Darwin's principle of natural selection, his *means* or explanatory mechanism for the modification and coadaptation of species. Based upon observations of the biosphere, thinkers in the past had hypothesized *that* evolution took place, but it was Darwin who explained *how* evolution occurred with the principle of natural selection, complete with several arguments, numerous pieces of evidence, and other principles that provided a coherent picture of the so-called "Tree of Life" (also see Ayala, 1985; Gould, 2002; Stebbins & Ayala, 1987; Strickberger, 2000; Mayr, 2001). We encourage you to read all of the *Origin*, as this is a classic and foundational piece of writing for anyone doing research in biology or philosophy of biology.

The reader can consult various other resources in philosophy of biology to get a sense of the scope and breadth of this discipline (e.g., Grene & Depew, 2004; Rosenberg & Arp, 2009; Rosenberg & McShea, 2007; Sarkar & Plutynski, 2008; Garvey, 2007; Hull & Ruse, 1998, 2007; Sober, 2000; Sterelny & Griffiths, 1999). The philosophy of biology has a long, varied, and complex history that would take several lifetimes to ingest completely. Besides references to research in the philosophy of biology, we have also included further reading material related to philosophy of science and the history of biology and science below. We hope that students and scholars of philosophy of biology alike will benefit from the material in this book.

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PART I

IS IT POSSIBLE TO REDUCE BIOLOGICAL EXPLANATIONS TO EXPLANATIONS IN CHEMISTRY AND/OR PHYSICS?

Introduction

Humans seek both unity and simplicity when classifying and explaining reality, and this is especially true in the sciences. This way, we can understand, control, and—in the words of the famous Modern philosopher, René Descartes (1596–1650)—"thus render ourselves, as it were, masters and possessors of nature" and other aspects of reality (Descartes, 1637/1998, p. 35). Consider the law of gravity, the kinetic theory of gas, or the principle of natural selection and how they form unified, simple, powerful bases for our explanations of many of the events that occur in the universe. This being the case, there is a tendency for researchers to *reduce* diverse and complex parts, processes, and principles to their most basic constituents, or "lowest common denominator" (as Evelyn Fox Keller calls it in the first paper included in this part), if this is possible.

Now *reductionism* is a complicated term that has many meanings, distinctions, and uses, and one can look at the material in the further reading section at the end

of this introduction for further insights and clarifications. It is arguable that wholes can be reduced to parts, higher levels can be reduced to lower ones, the complex can be reduced to the simple, the older theory can be reduced to the newer one—and there are other forms of reduction. For our purposes here, following the excellent work of Michael Silberstein (2002), we can distinguish (broadly) between a *metaphysical* form of reductionism and an *epistemological* form of reductionism, bearing in mind that there are multiple versions and subversions of each form.

Metaphysics concerns what exists in reality. According to metaphysical reductionists, there are really no entities, properties, or substances that arise out of more fundamental chemical or physical ones since, once the more fundamental ones have been described, that is all there is to the reality of an entity, property, or substance. Thus, for example, when people speak about water, they may take it to be a substance in its own right. However, according to the metaphysical reductionist, water *just is* hydrogen and oxygen—nothing new emerges when two hydrogen molecules combine with one oxygen molecule. The same goes for biological phenomena as, for example, a cell just is the basic chemico-physical parts, process, and principles of which it is composed. In the first paper of this part, Evelyn Fox Keller seems to be expressing this position when she claims: "I am committed to the position that all biological phenomena, including evolution, require nothing more than the workings of physics and chemistry."

Conversely, according to a metaphysical non-reductionist, there is something about water—for example, its liquidity or liquid property—that emerges from (or supervenes on) the hydrogen and oxygen molecules, making it such that this liquidity exists on a separate metaphysical plane from the molecules on which it depends. In the case of the cell, this emergence or supervenience is true even more so, since a cell is a complexly organized hierarchical system of interactions. After all, reasons the metaphysical anti-reductionist, *liquidity* and the *cellular system itself* appear to be something distinct from hydrogen, oxygen, and DNA molecules, chemical bonds, and other chemico-physical parts, processes, and principles.

In many researchers' minds, the possible reduction of biological phenomena to chemical and physical phenomena became all the more *probable* starting with James Watson and Francis Crick's elucidation of the molecular structure of DNA that occurred in a *Nature* paper from 1953. In that paper (1953a) and a quick follow-up paper (1953b), Watson and Crick got scientists and philosophers of science to think about what parts, processes, and principles were *really* at work in heritability. Could Mendelian genetics be reduced to molecular genetics in the same way that heat was reduced to kinetic motion or lightning to electrical discharge? Given that chemistry and physics were making incredible strides at revealing the real workings of other parts of the universe—and, thereby, reducing things to chemical phenomena, then physical phenomena—it would only seem to make sense that living things could be revealed as chemical and/or physical phenomena too. Thus, the possibility of biology being reduced to chemistry and/or physics became solidified as a topic in philosophy of biology with papers such as J.J.C. Smart's "Can Biology Be an Exact Science?" (1959), Kenneth Schaffner's "Approaches to Reduction" (1967), Francisco Ayala's "Biology as an Autonomous Science" (1968; also see Ayala & Dobzhansky, 1974), Alexander Rosenberg's "The Supervenience of Biological Concepts" (1978), Philip Kitcher's "1953 and All That: A Tale of Two Sciences" (1984), Ernst Mayr's "The

Autonomy of Biology: The Position of Biology among the Sciences" (1996), and Elliot Sober's "The Multiple Realizability Argument against Reductionism" (1999), among others.

Whereas metaphysics concerns what exist in reality, epistemology is concerned with how we can know, describe, model, and explain reality, as well as our justification for doing so. According to a standard epistemological form of reductionism—often called *theoretical reduction*—it is possible to replace one theory *A* by another more explanatorily powerful theory *B*; thus, in effect, intertheoretically *reducing A* to *B*. For example, the phlogiston theory of combustion (which stated that an undetected substance called *phlogiston* was released during combustion) was replaced by the oxygen theory of combustion, while the caloric theory of heat (which stated that a fluid substance called *caloric* was responsible for a thing's temperature) was replaced by the kinetic theory of heat. In Kitcher's above-mentioned "1953 and All That," he responds to the typical epistemological reductionist's claim that classical genetics can now be intertheoretically reduced to—and, hence, replaced by—molecular biology, especially now that we know "what's really going on" with heredity at the molecular level. By using the general line of reasoning that the biological sciences are subfields corresponding to multiple levels of complex organization in nature, Kitcher (1984) wants to show that, "despite the immense value of the molecular biology that Watson and Crick launched in 1953, molecular studies cannot cannibalize the rest of biology" (p. 373).

In his paper included in this part, John Dupré mounts a primarily epistemological argument for non-reductionism in biology—and would likely agree with Kitcher—since he argues that "properties of constituents cannot themselves be fully understood without a characterization of the larger system of which they are part" and, hence, the "notion that complex systems, such as those found in biology, can be fully understood from a sufficiently detailed knowledge of their constituents, is mistaken." In her paper included in this part, it would seem that Evelyn Fox Keller is advocating a kind of promissory note that chemistry and physics will be able to explain biology completely. She offers two examples—(1) bacterial chemotaxis and (2) regulating the levels of oxygen and CO_2 in the termite nest—that she believes "provide evidence of partial successes in the effort to 'reduce biological explanations to explanations in chemistry and/or physics.'"

Whatever the case may be, the best strategy for researchers in biology is probably to pursue a general form of *methodological* reduction until parts, processes, or principles in reality are encountered that cannot be reduced. If such non-reducibility is encountered in the metaphysical or epistemological senses, then, following Keller, it may be that what is required is "fundamental transformations in the conventional approaches of both physics and chemistry."

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CHAPTER ONE

It Is Possible to Reduce Biological Explanations to Explanations in Chemistry and/or Physics

Evelyn Fox Keller

Despite all the dramatic successes we have witnessed in recent years in unraveling the physics and chemistry behind biological phenomena, we still have a very long way to go. The most fundamental question of what is the difference between living and nonliving matter remains unanswered. Unlike most physical and chemical systems, living systems must—at the very least—be endowed with *function*, a concept that to this day remains indispensable to biology, yet is effectively missing from the vocabulary of physics and chemistry. Accordingly, we need an account of the evolution of function out of simple physical and chemical dynamics which we do not as yet have. Nevertheless, I retain a guarded optimism: we are, I believe, moving closer to answering this basic question. Yet doing so, I argue, requires fundamental transformations in the conventional approaches of both physics and chemistry.

1 Introduction

My task in this paper is to respond to the question "Is it possible to reduce biological explanations to explanations in chemistry and/or physics?" Forced to choose between only two alternatives—yes or no—I opt for the positive response. But I could as easily have gone the other way. Indeed, I suggest that the question is not well-posed. To the extent that a negative response would imply that I believe there is something beyond physical and chemical processes involved in the formation of living beings, I am obliged to respond affirmatively. I am an unambivalent materialist and do not harbor any such belief. But if by that proposition one means that biological explanations can be reduced to the theories of matter currently available

in physics and chemistry, then my response is no. And not, as Niels Bohr (1932/1958) once argued, because the study of biology can be expected to bring the discovery of new laws. But rather, paraphrasing the arguments put forth by Nancy Cartwright (1983), because laws of physics, by definition, have been developed with reference to a narrow range of possible physical and chemical phenomena; and, one might add, necessarily so. For that is the way with laws: they are developed to describe (or explain) the lowest common denominator of physical and chemical processes, or that which is said to *underlie* the manifest variety of these processes.

How is that lowest common denominator identified? In the actual practice of the physical and chemical sciences, this has generally been translated (and for good pragmatic reasons) into the assumption that that common denominator is coextensive with the simple. Thus, for example, highly oversimplified models, conspicuous for their lack of realism, are often justified by the tacit expectation that, despite their lack of realism (perhaps even by virtue of their simplicity), they contain the features of greatest importance (see Keller, 2002, Ch. 3). In this fashion, a tradition has developed in which concepts of *underlying*, *basic*, *fundamental*, and *simple* are all expected to cluster together.

Such an expectation has worked astonishingly well in physics; not so, however, in biology. As has often been observed, biology simply does not conform to these expectations. Whatever the meaning of *fundamental* in biology, it clearly cannot be equated with *simple*, nor is it at all obvious that it is common to all biological entities. For example, DNA might be argued as fundamental to biological systems, but DNA is an enormously complex molecule. Even in the early days, when molecular biologists believed that its primary function was to code for proteins, no one would have thought of DNA as simple. Today, biologists have learned that this molecule has a repertoire of capacities far richer than had originally been thought, with new dimensions emerging every day. Also, they have learned that DNA is not universal: some organisms make do with RNA, and some entities (e.g., prions) manage to sustain themselves and reproduce without either DNA or RNA. Furthermore, not only does biology *not* admit of a clustering of *fundamental*, *common denominator*, and *simple*, but also a number of philosophers (starting with Jack Smart in 1963) have concluded from this fact that biology, therefore, does not have laws of its own, at least not in the sense that physics is said to have laws. As Ernst Mayr (1982) famously put it, the only universal law in biology is that "All biological laws have exceptions" (p. 38; also see Beatty, 1995; Mitchell, 2000; Smart, 1963).

Which is not to say that biology is not a science (although Smart seems to have thought it was to say exactly that), but rather that it is a science in which the search for "laws" has not, at least so far, played much of a role. True, one finds the term used to describe many important biological discoveries: Mendel's laws of segregation, power laws (referring to the distribution of nodes in biological networks), and even the central dogma is sometimes referred to as a law. But crucially, these ought properly be called *generalities*, not laws. They apply over a broad range of conditions; but there is neither anything necessary about them (as things could have been otherwise), nor need they be free of exceptions. Furthermore, where they do apply, they may or may not be either fundamental or simple. They are simply empirical statements about what generally seems to be the case. As such, they may (at least in some cases) serve as explanations of what follows, but to the extent that they

come with no account of how such generalizations emerge from the vicissitudes of the evolutionary pathways that gave rise to such properties, the generalities themselves remain unexplained.

Where, then, is the role of physics and chemistry in the construction of biological explanations? As a materialist, I am committed to the position that all biological phenomena, including evolution, require nothing more than the workings of physics and chemistry. But this position requires neither a belief in the adequacy of our current understanding of physics and chemistry, nor any expectation that biology will lead us to the discovery of new laws. New *phenomena*, to be sure, but regarding the question of new *laws*, I concur with Jean-Baptiste Lamarck (1809/1984): "Nature has no need for special laws" (p. 132). I believe that analysis of the specific physical and chemical phenomenology involved in biological processes should, in principle, suffice for an understanding of what endows biological systems with the properties of life. I say "in principle" because there is no telling whether or not the complexity of these systems might, in practice, exceed our analytic capacities. But in any case, the more important question is, "What kinds of analysis, in fact, are required?"

2 Systems Biology

Efforts to develop explanations of biological phenomena based on the physical and chemical properties of highly idealized biological systems were not infrequently made over the course of the twentieth century, but they failed to persuade working biologists of the value of such analyses (see Keller, 2002). Eric Ponder (Director of the Cold Spring Harbor Institute from 1936 to 1941) summed up the sentiments of an entire community when, in his response to such early efforts by Nicolas Rashevsky (1934), he argued:

> [I]t is futile to conjure up in the imagination a system of differential equations for the purpose of accounting for facts which are not only very complex, but largely unknown. . . . It is said that if one asks the right question of Nature, she will always give you an answer, but if your question is not sufficiently specific, you can scarcely expect her to waste her time on you . . . what we require at the present time is more measurement and less theory. (cited in Wilson, 1934, p. 201)

Rashevsky, it must be said, was only proceeding as mathematical physicists generally did—indeed, given the analytical technologies available at the time, he had little choice but to begin with a highly idealized (and hence, simplified) model. Similarly, biologists' disdain for such models, and their demand for more measurement, was also based on the realities of their discipline; in particular, on the recognition of both the complexity of biological systems and their rootedness in evolution.

In any case, among biologists it was the demand for more data that prevailed throughout most of the twentieth century. But at the century's end, the dramatic successes of the reductionist program of molecular biology and, perhaps especially, the development of new technologies (e.g., for nucleotide sequencing, molecular labeling, and visual tracking) led to an avalanche of data that threatened to overwhelm the ability of experimental biologists to make sense of their findings using their traditional modes of analysis. In the physical and mathematical sciences, conditions

were also changing, primarily due to the development of powerful high-speed computers and, together, these quite different developments created the conditions for a new rapprochement between mathematical and biological science. Support for interdisciplinary collaborations skyrocketed and, in the early years of the twenty first century, new institutes, programs, and departments under the name of *systems biology* have proliferated like wildfire.

Far from being a unified endeavor, systems biology encompasses a great variety of mathematical, computational, and physical approaches to biological problems, and there is substantial disagreement among researchers as to how far computational analysis will enable one to proceed by working from the bottom up (i.e., from what is known about the relevant molecular elements and their interactions), and to what extent it is necessary to work downwards from known systemic properties to the identification of the relevant actors. Nonetheless, virtually all are agreed on the importance of grounding analysis in the data that have become available, and on the need to develop analytic techniques that would enable one to move beyond a list of parts (e.g., genes, proteins, etc.). There is also widespread agreement on the extent to which the analysis of systems with a large number of components yields (emergent) properties that could not have been predicted from the behavior of the components themselves. Of particular importance is the recognition of such key biological properties as robustness, evolvability, and quorum sensing as properties that emerge only on the systems level. Indeed, in less than a decade, work in systems biology has reconfigured the frontier of biological inquiry, bringing issues to the fore that had previously gone virtually unnoticed.

Thus far, the main strength of such analyses has come not from the application of simple, highly idealized models, but from close attention to the kinds of experimental data that recent biological research has provided. Through imaginative deployment of theoretical and computational techniques from the physical, mathematical, and computer sciences, from the application of methods and insights from the engineering sciences, and from the use of sophisticated technologies developed in experimental physics to obtain the kinds of micro-molecular data that analysis of the physical underpinnings of molecular processes requires, we are beginning to acquire the kinds of understanding that would permit the formulation of biological explanations in terms of physical and chemical processes. But it is becoming clear that these accounts need not resemble the kinds of explanations in physics and chemistry to which we are accustomed.

Among the most obvious differences are: (1) the large number and dense interactivity of parts; (2) the hierarchical (and multi-scale) organization of biological systems; (3) the dependence of the identity of parts, and the interactions among them, on higher-order effects; and (4) the robustness and adaptability of biological structures. It is commonplace to observe that biological systems are vastly more complex than physical systems (they might be said to be maximalist, in contrast with the minimalism of physical systems), and the question has recently surfaced as to why these systems are so complex. As Arthur Lander (2004) puts it, "What purpose does all this complexity serve?" (p. 713). Kunihiko Kaneko (2006) raises a similar question, observing that: "For the purpose of simply responding to the external environment, a simpler reaction consisting of a single level would be sufficient." Kaneko's answer, once again, points us to evolution. He writes that "the great diversity or complexity we

observe is a result of the history of organisms that experienced a great variety of circumstances. . . . The past experiences have come to be embedded in the structure and function of organisms" (p. 9), and that seems correct.

3 Function: A Minimalist Conception

But there are clearly qualitative, as well as quantitative, differences between biological and physical systems, and perhaps foremost among these is that biological systems are endowed with *function,* a concept effectively missing from the vocabulary of physics and chemistry yet, to this day, indispensable to biology. To avoid confusion, let me make clear from the start that my use of the term "function" departs from the convention that has developed in recent years among philosophers of science, whereby function is defined in terms of natural selection. Proper function—as first argued by Ruth Millikan (1984), and now widely asserted—should be understood solely in the context of natural selection: the function of *X* is that "which caused the genotype, of which *X* is the phenotypic expression to be selected by natural selection" (Neander, 1998, p. 319). This is *not* how I am using the term. Indeed, both logically and historically, the notion of function with which I am concerned must *precede* the onset of natural selection, at least as that term is usually construed.

Natural selection—at least since the neo-Darwinian synthesis and, probably, ever since Darwin—has been conventionally understood as requiring the prior existence of stable, autonomous, and self-reproducing entities. Minimally, it presupposes the existence of single celled organisms, or, simply, of stable, autonomous, cells capable of dividing. But these first cells contained, of necessity, numerous sub-cellular entities (or modules), endowing the primitive cell with the functions that would be minimally required for the cell to sustain itself and reproduce. In other words, even if the first cells lacked many features of the modern cell, they had to have had primitive mechanisms to support metabolism, cell division, etc., and there needed to have come into being primitive embodiments of function that would work keep the cell going and to protect it from insult.

To further clarify what I mean by function, let me turn to Michael Ruse's (2003) argument against the use of the term for inanimate systems and, more specifically, against the sufficiency of circular causality for an understanding of biological function. Ruse offers the familiar example of the cyclical process by which rain falls on mountains, is carried by rivers to the sea, evaporated by the sun, whereby it forms new rain clouds which, in turn, discharge their content as rain. The river is there because it produces or conveys water to form new rain clouds. The rain clouds are a result of the river's being there. But Ruse argues that we would not want to say the function of the river is to produce rain clouds, and he is right. What is missing, he claims, is the means by which "things are judged useful."

I will not follow Ruse in his deployment of such worrisomely adaptationist and intentional notions as *value* and *desire*. Instead, I want to salvage his observation by re-describing what he calls *judgment* as a measurement of some parameter, or, if you like, as an evaluation performed by a mechanical sensor that, when exceeding some pre-set limit, is fed back into a controller which is able to restore the proper range of parameter. In other words, reverting to another philosophical tradition, I use the

term *function* in the sense of a simple feedback mechanism. Like a thermostat. As William Wimsatt (2002) puts it, "To say that an entity is functional is to say that its presence contributes to the self-regulation of some entity of which it is a part" (p. 177). Once such a mechanism is added to the rain–cloud–river cycle (say, a mechanism that triggers a change in evaporation rates when the water level falls too low), we can, in this sense of the term, quite legitimately speak of function and say, for example, that the function of such a mechanism is to maintain the water level within a certain range of parameters. Furthermore, I argue, we can similarly refer to the many different cellular mechanisms (proof-reading and repair, chaperones, cell-cycle regulation) that maintain various aspects of cellular dynamics.

To make my minimalist conception of function more precise, I want to focus on those effects that contribute to the stability of such properties that are essential to the system's continuance: its water level, or perhaps its physical integrity. *Stability*, here, means little more than constancy, the fixity of some property of either the internal or external milieu, but it is important to note that maintaining a property within a certain range does not necessarily contribute to the survival of the overall system. For example, maintaining the water level within certain bounds does contribute to the stability of the rain–cloud–river cycle, whereas maintaining a constant temperature of the system need not have anything to do with the stability of the system as such. In order to distinguish the stability or constancy of a particular aspect of the system from the stability of the system as a whole, I will use the term *persistence* for the latter. The crucial point is that, when the stability of a property *does* contribute to the persistence of the overall system, a kind of selection—i.e., selection of the most stable (or most persistent)—will operate automatically. Here, if you will, is selection without reproduction. Like natural selection, it is entirely agnostic with respect to the forces that threaten the stability of a system; it cares only about persistence.

Indeed, if we think of reproduction as just one strategy for ensuring persistence—as Frederick Bouchard (2004), Rosenberg and Kaplan (2005), and others have advocated—we can think of natural selection as a subset of this more general category of *selection for persistence*. And just as in the case of natural selection, the more general selection process that kicks in with variable stability/persistence provides us with an explanation of why the arrangement of device-cum-system is likely to be common. It might also provide us with an explanation of progressive refinements of organization leading to increases in stability. It will not, however, contribute to our understanding of how such an arrangement of parts—an arrangement in which one part exerts an effect that enhances the stability of the larger system in which it is embedded—first came to be.

Put in these terms, the obvious question is how such a mechanism might arise. It is striking that, for all the promise of recent attempts to treat biological systems as instances of the kinds of non-linear dynamical systems encountered in statistical physics, and for all the power of non-linear dynamics to generate emergent patterns, biological function—as I am using the term—seems not to be on the horizon. Apparently, the emergence of function requires rather more than just non-linear interactions, and the question is what that more consists of.

Addressing just this question, Walter Fontana and Leo Buss (1996) have concluded that "the traditional theory of 'dynamical systems' is not equipped for dealing

with constructive processes. Indeed, the very notion of 'construction' requires a description that involves the structure of objects. Yet, it was precisely the elimination of objects from the formalism that make dynamical systems approaches so tremendously successful" (p. 59). They argue that the main problem is that, although conventional dynamical systems theory is "well-suited to treat changes in the magnitudes of quantitative properties of fixed object species," it is still "ill-suited to address interactions that change the objects themselves" (p. 63). And this, of course, is precisely what characterizes the objects of biological systems, which are necessarily subject to change by the internal dynamics of the system, and not only by external insult. As they put it: "Mutation is to construction like perturbation is to dynamics" (p. 63). Fontana and Buss call their own very interesting effort to expand the traditional theory to include objects, their internal properties, their construction, and their dynamics, *constructive dynamical systems*.

4 Kant and As-If Purpose

Of course, function is not the only property that distinguishes living from non-living systems (or, as a *Nature* (2005) editorial put it, that distinguishes a live cat from a dead one), but it lies at the heart of that question of just what kind of science a science of life might be. Certainly, it lay at the heart of Immanuel Kant's (1724–1804) concern about the prospects for a science of life. "Organisms," he wrote in the second Part of the *Critique of Judgment* (1790/1993), are the beings that "first afford objective reality to the conception of an *end* that is an end of *nature* and not a practical end. They supply natural science with the basis for a teleology . . . that would otherwise be absolutely unjustifiable to introduce into that science seeing that we are quite unable to perceive a priori the possibility of such a kind of causality" (pp. 558, 66). For Kant, it was precisely here that the fundamental difficulty for a scientific approach to life is to be found: in the fact that organisms are systems that behave *as if* having a purpose, goals, ends (or functions) of their own. (It is true that the term Kant actually used was not function, but rather *Naturzwecke*, translated as "an end of nature" or "natural purpose," but I would argue that all of these terms are equally effective in capturing what was essential to his concerns.)

Kant did not believe that organisms really were teleological, but rather that we have no way of understanding them other than by thinking of them as teleological. Perhaps he should have written, "They supply natural science with the basis of an *as-if* teleology"—something totally missing from natural science as he knew it (which basically meant Newtonian mechanics). Indeed, this was the reason that he famously (or infamously) concluded that "there will never be a Newton for a blade of grass." The kind of organization instantiated by organisms cannot be obtained either from Newton's laws themselves, or from any other set of laws of that kind. As he wrote, it is "contrary to reason that crude matter on its own should have structured itself originally in terms of mechanical laws, that life could have sprung from the nature of what is lifeless, and that matter could have molded itself on its own into the form of a self-preserving purposiveness" (Kant, 1987, p. 311). Accordingly, if biology is to be a science, it would clearly have to be a different kind of science.

As Kant saw it, the difference between the life and the physical sciences lay precisely in the peculiar part–whole organization of living beings, or, as he suggested (in what was, in fact, the first use of the term), in their "self-organization":

> In such a natural product as this every part is thought as *owing* its presence to the agency of all the remaining parts, and also as existing *for the sake of the others* and of the whole, that is as an instrument, or organ . . . the part must be an organ *producing* the other parts, each, consequently, reciprocally producing the others. . . . Only under these conditions and upon these terms can such a product be an *organized* and *self-organized being*, and, as such, be called a *physical end*. (Kant, 1987, p. 557)

An organism, in short, is "an organized natural product is one in which every part is reciprocally both end and means. In such a product nothing is in vain, without an end, or to be ascribed to a blind mechanism of nature" (p. 558). In fact, Kant does not exclude the possibility that such products are intrinsically mechanical, but only the possibility that we can ever fully explain them in terms of blind mechanism, given the limits to our powers of judgment. Which is not to say that we should not try, but only that we must accept our own cognitive limits. As Alix Cohen (2007) explains:

> It is reasonable, even praiseworthy, to try to explain natural products in terms of natural mechanism as long as there is some probability of success. Indeed, if we give up this attempt, we must not do so on the ground that it is *intrinsically* impossible to find the purposiveness of nature by following this route, but only on the ground that it is impossible *for us* as human beings. (p. 115)

Indeed, Kant's argument is that it is precisely because of our limitations that we must use the concept of teleology in our investigations of biological systems.

This work *Critique of Judgment*, written in 1790, might well be regarded as the first philosophical treatise on systems biology, even though that term never appears as such. Nor does it appear in any of the many research efforts that Kant's discussion inspired over the next half-century, especially in Germany. What these efforts shared was a commitment to finding a way of integrating teleology and mechanism in the analysis of biological systems, qualifying in Tim Lenoir's (1982) view as a research tradition that he dubbed "teleo-mechanist." But whatever the successes of this tradition, their unifying goal soon fell out of favor, effectively drowned out by the increasing polarization between mechanistic science and a purposeful divine architect, on the one hand, and by Darwin's great success, on the other. Indeed, even Kant's essay on teleological judgment, the second part of his *Critique*, fell into neglect. But with the rise of systems biology over the last few years, it is hard not to hark back to this earlier tradition, and, more specifically, to the concerns of Kant's inquiry.

5 Cybernetics and Bernard Machines

The chief difference between then and now, between Kant's despair of ever being able to provide a mechanistic account of natural ends, and the optimism of our own time, came out of the development of mechanisms that promised a self-directed

functioning, in the middle of the twentieth century. "Out of the wickedness of war," as Warren Weaver (1948, p. 540) wrote shortly after the end of World War II, emerged not only a new machine, but also a new vision of a science of the inanimate: a science based on principles of feedback and circular causality, and aimed at the mechanical implementation of exactly the kind of purposive behavior that so worried Kant. I am referring to the birth of *cybernetics*, a science that would repudiate the very distinction between organism and machine on which Kant's concept of self-organization was predicated.

Certainly, the early vision associated with cybernetics promised far more than it delivered, conspicuously failing to fill the gap between self-piloting aircraft and living organisms. Nevertheless, since then, appreciation of the central role of feedback in the design (or organization) of functional units has never flagged and, in recent years, this concept has come to play a more prominent role in the analysis of regulatory systems in biology. It seems to me that contemporary methods of analysis of feedback in cybernetics just might be able to fill that critical gap, providing a mechanistic (i.e., physical and chemical) account of, at least, some cases of biological function.

Scott Turner (2007), borrowing from his colleague Cosma Shalizi, has recently introduced a felicitous term into the literature that I believe will serve well for the minimalist conception of function I proposed above. Turner is interested in "agents (or organs) of homeostasis" and, referring to the famous physiologist most associated with homeostasis, Claude Bernard (1813–1878), he calls these agents *Bernard machines*. Bernard machines are devices for creating (and maintaining) a fixed milieu either inside the organism or immediately surrounding it; they are the mainstay of physiology, or, rather, they are embodiments of physiology.

In terms of my earlier discussion of function, a Bernard machine is exactly what is required to turn the rain–cloud–river cycle into a system with function; a Bernard machine is the mechanism that introduces function into such a system. Such devices are ubiquitous in biological systems, yet extremely complex from an evolutionary point of view (even when considered simple from an engineering perspective). Furthermore, and clearly attesting to the power of contemporary systems biology (especially of those efforts that proceed, simultaneously, from the bottom up and the top down), there are now a number of examples in which a naturalist account of such mechanisms is either available or close to available. I have chosen two cases to illustrate.

My first case is bacterial chemotaxis, the ability of bacteria to sense and move toward an attractant (e.g., a nutrient), or away from a repellent (e.g., a toxin). Over the last few years, biologists have unraveled virtually the entire structure of this system—indeed, bacterial chemotaxis might be said to be a poster child of systems biology. The machinery that has evolved to make chemotaxis possible, itself, is built out of relatively sophisticated, and pre-existing, structures, the main components of which are the flagella (the motor system) and the chemical receptors (the sensors). The paradigmatic example is *E. coli*, where each bacterium has several flagella, capable of rotating clockwise and counter-clockwise. Given the helical structure of the individual flagellum, counter-clockwise motion has the effect of aligning them into a single rotating bundle, leading to motion in a straight line; conversely, clockwise rotation disrupts the bundle, causing the bacterium to tumble in place. The net

effect is a random motion of alternating *straight swims* and *tumbles*. But the addition of a simple feedback between the receptors and flagella that regulates the direction of rotation according to chemical concentration leads, straightforwardly, to directional motion.

The actual system of feedback that *E. coli* employs is at least one step up from the simplest possible. In particular, it employs a mechanism allowing temporal comparison of concentrations, linked to the motor system in such a way that the time between tumbles is reduced if the bacterium is going in the "right" direction, thereby leading to a more direct chemotactic response than would otherwise have been possible. Over the course of evolution, still further feedback loops have been added, resulting in an extraordinarily robust and efficient device for maintaining a favorable environment for the modern bacterium—an environment free of toxins in the case of negative chemotaxis, or rich in nutrients in the case of positive chemotaxis.

This is a Bernard machine *par excellence*, but it is one that regulates the environment of the bacterium by moving the organism until its local environment is improved, rather than by directly changing that environment (as, for example, a thermostat would). It works by means of a hierarchy of feedback loops between the sensory and motor system that are now well-understood, and that have been effectively modeled in terms of the chemical and physical processes involved. How such a machine came to be built, however, is another question, and that process is not well-understood. Yet it can safely be said that it, too, depended on a kind of feedback; more specifically, on feedback between variations in chemical processes, changes in the physical, material environment these variations lead to, and shifts in the selective pressure operating on the chemical innovations caused by the changed physical environment. Here, function is built, as it were, by a kind of evolutionary transduction of the interactions between chemical reactions and the physical dynamics of the spatial-temporal-material medium in which these reactions occur.

My second example of a Bernard machine is one of Turner's own examples: the mound, built by a colony of termites that serves as a wind tunnel, regulating the level of CO_2 (and oxygen) in the termite nest. Here, the environment of the colony is maintained at a more or less constant level of comfort, not by moving the colony, but by the work of individual termites in constructing a physical device that regulates the flow of wind, and that, by so doing, works to stabilize the temperature and concentration of gases in the nest. Unlike the machinery of chemotaxis, this mechanism is not part of the body of the individual organism, but rather part of the organism's environment. Equivalently, it might be said to be a component (indeed, a necessary component) of the extended body of the colony. Without it, the colony could not survive.

Here is the problem the termites face: A typical colony begins with a single queen and king, housed under the surface of the soil, with impressive reproductive powers. In a short time, the colony reaches a population of approximately a million termites, with the respiratory requirement roughly that of a rabbit (Turner tells us that when you add in the respiratory needs of the accompanying fungi, the respiratory need of the colony approximates that of a goat). Yet, throughout this explosive growth in population, the atmosphere of the nest—viz., the temperature, concentrations of O_2 and CO_2—remains quite stable. The question is, "How is this accomplished?"

Turner answers the question as follows. The behavioral repertoire of the individual termite includes two actions: picking up grains of soil and gluing them down. Which action is performed depends on CO_2 level, with a high probability of picking up the grain of soil when the level of CO_2 is high concentration, and of gluing it down at low concentrations. In other words, just as with the bacteria undergoing chemotaxis, each termite is equipped with a sensor that is coupled to its behavior. The link between the levels of CO_2 recorded by the sensor, and the activity of the termite, guides the construction of a mound in which the rate of air circulation is effectively matched to the metabolic needs of the colony. The mound, one would have to say, has a function, namely, to maintain the atmosphere of the nest at comfortable levels. We might call this an *atmospherostat*, built not by intentional agents, but by mindless termites who know only how to pick up and deposit grains of dirt at rates that depend on the concentration of CO_2. But this simple sensor–behavior link in the individual termite, no more complex than that found in the bacterium, leads, on the level of the colony, to an ongoing and apparently purposive rearrangement of the soil between the nest and the open. Indeed, the net result is the construction and continual reconstruction of a mound that, at any given moment, operates as a well-designed wind tunnel adapted to the respiratory needs of the colony.

Once again, the presence of feedback loops is of key importance. The physical structure of the mound determines (feeds back on) the flow of air through the mound (the air, of course, includes CO_2), the distribution of CO_2 determines (feeds back on) the activity of the termites in their subsequent building activities, and the activity of the termites results in higher levels of CO_2. The net result of all this feedback is a regulatory system of air–mound–termites with exactly the same logic as that of the thermostat in your living room, with the striking difference that no engineer was required to either design, build, or install the termite mound.

Our understanding of the functioning of this Bernard machine is less well-developed than that of bacterial chemotaxis, but the broad outline is clear. And here too, nothing as yet has been said about the origin (or evolution) of such a machine. Yet, it seems safe to presume the operation of the same kind of feedback as I hypothesize for the evolution of the mechanism for bacterial chemotaxis, namely, between: variations in chemical processes (genetic mutations); changes in the physical, material environment to which these variations lead; and shifts in the selective pressure operating on the chemical innovations caused by the changed physical environment. Here too, I suggest that the coupling between sensor and behavior required for function emerged by evolutionary transduction of the interactions between chemical reactions and the physical dynamics of the spatial-temporal-material medium in which these reactions occur.

6 A Guarded Optimism

Still, we are not home free. At best, the examples I offer provide evidence of partial successes in the effort to "reduce biological explanations to explanations in chemistry and/or physics." Indeed, they reveal an ambiguity in the very question. It is one thing to explain how chemotaxis works, or how termites are able to construct wind tunnels that regulate their environment in terms of chemistry and physics, but quite

another to explain how such complex and exquisitely organized systems could have first arisen. Or, as Kant put the question, how "crude matter on its own should have structured itself originally in terms of mechanical laws, that life could have sprung from the nature of what is lifeless, and that matter could have moulded itself on its own into the form of a self-preserving purposiveness." I have put forth some very general suggestions, but the task of constructing concrete models that would actually be capable of generating such devices is enormously difficult, and efforts appropriate to the task have only begun. Such efforts would have to take into account processes of self-assembly and self-organization in multi-level systems, operating on multiple spatial and temporal scales, in which (as Fontana and Buss have emphasized) the internal structure and properties of the component elements are themselves responsive to the dynamics of the system.

Yet, it seems to me that the task is now defined with sufficient clarity to support some optimism. What kind of explanation such models will yield, whether it would fall within the range of human cognitive capacities to grasp, or whether it would require reliance on computers that are so much better able than we are to handle all this complexity—these, of course, are other questions altogether. It is just possible that, in the end, if and when we succeed in "reducing" biological explanations to physics and chemistry, Kant will still have proven to be right about the relation between such accounts and human judgment.

Postscript: Counterpoint

As I expected it would be, it is difficult to find much difference between John Dupré's position and my own. But there are a couple of places that indicate some small disagreement.

First, I would resist his paraphrase of Bedau's discussion of weak emergence; rather, I would say that it is the interactions among the parts—and *not* their behavior—that (apart from stochastic input) "determine" the behavior of the whole. In the next paragraph, he refers to the "properties of the parts." But, in my view, it is important to distinguish *interactions*, *behavior*, and *properties*, especially in constructing models of complex systems.

Second, our uses of the term *downward causation* probably need distinguishing. I use it to refer to a wide range of influences that global properties have on the parts, including not only their activity (in the case of genes), but their very identity. The properties of a cell, for example, are at least partly determined by transcription of DNA but, in turn, cellular properties also determine which sequences are to be transcribed, in which combinations, and in what order. Indeed, the very definition of what (if anything) a gene *is* depends on the properties of the cell in which the DNA is embedded.

Finally—and here is probably our biggest difference—I find Dupré's distinction between *context* and *interactions* problematic. Or, perhaps it would be better to say *artificial*. For example, I agree that one might argue that chaperones can be seen as embodying yet another kind of interaction between protein, and I can see nothing wrong with such an argument. Indeed, I would suggest that what Dupré calls *context* is simply all those other factors/molecules whose interactions with the object or system in question have not been made explicit and, hence, have not been included in the description.

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CHAPTER
T W O

It Is Not Possible to Reduce Biological Explanations to Explanations in Chemistry and/or Physics

John Dupré

In this paper, I argue that the traditional notion that complex systems, such as those found in biology, can be fully understood from a sufficiently detailed knowledge of their constituents is mistaken. My central claim is that the properties of constituents cannot themselves be fully understood without a characterization of the larger system of which they are part. This claim is elaborated through a defence of the concepts of emergence and of downward causation, causation acting from a system on its constituent parts. Although much of this argument can be read as having only epistemological or methodological force, the final section of the paper defends a more robust metaphysical reading: even purely metaphysical understandings of reductionism such as are commonly represented by supervenience theses are misguided.

1 Introduction: No Need for Special Biological Laws?

Kenneth Waters (1990) has referred to the "anti-reductionist consensus" in the philosophy of biology, so it is perhaps not too surprising that I find myself in agreement with most of what Evelyn Fox Keller says. At the beginning of her paper, she says that she "could as easily have gone the other way," and I would say that, perhaps, she might better have chosen the other side. However, there are some passing statements in her paper with which I am inclined to differ, and I shall explore a few of these to see whether there may be some significant disagreement after all.

Like Keller, I am a materialist. That is to say, I do not believe there is any kind of stuff in the world other than the stuff described by physics and chemistry. There are no immaterial minds, vital forces, or extra-temporal deities. Keller writes, however, that as a materialist she is "committed to the position that all biological phenomena, including evolution, require nothing more than the workings of physics and chemistry." Even as a materialist, I'm not sure I feel committed to this; but, of course, that depends upon exactly what the title question means. A little unpacking of this question may help to reveal where (if anywhere) there is a serious difference between Keller's position and my own.

One could start with a trivial interpretation of this sentence. If "the workings of physics and chemistry" meant no more than the workings of things that were made of physical or chemical stuff, then a materialist, such as myself, could hardly deny it. But an ambiguity immediately appears in the phrase "physics and chemistry." It could be read—as I just have read it—ontologically, as referring to the things of which physics and chemistry speaks. And for a materialist, therefore, it refers to the entire material world.[1]

But on a quite different reading, one might more naturally think of physics and chemistry as scientific domains, traditions of inquiry, or suchlike. Then, it is far from trivial to claim that biology requires no more than the workings of physics and chemistry. Why should there *not* be biological workings that are quite different from those of physics and chemistry? And here, I appear to be in agreement with Keller when she denies that biology could be derived from the *theories or laws* of physics and chemistry.

As a matter of fact, much of the recent history of reductionism, as a philosophical doctrine, has addressed the relation between theories or, perhaps, laws of nature. Classical versions of the doctrine held that the relation in question was logical: laws of biology should follow deductively from the laws of physics or chemistry. Within the philosophy of biology, something that has surely received the status of a consensus is that no such derivations are plausible. One holdout against this consensus might be Alex Rosenberg (2006), who seems to suggest that the laws of physics, supplemented by the principle of natural selection, would suffice to derive the whole of biology. I discuss this position critically elsewhere (Dupré, 2007). Even ignoring the rather serious problem that, as Keller notes, there do not seem to be many, or any, laws in biology, there is a further problem that the concepts employed in different sciences seem to be incommensurable. As David Hull observed over 30 years ago (Hull, 1974), the relations between Mendelian genes and molecular genes are many/many. So Mendelian genetics, a scientific project still very much alive in medicine and agronomy, employs concepts that are incommensurable with those that pertain to the molecular entities that, in some sense, underlie the Mendelian phenomena. And subsequent developments in molecular genetics have suggested that the problems are more severe even than Hull could have known (Barnes & Dupré, 2008).

If the question of reductionism were merely a question of whether all of biology could be derived from the laws of physics, then we could confidently assert that the issue had been resolved. As I noted in the preceding paragraph, it cannot. However, this is clearly not what Keller has in mind and, despite her ambivalence on the topic, she has some willingness to be counted as a reductionist. So, again, what are we to make of "the workings of physics and chemistry"?

2 The Reductionist Principle

Suppose we are interested in the ecological system that includes lynxes and hares. Everyone can agree, I assume, that, among the constituents of this system are lynxes; and everyone can agree (or everyone I am concerned to argue with) that the lynxes are made entirely of physical stuff. There is nothing else to be made of. The first of these propositions is the one that the anti-reductionist will tend to emphasize, while the reductionist will be more inclined to stress the second. But where should we look for a definite disagreement between these opposing camps?

We have agreed that the lynx is made only of physical stuff. Sometimes this is expressed as the claim that the lynx (or anything else) is "nothing but" an array of physical parts. While this claim might be endorsed by most reductionists, even the moderate reductionists who have abandoned deductive relations between successive theories, it is liable to be treated with more suspicion by anti-reductionists. So we might try to separate the claim about constitution from the "nothing but" claim. What more is there to what a thing *is* than what it is made of?

Of course, one answer to the last question that, again, everyone can agree on is that it matters how the physical (or chemical) constituents are put together. In fact, to make a lynx they have to be put together in a stunningly intricate way; and a pile of chemicals that happened to be the very same molecules that could, properly assembled, constitute a lynx would be no more than an inert heap of stuff. So the reductionist's claim should be that the lynx is nothing but a collection of physical parts *assembled in a certain way*. So here, finally, is a proposition that we might expect the reductionist and anti-reductionist to disagree about: if we knew everything about the chemicals that make up a lynx, and the way they are assembled into cells, organs, and so on, we would, in principle, know everything about the lynx. Reductionists will generally endorse something like this, whereas anti-reductionists will deny it. Let me call this, with the specific biological system lynx replaceable by any system we care to investigate, the *reductionist principle* (RP).

An important feature of RP is, of course, the phrase "in principle." Certainly, no one knows how to explain all the properties of a complex organism in terms of the properties and arrangements of its parts; the question is whether this is simply a reflection of the underdeveloped state of our current biology, or whether there are deeper obstacles, obstacles in principle, that will continue to prevent us from doing this. The kinds of principles involved will distinguish a variety of different versions of anti-reductionism or, as it is often called, *emergentism*, the belief that there are emergent properties, properties that could not have been predicted (even "in principle") from a complete knowledge of the constituents of a thing and their internal relations. (I should note, however, that this probably still does not capture any disagreement between Keller and me, as she acknowledges explicitly the existence of emergent properties. It may be that we differ as to what they are.)

3 Strong Emergence

One conception of emergence, championed recently by Mark Bedau (2003), proposes the obstacle to explanation as the lack of a general principle connecting features of

the constituents to features of the whole, but holds on to the reductionist intuitions with the idea that a fully detailed *simulation* of the interactions among the constituents would generate the behavior of the whole. The behavior of the whole is fully determined by the behavior of, and interactions between, the parts; but the only way to get from the latter to the former is by a complete simulation. Bedau calls this kind of emergence—which shares with reductionism the insistence on the dependence of the whole on the properties of the parts—*weak emergence*. Strong emergence, in contrast, denies such dependency.

I propose, here, to defend strong emergence. That is to say, I want to deny that the behavior of the whole is fully determined by the behavior of, and interactions between, the parts. And hence, the elements of behavior that are not so determined are what we don't know when we know everything about the parts and the way they are assembled; and thus, finally, what violates RP.

At this point, we need to be rather more careful with the relations between *dispositions to behavior* and *behavior*. No one believes that the behavior of a whole is, in general, determined solely by the properties of its parts, even for the most paradigmatically mechanical systems. A properly functioning grandfather clock, say, the action of which is powered by a slowly falling weight, will not function if the weight is supported so as to disconnect its gravitational force from the action of the clock. With few, if any, exceptions the properties of parts translate into the behavior of wholes under specific circumstances. So, the most any reductionist should claim is that dispositions of the whole are determined by properties of constituents, together with appropriate surrounding conditions. With the important qualification that these dispositions may be probabilistic rather than deterministic, let me, for the sake of argument, concede this much. Have I, then, conceded what is important about reductionism? In earlier work, I have distinguished further between the case in which probabilistic dispositions involve determinate probabilities of specific behavior, and the case in which the behavior is possible, but in which there is no reason to think that it occurs with any determinate probability (Dupré, 1993, Ch. 9). As a matter of fact, I see no reason why the second case should not be characteristic of much that happens, an idea to which I shall return briefly and tangentially in the conclusion to this paper.

One way of glossing the previous remarks about context is to note that many, at least, of the dispositional properties that appear to fall within the range of the preceding discussion are relational. An elephant gun has the capacity to kill elephants. This is a property that depends on many features of the gun and many features of elephants. One could deprive the gun of this capacity by fitting all elephants with suitable armor plating or, indeed, by killing them all so that nothing any more has the capacity in question. But it is natural to think that there is an intrinsic capacity of the gun, the capacity perhaps to project a lead pellet of a particular mass at a particular velocity in a determined direction, that underlies the relational capacity. Relational capacities of a thing are quite obviously not reducible to any information about the parts of that thing alone, since they depend also on features of the other party to the relation. But the reductionist might reasonably propose that all the intrinsic properties of the thing are reducible to properties of the parts, and that the relational properties were deducible from a knowledge of the intrinsic properties of the things related. The hardness of an elephant's skin, the distance from the skin to organs

necessary for the maintenance of life, and so on, combined with the intrinsic capacity of the gun to project a leaden missile, together entail the capacity of the gun to inflict fatal damage on elephants.

So, here is a possible ontological picture for the reductionist. Imagine arranging entities in a traditional ontological hierarchy: elementary particles, atoms, molecules, cells, organisms, etc.[2] At each level of the hierarchy, we can determine a set of intrinsic properties of the relevant entities. From the intrinsic properties of entities at one level, say atoms, and the relations between the atoms, we can infer the intrinsic properties of molecules. Thus, ultimately, the intrinsic properties of everything are consequences of the intrinsic properties of their constituents and, ultimately of their smallest (physical) constituents. Or anyhow, this will follow as long as we can take care of the relational properties smuggled into the story. (Keller probably would not endorse this reductionist picture. For she writes that biological explanations assume "the dependence of the identity of parts, and the interactions among them, on higher-order effects." As will emerge very shortly, I take this to capture a fundamental deficiency in the reductionist picture.)

4 Complex Relations in Biology

Unfortunately, taking care of the relational properties is not an easy matter. Sometimes these are simply a matter of location, as with the relative positions of the elephant and the gun, and spatial relations presumably belong comfortably in the realm of the physical. But many relational properties in biology are not that simple. Consider, for example, the characteristic properties of enzymes. An enzyme is a catalyst, generally a protein but in some cases an RNA molecule, which facilitates a generally highly specific biochemical reaction in a cell. The mechanisms by which many enzymes work are well-understood and involve a variety of spatial and electrochemical interactions between the enzyme and its substrate. Enzymes typically have an active site, a small part of an often very large and complex protein, which binds to the substrate and changes its spatial configuration or electrical charge pattern in a way that reduces the activation energy of the reaction the enzyme catalyzes.

We may, no doubt, assume that intrinsic properties of the substrate and the enzyme are sufficient to explain the capacity of the latter to act as a catalyst on the former. However, the intrinsic properties of a large and complex molecule such as a protein will very likely allow it to catalyze many different reactions. And as a matter of fact it has become clear that many proteins do, in fact, have multiple functions. Ramasarma (1999) lists over 50 proteins (or groups of closely related proteins) with more than one known function (also see Jeffery, 1999). These alternate functions include a range of activities in addition to serving as enzymes, such as binding or transporting various molecules, inhibiting various cellular processes, or forming subunits of larger proteins. Glyceraldehyde-3-phosphate dehydrogenase (GAPDH), for example, a common "housekeeping" enzyme, is believed to act as an acyl phosphatase, an esterase, a protein kinase, and a Uracil-DNA glycosylase, in ADP-ribosylation, microtubule-binding, t-RNA binding, amyloid protein binding, and membrane binding (Ramasarma, 1999). The number of *possible* functions of a protein molecule seems, in principle, quite indefinite.

Hence, finally, a complete knowledge of the physical and chemical properties of a protein will certainly not tell us what a protein does. When we know what the protein does, chemistry may certainly be able to explain how it does it; but that is a different matter. The distinction between explaining *how something does what it does* and explaining *what it does* was central to my earlier critique of reductionism (Dupré, 1993). The idea is developed further by Pigliucci and Kaplan (2006).

Note, also, that it is common practice to say that a protein *is* an esterase, a protein kinase, etc. In fact, the primary name of the protein just mentioned, GAPDH, indicates its role in catalysing the transformation of glyceraldehyde-3-phosphate; most proteins are named by reference to (one of) their functions. But being a GAPDH, a molecule with that particular catalytic function, requires not merely a particular chemical structure, but an environment in which there is glyceraldehyde-3-phosphate to transform.

I take it that the point I have just been trying to make is part of what Keller means when she writes of "the dependence of the identity of parts, and the interactions among them, on higher-order effects." But this dependence points, I believe, to a fundamental *objection* to reductionism. Chemistry, alone, cannot tell us that a particular protein is a GAPDH rather than one of the countless other functionally defined things it might have been. To be a GAPDH requires, in addition, an environment that includes the other elements that make the performance of its specific function possible.

The point can perhaps be more intuitively illustrated by thinking of the quite different case of human capacities. As is most definitively argued in Wittgenstein's *private language argument* (1953/2001), the ability most humans have to speak a language is one that would be impossible, in principle, without the existence of a linguistic community of which they were part. Countless human capacities—to write a check, make a promise, play chess, and so on—depend for their possibility on the existence of a social context in which conventions or rules create the conditions for such activities. No amount of knowledge of my physics, chemistry, neurophysiology, or the like, could determine whether I was able to write a check. But the point also applies, in important ways, to less socially embedded physical activities. My physical properties do not determine, for example, whether I am able to move vertically through buildings. If my legs function in the standard way, my ability to do this will depend on the availability of staircases. If I use a wheelchair, my vertical mobility will depend on the provision of ramps or elevators. Again, the capacities of a thing, as opposed to the countless merely possible capacities, can be seen to depend on the relationship between the thing and the environment in which it exists.

It may seem that by conceding that the actual capacities—the capacities that become actual rather than merely possible in an appropriate environment—can be explained in physical or chemical terms, I have conceded everything the reductionist really cares about. Certainly, I hope I have conceded what is necessary to account for the extraordinarily successful practices of scientists engaged with molecular aspects of biology. Here, however, it is useful to recall a banal point, though one that occasionally gets overlooked in such discussions, that we are talking about science, not Nature. Biological explanations are part of biology, not part of the world, and biology, like any other science, is an articulated conceptual structure and not a repository of things-in-themselves. I shall move into metaphysics and attempt to say a little about Nature at the end of this paper, but, for now, I shall continue to address science. And the

fact that biology—a science—works with *concepts* that depend on the larger systems of which they are part, as well as on their constituents, is a fatal objection to the claim that "it is possible to reduce biological explanations to explanations in chemistry and/or physics."

5 A Misinformed Slogan and Its Contributions

The preceding point can be developed, as well as some of the main dangers of reductionist thinking illustrated, by considering the view that the genome contains *all* the information required to build an organism. In fact, this view is still too-often promulgated by scientists who should know better, and widely asserted in popular science writing. Probably not a lot of people, biologists or otherwise, who have thought seriously about such things still believe this. However, a close look at why it is so deeply mistaken will be a useful way of elaborating the difficulties with reductionism. I won't dwell too long on the tricky concept of information. As developmental systems theorists, in particular, have pointed out for some time, the concept faces an impossible dilemma (an excellent introduction to development systems theory is Griffiths & Gray, 1994; Oyama, 1985, is the *locus classicus*). If it is interpreted in the everyday semantic sense, then it is obviously false. Genomes contain meaning only in a highly metaphorical sense. But if it is interpreted in the technical sense of information theory, according to which, very roughly speaking, a source conveys information about a target when it reduces the uncertainty about the state of the latter, then there is no sense in which the genome carries information that does not apply equally well to everything else that is necessary for features of the genome to have their normal effects on the cell. Without a complete transcription mechanism, for instance, the genome carries no information; and taking the genome as part of the channel through which the information flows, the transcription machinery carries information about the same targets. In what follows, I shall occasionally use the concept loosely to refer simply to casual determination.

But this rather technical issue points directly to a more fundamental difficulty even with less pedantic interpretations of the idea under discussion. Strings of DNA, or even real genomes replete with histones and other structural elements that make up real physical chromosomes, do nothing on their own. Their involvement in the production of proteins is as part of a system that includes a very large number of additional molecules and cellular structures, and although there are very special and biologically important features peculiar to nucleic acids, singling them out in the way suggested by the reductionist slogan makes no sense. There are several simplistic ideas that contribute to the continued popularity of the slogan, however, and I shall briefly discuss three of these.

The first is what was named by Francis Crick (1958), presumably with a trace of irony, the *central dogma of molecular biology*. According to the dogma, information flows from DNA, to RNA, to protein sequence, but never in the opposite direction. Although one should question whether merely specifying a list of amino acid sequences that constitute proteins would be sufficient to specify the entire organism, the doctrine that DNA is only a source of information, entirely immune to the influence of signals from its cellular environment, provides a powerfully reductive

perspective on the economy of the cell. But, at any rate, interpreted this way the dogma is entirely misguided. Apart from its dependence, already referred to, on the transcription mechanism that produces mRNA sequence from DNA sequence—not to mention the mechanisms including complex cellular structures, ribosomes, that facilitate the production of proteins in accord with mRNA sequence—the behavior of the genome is affected by countless other molecules in the cell. It does certainly remain the case that the important activity of the genome is providing a template for transcription of RNA molecules, but which RNA molecules are transcribed and in what quantities is dependent on interactions with many other constituents of the cell, and the actual structure of the DNA in the genome is constantly being modified by these other constituents. Information, then, flows constantly to the genome from RNA and protein molecules.

The importance of RNA molecules in modifying genome behavior is a field still in its infancy, but one that is thought by many to be likely to revolutionize cell biology. A second simplistic view that is relevant here is the idea, once widely cited, that most of the DNA in the genome, perhaps up to 98% of it, is "junk." It has been realized for some time that only a very small proportion of the DNA in the genome provided sequence that ended up translated into protein structure. In accordance with the central dogma, it was concluded that most of the rest lacked any function at all and that, perhaps, its presence reflected no more than competition for space in the genome among genetic elements that played no part in the functioning of the organism (Dawkins, 1982). However, it now appears that the large majority of the genome, at least 70%, is transcribed, and as more is understood about the variety of RNA molecules in cells and their diverse functions, it becomes increasingly imprudent to assume that these RNAs—and, therefore, the genomic DNA from which they derive—may not play essential roles in the economy of the cell.

At this point it might be said that, even acknowledging that the function of the genome is affected by numerous RNA and protein molecules, the latter are derived from genomic DNA sequence, so that all that has been added to the idea of the DNA as controlling molecule is a few feedback loops. Everything still begins with the genome. This leads to the third simplistic view I want to mention. Sometimes, it is imagined that all that passes from one generation to the next, through reproduction, is the genome. Clearly, this presupposes the idea that the genome contains all the information necessary to build an organism, since the organism is built, and nothing is there to build it from but the genome. But this view is quite wrong, as well. The smallest "bottleneck" in the developmental cycle is a single cell, the zygote. And this cell contains all the machinery necessary for the functioning of the DNA, and the rest of a normal cellular complement of molecular constituents and subcellular structures. The cell is an evolved structure which, far from being assembled through instructions contained in the DNA, is a product of several billion years of evolution. Naturally occurring DNA is always and everywhere found as part of such exquisitely complex evolved systems.

This brings me back to the central point that occupied the earlier parts of this paper. The capacities of DNA are not merely consequences of its molecular constitution, but are simultaneously determined by the systems of which DNA molecules are part. The best way to illustrate this point is by considering the ways that we divide the genome into functional parts, genes.

6 Genes

The concept of a gene has two rather different traditions of use. Its origin is in the breeding experiments, especially on fruit flies, of the first half of the twentieth century, though the classic experiments of Mendel in the 1860s are generally considered canonical precursors of this work, and this research tradition is often referred to as *Mendelian genetics* (Sturtevant, 2001). In this context, the gene was a theoretical term used to track inherited features of the organism under study. The "gene for red eyes" was the hypothetical cause of a pattern of inheritance of red eyes. Flies with red eyes were assumed to possess this gene, and this, through Mendelian patterns of inheritance and theories of dominance and recessiveness, explained the quantitative characteristics of the pattern. Such a gene made the difference between having and not having a particular trait, in this case a particular eye color, and it is an important point about this research tradition that genes were always, and only, difference makers. Where a kind of organism showed no variation in a trait, there were no genes for that trait to investigate.

It is uncontroversial that, in this tradition, genes were what they were only in a very complex context. Even a quite deterministic view of the action of genes would need to allow that only deep within the body of a fly would any molecular entity actually make a difference in eye color. The same entity (if indeed it is an entity) might appear in another organism with a quite different effect, or indeed might appear as a production stage in a chemical factory making parts for organic computers. Being a gene for red eyes is very far from being an intrinsic feature of a bit of chemical.

Of course, the reason for this is clear enough: we have identified the gene in terms of its effect in a much larger system. Nowadays, we are inclined to think of genes rather as sequences of nucleotides, and conceived that way surely they are simply chemical objects. And indeed, we might suppose that the genes for red eyes and suchlike could now be identified with sequences of nucleotides describable quite independently of the biological context in which they appeared. As a matter of fact, however, this turns out not generally to be possible. One of the major areas in which Mendelian genetics remains a thriving tradition is in the medical genetics that addresses single gene disorders. A standard example is cystic fibrosis, which is caused by a recessive gene with very serious health consequences. However, there is no particular sequence that corresponds to the gene for cystic fibrosis. Rather, cystic fibrosis results from a large range of mutations in a certain transcribed DNA sequence (known as the *cystic fibrosis transmembrane conductance regulator gene*) any of which render the gene incapable of producing a functional product. If both copies of the gene are dysfunctional, the disease state ensues. Currently, over 1,000 such mutations have been catalogued. But not every possible mutation of this stretch of the genome will render the gene dysfunctional, and in fact the severity of the disease will vary according to the precise mutation. So, whether a piece of DNA sequence is a cystic fibrosis gene is determined only by how it functions in the entire organism.

It might be supposed that things would be much more straightforward from the perspective of the second tradition, molecular genetics. In this research tradition, which developed out of the identification of DNA as the material out of which genes were made and the resolution of the chemical structure of that molecule, genes are generally identified as specific sequences of nucleotides. Surely being such a

sequence is something that occurs quite independently of any context? I have already explained part of the problem with this thought. Insofar as molecular genes are identified as templates for particular proteins, and proteins are distinguished by (one of) the specific function(s) they carry out in the cell, then the identity of the gene, as it is conceived as a gene with the function of providing a template for a particular protein, is, again, tied to a molecular context. And it is under some such description that we are motivated to distinguish a particular sequence as a discrete entity, a gene. But the difficulty goes deeper than this, arising from fundamental limitations to the gene concept itself.

In the early days of molecular genetics, and nicely encapsulated in the central dogma, it was supposed that genes were discrete DNA sequences that specified precisely the amino acid sequence for a particular gene product. Subsequently, however, things have proved far more complicated. First of all, it was discovered early on that genes (in eukaryotes, anyhow, the division of life that includes, among other things, animals such as ourselves and plants) were not typically unbroken coding sequences, but parts of the coding sequence (known as *exons*) were interspersed with non-coding parts (known as *introns*). The whole sequence, exons and introns, is transcribed into RNA; but, subsequently the introns are excised by further processing machinery. Subsequently it was discovered that in many (probably most) genes this excision process could be carried out in different ways, resulting in different RNA products, so-called *alternative splicing*. These RNA products were also liable to undergo further "editing," alteration of details of their sequence, prior to being translated into amino acid sequences. And these amino acid sequences might subsequently be assembled into a variety of different functional proteins. So, different and discontinuous parts of the sequence, initially thought to be a gene, might end up in a range of different functional proteins.

And this is not the end of the relevant complications. The early picture had discrete genes, separated by non-coding (either regulatory or "junk") sequences in a reasonably orderly sequence. But it now seems that coding genes may overlap one another; they may be embedded within the intron of another gene; and they may sometimes be read in both directions, as so-called sense and anti-sense genes, and a particular sequence might be part of both a sense gene and an anti-sense gene. A particular part of the genome might be part of several different genes. In fact, it now seems that something like 23,000 "genes"—in the sense originally assumed in molecular genetics—are involved in the production of perhaps as many as a million proteins. It is hardly surprising that the process from the former to the latter is not straightforwardly linear.

I have presented these complications in terms of genes, but the fact is that they raise serious worries as to whether there is really a coherent concept of the molecular gene at all. The philosophers Paul Griffiths and Karola Stotz carried out a research project in which they presented scientists with DNA sequences involved in various complexities of the kinds just described, and asked them how many genes there were in these sequences (Stotz & Griffiths, 2004; Stotz, Griffiths, & Knight, 2004). There was little consensus as to the right answer. The best way to understand this finding is certainly open to debate, but my own view would be not that it showed that there were no such things as genes, but that distinguishing part of the genome as a gene only makes sense in relation to some function that particular bit of sequence serves

in the general functioning of the cell. There is, therefore, no objectively unique division of the genome into genes. Again I conclude, the conceptualization of the genome, as an object of study and as divisible into discrete functional constituents, requires that it be placed in the wider *context* with which it interacts.

7 Causation

We can also approach the question of the reducibility of the biological by looking at intuitions about causation. One of the intuitions underlying reductionist thinking is that, whereas it is natural to think of parts of an entity as causally explaining the behavior of the whole, the reverse, causal explanation of the behavior of the parts in terms of features of the whole, so-called *downward causation*, is somehow considered mysterious. (Downward causation has been the subject of philosophical debate for some time, generally dated from a proposal of Donald Campbell [1974). Interest has been greatly increased recently in the context of systems biology, of which more below.) So it seems natural to explain, for example, the movement of my arm in terms of a series of biochemical processes leading to the contraction of bundles of fibers attached to parts of the skeleton. This constitutes a classic causal/mechanical explanation in terms of pushes, pulls, hinges, and suchlike. It seems strange to many, on the other hand, to suppose that the whole organism of which the muscle tissue is part could somehow cause the necessary molecular activities. Of course, philosophically untutored intuition may find the second possibility quite natural. The naïve explanation of my arm's going up is that I intended to reach for a book, say, which explains the bodily movement in terms of a feature of the whole, its intention. If the whole person is capable of raising the arm, and raising the arm is caused by (among much else) calcium being pumped into the sarcoplasmic reticulum, then it appears that the person is capable of causing calcium to be pumped into the sacroplasmic reticulum.

Downward causation seems a very natural way to think of much of what I have been saying about molecular biology. What causes the human genome to behave in the particular ways it does—for example, various sequences being transcribed or not at varying rate, changes in conformation and spatial relation of chromosomes, and so on—is a variety of features dispersed over the surrounding parts of the cell. The behavior of the part is to be explained by appeal to features of the whole.

Another example that fits naturally into this picture is the phenomenon of protein folding. A major problem in molecular biology is to explain the transition from an amino acid sequence to the baroquely complex structure that results as this sequence folds into a three-dimensional shape. The topology of this structure is essential to the proper functioning of the protein, yet in many cases it appears to be strongly underdetermined by the chemical properties of the links between successive amino acids. It is known that many proteins require specific collaboration from other proteins, known as chaperones, to accomplish this complex feat. One might argue that this was simply another interaction, between the folding protein and the chaperone, fully compatible with a traditional reductionist perspective.

However, even if, as is probably a great oversimplification, interaction with the correct chaperones were all that was required for correct folding, the kind of argument considered with regard to protein function applies equally here. There is a very specific environment, in this case one replete with appropriate chaperones, which endows the amino acid sequence with the capacity, or disposition, to fold in a particular, functionally desirable, way. Still more is it a specific environment that disposes the various relevant parts of the genome to produce, in the end, an appropriately folded protein. And again, this environment is not something that could possibly be generated *de novo* by the genome but, on the contrary, it is one that took a few billion years to evolve. The cell, I think we must say, with all its intricate structure and diverse contents, is what causes these contents to behave in these life-sustaining ways (Powell & Dupré, 2009).

8 Systems Biology

A scientific development that has brought these issues of downward causation, emergent properties, and reductionism to the fore is the rapid growth of *systems biology*. Systems biology can be seen as a response to the growing realization that the accelerating avalanche of molecular data from ever-faster gene sequencing and comparable technologies for assaying RNAs and proteins had not been matched by similar growth in the ability to assemble these data into adequate models or explanations of larger-scale phenomena. Systems biology was conceived as a collaborative effort among molecular biologists, mathematicians, and computer scientists to attempt to provide such integrative understanding. In earlier work (O'Malley & Dupré, 2005), my collaborator and I distinguished between top-down and bottom-up tendencies in current systems biology. The former, generally reductive tendency hopes to build up more global understanding by gradual integration of information from molecular censuses and knowledge of molecular interactions. Top-down systems biologists doubt whether this can be done, and insist on the need for more general principles that emerge at higher levels of organization, and constrain the behavior of constituents.

This is a relatively crude dichotomy, of course, and the consensus among biologists involved in these projects is that some combination of the two will be needed for systems biology to succeed (Krohs and Callebaut [2007] offer criticism of the dichotomy just mentioned). This is exactly what should be expected in the light of the preceding discussion: the capacities of parts will require explanation through reductive, bottom-up approaches; but a top-down approach is required to understand their actual behavior, and to identify the capacities that need to be explained. However, whereas some top-down systems biologists hold that this is a matter of identifying laws that govern complex systems, my own prejudice is that the top-down part is more a matter of higher-level description of particular systems. One cannot infer the behavior of a cell by treating it as a bag of chemicals; one might begin to make progress by describing the intermediate structures – viz., ribosomes, Golgi apparatuses, and so on – and the heterogeneous distribution of various molecules in relation to such things.

9 Metaphysical Coda

Reductionism is inspired both by observations of the methods of science and by more purely metaphysical reflections or intuitions. Though, as I have argued, I don't doubt that reductionist methods have a vital role in science, I think this role can be overstated, especially in biology. A science such as molecular biology tells us not only how particular entities come to have the complex capacities they do, but also how complex systems enroll some of these capacities to create stability, order, and function. In doing so, I have suggested, those systems constrain and causally influence the behavior of their molecular constituents.

An influential movement in recent philosophy of science has attempted to describe biological systems in terms of mechanism (e.g., Bechtel, 2006; Machamer, Darden, & Craver, 2000). I am generally sympathetic to this movement, and these accounts have strong parallels, for example, with the view of top-down systems biology mentioned at the end of the preceding section. Although these recent accounts of mechanism do not rest a great deal on the implicit parallel with machines, this parallel does have serious disadvantages, as well as some virtues.

On the positive side, machines, like organisms, exploit capacities of their constituents to create order and predictable behavior. But there are important differences. The machines we construct typically have a fixed set of parts, and those parts are invariably subject to decay and failure over time. Organisms, in contrast, constantly renew and replace their parts, often with different ones. Organisms have life cycles; machines have only a linear progression toward decay.

However, both machines and organism illustrate one very important point. Order is difficult to achieve. Machines achieve it with all kinds of ingenuity, and auxiliary devices that anticipate, and sometimes prevent, the common causes of failure. Organisms maintain order with stunningly complex arrays of interacting parts, the "Bernard machines" eloquently described in Keller's paper, and much else besides. Also, I maintain, these order-preserving systems work by creating synergies of mutual determination between different levels of organization. Although this last point may indicate a fundamental difference between machines and organisms, there is a crucial point in emphasizing their similarity, namely, to indicate the dubiousness of an intuition that underlies much reductionist thinking. This is the idea that order is everywhere, i.e., that everything is determined by the unvarying capacities of microscopic constituents; and it is not at all borne out by a close study of the systems that do manage to maintain order and predictability. Biological order, I argue, is the extraordinary achievement of systems honed by billions of years of evolution. It is not something that comes for free with the determinism of the physical and chemical worlds.

Throughout this paper, I have been concerned to engage with reductionism as a serious aspect of scientific methodology. I have tried to produce an account that does justice to the undoubted importance that working scientists attach to reductionist methods, while avoiding philosophical conclusions that go beyond what such a concern requires. However, a great deal of philosophical discussion involves much weaker notions that have no such connections with scientific methodology, actual or even imaginable. Perhaps the most widely discussed, and certainly of no threatened relevance to science, are various theses of "supervenience," a form of reductionism

sometimes considered so weak that any sane person must accept it. Indeed, supervenience is often thought to be a paradigmatic form of *anti-reductionism*. A thesis of the supervenience of the biological on the physical asserts that, however inaccessible are principles connecting lower levels to higher levels, nevertheless, the biological depends on the physical in the sense that for any biological system there is a physical state that constitutes it, and wherever we were to find an identical physical state we would find an identical biological state.

It should be obvious what my worry with such a position will be. Perhaps this would be true for any closed biological system, but then there are no closed biological systems. This is one way of understanding the dependence of the identity of biological entities on context that I have emphasized in this paper. Bounded biological systems do not supervene on their physical parts because aspects of what they are depend on the context with which they interact, a context always extending beyond any predetermined boundaries. Perhaps I should concede that everything in the universe supervenes on the total physical state of the universe? Perhaps. But, here, we are so deeply into the domain of purely speculative metaphysics that I am more than happy to remain agnostic.

Postscript: Counterpoint

I still find the differences between myself and Evelyn Fox Keller to be slight. I shall make some very brief responses to the points that Keller raises in the postscript to her paper. While I agree that "it is important to distinguish *interactions*, *behavior*, and *properties*," the properties with which I am most concerned are capacities, and capacities, I take it, are defined in terms of the behavior that is their characteristic exercise. Moreover, capacities, I argue, are jointly determined by intrinsic features of an entity and by features of its environment. So though interactions, behavior, and properties should be distinguished, they are, I would argue, intimately connected.

On downward causation, I cannot see anything in Keller's postscript with which I disagree. It may be that something in the main body of my text gives a contrary impression, but I am unable to see the disagreement. I am, of course, happy if there is none.

Finally on the question of context and interaction, I agree that there is no absolute or objective distinction here. However, it seems to me that there is a useful pragmatic distinction. I have used the word "context" to refer to features of an object's environment that are necessary to confer on the object a particular capacity, as just explained. Interactions are simply the exercise of such capacities with relation to some other entity that will presumably constitute all or part of that context. So clearly there is no way of saying of a particular feature of the environment that it is objectively part of the context rather than something with which the entity interacts. However, I would say that the project of characterizing the entity, which I have said requires reference to the context, and the project of describing what, on a particular occasion, it does, namely, interact, are distinct activities. And it is only as a means to discriminate these intellectual activities that I would defend the distinction in my text, such as it is, between context and interaction.

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Notes

1 Incidentally, the disjunction here and the inclusive disjunction in the title are somewhat problematic. If strong doctrines of reductionism are true, then chemistry is reducible to physics. Hereafter, I shall generally refer just to physics, though, of course, any practical scientific project will surely be happy with a reduction to chemistry.

2 I won't worry here exactly what are the appropriate steps in the hierarchy. As a matter of fact, I am increasingly skeptical whether there really is such a hierarchy rather than a number of points at which we have found it useful to abstract objects of particular degrees of complexity. For example, we typically think of multicellular organisms as distinct from the multitudes of microbes with which they are in an obligate symbiotic relationship, but this is not appropriate for all purposes. The argument below concerning the contextual dependence of the identity of biological entities points more generally in this direction, but I won't develop the point any further here.

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PART II

HAVE TRAITS EVOLVED TO FUNCTION THE WAY THEY DO BECAUSE OF A PAST ADVANTAGE?

Introduction

The parts and processes of organisms tend to operate in certain ways on a regular basis. This operational regularity not only aids biologists in identifying certain traits, but some would argue that it also enables biology to be considered an autonomous science with its own domain of laws (Mayr, 1996; Nissen, 1997). The heart pumps blood, the kidneys filter urine, the medulla oblongata controls breathing, mitochondria convert sugar to ATP—all of this happens in fairly predictable and reliable ways. Another way of describing the tendency for parts and processes of organisms to operate in predictable and reliable ways is in terms of the organism's *functions*.

In the biological realm, a complete explanation of a trait seems to include an explanation in terms of function. It is natural to ask of some trait, "What is its function?" or "What purpose in the organism does the particular trait serve?" or "What is the goal of its activity?" In fact, in her paper in Part I of this book, Evelyn Fox Keller notes that function is "a concept that to this day remains indispensable to biology." The ancient Greeks—especially Aristotle (1995a, 1995b)—were aware of this kind of regularity in nature, and many thought that the universe itself was teleologically ordered (*telos* is a Greek word meaning "end" or "purpose"; see Ayala, 1970). In this post-Darwinian age, such a view seems to be without warrant, although James Lennox (1993) makes some convincing arguments that Darwin was a teleologist, of sorts.

Still, to explain the existence of the human heart, for example, as *merely* a red mass of tissue located in the chest cavity of the torso that pulsates and reverberates according to the natural laws of chemistry and physics might strike one as an incomplete explanation—an important part of explaining the heart is to say that it exists *so as to* pump blood as its end or purpose, or that pumping blood is the heart's *operation* or *function*.

Beginning in the 1960s with Ernest Nagel (1961) and Carl Hempel (1965), philosophers of science and biology have proposed many definitions for the term *function* in the hope of capturing the role that evolution plays in biology. Furthermore, researchers since Nagel and Hempel have been trying to specify the conditions under which it is appropriate to predicate functions of natural and artificial things, and to appeal to these functions in explanations (Arp, 2006; Ayala, 1970; Cummins, 1975; Griffiths, 1993; Millikan, 1984). In recent years, two views of the definition and nature of function have emerged as the most viable among biologists and philosophers of biology.

According to what has been referred to as either a *selectionist*, *backward-looking*, *etiological*, *modern history*, or *Ruth Millikan-style* account of function, the function of some trait or process *X* in an organism is defined by what the trait was naturally selected for doing in the organism's species' past. This is to say that *past* advantages to the organism are what define functions. So for example, the heart pumps blood or the kidney filters urine in a cat because these traits enabled the cat's ancestors to survive. The hearts that pumped blood and the kidneys that filtered urine most adequately were naturally selected for as a trait in felines, and cats today have hearts and kidneys that function so as to pump blood and filter urine *precisely because* of this fitness. In his paper included in this part, Mark Perlman argues for this *selectionist* account of function:

> Evolution by natural selection shows us how and why parts get selected to have the structures they do, and it seems that they are selected, to a significant degree, according to whether or not they perform their function well. Well-functioning organs, parts, and processes make survival and reproductive success more likely. This is the general reasoning behind the view that traits have evolved to function the way they do because of a past advantage.

Now, many thinkers believe that talk of functions, in general, is still fraught with antiquated, pre-Darwinian, groundless ideas of either an *internal* kind of movement toward ends, as one finds with Aristotle's view (see, for example, Johnson, 2005), or an *external* kind of movement toward ends as one finds with designer-god views (see the discussion in Ayala, 2007). According to these thinkers, there is an inappropriate normative, value-laden, and anthropomorphic connotation often associated with functions. Biological parts and processes simply operate, and we humans have to be careful not to project normativity onto these operations (Churchland, 1989; Dennett, 1987; Ruse, 2003). In John Searle's (1990) words, functions are the products of intentional minds and "never intrinsic but are always observer-relative" (p. 414).

In the second paper in this part, Robert Cummins and Martin Roth caution advocates of selectionist accounts of functions not to project something onto organisms that is not there: "Organisms are not *supposed to* be fit; to think otherwise reflects

the pre-Darwinian mindset that ultimate ends or goals can be found in the natural world." Instead of a selectionist account, Cummins and Roth argue for what has been referred to as either a *systematic*, *organizational*, *present-looking*, or *Cummins-style* account of function. According to the *systematic* account, roughly, the function of some trait is defined in terms of the role it plays in maintaining the overall systematic organization and survival of the organism in its present state. Traits only have functions in relation to other traits within the organization of the system as a whole. So, the heart's function is to pump blood because, ultimately, this fulfills its causal role in relation to the organization of the animal as a whole.

Researchers have tried to reconcile the selectionist and systematic views, and Perlman hints at this near the end of his paper. Whatever the case may be regarding the definition and nature of function, thinkers cannot seem to get around Robert Trivers' (1985) claim that "even the humblest creature, say, a virus, appears organized to *do* something; it acts *as if* [italics added] it is trying to achieve some purpose" (p. 5) or Larry Arnhart's (1998) observation that "although the evolutionary process does not serve goals, the organisms emerging from that process do" (p. 245).

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CHAPTER THREE

Traits Have Evolved to Function the Way They Do Because of a Past Advantage

Mark Perlman

Talk of the functions of objects (i.e., what the function of *X* is, or what *X* is *for*) is common throughout biology, indeed in many areas of science, as well as in our everyday understanding of objects in the world. This talk of what things are for, or the purpose for which they function, is the subject-matter of the field of *teleology*. There is widespread disagreement among philosophers and scientists about how we should understand functions. The main debate is between those who would base teleological functions on history and evolutionary selection of the thing in question (the *selectionist* view) and those who would base function on the actual causal role the thing plays (the *systematic* view). This chapter will describe the various approaches to functions, and defend a selectionist answer, that biological traits have evolved to function the way they do because of a past advantage. The paper will also discuss the counterarguments from the systematic view, and show how they fail to account for some major constraints on explaining functions, such as *normativity*.

1 Introduction

Some objects just are, while other objects are *for* something—they have functions. Can-openers are for opening cans. Lawnmowers are for cutting grass. These human-invented things have their functions because people designed them to have those functions. Of course, even for some human-made objects, the origins of their functions may be more complicated than simply the designers' intentions, but it seems safe to say that such intentions are a major part of the source of the functions of most artifacts. Many natural objects have functions also, especially organs or parts of biological organisms. Hearts are *for* pumping blood, eyes are *for* seeing, and so on. We also attribute functions to traits, and even behavioral strategies. Thus, the function of

camouflage traits of many organisms is to conceal their presence, either from predators or from prey, and primates' grooming functions to keep their fur clean and free of parasites. We might try to account for biological functions the same way we do with artifact functions: link them to intentions as well, presumably, a divine Grand Designer. But science seeks natural explanations of the natural world and, hence, scientists seek to ground biological functions on something natural rather than supernatural.

We might also be tempted to simply reduce or eliminate talk of functions (as the logical positivists would have had us do—see, for example, Hempel, 1965; Nagel, 1961); after all, Aristotle's (2001; also Johnson, 2008) ancient metaphysical framework, with its massive doses of teleology of final causes, has been rejected by mechanistic modern science. The problem is that biology makes prodigious use of functions, and not merely in an isolated domain, but in virtually *all* its sub-disciplines. We are not about to give up on this idea; hearts really do have the function of pumping blood. Biology cannot do without functions, and it is certainly a bona fide science, so we must be able to explain what grounds functions. The question is—what?

This paper will discuss the constraints on theories of teleological functions, and present the main schools of thought on functions. It will defend the *selectionist* view that the functions of biological objects, processes, structures, and traits can be successfully grounded in the historical features of natural selection. It will also discuss the main contrary position, the *systematic* view of functions, and argue that it does not prevail over the selectionist view.

2 Function and *Mal*function

One constraint on any account of biological functions is that it should include the most interesting feature of functions, that something can *have* a function even when it does not actually *perform* that function. The eyes of a blind person are still *for* seeing, even when they fail to perform that task. They can be considered malfunctioning sense organs, but they still have that (unrealized) natural function. Millions of sperm have the function of fertilizing an egg, even if only one in 100 million actually succeeds in doing so. It is natural, if odd-sounding, that almost all sperm fail to perform their function. This feature is what is known as the *normativity* of functions, the sense that something with a function is *supposed to* act in a certain way even when it doesn't always do that, or that function contains a rule that is supposed to be followed. This feature is central to the notion of function—it is what makes appeal to functions so interesting, useful, and explanatorily powerful. As Millikan (1989a) writes: "Now an obvious fact about function categories is that their members can always be defective—diseased, malformed, injured, broken, dysfunctional, etc.—hence unable to perform the functions by which they get their names" (p. 295). It is not enough to say that normal members of that category do perform the function; the question is what makes the dysfunctional item count as a member of that category at all? What makes those round organs on the face of blind person count as eyes at all if they cannot see? Perhaps they are merely correctly functioning *schmeyes*, eye-like organs (but not "eyes") that don't allow vision. Any successful theory of the source of function needs to account for this possibility of malfunction. The most obvious candidate for the source of a function—the task(s) which the object in question actually *does* (or the causal role

it plays in the mechanism of which it is a part, or the actual capacities it has)—has a major downside: no theory of malfunctioning. This would omit central feature of functions. But if what the object actually does is not the key to its function, what is?

3 Function and Evolution

It is here that the idea of function being defined historically gets its impetus. Perhaps an object gets it function from the actions of its precursors. This answer may well be true for some (but not all) human-made artifacts (especially mass-produced objects), but it is very tempting for biological organs and parts of organisms. They were reproduced from a genetic blueprint, and they were grown because the blueprint directed growth in that way, with that structure. The view here adds that parts are grown that way also to function in the way that they do. Evolution by natural selection shows us how and why parts get selected to have the structures they do, and it seems that they are selected, to a significant degree, according to whether or not they perform their function well. Well-functioning organs, parts, and processes make survival and reproductive success more likely. This is the general reasoning behind the view that traits have evolved to function the way they do because of a past advantage.

Moreover, given that teleological function is so ingrained in biological explanations, and given that the general paradigm for biology is evolution by natural selection, it would be surprising if functions were *not* grounded in natural selection. If selection explains how the organism came to have the structures it has, why wouldn't selection also be behind why those structures have the functions they have?

Of course, there are many details and competing factors to consider before declaring this historical (or "etiological"), evolutionary, teleological account of function to ultimately succeed, and we also need to consider some of the major alternatives.

4 Function Based on Past Origins

The modern philosophical "source" of the historical or etiological origins view of functions is often designated as having come from Wright (1973).[1] The function of *X* is *Z means*:

1 *X* is there because it does *Z*.
2 *Z* is a consequence (or result) of *X*'s being there.

The core idea is that the function of an entity is what explains why that entity is there. The function comes about, and is reinforced, in the same way that the function of artifacts is created and reinforced by the human designers of the artifacts: the function of a screwdriver is to turn screws, the screwdriver was made in order to turn screws, and the turning of screws is a result of the screwdrivers being there to turn them. In the case of a natural item, it is nature, not people, that puts the function there, and we can see this by examining the history of reproduction of the item. But it is somewhat misleading to call Wright's account the modern genesis of historical and evolutionary accounts. For one thing, the historical factors Wright appeals to are

diverse, and do not focus solely on specifically evolutionary history as determined by Darwinian natural selection. His view applies both to historical formation of function in artifacts *and* to non-engineered natural objects and traits.

Moreover, there is a significant problem with Wright's general view, pointed out by Boorse (1976)—some causes should not count as the basis of functions. In Boorse's example, suppose a scientist in a lab is knocked unconscious by gas escaping from a leaky valve. Even though the presence of gas in the room is explained by the fact that the scientist is unconscious (and, thus, could not turn off the gas), surely we should not view the function of the gas as being to render the scientist unconscious. The explanatory factor of its proliferation should not yield an assignment of function in this kind of case, but Wright's view seems to do just that.

One way to avoid such counterexamples is to limit the forms of historical explanations to explanations that invoke *selection*. The proliferation of gas in the room is explained by the scientist's being unconscious, but it is not selected *because* of such unconsciousness. In this way, we can avoid having unsuitable historical causes or explanations generate functions, but we can invoke historical factors when they really do generate functions. Thus, in overcoming this problem, we see natural selection emerge as a powerful mode of understanding function.

In doing so, it may seem as though we lose a nice feature of Wright's account—his providing a common grounding of artifact and natural organic functions. However, this may be a virtue instead of a drawback. In creating artifacts, humans also do indeed select among alternative possibilities, so many of the historical origins of artifacts are, artificially, selected. On the other hand, some argue that we ought to separate natural selection in biology from artificial selection of artifacts. Griffiths (1993) points out that while natural selection only operates on what a trait actually does, human selection (in the intentions of human designers) can be propelled by mistaken beliefs about the advantageousness of a trait. Griffiths relates that designers of early racing cars gave them tapered tails because they believed such tails would be aerodynamically efficient. It turns out they were wrong, and such tails actually increase drag, but the designers' false belief was nonetheless the reason they selected that design, so it is still the source of their function. The tapered tails are *for* reducing drag even if it is a fact of aerodynamics that they never will do so. Here it looks to be a virtue that the natural selection source of biological functions doesn't follow the artifact side so closely.

5 Biological Functions Based on the Distant Past: Natural Selection

The real focus on evolution and natural selection origins of function came with Ruth Garrett Millikan's landmark 1984 book, *Language, Thought, and Other Biological Categories: New Foundations for Realism*. She sees organs or mechanisms of an organism having their functions due to their evolutionary history of natural selection, specifically. Having those functions has contributed to the reproduction of organisms of that kind, and this evolutionary contribution gives the organs (or mechanisms) the functions they have. Millikan develops a technical theoretical concept[2] she calls *proper function*. A proper function is a property of a mechanism in an organism

needed for survival in *Normal* conditions, that is, in conditions in which the majority of organisms of that kind have lived and evolved. *X* performs its proper function when it does the sort of thing that has been historically responsible for replication of *X*s. On this view, hearts have the proper function of pumping blood because pumping blood is the result of similar organs in ancestor organisms that contributed to the selective success of those ancestors and, thus, caused the persistence of hearts. The organ has the function it has now because ancestors' versions of that organ helped its evolutionary success; the organisms and the organs are here now because ancestral hearts did their job and are (partly) responsible for the organism's (and its organs') present existence.[3]

Proper functions can fail to be fulfilled fairly often and still be an adaptational advantage, as long as they are fulfilled enough times for survival. So a Normal *X* need not fulfill its proper function, most of the time; just often enough to keep the organism on track evolutionarily, that is, out of danger and reproducing in sufficient numbers for evolutionary success. A mechanism that indicates the presence of predators in an organism's immediate environment need not be reliable in the sense of being tokened only when a predator is around. It can give a lot of false positives and still be a good mechanism. (But one false negative may be too many, for just one will be almost a certain chance of being eaten.)

Even when organs or systems seldom perform their proper function, a few successful performances may be enough. Millikan's example of this is from reproduction: the function of sperm is to reach and fertilize an egg, and that is the function of all sperm even if very few actually make it. Bee dances are another example she cites—as the orientation of the bee's "dance" (a specific pattern the bee makes as it flies) tells the other bees where food is. If the dance is mis-oriented, its content is still *direction of food*; it just fails to fulfill its proper function, and is a misrepresentation of the direction of food.

Among the areas for which Millikan uses this view of function is the content of mental representations, and along with it linguistic meaning. The approach has become known as *teleosemantics*. Very briefly, a theory of mental content should explain intentionality (aboutness), that is, how my ideas can be about something (say, grass), and how I represent something (i.e., I can think that "grass is green") and also explain how I can misrepresent the world (as I do if I think "grass is orange"). The selectionist approach would ground mental representation in cognitive function, and invoke malfunction to support the notion of misrepresentation. Millikan's view of representational content comes in two versions, neither of which assigns proper functions to the individual representations themselves. In *Language, Thought, and Other Biological Categories* (1984) her view is that it is not the representations themselves but the cognitive mechanisms producing the representations that have the proper functions, to produce and process representations which represent the environment correctly. In "Thoughts without Laws: Cognitive Science without Content" (1986) (and also Millikan 1989a, 1989b) she emphasizes the proper function of the consumer side of the representations, the mechanisms that use the representations produced by perception. Others who advocate a teleological approach to mental content include Papineau (1987, 1993) and Neander (1995).

One well-known proposed counterexample to selectionist accounts, from Davidson (1987), is the possibility of a spontaneously created organism (resulting from a freak

lightning strike in an organic environment like a swamp), molecularly identical to organisms with an evolutionary history and organs with functions based on that history. (Such a spontaneous organism has been nicknamed "Swampman.") Yet, this organism has no selection history whatsoever and, thus, nothing for the selectionists to base its functions on. Thus, on the historical selectionist accounts of mental representation, such an organism, with representational systems having no evolutionary history, cannot have any representational powers. Moreover, the organism's heart, lungs, eyes, etc., also lacking selection histories, will not have any proper functions either. The objection is simply that if these Swampman organs are identical (down to identity of molecular structure) to organs of similar organisms which did actually evolve, then it is absurd to say that one heart has a function and its double does not, or that one can represent the greenness of leaves around it while the other cannot. Given its ability to register its surroundings and respond with appropriate behavior, it just seems absurd to say it cannot represent its environment. Given that its heart pumps its blood, it also seems utterly counterintuitive to say its heart does not have the function of pumping blood simply because Swampman lacks the right kind of history.

Millikan (1996), along with Neander (1996) and Papineau (1996), accepts this counterintuitive consequence, and does not see it as a problem for the multitude of real organisms whose functions her theory does explain. Sometimes a common-sense intuition can be overturned by a good theoretical account. I have to agree with Millikan et al.'s dismissal of this as a serious roadblock. In jurisprudence, there is a saying that "hard cases make bad law" and, perhaps, that also applies to philosophy of science. The Swampman objection is that if a miraculous event that no one thinks would ever happen (but is at least a logical possibility) actually did happen, then the selectionist view of functions would give us an odd-sounding account of its organs' functions. For such an odd (and utterly remote) possibility, perhaps there is nothing wrong with an odd answer.

6 Fitness and Goal-Contribution Accounts

Interestingly, the historical approach to functions, while it often grounds them on selection, need not do so. Buller (1998) points to *fitness* rather than selection, arguing that ancestral contributions to ancestral fitness are enough to bring about natural norms and functions, even when there is no variation of traits to be acted on by selection. As long as the ancestral contributions to fitness are heritable, they contribute to their own preservation even if not, strictly speaking, through selection. Buller terms the selective theories as *strong etiological theories*, and the view that grounds functions on some sort of historical success (but not necessarily selection) *weak etiological theories*. While Buller's view is still in the minority, even if he is right, this need not necessarily be considered a counterargument to the selectionist view of functions. The overall historical view is that traits have evolved to function the way they do because of a past advantage, which allows there to be numerous routes to the source of the advantage. While the so-called "standard view" is that it is primarily natural selection, to locate it in fitness can still be said to qualify as another way in which a trait's presence can be due to past advantage.

Another route to finding a past advantage looks at the more *recent* past, rather than distant past. Such theories are often known as *goal-contribution accounts*. For example, Godfrey-Smith (1993, 1994) has us ground functions on factors in modern history which *maintain* (rather than select) a trait. Part of the reasoning for this move is, again, that selection requires variation—different options from which to select. But this does not fit well with traits already present which are maintained for their advantageousness. Godfrey-Smith's (1994) example is feathers: they were originally developed in early bird-like creatures (descended from or still to be considered reptiles) as a mechanism for insulation and thermoregulation. Later, when descendants of those organisms developed musculature and bone structures that became wings, their feathers came to have a major contribution to efficient flying, and this took over as their main function. This new function then maintained the presence of feathers, rather than selected them from alternative forms. So, Godfrey-Smith argues, their function in flying was not actually *selected*, though flying was involved in maintaining and preserving their presence in descendant organisms.

These theories that ground function in the recent past can, in a way, be said to be in agreement with the general view that traits have evolved to function the way they do because of a past advantage, though they are often couched as being rejections of the evolutionary view. The question is *how* distant, and, of course, adaptation can occur in shorter time-frames as well, depending on the rate of change in the environment and other selection factors. So, focusing on the more recent past may still be seen as invoking evolutionary factors. The goal-contribution theories often cite behavioral patterns and strategies as the recent origins of functions, especially when they are learned behaviors, not instincts. But to consider this a critique of the selectionist view is to overlook an important fact: learning is also selection. Of course, it is not the natural selection of genes, but rather natural selection of one behavior among numerous behavioral options, as an adaptation to the environment. Function of behavior remains grounded on selection, just not selection of genes. Moreover, the goal-contribution approaches can be embraced by the selectionists as a way to account for changes in function over time. Feathers may have originally functioned as insulators, but their functions later evolved and changed, due to natural selection. The later advantage, allowing them to fly, is the historical factor that gives them this current function. Thus, selectionist and goal-contribution theories should really be seen as *complementary* theories of function.

7 Neo-Teleology

We have now seen the powerful reasons to say that the explanation of present traits of organisms can be traced to their function, and that function can be traced to the past advantage of having traits with that function. But even if we are sympathetic to teleological explanation of function, dubbed *neo-teleology* by Cummins (2002), we must not try to explain too much by relying on teleology and function. In explaining why-is-it-there for traits of biological organisms, there are always really two sides to the answer: the mechanism that creates it, and the force that drives its development. For any biological trait, it arises originally because of mutation, and it appears because of the expression of the gene.[4] If not fatal or too disadvantageous, and if

the organism is lucky enough to survive, then the gene and the trait may be retained and passed on to offspring. So genetics and the developmental history of the individual organism will explain why the trait arose in that organism; and that sense of why-is-it-there is to be explained *mechanically*, not teleologically (even for fans of neo-teleology). But we will also want to explain why it remains, that is, why successive generations came to have it, or a further developed version of it. Even then, the trait might not itself have any evolutionary advantage, and indeed might be disadvantageous, and still survive. Often genes will be linked, or several traits linked to the same gene, so a trait might persist merely because the gene that brings it about is responsible (or linked to the one responsible) for bringing about another trait which does indeed yield an evolutionary advantage. In these kinds of cases, the presence of traits that "come along for the ride" will not usefully be accounted for by a teleological explanation of their function.

That said, there will still be many traits that have clearly evolved, and which clearly yield their evolutionary advantage in virtue of their function. The best explanation for their having developed into their present form will be from looking back at how the earlier forms of the traits functioned in the ancestors. For these traits, their presence and their current form are because of the past advantage of that trait (or earlier less-developed analogues) for ancestors. Here, the teleological approach does explain the current function, answering a specific version of the why-is-it-there question: why-is-it-there-in-the-form-that-it-is? Answer: because in the past its function created an evolutionary advantage. How far into the past should be look for this function-setting history? That may depend of the particular path of development of the trait. We certainly must allow that structures and processes can change function over time. So how far back we look may depend on how long the trait has possessed the current function. But whether we look at the more recent past, as the fitness and goal-contribution approaches do, or the distant past, as the selectionist view does, past function sets current traits and their functions in virtue of function contributing to evolutionary success.

So we arrive at a *qualified* yes answer to the question "have traits evolved to function the way they do because of a past advantage?" The teleological explanation of function is applicable to some/many traits, but not necessarily all of them.

8 Looking at the Present: Causal Roles and Functions

The main alternative to selectionist accounts of function is an approach based on causal roles of components of a system. In his 1975 paper "Functional Analysis," Robert Cummins proposed a sophisticated causal view, that a capacity of some complex system can be explained as the combination of capacities of the system's constituents, yielding an account of the functions of those components. The functions are based on what the components in the system *do*, though not everything they do counts as a function. The circulatory system can be analyzed into components each contributing to the overall functioning of the system: the blood carries oxygen and nutrients, the blood vessels carry the blood through the system, lungs add oxygen to the blood as it passes through, and the heart pumps the blood through the entire system. Thus, we can say that the function of the heart, in this system, is to pump

blood. The heart also does other things, such as make a *lub-dub* sound, but in this system that is not its function, since that action does not causally link up with the other capacities of the components of the system to allow the entire system to operate.

One important feature of functional analysis is that it does not depend on how each component itself operates internally, or what it is made of, but only the job that it does (or has the capacity to do). Another virtue of functional analysis is that it can be applied to biological and non-biological systems alike; it can explain the functions of parts of organisms like the circulatory system or visual system and, also, analyze the functions of components of a computer, a circuit board, or an assembly line. Cummins' explanatory account has been hugely influential, and has led some, including Millikan (1989a), to refer to functions so analyzed as *Cummins functions*. Others call them *causal role functions* or *systems functions*. In this volume, we use Cummins' preferred term: *systematic functions*.

The proponents of neo-teleology need not find fault with the systematic view of functions, *per se*, unless it is being claimed that it is *the* analysis of functions. We can, and ought to, grant that functional analysis is a useful way of breaking down a system into components and analyzing how they fit together. But we need to look at two central criticisms of systematic functions: (a) promiscuity, and (b) lack of an account of malfunction.

Systematic accounts are often faulted for being too liberal about function attribution—which the critics have called *promiscuity* of function. Millikan (1989a, 1993) complains that Cummins' account incorrectly attributes functions to things that don't really have them, such as rain clouds:

> [A]ccording to Cummins's definition, it is arguably the function of clouds to make rain with which to fill the streams and rivers, this in the context of the water-cycle system, the end result to be explained being, say, how moisture is maintained in the soil so that vegetation can grow. Now it is quite true that, in the context of the water cycle, clouds function to produce rain, function *as* rain producers; that *is* their function in that cycle. But in *another* sense of "function," the clouds have no function at all, because they have no purpose.

Matthen (1988) similarly objects to the odd results of the systematic view. In his example, the large and awkward tusk of the narwhal whale will be assigned the systematic function of reducing the whale's mobility. Such a dysfunctional function assignment is far less plausible than the function we would get on a selectionist account —the large tusk is a conspicuous attribute for mating purposes and, thus, the "function of the tusk is to increase the sexual attractiveness of the animal; certainly its function is not to reduce mobility" (p. 15).[5]

Millikan grants that Cummins has no aspirations to be defining purpose, merely actual function. But still, this attribution both clashes with our intuitive sense of the term "function" and is based on the component's actually performing that function. The whole point of Millikan's selectionist proper function account was to allow us a sense of the function, even for things that not only do not ever actually *perform* the function (like the multitudinous unsuccessful sperm), or a malfunctioning component, which lacks even the capacity to do its function (like a diseased heart which

lacks the capacity of pumping blood). The diseased heart still has the function of pumping blood, but doesn't and cannot actually perform that function. Thus, the systematic view fails on two counts: it attributes functions where they should not be, and it cannot make sense of malfunction. Systematic functions are not normative in the way that Millikan argues we need functions to be, and which her proper functions are.

Cummins and his defenders have responses to both the promiscuity charge and the malfunction issue. As far as promiscuity of functions goes, there are two rejoinders: limit the application of systematic functions, or reject the validity of the complaint. Davies (2001) argues that when system function is restricted to systems that are hierarchically organized, the promiscuity objection falls away. Indeed, Cummins' (1975; also see Buller, 1999) original systematic account anticipated the promiscuity charge, and alleviates it by relativizing the systematic functions to:

(i) the extent to which the analyzing capacities are less sophisticated than the analyzed capacities,
(ii) the extent to which the analyzing capacities are different in type than the analyzed capacities, and
(iii) the relative sophistication of the program appealed to, i.e., the relative complexity of the organization of component parts/processes that is attributed to the system.

Thus, the heart's capacity to make noise is not its function, because the capacity of interest of the system is not to make noise, but to circulate blood (to which the heart's noise does not contribute). Similarly, Davies (2001) argues that Millikan's cloud example fails because the levels of analysis do not fit Cummins' limiting conditions: the analyzing capacity in this case, clouds producing rain, is not simpler or different in kind from the target analyzed system, the distribution of moisture. If we consider Millikan's mention of maintaining moisture "so that the vegetation can grow," then the system being analyzed is a much larger ecological system than merely the water cycle, so the objection's conclusion is based on considering the wrong analyzed system.

Another tactic to battle the promiscuity objection is seen in Prior (1985), who seeks to relativize function to that which is *typical* or *normal* for the type of item in question, distinguishing a special subset of functions, *s-functions*, which are functions that standardly contribute to the survival and/or reproduction of the organisms that perform it, and saying that failure to perform an *s-function* counts as a malfunction. Millikan (1989a) and Neander (1991a) reject this by pointing back to the case of the sperm—it is normal for almost none of the sperm to fulfill their functions.

Detailed responses aside, the larger issue for the systematic view is that Cummins' point is to reject the talk of something's being *the* function of an organ or subsystem, because that talk is too teleological, filled with purpose, intention, and things the system is *supposed to do*. Cummins is content to have found a way to analyze the manner in which a component functions, without worrying about whether in a metaphysically charged sense that is *the* function that it is *supposed* to have.

A more serious criticism for systematic functions is that systems functions cannot allow for malfunctioning. Cummins accepts this result, to some extent, and in the original account provides that systematic accounts can only yield malfunctions relative to the actual containing system being analyzed. Davies (2001, Ch.7) actually

argues that it is a virtue of the systematic approach that it does not allow for malfunctions, and vigorously argues that no notion of function should expect to be normative and account for malfunction. Boorse (2002) is also willing to reject the requirement that functions must account for malfunction: "If Carla's heart cannot pump blood, then pumping blood is not, in fact, the function of her heart; it has no function. Since blood-pumping is the normal function of a human heart, it would be the function of Carla's heart if Carla's heart pumped blood normally; but it does not, so it is not" (p. 89).

Still others argue that biology does not actually invoke functions in the normative sense. Biologist Ernst Mayr (1961, 1993, 1997) maintained that biology consists of two largely separated fields: functional biology (the study of individual-level mechanisms) and evolutionary biology (the study of the history of those mechanisms). So while, obviously, the evolutionary side is concerned with selection, it may be that the functional side need not invoke it for much of anything. On this basis, Wouters (2003, 2005a, 2005b) argues that biology is much less concerned with normative functions than the selectionists claim it is, and that the main reason philosophers are so focused on selectionist functions is their agenda to use them to carve out a theory of mental representation.[6]

But these defenses notwithstanding, many find failure to account for malfunction to be the glaring shortcoming of systematic functions, and the glowing virtue of selectionist accounts. We often seek to explain the behavior of a biological system in terms of success or failure, and failure often turns on whether its systems function properly or malfunction. Moreover, the systematic defense overlooks Millikan's point about categorization mentioned earlier—how can we do functional biology until we can categorize the component organs and parts of an organism? And how can we say that *X* is such-and-such *kind* of organ without its function? How can we understand a malfunction, or say what counts as a malfunction, without a selectionist view of function? This is a crucial part of understanding nature, and systematic functions seem to leave us without any way to deal with malfunctions.

It is notable that there is a growing momentum to view the selectionist and systematic approaches as somehow compatible. Some, like Kitcher (1993), seek to unify the two approaches. Others view one as a subspecies or instantiation of the other: for example, Griffiths (1993) proposes that selection functions are a kind of systematic function, so where systematic functions are not generally normative, the systematic functions that are selected for can indeed be normative. Walsh and Ariew (1996) present a similar view, arguing that such a combination theory gives us the resources to account for malfunction. Others, like Arp (2006), Godfrey-Smith (1993), Millikan (1989c, 1999, 2002), Perlman (2008), and Preston (1998), view systematic and selected functions as both valid alternative approaches, a position known as *pluralism*. This approach attempts to retain the virtues of both approaches. However, there is no consensus on any of these attempts at reconciliation, and Davies (2001) rejects all of the combinational views (unification, instantiation, and pluralist) as untenable, arguing that none of them can account for malfunctioning. Still, the growing acceptance of the pluralist view reminds us of how much agreement there is between the two sides.

Sometimes, in such debates, it seems that the opposing sides are very far apart when, in fact, they are battling over a very fine difference, and this is true of the

functions debate. Both sides agree that functional analysis can illuminate the interactions between components of a system. They differ only in whether the resulting capacities should be considered *the* function of a component, or merely one of many ways that it functions. So selectionists can, and do, acknowledge the useful information yielded by functional analysis. Both sides also agree that in biology, the right way to understand origins and development of organisms is through evolution, and that evolution is largely brought about by natural selection. Those favoring systematic functions are certainly not denying evolution or natural selection—they agree that selection is behind evolution. They just think that selection is not the way to explain why a component of a system has a certain capacity.

9 Evolution Bites Back: Vindication of the Selectionist Approach

Cummins (2002) presents powerful arguments concerning the change of function, in early forms of organs and appendages He points out that the early stubs of what would later evolve into descendants' wings were not capable of flight and, thus, they were not selected for flight, and cannot serve as the source of the flight function of modern wings. The same holds for the fins of some ancient fish, which were used to push them along when they flopped onto land. These fins are the precursors of what later evolve into the legs of a huge variety of land animals, but it seems that such fins' motions cannot be the source of the function of legs to walk and run. Cummins views this as the refutation of the selectionist theory of teleological functions. But is it?

I am tempted to agree with Cummins' nice example against the evolutionary source of functions—since wings develop from wing buds and wing stubs that obviously didn't have the function of flight, modern wings which have flight as their function didn't get that function from functions of proto-wings. But I wonder if this may be a *special case*, instead of a generality. And I wonder if we might acknowledge the example, but not agree that it is decisive as a refutation of neo-teleology.

Consider the function of eyes. Proto-eyes began as eye spots on skin and were only very vaguely photosensitive. While proto-eyes didn't have the function of forming detailed images, it does seem plausible that they had the function of giving the organism some visual information about the environment, yielding an evolutionary advantage. An eye spot could produce perception of a rapid light–dark–light progression in the immediate vicinity, which could tell the organism of the presence of a predator (or its shadow). Modern highly developed eyes (like human eyes) still have that function, but far improved and in vastly greater detail and resolution. Providing visual sensory information is something that eye spots and modern human eyes share. The difference is not one of kind, but of degree (though, granted, quite a large degree).

So, the evolutionary source of function seems more plausible for eyes than wings. Traits can be divided into those with developmentally modified function (such as feathers and wings)[7] and those with developmentally incrementally specific function (such as eyes).[8] If the incrementally developing traits are the more common type, then even if Cummins may be correct about special cases such as wings (and similar cases), we can still make the case that many, or most, traits, organs, and behaviors do inherit function from their past.

However, with this point in mind, the selectionist view may be able to deal with the argument about wings as well. Perhaps we are describing the behavior of these early precursors incorrectly, and a different description will be better-suited to the evolutionary selectionist view of functions. Consider those proto-wings again. Initially, they were merely limbs with no contribution to flight. But the proto-winged reptiles also developed early feathers which, as Godfrey-Smith stresses, were for thermoregulation, not flight. Together the limbs with feathers came to have a small aerodynamic capability—not flight exactly, but better than nothing. Even today, there are numerous species of birds whose chicks leave their tree branch nests before they can really fly. They fall from the tree furiously flapping their underdeveloped wings, and though they don't fly, they slow their fall just enough so that they land without injury. (Of course, this probably also depends on kind of surface they fall toward—cement might be fatally hard, but pine needles on a forest floor are soft enough for their slowed fall to end safely.) Flying comes in many varieties, and is actually a matter of degree. Eagles spend most of their time in the air soaring and floating on thermals, because such large birds would burn too much energy if they flew more actively by flapping away with their wings. People in hang-gliders "fly," though this is just floating on the air until they reach the ground. People in parachutes slow their fall, and they can even use high-tech parachutes to steer their descent. Compared to a blue jay or hummingbird, this "flying" is pretty unimpressive, but it is far better than unequipped humans can fly (which is not at all).

Perhaps we should say that anything short of falling at terminal velocity is, to some degree, a kind of flying. The baby birds can't take off into flight as they will in adulthood, but they don't drop like stones either. Their underperforming wings still do function to some degree—they give some amount of *aerodynamic lift*. Not much, but just enough. So those proto-wings of ancient feathered reptiles might not have provided take-off, but they likely did provide some amount of aerodynamic lift, if only slowing descent enough to fall safely. That aerodynamic action is enough to give even those proto-wings the function of some degree of "flight." Eons of evolution greatly increased the degree of aerodynamic lift, so now normally functioning adult birds can take off and really fly. Even flightless birds, such as penguins and ostriches, can get a minor amount of lift (though the penguin wings do better as fins in water).

So perhaps wings, even their early proto-wing stubs, have functioned all along to let the organism fly (to some degree). This providing of some amount of aerodynamic lift was there from the early forms of those structures, and that gives modern wings of ably flying animals their function—providing aerodynamic lift. It is just that most modern birds (and bats) have wings that provide this aerodynamic lift far better than their ancestors' early wing stubs did. Selection drove the gradual evolution of better and better wings, which provided more and more of that valuable aerodynamic lift, so that now it is quite a success story. But if we describe the function as providing aerodynamic lift, then it has been there all along. Only if we define the function by the extreme and call it "flying" does the selection story seem to get into trouble. Thus, we can escape Cummins' objection against the selection account by adjusting our specification of both the original and modern system function to a more neutral, less end-result description.

This same kind of story works for another kind of flying as well. Besides flapping of wings, there is an entirely different approach to flying: soaring with a flattened

body. Flying squirrels and numerous other animals have structures webbed between their limbs that give them aerodynamic lift to fly by gliding. Their habitat is the branches of trees, and all they need for excellent mobility among trees is a bit of extra lift. They don't flap their limbs to get off the ground, for they are already off the ground. They stretch out to soar from tree to tree and branch to branch. How did those small extra flaps of skin on their limbs arise? Their function does not explain the initial appearance—that came probably from mutation and natural variation. The trait was preserved because it wasn't fatal or excessively disadvantageous. But why did those flaps of skin between their limbs continue to develop into structures almost like wings that allowed these animals to soar from tree to tree? Function does explain that: it is because that trait gave the animals increased mobility, and thus increased survivability. The connecting skin between their limbs functioned to let them fly (by gliding), and that advantage is why the current animals have those structures today.

So even given Cummins' (2002) arguments, I think we can still say that teleological function is what explains why-is-it-there, in the sense of why-is-it-there-in-the-form-that-it-is? For many traits, the answer is that they performed a function, and that traits perform that function is what leads to reproductive success and continuation of organisms with traits like that.

Postscript: Counterpoint

Fortunately, the disagreement between selectionist and systematic views of about functions and teleology is not about whether Darwin is right about evolution being based on natural selection. Both sides agree that Darwinism is a fantastically well-confirmed model of explanation of the structure and change of biological organisms. Religious views like *creationism* and its whitewashed cousin *intelligent design theory* are non-starters for a naturalistic scientific account of biology. The questions are: What is the role of functions—can they be retained or should they be eliminated? What gives objects, processes, structures, and traits the functions they have? Do teleological functions have a role to play in biological explanation? The worry of logical positivists of the 1920s–60s was that functions and teleology were metaphysical views, and had no rightful place in science at all. The selectionist views of the late twentieth century and now the twenty-first century try to save functions and teleology by showing that they have a legitimate scientific basis, natural selection, and that they have a part to play in biological explanation, explaining the presence and form of biological traits, organs, processes, behaviors, and structures. The systematic view is that Darwinism eliminates the need for teleology altogether, whereas the selectionists think that Darwinism has shown us what functions are, and why they matter, and makes teleology scientifically acceptable.

Cummins and Roth (in this volume) effectively present the systematic view of functions, but there are some ways the selectionist can reply to their arguments and vindicate teleology in the philosophy of biology. First is the debate about normativity—the point is not that people knew that wings were for flight long before they knew about evolution. The point is that until the natural selection model, they did not know exactly *why* wings were for flight, that is, what grounded the functions of wings. Nor did pre-Darwinians understand how wings arose, and why they have the

structure they have. The historical selectionist view answers these questions, and does so partly by citing teleological functions.

Moreover, the very root of talking about functions is allowing for malfunctions. Malfunctions of objects, processes, structures, and traits help explain why organisms fail to succeed, survive, and reproduce. It is essential for talk of malfunctions that not all capacities count in determining function—if everything counts in determining function, then everything that happens counts as fulfilling that function, and malfunction is conceptually impossible. So some things the organism (or its parts) does must not count in determining function. Which ones count and which do not? Selectionism provides the answer—those that were selected for are the ones that count in determining function. This is the basis of the normativity of functions, and what allows for malfunction. Cummins and Roth seem at times content to give up on the notion of malfunction, but that seems a radical departure from the common understanding of function.

Moreover, their examples do not convince. Take their example of the function of the eye of a blind person. They argue that eyes as a kind (a type of object) have the function of seeing, but that the particular individual eyes (tokens) of a blind person do not have that function, because they lack the capacity of seeing. This type–token maneuver is clever but unconvincing. If a blind person's eyes are not *for* seeing, then why are they there at all? It would be odd to have such precisely shaped organs, which but for a small alteration would have the capacity for seeing, present on the blind person's face *for nothing*. The systematic view still uses evolution to show how it is that these eyes came to be there, but doesn't really capture the sense that, given the overall traits and abilities and capacities of that person, there is something their eyes should be doing, but aren't. The selectionist account gives us a much more sensible account of this—even a blind person's eyes are *for* seeing; that's why organs like that are there, on the front of the face, with links to optic nerves and a visual center of the brain. The fact that they do not perform this function is (presumably) due to some mechanical malformation of the structure, and that is why the eyes malfunction. (Of course, blindness can arise from malfunctions of the nerves and visual area of the brain as well, but the argument can easily be adjusted according to the specific malfunction.) Given the overall structure of the human body and its normal surroundings, one can compensate for not being able to see, but blindness is a significant disadvantage to creatures who normally depend so heavily on sight. The selectionist view accounts for this in a way that the systematic view glosses over.

Cummins and Roth also point out that function attributions are always relative to some capacity of the containing system, and they are correct, to a point. They even predict the selectionist reply—that there will ultimately be some unrelativized basis of function attribution. They reject this by saying that all this amounts to is that saying that the purpose of all living is to survive and reproduce, which they take to be a truism that doesn't help the selectionist case, commenting that "surviving and reproducing are just among the many things an organism does." But I think the selectionist should enbrace the point Cummins and Roth dismiss. While selectionists won't say functions of natural objects, processes, structures, and traits come from intelligent design, they should indeed say that not everything organisms do is equal. Some things they do are merely incidental to the biological mandates of survival and reproduction, and some are related to or essential to survival and

reproduction. These latter things are thus to be given a special status, and the whole point of selectionism is to say that this special status makes them the factors which determine function, whereas other (non-selection-related) things the organism does do not have a role in determining function. The explanatory value of the functions of these objects, processes, structures, and traits is that they are (at least in part) what drove the evolution of the organism. Thus they explain why it has the objects, processes, structures, and traits it has.

So, natural selection gives science a way to ground teleological functions in a naturalistically acceptable way. Proponents of the systematic view say that Darwin shows us that we don't need teleology. However, the advantage of the selectionist view over the systematic view is that teleological functions give us better, more intuitive, more informative, and more complete explanations than functional analysis does. This is not to diminish the usefulness and importance of the kinds of functional accounts the systematic approach provides. It is just to say that there is more to explain. We need functions in a fully teleological sense to adequately account for malfunctions, and we need the notion of malfunction to fully explain what is going on in the world of biology.

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Notes

1 Wright (1973, 1976) may be the *philosophical* inspiration, but he was not the first to come up with an historical (etiological) notion of function. Wouters (2005a) points out that the idea of using "function" to refer to those effects of a trait that explain why that trait was selected in the past was proposed by evolutionary biologist George Williams in *Adaptation and Natural Selection* (1966). Williams' account does not seem to have gained much acceptance among biologists, so perhaps that is why it is overlooked and Wright is seen as the modern originator of the historical view. Other initial moves can be found in Ayala (1970), Wimsatt (1972), and Woodfield (1976).

2 Millikan (1984, 1989a) consistently makes it clear that her view is the proposal of a technical theoretical concept, proper function, not an analysis of the common-sense notion or philosophical or scientific notion of function (though proper functions are a subset of functions). Yet, many people ignore, miss, or forget that point (even after she explicitly corrects them), and take her theory to be a definition or analysis of our concept of teleological "function" in general. However, others, most notably Karen Neander (1991a, 1991b, 2002), while acknowledging Millikan's avoidance of conceptual analysis, want to erect a Millikan-like theory as an analysis of the general teleological concept of function. Part of the difficulty is that pre-Darwinian scientists who discovered the function of an organ or system cannot be construed as having meant that they were discovering a selected function. Thus, Millikan seeks to avoid this problem by using a theoretical term. The dispute quickly

becomes embroiled in tricky questions of meaning of theoretical terms across changes in theory. For a good discussion of the conceptual analysis issue regarding proper functions, see Davies (2001, pp. 108–120).

3 In addition to Millikan and Neander, others who advocate versions of the evolutionary, historical view of functions as selected effects include Allen and Bekoff (1995), Brandon (1981, 1990), Griffiths (1993), Kitcher (1993), Matthen (1988), Mitchell (1995), Papineau (1987, 1993), Price (1995), and Shapiro (1998).

4 Of course, this issue is more much complicated than can be explained here. Besides presence or absence of a gene, there are often varying degrees of expression of a gene, multiple genes can affect traits, and developmental factors can play a significant role as well.

5 There are other promiscuity objections to systematic functions as well. Enç (1979) makes the point with astronomical interactions: The planet Neptune exerts a gravitational pull on its neighboring planet Uranus, resulting in perturbations in Uranus' orbit, and altering the conjunction Uranus would have with other planets in the solar system. Despite that this is what Neptune *does*, the capacity it has, or how it functions relative to Uranus, we would not want to say that *the function* of Neptune is to cause Uranus' orbital path to derivate from the perfectly elliptical one it would otherwise have.

6 Indeed, one of the main emphases of selectionist functions has been mental representation, as in Millikan (1984), Neander (1995), and Papineau (1987). Initially, Fodor favored a teleological view (1984, 1985, 1987), but later he vehemently rejected it (1990). However, even if one is generally in favor of selectionist accounts of function, this need not mean one automatically adopts it as a ground for a theory of mental content. Elsewhere (Perlman, 2000, 2002) I argue against such a teleological theory of content. Unfortunately, the details of the issue of teleological approaches to mental content are too complex to be dealt with adequately here.

7 Gould and Vrba (1982) call this kind of trait an *exaptation*, rather than an adaptation. An exaptation is a feature that performs a function but that was not produced by natural selection for its current use.

8 Mayr (1976, pp. 96–100) terms this distinction as between *intensification of function* vs. *change of function*. He points out that "Most evolutionary changes take place without the origin of new structures. . . . Most differences are merely shifts in proportions, fusions, losses, secondary duplications, and similar changes." One example Mayr cites is the eye; another is the homology of structure of forelimbs of mammals. The forelimbs of humans, dogs, pigs, horses, whales, and many other mammals have the same basic bones, but in significantly different shapes and proportions. This relatedness of structures is a major piece of evidence that these organisms have evolved from common ancestors.

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CHAPTER FOUR

Traits Have Not Evolved to Function the Way They Do Because of a Past Advantage

Robert Cummins and Martin Roth

Attributions of function are ubiquitous in biology and the sciences generally, and yet philosophers and scientists disagree over how we should understand functions and functional attributions. At issue are the *explanatory* and *normative* dimensions of functions and functional attributions, and in this paper we characterize two well-known accounts of functions—what we call *systematic* and *selectionist* accounts—and compare and evaluate their respective stances on these dimensions. We argue that whereas systematic accounts provide an approach to functions that is indispensable to biology and the rest of science, selectionist accounts do not. Furthermore, we argue that selectionist accounts introduce a conception of normativity that has no legitimate place in science, while systematic accounts do not. Our conclusion is that selectionist accounts of functions are deeply flawed, and that the attempt to ground function in selection history is a mistake.

1 Introduction

What is the word "function" for in the question that heads the title of this section, namely, "Have traits evolved to function the way they do because of a past advantage?" If we replace "function" with "produce effects," then our answer to the question is yes; for, then, the question is merely asking whether natural selection explains evolution. So what's the issue? We take it that what the question is really asking is something like this: Is there a difference between *having effects* and *having a function*, and does natural selection ground the distinction? Our answer is that there is a difference between merely having effects and having a function, but that natural selection does not account for the difference.

A useful way to approach the issues to be discussed in this chapter is to focus on function attributions. We attribute functions to things all the time; what motivates

such attributions, and under what conditions are such attributions correct? As we see it, there are two constraints on function attributions:

1 *Explanation*: Traits have many effects, but not all of those effects count as functions. Certain effects are called functions because of the explanatory role of appeals to those effects.
2 *Normativity*: To attribute a function to a trait is to do more than say what it does; it is to say what it is supposed to do, what it is for. Traits have many effects, but not all the effects traits have are effects they are supposed to have. Only the effects that traits are supposed to have count as their functions.

In this paper, we describe two approaches to function attribution, which we will call *systematic* accounts and *selectionist* accounts, and defend the former against the latter.[1] We will find that this is something of an intramural debate: there is more agreement than disagreement about the main points. So, part of what we will be doing is trying to put our finger on what all the shouting is about. In this section, composed of two parts, we contrast systematic and selectionist accounts of the role of function attributions in explanation. In section 2, we argue (following Davies, 2001) that selectionist explanations are special cases of systematic explanations and, thus, are not an alternative to systematic accounts of functional explanation. In section 3, we contrast systematic and selectionist accounts of norms and discuss their relative merits. We argue that, while systematic accounts provide an account of norms that can play a legitimate role in science, selectionist accounts do not.

1.1 Systematic accounts of functional explanation

Systematic accounts are built around a characteristic explanatory strategy we call *functional analysis*. Here are some examples:

(a) *Assembly line*: The capacity to produce a Ford is analyzed into an organized sequence of simpler capacities each of which is performable by unskilled/mechanical labor.
(b) *Cooking and calculating*: A recipe/algorithm specifies an organized sequence of simple operations, performance of which results in the desired dish/mathematical result.
(c) *Schematic diagram*: An amplifier circuit is analyzed into capacitors, resistors, power supply, etc., connected in a way that generates an amplified circuit.
(d) *Circulatory system*: The system is analyzed into various kinds of pipes (active and passive), pumps (mechanical and osmotic), and control systems (autonomic nervous system) in a way that explains how oxygen, hormones, and nutrients are delivered to cells, and wastes are removed.
(e) *Computer*: Computers need to be understood at many levels of functional analysis, from algorithm, through programs, interpreters/compilers, operating systems, firmware to circuitry and hardware. They are literally unthinkable without this kind of multilevel functional analysis.[2]
(f) *Glucose metabolism*: Understanding insulin resistance has become a major research effort in response to the obesity epidemic in North America. Figure 4.1 shows an example of functional analysis at work in this area.[3]

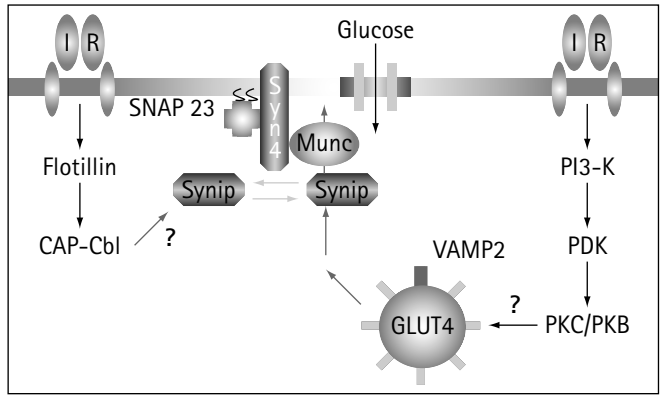

Figure 4.1: Schematic model indicating the presence of two potential insulin receptor-dependent signal transduction pathways (*Source*: Pessin & Saltiel (2000), Copyright ©2000 by the American Society for Clinical Investigation)

In all these cases, a capacity of some complex system is explained as programmed exercise of capacities of system constituents, or as programmed exercise of other capacities of that system. All of these systems can do what they do because they, or their constituent systems, do other (typically simpler) things in a certain orchestrated way. Making a cake gets analyzed into stirring, adding ingredients, baking, etc. Signal amplification gets analyzed into the capacities of resistors, conductors, capacitors, power supplies, etc. This kind of explanation by analysis is *functional* analysis because it operates at a level of abstraction that identifies constituent processes or parts in terms what they do or contribute, rather than in terms of their intrinsic constitutions: their functions rather than their forms.[4] Anything across which there is a drop in electrical potential is a resistor. Anything across which there is a pressure gain is a pump. Functional analysis, therefore, is a kind of *black-box* analysis, where a black box is something whose internal structure is irrelevant to the explanation (and perhaps unknown), and which appeals only to what the interactors do, namely, how they interact with each other.

Functional analysis allows us to understand how something works—e.g., a circuit or circulatory system—in abstraction from implementation details, in abstraction from how the resistor or pump itself works. The form–function distinction is evidently relative: the difference between an artificial heart and a natural one is one of form, from the point of view of an analysis of the circulatory system. But natural and artificial hearts work differently and, hence, themselves have different functional analyses, and these draw the form–function line in a different place. What is form at one level of analysis is function at another. Functional analysis, at any level, allows us to see how a complex system might be fixed, improved, or sabotaged by substituting more or less adequate functional equivalents for component parts or processes.[5]

Systematic accounts are rather liberal about function attribution (see Cummins, 1975). Functions are just the top half, as it were, of the form–function distinction: we have function, rather than form, when one is distinguishing what something does from what it is. Since containing systems may have different properties or capacities that need analyzing, and since something may be a constituent of more than one

containing system, things can have a multitude of functions relative to one or many containing systems. Relative to a system of medical diagnosis, the function of the heart may be to make thumping noises. That may be one of the things one needs to know to understand how that kind of medical diagnosis works. To some, this seems promiscuous: "*That's* not the [*the!*] function of the heart! That's not what the heart is for," they say. "*The* function of the heart is to circulate the blood. Systematic accounts cannot pick out *the* function of the heart from all the other things hearts do." We agree: it cannot. And we think this is as it should be. The idea that the heart is *for* something in particular is, we think, a vestige of an unscientific teleology (cf. Pennock, 2001).

It is, or course, perfectly possible to acknowledge that what we are calling functional analysis is both a useful and ubiquitous form of explanation in science and engineering, while denying that the analyzing capacities appealed to in such explanations are *functions*. That is, one could acknowledge what we are *calling* functional explanation without agreeing that it is *functional* explanation—explanation by appeal to functions—and, hence, without accepting our rather liberal account of function attribution. One could, for example, call it explanation by black-box analysis. Indeed, we are pretty sure that this is the majority position. In a later section, we will try to explain why we think the majority position gives us a messy picture of the relevant science. But it is really the explanatory strategy we are attached to. The rest of our position is mainly negative: it consists of what we do not like about selectionist accounts of function attribution and explanatory appeals to functions.

1.2 Selectionist accounts of functional explanation

Selectionist accounts hold that the function of a trait is the effect of having that trait for which it was selected (see Godfrey-Smith, 1994; Griffiths, 1993; Millikan, 1984, 2002; Neander, 1991, 2002). If the chain of magnetosomes in magnetotactic aquatic bacteria were selected for because they kept their anaerobic hosts from oxygen-rich surface water, then the function of those chains is to keep their hosts from oxygen-rich surface water. Thus, selectionist accounts attribute function only where there has been natural selection or, perhaps, artificial selection. The account is, therefore, biological and historical at its core: the current effects of a trait are irrelevant; what matters is the actual selection history. What we are calling functional analysis may appeal to a sparrow's or an airplane's wings role in enabling flight; but, on selectionist accounts, a strange selection history could reveal this as a mistaken function attribution. We prefer to accept the history, but hold on to the functional analysis.

Functional explanation, according to selectionists, is simply what is called ultimate explanation in evolutionary biology (Tinbergen, 1958/1984). The occurrence of a trait is explained as the result of natural selection: it appeared in some ancestral population, and spread through that population because of the positive contribution it made to the fitness of its bearers. The proximal effect (or effects) for which the trait was selected and maintained in the population is (or are) its function(s). Magnetotactic bacteria have magnetosomes because of the functions of those chains of magnetically polarized bits of ferrite.

Some caveats: (1) Traits may, of course, spread through a population without being selected for. Such traits have no functions, according to selectionist accounts. (2) The

circumstances that account for the selection of a trait do not account for the appearance of that trait in the first place. Selection can only select what is already there. Thus, it is not the appearance of a trait that functional explanation explains, according to selectionist accounts of function, but simply its spread through the population. Natural selection can, of course, explain the appearance of a trait as well, but this will not count as functional explanation on selectionist accounts, because "first appearances" must be explained by the selection of something else, namely, the elements in the developmental recipe for that trait, as well as the recipe itself.

2 Functional Attribution: Meeting the Explanatory Constraint

According to the selectionist, we can sometimes explain the presence of a trait in an individual or population by appealing to its function. So understood, selectionist explanation is apparently an instance of teleological explanation. The modern debate over functions was originally motivated by the suspicion that teleology—the idea that one can explain why something happens or why it is there by appeal to its function or purpose—is unscientific (see Hempel, 1965; Nagel, 1961; Woodfield, 1976). Acorns do not grow into oaks because that is what they are supposed to do. Such explanations appear to appeal to intentions and *intelligent design*[6] of the kind that is either absent in nature or itself in need of explanation. Thus was born the philosophical project of *naturalizing* teleology, i.e., of rendering it commensurable with hardheaded physical science. Early efforts along this line such as those cited above assumed that appeals to functions get into science in the context of teleological explanation, so the project of naturalizing teleology carried with it the project of naturalizing functions. This is part of what we deny: functions, we think, have a life outside teleology, in functional analysis. In that context, they evidently have no need of naturalization. It is *teleology* that is suspect and, hence, any appeal to functions arising in the context of teleological explanation.

Teleological characterizations of biological traits are ubiquitous in science and everyday life: eyes are for seeing, ears for hearing, hands for grasping, teeth and jaws for chewing. Contemporary teleological explanations of biological traits go beyond merely attributing functions to traits, however. Such explanations attempt to account for the spread of a trait through a population by appealing to the function of that trait. In order for such attempts to succeed, there must be some process or mechanism whereby traits spread, and spread because of their functions.

Historically, it was the failure to find such processes or mechanisms that doomed teleological explanations in mechanics and developmental biology. Teleological mechanics attempted to explain motion in terms of the goals or "final causes" of objects, but Newton's gravitational explanation of motion showed that objects would move as they do regardless of their functions or goals (Coleman, 1978). Thus, appeals to function to explain motion were rendered idle, and final causes were banished from mechanics. Appeals to the behavior of goal-directed inner agents ("entelechies") to explain biological development fared no better. Not only were such appeals regressive (since the behavior of entelechies itself was explained teleologically), but developments in cellular and molecular biology showed that organisms would

develop as they did, with or without entelechies. As in mechanics, development was shown to be insensitive to function, and so appeal to goals or functions was explanatorily empty.

Given the dismal track record of teleological explanations in science, why do teleological explanations hang on in evolutionary biology? According to the selectionist, appeals to function to explain the spread of a trait are legitimate because there is a function-sensitive natural process that spreads traits: natural selection. Since there is no real dispute over whether natural selection explains the spread of traits via the contribution of effects of traits to fitness, denying selectionist explanations of trait spread looks tantamount to denying natural selection. We have no problem with natural selection. So, if selectionists see functional explanation as simply a standard application of natural selection, then we can have no objections to selectionist accounts of functional explanation, so understood.

So why do we find selectionist accounts of functional explanation troubling? We do not have a deep, principled objection. Our objection is rather that what we will call *neo-teleology* (Cummins, 2002)—the idea that some traits are "there" because of their functions—tends to be misleading when coupled with standard examples of function. This objection has two parts. We have already covered the first, in passing. Selectionist accounts of function restrict appeals to functions to selection scenarios, while we think appeals to functions in black-box analysis—the abstraction from form and the focus on interactions in complex systems—is ubiquitous, important, and deeply entrenched. The suggestion that these are somehow not "real" functions is silly. The suggestion that there is *more* than black-box analysis involved in the appeal to functions in evolutionary biology is another matter, and this brings us to the second part of our objection.

It seems obvious that wings are for flight. But it is not at all obvious that, for example, sparrows, or birds in general, have wings because they enabled flight in an ancestral population. While it may be plausible to suppose that, at some point in time, a sub-population of the ancestors of today's birds developed wing-like structures ("proto-wings") that enabled a crude sort of flight, those structures are nothing like contemporary bird wings. Contemporary bird wings are the result of selection acting on variations in wing structures, all of which enables flight. Selection did not act on differences in function (enabling flight), but on differences in how well that particular function was performed.[7] Since selection acted on variations in traits that all shared the same function, appeal to function cannot explain why traits, for example, contemporary bird wings, are there.[8]

This objection can be avoided: one could retain the selectionist account of functions, and conclude that the function of the sparrow wing is not to enable flight. If selection is operating to discriminate among variants, it is *at the level of variation* that the relevant functions must be attributed, namely, flight-in-this-particular-way vs. flight-in-that-particular-way. Those two functions are *not* shared by the two variants.

But this tactic has its costs. It flies in the face of almost all of the function attributions we actually make, attributions that would be licensed by functional analysis. It seems obvious that the functions of biological traits, structures, and designs are often known without knowing what, if anything, was actually under selection (vs. merely hitch-hiking) and for what (better flight, mate attraction, durability,

developmental cost). This, we think, is because the form–function distinction is often pretty obvious: wings enable flight in sparrows, bats, and butterflies. There are serious issues surrounding how to discover what is under selection and why, but there seems no reason to burden our relatively transparent function attributions with these issues while, at the same time, banning such talk from black-box analysis. What is to be gained? Does calling the effects of traits that explain their spread "functions" provide any explanatory leverage that is not already provided by a combination of black-box analysis, selection, and development? We are skeptical that it does.

What biology needs are the resources to explain how the effects of an organism's traits in a particular environment contributed to the reproductive success or failure of that organism. In order to tell that story, biologists need to know what the various capacities of organisms were, how those capacities were exercised by organisms, and how the interaction of organisms with those capacities with a particular environment affected fitness. What biology requires, in short, is functional analysis. Of course, after having actually done the science, we can feel free to give a new name to those effects of traits that explain spread, if we wish (perhaps we can call them "selectionist functions' "). In so doing, we can say that the selectionist function of a trait explains why it spread throughout a population, but at the cost of trivializing the appeal to such a function, for now the selectionist function is simply being picked out as whatever effect it was that explains spread. Function attributions, so understood, do not do any additional work when it comes to telling the evolutionary story; they simply piggy-back on whatever the science reveals to be the relevant effects.

The kinds of function attributions that functional analysis yields, by contrast, are not so dispensable, and for reasons selectionist accounts must admit. If the goal is to explain why a trait spread by appealing to certain of its effects, then we need to be able to show how those effects of traits contributed to the capacities of their containing systems (viz., organisms), which is precisely what functional analysis delivers. And once we understand how something works in the way provided by a functional analysis, we understand how others might be constructed and how other instances of the same design could perform the same task, or do it better. Because the system and its components are specified functionally, we can see how substitution of functional equivalents at various points in the design—the result of natural or artificial variability in heritable traits—can make incremental changes in the system while preserving its overall viability. This is precisely how we must understand a system in order to see how it can evolve by natural selection.

3 Functional Attribution: Normativity

What is common to the selectionist and systematic accounts of function is that to ascribe a function to something is to do more than say what the thing does; it is to say what a thing is *for* and, thus, what it is *supposed to do*. This is the sense in which functions are normative, and any account of function attribution needs to accommodate this normative dimension. In evaluating the correctness of function attributions *vis-à-vis* normativity, there are at least two questions to keep in mind: (1) How well do such attributions square with judgments about cases, both in common sense

and in science? (2) How does the account of norms provided fare with respect to the role of norms in science generally?

3.1 Norms in systematic accounts

Systematic accounts relativize failures to function properly to a target *explanandum*:[9]

> component x is failing to function properly, relative to a capacity C of the containing system S, if (other things equal) S fails to have C (or has a relatively diminished capacity) because of what x is doing.

Thus, systematic accounts allow for a kind of relativized or instrumental normativity: what something needs to do for the containing system to exercise the target capacity (e.g., multiply, produce Fords, block serotonin re-uptake). Because such accounts offer an *a*historical notion of norms, they can easily accommodate the intuition that people knew wings are for flight and eyes for seeing *long before* they knew anything about selection.

A common complaint against this view is that such accounts attribute (or would attribute) functions where common sense would deny them—such accounts are too liberal. For example, relative to a medical diagnosis, we would say that the function of the heart is to make a thumping noise. In attributing such a function, we are claiming that the heart is supposed to make a thumping noise. The objection is that hearts are not supposed to make thumping noises; that is not what hearts are for. We think this objection reflects the interest relativity of function attribution: some capacities of containing systems are emphasized more than others and, so, when we talk about a/the function of a trait, we tend not to relativize the attribution to a capacity explicitly; the salient explanandum goes without saying. To damage systematic accounts, one needs to defend the idea that some capacities, but not others, *really* count when it comes to function attribution. We think such a defense cannot be made, a point we will return to later.

A perhaps more serious objection is that this sort of instrumental normativity—viz., the you-ought-to-do-x-to-achieve-g sense—will not accommodate the fact that a blind person's eyes are still for seeing, or that the function of a sperm is to fertilize an egg even though few ever do it, for example.

We will begin with blindness. The objection is that since the eyes of blind people never perform the function of enabling sight, systematic accounts should deny that a blind person's eyes are for seeing (and, thus, deny that the eyes are not functioning properly). To us, this appears to rely on a type–token ambiguity. Eyes generally (the type) enable seeing. A blind person's eyes (here, the token) are not *for anything* in *that individual*. If, for reasons too gruesome to imagine in detail, our descendants are all blind, then eyes will be, like the appendix, vestigial. Like the appendix, they will have no function. They will have *had* one, but they will have lost it. The sense that the eyes of a blind person are for seeing is simply the recognition that other humans do see, and that the eyes are an essential part of the human visual system. Thus, the blind person's eyes are not functioning properly (assuming here that the problem is really with the eyes) because they are not functioning in the way required for humans to see. The eye is for seeing (type); the blind person's eyes are not (token). The same can be said for color blindness, deafness, and so on.

The same treatment applies to the sperm case. Individual sperm are not for anything in an individual. Sperm, generally, (the type) enable reproduction. Imagine that cloning replaces sexual reproduction as the main form of human reproduction. Human sperm become vestigial. They lose their function. The sense that individual human sperm are for fertilizing eggs is simply the recognition that other human sperm do fertilize eggs, and that fertilization is an essential part of the human reproductive system. Thus, sperm that fail to fertilize eggs are not functioning properly (assuming here that the problem is really with the sperm) because they are not functioning in the way required for humans to reproduce. If we imagine a functional analysis of the reproductive system that is designed to explain reproduction, it will feature a sperm fertilizing an egg. That is what needs to happen for the whole thing to work. The analysis will say that the job of a cloud of sperm is to achieve fertilization, and will emphasize that the system makes up, in numbers, what is lacking in individual efficiency. Asking for the function of an individual sperm is like asking for the function of an individual oil molecule in the oil pan in your car. It reveals a misunderstanding of how the system actually works.

To repeat, we have no objection to ultimate explanations; no objection, that is, to explaining why something works the way it does by appeal to development, learning, and evolution. Admittedly, you can do this by refusing to treat the workings of the constituents of complex systems as functions, and save the word for selected-for effects of traits and structures. But you pay a two-fold price: (a) you fly in the face of ubiquitous function talk in black-box analysis; and (b) you fly in the face of the fact that functions, as ordinarily attributed, do not track selection, i.e., that selection is typically selection among things that share a function as functions are ordinarily understood. This does not make selectionist thinking wrong; it just makes it potentially misleading, and not well-poised to interface with the rest of science and common sense.

3.2 Norms in selectionist accounts

Selectionist accounts of functions face a serious problem when it comes to making sense of norms in science. The problem can be put in the form of a dilemma: either the selectionist account offers an account of the appeal to norms in science that differs from the systematic account, or it does not. If it does not, then the dispute about norms is just a dispute about capturing intuitions about cases. If it does, then it must offer some non-instrumental notion of what a biological trait or structure is for or supposed to do. But what on earth could this be, and how could we make sense of it, short of saying that normativity is built into the very fabric of reality?

The virtue of systematic accounts is that, outside of perhaps biology and psychology, the only notion of norms that science is thought to need is the instrumentalist one that systematic accounts provide. It is the only one biology and psychology need as well. These sciences need to be able to explain how effects of traits contributed to the capacities of containing systems, and how the failure to produce those effects explains the failure of a capacity to be exercised (or how variations in effects of traits explain variations in the capacities of containing systems). The same point applies to the containing systems themselves. To say that a containing system is supposed to have a capacity is just to say that its having that capacity is necessary for the exercise of some other capacity of a larger containing system. At no point

do we reach an unrelativized effect or capacity that a trait or containing system is supposed to have.

Perhaps the selectionist can respond that, although function attributions are always relative to some capacity of a containing system, some capacities are more important than others—indeed, some are critical—in determining when it is appropriate (true) to attribute functions. But privileging the capacities of containing systems that mattered to selection gives us normativity beyond the instrumental only if we can make sense of the idea that organisms are supposed to have those capacities in some unrelativized sense. We agree, of course, that we can make sense of the claim that a creature needed to have certain capacities in order to survive and reproduce, relative to a certain environment. However, short of saying that the point or purpose of living is to survive and reproduce (that is the ultimate goal of life), surviving and reproducing are just among the many things an organism does.

Organisms are not *supposed to* be fit; to think otherwise reflects the pre-Darwinian mindset that ultimate ends or goals can be found in the natural world. But, of course, this is not what those championing selectionist accounts of functions have in mind. Rather, they have in mind that privileging selected for effects gives us an *explication* of the concept of function in evolutionary biology. It gives us an account that is in reflective equilibrium with normal informed function attributions in evolutionary biology. We doubt this: as standardly attributed, functions do not track selection, as we pointed out above. But we wouldn't be impressed, in any case. What philosophy has to offer here, we think, is a conceptual framework that enables an economical and fruitful way to express, and assimilate, the science and methodology, not a set of conditions that "captures" our (or their) intuitions about functions. We think the systematic account is clearly superior in this respect.

To repeat: we think the fundamental thing to understand about functions is that the form–function distinction enables a kind of abstraction that allows us to understand how complex systems work, and how they can be modified. Like Galileo's idea that geometry can discipline a system of representations in a way that mirrors the way nature disciplines a system of magnitudes, functional analysis is one of the central intellectual innovations that makes science and technology possible.

It is dangerous to start thinking that natural objects, processes, structures, or traits are *for* something. It inevitably suggests intelligent design. And it should. For this, after all, *is* the only source of purpose. Our artifacts, actions, and beliefs are often for something or other. But *we* are not for anything. Nor are our eyes of the flora that thrive within us.

Table 4.1 summarizes the differences between systematic and selectionist accounts of functions.

Postscript: Counterpoint

Perlman's paper confirms our initial suspicion that the current debate over functions has little or nothing to do with how we should understand the science. We all agree that traits have effects which contribute to the capacities of organisms, that having those capacities (in part) explains why an organism was more or less successful in reproducing, and that reproductive success accounts for the differential proliferation

Table 4.1: Systematic and selectionist accounts of functions

	Systematic accounts	Selectionist accounts
Explanandum	Capacities or properties of complex systems	The occurrence or presence of a trait or structure in an organism or artifact
Explanatory strategy	By an analysis of components that abstracts away from implementation details and focuses on what they do: black-box analysis (bb analysis)	By appeal to natural selection
Function attribution: Explanation	What something does in a particular bb analysis	An effect of a trait or structure that accounts for its having been selected for and/or maintained in a population by selection pressures.
Function attribution: Normativity	Relativized to the target *explanandum*: component *x* fails to function properly relative to a capacity *C* of the containing system *S* if (other things equal) *S* fails to have *C* (or has a relatively diminished capacity) because of what *x* is doing.	Fixed by selection history: failure to function properly is failure to manifest the effect(s) for which the trait/structure was selected.
Scope	Any venue employing bb analysis (every branch of engineering, medicine, physiology, ecology, chemistry, sociology, art, music, literature . . . pretty much everything except, perhaps, basic mechanics)	Evolutionary biology (and, perhaps, artifacts)

of some genotypes as opposed to others. So, if the science is not driving the debate, what is? We think there are two principal sources: (1) a desire to "capture the intuitions," and (2) the idea that explanation in biology is somehow special, *sui generis*, a place where teleology of a sort actually works (Cummins, 2002).

Regarding (1), the alleged defects in systematic accounts of function attribution are that they are too liberal in attributing functions, and that they cannot accommodate the normative nature of functions.

With respect to the charge of *liberality*, we agree that the account does not square with intuitions about functions, in many cases. Relative to the capacity of some systems of medical diagnosis to identify disease, heart sounds do have a function, according to systematic accounts, and many find this counterintuitive. But how seriously should we take this? Scientific treatments of motion have increasingly diverged from intuition since the seventeenth century. The proper response is surely to say, "So much the worse for intuitions," and it is the proper response here. Biology should be no more constrained by intuitions concerning functions than physics should be constrained by intuitions about motion. Physics is, in large part, counterintuitive and,

so, it would be no knock against biology if it turns out it makes counterintuitive function attributions. Functional attributions should earn their keep by making for better science, not by placating the folk. Ironically, selectionist accounts of functions implicitly agree that intuitions can be unreliable, since there is no guarantee that a trait's selection history will track common-sense intuitions about that trait's function. That said, we are suspicious of the opposition's view of what the intuitions are. Evolutionary biologists probably will not say that a function of the heart is to make sounds. But an ethnologist studying medical diagnosis probably wouldn't blink an eye. This relativity to a containing system and target capacity is just what the systematic account would predict, if it were in the business of predicting intuitions. Predicting intuitions, however, is (or ought to be) the business of psychology and should not, in any case, be probative in the current dispute.

With respect to *normativity*, while we think systematic accounts can accommodate instrumental normativity, we are deeply suspicious of the idea that non-instrumental norms have a legitimate role to play in biology, or in any other science. You can make an instrumental norm *look* like a *Norm* by privileging a particular goal-state, but this is still just instrumental normativity—hence, relativized normativity—thinly disguised. We suspect that the feeling that there is an unrelativized Norm at work in evolutionary biology is simply an artifact of a choice of target. If you want to account for ("capture") the function attributions—including malfunction and failure of function attributions—of evolutionary biologists talking about natural selection, you can probably get a pretty good fit by relativizing to fitness, in one way or another. But this isn't evidence for a selectionist account of functions, unless you think functions are special to evolutionary biology. We think it is more important to see evolutionary biologists using an explanatory strategy ubiquitous in science—viz., black-box (functional) analysis—than it is to salvage the appearance of an outdated teleology.

Meanwhile, the alleged Norm contributes nothing to the science, but engenders considerable misunderstanding. On the one hand, explaining the evolution and development of sight in humans requires no reference to any Norm. On the other, we get the suggestion that there is something eyes are supposed to do. From the fact that eyes in a person's ancestors enabled them to see, and that this fact in part explains why the person has eyes, it does not follow that the person's eyes are supposed to do anything. There are things eyes do, and things ancestral eyes were selected for doing and current eyes are maintained for doing. But there is nothing that eyes are *supposed to do*.

This leads us directly to (2), the idea that evolutionary biology is somehow special in providing a home for a robust notion of function not available in other venues. Recall that, whereas what we are calling functional analyses are ubiquitous in science, selectionist accounts are more or less relegated to biological traits and designed artifacts. One does not have to be the village unificationist to appreciate the advantages of seeing function attribution in evolutionary biology as an instance of a common and well-understood strategy in science generally. Of course, we have already seen the source of the resistance to such replacement: biological explanation is supposed to have a teleological character that functional analyses lack.

We acknowledge that systematic accounts are not teleological in character. Our argument is simply that the alleged teleology, if it were there, would be an

embarrassment, not a virtue, and that there is no reason to suppose that it plays any substantive role in the powerful tool Darwin bequeathed to us. It is, to repeat, an illusion created by the strategy of accounting for some (suspect) intuitions by, in effect, relativizing function attributions to fitness. Darwin didn't show us how to ground teleology; he showed us how to get along without it.

Notes

1 Buller (1998) has argued that it is fitness, rather than selection, that is required to ground functions. Because our argument is directed against any theory of functions that appeals to past advantage, our arguments against selectionist accounts of functions apply to fitness accounts as well, and sometimes our points are couched in terms of fitness rather than selection.

2 Teleology is undoubtedly older than functional analysis, which probably did not become common until the introduction of relatively complex artifacts. It was then possible to think of natural systems as having a functional structure ("design") analogous to complex machines; indeed, to think of the body and its organs as natural machines.

3 In this model, insulin stimulation results in the activation of a PI 3-kinase-dependent pathway that is necessary but not sufficient to induce GLUT4 translocation. In parallel, the insulin receptor activates an additional pathway leading to Cbl tyrosine phosphorylation through its interaction with the CAP protein (Syn4, syntaxin 4; PI3-K, PI 3-kinase).

4 This is the sense of "function" at the heart of functionalism in the philosophy of mind. The idea is that our mentalistic vocabulary is a functional vocabulary: it identifies mental states and processes in terms of what they do, not what they "are." Mental concepts, thus, resist physical reduction for the same reason function concepts generally resist reduction: physically disparate things can have the same function. Since the prospects for physical reduction for *doorstop* are no better than they are for pain or belief, and since no one wants to be a dualist about doorstops, failure of reduction is no argument for dualism.

5 This, as it turns out, is just what we need to understand how natural selection works, a point to which we return below.

6 We use this phrase *on purpose* to emphasize that teleology, as an ultimate explanatory strategy, is still with us.

7 Even this is tricky: a variation in wing design might be selected for even though it did not improve flight, but, for example, because it made wings more durable or less developmentally expensive, or more attractive to the opposite sex.

8 Perlman (this volume) makes the point that, since it was selection pressure for better flight that drove the design improvements in wings, the function of wings (in the selectionist's sense) explains why contemporary wings have *the specific designs they do*. We have no objection to this point.

9 The *explanandum* is the thing to be explained, while the *explanans* is the explanation itself or the thing doing the explaining. For example, "The Earth revolves around the sun in a fixed orbit [*explanandum*] because of the sun's immense gravitational pull upon it [*explanans*]."

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PART III
ARE SPECIES REAL?

Introduction

"There is probably no other concept in biology that has remained so consistently controversial as the species concept"—so claimed the famous biologist, Ernst Mayr, back in 1982 (p. 251). The same is still true today, without a doubt. Most of us take it for granted that there are obvious ways to define a species and distinguish one species from another. Further, many people assume that species are really things, existing in their own right. Consider that, at a very early age, children are already able to distinguish different types of animals: an elephant is different from a lion, which is different from a butterfly. So, there are elephants, lions, and butterflies that really exist at the zoo, but then we may think that there are the species *elephant*, *lion*, and *butterfly* which really exist, somehow, too. Aristotle (1995a, 1995b, 1995c), for example, thought that species were real things, with real essential characteristics which enabled people clearly to distinguish one species from another, and this kind of thinking was entrenched in biology until Darwin. Given the fact of evolution (especially macroevolution, see Part V), the essential nature of species has been called into question, along with any universally applicable definition of the term.

In recent years, there have been several so-called *species concepts* that have emerged whereby thinkers have tried to specify the necessary and sufficient conditions for the definition of species, with many of these attempts failing. Jerry Coyne and H. Allen Orr do a detailed job of laying out all of the standard species concepts, complete with their problems, in the book titled *Speciation* (2004). For example, one of the oldest ways in which to distinguish one species from another has been through noting basic differences in shape or form. This is how Aristotle did it, and probably how we can distinguish elephants from lions and butterflies at such an early age. This way of distinguishing species has been termed a kind of *morphological species concept* (*morpho* is the Greek word for "shape" or "form").

Without a doubt, the most influential species concept for the biological sciences in the twentieth century was Ernst Mayr's *biological species concept* (BSC). According to the BSC, one species *A* is defined and distinguished from another species *B* by virtue of the fact that the members of species *A* can breed in nature *only* with other members of species *A*, and not with species *B* or any other species *C*, *D*, *E*, etc. Thus, species *A* is reproductively isolated from species *B*, *C*, *D*, *E*, etc.—and vice versa—and that is how we can distinguish one species from another. The BSC has encountered numerous problems; an obvious one being that there would be no way to classify fossils according to this standard since there is no way to check for reproductive isolation among the dead and buried! Yet, we still think that a fossilized *T. rex* is a distinct species from a fossilized stegosaurus or member of the Australopithecene lineage, like the famous Lucy.

In the first paper of this part, Michael Claridge recognizes the importance of the BSC, noting that: "Interbreeding in the field, crossability, is the important criterion for determining species status, but this cannot be absolute." Following Mayr (2004), he claims that the various species concepts are simply recipes for recognizing particular species taxa and that many people have thus confused species concepts with species taxa.

In his paper included in this part, Brent Mishler claims that species "properly defined *are* real entities, but not *uniquely* real" and argues that the "so-called 'species problem' is really just a special case of the taxon problem. Once a decision is made about what taxa in general are to represent, then those groups currently known as species are simply the least inclusive taxa of that type." Ultimately, Mishler advocates something called the PhyloCode, a completely rank-free phylogenetic classification that he thinks is "far better for teaching, research, communicating with other scientists, and interfacing with the larger society."

It may be that the definition and nature of species will always be a problem for researchers because of the "subjective dependence" of species concepts on the particular view of the taxonomist, as noted by Claridge. Yet, given the myriad attempts at a clear and coherent *universalizable* definition of species still occurring, it may be that researchers are resistant to this subjectivity.

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CHAPTER
F I V E

Species Are Real Biological Entities

Michael F. Claridge

Species concepts are central ideas to most areas of biology and are certainly not just the property of systematists. Biological systems are all characterized by variation and are thus complex. Species concepts must take account of this variation and complexity. The term "species" derives from classical Greek logic, which dictates that they are absolutely distinct from other species with no overlap. This *typological* approach also characterizes many more modern concepts of species, but cannot take account adequately of biological variation. Most biologists accept a broadly biological species concept where species is that level in divergent evolution when two lineages are genetically divergent and intergrading, or interbreeding, between them is rare or non-existent. Interbreeding in the field, crossability, is the important criterion for determining species status, but this cannot be absolute. Problems of geographical variation and non-sexual reproduction greatly complicate the problems, but that is the nature of biological systems. Ranks of classification above, or indeed below, the species level are much more subjective than the species itself and depend on the views of particular sytematists and the characters that they use. Ranks above the species are purely relational within particular groups of organisms and lack the biological reality of species.

[T]he Swiss sometimes find it difficult to say exactly where the Jungfrau and neighbouring Monch mountains start and stop, but this does not lead them to doubt the reality of these two mountains because their limits are unclear.

M.H.V. van Regenmortel, "Viral Species" (1997)

1 Introduction

A fundamental feature of biological systems that differentiates them most obviously from physical ones is the all-pervading presence of variability. Variation is characteristic of living organisms at all levels of organization. As the late Ernst Mayr

pointed out forcibly many times during his long life, and most recently in 2004, any philosophy of biology must be able to accommodate both genetic and evolutionary variation. The earliest biologists in Europe naturally followed the classical Greek philosophers, most particularly Plato and Aristotle, in their systems of logical division and the consequent absolute separation of different resulting categories. One of Mayr's greatest contributions was repeatedly to make quite clear the need for population thinking in systematics and evolutionary biology (e.g., Mayr, 1942, 1963, 1982, 2004). Animals and plants in nature exist as populations of individual organisms. These organisms show statistical patterns of variation in their various characteristics, both within and between populations. Since the eighteenth century, the so-called "species problem" has been essentially an argument about and between different philosophies of biology.

No topic in evolutionary and systematic biology has been more contentious and controversial than the nature and meaning of *species*. In the words of Ernst Mayr (1982), "There is probably no other concept in biology that has remained so consistently controversial as the species concept" (p. 251). One of the aspects of the species problem that has made it, on the one hand so intractable, but, on the other, so rewarding is that it is not only a very practical problem for all taxonomists and biologists, but also a deeply philosophical and theoretical one (Hey, 2006). In addition to arguments about different philosophical approaches, much of the controversy has centered on the confusion between, on the one hand, the philosophical concepts of species and, on the other, the practical recognition of species taxa themselves. The frequent confusion of these two different aspects of the species problem continues to cause much argument and controversy among biologists and philosophers. So much so that the expanding interest in these problems over the past 10 or 20 years has led to the publication of many relevant books and review articles, notably in Claridge, Dawah, and Wilson (1997c), Ereshefsky (1992), Foottit and Adler (2009), Paterson (1993), Wheeler and Meier (2000), and R. Wilson (1999). Species are widely regarded by biologists as the primary units of biodiversity and conservation (E. Wilson, 1992, 2005), so it is obviously practically important, so far as possible, that we agree on their nature. Perhaps surprisingly, most biologists and philosophers, often with very different attitudes to species concepts and definitions, have broadly agreed that species taxa represent attempts to recognize real biological entities in the field, the units of diversity and conservation of E.O. Wilson. This view, which might be seen as the traditional one, has been attacked strongly in recent years particularly by Mishler (1999) and Mishler and Theriot (2000a, 2000b, 2000c), but also by Ereshefsky (1999). On the other hand, it has been defended by me and my colleagues (Claridge, 2009; Claridge, Dawah, & Wilson, 1997a, 1997b), and, from a quite different standpoint, by de Queiroz (1998, 1999, 2005). In this paper, I again defend the traditional view. To do this, it will be helpful to review briefly the history of the application and development of species concepts in biology. This review section is largely based on a recent paper of mine on insect species (Claridge, 2009).

2 Early Species Concepts–Linnaeus

The term *species* is a very old one and derives from the writings of classical Greek philosophers, most notably Aristotle (see Cain, 1958). It was natural for scholars and

naturalists of the seventeenth and eighteenth centuries to adopt the systems of Aristotelian logic in attempting to classify and make sense of the natural world. This was taken at the time as the only possible system of reasoning, despite the fact that such logic requires that all entities classified must be absolute and clearly separated from other comparable entities with no overlap between them. Technical terms from Aristotelian logic that were used in attempts to classify living organisms included *definition*, *genus*, *differentia*, and *species*. Here the *genus* referred to the general kind, while *species* referred to the particular kind within the genus, as qualified by the *differentia*. Carl von Linné (1707–1778), better known to us as Linnaeus and the founder of the binomial system of nomenclature that we still use for living organisms, clearly documented his principles and practice in producing classifications of both animals and plants (Cain, 1958, for a full account). Indeed, the binomial system itself is a result of the use of the Aristotelian system. Authors previous to Linnaeus, and Linnaeus himself in his early works, had given multinomial names to organisms; the genus being one word, but qualified by a descriptive phrase, the *differentia*, to describe and delineate particular species. Linnaeus, from the tenth edition of the *Systema Naturae* in 1758, probably primarily because of the pressures to describe such large quantities of new material, reduced the differentia for animal species to a single word—the specific name—and so established the binomial that has been used for plants and animals since then and continues to this day, despite a spate of criticism.

To Linnaeus, species were simply the lowest category of particular kinds in his classifications, though he did also in practice often recognize *varieties* within species! Not only did Linnaeus publish classifications and descriptions of many animals and plants, but he also wrote books in which he detailed his methods and philosophy (e.g., Linnaeus, 1737/1938). It is clear that Linnaeus, as a practicing taxonomist, frequently found it difficult to adhere to his strict philosophical principles, and his works were sometimes an uneasy compromise between philosophy and pragmatism (Cain, 1958). In a detailed study of the many writings of Linnaeus, Ramsbottom (1938) showed that in developing a practical concept of species, he recognized three main criteria. Species were: (1) distinct and monotypic; (2) immutable and created as such; and (3) true breeding. Criteria (1) and (2) here are to be expected in pre-evolutionary philosophy, but (3) may be a little more surprising and clearly results from practical field experience. The idea that species had a single norm of morphological variation and were clearly each distinct from, and did not overlap with, other species within the same genus was widely accepted following the broadly typological thinking of the times (Mayr, 2004) deriving directly from Aristotelian logic.

Linnaeus was, of course, working in very exciting times when European explorers were travelling widely in regions of the world previously unknown to them and bringing back large collections of plants and animals for study. For obvious reasons these samples consisted of dead and often poorly preserved material. Thus, in order to describe new species and to classify them, early taxonomists had little recourse but to use only morphological characters. The total immutability of species was widely accepted in the eighteenth and early nineteenth centuries, but it is now clear that even Linnaeus later in his life developed some complicated theories of speciation by hybridization (Cain, 1993).

Before the enormous influx of largely tropical material into European museums, most early taxonomists, including Linnaeus, were field naturalists themselves and familiar

with the organisms on which they worked as living entities. They certainly knew that the species they described from local fauna and flora using morphological characteristics to differentiate them were also biologically distinct and differed in obvious features of their natural history, in addition to the differences observed in their museum specimens. The swamping of museums by large collections from overseas inevitably led to the almost exclusive use of morphological differences both to describe and to recognize species. This obviously still remains so today for most groups of animals, and particularly for species-rich groups such as insects. The morphological species or morphospecies has evolved from these early classifications solely for reasons of convenience. Interestingly, today such "morphospecies" ideas are now being developed further by the use of molecular characters for recognizing species (see Blaxter, 2004; Tautz, Arctander, Minelli, Thomas, & Vogler, 2003). It is important to emphasize that the morphospecies is not a philosophical concept, but simply a practical methodology used to differentiate species taxa. No doubt Linnaeus himself would have been very unhappy that his philosophy should be reduced to such a purely practical matter! Following the lead of Linnaeus and his many followers, taxonomists were forced more and more to use the practical morphospecies for what they largely knew only as dead museum specimens. The amount of difference required to recognize and separate species became inevitably more and more subjective, as illustrated by different variants of the well-known quote, "A species is a community, or a number of related communities, whose distinctive morphological characters are, in the opinion of a competent systematist, sufficiently definite to entitle it or them to a specific name" (Regan, 1926, p. 75).

Unfortunately, as taxonomy and systematics were, through force of circumstance, based more and more only on morphological differences between dead museum specimens, so two quite different traditions of studying the natural world diverged, with what we might call the morphologists on the one side and the naturalists on the other. Naturalists, even in the late eighteenth century, were well aware that species had some real biological basis in the field. For example, Gilbert White (1789) first showed that several morphologically very similar species of song birds in Britain of the genus *Phylloscopus*, the warblers, were very clearly separated in the field by their quite distinctive male songs, now known to function as important elements of their specific mate recognition systems. This interest in breeding barriers and species as reproductive communities was an essential element of the naturalist tradition. Later in the nineteenth-century English-speaking world, most notable as part of the naturalist tradition were Charles Darwin and Alfred Russell Wallace. It was from this tradition that they independently developed the theory of evolution by natural selection and accumulated overwhelming evidence for descent with modification. After the general acceptance of evolution, species were then recognized as the end terms of different lines of descent. The controversies around evolution meant that the nature of species was not regarded at that time as a high-priority subject. Darwin himself certainly normally regarded species as more or less arbitrary stages in the process of evolutionary divergence.

3 Biological Species Concepts

In addition to developing the idea of species as morphologically discrete entities, Linnaeus had the rather more vague idea of species as breeding units that generally

breed true (Ramsbottom, 1938). However, it was not until the late nineteenth and early twentieth centuries—largely through the writings of the British entomologists Karl Jordan (Mayr, 1955) and Sir Edward Poulton—that these ideas were clarified and became central to species philosophy. Probably the most important contributor to this way of thinking was Poulton. First in 1904, in his Presidential Address to the Entomological Society of London, and later expanded in a volume of essays on various aspects of evolutionary biology (Poulton, 1908), Poulton made the most important advance toward what has since become known as the *biological species concept*. He emphasized the importance of interbreeding in the field as the most critical species criterion. This was what later Mayr (1942, 1963) termed *crossability* and contrasted strongly with *interfertility* or simple ability to hybridize. Poulton was one of the first authors to make this clear differentiation and thus effectively to develop the modern biological species, for which generally he receives insufficient credit (Claridge, 1960; Mallet, 1995; Mayr, 2004).

During the early and mid-twentieth century the revolution in evolutionary thinking, often known as the evolutionary synthesis (Mayr & Provine, 1980), was developed by the attempted unification of systematics, genetics, and evolution, exemplified by the publication of major seminal volumes, including *Genetics and the Origin of Species* (Dobzhansky, 1937) and *Systematics and the Origin of Species* (Mayr, 1942). The so-called "biological species concept" was central to these ideas and was formulated by Mayr (1942): "[A] species consists of a group of populations which replace each other geographically or ecologically and of which the neighbouring ones intergrade or interbreed wherever they are in contact or which are potentially capable of doing so (with one or more of the populations) in those cases where contact is prevented by geographical or ecological barriers" (p. 120). More useful from a practical point of view, and certainly the most widely cited since the original publication, is his shorter definition: "[S]pecies are groups of actually or potentially interbreeding natural populations which are reproductively isolated from other such groups" (p. 120). Reproductive isolation in nature, as also for Poulton, was the key factor in identifying and maintaining species as discrete entities. Such reproductive isolation was maintained by what Dobzhansky (1937) termed *isolating mechanisms*, which were any attributes of species populations that reduced the likelihood of interbreeding between them. Clearly such a category is a very broad one, including not only all sorts of post-mating genetic incompatibilities as well as behavioral and ecological differences that act before mating and fusion of gametes, but also totally extrinsic geographical barriers. The latter are clearly not properties of the organisms and are not now generally classified with the intrinsic factors. To Dobzhansky, speciation was the origin of reproductive isolating mechanisms and, thus, of reproductive isolation. Dobzhansky's system of classification of isolating mechanisms was followed and modified by many authors during the twentieth century, including particularly Mayr (1942, 1963, 1982) and Cain (1954).

A major set of criticisms of the biological species concept has been developed over some years by Hugh Paterson (see Paterson, 1985, 1993). One of his main concerns is with the concept of species isolating mechanisms and with the implication that they have evolved as adaptations under natural selection to achieve and maintain reproductive isolation. Clearly Paterson must be correct, at least for all so-called "post-mating mechanisms," which logically cannot be due to such adaptation (see also Claridge,

1988; Mallet, 1995). Avise (1994) and, more recently, Coyne and Orr (2004) have suggested the neutral term *isolating barrier* to replace *isolating mechanism*. This is a useful suggestion, but does not deal with Paterson's further criticisms. Maybe there is no need anyway now for a term to include such a diversity of phenomena?

Paterson (1985) regards species as groups of organisms with common fertilization systems: "We can, therefore, regard species as that most inclusive population of individual biparental organisms which share a common fertilization system" (p. 25). He recognized an important subset of the fertilization system that he termed the *specific mate recognition system* (SMRS), "which is involved in signaling between mating partners and their cells." Thus, the often complicated reciprocal signals and signaling systems of mating and courtship (well-documented and reviewed by ethologists including Brown, 1975; Eibl-Eibesfeldt, 1970; and Tinbergen, 1951) have ensuring specific mate recognition as one essential function. In such behavior sequences successive signals release in turn successive responses via tuned receptors in the opposite sex. These sequences are usually, but not exclusively, initiated by males. Unless appropriate responses are received at each stage and the signals are recognized as appropriate, exchange will be terminated and ultimate exchange of gametes will not occur. The exchanges of signals between partners may be broken off at any stage. Thus, to Paterson, species are defined by their unique SMRSs, and the evolution of new species, speciation, is the origin of new SMRSs. He has argued at length over the years that his concept of species is quite distinct from the biological species *sensu* Mayr. He terms the latter the *isolation concept*, because it is defined by reproductive isolation from other species, and terms his own system the *recognition concept*. Lambert and Spencer (1995) and Vrba (1995) have strongly supported this line of argument, but others have doubted the clear demarcation between isolation and recognition concepts (Claridge, 1988, 1995a; Claridge et al., 1997a; Coyne & Orr, 2004; Coyne, Orr, & Futuyama, 1988; Mayr, 2000a, 2000b, 2000c).

Thus, in practice a broadened biological species concept would recognize that different species are characterized by distinct SMRSs which result in the levels of reproductive isolation between sympatric species observed in the field. However, species taxa are only rarely recognized by direct studies of the SMRS, which are themselves equally rarely understood with any certainty, but they are the final arbiters for determining biological species boundaries (Claridge, 1988, 1995a, 2009; Claridge et al., 1997a, 1997b). Normally, biological species are recognized by markers that are thought to indicate the existence of reproductive isolation. In the past, these were usually morphological markers, so that, after the evolutionary synthesis of the mid-twentieth century, taxonomists hypothesized that the differences they recognized and used to separate morphospecies were also indicators of biological species boundaries. More recently, morphological markers have been supplemented by a wide range of others, including cytological, behavioral, and biochemical ones. In particular, increasing use is now made of molecular markers involving characters derived from the amino-acid sequences of specific pieces of DNA (Avise, 1994). Indeed, it has even been suggested that all species taxa should be diagnosed by such molecular differentiation (e.g., Blaxter, 2004; Tautz et al., 2003). However, the enormous diversity of markers currently available to taxonomists simply indicate levels of reproductive isolation. Levels of gene flow between populations, and therefore levels of reproductive isolation, are now routinely estimated by molecular divergence.

A particular feature of the biological species concept is that reproductive isolation may occur between species populations without any obvious accompanied morphological differentiation. This phenomenon of real biological species existing in nature without obvious differentiation to the human observer has been recognized since early in the twentieth century and was well discussed by Mayr (1942). Such species are usually known as *sibling* or *cryptic* species and have been clearly demonstrated in many groups of living organisms, most particularly insects (Claridge, 1960, 1988, 2009; Claridge et al., 1997b; Mayr, 1942; Thorpe, 1940).

During the primacy of the biological species in the middle years of the twentieth century, a large body of opinion among museum taxonomists was nevertheless opposed to it (e.g., Sokal & Crovello, 1970) and preferred either some overtly morphological species approach or a purely phenetic one. Such views have always been strongly supported by botanists on the grounds that interspecific hybridization is so common in plants that reproductive isolation is not a useful criterion (Gornall, 1997; but see Mayr, 1992). However, entomologists have also often led the criticism of the biological species.

There are two important problems in the practical use of biological species that are acknowledged by all of its proponents. These concern the status of (1) asexual and parthenogenetic forms and (2) geographically or spatially isolated (allopatric) populations:

1 *Agamospecies*: The biological species in its various manifestations can only be applied to biparental sexually reproducing organisms in which a distinctive SMRS leads to reproductive isolation. Neither asexual nor obligate parthenogenetic organisms have a functional mate recognition system that leads to the fusion of gametes, so that the biological species cannot strictly apply to them. These organisms exist as clones which may differ in morphology, biochemistry, cytology, behavior, ecology, etc. (Foottit, 1997), and which, contrary to some opinion, may show considerable genetic variation (Loxdale & Lushai, 2003). Although distinctive and diagnosable clones are often described as species, they cannot truly be biological species. They have, however, been given the useful name agamospecies (Cain, 1954). Such agamospecies often differ between themselves in important features of behavior, such as feeding preferences and ecology (de Bach, 1969). They are thus practical categories like the morphological species. Many groups of living organisms, including most micro-organisms, can only be agamospecies, but of course such organisms show obvious patterns of variation and are clearly subject to evolution.

2 *Allopatric forms*: A practical problem with applying the biological species is that reproductive isolation in the field can be determined only for sympatric populations, where alone there are possibilities of testing the effectiveness of SMRSs in the field. Geographical variation and the status of allopatric populations have long been of major interest to both taxonomists and evolutionary biologists. Degrees of observable differentiation between allopatric populations vary from almost nothing to large differences, at least comparable with those observed between distinct sympatric species of the same taxonomic group, but the criterion of gene flow and reproductive isolation in the field cannot be conclusively tested. Experimental crossings of allopatric forms under laboratory and experimental conditions yield results of only limited value.

The polytypic nature of biological species has for long been recognized, and a series of taxonomic categories, from superspecies to subspecies, has been developed to describe such essentially continuous geographical variation (Cain, 1954; Mayr, 1942). This continuum, on the one hand, has provided vital data for the development of theories of allopatric, or geographical, speciation (Cain 1954; Mayr, 1942); but, on the other, it has led many workers also to regard the species as no more than a rather arbitrary stage in the divergence of local populations. For example, Alfred Russell Wallace (1865), when confronted with the bewildering range of geographical variation and polymorphism in the swallowtail butterflies of South East Asia, in a widely cited quote, stated:

> Species are merely those strongly marked races or local forms which, when in contact, do not intermix, and when inhabiting distinct areas are generally believed to . . . be incapable of producing a fertile hybrid offspring. . . . [I]t will be evident that we have no means whatever of distinguishing so-called "true species" from the several modes of variation . . . into which they so often pass by an insensible gradation. (p. 12)

Interestingly Wallace (1889), in a wider discussion, later supported a much more biological type of species concept. Wallace, as of course also Darwin, was impressed with variation within and between natural populations as the basic material for evolution by natural selection. Equally today the allocation of allopatric populations within the superspecies/subspecies continuum is largely subjective. Drawing a line through any continuum must indeed be to some extent arbitrary. This is undoubtedly a practical weakness of the biological species, but I would argue equally that it is a weakness of all discrete species concepts.

These obvious complications, together with a frequent desire to eliminate the priority given to one set of organismal characters—the SMRS and resulting reproductive isolation—over all others, have persuaded many systematists to abandon the biological species concept in favor of what we might term a *general phylogenetic species concept* (Claridge et al., 1997a).

Before moving on to consider phylogenetic concepts in more detail, it is appropriate to discuss the interesting ideas of Mallet (1995) on his "genotypic cluster criterion" or concept of species. Mallet is a geneticist who has worked on widely distributed species and populations of tropical butterflies, like those studied earlier by Alfred Russell Wallace (see above). One of his concerns is that the biological species is absolute and does not allow for interspecific hybridization and intergrading, a subject which he himself has recently extensively reviewed (Mallet, 2005; 2008). However, since intergradation is the basis of the polytypic biological species espoused by Mayr, Cain, and others, this criticism cannot be a real problem. Even in sympatric interactions, re-productive isolation does not need to be absolute in order to maintain species integrity. Indeed, the acceptance of the reality of evolution demands that species cannot always be completely reproductively isolated. Intermediates and intergradation must be expected. Thus, all realistic species concepts must allow for such intergradation, and the broadly conceived biological species certainly does this.

Another interesting contribution, along similar lines to that of Mallet (1995), is the *genomic integrity species* definition of Sperling (2003). Here, species are "populations

that maintain their genomic integrity when they contact each other, even if they occasionally exchange genes" (p. 432). Clearly, biological species in the broad sense that I follow here must be both distinct genotypic clusters and maintain their genomic integrity, as argued by Mallet and Sperling, respectively. These various attempts to formulate more inclusive and realistic species concepts appear to me, then, to be quite compatible with, and indeed very similar to, the broadly based biological species concept advocated here.

4 Phylogenetic Species Concepts

A revolution in the philosophy and practice of systematics took place in the English-speaking world after the publication of Hennig's *Phylogenetic Systematics* in translation in 1966. Few systematists today do not use some variant of the cladistic methodologies pioneered by Hennig. Coincident with this widespread acceptance of cladistic methods for constructing phylogenetic hypotheses and making robust classifications came increased published dissatisfaction with, and rejection of, the biological species concept by some systematists (e.g., most authors in Wheeler & Meier, 2000). Oddly enough, Hennig himself thought of species as reproductive communities, so his species concept was broadly similar to the biological species of Mayr (1942)! Of course, Hennig was interested primarily in extending species back in time as diagnosable clades and lineages. In this he was developing what Simpson (1951) had begun as a broader evolutionary species concept based on phylogenetic lineages, which has since been taken up by many others (e.g., Cain, 1954; Wiley, 1978; Mayden, 1997; Wiley & Mayden, 2000a, 2000b, 2000c; and see Hey, 2006). Most recently, de Queiroz (1998, 1999, 2005) has developed what he terms the *metapopulation lineage concept* based on similar reasoning.

Cladists have certainly not spoken with one voice on the nature of species. Hennig (1966) saw species as that unique level in the taxonomic hierarchy at and above which cladistic methods could be applied to determine phylogenies and below which they could not. Within species, interbreeding relationships dominate, and these Hennig differentiated from phylogenetic relationships as *tokogenetic* ones, a term that has not been widely adopted in the general literature. Many more recent cladists have followed this view of species. For example, Nixon and Wheeler (1990; Wheeler & Nixon, 1990) clearly stated that species are uniquely different from higher-level taxa, in that they lack resolvable internal phylogenetic structure. On the other hand, other cladists, including Nelson (1989) and Mishler and Brandon (1987), clearly state that the species represents just one rank in the taxonomic hierarchy and is of no more or less significance than any others, such as genus, family, order, etc. Mishler (1999) and Mishler and Theriot (2000a, 2000b, 2000c) developed this line of argument in further detail. For example, Mishler (1999) concluded: "[W]e have no and are unlikely to have any criterion for distinguishing species from other ranks in the Linnean hierarchy, which is not to say that particular species taxa are unreal. They are real, but only in the sense that taxa at all levels are real Species are not special" (p. 309). In criticism of this view, Wheeler (1999, p. 136) commented that species "exist in nature is one aspect of species about which I can agree with Mayr (1963)," and most taxonomists also seem broadly to agree with this long-established view.

Many authors have attempted to formulate an expressly phylogenetic species concept. In a valuable volume devoted to a debate about species concepts and phylogenetic theory, proponents of three different such phylogenetic concepts, including what was termed the *Hennigian species concept* (Meier & Willmann, 2000) and two quite different versions confusingly each termed the *phylogenetic concept* (Mishler & Theriot, 2000a, on the one hand, and Wheeler & Platnick, 2000, on the other), presented their arguments and many disagreements, not only with the biological species concept and the evolutionary concept (supported by Wiley & Mayden, 2000a), but also with each other. In the same volume, Mayr (2000a) took a lone stand to defend his view of the biological species concept. Clearly, there are fundamental disagreements between the three sets of authors claiming to base a species concept on phylogenetic and cladistic theory. However, despite these disagreements, there is some practical consensus, and perhaps the most widely cited definition of the phylogenetic concept is that of Cracraft (1983, 1997), who stated that the species is the "smallest diagnosable cluster of individual organisms within which there is a parental pattern of ancestry and descent" (Cracraft, 1983, p. 170). Some critics have suggested that this definition applies only to individuals and not to populations, a view fiercely refuted by Cracraft (1997). Nixon and Wheeler (1990) also emphasized this when they defined phylogenetic species as "the smallest aggregation of populations (sexual) or lineages (asexual) diagnosable by a unique combination of character states in comparable individuals" (p. 218). Thus, to me it seems that the essence of the dominant phylogenetic concept in its various forms involves the recognition of diagnosable clades. The major question then has to be just exactly what is diagnosable? How different do two populations or lineages have to be to be diagnosably and recognizably distinct? These judgments must surely be subjective, particularly since what is distinct to one taxonomist may well not be to another.

Leaving aside the latter difficulty, it is clear that any of the markers discussed above as useful for delimiting biological species, including molecular and behavioral ones, may also be used to characterize phylogenetic species, though in most groups such characters have tended to be exclusively morphological. My colleagues and I (Claridge et al., 1997a) concluded that the purely practical differences between a phylogenetic concept and a broadly biological one for determining the limits of species taxa were not very great, a view which I still hold. To me, the great disadvantage of the phylogenetic concept is the difficulty in agreeing on what precisely is a diagnosable difference and that therefore such species can only be the subjective judgments of particular systematists. A major advantage of the biological concept is that it does attempt, however difficult that may be, to identify real reproductively isolated and therefore independently evoloving populations, even though isolation may not always be complete. Clearly, the phylogenetic concept can be applied to asexual or parthenogenetic lineages which are effectively agamospecies (Cain, 1954), though the subjective judgment is still central. For the phylogentic species, the problems of differentiating allopatric populations are no different to those involved in differentiating sympatric ones. Thus, diagnosably distinct allopatric populations will be regarded as separate species. The result will almost always be that more allopatric populations will be recognized as distinct species than will be the case with an application of the polytypic biological concept. For example, the well known analysis of the Birds of Paradise (*Aves*, *Paradisaeidae*) by Cracraft (1992), using his phylogenetic

concept, established more than twice as many species (90) than had previous applications of the biological species to the same data set! However, that judgment will always be essentially subjective under both concepts, though various authors, from Mayr (1942) to Sperling (2003), have attempted to provide more objective criteria for assessing the species status of totally allopatric populations. Though there are fundamental differences of philosophy and theory between the biological and phylogenetic concepts, I see little difference generally in practice when applied to species taxa by competent taxonomists.

Despite the apparent advantages of the phyologenetic concept in breadth of application, in my view it has at least one major practical disadvantage compared to biological concepts, and that is the improbability that its application will reveal the existence of complexes of sibling species and thus a full picture of biological diversity. The philosophy of the phylogenetic species gives no incentive or reason to search for further divisions once diagnosably distinct forms have been established. On the other hand, the emphasis of the biological species on reproductive isolation and specific mate recognition means that sibling species will be revealed by its diligent application. Among most groups of living organisms, sibling species are now widely known and are of great biological significance (see contributions in Claridge et al., 1997c).

5 Species Concepts and Speciation

Theories of speciation, the evolutionary diversification of species, have often been closely tied to the development of particular species concepts, so that a brief review is needed here. Most modern authors will agree that in recognizing and describing species, taxonomists are providing a framework for understanding the diversity of living organisms and their evolutionary relationships. However, the philosophical interactions between different species concepts and particular theories of speciation are longstanding and still not fully resolved. It seems obvious that a system for describing observed diversity should be independent of the various possible modes by which that diversity may have evolved (but see Bush, 1994, 1995; Claridge, 1995b).

Probably the most widely accepted mode of animal speciation is that of *geographical* or *allopatric* speciation (Cain, 1954; Mayr, 1942, 1963). The essence of such theories is that an ancestral population is subsequently divided into at least two daughter populations, isolated in space, where they diverge and develop genetic isolation prior to any subsequent meeting and sympatry. The most extreme view of this is that the daughter species must have diverged to the extent that they do not interbreed on meeting: that is, they have developed completely separate specific mate recognition systems, in the terminology of Paterson (1985, 1993), perhaps the strongest current supporter of this view. Contrary to this theory of speciation involving complete allopatry, many authors—most notably, Wallace (1889) and Dobzhansky (1940)—developed theories of the reinforcement of species-isolating mechanisms in sympatry by natural selection, after the partial divergence of incipient allopatric species. This is still a controversial theory, which has been well-reviewed recently (Coyne & Orr, 2004).

Quite distinct from the various theories of allopatric speciation are those of sympatric speciation, where no period of allopatry is necessary for two species to diverge from one previous one, normally by powerful disruptive selection. Though

not strongly supported in the early years of speciation theory, such ideas have always been advanced by some entomologists and others working with large groups of sympatric specialist feeders, including parasites and herbivores (Bush, 1975, 1993, 1994; Walsh, 1864; Wood, 1993). Here, descendant species diverge within the range of the ancestral species and therefore all stages of such divergent populations may be expected to exist in the field together. These ideas have become more and more acceptable to mainstream evolutionary biologists in recent years (Coyne & Orr, 2004), to the extent that even Ernst Mayr, the strongest opponent of such theories since his 1942 book, in his final work accepted that sympatric speciation is probable at least in some parasitic organisms (Mayr, 2004). In fact, there appears to be a developing consensus that there may be a continuum from pure allopatric to pure sympatric speciation, where intense natural selection may outweigh the swamping effects of gene flow by hybridization (e.g., Feder et al., 2005).

Whatever the final consensus on speciation, there surely can be little doubt that the nature of our species concept should not depend on the mode of speciation. Thus, in principle I agree with most cladists at least on the particular point that we should describe the patterns of diversity that we see in nature, so far as possible, independently of the theories concerning the evolution of such patterns (Wheeler & Nixon, 1990). However, I cannot agree with Wheeler and Nixon (1990) that "the responsibility for species concepts lies *solely* with systematists" (p. 79). Aside from the essential arrogance of such a statement, an evolutionary view of species inevitably must involve at least genetics and evolutionary biology, in addition to systematics. If we accept the generality of evolution and species as the results of evolutionary divergence, then it follows that the species concept itself must be an evolutionary one. As noted above, Simpson (1951) first attempted to fuse the then biological species, the agamospecies and the paleospecies, into a unitary all-embracing evolutionary concept. Cain (1954) developed further and clarified these ideas, as later, particularly following the general acceptance of cladistic methodologies, did Wiley and Mayden (Mayden, 1997, 1999; Wiley, 1978; Wiley & Mayden, 2000a). Such theories, like those of de Queiroz (2005), provide a reasonably satisfactory philosophical fusion of the variety of species concepts that account for the diversity of living organisms and their relationships over time, but do not help much in the practical recognition and identification of species taxa.

6 Conclusions

The species problem has always confused two almost completely separate phenomena—species concepts and species taxa—as emphasized frequently over a period of more than 60 years by Ernst Mayr. Species taxa are recognized and described by taxonomists according to their own preferred species concepts. Taxonomists may also be influenced in their choice of concept by the particular groups of organisms on which they work. I follow Mayr (2004) in the view that many species concepts are in reality recipes for recognizing particular species taxa and not themselves significant and distinct concepts. However, the question that is being posed in this volume by Mishler (next paper) concerns the idea that species do not represent any biological reality distinct from that of other levels in the taxonomic hierarchy, such as genera,

families, orders, etc. Clearly from what I have said earlier in this paper, unlike Mishler, I certainly do think that species are of unique and real biological significance. Higher-level categories are to me clearly subjective, depending on the views of any particular taxonomist, despite the added objectivity of cladistic methodologies. I agree with Dobzhansky when, as long ago as 1935 (translated in 1937), he said the species is "that stage of the evolutionary process at which the once actually or potentially interbreeding array of forms becomes segregated in two or more separate arrays which are physiologically incapable of interbreeding" (p. 312). Later, he put this more succinctly in describing a species as "the most inclusive Mendelian population" (Dobzhansky, 1951, p. 263), as did Carson in his widely cited title for a paper, "The Species as a Field for Gene Recombination" (1957). This view is very close to that advocated more recently by Paterson (1985), who regarded species as "that most inclusive population of individual biparental organisms which share a common fertilization system" (p. 25). Clearly, any such evolutionary view of species must recognize that the process of speciation is a continuous one, so that drawing real lines between species as they evolve will be very difficult and intermediate stages must be expected.

The quotation that I have given at the beginning of this paper from a little cited, but very significant, contribution by the virologist van Regenmortel (1997) concerning the Jungfrau and Monch mountains in Switzerland to my mind provides an excellent analogy for understanding the species problem. Here, he introduced the notion of the species as a *polythetic class*, after Beckner (1959), to replace the classical idea of universal classes. Such polythetic classes are "defined by a combination of characters, each of which may occur also outside the given class and may be absent in any member of the class. . . . Contrary to the situation with universal classes, no single property is either necessary or sufficient for membership in a polythetic class" (van Regenmortel, 1997, p. 21). There can be few practicing taxonomists who have not had this type of problem when confronted by large assemblages of apparently related species. A further very relevant and clearly related philosophical concept that van Regenmortel also introduced in the same publication was that of *fuzzy logic* and *fuzzy sets* (after Kosko, 1994, and Zadeh, 1965). This philosophy contrasts strongly with the rigid Aristotelian systems of thinking about classification that Ernst Mayr called typological. Such typological approaches still, often unwittingly, underlie some species thinking. Because intermediates or hybrids between otherwise distinct populations may occur in the field, it does not mean in principle that we cannot and should not recognize distinct species within the continuum of variation. The reality of species as distinct from higher taxonomic categories is not invalidated by such fuzzyness, as, for example, Ereshefsky (1999) has argued. This brings me back full circle to my introductory comments on the all-pervading nature of variation in biological systems and the need for us to recognize it in discussions on species.

Postscript: Counterpoint

Mishler argues from the narrow viewpoint of his own preferred species concept, which is one of several phylogenetic concepts and the most strictly cladistic of them all. He follows his previous definition (Mishler & Theriot, 2000a) where: "A species is

the least inclusive taxon recognized in a formal phylogenetic classification" (p. 46). To me, this is not very helpful and certainly not very precise. Of course, it begs the question of just what is a formal phylogenetic classification; there would certainly be much controversy over that even within the cladistics community. On several occasions, Mishler mentions the biological species as being too simple, and he appears to misunderstand the criterion of interbreeding, which is certainly not just simply the "ability to interbreed." If nothing else, I hope that my discussion above has demonstrated the enormous complexity of the modern biological view of species and particularly the nature of an interbreeding criterion. Contrary to Mishler's comments, the biological view of species is certainly not absolutist or typological: above all, it allows for the natural variation in biological systems, and particularly in populations. Mayr was the first author clearly to differentiate what he called typological concepts in contrast to populational or biological ones. Mishler, uniquely I think, sees a similarity between the biological species and creationism through a common inheritance from classical and Christian thinking, which he believes "is so ingrained in Western thought . . . that most evolutionary biologists and ecologists have serious trouble letting go of it." I object to this view, which clearly implies that all who accept the biological view of species are unable to think critically about these matters! The biological species has been the basis and focus over more than 50 years for most recent discussions on evolution in general, and in particular on speciation—the origin of new species. Ernst Mayr (1942) certainly did more than any other recent author to develop and establish these ideas. Mishler contrasts what he terms the "simplistic Mayrian view" with "Darwin's richer conception" of species. In fact, in *The Origin of Species* Darwin clearly regarded species as more or less arbitrary stages in the process of divergent evolution. I find it difficult to think of this as a richer conception of species than the "Mayrian" view! Of course, few will argue that during the process of speciation and divergence between two populations, the exact point when species status is achieved may be an arbitrary one. In the 1850s, Darwin was so concerned to establish both the fact of evolution and the process of adaptation by natural selection that, unsurprisingly, he did not concern himself with the detailed nature of species and the process of speciation. However, many of his contemporaries, including Wallace and Poulton, certainly predated Mayr with biological species thinking, as discussed above. It is interesting that Darwin himself, in his taxonomic studies, spent much time thinking in detail and worrying about the limits of species in the particular group that he was studying, most notably the barnacles (*Crustacea*, *Cirripedia*). This hardly fits with his view of species as completely arbitrary constructs.

I certainly do agree with Mishler that the particular groups of organisms studied influence greatly the attitudes of different biologists to species concepts. It is obviously a very different problem trying to understand the diversity of, for example, bacteria, on the one hand, and birds, on the other. The nature of reproduction and breeding systems in different groups is clearly critical and may in part account for some of my differences, as an entomologist, with those of Mishler, a bryologist.

After a brief statement of his preferred species concept, much of Mishler's paper here is concerned with what appears to me to be an important discussion, but one peripheral to our present argument, on the nature of ranking in classification and his perception of the advantages of a rank-free classification! Of course, it is true

that particular ranks, such as family, order, etc., in different groups of living organisms are not, and probably cannot be, truly comparable. Mishler suggests that users of classifications assume "that taxa placed at the same rank must be comparable in some way." However, even the most naïve ecologist is unlikely really to expect that, for example, orders of bacteria, flowering plants, and insects are strictly comparable entities! These rankings are surely simply relational within any major group. To me this provides a powerful argument as to just why species are real biological entities and why other higher- or, indeed, lower-ranked groups within a classification are not.

In conclusion, I find it hard to believe that establishing a totally novel rank-free classification and a totally new system of biological nomenclature, the so-called "PhyloCode," can possibly be helpful in the current biodiversity crisis. We need broadly applicable species concepts and the existing Linnean system of nomenclature, certainly for species names. In my view, revolutionary new systems designed to replace the current system of taxonomy and nomenclature can only cause unnecessary diversions from the real and urgent problem of documenting biological diversity.

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CHAPTER
S I X

Species Are Not Uniquely Real Biological Entities

Brent D. Mishler

Are species uniquely real biological entities? This question is one of the most controversial topics today in such areas of biology as ecology, systematics, conservation, population genetics, and evolution. "Species" currently play a central role in both theory and practice in these areas, and have a large place in the public's perception of biological diversity as well. This question can be decomposed into two parts: (1) Are species real, and in what sense? (2) If real, is their reality the same as entities smaller or larger than them—i.e., are they real in a sense that genera or subspecies are not? This paper will briefly review historical and current opinions on these questions, but will primarily advocate one particular position that appears to fit biological reality as now understood: that species properly defined *are* real entities, but not *uniquely* real. The longstanding "species problem" can be solved by realizing that there is no such thing as species after all! The so-called "species problem" is really just a special case of the taxon problem. Once a decision is made about what taxa in general are to represent, then those groups currently known as species are simply the least inclusive taxa of that type. As I favor a phylogenetic basis for taxonomy, I want to look at how to include terminal taxa in the PhyloCode, currently a controversial topic even among PhyloCode supporters. In brief, my argument is: (1) life is organized in a hierarchy of nested monophyletic groups—some of them quite fine-scale, well below the level we currently call species; (2) not all known monophyletic groups need be named, just the ones that are important to process or conservation studies and that have good support; (3) those that are named taxonomically should be given unranked (but hierarchically nested) uninomials; and (4) formal ranks, including species, should be abandoned. I will conclude with a brief discussion of the implication of my position on species for academic studies in ecology and evolution as well as for practical applications in biodiversity inventories and conservation biology.

But be warned, you who thirst for knowledge, be warned about the thicket of opinions and the fight over words.

Hermann Hesse, *Siddhartha*

1 Historical and Current Views of Species

Over the history of science, people have taken a number of different positions on these issues involving the reality of species. The fundamental view throughout the classical period (basically from the ancient Greeks until Darwin) was that species are indeed the basic, real units of life. The basis for their reality was initially conceived of in a typological or idealistic framework; species were viewed as the basic kinds or types of living things. Later, under the influence of Christian theology, the basis for the fundamental reality of species changed somewhat. They were still viewed as basic kinds, but now as specially created "ideas" in the mind of the creator. Taxonomic groups at more inclusive levels were also manifestations of the creator's ideas, but species were the fundamental kinds, the building blocks of life. This idea continues to the present in the attitudes of the majority of the general public in the United States, under the influence of creationism.

The course of science took a somewhat different path than the public view. The Darwinian revolution did not question the reality of species in scientists' thinking (although see below for Darwin's contribution to a shift in thinking about their uniqueness), but changed the perception of their nature greatly. Instead of representing a natural kind defined by certain necessary and sufficient characteristics, species came to be seen as a natural genealogical unit composed of organisms historically related to each other, with a beginning and an end, *not* defined by any characteristics (i.e., "individuals" in the philosophical sense; Ghiselin, 1974; Hull, 1978; Mishler & Brandon, 1987). They were viewed as a cross-section of a lineage (de Quieroz, 1999). In the Modern Synthesis (called such at the time, but looking rather dated these days!), a view solidified of species being the largest group of interbreeding organisms (the gene pool) and as such the most fundamental unit in which evolutionary change takes place (*the biological species concept*; BSC; Mayr, 1942, 1982). Species came to be regarded as a fundamental level in the hierarchy of biological organization (e.g., molecule, cell, tissue, organism, population, species, community, ecosystem).

This view was nearly unanimous until the 1960s, when, under the influence of highly empirical operationalist philosophies of science then in style, and the seemingly "objective" application of computer algorithms to science, an approach emerged called "numerical taxonomy" or "phenetics." In this view, taxa at all levels, including species, were viewed in a nominalistic manner. A species was just a cluster of similar organisms grouped at some arbitrary numerical level of similarity (*the phenetic species concept*; Levin, 1979; Sokal & Crovello, 1970). It was considered to be unnecessary and wrong-headed to require anything about a deeper basis for reality, whether relatedness or interbreeding ability, to describe species. If named species later turned out to be something useful for inferences about evolutionary or ecological processes, then fine, but their recognition as species was best kept separate from process considerations.

One trend apparent in the history of thinking about species has to do with organismal specialty; to a large extent, there has been a sociological difference among communities of systematists studying different kinds of organisms. Zoologists tended to favor the biological species concept (Coyne, Orr, & Futuyma, 1988), while botanists and bacteriologists tended to favor the phenetic species concept (e.g., Levin, 1979; Sokal & Crovello, 1970). There have been some exceptions: for example, Grant

(1981), Rieseberg and Burke (2001), and Stebbins (1950) represent a minority BSC tradition viewpoint among botanists, while Wheeler (1999) represents a minority non-BSC viewpoint among zoologists. This striking distinction is probably mostly due to actual differences in reproductive biology among different branches of the tree of life. Specialists on organisms with either very open mating systems or highly clonal reproduction have always had trouble applying the BSC and have looked for alternatives.

The Hennigian phylogenetics revolution that began in the 1970s altered many aspects of theory and practice in systematics, but did not do much to prune the existing variety of species concepts, and in fact added several more. Hennig himself (1966) held to a version of the biological species concept, while other Hennigians preferred the *evolutionary species concept* (basically an interbreeding group viewed through time as a lineage; Wiley, 1978) or various versions of a *phylogenetic species concept*. The latter are a heterogeneous set of concepts as well: some quite similar to the phenetic species concept (i.e., species viewed as a unique set of character states; Cracraft, 1997; Nixon & Wheeler, 1990; Platnick & Wheeler, 2000; Wheeler & Platnick, 2000a, 2000b), others applying Hennigian concepts of apomorphy and monophyly to the species level (Mishler & Donoghue, 1982; Mishler & Theriot, 2000a, 2000b, 2000c; Rosen, 1978).

In comparing different views of species it is important to distinguish two components of any species concept: *grouping* vs. *ranking* (Horvath, 1997). The grouping component of any species concept indicates the criteria for group inclusion, whether ability to interbreed, phenetic similarity, or sharing of apomorphies indicating monophyly. The ranking component of any species concept indicates the criteria for deciding whether a group counts as a species rather than a taxon at some other rank. Both components are necessary because all concepts define groups within groups, and the level of group corresponding to species needs to be specified. Some of the controversy over species concepts has been because people are not clear about this distinction.

The phylogenetic species concept in the sense of my work with Brandon and Theriot (Mishler & Brandon, 1987; Mishler & Theriot, 2000a, 2000b, 2000c) is clear about this distinction, and basically treats species as just another taxon (see also Nelson, 1989), taking the perspective that if we are going to be phylogenetic about taxa in general, we need to be phylogenetic about species. Theriot and I (Mishler & Theriot, 2000a) defined species as follows: "A species is the least inclusive taxon recognized in a formal phylogenetic classification" (p. 46). As with all hierarchical levels of taxa in such a classification, organisms are *grouped* into species because of evidence of monophyly. Taxa are *ranked* as species rather than at some higher level because they are the smallest monophyletic groups deemed worthy of formal recognition, due to the amount of support for their monophyly and/or their importance in biological processes operating on the lineage in question. One obvious question follows from the definition given above: doesn't the ranking decision sound arbitrary? The short answer is: Yes! If not completely arbitrary, the decision does depend on local context—ranking criteria are pluralistic rather than universal (Mishler & Donoghue, 1982).

The ranking decision in the phylogenetic species concepts discussed above is present because of the way the current codes of nomenclature are written. Monophyletic taxa not only have to be discovered and diagnosed, they must be given a specific rank, including species. But this doesn't have to be so. We can remove this arbitrary aspect of taxonomy; the best approach is arguably not to designate any ranks at all.

I now advocate an extension of the recent calls for rank-free phylogenetic taxonomy to the species level (e.g., Mishler, 1999; Pleijel, 1999), and will develop this position in the following sections.

2 Return to a Darwinian View of Species

Let's consider the two-part question introduced above: (1) *Are species real?* (2) *Are species uniquely real?* All working biologists today think that the answer to the first question is yes: species are real entities in some sense (although the grouping criterion considered to be the basis for their reality varies as described above). The current debate concentrates on the second question: whether or not species are a special level either in biological organization or in the taxonomic hierarchy. In other words, is there a unique ranking criterion for species? The two possible answers to this question can be contrasted as the *Darwinian view* vs. the *Mayrian view*.

One of Darwin's important novel contributions to biology was the explicit recognition that the species level is an arbitrary point in the divergence of two lineages. The *Origin* (Darwin, 1859) is full of passages indicating Darwin's view that the species rank is arbitrary, even though the lineages are quite real. His view was that divergence between two lineages happens, and at some point it is convenient to call the two lineages species according to the judgment of a competent taxonomist, but nothing particularly special or universal occurs at that point.

The Modern Synthesis, in its bringing together of population genetics and taxonomy, emphasized a different point of view on species than Darwin. Species were now viewed as an important and distinct level of biological organization (like "molecule" or "cell"), the largest group within which evolution occurs, held together by sharing a gene pool. Ernst Mayr is particularly responsible for pushing this viewpoint (Mayr, 1982). Following Mayr, many today (scientists and public alike, in a strange convergence between evolutionary biologists and creationists) see species in this special light. Note that I am not calling Mayr or any evolutionary biologist a creationist. I am only pointing out an interesting parallel to their position in this one particular area. I don't think the parallel is an accident, however. I think that the idea of distinct, basic, natural units (i.e., species as the building blocks of biodiversity) is so ingrained in Western thought (coming from before the Christian era so not due to creationists directly) that most evolutionary biologists and ecologists have serious trouble letting go of it. Darwin was a really original and courageous thinker whom many biologists even today have trouble emulating.

There is abundant empirical evidence presented since Darwin's time that shows he had the right view and that the actual "species situation" is much more complex than modeled by the Modern Synthesis adherents (Mishler & Theriot, 2000a, 2000b, 2000c). Gene pools (potential horizontal transfer of genes at some level of probability) usually occur at many nested levels within one lineage, and the most inclusive level is often higher than anyone would want to call species (e.g., corresponding to the current generic and even familiar level in flowering plants). On the other hand, sometimes gene pools don't exist at all in a lineage, in the case of asexual organisms. Alan Templeton (1989) succinctly summarized this spectrum of problems with the Mayrian BSC as ranging from "too much sex" to "too little sex."

It would be conceptually cleaner if Mayr was right that there is a particular, unique level, comparable across the tree of life, at which "species-ness" arises as two lineages diverge. However, empirical reality intrudes on this tidy BSC concept; we need a more flexible concept since such a clean species break rarely if ever appears to be the case. My own view is that Darwin's richer conception is better, and that the supposed advances of the Modern Synthesis were actually retrograde, at least as far as species concepts are concerned. To make progress in this area we need to reject the simplistic Mayrian view and emulate Darwin's view.

My own answer on the twin questions italicized at the beginning of this section is this: entities that are currently called species are indeed real, if grouped correctly as monophyletic groups, but they are not uniquely real, i.e., they are only real in the sense that other levels of monophyletic groups are—there is no special ranking criterion for species. The processes causing divergence of lineages, and keeping them separate afterwards, are many. We must develop a richer view of the tree of life and how best to understand and classify it. Such a view must consider the many nested levels of divergence and reticulation in the tree of life, not just the one we arbitrarily happen to call species.

To develop this view, we need to look closely at several related concepts. One is the nature of *monophyly*. There have been two basically different ways of defining monophyly within the Hennigian tradition of phylogenetic systematics: one is synchronic (i.e., "all and only descendants of a common ancestor"); another is diachronic (i.e., "an ancestor and all of its descendants"). I have argued elsewhere (Mishler, 1999) that the former view (Hennig's own view) is better, because it avoids the time paradoxes inherent in placing the ancestor in a group with its descendants. Just like a zygote is not one of the cells of an adult organism (instead it is *all* the organism at its beginning), the ancestor is not a member of a synchronic monophyletic group when looked at later—it was the whole monophyletic group back in its day.

A further consideration is that the word "species" appears in many definitions of monophyly (including Hennig's). This obviously matters if we are discussing the application of monophyly to the species level, because of circularity concerns. We need a definition that is both synchronic and neutral about taxonomic ranks, like this: a monophyletic group is all and only descendants of a common ancestor, where "ancestor" is interpreted broadly to mean an individual in the philosophical sense of Ghiselin (1974) and Hull (1978), e.g., an organism, or breeding groups of various sizes.

Another distinction that is needed is between *clades* and *lineages*. While sometimes treated loosely as synonyms, they are not exactly the same thing—some refinement of terminology is needed. Figure 6.1 shows the difference. A "clade" is a synchronic entity, a monophyletic group as discussed above (a group composed of all descendants of a common ancestor). A "lineage," by contrast, is a diachronic concept, a series of ancestors and descendants (replicators in the abstract sense of Hull, 1978) through time. They are related terms, of course: a clade could best be viewed as an instantaneous snapshot of a lineage.

This distinction helps us clarify some of the murky debates over phylogenetic species concepts. Some phylogeneticists have focused their species concepts on clades (e.g., Baum & Shaw, 1995; Mishler & Brandon, 1987; Mishler & Theriot, 2000a, 2000b, 2000c), and some on lineages (e.g., de Quieroz, 1999; Wiley, 1978), but it is important to note that both clades and lineages form hierarchies. Clades are obviously nested inside

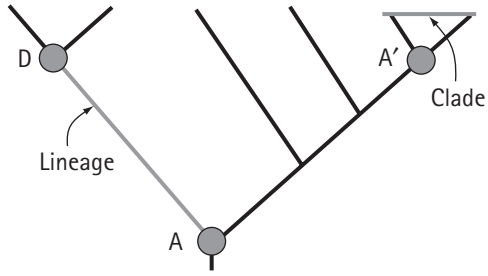

Figure 6.1: The distinction between clades and lineages. A clade is a synchronic, monophyletic set of lineage-representatives, where monophyly is defined synchronically as "all and only descendants of a common ancestor" (represented by A' in this case). A lineage is a diachronic ancestor–descendant connection (between A and D in this case): "species" in the de Queiroz sense.

of other clades, but less widely understood is that the same is true of lineages. A smaller-scale lineage (say of cells) is nested inside of larger-scale lineages (such as organisms or larger groupings acting as individuals in a philosophical sense). There is no privileged level that can be recognized in either of these nested hierarchies; there is no unique species rank in either clades or lineages.

I prefer applying our formal classification system to name clades (i.e., monophyletic groups) for the following reasons: (1) clades are more nicely nested hierarchically than lineages; (2) we have a well-thought-out code of nomenclature available for naming clades (the PhyloCode); and (3) there are many more kinds of lineages, biologically speaking, due to the multiple kinds of replication which can occur in nature. Some recent workers have thought about providing a code to name lineages, i.e., a "Species Code" (see discussion in the PhyloCode preface at: http://www.ohiou.edu/PhyloCode/preface.html), perhaps to complement the PhyloCode (which is based on clades), but this will prove to be very challenging.

The generalized view presented above, abandoning species in favor of describing clades at several nested levels, has many advantages in theory. Yet it requires considerable further explication before being applied in practice, since so many other areas of biology are accustomed to using species as a unit. I will go through several of these areas below, beginning with systematics, where it all starts.

3 Practical Implications

3.1 Truly rank-free classification, all the way down

As covered in many previous papers (summarized by Mishler, 2009), it has become clear that the ranks in the Linnaean system are problematic for classification, both theoretically and practically. Let me just summarize these general arguments briefly here. Rapid advances in phylogenetic research have made it obvious that there are not nearly enough ranks to suffice in classifying the tree of life, with its thousands of nested levels of clades. The need to maintain the hierarchy of the ranks leads to instability, with names being changed without good reason, as, for example, when

one currently recognized genus is found to be nested inside another (a common occurrence). Ranked classifications can lead to bad science in such fields as ecology or macroevolution, if a user of a classification naïvely (but understandably) assumes that taxa placed at the same rank must be comparable in some way.

The current codes of nomenclature can be tweaked to name monophyletic groups, but they are far from ideal for that purpose. The current codes are used to name *all* kind of groups; thus a user has no way of easily knowing if a given taxon is thought to be monophyletic. Only a search into the literature can uncover the basis for a particular taxon name under the current codes, while under the PhyloCode one knows that the author of the name hypothesized it to be a monophyletic group. A name that can be used to convey anything really conveys nothing.

It has become clear that the current codes don't lend themselves well to naming monophyletic groups unequivocally, primarily because there is only one type specimen. It is possible to patch the current codes of nomenclature to name phylogenetic taxa, as suggested by (Barkley et al., 2004). But, for many reasons it would be better to develop a new code of nomenclature specifically designed for phylogenetics. It really is time to bite the bullet and complete a synthesis between the Darwinian revolution and the Hennigian revolution (de Queiroz, 1988). Ranked classifications are a hold-over from the pre-Darwinian creationist mindset (Ereshefsky, 2002). They are not just a quaint anachronism; they are resulting in miscommunication at many levels. Completely rank-free phylogenetic classifications are far better for teaching, research, communicating with other scientists, and interfacing with the larger society.

What about the fundamental taxonomic level, species? Most published discussions about rank-free taxonomy are based on considerations of higher taxa alone, yet all the criticisms of taxonomic ranks summarized above can be extended to species—it is clear that all the arguments about the inadequacy of the current codes for naming phylogenetic taxa apply to the species level also.

The developing PhyloCode may be accessed online (http://www.ohiou.edu/PhyloCode/). This code maintains many of the features of current rank-based codes, but removes all ranks from clade names, and also uses multiple types (called "specifiers") to precisely fix the name of a clade. Important to this discussion, the current draft of the PhyloCode unfortunately does not deal well with providing names for what have been called species. Many uncomfortable special conventions are currently suggested for dealing with this particular rank. Thus even the community of supporters of the PhyloCode is conflicted about what to do about species! More work is needed to make the PhyloCode work seamlessly at all taxonomic levels.

How could rank-free classification be applied to terminal taxa? Exactly as at other levels: names of all clades (including the terminal level) should be hierarchically nested uninomials regarded as proper names (current usage should be followed as much as possible to retain links to the literature and collections). As at all taxonomic levels, we could use either node-based or stem-based names with multiple internal specifiers (I personally think the use of apomorphy-based names is incoherent at any level, but that is another argument!). Specifiers should be actual specimens (this should be true at all levels).

In my opinion, species names should be converted from the current epithets (despite the current prohibition of this is the PhyloCode draft; see example of this in Fisher, 2006). The overriding principle is to achieve maximum continuity with previous

literature for the sake of preserving connections to databases, literature, museum specimens, etc. There are two additional important principles, in my opinion: the naming system should be consistent for clades at all levels, and the PhyloCode should be distinct from the existing codes in terms of rules. In this approach, then, each clade named under the PhyloCode, including the terminal-most clade, has a uninomial given name, but also has associated with it a set of more and more inclusive "family names" (its clade address). In a database at least, all the higher clades to which a taxon belongs would be regarded as part of its complete name; this would help computers (and users) keep track of information in the database. Homonyms, which would result when converting species epithets to uninomials, can thus be told apart by higher-level clade names if their context is unclear, just as a teacher uses last names to distinguish among several children in class having the same first name.

3.2 Phylogenetic monography

How can monographs be done under this view of species? In a rank-free framework, they can be done as well as or better under the current codes, as exemplified through the pathbreaking approach by Fisher (2006). Her approach was as follows: (1) use earlier taxonomies as a criterion for stratified-random selection of specimens to study (Hennig's semaphoronts); (2) after that, ignore taxonomic designation during character analysis and character scoring; (3) once operational taxonomic units are established (based on scored characters), conduct phylogenetic analysis; (4) use the resulting phylogenetic tree to inform taxonomic decisions, including naming of terminal clades consistent with the PhyloCode's treatment of more inclusive monophyletic groups. Specifiers used are specimens on deposit in an herbarium or museum, and the formal specifiers as well as other specimens studied are cited much as in traditional monographs.

3.3 DNA barcoding

This discussion touches upon the current debates over DNA barcoding, another recently proposed system for characterizing species, which uses a short stretch of DNA sequence from a standard gene. Similarity above a certain percentage, say 2%, equals species status. This approach has gained popular appeal, but suffers from obvious philosophical problems. Contrary to their posturing as cutting-edge, DNA barcoders are actually returning to an ancient, typological, single-character approach, and are maintaining a pre-Darwinian view of species. There are two aspects to DNA barcoding, one good (but not new), the other new (but not good): DNA-based identification (i.e., using sequence data from a standard gene) and DNA taxonomy (i.e., using sequence data from a short stretch of a standard gene to recognize and name taxa). All critics (including me) are strongly in favor of the good idea of using DNA for identification of already well-characterized taxa, but that is old hat—the important use of DNA for identification goes back to the beginning of molecular systematics. The DNA barcoders can't take any credit for that—the most that they can claim is that they will scale-up, standardize, and database. But, there is really no need to set up a new bureaucracy or new databases (wasting the money of naïve funding agencies, who could be directing their attention toward real phylogenetic

systematics)—current efforts elsewhere (such as GenBank) are more than sufficient. The new idea that DNA barcoding can replace normal taxonomy for naming new species and studying their relationships is not only bad philosophically, it is destructive in a practical sense. We should use all available resources to build real capacity to do systematics right (Will, Mishler, & Wheeler, 2005).

3.4 Implications for ecology, population genetics, and evolution

The species level is highly embedded in current ecological theory and practice. It is widely accepted that within- and between-species interactions are different in kind. Niche theory is usually conjoined with a view that the species level provides a fundamental break. Gause's (1934) theory of competitive exclusion talks about the ability of species needing to differentiate in order to live in the same environment. The species level is likewise highly embedded in studies of population genetics. The species is thought to be the largest unit in which gene flow is possible, thus the largest group that can actually evolve as a unit.

It is beyond the scope of this paper to elaborate on the ways to modify ecological theory to fit with a rank-free view of phylogenetic diversity (i.e., no species or other ranks). It needs to be done, however—based on the arguments presented above it is clear that the world is more complex than the current BSC allows for. If the systematic community moves to a rank-free view of biodiversity, then basic ecological and evolutionary research must be modified to account for this view. Fortunately, phylogenetic comparative methods are under active development in many areas (beginning with seminal studies such as Burt, 1989; Cheverud & Dow, 1985; Felsenstein, 1985; Harvey & Pagel, 1991; Huey & Bennett, 1987; and Martins, 1996). Studies can go forward on niche differentiation, competition, coexistence, species-area curves, community assembly, gene flow, macroevolutionary diversification, etc., but in a more rational manner taking into account nested hierarchical levels in these phenomena, without using ranks.

3.5 Implications for conservation biology

As argued in detail above, biodiversity isn't species—biodiversity is the whole tree of life, not just the arbitrary place at which species are named. There are clades smaller and larger than the traditional species level. Species are not comparable between lineages in any manner, just an arbitrary cut-off somewhere along a branch in the tree of life. Thus only someone sharing the BSC view that species are fundamental (a view interestingly shared by creationists, as discussed above) should think that species are the basic units of biodiversity, or that a list of currently named species in some way provides an inventory of biodiversity. Biodiversity is a much richer tapestry of lineages and clades.

So how can we inventory biodiversity without species? Since counting species or measuring their ranges and abundances is a poor measure of biodiversity, what should be done? New quantitative measures for phylogenetic biodiversity need to be applied which take into account the number of branch points (and possibly branch lengths) that separate two lineages. Phylogenetic measures of biodiversity have been developed

that could be used as a basis for rank-free measures of biodiversity (Faith, 1992a, 1992b; Mishler, 1995; Vane-Wright, Humphries, & Williams, 1991). There are two possible approaches: counting of number of nodes separating two terminal clades, or summing the branch lengths separating two or more terminal clades. Advantages and disadvantages of each exist, and more work needs to be done, but the direction to move is clear.

What does "rarity" mean without ranks? This relatively new phylogenetic worldview can clarify greatly this term (Mishler, 2004). Rarity fundamentally means having few living close relatives, and these days "few" and "close" can be defined quantitatively on cladograms. Conservation priorities can actually be better guided by phylogenies rather than by taxonomy *per se*. Phylogenies provide a richer view of our knowledge of nested clades, and are directly associated with the evidence used to build them. Just like in the more theoretical areas discussed above, the most practical application of systematics in the modern world, conservation, needs to drop its reliance on species.

Postscript: Counterpoint

I agree with the quote at the beginning of Dr. Claridge's paper (about mountains in Switzerland) and with his statement that "species taxa represent attempts to recognize real biological entities." I believe mountains and taxa are real; as I explained in detail above, the issue for me is not whether taxa are real (they are, if monophyletic), but whether entities given the rank of species are real in a unique and special way that entities larger and smaller than them are not. Claridge and I agree that the entities we call species are real biological units. Our main difference is in what that reality is due to: for me it is monophyly, for Claridge it is sharing reproductive bonds. In either case, my point is that there are such real entities deeply nested inside each other, with no one level fundamental or unique. Species are real, but not in a unique and special way.

Claridge understates the fundamental differences between interbreeding groups and monophyletic groups; they are not the same thing theoretically or practically. In fact they are diametrically opposed, by definition. As was first pointed out by Rosen (1978), the ability to interbreed is certainly a plesiomorphy and thus not a good guide to monophyly. It is the derived *inability* to interbreed, say the origin of a new mate recognition system, that can be an apomorphic feature useful to diagnose a monophyletic group. Any empirical test for reproductive compatibility is certain to be measuring plesiomorphic similarity. The BSC is (and should be) anathema to a cladist, which makes it puzzling how someone could be a solid cladist at all levels but species.

I agree with Claridge that breeding relationships are very complex and diverse—but would point out that this observation actually strengthens my point. There are smaller inbreeding groups (sometimes actual, sometimes potential) nested inside of larger interbreeding groups: local populations, clusters of populations, geographic regions, even up to the intergeneric level in flowering plants like orchids. The potential to successfully interbreed gradually trails off as one looks at more and more distantly related populations (as Darwin pointed out). Claridge acknowledges this when he says that "the process of speciation is a continuous one, so that drawing real lines between

species as they evolve will be very difficult and intermediate stages must be expected," but then he contradicts himself in the same paragraph by saying: "species are of unique and real biological significance." In most organisms there is no magical level at which the probability of successfully interbreeding suddenly drops from near 100% to near 0%. Thus even under the biological species concept there is no unique and special level. Again, keep in mind the important distinction between grouping and ranking: breeding *groups* are very real—no one is denying that—it is the *ranking* decision about which level among many levels of nested breeding groups is to be called species that is arbitrary. Darwin was very aware of this distinction; we should still take his views seriously.

Evolutionary biology will be richer and much more accurate in its models of the world if this Darwinian hierarchical perspective is accepted. Evolutionary and ecological processes are occurring at many nested levels. "Speciation" is a major field of study, with many books and papers to its credit, which my point of view would seem to denigrate. But while I do think that "speciation" is an oversimplified concept, like the biological species concept on which it is based, I believe that there are important processes being studied by these researchers. I call it "diversification," the splitting of lineages influenced by a variety of interesting processes (ecological, reproductive, genetic, developmental, etc.). The important distinction I make is that diversification happens at many nested levels, not a single magical one, and full accounting of these is needed for a complete understanding of evolution. Focusing at the level of the entities taxonomists happen to call species in a particular case, as in standard studies of "speciation," is a one-dimensional look at a multidimensional, hierarchically nested process.

We can do better with a completely rank-free view of taxonomy. Claridge thinks that my discussion of rank-free classification is peripheral to our argument over species, but if course it is central to my position. The arguments against comparability of entities at a particular rank apply to "species" as much as "families" or "orders." Evolutionary processes are not just operating to produce what we happen to call species; they operate at many nested levels in producing the tree of life, "which fills with its dead and broken branches the crust of the earth, and covers the surface with its ever branching and beautiful ramifications" (Darwin, 1859, pp. 170–171).

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PART IV
DOES SELECTION OPERATE PRIMARILY ON GENES?

Introduction

Consider the worker honey bee, which will sacrifice itself to protect the hive when it stings an intruder. A case like this is interesting from the standpoint of natural selection, since one would think that an organism's traits are naturally selected to benefit the organism in some environment—in which case, a stinger on a bee should not have evolved. However, since stinging some predator or intruder assists in saving the entire hive (and its own species), then we can see how the worker bee has made it through the evolutionary sieve. The same goes for many other species, especially those group or cooperative species that form tight, interconnected networks. The worker bee causes us to wonder which biological entity *exactly* is being selected in natural selection: is it the gene, individual, population, or species? Also, which of these entities might be most influenced by natural selection? These questions comprise the so-called *unit of selection debate* in philosophy of biology. After all, in the following paragraph of Chapter 4 of the *Origin*, Darwin (1859/1999) seems to indicate that natural selection works on individuals, populations, *and* species:

> Natural selection will modify the structure of the young in relation to the parent, and of the parent in relation to the young. In social animals it will adapt the structure of each individual for the benefit of the community; if each in consequence profits by the selected change. What natural selection cannot do, is to modify the structure of one species, without giving it any advantage, for the good of another species. (pp. 73–74)

In his book titled *Adaptation and Natural Selection* (1966), George C. Williams suggested that the gene is the principal unit of selection, and Richard Dawkins (1976, 1978, 1982) added further arguments and evidence for what has become known as the *gene's-eye view*. According to the gene's-eye view, the gene is a replicator (and interactor, see Hull, 1980) that passes on its basic pattern and structure in replication, and is housed in organisms conceived of as vehicles for replication (and interaction). This seems sensible enough for anyone who has had a modicum of biology where heredity is studied. However, here is where the controversy comes in: although it may seem that natural selection works at the vehicular level, the *real* selective forces are taking place at this genetic, replicator level.

This has caused a continual flurry of responses over the years. For example, in a paper titled "Artifact, Cause, and Genic Selection" (1982), Elliott Sober and Richard Lewontin offer a critique of the gene's-eye view, arguing that the "general perspective of genic selectionism is mistaken" (p. 157). In their paper titled "The Return of the Gene" (1988), Kim Sterelny and Philip Kitcher respond to Sober and Lewontin and, through numerous clarifications, arguments, and evidence of their own, offer a more balanced perspective that includes the "return" of the gene's-eye view. In his more recent paper titled "The Levels of Selection Debate: Philosophical Issues" (2006), Samir Okasha provides us with the present status of the debate. The units of selection debate is intriguing for biologists and philosophers of biology alike, as Okasha mentions, because it is a "mix of empirical, conceptual and methodological questions which makes it ideally suited to, and much in need of, philosophical scrutiny" (p. 1).

In the first paper in this part, Carmen Sapienza argues that natural selection acts on individual genes. In his words, for example, "the colonization of hospitals by microorganisms that are resistant to various antibiotics is a clear case where organisms that differ only by the acquisition of a single gene come to dominate an environment in which both might persist for many generations in the absence of the selective agent." As another example of natural selection working directly on genes, Sapienza speaks about genetic variability in the structure of centromeres involved in meiotic drive, which has to do with the non-random segregation of chromosomes in meiosis. The upshot of his paper is that "the bulk of the variance" in phenotypes "is attributable to variation in a small number of genes."

In the second paper, Richard Burian cautions people who take the gene's-eye view to be careful not to draw conclusions too hastily. Using a few examples—including the case of sickle cell anemia (originally put forward by Sober and Lewontin, 1982)—Burian shows that there are probably many cases where we cannot draw the conclusion that selection is operating on a particular gene (or allele) because the phenotype under selection is elicited only in combination with another gene (or allele). In fact, standard accounts of phenotypes nowadays are intimately tied to genotypic factors that make the distinguishing between the two—phenotype and genotype—very difficult to accomplish (see the papers in Pigliucci & Preston, 2004). Burian also uses his examples to show that, over time, selection probably acts on populations to increase the frequency of causally relevant genetic complexes in the population, rather than on the genes themselves. Burian's final point in his paper is telling about the debate, which we leave with the reader to ponder:

Resolution of the debate about whether selection acts primarily on genes will remain unresolved unless it integrates considerations of life histories, diet, hormonal mechanisms, neurological mechanisms, and genetic and epigenetic mechanisms. But the very fact that all these considerations are relevant suggests that selection acts in multifarious ways. It also suggests that although selection "acts" on genes, it does not act primarily on genes.

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CHAPTER SEVEN

Selection Does Operate Primarily on Genes

In Defense of the Gene as the Unit of Selection

Carmen Sapienza

Natural selection is an important force that shapes the evolution of all living things by determining which individuals contribute the most descendants to future generations. The biological unit upon which selection acts has been the subject of serious debate, with reasonable arguments made on behalf of populations, individuals, individual phenotypic characters, and, finally, individual genes themselves. In this paper, I argue that the usual unit of selection is the gene. There are powerful logical arguments in favor of this conclusion, as well as many real-world examples. I also explore the possibility that epigenetic differences between individuals may be heritable between generations. Although few such examples exist, epigenetic differences provide an exciting source of potentially heritable variation that may allow rapid evolutionary change to occur, perhaps in response to environmental influences.

1 Introduction

Natural selection may be defined as a mechanism that distinguishes differences between biological entities and results in a net reproductive advantage for one of them. I will assume that natural selection is a significant force in evolution and will not debate whether it is the only force shaping evolutionary change. I will defend the idea that the "gene" is the usual and most important level at which natural selection distinguishes differences between biological entities. The notion that the gene is the unit of selection assumes, further, that differences between genes underlie almost all forms of heritable variation. In this paper, I have attempted to argue from first principles, accompanied by a few real-world examples that I believe make the case that natural selection has shaped important complex traits and that these traits are controlled by one or a few genes. This form of argument has been aided considerably by Professor

Burian's thorough and thoughtful discussion of the history of this debate in the companion paper. I refer the reader to Professor Burian's paper and wish the reader to know that I am in substantive agreement with much of his discussion.

2 Natural Selection Operates *within* Genomes without Regard for Phenotypic Effect

I suspect that the reason I was invited to defend the idea that natural selection operates primarily on genes is because W. Ford Doolittle and I co-authored a highly controversial article in 1980, entitled "Selfish Genes, the Phenotype Paradigm and Genome Evolution" (Sapienza & Doolittle, 1980). In that piece—and a companion article written by Francis Crick and Leslie Orgel (1980)—we extended Richard Dawkins' (1976) selfish gene argument to the level of genome structure. We argued (correctly, I believe, to this day) that much of present-day genome structure is the result of natural selection operating directly on DNA sequences for the capacity to make more than one copy of themselves prior to cell division/meiosis. In other words, many elements in the genome are present simply because they have the capability of making copies of themselves and spreading these copies around the genome. In conjunction with sexual reproduction, such behavior becomes the equivalent of "meiotic drive" and the new copies of the elements will spread throughout the genome, much like an intra-genomic parasite. In fact, members of a small number of families of these elements make up more than a third of the human genome and some individual families have more than a million members. Such sequences are sometimes erroneously referred to as *junk DNA*, inferring that the sequences have no function (as far as individual phenotype is concerned). A less anthropomorphic explanation is that their function is to make more copies of themselves (much like the function of a virus is to infect cells and make more viruses). In rare cases, insertion of an element into or near a gene may have an effect on the phenotype of the individual (and so be subject to natural selection operating on organismal phenotype); however, it is impossible to imagine that such has been the case for all one million accumulated members of the human Alu family (Batzer & Deininger, 2002), for example, insofar as most of them are not present within or adjacent to genes (Ensembl, 2009). I have always found this logical argument compelling, in the extreme, and have concluded that the vast majority of these "transposable elements" have survived and increased in number within genomes largely in the absence of supervision by any selective force operating on organismal phenotype.

In the remainder of this paper, I will argue that there is at least one additional way that natural selection can, and does, operate directly on genes—via non-random segregation of chromosomes—and that, even in those cases where natural selection appears to operate on some complex phenotypic difference between individuals, the difference is most likely traceable to genetic differences at one or a few loci.

3 Selective Forces, Heritable Variation, and the Definition of Function

During my graduate student days, I was fortunate to attend a small meeting on genome evolution at which the late John Maynard Smith was a featured speaker. Professor

Maynard Smith was a wonderfully eloquent communicator who had the ability to reduce complex problems to manageable components. His definition of the term *function* has served as a guiding principle in my attempts to explain biological variation throughout my career. He noted that when an evolutionary biologist made the statement "the function of the heart is to pump blood," what he/she actually meant was *not* simply that the heart *did* pump blood but that those individuals whose hearts were superior in pumping blood survived and left more descendants than those individuals whose hearts were inferior in this function (for more on this, see the papers by Perlman and Cummins & Roth in this volume).

As was the case with many of Maynard Smith's simple examples, a complex web of cause and effect was concealed just below the surface. There is the assumption that hearts of different blood-pumping abilities are carried by different individuals in the population and, further, that the different blood-pumping abilities of the different hearts is heritable, so that individuals with superior hearts are more likely to have offspring with similarly superior hearts than are individuals who have hearts of inferior blood-pumping ability. Layered on top of these caveats is the question of how, exactly, individuals with superior hearts come to leave more offspring than individuals with inferior hearts. Is it because they can run faster or for longer distances, thus escaping predators? Or is it because they are less likely to die as a result of myocardial infarction and so have a longer reproductive lifespan? There are many possibilities, but the gist of determining the "function" of biological structures or processes is the formal identification of selective forces and the determination of how each force distinguishes between variants.

So, in Maynard Smith's example, we would be left with the question of what the selective force "sees" and whether what is seen is attributable to one gene, a few genes, or some higher collective property of the organism. Of course, it is true that a complex organ, like a heart, is not seen by selection on its own but in the context of the creature bearing that heart. Having a heart attack while running the Boston Marathon cannot be ascribed to variation in a single gene—or can it?

My argument is that the things about heart performance that are likely to matter most—for example, systolic and diastolic blood pressure, likelihood of myocardial infarction, and serum cholesterol levels—all show very high heritability (Jorde, Carey, Bamshad, & White, 2000). In other words, the variation that is seen in blood pressure between different individuals in the population can be explained, in large part, by differences in genotype. The number of genetic differences required to explain these phenotypic differences is not known precisely. However, millions of additional years in patient lifespan (not to mention, billions of dollars in drug company profit) have been realized as a result of treatment of two of the most common and dangerous cardiovascular phenotypes: hypertension and high serum cholesterol. Both of these conditions can be alleviated by drugs (ACE inhibitors and statins) that target the products of single genes. Insofar as variability in cardiovascular phenotype is a product of heritable variation (even those of us who are loath to be gym rats must admit to some environmental effects), it seems probable that the phenotypes under selection are controlled by small numbers of genes.

4 Natural Selection Can, and Does, Act on the Products of Individual Genes

Given the complexity of living organisms and the likelihood that many phenotypic characters are the result of the action of multiple genes, it is worth entertaining the question of whether there are examples of natural selection acting at the level of individual genes. Simple, real-world examples from bacterial genetics come to mind: for example, the colonization of hospitals by micro-organisms that are resistant to various antibiotics is a clear case where organisms that differ only by the acquisition of a single gene come to dominate an environment in which both might persist for many generations in the absence of the selective agent. Because the enzymes that break down penicillin, for example, tend to be shuttled from bacterium to bacterium on easily mobilized genetic elements—small pieces of DNA, called *plasmids* and *transposable elements*, that contain one or a few genes—the accumulation of antibiotic-resistant micro-organisms in hospitals has occurred with breathtaking speed.

In such cases, the selective force (the presence of antibiotics) has distinguished between, and among, genetic variants by killing those that do not produce the product of the gene encoding antibiotic resistance. In fact, in this instance what is being selected is not the gene, *per se*, but the presence of the product of that gene. While there is only minimal argument that without the gene, there would be no gene product, the reciprocal statement is not true: there are many examples in which a particular gene is present but the gene product is not. The most obvious examples are carried around with each of us while we go about our daily business. Even though each of our 10^{14} cells contains all ~25,000 of our genes, every cell is not producing all of the gene products of all of our genes. The processes of development and differentiation lead to these characteristic epigenetic differences between cells that are not based on genotypic differences (with a few notable exceptions). Instead, the DNA of liver cells is packaged differently within the cell nucleus from the DNA of brain cells, and the selection of genes that is accessible to the RNA transcription machinery is different in each type of cell. Given that it is possible for genes to be present in cells (including sperm cells and egg cells) without expressing a gene product, it is worth asking whether it is possible for natural selection to operate on genes themselves directly.

5 Natural Selection Can Act Directly on Genes Themselves

Perhaps the most convincing demonstration that natural selection may operate at the level of individual genes comes from examples of *true meiotic drive*, i.e., non-random segregation of a chromosome at meiosis. The process by which an egg is formed in females of most species is an asymmetric meiosis. Instead of producing four gametes that are equal in size and genetic content, females produce eggs by producing one large gamete (the egg) and two smaller meiotic products (the first and second polar bodies). The genes contained in the egg have the possibility of contributing to the next generation, while the genes in the polar bodies do not.

My colleagues and I (Wu et al., 2005), and others (Agulnik, Agulnik, & Ruvinsky, 1990), have found that it is possible for individual chromosomal variants to influence the probability that they are segregated to the egg half of a meiotic division directly—thus enhancing their chances of survival for another generation—rather than the polar body half, where they have no chance of being represented in the next generation. In most of these instances, the chromosomal structure upon which natural selection is operating is the *centromere*, i.e., the complex of DNA sequences and proteins responsible for attaching the chromosome to the meiotic spindle (the cellular structure responsible for the actual partitioning of chromosomes between daughter cells) so that the spindle microtubules can move the chromosome to one pole or the other.

When chromosomes are observed under the microscope, they appear as strands of nearly uniform thickness, except for a single constriction called the *centromere*. The centromere is the structure responsible for the physical movement of the chromosomes between daughter cells at cell division. The centromere contains proteins to which microtubules (cellular "motors" responsible for the movement of many cellular components and proteins) attach and which enable the chromosomes to be moved away from the plane of cell division, thus ensuring the orderly and equal segregation of the chromosomes to each daughter cell.

Evidently, all chromosomes have centromeres, but not all centromeres are equal in their ability to attach a chromosome to the egg side of the spindle (Pardo-Manuel de Villena & Sapienza, 2001a). Interestingly, such *meiotic drive* occurs even when the outcome is disadvantageous to the organism. In the case of the *Ovum mutant* allele carried by the DDK inbred mouse strain, for example, inclusion of the DDK allele in the egg risks embryonic death if the egg is fertilized by sperm from some other inbred strain males; yet the DDK allele is included in the egg more than 60% of the time in females who are heterozygous for a DDK allele and a "wild-type" allele (Pardo-Manuel de Villena, de La Casa-Esperon, Briscoe, & Sapienza, 2000; Wu et al., 2005). Even in the face of a net reduction in the overall fitness of individual heterozygotes, natural selection (in the form of the DDK allele segregating, preferentially, to the egg) is predicted to result in a net increase in the fraction of DDK alleles in the population, simply because a higher fraction of eggs will contain DDK alleles.

Before our laboratory started working on this problem, I had been under the impression that examples of meiotic drive were rare. I was wrong. One of the more interesting examples of biological variation, without direct phenotypic consequence to the carrier, is the formation of *Robertsonian translocation chromosomes*. Such chromosomes are end-to-end fusions of smaller *acrocentric* chromosomes (each having a centromere at the tip of the chromosome) into a larger chromosome with two arms and a centromere in the middle. These chromosomal variants are created with extraordinary frequency—approximately 1 per 1,000 meioses in human females (which is several orders of magnitude greater than the frequency of any other genetic event)—and result in a meiotic pairing configuration in which one member of the pair must attach a single centromere to the meiotic spindle, while the other must attach two centromeres to the spindle (see Hamerton, Canning, Ray, & Smith, 1975). As a group, mammals have an unusually constant genome size of approximately 3×10^9 base pairs per haploid genome but vary widely in the number of chromosomes over which the genome is distributed, from 6 chromosomes (in the Indian muntjac deer) to

102 chromosomes (in a small South American rodent) (Scherthan, 2007). As it turns out, cycles of chromosome fusion/chromosome breakage and meiotic drive have shaped all of mammalian karyotypic evolution (Pardo-Manuel de Villena & Sapienza, 2001b), with individual karyotypes being driven to favor all, or most, chromosomes to be of one form (i.e., acrocentric, as in many mice) or the other (metacentric, as in humans and great apes).

This variation in chromosome form has proven a major mechanism of reproductive isolation (and, therefore, speciation) in mammalian evolution. Individuals from different populations that carry/do not carry the fusion chromosome are often perfectly interfertile. However, the resulting heterozygotes often have meiotic segregation difficulties, resulting in aneuploid gametes (having an abnormal number of chromosomes) with phenotypic consequences for the next generation (Gropp & Winking, 1981). In fact, closely related species exhibit such karyotypic differences far more often than might be expected by chance. A notable example is that human chromosome 2 appears as two separate chromosomes among the karyotypes of the great apes (Dutrillaux, 1980).

These examples of direct selection at the level of the gene (as represented by genetic variability in the structure of centromeres) affect reproductive fitness, which is the most important outcome measure in any discussion of population genetics. Although one might hedge that centromeres themselves are epigenetic structures, the innate biological variability between centromeres that allows them to be subject to meiotic drive is fundamentally genetic (Henikoff & Malik, 2002).

6 What Are the Limitations on the Unit of Selection Being "the Gene"?

I have a wonderfully clever and inventive colleague who works on G-protein-mediated signaling. We are often like-minded, politically and socially, and agree on many divisive subjects that might bring others to blows. However, we have never seen eye-to-eye on the "function" of G-proteins. Fortunately, we are each dismissive of the other's views on this topic, rather than confrontational, and so our disagreement does not cause much friction between us. Our disagreement stems, I think, from our views on natural selection and how natural selection might affect the function of G-proteins. My colleague believes that it is likely that selection has optimized all of the G-proteins to serve a unique function. I do not.[1]

This difference of opinion is likely to reflect our level of biological focus. My colleague would characterize himself as a molecular biologist. I characterize myself as a geneticist. Both of us are comfortable with the notion that natural selection can act on genes or, in this case, gene products. However, my colleague is much more reductionist than I am, and sees *all* genes or gene products as the product of natural selection. I am of the opinion that traits controlled by large numbers of genes are difficult, if not impossible, for natural selection to "see." I believe this must be so because, although it is likely that selection can optimize a particular subunit of a G-protein to bind guanosine triphosphate, I think that the very large number of combinations of protein subunits observed to participate in various forms of G-protein-mediated signaling is too large for natural selection to "see."

My colleague tells me that functional G-proteins are *heterotrimeric*, meaning that the functional proteins contain alpha, beta, and gamma subunits and that G-proteins are encoded by a family of genes, 35 in total: 16 alpha, 5 beta, and 14 gamma, all of which are scattered over several chromosomes (Dutrillaux, 1980). The interplay between various G-proteins and various receptors gives rise to a large number of complex phenotypic traits, many of which can be seen, with little imagination, to have adaptive significance. Our ability to discriminate between the odor of roses and the odor of manure, for example, is the result of the interaction of particular G-proteins with particular olfactory receptors in particular olfactory neurons. One might think, then, that particular G-proteins would have evolved to play specific roles.

However, my argument, simply stated, is that even if rare variants of a particular alpha, beta, and gamma subunit were to come together in an individual to reward him/her with the most exquisite sense of smell (rather like the character in the popular novel *Perfume* [Suskind, 1986]), the allelic combinations giving rise to this trait would be broken in the next generation because the genes are on different chromosomes and segregate independently. If only three unlinked genes were involved in this trait (a particular alpha, a particular beta, and a particular gamma subunit, for example), then $^1/_2 \times {}^1/_2 \times {}^1/_2 = {}^1/_8$ of gametes would carry the proper combination. Going beyond three genes requires ever greater population sizes and numbers of offspring or ever greater selective advantage for the variant trait, in order to bring natural selection face-to-face with the trait. If we use the minimum reproductive advantage necessary to maintain an advantageous trait in the population (we may use the one given in Professor Burian's paper: 1 additional offspring in $\sim 10^4$), then natural selection cannot, by definition, "see" beyond ~13 independently segregating genes ($2^{13} = 1$ in 8,096).

In practice, the minimum reproductive advantage required to maintain the trait is likely to be substantially greater, given changing environments or weak selection, reducing further the number of gene variant combinations that natural selection can see. The easiest way to get around this problem is to create situations in which the desirable gene variants do not segregate independently: to "link" the gene variants affecting the desirable trait on the same chromosome in gene clusters. Numerous examples of this strategy are available, including the large family of olfactory receptor genes whose signals are transduced by the G-protein signaling molecules with which we opened our discussion. If an advantageous combination of variant alleles at two separate genes were to become closely linked (as a result of a chromosome rearrangement, for example), they would ensure the inclusion of the advantageous combination in a half of gametes rather than a quarter of gametes (if unlinked). Given the demonstrated tendency for genomes to link genes that work in the same developmental or biochemical pathway in this fashion, I would argue that this outcome is not required if the unit of selection is something other than the gene but is a prediction of the hypothesis that the unit of selection *is* the gene.

7 The "Complexity" Argument: Do Complex Phenotypes Require Complex Explanations?

The notion that natural selection "sees" only traits that are the result of the action of multiple genes (i.e., organs or organisms) seems, to me, a bit like the irreducible

complexity argument used by supporters of creation science. The gist of their argument is as follows: The eye is a very complex structure that is able to receive visual stimuli and transmit those signals to the brain, where they can be processed into information upon which decisions that affect fitness may be made (climb the tree and avoid the wolves, stay on the ground and fight, or run?). If any one part of the eye were removed/did not function properly, it would fail to fulfill its function and, thus, the eye could not have been designed by natural selection but must have been designed by an intelligent designer. In the same way that I would argue that a badly functioning eye is better than no eye (ask any visually impaired person whether they would prefer to be completely blind or badly impaired), I *do* see how it is possible to add/subtract layers of functional complexity by changing one gene at a time.

I think that the ultimate argument under this heading is illustrated by the ultimate quantity that all evolutionary arguments must take into account when hypothesizing a selective advantage/disadvantage for *any* trait: biological fitness. At bottom line, fitness is simply the number of offspring provided to the next generation. Fitness is the sum-total of all of the biological, social, and environmental variables in operation during the lifespan of any organism. One might argue that fitness in the human population would be an incredibly complex issue, affected strongly by economic, social, environmental, and biological variables. I would have predicted, a priori, that tracing reproductive success to any particular variable would be impossible. Fortunately, my certainty on this subject can be listed under the comment of my former supervisor that "not everything I know is true."

A few years ago, a company called deCODE genetics Inc. was formed in Iceland, with the intention of "mining" the genetic variability of the Icelandic population in order to find genetic variation associated with common diseases. The social and political structure of Iceland makes such an endeavor easier, in some ways, than it might be in a more diverse population. The integration of birth and medical records with genotype information has made it possible to analyze fitness among virtually the entire population of Iceland as well as ask whether there are genetic factors that are correlated with fitness (Kong et al., 2004). The conclusion, from analyzing more than 14,000 offspring in more than 5,000 families, is that the women who had the largest numbers of offspring (women being the important variable) were those who had managed to reproduce at the oldest ages. In other words, all things being factored out across the entire Icelandic population, the women with the longest reproductive lifespan had the most children.

This may not seem terribly surprising, upon short reflection. However, what *was* surprising, to my mind, is that fitness correlated with the number of recombination events observed in their offspring. Females who had higher levels of recombination were able to reproduce at older ages, and had more offspring, than females who had lower levels of recombination. Because failure of recombination is a risk factor for aneuploidy—and 50% of spontaneous abortions are aneuploid (Hassold & Hunt, 2001), so this factor has a major effect on reproductive success—the suspicion is that the ova of females with more recombination events are less likely to be aneuploid at older ages than the ova of females with fewer recombination events.

It is possible that many genes could affect recombination rate, but it is certainly true that we, and others, have demonstrated inter-strain differences in recombination that are attributed to single loci (specifically, in mice) and that there are many cases

of one or a few genes affecting recombination rates dramatically in many organisms (de La Casa-Esperón et al., 2002; Kong et al., 2008). Overall, I would argue that, while it is certainly possible to envision situations in which the unit of selection might be something much more complex than an individual gene or small numbers of genes, the availability of real-world examples of the opposite tendency make me question the widespread utility of more complex explanations.

8 Do "Epigenes/Epialleles" Provide a "Non-genetic" Source of Heritable Variation Upon Which Natural Selection May Act?

Because I have taken the position that all or most heritable biological variation is based on true genetic differences between genes, it is important to address the possibility that variation caused by epigenetic differences is also heritable. By my definition, very little of this form of biological variation is heritable, because by *heritable*, I mean the variant epigenetic form must be transmitted to the next generation, unaccompanied by a causal genetic difference.

Most epigenetic differences survive somatic cell division. Somatic cells are all of the cells of the body except for sperm cells or egg cells. Indeed, faithful replication of epigenetic differences is the basis for differentiation and development: for example, progenitors of liver cells continue to produce liver cells and not brain cells (and it is this form of programming that is at the practical root of much of the embryonic vs. adult stem cell debate). The phenotypic difference between these genetically identical cells is based on the somatic heritability of *chromatin* structure. Chromatin is the complex of DNA, histone proteins, and other proteins that bind to DNA and small molecular modifications of DNA (the addition of methyl groups to certain combinations of letters in the DNA code) and modifications of histone proteins (the addition of methyl or acetyl groups to certain amino acids). The DNA of liver cells is packaged into chromatin differently than the DNA of brain cells, enabling different groups of genes to be expressed in each cell type. Liver cell-specific chromatin packaging is replicated from one liver cell division to the next. However, few, if any, of these epigenetic differences are transmitted through the germline to the next generation. There are a few examples of such "transgenerational" epigenetic inheritance (Rakyan et al., 2003), but most are also accompanied by genetic differences (Chong, Youngson, & Whitelaw, 2007).

Nonetheless, I must admit to being intrigued by the formal possibility of transgenerational epigenetic inheritance. It is certainly possible, if so far rare, for epigenetic variability to provide a *non-genetic* form of biological variability. This form of variability could provide a mechanism for the inheritance of acquired characteristics: if a particular environmental factor resulted in selection for turning on a particular combination of genes early in development and these genes were newly expressed in both brain and testes, for example, it is also possible that particular behavioral patterns dictated by the newly expressed genes could be programmed in the next generation by their chromatin packaging in some fraction of sperm cells. The demonstration of such a development would be exciting, indeed.

9 Summary Points: The Usual Unit of Selection Is the Gene

The following is a summary of the main points of this paper:

1 Much of complex genome structure reflects the accumulation of mobile elements that increase in number, regardless of effect on organismal phenotype.
2 There are examples of selection leading to replacement of one population of organisms by another—viz., near isogenic population—that differs from the original at only a single or small number of loci.
3 Selection, via meiotic drive/non-random segregation of chromosomes, can act directly on DNA sequences or chromosomal structures in the absence of gene products encoded by those particular sequences.
4 A substantial fraction of the observed variation in complex traits that are likely targets of selection can be traced to genetic differences at a small number of loci.
5 Even under the most generous estimates for how small a difference between entities natural selection can "see," no trait that is the product of more than 10–12 unlinked genes can be selected.
6 The problem of independent segregation of unlinked genes predicts that if traits are encoded by multiple genes, those genes will tend to become associated in fewer linkage groups and become easier for selection to see.
7 Although epigenetic variation would provide a rich source of potentially heritable, but non-genic variation, very few examples of transgenerational epigenetic inheritance have been documented. Most such differences are accompanied by underlying genetic changes or are strain/population-specific.

Postscript: Counterpoint

Of the arguments raised by Professor Burian against the idea that the usual target of natural selection is the gene, there are a small number with which I disagree, on the basis of the evidence or strength of argument. On the other hand, I am in agreement with Professor Burian that there is one issue that does require (or, at least, may require) selection to act on something other than individual genes. First, let us look at our disagreements, as these are the easiest to address.

1 What does selection at the level of the gene explain?

Professor Burian takes issue with the notion that quantitative traits—such as height, blood pressure, and serum cholesterol—that are influenced by environmental factors as well as genetic factors could trace the genetic component of their variance to a small number of genes. He is correct in that, indeed, there may be hundreds of genes that influence these phenotypes. However, my point is that the bulk of the variance, in these and many other quantitative traits, is attributable to variation in a small number of genes. The simple proof of this statement is that while diet and exercise

undoubtedly influence hypertension and/or hypercholesterolemia, a large fraction of the hypertensive or hypercholesterolemic population have altered their blood pressure or their serum cholesterol by taking drugs that target the product of single genes. Does this mean that there is only one gene that controls blood pressure or serum cholesterol? The short answer is no, but there are individual genes that have a disproportionate effect. Most models of quantitative trait variation suggest that multiple genes are at work and that their effects are *additive*, i.e., genetic variation at genes "1" through "*n*" results in the total genetic variance observed. However, the effect of each gene need not be equal. Although genetic variation at 20 loci may contribute to the total genetic variation, 50% of the trait variance may be due to the effects of two genes, with the remaining 18 genes each contributing a small amount to the remaining 50% of genetic variation. Under this scenario, selection may act strongly on the two genes with large effect and weakly, or not at all, on the remaining 18.

Additional evidence that multiple genes with unequal effect is a common feature of quantitative traits comes from whole genome association studies of other phenotypes. The completion of the human genome sequence and resequencing efforts to determine the amount of genetic variability between individuals has allowed geneticists to take an unbiased approach to determining what fraction of phenotypic variation maps to genetic variation in individual genes. Height, as Professor Burian notes, is a good example of a trait controlled by the effects of many genes. However, with respect to individual populations, whole genome association analysis has found that, although height is a polygenic trait, there are likely a few genes that explain the majority of the variation, while many other genes contribute to background "noise" (Liu et al., 2006). Different genes may be found in different populations (Perola et al., 2007), but whole genome association analysis, by nature, would not be so successful in finding genes with significant effects on these phenotypes if they were truly the result of hundreds of genes, each with equal effect.

In fact, I believe it is this difference in focus that underlies the disagreement between Professor Burian and me over the usual target of selection. If there are complex regulatory networks underlying most phenotypes, then my argument that natural selection cannot "see" so many genes (if they are unlinked) can be used as an argument that selection does not operate on genes. If, on the other hand, most phenotypic differences in complex traits have smaller numbers of genes at their root, there is no need to postulate that selection operates at a higher level. My view on this difference is reminiscent of the complex charts detailing the reactants, enzyme catalysts, and products of the linked biochemical reactions that make up glycolysis or the Calvin cycle or Kreb's cycle. Anyone who was forced to memorize the intricacies of these charts as a student also remembers that there were "rate-limiting" steps in most of these pathways. While all steps were important, the flow of reactants and products through the pathway was not controlled equally at every step, and mutations in genes in some steps in the pathway had a much greater phenotypic effect than mutations in others. A less academic example is the towing of cars stopped on the shoulder of the highway. While automobiles are enormously complex and have thousands of moving parts, the selection of any particular car by a tow truck does not often have to do with the failure of many parts (barring a crash) but is usually the result of failure of a particular part in one system or another and each

system (ignition, transmission, steering, braking, etc.) tends to have a hierarchy of components that are most frequent to fail.

My last minor disagreement with Professor Burian is over the role of epigenetics in producing heritable variation. A possible, and quite exciting, discovery would be that epigenetic variation could provide an important "non-genetic" source of heritable variation. In the case of the particular trait that I addressed earlier—viz., the contribution of genetic vs. epigenetic variation to differences in recombination rate—the difference between the male recombination rate and the female recombination rate may be almost entirely epigenetic. However, it is most likely that the inter-individual variation between females (which is the variable of interest in this case) is due to true genetic variation at loci involved in DNA repair rather than to epigenetic differences between females.

2 What *doesn't* selection at the level of the gene explain?

Professor Burian makes a valid point that there are cases in which it is difficult to argue that selection is operating on a particular gene or allele because the phenotype under selection is elicited only in combination with another gene or allele. Examples of *hybrid vigor* or *overdominance* (like Professor Burian's sickle cell example) have long fascinated geneticists because they represent cases in which a trait that is present in neither parent shows up in the offspring. However, it is worth pointing out that hybrid vigor (and cases in which the hybrid is less fit than either parent) is a relatively rare circumstance. Most hybrids, in fact, have phenotypes that are similar to one parent or the other (dominant traits) or some average/intermediate value between the two (additive traits).

Because the goal of this exercise was to present arguments for what is the usual and most likely target of selection, I believe some quantitative data are in order. The sequencing of human, mouse, and other animal genomes has been accompanied by the development of genome-wide and high-throughput methods in which it is possible to determine which alleles of which genes are being transcribed in tissues or individuals. Such transcription profiles may be compared between any two parents and their offspring to determine whether the amount of transcript of any particular gene resembles that of one parent or the other or is some average between the two. Although the quantitative trait being analyzed in this case (amount of transcript produced from an individual allele) may not always be the best measure of a phenotype produced by any gene, it is a useful phenotype for which one may ask, of thousands of individual genes, whether it is heritable, dominant, additive, or overdominant.

The most comprehensive analysis provided, so far, is from the work of Cui, Affourtit, Shockley, Woo, and Churchill (2006). These investigators compared transcript levels in two inbred strains of mice and their reciprocal F1 hybrids. Nearly 9,000 transcripts showed evidence of heritability in transcript level, with a median heritability of ~70%. Approximately 20% of the heritable transcripts exhibited dominance (levels similar to one parent), while the bulk of the remainder showed additive inheritance. Only 167 transcripts (less than 2% of the total heritable transcripts) exhibited overdominance. Consequently, these data suggest that it is comparatively rare to encounter cases in which natural selection is presented with a phenotype that could not be predicted by one or both alleles at each locus.

Note

1 G-proteins mediate cell signaling via guanine nucleotide binding and additional couplings with more than 800 different receptors (in humans). Cell signaling is a crucial biochemical process by which signaling molecules bind to receptor molecules that are specific and the binding of the signal to the receptor keys a change in a biochemical pathway. The details of the biochemistry are not important for this discussion (although they were important enough that their discoverers were awarded a Nobel Prize in 1994).

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CHAPTER EIGHT

Selection Does Not Operate Primarily on Genes

Richard M. Burian

This paper offers a review of standard views about the requirements for natural selection to shape evolution and for the sorts of "units" on which selection might operate. It then summarizes traditional arguments for genic selectionism, i.e., the view that selection operates primarily on genes (e.g., those of G.C. Williams, Richard Dawkins, and David Hull) and traditional counterarguments (e.g., those of William Wimsatt, Richard Lewontin, and Elliott Sober, and a diffuse group based on life history strategies). It then offers a series of responses to the arguments, based on more contemporary considerations from molecular genetics, offered by Carmen Sapienza. A key issue raised by Sapienza concerns the degree to which a small number of genes might be able to control much of the variation relevant to selection operating on such selectively critical organs as hearts. The response to these arguments suggests that selection acts on many levels at once and that sporadic selection, acting with strong effects, can act successively on different key traits (and genes) while maintaining a balance among many potentially conflicting demands faced by organisms within an evolving lineage.

1 Introduction

This paper addresses how selection "acts" on genes, organisms, and lineages of organisms and examines how to determine what sorts of "units" are selected.[1] I'll start by saying a few things about selection and about genes, and then review some traditional arguments about genes as units of selection. After that, we will examine some of Sapienza's arguments in the companion to this paper and develop some arguments against the view that selection acts primarily on genes.

2 Four Criteria for Natural Selection

This is not the place to examine closely what natural selection is or how it works. Ever since Darwin (1859) used artificial selection as a model for natural selection, it

has been important to recognize that natural selection differs in important ways from other forms of selection. The conditions set forth here concern *natural* selection, not selection in general. It suffices to set out four criteria that, according to a wide consensus, must be met for selection to influence the course of evolution. The criteria include (1) variation that (2) affects fitness, and (3) is heritable. Furthermore, (4) environmental conditions must be sufficiently regular to permit selection to be cumulative.

The entities subject to selection must go through iterated generations, and their properties (traits) must vary from individual to individual. When a biased sample of available variants survives over a series of generations, selection *may* be occurring. Effective transgenerational selection requires there to be heritable variation in fitness (Lewontin, 1970) and conditions permitting cumulative selection. *Variation* (criterion #1) is required for entities with different properties to be present in the next generation(s). Evolutionarily relevant variation must affect *fitness* (criterion #2), where fitness differences are, by definition, differences in *the propensity to survive and reproduce*.[2] In addition to variation and differences in fitness, *heritability* is required (criterion #3)—i.e., a positive correlation, on average, between the trait of the parent(s) and the offspring. This is necessary for differential survival to allow selection to favor or alter certain traits of the selected entities within lineages. If an entity's traits had no correlation with those of its offspring or with the distribution of those traits in the next generation, cumulative change over long periods would be extremely difficult to achieve and (if it occurred) would probably not be caused by selection. Finally, if, the entities in question faced different regimes with the odds of survival shifting arbitrarily in direction in every generation, the distribution of traits or phenotypes would zigzag with no cumulative effect. Thus, the environment (and environmental cycles) must be sufficiently regular for selection to be effective (criterion #4). The rationale for combining all four conditions is nicely presented in Sterelny and Griffiths (1999, pp. 29ff.). Their initial example of cumulatively effective selection in organisms is the cryptic coloration of a ground-dwelling bird living in a reedy marsh habitat. If the habitat varied rapidly enough, cumulative stabilization of the requisite pattern of mottling and barring would be most unlikely.

Even without a definition of natural selection, we have set out fairly stringent necessary conditions for effective selection to occur: heritable variation of fitness, with sufficient regularity of (environmental) conditions to allow cumulative selection to maintain or alter the traits of the organisms (or other relevant entities) over a long series of generations. The four conditions do not, as such, exclude natural selection from acting primarily on genes, which reproduce over a series of (cellular or organismal) generations. Although *illustrations* of selection in terms of *organisms* may prejudice one against selection acting *primarily* on genes, the four conditions set forth above do not, by themselves, reinforce that prejudice.

3 Genes

The term *gene* is about a century old. A Danish biologist, Wilhelm Johannsen, coined it in 1909 to stand for something almost wholly unknown (Johannsen, 1909). He thought, following Mendel, that the only thing known about genes was that, when organisms were cross-bred, different variants of a gene caused organisms to inherit

alternative versions of the affected trait(s) in a "Mendelian" pattern. Because male and female parents typically contributed equally to Mendelian traits, something contained in sperm and eggs (and therefore also contained in fertilized eggs) caused Mendelian differences between organisms. Mendel called the things that differed *factors*. When Mendel's work was rediscovered in 1900, many biologists tried to locate Mendel's factors, in specific ways, especially in germ cells, and tried to understand them in terms of cytological theories (e.g., as parts of chromosomes) or theories of organismal development. Johannsen argued that such moves were a mistake: no one knew what genes are—they might, for instance, be properties or states of the relevant cells rather than separate entities. The term *gene*, therefore, was supposed to be free of theories or hypotheses about the material constitution of genes and about the means by which they brought about their effects. The claim that a particular allele (gene variant) is present simply summarizes the phenomena; it is another way of stating the Mendelian behavior of the trait(s) caused by that variant (Burian, 2000; Carlson, 1966).

For the rest of his life, Johannsen argued that we did not know the intrinsic properties of genes or how to localize them. It was not even safe to claim that there were two copies of genes in fertilized eggs: if genes were something like a state of the egg or sperm and the fertilized egg was composed of a sperm and an egg in the same state, the egg would be in a single state; what we now call homozygotes would have a single state of that gene, whereas heterozygotes would be fertilized eggs (and organisms) with a mixture of two states of the relevant gene—states that separated in the formation of sperm (in males) or eggs (in females). Little wonder that the material constitution of genes was contested well into the 1950s; even those who were strongly convinced that genes are material parts of chromosomes did not know whether genes were made of proteins, nucleic acids, or some combination of the two.

Since 1909, there have always been multiple contenders for *the* definition of the gene, and every 15 or 20 years or so at least one new contender for *the* definition has been taken seriously by biologists (Burian, 2000, 2005; Carlson, 1966; Falk, 1986; Portin, 1993; Snyder & Gerstein, 2003; L. Stadler, 1954). This definitional problem sometimes resulted in sterile terminological debates, but the ruckus over defining genes raised major issues—many of which remain to be resolved—about the causes of biological inheritance and the extent to which specific factors caused inherited traits.

For the entire century since 1909, there have been intense struggles about how to define genes. A point that deserves immediate attention is that alternative definitions of genes always reflected major tension, never fully resolved, between attempts to provide *structural* (e.g., narrowly physico-chemical) and *functional* definitions of genes. A gene must, in the end, be some sort of material entity that performs immensely complex functions. It follows that structural and functional criteria for what should count as a gene are inextricably intertwined.

As it turns out, there is considerable advantage in combining structural and functional criteria for identifying genes. One can use criteria of each sort to help correct mistakes on the other front. This has been valuable ever since Johannsen's day, as geneticists and others worked to specify the factors that cause inherited traits. An easy example comes from the early stages of the chromosomal theory of the gene. Work on this theory began with T.H. Morgan's discovery in 1910 of a sex-linked

mutation for white eyes in the fruit-fly *Drosophila*. He showed, in effect, that the mutant gene is inherited together with the X, i.e., a sex chromosome.

In *Drosophila*, males have one X and females two Xs. Mutations carried on the X chromosome are therefore not recessive on males, so mutations are easily detected. X chromosomes could be followed from grandfather to grandson. By crossbreeding X-carrying males with the daughters of fathers with the same X, one can increase knowledge of the mutations it carries (by testing the effects on both homozygous and heterozygous females) and of how the mutation is transmitted. Cytological aberrations, such as an attached fragment of a foreign (previously tested) X chromosome, allowed additional predictions and tests. Standard histories of the Morgan group's contributions to Mendelian genetics follow the development of this line of work.[3]

Morgan and others in his laboratory quickly developed techniques for locating genes on specific chromosomes. Once genes were closely associated with particular positions (loci) on chromosomes, two genes with identical-seeming effects located on different chromosomes were assumed to be different structures that played a distinct role in the development or economy of the organism. This was verified fairly quickly by finding biochemical and other detailed differences in the effects of some genes yielding a common visible phenotype. A famous example concerns two genes that altered eye color in the same way: *vermilion* (located on the X chromosome) and *cinnabar* (located on chromosome 2). After about 20 years, it was shown that the *vermilion* mutation blocked a biochemical step in the formation of brown pigment in the *Drosophila* eye and the *cinnabar* mutation blocked a later step in formation of the same pigment. The twisty path by which this was accomplished, combining biochemical, developmental, and genetic tools, is sketched in Burian, Gayon, and Zallen (1988, pp. 389–400).

By the 1960s, when molecular biology was far enough developed to begin identifying genes with particular sequences of DNA, the traffic back and forth between structure and function became much more intimate. It turned out that genes could not be defined simply as sequences of nucleic acid, as lots of sequences are too short, too long, or seem to be junk, i.e., to have no known function or to perform a non-genetic function (e.g., to serve as spacers; see Sapienza's paper). This complexity reflects and supports the fundamental commitment retained through most of the history of genetics, namely, that proper identification of genes must combine functional with structural criteria. By now, effectively by definition, genes are structures of DNA (or, occasionally, RNA, as in RNA viruses) that are copied across organismal generations and that have some additional function(s) in virtue of being transcribed onto RNA that has specific potential to enter into, or to affect, additional biological processes. One consequence of this account is that there is no single answer as to what, exactly, should count as a gene, for that depends on exactly what parts of the genome count as having a function.

Interestingly, there are those who would retain the word *gene* only for protein-encoding genes. But this causes problems: for example, it would not count sequences of DNA encoding transfer RNAs and ribosomal RNAs as genes. Nor would it yield a univocal count of genes, since (by the standard criteria of current molecular biology) many plant and animal genes encode a few to hundreds of distinct proteins obtained by alternative splicing of their RNA transcripts, and some rare proteins are made from pieces assembled from distant transcripts of distinct genes.

4 Genic Selectionism

With this background, we are ready to turn to the question of whether or not selection operates primarily on genes. To begin, let's review some important early doctrines that influenced recent debates about our topic. A book that influenced many philosophers is G.C. Williams' *Adaptation and Natural Selection* (Williams, 1966), in which Williams argued that group selection plays a minor role in evolution, in comparison with individual selection, and that selection acts primarily on genes. The latter claim is what interests us. One of Williams' key arguments is that genes can be beneficiaries of selection, but organisms cannot because *organisms do not survive in evolutionary time, but genes do*. Understood as sequences or structures of DNA, genes survive recombination, death of the organism, and death of the species. Thanks to the mechanisms by which DNA is copied and by which sequences are maintained, genes have the potential to survive indefinitely. Thus, with an eye to meiosis, in which chromosomes may be broken up by "crossing over," Williams (1966) writes:

> I use the term gene to mean "that which segregates and recombines with appreciable frequency." Such genes are potentially immortal, in the sense of there being no physiological limit to their survival, because of their potentially reproducing fast enough to compensate for their destruction by external agents. They also have a high degree of qualitative stability. Estimates of mutation rates range from about 10^{-4} to 10^{-10} per generation. . . .
>
> Natural selection would produce or maintain adaptation [of genes] as a matter of definition. Whatever gene is favorably selected is better adapted than its unfavored alternatives. This is the reliable outcome of such selection, the prevalence of well-adapted genes. The selection of such genes of course is mediated by the phenotype, and to be favorably selected, a gene must augment phenotypic reproductive success as the arithmetic mean effect of its activity in the population in which it is selected. (pp. 24–25)

Mutation is inevitable, but evolved defenses against mutation protect organisms (and genes) well enough that when a variant of a gene confers an average selective advantage of one extra survivor/10^4 offspring, that variant will normally survive indefinitely. Although a number of qualifications are needed here, they don't matter for present purposes. On an evolutionary time scale, organisms, populations, groups of organisms, and even species exist for far shorter times than the genes (with their evolving variant forms) they contain. According to standard textbooks, species of mollusks and clams, which are longer-lived than most land species and are well-preserved as fossils, have a half life of about 10 million years. A standard estimate suggests that at most 3–4% of the mollusk species extant 35 million years ago are still in existence. By standard criteria, most genes (i.e., such gene *kinds* as transfer RNA genes, alcohol dehydrogenase genes, etc.), preserving their function through a lineage of descent, are far longer lived than that.

If these are the relevant comparisons, it is the genes that are the beneficiaries of selection. However, I note that once we recognize that we should compare *lineages* of genes with *lineages* of organisms, it is not clear that these are the relevant comparisons; lineages of organisms, like lineages of genes, last until extinction.

In *The Selfish Gene* (1976) and *The Extended Phenotype* (1982), Richard Dawkins popularized the above argument and expanded the scope of the controversy. Both

Dawkins and David Hull (1980, 1981) sharpened the argument by distinguishing between replicators and interactors, arguing that replicators are the main unit of selection. For Hull, a replicator is an entity whose structure is passed on fairly directly via a process (replication) that yields one or more reasonably accurate copies of the replicator. Evolutionarily relevant replicators must meet criteria of longevity, fecundity, and fidelity. The relevant form of longevity is *preservation of structure in the form of copies*. Hull's example makes the point clear: atoms of gold, structurally, are very similar to each other and quite long-lived; but because atoms of gold are not made by a copying process from previous atoms of gold, they do not count as replicators and are not subject to evolutionary processes like those affecting organisms and other biological entities. Dawkins' version of replication is more stringent: he requires nearly exact copying and, for relevance to selection, focuses on "active germ-line replicators," i.e., those that play an active role in favoring their own replication across organismal generations. Thus, on Dawkins' account, since organisms reproduce not by a process of copying, but by (re)constructing most of the materials (e.g., proteins) and structures out of which they are constituted, organisms are not replicators, not even single-celled asexual organisms. On Hull's less stringent account, single-celled asexual organisms are normally close copies of the parent from which they are produced, so they are perfectly good replicators. In spite of this difference, Dawkins and Hull agree that genes are exemplary replicators since their structure is preserved across enormous numbers of generations by means of the copying mechanisms that replicate DNA structure. Williams' (1966) definition of a gene as "that which segregates and recombines with appreciable frequency" is, effectively, a definition of a replicator. It is non-committal, at least on the surface, about the structure or function of genes as units, and Williams explicitly points out that this definition treats as genes what, by more conventional criteria, might be counted differently, e.g., as several linked genes or as no genes at all.

Hull's (1980) term for the other principal sort of unit, *interactor*, is preferable to Dawkins' term, *vehicle*. An interactor is an "entity that interacts as a cohesive whole with the environment in such a way that replication is differential" (p. 318). Dawkins' vehicles are explicitly restricted to entities within which genes (and perhaps other replicators) are contained. Organisms are the main entities that fit this description, but mitochondria and other DNA-containing organelles and groups and populations of organisms may also qualify. There are no exact boundaries for either replicators or interactors; they come in degrees of exactness of replication, and of cohesiveness plus relevance to differential replication. Still, organisms are built anew in each generation by an interactive process with the environment, not by a copying mechanism. This makes Dawkins's distinction between replicators and interactors quite rigid: replicators are made by copying, while interactors are made by some kind of construction. (Construction is my term; the classical embryological term is *epigenesis*, a process in which the entity is *built* rather than copied.) Hence, organisms cannot be replicators. Something is a vehicle for Dawkins only if it contains (and is built from, or by) replicators. A vehicle is evolutionarily important only insofar as its properties bias the likelihood of its replicators making it into the next generation. For Hull, too, an integrated whole counts as an interactor because its properties or behaviors affect the likelihood of replicators making it into the next (organismal) generation, but only some interactors contain the replicators in question.

The take-home message of these arguments is that replication involves entities beyond the replicators themselves, and that evolution is the result of interactions that bring about transgenerational replication of sufficient exactness to enable construction or reconstruction of organisms and other evolving entities. All this is supposed to be required for evolution via natural selection. Orthodox evolutionary theory holds that genes are generally the primary replicators and that organisms interact as unitary beings with each other and with the rest of their environments in such a way as to affect their likelihood of reproducing and transferring copies of the genes within them into the next generation. As Hull insists, replication and interaction are distinct processes; both are required for evolutionary processes. But the boundaries of these processes may overlap. Thus, on Hull's view, the same entity—e.g., an asexual single-celled organism when it divides—can be correctly considered to have replicated or to have reproduced.

5 Three Traditional Arguments

We are ready to present arguments that show that genes are not the primary units (targets or beneficiaries) of selection. Sapienza argues that selection *can* and (at least sometimes) *does* act on genes. He is right. But that's not our main question, which is whether selection acts *primarily* on genes. Nor does the question whether selection benefits individual genes or organisms solve our problem. Selection does not benefit individual copies of genes (gene tokens) any more than it benefits individual organisms. And successful *lineages* of organisms are just as long-lived as successful lineages of genes, so a surviving lineage of organisms is, as such, just as much or as little a beneficiary of selection as a surviving lineage of genes is. Given this, I will leave the issue of beneficiaries behind and concentrate on whether selection acts primarily on (lineages of) organisms or genes.

The arguments considered here apply directly to sexual organisms, especially multicellular eukaryotes, i.e., organisms whose cells have true nuclei containing chromosomes that are complexes of histones and DNA. On this score, the differences between prokaryotes and eukaryotes are important largely because of the specialized regulatory processes affecting the development and evolution of eukaryotes, and because the relationships between genes and their products, and between genes and functional traits, are much more indirect in eukaryotes than in prokaryotes. These features of eukaryotes make the argument easier and more decisive, but similar issues can be raised for prokaryotes.

(1) The first argument focuses on the causal processes involved in selection. Selection acts on marginal differences between organisms with systematically different traits in the actual ecological and competitive circumstances faced by actual organisms. It "acts" when some heritable genotypic or phenotypic traits increase the probability of reproductive survival. The conditions for this are already familiar: heritable variation in fitness plus a fairly consistent selective regime. As Sapienza argues, genetic differences (almost) always underlie trait differences when these conditions are met, so favoring a trait typically favors some constellations of genes over others. However, this cannot be *universally* true since genetically identical organisms (e.g.,

identical twins) do not have strictly identical fitness-affecting traits and since, quite generally, all kinds of environmental differences and learned responses alter the course of development, the response repertoire, and even the anatomy of organisms (Jablonka and Lamb, 2005; Gilbert, 2009). Not all heritability is genetic!

Thus, as a matter of bookkeeping, selection favoring a trait within a population favors not only that trait on average, but also some genes or gene combinations. Sometimes this may turn out to be one gene that is closely correlated with the selected trait. William Wimsatt (1980) offered a complex and influential argument that, in general, we should treat genetic changes that reflect phenotypic selection as a way of bookkeeping. The bookkeeping works because genetic changes track trait changes in evolutionary time—but not because changes of the individual genes, as such, yield a correct causal explanation of the evolutionary trajectory followed by the lineage of organisms.

One of Wimsatt's examples makes the point clearer. The case involves frequency-dependent selection involving simultaneous variation of two different genes, in which the selective effect of a given allele depends on the proportions of alleles present in the population at *both* loci. In such circumstances, whether selection favors or disfavors a given allele may depend on the frequencies of the alleles present at the other locus.[4] The result is a dynamic system driven by trait relations to ecological circumstances and by causal interrelations involving two genes and their products or effects; but it does not, in general, allow selection to favor any one gene variant across the full range of available environments and population distributions. Since seasonal and sporadic changes may dramatically affect the proportion of alleles at the other locus, no prediction of evolutionary outcome can be made from the selective advantage for a particular allele in a given situation in which the population has a particular composition. Although this only sketches the complexities involved, it makes the key points: without a clear and correct account of genotype–phenotype relationships and the relevant selective regime, one cannot determine, from the local fitnesses of alleles plus the specific trajectory of the genome in the lineage, whether the individual alleles that gained in frequency were the primary targets of selection. In general, then, even granting strong genetic causation of traits, it does not follow that gene-by-gene analysis suffices to explain the evolutionary trajectory of the organisms in question. Genes (and their alternative alleles) have the effects they do as part of a system of tightly integrated interactions. This is the stumbling block at the heart of the issue we are debating.

(2) In a classic paper, Elliott Sober and Richard Lewontin (1982) present a second, related, argument using the familiar example of sickle cell anemia. The sickle cell allele of a gene for hemoglobin is prevalent in populations exposed to mosquitoes carrying the malaria parasite. Without medical intervention, which only recently became feasible, individuals homozygous for this allele nearly always suffer excruciating death before reaching puberty from sickle cell anemia. Nonetheless, the allele is maintained at fairly high frequencies in malarial regions because it provides human heterozygotes with considerable resistance to malaria. In most common ecological circumstances, one copy of the normal allele produces blood cells with enough normal hemoglobin to prevent them from sickling and enables them to carry enough oxygen for carriers of the allele to be unharmed, but the amount of oxygen carried is low enough to

reduce the rate at which the malaria parasite can reproduce. In spite of the terrible cost of killing off homozygotic "sickler" children before they reproduce (roughly a quarter of the offspring of matings between heterozygotes), the net effect of carrying the allele in severely malarial environments is more favorable than not having this protection against the disease. Thus, in favoring resistance to malaria, selection has the most unfortunate side-effect of carrying sickle cell anemia along with that resistance.

The sickle cell story is actually more complex than this exposition suggests, but the complications don't affect the point. We have here, approximately, a simple case of heterozygote advantage. What we need to determine is whether selection acts primarily on the sickle cell allele. According to Sober and Lewontin, the answer is *no*: in malarial environments, selection favors heterozygotes, penalizes homozygotes drastically, and does not act on the allele as such. The allele has an average effect, of course, in a given mixture of environments (this is Wimsatt's bookkeeping!), but selection acts on the effect of the distribution of different hemoglobins in blood cells in environments with varying degrees of malarial severity. The hemoglobins in blood cells of homozygous normals favor malaria; the hemoglobins in homozygous sicklers alter blood cells so that they block capillaries and cause lethal oxygen starvation, those in heterozygotes allow blood cells to function well enough and yet weaken the malaria parasite. These are the differences on which selection acts. Sober and Lewontin argue that if it acts on any genetic entity, it is the genomic states of being homozygous normal, heterozygous for sickling, or homozygous for sickling. That is, genetically speaking, the conditions causally relevant to the phenotypic states that influence survival are the combinations of alleles, not the alleles themselves or their frequencies in the population. If so, at the genetic level, selection acts primarily on the genotypes, not the genes.

From this perspective, *selection acts on populations to increase the frequency of causally relevant genetic complexes* in the population over time. What this means in general depends on how genes or genotypes correlate with phenotypes. To get the causal story right, as the case of heterozygote advantage makes clear, one must examine the *genotypes* available in the populations and the dynamics of genotypic and phenotypic change over time.

Changes in the distribution of organismal genotypes will, of course, be tracked by changes in gene frequency, but the use of frequencies of individual genes, taken one at a time, as a surrogate for genotype frequencies ends up misrepresenting the actual causes of genetic change. This is made even clearer in a recent paper by Brandon and Nijhout (2006). They argue that genic selectionists must offer a false account of the causes that maintain a dynamic equilibrium when the sickle cell and normal alleles are maintained at a stable frequency. Baldly expressed, the correct causal account is based is on balancing selective pressures favoring the heterozygote and strongly disadvantaging the two homozygotes. However, since genic selectionists treat alleles (rather than combinations of alleles) as what is selected, they must view selection of alleles in this sort of case as frequency-dependent—and when the alleles are at an equilibrium frequency, there is no selective pressure acting to change the frequency of the alleles. This is obvious, because at equilibrium the average fitnesses of the normal and mutant alleles are equal: *there is no net force acting to increase or decrease the frequency of either allele*. In fact, however, *selection* is acting. It acts on

heterozygotes (who are moderately susceptible to malaria, but otherwise undamaged) and, more harshly, on homozygotic normals (who are highly susceptible to malaria) and homozygotic sicklers (who die from sickling of blood cells). Thus, in cases of heterozygote advantage, genic selectionists are forced to offer a drastically false account of the effective selective forces in a dynamic balanced equilibrium. More generally, *when the allele or the gene is used as the unit of analysis and interaction effects alter heterozygote fitness non-additively, calculating allele by allele yields a mistaken calculation of how selection acts.*

(3) A more diffuse literature concerning complex life histories provides a third argument. Take a relatively straightforward example: aphids. Here is a simplified account of a typical annual aphid life cycle (Ragsdale, 2002). Most species of these plant-sucking insects live in temperate climates and go through a kind of alternation of generations. During early spring, the offspring of an overwintering female, who mated at the end of the previous summer, are all females. These females yield a series of all-female generations that typically live for three to four weeks and reproduce parthenogenetically (without fertilization). The females just described are viviparous, that is, they do not lay eggs, but produce free-living daughters, already containing embryos. Even though these females and their daughters are genetically identical, what they eat can alter the size, morphology, and fertility of their daughters and granddaughters (B. Stadler, 1995). For example, it can determine whether those offspring are winged (and seek another host plant) or wingless (and stay on the same host). At the end of the summer, triggered by light, nutritional, and temperature signals, some of the females produce winged males and sexual females. Those females are oviparous, not viviparous, and lay overwintering eggs. The eggs that hatch in the spring produce stem females, who produce both winged and wingless parthenogenetic daughters and start the cycle anew.

Many organisms have yet more complex life cycles than those of aphids. Here are two major points to be taken from these sorts of cases. (A) Ecological (or nutritional, etc.) circumstances determine major features of offspring produced from organisms with a given set of genes. Indeed, in general, *the traits of the offspring of an organism are co-determined by environmental and genetic causes.* (B) Selection is episodic, but how it acts must be calculated over entire cycles of the right scale. This is part of the reason for the requirement that there must be suitable conditions permitting cumulative selection for natural selection to be effective. For example, the timing of environmental cycles must be short enough to be "visible" to selection, or selection cannot act to take them into account. Annual cycles work; 10,000-year cycles probably do not (even for redwood trees) and million-year cycles certainly do not.

Major genetic changes in aphids occur approximately once a year, during the sexual generation. That is when recombination and most other germ-line changes occur. The cumulative effect of many selective episodes on a lineage will be expressed, in part, by the robustness and the fecundity of the sexual female and the overwintering eggs she produces. And those properties of the female are affected by the circumstances encountered by the all-female lineage that preceded her for an entire season. There is, no doubt, a net average effect of the seasonal success and failures within her lineage on the female and a net average effect of the successes and failures of the clone within its local habitat, but there will be cases in which a

key selective episode that affected one of the great, great, . . . grandmothers of a sexual female is a key determinant of some features of her offspring. Thus, certain seemingly genetic traits—e.g., the fecundity of the sexual female or what proportion of the daughters of a parthenogenetic female have wings—may be determined less by her genes (which determine her capacities in these respects) than by her nutritional status and that of her maternal lineage during the current season.

Generalizing over the life histories of organisms that succeed in reproducing and about the ways in which selection manages to take account of what happens on regular cycles over long periods of time (which is a major part of what is at stake in evolution), *selection normally "acts" not as a single force on an isolated gene or trait, but as a balancing device in response to situations affecting multiple traits in scattered episodes over extended periods of time. In consequence, a balance of selective pressures affecting multiple traits is the norm, not a single force affecting an isolated gene (or trait). Successful lineages must cross all of the distinct thresholds that recur regularly during the relevant long cycles that arise over multiple years and/or generations.* This result shows why it is necessary to analyze the conditions for *cumulative* selection with great attention to long cycles affecting life history patterns. If all this is right, systematic devices for handling diverse difficulties—at once and in series—are typically involved in selective processes. For this reason, the genic selectionists' claim that selection acts primarily on single genes is prima facie implausible. The burden is on them to show how selection can work over such long periods in such complex ways by acting gene by gene in the manner described in Sapienza's contribution to this volume.

6 Response to Sapienza's Arguments: (A) What Does Selection Act On?

Let's start with hearts. Nearly everyone agrees that the function of a heart is to pump blood. (For an exception, see Cummins & Roth, this volume.) It must do so (in the absence of human intervention) for the animal's entire life. In every extant lineage, animals have almost certainly faced recurrent contingencies affecting hearts. When they present significant challenges to the heart (as do malaria and drought, for example), the lineage is likely to have evolved some provisions to deal with, or avoid, those challenges. As an engineering matter, one cannot simultaneously optimize all of the traits of an organ to meet all expectable contingencies. If optimizing blood pressure causes increased risk of myocardial infarction, selection must act in a way that balances the resulting trade-off. And since ecological and seasonal cycles present various stresses and demands that hearts must meet, the heritable properties of hearts must permit or enable them to adjust to the full range of circumstances that recur sporadically or regularly over tens or, even, hundreds of generations. The penalty for failure to do so is simple, but drastic: extinction.

In game-theoretic terms, selection does not optimize traits or organs, it satisfices (does at least well enough to get by). For our example, heart designs must make (or at least allow) trade-offs adequate to meet the expectable tasks and challenges posed over a long series of generations. Organisms in a lineage must overcome (at different times) the effects of muscular and nutritional stress, low blood pressure, high blood

pressure, blocked blood vessels, changes in oxygen supply, specific infections, drought, floods, temperature extremes, and so on, indefinitely. Lineages like ours, in which individuals grow hearts somewhat differently depending on ecological conditions and life histories (e.g., altitude, nutritional limitation, a sedentary life, or a life of long-distance hunting), do not have rigidly fixed genetic programs that determine all the traits of their hearts, for developing hearts respond differently to differing circumstances encountered in different ecologies, situations, and thanks to different life habits. Such phenotypic plasticity conflicts with rigid genetic control of traits.[5]

Two of Sapienza's claims about hearts are hard to reconcile unless they are carefully circumscribed. These are (1) that the variation in blood pressure between different individuals can be explained, in large part, by differences in genotype and (2) that the phenotypes under selection are controlled by a small number of genes. Take the first: variation in blood pressure can also be explained by variation in lifestyles (e.g., amount of exercise, daily hours of sleep), ecology (altitude and climate, availability of meat), and social and cultural factors (cf. correlations between heart health and various lifestyles). If Sapienza means (as is unlikely) that genotypic differences explain high blood pressure in general, independently of these other factors, the evidence is against him. Since we know that the blood pressure of most individuals can be altered considerably by controlling diet and exercise, and that early lifestyles and circumstances can have life-long effects on heart function, the claim I just ascribed to him is initially implausible and needs to be supported by specific evidence. If, instead, he means merely that the blood pressure has high (genetic) heritability, then he is right, but all that shows is that genetic variation has important effects on blood pressure, which is consistent with considerable environmental influence on it as well. Turning to the second claim, high heritability does not, as such, provide evidence that the phenotype in question is controlled by a small number of genes. Height is highly heritable, but it is not controlled by a small number of genes.

I believe that most organs and quantitative traits examined to date are like height in being controlled by a large number of genes. All in all, Sapienza's claim that blood pressure is controlled by a small number of genes is initially implausible. And since selection has been acting for many millennia to keep blood pressure within the bounds required by adequate health for reproductive survival, the fact that so much heritable variation is still available within human populations makes it likely that this phenotype, in particular, either is involved in trade-offs along the lines suggested above or is not after all controlled by a small number of genes.

This argument does not prove that Sapienza is wrong. Nor does it establish what claims in this neighborhood are right. Rather, it shows that specific and detailed knowledge of the relevant genotype–phenotype map is required to settle the matter. To argue more than this without going into serious molecular detail is, I think, hand-waving. Now that we can study such issues at the molecular level, their resolution requires successful analysis of the relevant genotype–phenotype relations.

6.1 Meiotic drive

Professor Sapienza's remaining examples don't bring us any closer to a resolution of our debate. I shall deal with them briefly and then close by taking up a more

general issue. Sapienza is entirely right that selection can act directly on genes. His example of meiotic drive in DDK mice illustrates the point nicely. Here, selection acts primarily on a particular allele because the relevant variation is between chromosomes containing the allele and chromosomes containing alternative alleles. Since the probability of the allele (and its chromosome) getting into the next organismal generation is altered by the direct effects of the allele itself, specifically its effect on the likelihood that its chromosome will make it into the egg, selection *acting primarily at the level of the gene* and not on other phenotypic traits of the organism secures the representation of this allele in the next organismal generation.

However, our main issue is not whether selection can act in this way, but whether it *typically* does so. And comparison with the heart example shows why this example of meiotic drive is not typical. The selectively relevant properties of hearts are not typically like the distribution of alleles or chromosomes into egg cells or polar bodies; they include such organ-level properties as the rate of pumping blood, the robustness of the organ, and its ability to operate continuously and withstand stress. In this connection, selection acts primarily on properties relevant to the demands of organismal survival imposed by the ecology and by competitors. In favoring organisms with more robust hearts, ability to maintain blood pressures within a suitable range, etc., it alters genomic organization and the content of genes over generations, but it does so secondarily, for it acts, in the first instance, on heritable phenotypes. As Sapienza explicitly grants, meiotic drive and organismal selection often favor different outcomes; selection on multiple levels requires a difficult balancing act.[6] Since selection does not act purely at one level, one needs to know the balance of selective pressures at different levels to calculate the net selective forces acting on (and net fitness of) a gene, genotype, or trait. Such a calculation complicates (and I think will ultimately defeat) all attempts to treat selection as acting primarily on genes.

6.2 Robertsonian translocations

This way of thinking, in terms of multiple levels and units of selection, carries over to the examples of Robertsonian translocations and G-proteins, both of which can be turned against the claim that selection acts primarily on genes. As Sapienza indicates, reproductive isolation of populations is a major step in speciation. Robertsonian translocations enforce reproductive isolation because members of a population that differ by a Robertsonian translocation cannot produce viable or fertile offspring. Where there is contact (or recontact) between populations separated by such a translocation, selection acts within each population to sharpen and tighten any other isolating mechanisms that reinforce the separation of the populations (e.g., by preference for mates from their own population), or to reduce the competition between them. If contact is maintained in the absence of additional isolating mechanisms, it is likely that members of one or the other population will eventually be eliminated (at least locally) by competitive exclusion or by reproductive failure.

I argue that Sapienza has not yet shown that the chromosomal variation and allied phenomena are, as he claims they are, "examples of direct selection at the level of the gene." He assumes, but does not argue, that when a change in centromere affinities

causes a chromosomal mismatch that leads to a Robertsonian translocation, *selection* is responsible for the chromosomal differences or acted to favor the allele(s) that favored the centrosomal change. But in principle and perhaps in fact, the chromosomal change may, rather, present the raw material on which selection may or may not act, resulting (if it acts favorably) in the formation of a separate population and, ultimately, a new species. If an isolated family, separated geographically from the main population, obtained the Robertsonian translocation by mutational accident(s), the new species might have been formed by mutation affecting chromosomes and drift alone! The fewer the mutations required and the smaller the initial populations, the harder it is to determine whether or not selection was involved. The role of selection in such a case is, thus, an open question. What is required, once again, is detailed knowledge of the relevant genotype–phenotype relations and enough information about the relevant selective regimes. Knowledge of the relevant properties of centrosomes and the distribution of genotypes is crucial, but it is not enough. *We also need to know the relevant grounds for the reproductive success of the affected organisms of the different genotypes in relation to competitors within and between populations* before we can decide the issue.

6.3 G-proteins

Sapienza's nicely nuanced account of the limitations selection faces in acting gene by gene on genes for G-proteins is very helpful. I agree with much of what he writes, but also with part of what he implies that his colleague maintains (though not the strong gene selectionism the colleague advocates). Sapienza is almost certainly correct that selection cannot act directly on the 35 genes encoding material used in various proteins to maintain, simultaneously, all of the distinct contributions of each gene to the G-proteins utilized by animals for recognition of scents, signal detection, and the many other functions that those genes affect. As he argues, the combinatorics for gene-by-gene selection are prohibitive. Yet, his colleague is surely correct that an adequate theory or mechanism is needed to explain how selection maintains these complex relationships. It is important to figure out the exact *explanandum*, that is, exactly what it is about G-proteins that needs to be explained (and that is not obvious!), but the coordination of G-proteins with distinct receptors is clearly the result of selection acting in some manner or other on the substrates out of which both the receptors and the proteins are manufactured. The puzzle about how selection can do this has not been removed simply by showing, as Sapienza has shown in outline, that it does not do so by acting gene by gene.

Some important disciplinary differences and differences in background assumptions underlie the differences between Sapienza's and my positions. These differences affect which phenomena and *explananda* we think are most important, our divergent accounts of how selection integrates effects from many genetic and non-genetic sources, and our views about what sorts of evidence are required to decide the issue. These disagreements are partly philosophical in character, but they are also scientifically productive. In the examples we have been discussing, they can lead us to experimentally concrete problems, potentially resolvable by experimental and populational studies.

7 Response to Sapienza's Arguments: (B) On the Importance of Epigenetics

Sapienza is right that biologists ought not (and, I am sure, will not) resort to arguments based on irreducible complexity; that would amount to the abandonment of science. But the first step in working toward new explanations in new domains is finding regularities that describe thus-far uncharacterized or unexplained phenomena. Recently, some philosophers of science have argued for the importance of exploratory experimentation in such contexts. It was suggested as a focal topic in philosophy of science a decade ago (Burian, 1997; Steinle, 1997) and its role in current molecular sciences has been explored in four recent papers (Burian, 2007; Elliott, 2007; Franklin, 2005; O'Malley, 2007). Given the complex regularities being teased out in post-genomic molecular biology (those concerning G-proteins are the tiniest tip of an enormous iceberg), Sapienza's argument serves, I think, as a *reductio* of classical versions of the position he set out to defend—i.e., the position that selection acts primarily on genes. He showed that the combinatorics of gene-by-gene selection are incompatible with fine-grained selection of G-proteins or maintenance of matches between G-proteins and their receptors. What is sought are alternative mechanisms by which selection can act, effectively, to build and maintain complex traits (involving, in this case, coevolution of multiple independent complexes of genes). In this closing section, I suggest that new work on epigenetics (and epigenetic inheritance) offers the prospect of solving at least some of these problems. Sapienza rightly left this possibility open in the concluding section of his paper and has explored possibilities along these lines in some of his technical papers (e.g., Sandovici et al., 2006).

Sapienza maintains that when traits are too complex to be maintained by selection acting on genes, they must have enormous selective advantage or they will be lost. This would be true if selection acted primarily on genes, but that is the very question we set out to debate. Phenotypic studies establish, I maintain, that selection "sees" or acts on complex organismal traits—and that G-proteins are a plausible example of a case in which selection maintains traits that can't be explained by (classical) genic selection. Sapienza argues correctly that we do not yet have a good theoretically grounded understanding of how complex traits are altered and maintained. At least two lines of research, however, may provide serious mechanistic answers to such puzzles.

The lines of research I have in mind overlap somewhat, but address a variety of different mechanisms. One line concerns RNA regulatory networks, the other concerns epigenetics (and specifically the study of epigenetic inheritance). I close with a couple of paragraphs on each to indicate some of their promise and hint at how they overlap.

Recent genomic work has yielded some surprises concerning the amount of genetic material that is transcribed and the great variety of regulatory RNAs included in genomic transcripts. John Mattick has published several articles summarizing the evolutionary importance of regulatory RNAs in the evolution of eukaryotes (e.g., Mattick, 2004). In mammals, for example, it appears that at least 80% of the DNA is transcribed (in humans it is probably 97% or more), and that much of it forms regulatory RNAs that enter into complex networks. These play a major role in regulating development

and determining what products will be made from which genes in which circumstances. In eukaryotes, the correspondence between genes and gene products is not 1:1: most protein-encoding genes make several (and some make up to several hundred) products by alternate splicing and/or by contributing to products by splicing of RNAs from distant parts of DNA.[7] Many of the controls that regulate which products are made in which cells and at which stage of development are located in RNA regulatory networks. These controls deal with everything from nutritional switching to responding to infectious agents, from switching cell type in differentiation to coordinating responses to heat or cold or infectious agents. There are at least a dozen major types of RNA involved in these networks, some of them very short—e.g., short interfering RNAs and microRNAs, each about 21–22 nucleotides long and capable of rapid response. Such controls alter the protein content of a cell and its descendants by interfering with mRNAs or altering the regulatory signals contained in mRNAs to regulate the developmental stage at which they are translated. These controls also respond to nutritional state of the organism, the presence of specific toxins or pathogens, the entry of signal molecules into the cell, and so on.[8]

Some mechanisms deployed to regulate development in RNA regulatory networks coordinate gene expression for large numbers of genes. For example, a developmental switch that coordinates the transition from maternal to zygotic mRNAs in the zebrafish (a standard model organism for such work) is a microRNA, labeled miR-430 (Schier & Giraldez, 2006). Expression of miR-430, itself produced in large numbers at a specific moment in embryonic development, blocks a regulatory region in the untranslated regions of at least 700 maternal mRNAs in somatic cells or the embryo, thereby ensuring that they are degraded quickly rather than slowly over an extended period. It does so by base pairing with its target while attached to a protein complex that then disrupts the target mRNAs. The disruption occurs coordinately in all somatic cells of the embryo. However, in the germ cells, the same microRNA protects the maternal mRNAs from degradation. Thus, the regulation is highly specific and coordinates the timing or loss of expression of immensely complex batteries of gene products (and, apparently, gain of expression of their zygotic replacements as well) in cell-specific and stage-specific ways (Schier & Giraldez, 2006). This example illustrates coordinate regulation of immensely complex traits and processes that can be regulated in many fine-grained ways by changing only a few nucleotides or a relevant environmental variable, e.g., by altering the timing of the expression of miR-430, by altering its sequence, or by altering the relevant regulatory sequences (and hence the mRNAs) of the genes of affected maternal proteins one at a time. We do not understand this or related mechanisms in detail, but it is immediately obvious that they provide strong controls of complex coordinated batteries of genes.

It has been experimentally demonstrated that such controls can be modulated in fine-grained ways to alter the timing of developmental changes, the number of genes affected, and the size of the effect. The combinatorics of such regulation, unlike those of G-proteins proceeding allele by allele, appear to be tractable and their flexibility quite extraordinary. The connection with the second line of research, on epigenetics, is straightforward. Many changes in RNA regulatory networks are heritable, some of them genetically (e.g., by mutation of the DNA from which microRNAs are constructed), some of them epigenetically.

The term *epigenetic* goes all the way back to Aristotle (see the papers in Van Speybroeck, Van De Vijver, & De Waele, 2003). The historical use that fits most readily with present usage contrasts the theory of predetermination of embryos (which held that the fertilized egg already contains, in miniature, the "form" of the adult or all of the major organs of the embryo) and the theory of epigenetic development of embryos (which held that the fertilized egg does not contain the form of the adult, or all or its organs, but that these are built by a series of developmental changes requiring internal and external resources in an extended developmental process). In the last few decades, the term has acquired a new meaning. An epigenetic change is a heritable change independent of any change of nucleotide sequence in the DNA (or genetic RNA) of the organism in question. A clear example of epigenetic change in development is provided by gene or chromosome silencing. For example, methylation of chromosomes (particularly at CpG sites—i.e., DNA sites at which the nucleotides cytosine and guanine are linked by a phosphate) can alter the conformation of the DNA in such a way as to prevent transcription of that region. This is one way of achieving so-called "genomic imprinting," in which chromosomal regions are silenced by epigenetic mechanisms. Genomic imprinting is a crucial control of gene expression in development, required, for example, for proper differentiation of secondary sexual characteristics. In mammals, it is now well-established that many chromosomes are imprinted differentially according to whether they come from the male or female parent, and that imprinting is epigenetically inherited (see, e.g., Sandovici et al., 2006; Wood & Oakey, 2006). There is a large literature establishing that imprinting is removed from chromosomes very early in embryonic development, but various marks that serve as cues for reestablishing imprinting are left, that this process involves no change of DNA sequences, that one of the major mechanisms involved is methylation of CpG sites, and that "correct" or nearly "correct" imprinting is required for normal development.[10]

I mention this example for a particular reason. At the close of his paper, Sapienza cites the work of deCODE genetics Inc. on differences in reproductive rates in Icelandic families and their finding that the women with the highest reproductive rates are those had the highest rates of meiotic recombination. Another of their findings suggests that there may be some connection with epigenetic inheritance involved in this result. The single genomic marker with highest correlation with a high rate of recombination was the fraction of the genome with a CpG motif, i.e., with the markers for methylation and silencing of chromosomal regions. Like some of the work in Sapienza's lab (Sandovici et al., 2006), this suggests, but does not provide definitive evidence, that epigenetic inheritance may play a role in the mechanisms affecting recombination rate. I do not understand these mechanisms and do not pretend to know whether this suggestion will pan out, but it is a nice illustration of the range of open questions still to be faced before we can reach a definitive answer about the mechanisms underlying selection of complex phenotypic traits. It also is a marker of the fact that an adequate case for selection acting primarily on genes has not yet been made.

One more example of an epigenetic process illustrates the power of epigenetic change to release hitherto hidden phenotypic potential of the organism and then to "lock in" the altered phenotypic properties of the survivor. This concerns the research on heat shock protein 90 (hsp90) (Rutherford, 2003; Rutherford & Lindquist, 1998; Wagner,

Chiu, & Hansen, 1999). In barest outline, the story goes like this: hsp90 is a "chaperone" protein that marks cells to be killed if they have certain abnormal proteins. Experiments with mice and fruit flies have shown that if hsp90 is depleted by a heat shock early in embryonic development, it allows molecular variants with strong phenotypic effects to survive the molecular controls that normally would kill cells with those abnormal proteins. The result is unusual phenotypic variation in the surviving animals. Since hsp90 recognizes normal proteins by the conformation of the relevant proteins present in the earliest stages of development and the altered proteins are transmitted maternally, they are present during early development of the offspring. Thus, the surviving conformal variants of the affected proteins are processed as normal in the offspring of the heat-shocked animals, so the standards employed at the molecular level for recognizing normal proteins have been inherited epigenetically with significant phenotypic consequences. As I understand it, this outline is correct—but the details matter immensely and a great deal of important research must be examined with great care before it is clear whether an account like the one given here can be generalized to resolve significant puzzles of the sort raised by Professor Sapienza.

Postscript: Counterpoint

Like Professor Sapienza, I begin the postscript by pointing out some points of agreement. He is correct that selection "acts" directly on genes that are able to spread themselves within genomes over organismal generations. Among such "selfish" genes are those that produce meiotic drive or produce transposable copies that incorporate themselves in the genome. Sapienza is right that the importance of such phenomena has not been adequately appreciated and needs to be carefully assessed—the high prevalence of "ghosts" of retrotransposition events in the human genome provides ample evidence of that! It will be particularly important to evaluate the impact of retrotransposition on genome organization, genome evolution, and the evolution of organisms.

Given this, I have no doubt that, as Sapienza argues, selection acting directly on genes has had important effects on evolution and evolutionary history. So far so good. But our core issue concerns the centrality of such phenomena in evolution. In this connection, Sapienza's arguments do not establish that selection acts primarily on genes (or on DNA). In spite of the important genetic findings and mechanisms he uses to suggest difficulties for some of my arguments, the fundamental points I made are not touched by those difficulties. The key issue is the balancing act that is required when selection acts at several levels over periods of time sufficient for evolutionary shaping of genomes and organisms. Genes and DNA are as susceptible to this problem as any other traits that are affected by natural selection. My counterarguments in this brief reply illustrate this point as well as shortcomings in some of Sapienza's key arguments.

The fact that variants of some genes (currently) have disproportionally larger selectively relevant effects than variants of other genes on phenotypes of interest does not prove very much. Some of the recent literature on genetic influences on human diseases shows that *which* genes have disproportionate influence changes with

the ecology. Our knowledge here is only now being developed and is not very secure, but in a couple of examples it is both strong enough and suggestive enough that I think it makes the point rather nicely. One example concerns genetic resistance to AIDS. Recent work, reviewed by Dean et al. (2002), has shown that some HIV viruses interact with chemokine receptors to enter cells. The dominant HIV viruses in initial HIV infection are HIV-1 viruses that enter solely through the CCR5 receptor. (These are called HIV-1 R5 strains.) Several groups of humans (mainly with ancestors who lived in northern Europe) have a 32-nucleotide deletion in the gene encoding the CCR5 receptor. (This allele is known as *CCR5-Δ32*.) Homozygotes exhibit a near (but not quite total) resistance to HIV-1 infection, and heterozygotes exhibit significantly slower than normal progression of AIDS. Subsequent population genetic studies, yielding exceptionally high concordance with the data on the distribution of the *CCR5-Δ32* allele in tested populations, explain the distribution of this allele as the result of strong episodic selection favoring this allele because it also produced resistance to the black plague (Duncan, Scott, & Duncan, 2005). Given very detailed knowledge of the timing and incidence of plagues, the expected distribution of the allele matches the available data closely; other suggested selection pressures and histories that might account for the distribution of the allele do not come close to doing as well.[9]

Such population genetic data and scenarios, though made plausible by our understanding of the specific mechanism of action of both HIV-1 and our (less thorough) understanding of its contribution to resistance to black plague, are not by themselves wholly decisive. But in this instance they appear to provide the best currently available account of a significant source of genetic resistance to HIV infection. And even though it does not decisively establish the selective grounds for the prevalence of *CCR5-Δ32* in various populations, it illustrates exactly the sort of scenario that I think will prove to be quite common: episodic, strong selection, acting sporadically, crucial in maintaining a balance in the arms races between hosts and disease vectors, predators and prey, invading competitors, and "physical" environmental conditions such as drought, flood, climate change, and the like. Such sporadic selection will heighten the impact of one or another genetic or genotypic variant in a population (especially at population bottlenecks), with a sometimes dramatic effect on the genetic and/or phenotypic composition of the population. Just like the "ghosts" of episodes in which numerous transpositions occurred, so the "ghosts" of these strong selection episodes will remain in affected populations, available for mobilization when ecological conditions prove favorable. Selection must balance the impact of such episodes over long intervals of evolutionary time, while maintaining sufficient genomic stability, organismic viability, and reproductive capacity for maintenance of the lineage. This is the image of evolution with which I operate. If it is sound, it is not plausible that selection operates primarily on genes.

I have just argued that to understand selection pressures on key genes we often need to understand the history of the relevant ecology/ies. Although I have no direct evidence bearing on the genes that affect heart function, there is a plausible view in the semi-popular medical literature that illustrates the sort of issue that needs to be evaluated in connection with Sapienza's arguments about the high selective value of variation of only a few of the genes affecting heart function. It is often suggested that the ecological change wrought by modern agriculture, together with increased

wealth and food availability in many cultures, has radically altered the dietary regime of humans. Large populations now have access to sweets, fats, and an abundance of calories that was extremely rare in human history. (This is combined, of course, with radical changes in public health measures, medical care, protection from infection, and much more of the sort.) The increase in longevity, but also in obesity, type II diabetes, hypertension, and heart attack, is related to the changes in diet and lifestyles. Heart attacks occur predominantly at an age beyond that to which most humans lived until a relatively few centuries ago. It is not implausible that the selective value of the genes that Sapienza discusses, the ones with highly heritable effects on hypertension, etc., have altered significantly with the alteration of dietary regime, the altered longevity of humans, and the other changes just alluded to. Again, the genes that affect hypercholesterolemia surely have other effects than their effects on cholesterol levels. I do not know what these effects are, or whether they are relevant to the long-term selective values of the alleles at issue. Before one jumps from the positive impact of the medications blocking excessive cholesterol in medically relevant circumstances to the selective values of certain alleles, a vast range of additional information is required. Such information may be available (though I suspect it is not), but until it is presented and subjected to careful evaluation, the case has not been made that these medical results suffice to determine the selective value of the genes whose action has been blocked. As best I can see, Sapienza's argument about the relative paucity of overdominance in high-throughput tests does not address these points.

My argument is not decisive. It does not prove that selection acts primarily via multi-level and multi-factorial causation, but neither does Sapienza's argument make a decisive case that selection acts primarily on genes. It is, of course, problematic for his view that selection can "see" a relatively small number of independent genes bearing on a selectively relevant trait at once, caught up as they are in immensely complex networks. In general, it seems more plausible that selection "acts" on some of the specific traits that result from the workings of genes and gene control networks and that the chips fall as they may for individual genes. This would be one way in which selection could "act" on phenotypes (or intermediate steps toward phenotypes) such as pathogen and drought resistance and leave the appearance that much mutation is neutral. But it is also important to recognize (as the case of *CCR5-Δ32* suggests) that selection can "act" episodically, and when it does so, it need not "see" all the genes on which it acts at once. Selection can act sequentially on different genes, during relatively short periods of intense selection when, due to some salient circumstance (such as the flourishing of a major new pathogen), the impact of one allele rather than another, given the stable configuration of the rest of the network, makes a large difference to organismal survival or reproductive success. It can act on the few salient genes in humans that affect hypercholesterolemia rather drastically when they lead sedentary lives, overeat drastically, and don't have the right medical remedies available for averting the consequences of doing so. Similarly, to support a point that Sapienza made in his postscript, when a transposable element breaks the restraints that have kept its transposition in check or enters an organism that has no such restraints, it can spread enormously rapidly until controls on its spread are established. But unless such controls are ultimately established, the genomes in which it is spreading will ultimately break down—and when adequate

constraints to prevent genomic breakdown are established, they will involve a balance between organismal and genic factors. It is precisely the need for such a balance that I believe is omitted from Sapienza's account of the matter.

Let me return, finally, to Sapienza's position about the relatively few genes that, he suggests, are salient for the maintenance of hearts. It is a non-trivial question whether the situation here is more closely analogous to sporadic selection altering the genes affecting cystic fibrosis and resistance to AIDS along the lines I sketched or to genes affecting height in the manner Sapienza suggests. This question is far from closed by our arguments. My response to Sapienza's argument is a bit like a just-so story—but with a difference. I don't pretend to know the truth in this matter. Rather, I am putting forward a plea for major extension of research to determine the balance of circumstances that influence the selective values of alleles *and other biological factors* that affect heart function. The research required should take full account of the wide range of relevant conditions at many different levels that must be considered before reaching a firm conclusion about the balancing act required of selection in cases like this. Arguments about the percentage of trait variance that is currently due to a given gene or allele don't carry much weight until they have been put through studies that cover relevant intervals of evolutionary time and relevant ranges of environments, and until the full range of (presumably pleiotropic) effects of the relevant genes have been taken into consideration. If my argument is correct, resolution of such questions requires an integrated examination of selectively relevant systems and contingencies at many levels, including the genetic, genomic, cellular, organismal, and ecological. Resolution of the debate about whether selection acts primarily on genes will remain unresolved unless it integrates considerations of life histories, diet, hormonal mechanisms, neurological mechanisms, and genetic and epigenetic mechanisms. But the very fact that all these considerations are relevant suggests that selection acts in multifarious ways. It also suggests that although selection "acts" on genes, it does not act primarily on genes.

Notes

1 The term *unit of selection* is due to Lewontin (1970), which is a fundamental paper for the theory of selection. A useful anthology of the literature to 1983 on this topic is Brandon and Burian (1984); an excellent brief review is Mayr (1997).

2 The addition of "and reproduce" causes difficulties because conditions increasing the likelihood of survival may conflict with conditions favoring reproduction. Again, exactly how should we calculate the number of offspring relevant to *reproductive survival*? Take a pair of nesting birds: should we count the number of eggs, the number of hatchlings, the number of fledglings, or the number of sexually mature offspring? If some offspring are sterile, should that reduce the count?

3 Darden (1991) provides a particularly useful philosophical treatment of the importance of cross-disciplinary interactions between cytological study of chromosomes, genetic study of mutations, and the co-association (linkage) between mutations when they are on the same chromosome.

4 Concrete examples of such situations are well-known; Wimsatt includes one in his paper.

5 Many biologists have taken up phenotypic plasticity recently: Massimo Pigliucci (2001) and Mary Jane West-Eberhard (2003) published important books on the topic that are

philosophically sophisticated, and Brandon (1985) put forward an early argument showing its importance for human genetics.

6 A classic paper by Lewontin and Dunn (1960) provides a beautiful demonstration of selection acting simultaneously in different ways at three different levels in house mice. The mice experience meiotic drive, organismal selection, and group selection between small demes in a relatively closed environment.

7 There are many other regulated devices that alter the correlation between coding DNA and the products derived therefrom. Examples include RNA editing and post-translational modification and splicing of polypeptides.

8 For a review of work on microRNA pertaining to the issues of this paper, see Burian (2007).

9 In the interest of brevity, I omit a second medical example discussed briefly by Dean, Carrington, and O'Brien (2002). This concerns cystic fibrosis, a genetic disease (or condition) caused by a gene that appears to have been strongly selected because it produces to typhoid fever produced by *Salmonella typhi*, as was demonstrated by Pier et al. (1998). And because both the evolutionary issues about CF and AIDS resistance are not tractable to direct molecular resolution, I should also cite a paper that has demonstrated at the molecular level that severe selection such as that produced by typhoid fever and Black Death can produce stably modified populations with novel resistance genes present in high proportions. The paper (Navas et al., 2007) reports experimental evolution of *C. elegans* challenged to survive on food laden with a fatal pathogen.

10 See Jablonka and Raz (2009) for a recent review of epigenetic mechanisms and their biological importance.

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PART V

ARE MICROEVOLUTION AND MACROEVOLUTION GOVERNED BY THE SAME PROCESSES?

Introduction

Microevolution usually refers to the changes in allele frequency *within* a species that ultimately affect the phenotype of organisms that make up that species. *Macroevolution* refers to the changes that are *across* species, such as when a new genus, phylum, or family emerges (forms of speciation), or when species go extinct (for more on species, see Part III of this book). This is why, in the first paper in this part, Michael Dietrich notes that: "Patterns of variation within a species are classic examples of microevolutionary phenomena, while patterns of phyletic change associated with either punctuated equilibrium or mass extinction are recognized as examples of macroevolutionary phenomena."

With respect to microevolution, common examples of patterns of variation within a species exist all around us, including the emergence of coloring that camouflages several species of animals like birds, butterflies, beetles, and moths. This sort of microevolutionary change in phenotypic quality (here, coloring) is usually caused by the change of allele frequency in a gene pool of a population. There are turkeys that blend in with the brush, an order of insects called *Phasmatodea* that includes "walking sticks" which can be mistaken for small tree branches, and viceroy moths that look almost exactly like monarch butterflies (Owen, 1980).

There is a famous case study of peppered-colored and black-colored moths in industrial England that is regularly spoken about in books in the biological sciences (e.g.,

Audesirk, Audesirk, & Beyers, 2008; Berra, 1990). Because of the changes of gene frequency in their population, some of these moths were pepper-colored while some were black-colored. The pepper-colored moths could blend in nicely with the lichens on England's trees, camouflaging them from predators (mostly birds), while the black moths would be eaten because they did not blend in as well. Now, as England was industrializing in the eighteenth and nineteenth centuries, more and more soot from factories accumulated on the trees, making them blacker in appearance. During this time, the moths that just so happened to have developed black pigmentation were camouflaged from predators on the trees, while the peppered moths would be eaten by predators. The change of allele frequency in the gene pool of this population of moths that gave rise to either peppered coloring or black coloring is a clear example of straightforward microevolution.

Conversely, with respect to macroevolution, patterns of phyletic change are most readily available from fossil evidence. Consider the so-called "Cambrian explosion" of a multitude of new species that probably arose as a result of punctuated equilibrium (Morris, 1998). Or, think of the various transitional forms that link one species to another in macroevolutionary events such as the now widely accepted view that certain species of theropod dinosaurs gave rise to birds. *Archaeopteryx* is one clear candidate for this transition (Witmer, 2002).

So, there is no doubt or debate *that* these kinds of micro- and macroevolutionary events have occurred (and are continuing to occur). There is a debate, however, as to whether macroevolutionary processes are *reducible* to microevolutionary processes (for more on reductionism, see Part I of this book). In his paper in this part, Michael Dietrich wants to argue that, in fact, microevolution and macroevolution are governed by the same processes—to a certain extent. We say "to a certain extent" because he does claim: "Rather than deny that distinct macroevolutionary processes are possible and present in nature, I claim that such processes are possible in the case of species selection, but are relatively rare and so are of minor evolutionary consequence when the entirety of the domain of evolutionary biology is considered."

In his paper, Doug Erwin argues that microevolutionary and macroevolutionary processes are distinct through examples such as the occurrence of species selection in Cretaceous mollusks, the origin of morphological novelties in higher taxa of the fossil record, and the very distinctiveness itself of patterns of selectivity during mass extinctions like the ones that occurred at the end of the Cretaceous (65.6 million years ago) and at the end of the Permian (252.4 million years ago). So, the debate still rages on and, in the words of Todd Grantham (2007), the question still remains: "Is macroevolution more than successive rounds of microevolution?"

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CHAPTER
NINE

Microevolution and Macroevolution are Governed by the Same Processes

Michael R. Dietrich

When Theodosius Dobzhansky discussed the distinction between micro- and macroevolution in his landmark book *Genetics and the Origin of Species* (1937), he accepted that evolution below and above the species level could be distinguished, but that they were not produced by fundamentally different processes. In doing so, Dobzhansky advocated a theory of evolution unified at different levels by common processes, most notably gradual change over time shaped by natural selection (Dobzhansky, 1937; Provine, 1988; Smocovitis, 1996). Dobzhanksy's unification was quickly pulled into controversy as Richard Goldschmidt (1940) argued for a "bridgeless gap" between micro- and macroevolution. Today the divide between micro- and macroevolution still exists, but not as Goldschmidt imagined it. Most scientists would accept that there are distinct phenomena that can be categorized as microevolutionary and macroevolutionary. Patterns of variation within a species are classic examples of microevolutionary phenomena, while patterns of phyletic change associated with either punctuated equilibrium or mass extinction are recognized as examples of macroevolutionary phenomena. The question, however, is whether there are also distinct processes that underlie these phenomena. Dobzhansky's answer and the answer from the evolutionary synthesis was that there is not. In this paper, I will review the historic controversy over the distinction between micro- and macroevolution in order to clarify the terms of the dispute. I will then turn to contemporary arguments for macroevolutionary processes. Rather than cast this debate in strongly polarized terms that simply deny that distinct macroevolutionary processes exist or could exist, I will reframe the debate in terms of the relative significance of distinct micro- and macroevolutionary processes. I will argue that microevolutionary processes are much more frequent than uniquely macroevolutionary processes, and so of greater significance for the domain of phenomena that encompasses both micro- and macroevolution.

1 The Bridgeless Gap?

Theodosius Dobzhansky's 1937 book *Genetics and the Origin of Species* was a landmark for the evolutionary synthesis not because it offered a new theory of how Mendelian genetics and Darwinian evolution could be integrated, but because it articulated a program of experimental research to support that integration. This methodological orientation is crucial for understanding Dobzhansky's perspective on the relationship between micro- and macroevolution (Eldredge, 1989; Gould, 1982b). Dobzhanksy (1937) thought of macroevolution as requiring "time on a geological scale," but he argued that the only way to understand "the mechanisms of macro-evolutionary changes" was through "a full comprehension of the micro-evolutionary processes observable within the span of a human lifetime." This epistemic limitation leads him "reluctantly to put a sign of equality between the mechanisms of macro- and micro-evolution, and, proceeding on this assumption, to push our investigations as far as this working hypothesis will admit" (p. 12). Perhaps this reluctance was also a product of his mentor's advocacy of distinct mechanisms for the two processes, but, for present purposes, I want to point out that Dobzhansky's aim was to elucidate the mechanisms or processes of evolution, and he thought the best path to such understanding was through a program of experimentation that could be accomplished during a researcher's lifetime. Dobzhansky did not deny that there may be macroevolutionary processes; he was making a methodological claim about what could be known with the program of experimental evolutionary genetics that he had proposed.

Goldschmidt wrote his 1940 book *The Material Basis of Evolution* to demonstrate that the then known facts of evolutionary biology and genetics could support exactly the opposite conclusion suggested by Dobzhansky's unifying synthesis. Reviving a tradition of saltationism, Goldschmidt argued for a bridgeless gap between species and so a bridgeless gap between the processes of micro- and macroevolution. Building on his views on genetic organization and mutation, Goldschmidt proposed a new genetic mechanism, called *systemic mutation*, for the production of the large-scale morphological changes that he thought marked the creation of a new species. Systemic mutation was the result of many chromosomal rearrangements that create a new pattern or genetic structure. This new pattern in turn creates a new chemical system—new sets of reaction pathways corresponding to new phenotypes (Goldschmidt, 1940, p. 203). In Goldschmidt words,

> [W]hether this model is good or bad, possible or impossible, the fact remains that an unbiased analysis of a large body of pertinent facts shows that macroevolution is linked to chromosomal repatterning and that the latter is a method of producing new organic reaction systems, a method which overcomes the great difficulties which the actual facts raise for the neo-Darwinian conception as applied to macroevolution. (p. 249)

The idea for systemic mutation arose from research on position effect, especially H.J. Muller's work on the effects of minute rearrangements over an extended section of chromosome that produced different scute mutations in *Drosophila* (Dietrich, 2000b). Goldschmidt sought to bolster the case for this new mechanism of speciation by creating an analogy to the possibility that large phenotypic effects could be produced by small mutations in developmentally important genes. These developmental macromutations could very rapidly lead to new phenotypes and new species, which

Goldschmidt called *hopeful monsters*. This second process for macroevolution relied on the idea that the developmental and physiological processes of gene action could be extremely powerful and as a result transform a small genetic change into a large phenotypic change. Years of research in physiological genetics and well-known phenomena such as homeotic mutants made the idea of developmental mutations with large effects much more acceptable than the supposed repatterning underlying systemic mutation (Dietrich, 2000a). Theodosius Dobzhanksy and George Gaylord Simpson accepted that developmental mutations could occur and could lead to rapid morphological change. Sewall Wright even incorporated these mutations with large phenotypic effects into his shifting balance theory of evolution. Acceptance of these "large mutations" does not mean that Dobzhanksy or Simpson accepted that they were mechanisms of rapid speciation. Indeed, they maintained that small mutations in developmentally important pathways were another source of variation that could produce variation within populations that was then subject to all of the processes admitted for microevolutionary change and, by accumulation, macroevolutionary change.

Sewall Wright (1949), however, was open to the possibility that

> a homeotic gene, capable of being carried at low frequency throughout the species because of low penetrance . . . may thus be tried out in all localities with a reasonable chance of encountering a genetic and environmental milieu in which it is superior to type and in which there is sufficient isolation to permit crystallization about a new species type. (p. 48)

Wright was careful to note that mutations with large effects did not necessarily have any direct effect on reproductive isolation and speciation. They could accelerate the process of divergence among geographically isolated populations and so lead to a new species. Even for Wright, then, developmental mutations did not contribute to a process that was distinct from the microevolutionary mechanisms championed in the synthesis (Dietrich, 2000b).

The divide between micro- and macroevolution did not fade away after Goldschmidt's genetic mechanism of systemic mutation was rejected during the evolutionary synthesis. Indeed, Goldschmidt's ideas were revived by Stephen Jay Gould beginning in 1977. In an article on Goldschmidt's hopeful monsters, Gould (1977) claimed that, as "a Darwinian, I wish to defend Goldschmidt's postulate that macroevolution is not simply microevolution extrapolated, and that major structural transitions can occur rapidly without a smooth series of intermediate stages" (p. 24). These postulates formed the core of the idea of punctuated equilibrium that Gould and Niles Eldredge were championing at the time. In his introduction to a new printing of Dobzhanksy's *Genetics and the Origin of Species*, Gould (1982b) wrote that he and others were critical of the "synthesists' premise that gradual change of gene frequencies within populations serves, by extrapolation, as an adequate model for all evolutionary events." Instead, Gould and his allies believed that

> a common set of genetic principles produces different patterns of change at various levels of the evolutionary process, and that several bulwarks of traditional microevolution—change by gradual and sequential allelic susbstitutions, each with small effect, and the adaptive nature of virtually all change, for example—do not always apply to macroevolution. (p. xxiv)

While Gould appreciated Goldschmidt's argument for a distinction between micro- and macroevolution, he did not approve of Goldschmidt's proposed mechanism of

macroevolution by systemic mutations (Gould, 1982a). If Gould had a real interest in the evolutionary processes proposed by Goldschmidt, it was in the production of hopeful monsters by mutations in developmental processes, not systemic mutations (Gould, 2002, p. 68).

The distinction between micro- and macroevolutionary processes remained with evolutionary biology throughout the twentieth century, as did skepticism that there are any causal processes unique to macroevolution. Goldschmidt's proposal of a mutational mechanism used only in macroevolution was quickly rejected by leaders in the evolutionary synthesis. His proposed mechanism of producing hopeful monsters fared better, but it is not a uniquely macroevolutionary process. Indeed, the fact that changes in developmentally important genes could be another source of microevolutionary variation was a key to its acceptance.

I understand the current debate over distinct macroevolutionary processes in terms of a claim that there are significant causal processes that are found only in evolution above the species level. Goldschmidt's ill-fated systemic mutations constituted this kind of uniquely macroevolutionary process. His developmental mutations or hopeful monsters could produce macroevolutionary outcomes, but they could also produce microevolutionary effects. As a result, mutations in developmentally important genes do not constitute a distinct and uniquely macroevolutionary causal process.

This distinction between process and outcome is crucial for contemporary research as well (see Millstein, 2002). For instance, in 2002, Matthew Ronshaugen, Nadine McGinnis, and William McGinnis published the results of a series of experiments on the Hox gene, Ultrabithorax (*Ubx*). Comparing *Ubx* genes from *Drosophila* and the crustacean *Artemia*, they found that crustacean *Ubx* proteins had a series of serine and threonine residues at the C-terminus, while insect *Ubx* proteins did not. Deleting these serine and threonine residues or replacement by alanine produced *Ubx* proteins that repressed limb formation in a manner characteristic of an insect hexapod body plan. Ronshaugen, McGinnis, and McGinnis (2002) conclude that they have found the "first experimental evidence that links naturally selected alterations of a specific protein sequence to a major morphological transition in evolution." They propose that the "successive removal of Ser/Thr residues might quantitatively influence repression function and morphology, allowing viable microevolutionary steps toward 'hopeful monsters' with macroevolutionary alterations in body shape" (p. 917). Note that the outcome is macroevolutionary (a dramatic change in limb number and body plan), but the process is microevolutionary (the gradual deletion or substitution of serine and threonine in the *Ubx* C-terminus). This paper and many others in the evo-devo literature are uncovering the mechanisms for the production of macroevolutionary outcomes. The question here is whether those mechanisms are unique to macroevolution. Mutation in developmentally important genes does not constitute such a macroevolutionary process that is distinct from microevolutionary processes, even if they produce macroevolutonary outcomes.

2 Species Selection

While Gould revived Goldschmidt's memory in the 1970s and 1980s, the introduction of species selection by Steven Stanley in 1975 represented the true return to the

discussion of causal processes unique to macroevolution (Stanley, 1975, 1979). Species selection represents the most clearly articulated macroevolutionary process in part because it is explicitly a hierarchical expansion of microevolutionary processes. Building on earlier discussions of patterns of species births and deaths in Niles Eldredge and Stephen Jay Gould's work on punctuated equilibrium (Eldredge & Gould, 1972) and discussion of species as individuals, proponents of species selection developed it as a higher-level analogue to organismal selection (Eldredge, 1989; Gould, 2002; Stanley, 1975).

So, just as organisms each have a birth and a death as well as properties that allow them to act as cohesive individuals, species have a birth and extinction and properties that allow them to act as cohesive individuals (Gould, 2002; Hull, 1980). Natural selection at the organismal level is understood in terms of differential reproduction and survival of organisms caused by heritable variation in fitness, which in turn is a product of the interaction between an organism's traits and its environment. At the species level, species selection refers to the differential reproduction and/or extinction of species caused by heritable variation in fitness (Grantham, 1995). While organismal selection and species selection are deliberately analogous, species selection must represent a distinct causal process if it is to be considered a unique macroevolutionary process.

In order to understand species selection as a causal process, we must first distinguish it from sorting, which is the "differential birth and death of individual entities in a population" (Lloyd & Gould, 1993, p. 595). Sorting makes no claims about causal processes. Sorting can be the outcome of a number of different processes, including selection of traits at a different level. Indeed, selection for certain traits possessed by organisms in a population can create a pattern of differential reproduction at the species level. Because the traits and causal interactions relevant to selection are at the organismal level, this is a case of organismal selection and species sorting.

Further thinking about species selection divides roughly into two camps: those who favor an emergent character approach and those who favor an emergent fitness approach (Grantham, 1995; Lloyd, 1988; Lloyd & Gould, 1993). Since both of these approaches deploy concepts of emergence, a short detour is in order. Emergent properties are properties of a physical system that cannot be reduced to the properties of its constituent parts at lower levels. Put another way, emergent properties are those that arise from the ways that constituent parts interact with one another and are generally considered to be not predictable from simply knowing the constituent parts, and as such constitute phenomena distinct from any lower-level phenomena (Grantham, 2007). Regardless of the many nuanced ways in which emergence can be elaborated philosophically, what is needed for the discussion of macroevolution is a sense in which emergence captures causal processes that cannot be reduced to the interaction of lower-level entities alone—emergent properties must have some causal power of their own at their higher level (Grantham, 2007).

Elisabeth Vrba is the principal advocate of the emergent character approach to species selection. In a series of papers, she and her co-workers contrasted the selection operating on "aggregate organismal characters" with selection acting on "true species-level characters" (Lieberman & Vrba, 2005; also see Lloyd, 1988). Only selection acting on species-level characters qualified as species selection. As such,

Vrba (1984) defined species selection as "that interaction between heritable, emergent character variation and the environment which causes differences in speciation and/or extinction rates among variant species within a monophyletic group" (p. 323). The strength of this definition is that it identifies unambiguous cases of species selection by excluding aggregate characters. Aggregate characters are species-level features that are the sum of lower-level parts. Population means, medians, and many other statistical measures are properties of the population, not their constituent organisms alone, but they are calculated directly from those organismal values (Gould, 2002, p. 658). Selection can alter an aggregate character, but it does so by acting on the underlying organisms. Vrba (1989) designates as effect hypotheses these cases where causal interactions at the organismal level have effects at the species level. The effect hypothesis describes macroevolutionary phenomena, but it does not describe a distinct macroevolutionary process. The causal process generating the species-level effect occurs at the organismal level and, so, the species-level effect is reducible to a microevolutionary process.

The emergent fitness approach to species selection advocated by Elisabeth Lloyd and Stephen Jay Gould admits aggregate characters as long as they contribute to irreducible species-level fitness (Grantham, 1995; Gould, 2002). According to this approach, "any species-level trait that imparts an irreducible fitness to species in their interaction with the environment defines a true process of selection at the species level" (Gould, 2002, p. 659). The emergent fitness approach depends on a correlation between a species-level trait and species-level fitness. These species-level fitnesses are themselves emergent in that they cannot be the result of correlations between traits and fitnesses at lower levels (Grantham, 1995; Lloyd & Gould, 1993). The emergent nature of species-level fitness is crucial if this approach wants to claim that it is reflecting uniquely macroevolutionary causal processes. According to Vrba (1989), the "acid test of a higher level selection process is whether it can in principle oppose selection at the next lower level" (p. 388). If species selection is a distinct macroevolutionary process, then it should be able to oppose selection at the organismal level. Vrba's emergent character approach passes this acid test by refusing to consider aggregate species-level characters. Lloyd and Gould's emergent fitness approach passes this acid test by demanding that species selection applies only to those species-level traits that are correlated with emergent fitnesses at the species level.

Defining species selection in terms of species-level fitness has one important advantage over species characters: the emergent fitness definition is much more expansive (Grantham, 1995; Lieberman & Vrba, 2005; Lloyd & Gould, 1993). The stricter emergent character definition includes fewer cases and has less potential for future cases. The emergent fitness definition, because it can apply to aggregate traits as well as emergent traits, can include more cases of species selection.

Gould also argues that the emergent fitness approach offers a better explanation of a classic case of species selection: David Jablonski's studies of species survivorship and geographic range in Cretaceous mollusks (Gould, 2002; Jablonski & Hunt, 2006). In 1987, Jablonksi proposed that geographic range size in Cretaceous mollusks was an example of species selection because "range size was heritable and correlated with species survivorship" (Jablonski & Hunt, 2006, p. 556). Other paleobiologists proposed that larval ecology determined geographic range size and population structure, thereby rendering this a case of organismal selection. To distinguish

between these alternatives, Jablonski sampled mollusks that had planktotrophic larvae, which swim and feed and, so, are capable of dispersal, and non-planktotrophic larvae, which do not feed and are less capable of dispersal (Jablonksi & Hunt, 2006, p. 557). Species with planktotrophic larvae should have a larger geographic range and longer species survivorship as a result of dispersal. Species with larger ranges survive longer because they are less susceptible to local forces that might lead to extinction of a more restricted species. Gene flow across the large geographic range would also reduce the rate of speciation for planktotrophic species. Non-planktotropic species will have smaller ranges and less gene flow as a result of dispersal. As a result, populations will become genetically isolated more quickly and so have a higher speciation rate. Jablonski also demonstrated that geographic range was heritable—parents and offspring have similar ranges.

Geographic range, then, is a species-level property that is heritable, varies across species, and is correlated with species-level fitness measured in terms of speciation rate (Grantham, 1995). Geographic range, when it is understood as the product of complex interactions of many lower-level factors, seems like an emergent character. However, if geographic range is dependent on an organismal trait, like planktotrophy, then geographic range seems like an aggregate character—a product of the action of many planktotrophic organisms. Nevertheless, Gould argues the geographic range that is produced by planktotrophic or non-planktotropic organisms is correlated with emergent species-level fitness (Gould, 2002, p. 661). Jablonksi reaches the same conclusion in his 2006 paper with Gene Hunt. In their words, "The consistent positive relationship between range size and species survivorship regardless of underlying larval biology suggests that emergent fitness . . . in these mollusk species was causally related to the sizes of their geographic ranges" (p. 561). With regard to the emergent character and emergent fitness approaches, the point here is that the emergent fitness approach allows Gould, Jablonksi, and Hunt to claim this as a genuine case of species selection without having to demonstrate that geographic range is itself an emergent character.

Species selection represents the best case for a distinct and unique causal process operating only in macroevolution. Once species selection is recognized, however, the question becomes: how frequently does it occur? In 1995, Todd Grantham noted that "at present, only a handful of well-documented cases require hierarchical explanations. I suspect that number will remain small" (p. 318). In his 2002 book, Stephen Jay Gould wrote, "I freely admit that well-documented cases of species selection do not permeate the literature" (p. 710). But, he notes, this paucity may be simply the result of the recent recognition that scientists ought to be looking for species selection at all. The emergent fitness approach broadens the range of possible cases of species selection and in doing so strengthens the case for the prevalence of distinct macroevolutionary processes.

3 The Macroevolution Dispute as a Biological Controversy

By definition, controversies are extended disputes. However, they need not be disputes between diametrically opposed positions (even if the various chapters in this book

sometimes give that impression). Many of the most well-known controversies in biology have been "relative significance" disputes (Beatty, 1997). What is at issue in these disputes is how much significance should be ascribed to any number of rival theories, where the relative significance of a theory is defined as "roughly the proportion of phenomena within its intended domain that the theory correctly describes" (Beatty, 1997, p. S432). Historical disputes from the Mendelian–Biometrician controversy to the Fisher–Wright debates, to the Classical–Balance controversy and the Neutralist–Selectionist controversy can be understood as disputes over how much of the domain of evolutionary genetics could be correctly explained by either position (Beatty, 1997; Dietrich, 2006; Skipper, 2002).

Of course, the domain of evolutionary genetics, like the domains of every field within science, also has changed over time. These changes in the domain of phenomena can have profound effects on an ongoing controversy. Like many disputes in biology over the past 100 years, the dispute over the existence of distinct processes for micro- and macroevolution is a matter of relative significance. Lloyd and Gould (1993) note as much in their discussion of species selection when they claim that: "Almost all major questions, and great debates, in natural history revolve around the issue of relative frequency: for example, selection and neutrality, adaptation and constraint." For them the question is, does species selection "display a high relative frequency among the causes of trends?" (p. 598). Although the dispute over distinct macroevolutionary processes has been polarized, denying the existence of any uniquely macroevolutionary process is not necessary. The dispute over distinct micro- and macroevolutionary processes is not an all-or-nothing affair. Instead the debate should be framed in terms of the relative frequency and significance of macroevolutionary processes.

Rather than deny that distinct macroevolutionary processes are possible and present in nature, I claim that such processes are possible in the case of species selection, but are relatively rare and so are of minor evolutionary consequence when the entirety of the domain of evolutionary biology is considered. The scope of the domain of evolutionary biology is crucially important here. I do not deny that there are well-established cases of unique macroevolutionary processes in the form of established cases of species selection. I do not deny that more will be found. My claim is that these form a small portion of the domain of evolutionary phenomena that includes evolution, both above and below, the species level. This does not deny their existence or historical impact as evolutionary processes—it merely notes their *current* relative significance. As more cases of species selection are recognized, its relative significance within the domain of evolutionary biology may increase. However, because species are part of a hierarchy of biological levels at which selection could occur, it can never become more frequent than selection at lower levels. Put another way, species are composed of individual organisms, which themselves are under selection for many different traits. Selection for these many organismal traits will always outnumber cases of species selection simply because there are many fewer species than organisms. The process of selection at the organismal level is therefore of greater relative significance within the domain of evolutionary biology when compared to the process of species selection.

Macroevolutionary processes can be made much more significant if the domain in question is limited to macroevolutionary outcomes. Within a macroevolutionary

domain, both microevolutionary and macroevolutionary processes would be recognized by most scientists, but their relative ability to produce macroevolutionary outcomes would be in dispute. Restricting the domain to macroevolutionary outcomes will have obvious appeal to advocates of distinct macroevolutionary processes, but it generates greater relative significance only by excluding evolutionary phenomena. To those who advocated a unified evolutionary biology during the evolutionary synthesis, this restriction may seem undesirable. To those biologists engaged predominantly with macroevolutionary outcomes, this restricted domain will seem more natural. Even with a restricted domain, however, the relative paucity of established cases *in the current literature* of macroevolutionary processes, such as species selection, would give greater relative significance to microevolutionary processes.

So, where does this leave the dispute over macroevolutionary processes? In a winner-takes-all controversy between opposing positions, we might expect the accumulation of evidence to eventually convince most participants of the veracity of a single theory. Relative significance controversies in biology typically have not ended this way. Instead, they gradually depolarize as participants accept a form of pluralism that recognizes both theories and some evaluation of their relative significance. As more cases of species selection accumulate, unique macroevolutionary processes will be acknowledged. How long it will take evolutionary biologists to reach agreement regarding their relative significance will depend on an array of factors from the scientific to the sociological. If the track record of other relative significance controversies in biology is any indication, however, we will have a long time to wait to see this form of the dispute over macroevolutionary processes resolved (Beatty, 1997; Skipper, 2002).

Postscript: Counterpoint

In his contribution, Erwin argues for a "hierarchical ordering of the evolutionary processes through time" that renders it immune from reduction to microevolution, because explaining the origins of these hierarchies requires "a historical theory of evolution, one that encompasses an understanding of how evolution itself changes the evolutionary process." To support his contention, Erwin offers the example of the evolution of regulatory "kernels" (Davidson & Erwin, 2006a). These kernels are "evolutionarily inflexible subcircuits" of gene regulatory networks (Davidson & Erwin, 2006a, p. 796). Erwin and Davidson suggest an important role for these regulatory kernels in the evolution of animal body plans. Moreover, since these kernels are highly conserved and so resistant to change, they are offered as non-reducible to types of genetic change ("from single base substitutions to gene duplications") that are associated by Erwin and Davidson with microevolution (Davidson & Erwin, 2006a, p. 800).

While it is true that evo-devo has produced exciting new results that have created a new synthesis that more fully incorporates developmental biology with evolutionary biology, has it produced evidence of unique macroevolutionary processes? Does the recalcitrance of regulatory kernels to change mean that the evolution of these kernels is a macroevolutionary process? Erwin and Davidson admit that kernels originate through a process of microevolution. Once they have formed, however, they resist small modifications and so force natural selection to accept or reject the

kernel as a whole. The phenotypic result is that kernels "lock in structures" and act as a "mechanism for developmental constraint" (Davidson & Erwin, 2006b). If we grant that this is how regulatory kernels act, the key question is, do they have effects on morphological structures *only* at the species level and higher? If kernels produce effects only at the macro-level, then they would qualify as elements of *uniquely* macroevolutionary processes. This is a strong claim, however, and one that Erwin has not fully supported. In their 2006 response to a paper on regulatory kernels, Erwin and Davidson claim that kernels are not macromutations (mutations that produce new species, see Dietrich, 1992). They also claim that there is an only "imperfect" link between "Linnean hierarchy and the hierarchical levels of regulatory evolution" (Davidson & Erwin, 2006b). Both of these claims seem to admit the possibility that regulatory kernels could have morphological impacts both above and below the species level. If kernels (or any other regulatory change) can give rise to microevolutionary outcomes, then they cannot be heralded as uniquely macroevolutionary.

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CHAPTER
T E N

Microevolution and Macroevolution Are Not Governed by the Same Processes

Douglas H. Erwin

Do evolutionary trends encompassing large clades and extending over millions to tens of millions of years require a different suite of evolutionary processes than those amenable to investigation over shorter time scales? This question is the heart of the distinction between the large-scale patterns of macroevolution and the population to species-level patterns of microevolution. This is not a theoretical debate, for while in principle there is no reason why macroevolutionary patterns could not be explainable by microevolutionary processes, there are equally valid theoretical arguments establishing the possibility of macroevolutionary processes. Rather, it has been an empirical debate about whether larger-scale patterns require sorting and selection between larger entities, generally species or clades. The argument may, however, be too narrow. Both microevolution and macroevolution are inherently uniformitarian, failing to consider the historical dimensions of evolution. The resolution to the apparent schism between macroevolution and microevolution lies in the recognition that through time history has progressively packaged the variation in more inclusive ways, changing the locus of selection.

1 The Domains of Microevolution and Macroevolution

The fossil record documents the rapid diversification of animals in the Cambrian (530 million years ago [mya]), and flowering plants in the Cretaceous (ca. 120 mya), trends of increasing or decreasing body size that may persist for millions of years and the abrupt appearance of new species. Each of these *patterns* of evolutionary change has been characterized as *macroevolutionary* in nature, reflecting events that have happened above the species level. Other macroevolutionary events include large-scale temporal and

geographic patterns such as large-scale diversification patterns, mass extinctions, the origins of evolutionary novelties, and patterns of morphological disparity. Since Dobzhansky introduced the term to English-speaking evolutionary biologists in 1937, its use has engendered considerable controversy over whether distinct *processes* must exist to account for such patterns, a debate that persists today. In other words, are microevolutionary processes a sufficient explanation of macroevolutionary patterns, particularly as documented by the fossil record, or do emergent properties exist at or above the species level that require reference to macroevolutionary processes?

In this paper, I will introduce the variety of meanings of *macroevolution*, address the issue of whether any disjunction between macro- and microevolution reflects a hierarchical expansion of the levels at which selection operates, and then turn to the view that novel mechanisms may be responsible for macroevolutionary change. I will also address the issue of whether mass extinction represents a sufficient alternation of regimes from microevolution to represent a distinct process. Finally, I will suggest that while hierarchical expansion may be operating, both macroevolution and microevolution suffer from a similar flaw: the implicitly uniformitarian underpinnings of evolutionary thought. The process of evolutionary change itself changes the probabilities of subsequent evolutionary change (having become an elephant, learning to fly is rather difficult). Population genetics, the quantitative underpinning of the microevolution of the modern synthesis, is fundamentally ergodic; but evolution, like economics, is not. Curiously, this perspective receives support from an unlikely source: the work of John Maynard Smith, arch opponent of Stephen Jay Gould's macroevolution. Thus, strong challenges to conflating the processes of macroevolution with microevolution come from three separate and distinct areas: (1) selection among species, (2) the sources of variation and constraint, and (3) the non-uniformitarian nature of the evolutionary process itself. Establishing a disjunction in any of these three areas would be sufficient to demonstrate that macroevolution and microevolution are not governed by the same processes.

2 Changing Meanings of Macroevolution

The meaning of the term *macroevolution* has evolved considerably over the past 80 years, reflecting developments in evolutionary theory. The Russian geneticist and developmental biologist Iurii Filipchenko introduced the term in 1927, proposing that non-Mendelian cytoplasmic inheritance was responsible for the formation of taxa above the species level (Filipchenko, 1927, cited by Burian, 1988; see discussion in Erwin, 2000). Filipchenko's student, Theodosius Dobzhansky, introduced the term into English in 1937, writing: "[T]here is no way toward an understanding of the mechanisms of macroevolution, which require time on a geological scale, other than through a full comprehension of the microevolutionary processes. . . . For this reason, we are compelled, at the present level of knowledge reluctantly to put a sign of equality between the mechanisms of macro- and micro-evolution" (p. 12). Dobzhansky's statement must be put in context. When he wrote, discussions of larger-scale patterns in evolution often invoked either macromutationism (e.g., Goldschmidt, 1940), or internally driven orthogenesis, each of which rejected the primacy of natural selection in evolutionary change. Dobzhansky was primarily concerned with solidifying a

rigorous, experimentally tractable approach to the study of evolution with natural selection at its core. He thus rejected any unique macroevolutionary processes that did not involve natural selection. Neither macromutationism nor orthogenesis was supported by experimental investigations of the time, and no other theory of macroevolutionary process was on offer, so Dobzhansky argued that emphasis must be placed on the microevolutionary processes that were subject to experimental study.

Dobzhansky was joined in the construction of the Modern Synthesis by the paleontologist George Gaylord Simpson, who was concerned about accommodating the seemingly discontinuous origins of major clades, as seen in the fossil record, within a Darwinian framework (Simpson, 1944). In a statement that would presage much of the career of Stephen Jay Gould, Simpson (1944) wrote: "Macro-evolution involves the rise and divergence of discontinuous groups, and it is still debatable whether it differs in kind or only in degree from micro-evolution. *If the two proved to be basically different, the innumerable studies of micro-evolution would become relatively unimportant and would have minor value in the study of evolution as a whole* [italics added]" (p. 97).

Just such a discontinuity lies at the core of Niles Eldredge and Gould's (1972) theory of punctuated equilibrium. They explained the well-established observation (dating back at least to the English paleontologist John Phillips' study of the fossils of Oxfordshire in the 1840s) that most fossil species appear abruptly in the fossil record, then disappear just as abruptly, as a fundamental discontinuity between intra-specific and inter-specific patterns. Darwin had explained these abrupt appearances as a consequence of the incompleteness of the fossil record, but Gould and Eldredge argued that the fossil record was oftentimes more than sufficient to preserve gradual changes, if they occurred. They argued that speciation occurred in rapid event, and that any change within species might be largely irrelevant to speciation itself.

Steve Stanley (1975) soon clarified some muddled thinking in the initial paper by Eldredge and Gould, observing that if speciation occurred randomly with respect to the future evolutionary needs of the lineage, selection could occur between species as well as between individuals. Stanley's arguments united the debate over the mechanism of speciation with a longstanding interest among paleontologists in the mechanisms that drove evolutionary trends. Arguments over species selection reinvigorated the controversy over whether macroevolutionary patterns were reducible to microevolution, for if species selection does occur, then Simpson's prediction is met, and large-scale evolutionary patterns may, at least in part, be disjunct from intra-specific adaptive change.

The critical issue is whether evolutionary trends over longer time scales oppose those at shorter time scales, and if so, whether this requires different processes at each time scale. Horses provide an illustration of the problem. Through the Cenozoic the size of horses increased while they also underwent a reduction in the number of toes to the single hoof present in modern *Equus*. The classic microevolutionary account of this trend would posit continuous selection for increased body size and reduction in toes (this is the picture presented in textbooks of the 1960s–1980s, and in more than a few places even today). Modern studies of the evolutionary (phylogenetic) relationships of horses demonstrate that numerous species and often genera of horses were present simultaneously during this interval. But what mechanism produced this long-term trend? To the extent that morphologic evolution within species, or

between species over short time spans, opposed the long-term evolutionary trend, the patterns would appear to support a discontinuity between micro- and macroevolution.

Through the 1980s and 1990s the dynamics of mass extinctions were incorporated as an additional domain of macroevolution. To the extent that the dynamics of mass extinctions represent a filtering process uncorrelated with selection between mass extinctions, they obviously, if rather trivially (given the number of mass extinctions), represent a macroevolutionary process that is not governed by microevolution.

Thus, the concept of macroevolution has changed over time. Filipchenko invoked distinct macroevolutionary mechanisms; Dobzhansky and Simpson recognized the reality of macroevolutionary patterns, but questioned whether they required anything other than microevolutionary explanations. By the 1970s, with Gould, Eldredge, Stanley, and other paleontologists, the emphasis shifted to question about whether macroevolutionary trends reflected selection among species rather than within them. In the past decade, however, the discussion has come full circle with advances in comparative evolutionary dynamics generating new controversy over the sources of evolutionary variation. The sources and constraints on variation are increasingly well-understood on a molecular level, and some of these new discoveries have suggested a return to a more mechanism-based macroevolution rather than the selection-based arguments of Gould and Stanley.

A point of clarification is needed before proceeding. As Arthur (2003) noted, macroevolution has been used to describe both Linnean taxa (phyla, classes, etc.) and the amount of time since two taxa diverged. Such a conflation is misleading, however, as there is no necessary connection between the magnitude of morphologic change and the time since divergence. Thus, Gingerich (1987) argued that distinctions between micro- and macroevolution reflected a conflation of rates, but he was employing a time-based definition. As in most other discussions of this topic, I will address the distinctiveness of clades rather than time since divergence. There is a more prosaic turf battle underlying this controversy, however. To population geneticists and some other microevolutionary biologists, the primacy of an experimental approach to evolution demands a continuity of process across scales. In contrast, paleontologists can rarely resolve time sufficiently to observe intra-specific evolutionary changes, and their own professional esteem may produce a bias in favor of the efficacy of macroevolutionary processes. Thus, in each case parochial concerns with disciplinary progress may blind workers to underlying evolutionary reality.

3 An Expanding Hierarchy of Selection

Some clades persist and diversify more than other clades: flowering plants (angiosperms) are far more speciose than the gnetales or other closely related sister clades. Microevolutionary processes can produce such patterns, but if speciation is often punctuated, as suggested by the fossil record, then macroevolutionary trends may result from differential orgination and extinction of species within clades (Stanley, 1979). Two different processes have been proposed to explain such differential persistence: species selection and species sorting. Indeed much of the confusion over macroevolution in the 1970s and 1980s can be traced to a failure to distinguish between these processes.

Species sorting is the differential success of species from whatever cause, and may reflect species selection, natural selection, drift, or some other process. Species selection is far more contentious, for it suggests species must have properties upon which selection can act independent of lower-level processes; in other words, emergent properties. Furthermore, for species selection to be a viable and independent evolutionary process, species must generate daughter species with sufficient fidelity to make species selection analogous to natural selection. In other words, these species-level characteristics must be heritable (Eldredge, 1989; Erwin, 2000; Gould, 2002; Gould & Loyd, 1999; Grantham, 1995).

Initially, the origin of species selection from the theory of punctuated equilibrium led to the proposal that the punctuated nature of some speciation events means that species have a discrete birth, persistence, and death, and thus constitute independent evolutionary individuals. There has been a lengthy debate over the causes of morphological stasis as well as the reality of punctuations associated with speciation, both elements that have been critical in discussions of the nature of species. In a significant recent paper, Estes and Arnold (2007) tested the relevance of six different models of phenotypic change in explaining stasis. Testing the results of the models against a compilation of phenotypic change over time scales ranging from a few to 10 million generations, they show, surprisingly, the best fit with a simple model in which populations have limited movement around a fitness optimum. If we think of these results in terms of an adaptive landscape of hills and valleys, with the hills being morphologies that are better adapted to the environmental conditions, the analysis by Estes and Arnold appears to suggest that the hills persist for very long periods of time and species jump around on them. Stabilizing selection appears, in this view, to be a very important control on evolutionary change. There are some scaling issues associated with this analysis, so that it is not clear that the results are not dependent on how the model was constructed. If Estes and Arnold are correct, many of the apparent punctuations seen in the fossil record may actually represent an adaptive response to the constrained movement of the optimum phenotype on an adaptive peak. The result does not, however, explain persistent morphological trends, and, as we will see, makes the issue of emergent, species-level characters even more significant. Today the issue of whether species constitute discrete individuals is more properly viewed as whether there are emergent characters for selection to operate on at the level of species, and it is best to focus on this issue.

The geographic range of species of Late Cretaceous marine mollusks has become the battleground for species selection. Jablonski (1987, 2005) found that bivalves and gastropods with planktotrophic development (larvae that swim and feed in the water prior to settling on the substrate) have greater dispersal ability and thus a statistically significant greater geographic range and lower extinction rates than non-planktotrophic species (those with larvae that settle without feeding). In a comparison of closely related species, Jablonski concluded that geographic range at the species level is variable and generates differential survival, and, most importantly, that geographic range is heritable, with daughter species having a similar geographic range to the parent species. The counterargument has been that geographic range in these taxa is primarily a reflection of larval ecology, a characteristic of individuals not the species, and, thus, can be explained by natural selection with no need to invoke species selection (see particularly Webb & Gaston, 2005).

Using updated data and new statistical approaches, Jablonski and Hunt (2006) showed that the effects of geographic range and larval ecology could be deconvolved. The results show that geographic range is heritable, with high statistical significance. Moreover, this result holds even within the same types of larval ecology. These results suggest that geographic range, independent of larval ecology, is a significant predictor of the range of the daughter species, and of duration, supporting the claims of a species selection model.

But is geographic range truly an emergent phenomenon, sufficient to justify the claims of species selection? Grantham (2007) recently summarized the philosophical debates over this issue, identifying four different classes of emergence (see also Bedau, 1997). The relevant class of emergence in the context of species selection on geographic range is weak emergence. The critical issue for weak emergence is whether an explanation of the higher-level, emergent property "cannot be predicted from the lower level without simulating the full suite of processes that have historically led [to the property]" (Grantham, 2007, p. 79). The emergent property is thus "causally incompressible" to lower-level properties, even if, in principle, one could describe each of the lower-level properties that generated the events leading to the emergent property. Thus, one might be able to explain geographic range as a consequence of larval dispersal, climate, and population dynamics, but because these and other factors interact in complex ways, the actual geographic range of a species cannot be predicted except by observing the actual process play out, or by fully simulating it. To the extent that geographic range is a result of such complex and non-linear processes, it is not explainable as a result of microevolutionary processes and is emergent.

What of the persistence of trends? Recall that earlier I noted that another argument for the distinct nature of macroevolution was the persistence of trends that appear to run counter to adaptive trends within species. In 2002 Simons suggested that the problem of reversal of trends was more apparent than real, reflecting a reliance upon the short-term mean fitness. If the environment is highly variable, apparent reversals of evolutionary trends (and thus an apparent disjunction between short-term and long-term selection) are actually expected if geometric mean fitness is considered. Without going into details, over longer time spans, the environmental variability means that the geometric mean fitness will often be different from the shorter-term arithmetic mean fitness. Populations and species may arise which are adapted to short-term optima, but will then disappear as the environment changes, returning to the lineage to the long-term mean. Thus, Simons argues, the apparent reversals just reflect the importance of the geometric mean fitness and not any divergence between macro- and microevolutionary processes.

While Simons' argument may explain shorter-term morphological variability, there are two problems that cast doubt upon the generality of his explanation. First, he seems to have failed to understand the duration of many of the evolutionary trends documented by the fossil record. Many of these persist through considerable environmental change, including mass extinctions and dramatic climatic shifts, making claims for the persistence of adaptive peaks questionable. Second, Simons' argument would produce punctuated, dynamic stasis, but not a long-term directional trend in morphology, and, thus, fails as a criticism of one of the more pervasive features of the macroevolutionary record.

4 Origins of Novelty

The importance of a hierarchical view of evolution is also supported by apparent discontinuities in the appearance of novel morphologies in the fossil record. One approach to this is to use the appearance of short-lived new families as an index of innovation, under the assumption that they represent relatively novel morphologies. As it happens, there is a disjunction between the appearance of new families of marine organisms, which is relatively constant through the 543 million years of the Phanerozoic, and the highly non-random and clustered appearances of morphologies with the Linnean rank of order, class, and phyla (Erwin, Valentine, & Sepkoski, 1987). Phyla and classes are clustered in the early Paleozoic, while orders predominantly first appear then and immediately after the end-Permian mass extinction. Jablonski's analysis of ordinal appearance patterns in the post-Paleozoic (after 252 mya) shows that well-skeletonized orders are significantly more likely to appear in near-shore habitats rather than off-shore habitats (80% vs. 40% for the control group of poorly skeletonized orders; Jablonski, 2005). In contrast, the first appearances of families and genera of echinoids, crinoids, and bryozoa (all well-preserved clades) are consistent with the environmental distribution of each clade. The reasons behind the apparent discontinuity of high-level originations is unclear, in particular whether it reflects a greater likelihood of the genetic and developmental changes appearing in near-shore habitats, or a greater probability of success (and thus preservation) (see discussion in Jablonski, 2005). In either case, the critical point is the disjunction between patterns at higher and lower taxonomic levels, which suggests a similar discontinuity in process.

Studies in which Linnean ranks are employed as an index of morphological uniqueness or innovation have been criticized for a number of reasons: the taxonomic ranks may not be strictly monophyletic, or represent a single clade; and ranks are not equivalent across taxa, so that a family of bivalves is not necessarily similar to a family of trilobites. True, but irrelevant. The use of taxonomic ranks is a shorthand for morphologic disparity: their morphologic distinctiveness. A large number of quantitative morphmetric analyses have convincingly established that the morphologic distinctiveness of these clades can be established independent of Linnean categories, and that major clades are most often morphologically very distinct during their initial diversification (reviewed by Erwin, 2007). In other words, the morphological disparity of high taxa is *not* a consequence of subsequent diversification. Even more curiously, in many clades of marine invertebrates, the increase in morphological disparity occurs *before* an increase in taxonomic diversity. These are not patterns expected from analysis of microevolution, although whether it requires a purely macroevolutionary process remains to be established. When these results are combined with new insights from the comparative study of animal development across different clades ("evo-devo") and the nature of evolutionary novelties, they do pose a challenge to microevolution as a sufficient explanation of macroevolutionary pattern. Indeed, the most substantive current challenge to the Modern Synthesis comes not from hierarchical expansion of selection to multiple levels, but from new insights into the developmental basis of evolutionary novelties, as discussed in section 6.

5 Mass Extinctions

Both the end-Cretaceous (65.6 mya) and the end-Permian (252.4 mya) mass extinctions were catastrophic, occurring in just a few hundred thousand years or less and affecting virtually every ecosystem (see Erwin, 2006b). It is not the intensity of these episodes alone that has raised concerns about their macroevolutionary role, but rather the possibility that factors favoring survival during these events may have differed from those during "background" times between these crises, adding, as Gould (2002, p. 1330) termed it, a "third tier" to evolutionary processes in addition to microevolution and macroevolution.

One argument against the uniqueness of mass extinctions as an evolutionary process is that they simply represent the end-members of a continuum of extinction intensities though the Phanerozoic. While it is certainly true that one can plot a histogram such that the major mass extinctions appear to represent such end-members, this says nothing about underlying process. The unique nature of mass extinctions stems from the rapidity of at least some of the events, the disappearance of major clades (e.g., blastoids and trilobites during the end-Permian, ammonoids and rudist bivalves during the end-Cretaceous) and ecosystems (e.g., reefs), and most notably the evidence for differences in selectivity. In contrast to most intervals, the geographic range of genera (rather than species) appears to be an excellent predictor of survival for a wide range of marine groups during each of the five major mass extinctions (Jablonski, 2005). Thus, even well-established, dominant clades were at risk of significant reductions in diversity, or extinction, during these events. With the possible exception of trilobites during the end-Permian event (already reduced to insignificance), it is difficult to argue that many of the major marine clades that disappeared were on the way out prior to the onset of the mass extinction. These effects suggest that mass extinction represents both a quantitatively and a qualitatively different phenomenon than microevolutionary evolution during background times. As they sort and sift through existing clades, mass extinctions have often removed ecologically dominant groups and led to the pervasive reorganization of ecosystems during the post-extinction biotic recoveries.

6 Is Evolution Uniformitarian?

One of the fundamental principles of geology is *uniformitarianism*, the idea that the present is the key to the past. Established by geologists James Hutton and Charles Lyell in the late 1700s and early 1800s, this concept assumes that the rates and mechanisms of geological processes operating today can be used to explain the patterns seen in the geological record. Thus, evidence associated with glaciers today, such as pebbles with parallel scratch marks and unsorted till, is a good indicator of past glacial activity. One problem with uniformitarianism is that it breaks down when the time span over which events occur is longer than the historical record. Consequently, geologists had a long debate before accepting that craters on Earth could be produced by impacts of extra-terrestrial objects as well as by volcanism, because we have not (yet!) had a major impact in recorded history. Despite this, there were sound reasons

for accepting uniformitarianism as a guiding principle: Hutton and particularly Lyell were contending with other geologists who, inspired by the Bible, interpreted geological evidence as documenting a series of catastrophes. So, the development of geology as a science was critically dependent upon uniformitarianism.

Evolutionary theorists have paid far less attention to this philosophical issue than geologists, but I believe uniformitarianism has been, albeit implicitly, just as much a part of evolution as it has of geology, and for a very similar historical reason. Evolutionary uniformitarianism is the assumption that the rates, mechanisms, and processes that can be observed experimentally today are sufficient to explain the sweep of evolutionary patterns through geologic time. There is no necessary reason why evolutionary uniformitarianism should be correct; it is simply an implicit assumption of most evolutionary biologists. It could be the case that rates of change were more rapid, or slower, in the past or that different mechanisms of genetic change operated. Evolutionary biologists have tended to reject such explanations because they are difficult to test, not because we can demonstrate from first principles that they are untrue.

Evolutionary uniformitarianism was adopted for much the same reason that geologists invoked geological uniformitarianism a century earlier. The crisis in evolutionary biology during the latter part of the 1800s involved strong disagreements over the causes of evolutionary change. Macromutationist views were widespread (Bowler, 1992), and orthogenesists such as Henry Fairfield Osborn at the American Museum of Natural History argued for internally driven evolutionary trends. To counteract these views, I believe that geneticists such as Thomas Hunt Morgan, and later the architects of the Modern Synthesis, implicitly adopted a uniformitarian approach to stymie their opponents. Population genetics, like statistical physics, lacks an historical dimension. In other words, the properties of the objects of study do not change over time. So, to the extent that it is the core of the Modern Synthesis, biological uniformitarianism has been an implicit assumption. Curiously, Gould, who one might have expected to be aware of this conceptual constraint, only touches on it obliquely in three brief passages in *The Structure of Evolutionary Theory* (2002). I suspect that his emphasis on the hierarchical expansion blinded him to the issue of uniformitarianism in evolution.

There are two different ways in which evolutionary uniformitarianism could be incorrect. First, just as geologists initially did not recognize that they needed to enlarge the scope of the processes they considered to include the impact of extra-terrestrial objects, it could be that the temporal restriction of modern biology to the past 150 years or so is too limited to provide experience with the full range of evolutionary dynamics. Merely by enlarging our scope, however, we can re-establish evolutionary uniformitarianism. We have already done this, implicitly, in considering rates of evolutionary change as documented by comparative studies rather than experimental approaches (Erwin, 2006b; Gingerich, 1993; Hendry & Kinnison, 1999; Roopnarine, 2003). The hierarchical expansion of levels of selection also corresponds to an enlargement of the scope of evolutionary uniformitarianism, rather than a fundamental challenge to the principle. A difficulty is the tension that exists between experimental and comparative approaches to evolution, for there may exist disjunctions between changes documented through comparative approaches and those amenable to experimental investigation. One such example will be discussed in detail below.

The second alternative is that evolutionary uniformitarianism is deeply misleading, and that the nature of evolutionary change has itself evolved over time. There is now considerable evidence that this is true for the major evolutionary transitions such as the origin of eukaryotic cells (those with DNA packaged in a nucleus) and the origin of multicellularity. Furthermore, evidence from the structure of gene regulatory networks in animals also indicates that the nature of the variation available for selection to act upon has changed over time. These are two examples of how the evolutionary processes has itself evolved over time, and I believe that this may impose another way in which macroevolutionary patterns are not reducible to microevolutionary processes, at least as they are currently defined by microevolutionists.

In *The Major Transitions in Evolution* (1995), Maynard Smith and Szathmáry developed the argument that increases in biological complexity reflect eight major evolutionary transitions, each involving new ways of packaging and transmitting information between generations, and that these transitions permitted increases in biological complexity. Their book raised some fascinating issues for evolutionary biologists and has sparked considerable discussion and research. In each of the transitions they discussed, formerly individually reproducing units became subunits of a larger body, which also replicated. This created the potential for conflict between replication at different levels: the replication of cell lineages may, for example, be in conflict with the need of the whole organism to grow before replicating. To alleviate such potential conflict, formerly freely replicating entities within some larger whole may lose the potential for independent reproduction. For example, the origin of eukaryotes involved the establishment (via symbiosis) of the mitochondria and chloroplast as energy-producing organelles within a eukaryotic cell. Although DNA still persists in mitochondria, many genes have been transferred to the nucleus of the host cell, so that today it is impossible for mitochondria to live on their own. This loss of independence allowed a greater division of labor and increased specialization. Such a division of labor is a characteristic feature of these evolutionary transitions.

Through each of these transitions the nature of the hierarchical structure of life has itself evolved, a macroevolutionary event of the first order, and created new and emergent evolutionary structures. While these events can be studied comparatively, they are difficult or impossible to directly study experimentally, because they have changed the rules of the game, and changed the nature of the variability upon which selection can act.

A second and related area of non-uniform evolutionary processes has been revealed by detailed comparative studies of gene regulatory networks. Over the past two decades biologists have developed new tools to explore the molecular and genetic basis of development. By examining the behavior of the same genes in different organisms, they can also examine how, for example, the genes controlling limb development are similar and different between flies and mice. To their considerable surprise, they have discovered that most of the basic toolkit for producing appendages—eyes, gut, brain, and many other body parts—is shared across all bilaterian animals (those with bilaterial symmetry, including various worms, mollusks, arthropods, and vertebrates). Of even greater surprise has been the recent recognition that many of these genes were also present in animals as simple as sea anemones, the Cnidaria. The diversification of major morphological groups, often called body plans, apparently lagged the appearance of the developmental tools required to produce the wide

diversity of animal forms. Why this should be so is unknown, although it almost certainly relates to changes in ecological relationships and the nature of the environment, particularly increases in oxygen levels in the ocean.

This work on evolutionary developmental biology has revealed a hierarchical structure to these conserved regulatory networks responsible for the patterning of major aspects of the body plan. The best-studied example involves the development of the gut (strictly, the endomesoderm) of echinoderms. Experimental studies by Eric Davidson's lab at CalTech have revealed the complete wiring diagram of genetic interactions, including the genes involved, the various *cis*-reglatory control regions, and their logical relationships (Davidson, 2006). Comparison of two evolutionarily distant clades, such as sea urchins and starfish, which split some 500 million years ago, shows that there are about five genes whose regulatory interactions have been completely conserved. Even more curiously, when the elements of this regulatory "kernel" are experimentally perturbed, no viable larvae are produced. Perturbation experiments of other parts of this network may still produce larvae, and there have clearly been many evolutionary changes of other elements of the regulatory network. From this, we conclude that the early evolution of animals was associated with the formation of critical regulatory interactions that were responsible for particular parts of the body, including the gut and heart. After the kernels were formed, experimental evidence shows that they are essentially immune from subsequent modification. Other evolutionary changes occurred within the networks (hearts haven't stopped evolving), but the changes were forced to other parts of the network. Because these kernels are so refractory to subsequent change, selection is essentially binary: keep what you have, or you get nothing at all (Davidson & Erwin, 2006).

Consideration of any complex organism, indeed any organism at all, suggests that this hierarchical ordering of the evolutionary processes through time has had a pervasive impact on evolution. These events are not reducible to microevolution, nor explained by the Modern Synthesis, because they require a historical theory of evolution, one that encompasses an understanding of how evolution itself changes the evolutionary process.

7 Conclusions

Macroevolution and microevolution address different aspects of the evolution of life, and a complete evolutionary theory necessarily must encompass both the patterns and the processes at each level. I have argued here that macroevolutionary processes are distinct from those of microevolution in several different ways. First, paleontologists have developed good evidence for the occurrence of species selection, as distinct from species sorting, in the geographic range of Cretaceous mollusks. Convincing demonstrations have been provided that geographic range is a weakly emergent, heritable trait that cannot be reduced to microevolution. It seems likely that other examples await discovery. Second, patterns of selectivity during mass extinctions often differ from those during background extinctions, suggesting that mass extinctions represent a distinct level of selection. Finally, the origin of morphological novelties documented by the fossil record indicates a disjunction between the processes responsible for the origin of "higher taxa" and those responsible for most genera and

families (suggesting that different processes exist even within macroevolution). I have also suggested that our traditional views of both microevolution and macroevolution have been hampered by an unduly uniformitarian view of the evolutionary processes. At least some of the most emergent phenomena in the history of life appear to have changed the processes of evolution by changing the nature of what selection can act upon, by constructing new and more complex evolutionary entities, and by subsuming previously independent entities to manage conflict.

Postscript: Counterpoint

Dietrich addresses the distinction between process and outcome in clarifying whether there are unique processes associated with macroevolutionary patterns. This issue is indeed one of the critical questions that must be addressed. One where I think the issue is much less settled than Dietrich appears to believe concerns developmentally significant genes, which he argues "do not constitute a distinct and uniquely macroevolutionary causal process." As I discuss in my paper, I view this issue as far less settled. What is strikingly absent from virtually all microevolutionary thought, and even from most macroevolutionary thought, is a sense of history, of the impact of evolutionary changes on the range of variation that is possible, and of how that range of variation has itself changed over time. These changes in the range of variation over time have limited some opportunities (by foreclosing the viability of most changes in the genetic code, for example) yet created others. Dietrich focuses on particular mutation identified by comparative developmental biology, but the useful information actually comes from the network of developmental interactions, which are now being worked out. And the lesson from such studies is that one of the paradigmatic examples of macroevolution—the establishment of animal body plans during a relatively short interval of the Cambrian Period—in part reflects the establishment and subsequent conservation of particular components of developmental gene regulatory networks.

In his conclusion, Dietrich argues not that macroevolutionary processes do not occur, but that, for species selection at least, they are rare and "of minor evolutionary consequence." In this paper I have argued for a plausible role for a larger range of macroevolutionary processes, but I also suggest that Dietrich's emphasis on rarity is inappropriate. Mass extinctions have (fortunately) been rare as well over the past 500 million years. But it is abundantly evident that these rare and infrequent events have had a profound and continuing impact on the history of life. Dietrich is correct that a greater understanding of relative frequency is important, but frequency is not necessarily a proxy for generative impact, which must be assessed independently.

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PART VI

DOES EVOLUTIONARY DEVELOPMENTAL BIOLOGY OFFER A SIGNIFICANT CHALLENGE TO THE NEO-DARWINIAN PARADIGM?

Introduction

This book offers contemporary debates in philosophy of biology; thus, we have included papers pertaining to the new and burgeoning science of evolutionary developmental biology (evo-devo). Evo-devo's basic claim is broadly threefold:

(1) We have discovered that many genes produce—or can produce, when we manipulate them—a variety of different traits in different organisms, depending on which genes are being expressed or not (what is colloquially called the switching on and off of genes) in the very development of the organism. To take a simple and common example, genes in the larvae of fruit-flies can be moved around or replaced, and doing so causes eyes to grow where antennae should be, legs where heads should be, and other monstrosities in these flies. Also, such monstrosities are witnessed naturally without manipulation. Research on the genetic development of roundworms reveals similar phenotypic adjustments based upon genetic manipulation during development (e.g., Callaerts, Halder, & Gehring, 1997; Duncan, Burgess, & Duncan, 1998). This is the significant *developmental* part of evo-devo.

(2) Further, for some time now, we have been mapping genes and manipulating the genetic toolkit that controls the phenotypic traits of animals like fruit-flies, mice, worms, and others. Through that work, we have discovered that all animals are built out of essentially the same genes. For example, the Pax6 gene seems to be responsible for eye development in both mice and fruit flies (Quiring, Walldorf, Kloter, & Gehring, 1994). Also, the melanocortin 1-receptor (MC1R) gene seems to be responsible for hair color in mice and humans, as well as plumage color in certain birds (Takeuchi, Suzuki, Yabuuchi, & Takahashi, 1996; Valverde, Healy, Jackson, Rees, & Thody, 1995). And there are many other examples (see Minelli, 2003).

(3) Here is the fascinating claim made by evo-devotees: evolution is not so much the result of wholesale genetic variation in terms of mutation as it is a matter of changing *when* and *where* genetic switches will be turned on and off in the development of an organism. Thus, according to many evo-devotees, the various species we see around us today actually are the result of genetic switches being turned on or off at various points in the development of an organism throughout evolutionary history. If true, this is a fascinating discovery—although, the development of organisms has been a significant area of research since Darwin's time; and Darwin (1859/1999) himself claims in the *Origin* that "characters derived from the embryo should be of equal importance with those derived from the adult, for a natural classification of course includes all ages" (p. 342).

There is now ongoing debate as to the extent to which evo-devo might challenge or complement various standard evolutionary principles since, according to Sean Carroll, Benjamin Prud'homme, and Nicolas Gompel in a fairly recent issue of *Scientific American*, "evolutionary changes to anatomy, particularly those involving pleiotropic genes, are more likely to happen via changes to gene enhancers than to the genes themselves" (p. 67). In the first paper included in this part, Manfred Laubichler wants to argue that, to a certain extent, evo-devo does offer a significant challenge to the neo-Darwinian paradigm that evolution is mostly the result of wholesale genetic variation in terms of mutation. Based upon recent evidence, Laubichler expands an important tenet of evo-devo that adaptive mutations which affect phenotypic characteristics are more likely to occur in the *cis*-regulatory regions of genes than in the regions where protein-coding occurs.

In his paper included as the second one in this part, although Alessandro Minelli wants to argue that evo-devo does not offer a significant challenge to the neo-Darwinian paradigm, he also maintains that "evo-devo is not simply developmental biology grafted onto evolutionary biology; rather, it deserves to be acknowledged as a research field of its own, with a specific agenda and a specific conceptual endowment." Through a discussion of the concept of *constraint* in evolutionary biology, complete with a few examples, Minelli ultimately claims that evo-devo "provides its unique contribution to understanding the evolutionary process by a description and analysis of *developmental* [emphasis ours] constraint and its elements."

Even as early as 1954, Gavin de Beer could maintain that it "has become increasingly clear from research in embryology that the processes whereby the structures are formed are as important as the structures themselves from the point of view of evolutionary morphology and homology" (p. 136). Research in evo-devo seems to be underscoring de Beer's point.

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CHAPTER ELEVEN

Evolutionary Developmental Biology Offers a Significant Challenge to the Neo-Darwinian Paradigm

Manfred D. Laubichler

In this paper, I note that in evaluating present-day evo-devo the question for us is not only whether evo-devo is revolutionary in some sense, but also whether sometimes a "revolutionary" position in science can actually resemble more a "counter-revolution." I argue that the relationship between evo-devo and the neo-Darwinian paradigm can be interpreted exactly like that, and that the revolutionary nature of evo-devo lies precisely in its return to a more inclusive conception of phenotypic evolution, one that more closely resembles the conceptual framework of Darwin and the first few generations of evolutionists than the more narrowly focused interpretation of the Modern Synthesis. However, I also argue that evo-devo is by no means "reactionary" as it approaches these more traditional conceptual problems with all the methodological, technological, and empirical advances of the last six decades of biological research.

1 Introduction

Scientific revolutions, those rare episodes of paradigm shifts in science, are largely conceptual in nature (Kuhn, 1962). Therefore, any claim that evolutionary developmental biology (evo-devo) offers a significant *conceptual* challenge to the neo-Darwinian paradigm in evolutionary biology has, to a certain degree, revolutionary implications. On the surface this assertion might seem preposterous. Surely, present-day evo-devo is not transformative in the same way as Einstein's theory of relativity or Darwin's first formulation of evolution by means of natural selection! At most, one could argue that we are dealing with a small-scale revolution, an incremental change in the conceptual framework of evolutionary biology. Or, as some have argued, is evo-devo actually

nothing but the completion of the Modern Synthesis, finally bringing into the fold the last renegade disciplines, those that, for whatever reason, did not participate in the transformation of the 1940s (Hamburger, 1980; Ruse, 2006)? Alternatively, if we accept Alessandro Minelli's claim in this volume that the core ideas of evo-devo are actually not much different from those held by the founders of the Modern Synthesis, then the whole discussion about "revolution" or "challenge" would indeed be pointless as evo-devo certainly would be squarely located within neo-Darwinian paradigm.

So why I am arguing here that evo-devo represents a paradigm shift? My arguments for the revolutionary nature of evo-devo are rooted partly in an understanding of the scientific problems and explanatory structure of what I consider to be the genuine core of present-day evo-devo—the patterns and processes of phenotypic evolution and especially the problem of innovation—and partly in a careful reading of the history of biology paired with a slightly idiosyncratic interpretation of the nature of scientific revolutions. By and large we look at the history of science as progressive in the sense that new data, concepts, and theories have gradually added to our understanding of nature. To be sure, we sometimes followed dead ends (remember the ether or phlogiston) and sometimes things have changed rather dramatically (those rare revolutionary events), but, so this view, in general science has pursued a more or less clear direction of continuous progress. While no one can question the quantitative progression of science—we have been adding new knowledge at an ever-increasing rate—this linear (or, rather, exponential) model of growth does not necessarily apply to conceptual insights or the frequency and even directionality of scientific revolutions. In evaluating present-day evo-devo, the question for us is thus not only whether evo-devo is revolutionary in some sense, but also whether sometimes a "revolutionary" position in science can actually resemble more a "counter-revolution." Here I will argue that the relationship between evo-devo and the neo-Darwinian paradigm can be interpreted exactly like that and that the revolutionary nature of evo-devo lies precisely in its return to a more inclusive conception of phenotypic evolution, one that more closely resembles the conceptual framework of Darwin and the first few generations of evolutionists than the more narrowly focused interpretation of the Modern Synthesis. But evo-devo is by no means "reactionary" as it approaches these more traditional conceptual problems with all the methodological, technological, and empirical advances of the last six decades of biological research.

The "counter-revolutionary" nature of evo-devo lies in its conceptual emphasis of the phenotype as both the *explanandum* and the *explanans* of evolutionary biology. This phenotypic conception of evolution is different from the traditional gene-centered neo-Darwinian paradigm, where the phenotype is ultimately seen as an epiphenomenon of what are considered to be the more fundamental dynamics of genes and populations. In the middle of the twentieth century the Modern Synthesis and its conceptual innovations were a major theoretical breakthrough. It was no small accomplishment to combine the mathematical abstractions of population genetics, which described the dynamics of alleles within populations under a variety of conditions, with empirical studies of variation and speciation. This approach allowed us to study certain elements of the evolutionary process from both theoretical/mathematical and empirical perspectives and it firmly established evolutionary biology as an exact science (Mayr, 1982; Mayr & Provine, 1980). The molecular revolution in biology, which followed at the heels of the Modern Synthesis, further supported the basic

assumptions of mid-twentieth-century evolutionary biology as it provided us with a molecular interpretation of the central concepts of population genetics, such as gene, allele, and mutation. But what both the Modern Synthesis and its later molecular interpretation missed was a mechanistic understanding of phenotypic evolution. This would have required a clear understanding of development, i.e., how a particular genotype actually causes a particular phenotype. Most of twentieth-century evolutionary biology considered development as a "black box" or as something that did not really affect the underlying dynamics of genes and populations. In the context of genetic or molecular models of evolution, development was hidden in the structure of the genotype–phenotype map, a formal abstraction that translates abstract genotype values into phenotype values, or, in reverse, allows us to ascribe fitness values measured for phenotypes to underlying genotypes.

As I have said before, these assumptions and conceptual abstractions have been the basis of evolutionary biology for more than half a century and have led to many important insights. But they did not provide us with a mechanistic understanding of phenotypic evolution, which is arguably the main scientific problem of evo-devo (Laubichler, 2007). Here I will review, in form of a brief summary, several specific research programs and types of explanations that are part of modern evo-devo and that highlight its specific focus as a mechanistic theory of phenotypic evolution, and briefly sketch how evo-devo's emphasis on a mechanistic explanation of phenotypic evolution can help to resolve this issue. But before we discuss the specific elements of present-day evo-devo, let me briefly place these developments in their historical context.

2 A Brief History of Developmental Explanations of Phenotypic Evolution

The starting point of any theory of phenotypic evolution is the enormous diversity of extant and extinct organisms. Estimates of the total number of species (living and extinct) run in the tens of millions or even more. What is astonishing, however, and most in need of an explanation, is the fact that this diversity is highly structured. All multicellular animals and plants can be grouped into a small number of distinct phyla, each characterized by its unique *Bauplan* or body plan, and, furthermore, these body plans themselves share a number of fundamental architectural principles of organismal design, such as modular elements, symmetries, or a hierarchical organization. For at least two centuries, development has been featured prominently in explanations of these unique features of organismal variation (Amundson, 2005; Laubichler, 2007). Among the many nineteenth-century proposals connected with embryological explanations of the hierarchical nature of phenotypic diversity we find von Baer's laws, which state that early stages in development will more closely resemble each other than later stages (thus accounting for the similarity of all those body plan features that are laid down early in development), or various versions of recapitulation theory, which present a more linear model by assuming that more complex organisms pass through the stages of less complex ones and novel features thus represent a form of terminal addition (with some debate as to whether the earlier stages represent adult or embryological stages of less complex organisms). And even for Darwin, development was an important part of the argument for this theory of evolution,

especially with regard to explaining patterns of phenotypic variation (Richards, 1992). But, lacking of a particulate theory of inheritance based on "genes" or distinct "factors," the three elements of what used to called "generation"—development, inheritance, and evolution—were not yet separated into distinct processes; thus, to put it into modern terms, the organism, or the phenotype, was still the focus of study and the principle of explanation within biology (even the life force of vitalistic theories was tied to organisms as it was thought that their specific properties, such as regulation or purposefulness, could only be explained by such a distinct organic force) (Laubichler & Maienschein, 2007a, 2007b).

The turn of the twentieth century was characterized by several conceptual shifts and the emergence of distinct experimental research programs connected to the problem of phenotypic evolution. As a result, what had been a single unified problem of phenotypic diversity and evolution split into three distinct areas of investigation—inheritance, development, and evolution (Allen, 1975; Laubichler & Maienschein, 2007a). Furthermore, in part as an antithesis to what some perceived as overly reductionist tendencies within experimental biology, a renewed interest in form led to several new proposals, ranging from D'Arcy Thompson's conception of "Growth and Form" to discussions within paleontology and morphology that are now considered part of the revival of "idealistic morphology" (Meyer, 1926; Naef, 1919; Thompson, 1917; Troll, 1928). Many of these proposals were attempts to fill the void left by what Peter Bowler (1983) has called the eclipse of Darwinism, referring to the dissatisfaction with natural selection as the main explanatory mechanism of organic evolution. The argument in this context was that additional "internal" forces are needed to understand the observed patterns of phenotypic evolution. Some of these proposals focused on how the environment could influence hereditary material; others emphasized the apparently directional patterns of phenotypic transformations (especially within the fossil record) and argued that such orthogenetic series are the product of internal mechanisms. And among those, developmental processes ranked high.

The scientific problems that many of these suggestions attempted to address were, of course, the old questions of the origin and patterns of phenotypic variation. A number of the solutions proposed in the course of the last century to address these problems thus represent earlier attempts of what we now call evo-devo. Several of these twentieth-century research agendas involved a broader conception of the gene than the one that dominated evolutionary biology during that period. For instance, Richard Goldschmidt and Alfred Kühn and their schools of developmental physiological genetics emphasized the functional role of genes in development (Laubichler & Maienschein, 2007b; Laubichler & Rheinberger, 2004), proposing several mechanistic models of how developmental systems produce phenotypes and how changes within these systems contribute to corresponding changes in phenotypes. This differs from the Modern Synthesis view that evolutionary processes are driven largely by (random) genetic changes, on the one hand, and by functional interactions of organisms with their environment, on the other hand, and which considered development as a black box that does not alter fundamental dynamics of natural selection. Developmental physiological genetics of the first half of the twentieth century is thus a legitimate predecessor of evo-devo.

In addition, the views of the Modern Synthesis on the agents of evolutionary change, especially in its so-called "hardened" form, were soon challenged by individuals trained

in different fields such as morphology, paleontology, comparative embryology, and developmental biology. What were first individual voices of dissent would, by the late 1970s and early 1980s, be organized into a movement that would soon be known as evolutionary developmental biology, or evo-devo (Hall, 1998). Initially each group objected to specific perceived shortcomings of the Modern Synthesis. Paleontologists argued that the implicit gradualism of evolutionary models does not correspond to observed patterns of the fossil record and proposed a whole range of macroevolutionary principles, some, like heterochrony, having a developmental basis. Morphologists complained that the gene-centered perspective of the Modern Synthesis does not explain the structured hierarchy of forms and the nested nature of homologies. They also provided alternative theories of morphological evolution that included developmental principles, such as burden or developmental constraints, in explaining the conservation of specific characters. Developmental biologists also objected to the gene-centered view, arguing that the mechanisms of morphogenesis need to be an important part of any explanation of form. During this first period, what is now known as evo-devo was represented by a variety of theoretical positions and concepts aimed to correct or expand the Modern Synthesis (Laubichler & Maienschein, 2007b; Love & Raff, 2003).

But it was the field of developmental genetics during the 1990s that, building on earlier insights, provided evo-devo with a new conceptual foundation that had the potential to address many of the open questions within a coherent framework and thus challenged the Modern Synthesis assumptions about phenotypic evolution. This new conceptual framework was built on the discoveries of (1) the conservation of Hox genes and other developmentally relevant transcription factors; (2) the structured regulatory logic of developmental systems, including the complete description of the first set of regulatory gene networks; and (3) the limited number of structural genes in higher organisms, which proved to be critical in this context. The new paradigm emphasizes that in order to understand phenotypic evolution, one first has to understand the genetic toolkit of development and the nature of gene regulation in development. These two developmental concepts then provide the basis for the study of variational properties of phenotypes, which are after all the raw material for natural selection to act upon. And, as I will argue below, the current incarnation of evo-devo actually has a good change of accomplishing a conceptual solution to these longstanding problems

3 Research Questions of Evo-Devo

Presently evo-devo investigates a whole range of specific questions (Laubichler, 2005; Laubichler, 2007; Müller, 2005, 2007; Wagner, Chiu, & Laubichler, 2000) that all focus on explaining the patterns and processes of phenotypic evolution. In this context, evo-devo represents a reorientation of evolutionary biology. The main difference is that in the context of evo-devo, phenotypes are seen no longer merely as the passive by-products of evolutionary processes that are mainly driven by internal genetic changes and external evolutionary forces, such as natural selection or genetic drift, but rather as the locus of integration of a whole range of mechanisms, from molecular and developmental to physiological and environmental. Consequently, the patterns and

processes of phenotypic change result from a combination of all these diverse causal mechanisms from a variety of organizational (from genes to the environment) and temporal (developmental, life history, and evolution) scales.

For evo-devo to be successful in the long run it has to overcome several obstacles, some of a deep conceptual nature, others more a consequence of historically entrenched habits of scientific practice. Conceptually, the different frameworks of organism-based experimental developmental biology and population-based evolutionary biology are arguably the largest stumbling block to an evo-devo synthesis. This can best be seen by the ways both traditions understand the effects of a gene. In developmental genetics an effect of a gene is defined by its role in such processes as differentiation; the main emphasis therefore is on the regulation of gene expression. Evolutionary biology, on the other hand, emphasizes the role of genetic variation within and between populations, and its model of evolutionary change uses the metric of fitness differences to evaluate the functional importance of genes.

Thus, as long as both disciplines are still limited by their respective conceptual positions, it is difficult to accomplish an integrative theory of phenotypic evolution. For evo-devo to be successful it will have to develop conceptual innovations that overcome these incompatible views. One such conceptual innovation that can help us overcome this conceptual divide is the notion of regulatory evolution, which provides a mechanistic understanding of phenotypic diversity from a developmental perspective. It also has the potential to be incorporated into models of evolutionary change, once the genetic variation governing these changes in regulatory networks has been identified. The notion of regulatory evolution thus shows how a combination of conceptual and empirical advances has led to a change in the conceptual framework of explanations of phenotypic evolution. But the notion of regulatory evolution is just one of a set of concepts and unifying themes that together make up the proposed integration of evo-devo. Others include such notions as constraint, heterochrony, modularity, hierarchy, homology, evolvability, emergence, plasticity, innovation, robustness, regulatory networks, among several others.

4 Unifying Themes of the Conceptual Basis of Evo-Devo

The conceptual basis of evo-devo is diverse. Among the many concepts that either originated within evo-devo or have been transformed by evo-devo perspectives I want to briefly sketch four that are most closely connected to its revolutionary potential. These are regulation (including regulatory evolution and regulatory gene networks), modularity, plasticity, and innovation. All of these are reflective of an organismal perspective on development and evolution. There are, of course, many additional concepts that are part of the theoretical framework of evo-devo. Some, like heterochrony, developmental constraints, burden, or hierarchy, have been part of the first wave of evo-devo proposals. Others, such as robustness, evolvability, or homology, are the subject of intense discussions today, but within the space here we will limit their discussion within the context of our four examples.

Regulation has long been recognized as a fundamental property of organisms as well as other biological and social systems. Regulation is thus a property of complex

systems and it enables them to maintain their structural cohesiveness in light of a variable environment. What is of special interest for us here is that development, or the origin of organismal form, is also a regulative process. In today's evo-devo, regulation is central to our understanding of both the development and the evolution of organisms. It is an essential part of the emerging mechanistic theory of phenotypic evolution, as gene regulatory networks provide us with a clear explanation of the problem of differentiation. Different cell types are ultimately characterized by the expression of different sets of genes, which are, in turn a consequence of differential gene regulation. Uncovering the complex molecular machinery that regulates these expression patterns has been one of the major accomplishments of developmental genetics during the past decades.

In addition, it has also become clear not only that regulatory networks act within the genome, but also that different levels of phenotypic organization have their own regulatory control mechanisms that guarantee their stability in light of the variation of its parts. As we have found out more about the molecular details of development, it has become clear (1) that a large degree of conservation exists among the different elements of developmental systems; (2) that there are many cases in which we find phenotypic stability despite substantial changes among the elements of the developmental systems, in other words that the same phenotype can be realized through the action of different molecular factors; and (3) that these regulatory networks have their own conserved architectural structures that show different degrees of conservation. There are some elements that are extremely conserved and whose action is essential for any normal development ("kernels"), while others are more variable and can also be deployed in a variety of different context ("plug-ins" and "switches") (E. Davidson, 2006a; E. Davidson & Erwin, 2006).

This last property of regulatory (gene) networks also helps us understand the developmental mechanisms of phenotypic evolution, which are now understood as largely a consequence of regulatory evolution. One immediate consequence of the various genomics projects has been the recognition that there are fewer genetic differences between different species (such as *Drosophila* and human) than originally thought. These results imply that phenotypic differences and phenotypic evolution are more the result of changes in the expression patterns of genes than they are of novel genes. These findings undermine any simple causal model of phenotypic evolution that connects novel phenotypic characters with new genes as it is now clear that phenotypic differences are a consequence of differences in the regulation of the developmental systems. The architecture of these regulatory (gene) networks is also an instance of modularity, another general principle of biological organization that is also a prominent unifying theme within evo-devo.

Modularity is a fundamental characteristic of all biological organizations (Callebaut & Rasskin-Gutman, 2005; Schlosser & Wagner, 2005). We find modules on all levels of organization, from the genome to cellular, anatomical, and behavioral structures. In addition to structural modules there are dynamic modules such as specific developmental processes that remain stable across a wide range of different organisms, for example certain elements of regulatory networks, signaling cascades or processes of induction. Modularity is also closely connected to the concept of homology and the basic principles of phenotypic organization. Several major research questions of evo-devo relevant to the form and function problem are directly connected to issues

of modularity. Most models of adaptation or optimization of individual organismal character depend on what Herbert Simon (1995, 2002) called "near-decomposability," in other words on the existence of both phenotypic and corresponding genotypic modules. Genotypic modules are characterized by reduced levels of pleiotropy, i.e., the genes connected to the particular phenotype under selection should not also affect too many additional phenotypic features as this would greatly limit the ability of natural selection to optimize any given function. Expressed in terms of the genotype–phenotype map, this implies that due to the modular architecture of this map it is possible to decompose it into several smaller independent maps of lower dimension that each can then be optimized independently by natural selection. As such a structure is itself an adaptive feature of organismal design—it does, after all, allow for selective optimization of traits—it is reasonable to assume that the modular structure of the genotype–phenotype map has itself been the product of evolutionary change. We have already mentioned that the genotype–phenotype map is a formal representation of the developmental system. Therefore, the observed modularity of organismal design is a consequence of regulatory evolution of the developmental system.

Phenotypic plasticity is the capacity of a single genotype to generate different phenotypes in response to variable environmental conditions (West-Eberhard, 2003). Plasticity is thus a major functional response of organisms to cope with their unpredictable environment. As such, it is another case for the regulatory potential of organisms. Research into the mechanisms of phenotypic plasticity has recently emerged as a major theme within evo-devo, especially in light of recent efforts to add an ecological dimension to its portfolio.

One organismal property that displays high degrees of phenotypic plasticity is behavior (Laubichler & Gadau, 2009; Page & Amdam, 2007). Studying behavior, and especially social behavior, from an evo-devo perspective has become an active area of research during the last few years. Social systems are interesting as they are built from linear and non-linear behavioral interactions among individual organisms involving communication (signaling) and stimulus–response systems. They develop at the level of the individual through ontogenetic processes, and at the social system, or superorganismal, level through growth and differentiation of the social group. Social systems, such as those represented by social insects, are also the product of evolution, and, indeed, the emergence of social behavior is considered one of the key innovations in the history of life. We also know that there are allelic substitutions at variable gene loci that affect developmental events with downstream effects on interactions and response systems at the level of individuals, resulting in adaptive social patterns and emergent colony-level traits. However, in order to differentially affect behavior, allelic differences must be translated into differential expression or function of peptides and proteins that themselves are parts of signaling systems and which in turn form control systems that effect changes in the signaling and stimulus–response systems of individuals. In other words, the function of these allelic variants is determined by both the developmental and the social system.

Furthermore, these control systems directly affect and are affected by developmental processes in both immature and adult insects and by the developing colony social structure. Behavior is thus the product of complex regulatory processes during ontogeny, phylogeny, and the life history of individuals, and, in the case of social organisms, also the colony or social system. The advantages of studying social behavior

from an evo-devo perspective are manifold and are of a pragmatic as well as a theoretical nature. Conceptually, behavior, as the ultimate integrator of form and function, helps us to expand the framework of regulatory gene networks to include organismal (physiology) as well as colony-level and environmental effects.

Innovation or the origin of evolutionary novelties has often been identified as the genuine evo-devo problem (Laubichler, 2007; Müller & Newman, 2005a, 2005b). We have already seen that recent evidence strongly suggests that any new phenotypic feature is the consequence of one form or another of regulatory evolution, i.e., that the developmental system determines whether or not a new phenotype is produced in the first place. Natural selection, of course, then decides its future fate. Innovation is thus connected to what Darwin already identified as the problem of the origin of variation. But while previous suggestions often invoked some sort of special cause or mechanism, such as macro- or systemic mutations, in order to account for phenotypic innovations, within the evo-devo framework these features are seen as a consequence of regulatory or developmental evolution. Understanding the limits and possibilities of the developmental systems thus becomes a major part in any explanation of macroevolution.

5 Conclusion: A Mechanistic Theory of Evo-Devo Challenges the Modern Synthesis

Twenty-first-century evo-devo offers a promising conceptual framework for addressing the problems of phenotypic evolution. It also ties these current issues, represented by the various research programs and concepts discussed above, to a diverse history, which has been crucial in informing present-day debates. Throughout the last 150 years, concepts such as form and function, ontogeny and phylogeny, constraint and selection, micro- and macroevolution have structured our understanding of these problems and provided analytical perspectives that have helped to illuminate the challenges and possibilities of such integration.

The main challenges of evo-devo are connecting different temporal scales (ontogeny and phylogeny), perspectives (molecular mechanisms of development and dynamics of populations), and analytical traditions (experimental and mathematical). Ron Amundson (2005) suggests that these differences represent the polar opposites of populational and typological thinking. The latter, in his view, has been dealt a bad hand, and he sees evo-devo as a way to rectify this. In my opinion, none of these dichotomies are particular helpful or informative; rather the solution lies in a more balanced approach grounded in a solid understanding of the underlying mechanism of developmental and evolutionary change. Within such a framework the recent discoveries of the enormous amount of conservation of key elements of developmental systems, such as the Hox genes, other transcription factors, and the structure of gene regulatory networks (E. Davidson, 2006a, 2006b; E. Davidson et al., 2003; Hinman et al., 2003; Howard & Davidson, 2004; Levine & Davidson, 2005; Manzanares et al., 2000; Negre et al., 2005; Santini, Boore, & Meyer, 2003), become the foundation for a more mechanistic account of phenotypic evolution than the ones that are built on the conceptual abstractions of the Modern Synthesis.

These discoveries have led to the idea of a general toolkit for development, shared by organisms from rather distinct phyla (Carroll, Grenier, & Wetherbee, 2001; E. Davidson,

2006a). As a rule of logic, phenotypic innovations or variation would have to be explained by corresponding changes in these developmental systems. The challenge for evo-devo, then, is not only to identify these changes between distant organisms, such as *Drosophila* and the mouse, but also to find those within populations or at the very least between closely related species. Such data would then allow us to integrate developmental with evolutionary processes in explanations of phenotypic evolution (E. Davidson, 2006b; Kruglyak & Stern, 2007; McGregor et al., 2007; Orgogozo, Broman, & Stern, 2006; Stern, 2007; Sucena, Delon, Jones, Payre, & Stern, 2003).

Furthermore, it has now also become clear that, for the most part, phenotypic novelties are a consequence not primarily of novel genes but rather of differences in the regulation of gene expression (in space, time, and quantity, as has been revealed by several long-running research programs that focus on the details of the regulatory logic underlying gene expression and differentiation: B. Davidson & Levine, 2003; Harafuji, Keys, & Levine, 2002; Levine, 1999; Levine & Davidson, 2005; Levine & Tjian, 2003; Mannervik, Nibu, Zhang, & Levine, 1999; Markstein & Levine, 2002; Markstein, Markstein, Markstein, & Levine, 2002; Shi, Levine, & Davidson, 2005; Stathopoulos & Levine, 2002, 2004).

The emerging picture is one of structured complexity with the regulatory networks exhibiting a modular and hierarchical structure that allows us to correlate different aspects of network architecture with different types of variational properties among organisms. Based on these results Eric Davidson and Douglas Erwin have suggested that different elements of the network architecture are connected with different morphological features of body plans that show varying degrees of conservation (E. Davidson, 2006a; E. Davidson & Erwin, 2006). Their model, initially derived from the molecular analysis of the genomic control of differentiation, thus provides the mechanistic underpinning for older observations in comparative anatomy and morphology that also described the nested hierarchy of body-plan features and homologies (E. Davidson & Erwin, 2006; Riedl & Auer, 1975; Wimsatt, 2007). Modern developmental genetics has thus led us to a molecular understanding of the fundamental principles of phenotypic diversity.

These observations also suggest a roadmap for accomplishing a true integration of developmental and evolutionary processes. What the molecular analysis of developmental processes and regulatory gene networks provides is a mechanistic understanding of both the development and evolution of phenotypic characters. On the developmental side the analysis of these mechanisms has revealed general architectural principles of developmental systems, including the properties of regulatory gene networks, the logic of differentiation, and the properties of morphogenetic processes. Comparing these mechanisms across a range of different taxa has provided us not only with well-established correlations between the variation in gene expression patterns and phenotypic characters, but also with several concrete hypotheses about how changes in the developmental systems can explain evolutionary transformations of phenotypic characters and about the evolutionary mechanisms driving these phenotypic changes (Arthur, 2002; Brakefield, 2007; Cameron, Peterson, & Davidson, 1998; Cebra-Thomas et al., 2005; B. Davidson & Levine, 2003; Hansen, 2006; Nijhout, 2003; Peterson, Cameron, & Davidson, 2000; Prum, 2005; Salazar-Ciudad, 2006).

The mechanistic framework of evolutionary developmental biology thus allows us to (1) understand the general principles of developmental systems governing phenotypic

patterns; (2) identify the causal connections between variations in those developmental systems and observed patterns of phenotypic variation; (3) suggest concrete evolutionary scenarios of how underlying developmental changes govern evolutionary transformations of phenotypes; and (4) develop experimental tests to uncover the selective forces driving these evolutionary transformations. In this way the mechanistic framework of developmental evolution unites the perspectives of form and function that have so often been associated with separate explanatory frameworks and sub-disciplines.

Postscript: Counterpoint

Any claim that evo-devo offers a significant conceptual challenge to the neo-Darwinian paradigm in evolutionary biology has, to a certain degree, revolutionary implications. One could argue that we are dealing with a small-scale revolution, an incremental change in the conceptual framework of evolutionary biology. If we accept Alessandro Minelli's claim in this volume that the core ideas of evo-devo are actually not much different from those held by the founders of the "Modern Synthesis," then the whole discussion about "revolution" or "challenge" would indeed be pointless as evo-devo certainly would be squarely located within the neo-Darwinian paradigm.

In returning to our initial question, does evo-devo offer a substantial challenge to neo-Darwinism, the main conclusion from these brief discussions is that it clearly does. My argument is based on the observation that the revolutionary implications of evo-devo lie in the conceptual synthesis based on the mechanistic integration of developmental and evolutionary processes; that this synthesis is built around a set of concepts, such as modularity, hierarchy, constraints, evolvability, novelty and emergence, robustness, history, etc.; and that reaching the promise of evo-devo will require not just continuous experimental work and more sophisticated mathematical models, but also a thorough analysis of the conceptual structure and a critical reading of the historical developments of the field. This last point gets us back to the initial discussion about the revolutionary nature of evo-devo. What we can observe in this context is that the promise of evo-devo lies in its potential to overcome the joint conceptual abstractions of "gene" and "population" that were so instrumental to progress in twentieth-century biology. But the reason for the success of these abstractions—that they reduced complexity and allowed the development of predictive models and productive research programs—also is the reason for their limitation with regard to capturing the complexity of underlying mechanisms. Insofar as evo-devo focuses on these mechanisms and takes full advantage of the changing nature of twenty-first-century century biology with all its data-richness and bioinformatics tools, it can truly become a bridge between (unanswered) nineteenth-century questions and twenty-first-century technology.

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CHAPTER TWELVE

Evolutionary Developmental Biology Does Not Offer a Significant Challenge to the Neo-Darwinian Paradigm

Alessandro Minelli

In this paper, I do three things. First, I show that it is not true that developmental biology has been always extraneous to the thought of neo-Darwinists, as it has become fashionable to say. Second, I maintain that developmental biology's broad absence from mainstream neo-Darwinism does not mean that development cannot be profitably and easily integrated with this view of evolution. Third, and most important, I argue that evo-devo is not simply developmental biology grafted onto evolutionary biology; rather, it deserves to be acknowledged as a research field of its own, with a specific agenda and a specific conceptual endowment.

1 Introduction

There are brands of scientists who should find it intrinsically difficult to identify their views, or those of their colleagues, with a frozen set of tenets. These scientists specialize in the study of change, be it the change an individual organism undergoes throughout ontogeny, or the change a population or lineage of living beings undergoes throughout a series of generations. Alongside developmental biologists (the scientists accustomed to work with changes in ontogenetic time) and evolutionary biologists (the scientists interested in the changes occurring in populations and species lineages), there are *evolutionary developmental* (evo-devo) biologists, who should be interested in both kinds and/or dimensions of change. What actually is evo-devo? The safest, although obviously unsatisfactory, answer to this question is that evo-devo is a discipline still in search of its identity (for a detailed analysis of the problem, see Müller, 2008). More technically, but not necessarily more satisfactorily, it is often

portrayed (e.g., Carroll, 2005) as the discipline aiming at understanding the evolution of complex organismal structures by applying the methods of developmental genetics comparatively. Others, however, acknowledge an autonomous research program of evo-devo, focusing on the evolution of development and, more characteristically perhaps, on less traditional topics like evolutionary innovations and evolvability (Hendrikse, Parsons, & Hallgrímson, 2007).

Somehow less controversial, but equally far from definitely settled, is the meaning of neo-Darwinism. The term was coined in the late nineteenth century to characterize the view according to which natural selection, together with hereditary variation, is the only cause of evolution. Eventually, this idea was newly and more effectively launched between the two world wars by the intellectual movement known as the Modern Synthesis, after the subtitle of an important collection edited by Julian Huxley (1942).

In the context of the debate about the challenge evolutionary developmental biology may offer to the neo-Darwinian view of evolution, the problem cannot be seriously attacked by limiting the comparison to sets of specific statements expressed by individual scientists, in either camp at some specified time, along the evolution of their personal thought. Indeed, it would be dangerously restrictive even to extract definitive sets of principles from mainstream textbooks or current research practice to be aseptically compared. Specifically, it can be seriously doubted whether the history of the disciplines relevant to our discussion can really offer evidence for neatly defined alternative paradigms and neatly defined revolutions. In my opinion, most of the apparent clashes between different research programs in this broad area of science can be explained by one or more of the following conceptually shallow reasons: (1) *local* research programs have been crowned with success despite the fact that they were developed without borrowing concepts, or evidence, from other fields; (2) semantic misunderstandings often occur, due to multiple use of the same terms for different concepts; and (3) reason of strategy may dictate opportunistic raising (or lowering) of boundaries between disciplines and research programs within the academic landscape.

I will articulate my argument along the following lines. First, it is not true that developmental biology has been always extraneous to the thought of neo-Darwinists, as it has become fashionable to say. Second, its broad absence from mainstream neo-Darwinism does not mean that development cannot be profitably and easily integrated with this view of evolution. Third, and most important, evo-devo is not simply developmental biology grafted onto evolutionary biology; rather, it deserves to be acknowledged as a research field of its own, with a specific agenda and a specific conceptual endowment.

2 The Evolving Evolutionary Synthesis

To put the present-day debate in context, it is useful to note that some key contributors to the Modern Synthesis of the 1930s, through which neo-Darwinism was re-launched as the standard paradigm of evolutionary theory, were keenly interested in embryology, and overtly acknowledged its relevance for understanding evolution (Love, 2003). This is not to deny, of course, that subsequent history eventually witnessed a vanishing of neo-Darwinians' interest toward development (e.g., Amundson, 2005),

but a couple of quotations from J.B.S. Haldane, Julian Huxley, and Gavin R. de Beer may help in appreciating the broader roots of neo-Darwinian thoughts. Some of these texts are particularly relevant from the perspective of a history of the relationship between evolutionary biology and developmental biology, as they were not published in journals or monographs primarily devoted to developmental biology, or in generally obscure publications. Instead, these texts are found in some of the most important monographs universally regarded as standard reference for the then consolidating neo-Darwinism, such as Haldane's *The Causes of Evolution* (1932a) and the collection edited by Huxley (1940) under the title *The New Systematics*, including a chapter by de Beer, which is illuminating in the context of our problem.

In a paper published in *American Naturalist*, Haldane (1932b) proposed a classification of genes according to what we would now call their *heterochronic effects*, that is, according to the time their expression is turned on or off during the development of an animal or a plant. Then, moving from semantic questions to the explanation of facts, Haldane suggested that a major difference between fishes and amniotes (gill clefts remaining open throughout life in the former, but being transformed and eventually eliminated in ontogeny in the latter) is probably explained by different temporal patterns of gene expression. Quite presciently, he also suggested that the characteristic body asymmetry of the gastropod mollusks (slugs and snails) is not necessarily a larval adaptation, as had been suggested by Garstang, but could perhaps be "a violent evolutionary novelty" due to heterochronic change, the anticipation of the larval stage of gene effects originally responsible for a relatively trivial asymmetry in the adult. Haldane (1932b), anyway, was convinced that "the time of action of genes not only merits further study by the geneticist, but is essential for a detailed discussion of evolution" (p. 22). He was even open to acknowledge that the combined effects of multiple genes expressed during development could sometimes lead to "apparently useless evolution" (p. 20).

In the Preface to *Evolution: The Modern Synthesis* (1942), Julian Huxley listed "developmental physiology" immediately next to genetics—and before ecology, systematics, paleontology, cytology, and mathematical analysis—as one of the disciplines that had contributed new facts or new research tools to the understanding of evolution. Specifically, 18 pages of this book illustrate "the consequences of differential development" on evolution. In a later chapter, following a summary of his personal studies with E.B. Ford on the "rate-genes" (heterochronic genetic effects) in *Gammarus*, Huxley remarked critically that attempts "have been made by representatives of the Morgan school (see e.g. Schultz, 1935) to minimize the importance of these discoveries, by asserting that they constitute only a re-description of old phenomena and add nothing truly new" (p. 528). Huxley rejected these criticisms outright: "On the contrary, I would maintain that the concept of rate-genes is as important for biology as is the concept of genetic balance or the gene-complex. I need not go into . . . the problem of how the genes become translated into characters. . . . Here we are concerned with [their] evolutionary implications" (p. 528). In the remaining of the chapter, Huxley mainly referred to heterochrony, a class of phenomena for which de Beer (1930, 1940) had already provided an articulated and, for that time, satisfactory treatment. Most interesting for the subsequent evolution of neo-Darwinism and its increasing obsession with adaptation is Huxley's observation that the study of developmental processes controlled by rate-genes—in addition to the light

it has thrown on the evolutionary aspects of recapitulation, neoteny, and fetalization—has provided cues to understanding "clandestine evolution, and apparently useless characters," topics that perhaps remain somehow indefinite in their nature and extent, but are clearly outside the limiting boundaries of adaptive evolution to which neo-Darwinism would be later restricted by many of its practitioners.

In the meantime, in the first edition of his very influential book *Genetics and the Origin of Species* (1937), Dobzhansky introduced the well-known definition of evolution as change in gene frequencies, a formula around which the most reductionist version of neo-Darwinism was eventually to be restricted. One wonders how far the success of this formula depended on the fact that this gene-centric approach was naturally prone to a quantitative, mathematical analysis, something that attracted to this research program the efforts of skilled mathematicians and undoubtedly contributed to raise this discipline's prestige by moving it away from a less appreciated descriptive natural history, and in the direction of scientific disciplines with a strong mathematical tradition, like chemistry and physics.

To deny the internal consistency and the explanatory power of the research program eventually developed from this core concept would be obviously foolish. Its success in providing a theory of adaptation has long provided stuff for textbooks. However, the point to be debated in this paper has little to do with the adequacy of population genetics, as a science of changes in gene frequencies, in explaining adaptation as the result of the action of natural selection on naturally occurring variation. The objection is that there can (and should) be more to evolutionary biology than a research program restricted to the concepts and tools of population genetics.

3 Population Genetics vs. Developmental Genetics

A first problem is that population genetics is, by definition, the study of genetic change at the level of population; the broadest scenario to which we can imagine to apply it is a set of loosely and marginally interbreeding, closely related species. It would be hard, indeed, to imagine how this approach could sensibly help in understanding macroevolution, that is, non-trivial changes in evolution such as those that accompanied the water-to-land transition of the early tetrapod vertebrates, or the origin of flowers, eyes, and wings. We do not advance much by simply denying that macroevolution is a phenomenon with its peculiar causative rules and suggesting, instead, that it is only an historical pattern whose actual mechanism is nothing but microevolution (natural selection on naturally occurring variation, at population level) repeated over thousands or millions of generations. This view looks attractively parsimonious, indeed, but its empirical content is virtually zero, for a good reason.

Population genetics can explain microevolution, but it is impotent in respect to evolutionary questions beyond the limit of the species. Being based on the natural mechanisms of gene transmission, it can only investigate the behavior of systems whose components are bound by the links of heredity (Amundson, 2005). Thus, how to compare species belonging to different orders, classes, or phyla? Population genetics and the study of adaptation have nothing to say about the differences between the membranous forewing of a fly and the sclerotized forewing (or elytron) of a beetle which may visit, with seemingly similar success, the same flower or the same heap

of dung. The fact that the fly and the beetle do not compete for the same reproductive resources only means that their differences cannot be explored by using the tools of population genetics. Microevolutionary analysis will never help us in reconstructing the features of their last common ancestor, or the divergent evolutionary histories of the fly and beetle wings. We can perhaps be satisfied with comparing the two appendages in terms of anatomical structure or functional morphology, or with the reconstruction of the wing body plan (*Bauplan*) in the last common ancestor of the fly and the beetle, as would result by using the algorithmic tools of pattern cladistics: that is, abandoning any hope to place the differences between fly's wing and beetle's elytron in a real evolutionary context. But this is exactly the point at which we can take advantage of recent progress, experimental and theoretical alike, in the study of animal development.

The cover of volume 1, issue 1 of *Evolution & Development* (published in 1999)—now one of the two major journals in the field—features a photo of a geophilomorph centipede. The reason why Wallace Arthur, one of the journal's editors, chose it for that eventually historical page was that a geophilomorph centipede offers a textbook example of an evolutionary problem that cannot be solved without the help of developmental biology. In this animal group, the number of leg-bearing segments varies, as far as known, between 27 and 191; but no even number in this interval ever occurs. This is particularly conspicuous—and obviously relevant (or puzzling) in terms of population genetics—since, for most species in this group, individuals with different (always odd, anyway) numbers of segments coexist in the same population. Why are there no individuals with an even number of leg-bearing segments? Can we explain their non-existence as an effect of selection? An alternative, and more plausible, explanation is perhaps that individuals with an even number of leg-bearing segments simply cannot be generated by the developmental mechanisms of segmentation. Clearly, a selective explanation looks quite unlikely in this case, but only experimental evidence can tell us whether the alternative explanation is valid.

Thus, the evidence required is about development, not about adaptation. That is, it must be provided by *developmental* genetics rather than by transmission genetics. Nevertheless, this developmental evidence will have important implications for evolutionary biology. So, the discontinuous variation in segment number is a typical target of *evolutionary developmental biology*. It tells us that—in some instances, at least—before asking questions about the relative fitness of alternative phenotypes, we must know which phenotypes can actually be produced. Using a currently fashionable term, we need to know whether the production of alternative phenotypes is in any way constrained, or biased.

As noted by Amundson (1994), when neo-Darwinians talk of evolutionary constraints, they generally mean constraints on adaptation: variation is taken, in principle, as available; thus, it is selection that must be responsible for the population not being able to get the highest peak in Sewall Wright's adaptive landscape. From a developmental biologist's perspective, on the contrary, to talk of constraints means to focus on constraints on the generation of phenotypic variation. Arthur (2004) has aptly remarked that the term *constraint* should be replaced by the neutral term *bias*, because intrinsic or extrinsic factors may affect the production of variation both negatively (e.g., Gould & Lewontin, 1979; Maynard Smith et al., 1985), in agreement with the common-sense meaning of *constraint*, but also positively, thus causing what Arthur

(2004) himself had termed *developmental drive*. Starting with mutation (Yampolsky & Stoltzfus, 2001), through the developmental generation of a phenotype, eventually accompanied by phenotypic plasticity (e.g., West-Eberhard, 2003), phenotypic variability is affected "by the structure, character, composition, or dynamics of the developmental system" (Maynard Smith et al., 1985, p. 265).

Acknowledging the existence of developmental constraints does not mean to deny the role of selection on the evolution of a given trait in a specified lineage of animals or plants, but to realize that both sides (variation and selection) of the Darwinian and neo-Darwinian model of evolution, not selection only, have structure of their own. One might say that in this way a generative or instructive, rather than permissive, element has been introduced into evolutionary theory, and that this represents a major breach in respect to the neo-Darwinian paradigm (e.g., Müller, 2003). But this is not true. Neo-Darwinism has been able to accommodate for directional selection without risking the acceptance of orthogenesis; today, evolutionary developmental biology is simply asking to look more closely at the variegated topology and metrics of intraspecific variation (e.g., Fusco, 2001).

4 Evo-Devo's Central Target: The Study of Evolvability

Consider, again, the variation in segment number among the geophilomorph centipedes. Population geneticists are accustomed to equate limits to the range of available phenotypes with limits in the extent of available genetic variation. This is an obviously sensible point, but it does not help us solve the problem with centipede segment numbers. The fact that individuals with 28, 30, or 32 pairs of legs are never produced, in a population where individuals with 27, 29, 31, and 33 pairs of legs are present, is not easily accommodated within a simple model of variation, by which a continuous distribution of segment number would be expected instead. Rather than lack of genetic variation, what forbids their generation are the mechanisms of embryonic segmentation, some critical steps of which restrict the resulting variation to a discontinuous set of phenotypes. We are still far from a detailed knowledge of segmentation mechanisms in these animals. Nevertheless, we can affirm with confidence that in centipedes evolutionary changes from an odd segment number to another odd segment number are extremely more likely than the apparently trivial, but seemingly impossible, change from a current, odd value N to the deceivingly close even value $N + 1$ or $N - 1$. Thus, the case of segment number in geophilomorph centipedes provides a very clear example of developmental bias on the *evolvability* of a trait.

From the point of view of evolutionary developmental biology (evo-devo), evolvability is largely a function of developmental systems' ability to generate variation (Hendrikse et al., 2007). Through development, genetic variation is translated into phenotypes subject to what has been called *developmental constraint* or *bias*, which include modularity, canalization, heterochrony, allometry, and integration. In fact, evo-devo provides its unique contribution to understanding the evolutionary process by a description and analysis of developmental constraint and its elements.

The concept of evolvability could be construed as extraneous to the neo-Darwinian program (because the latter simply identifies variation with genetic variation), but it is actually amenable to a neo-Darwinian definition, for example, as the ability of

the genetic system to produce and maintain potentially adaptive genetic variants (Hansen, 2006). The difficulties experienced until now in bringing together the traditional research program based on population genetics and the new program based on a comparative evaluation of developmental genetics are mainly due to an external, *technical* reason: there are a very small number of studies in evolutionary developmental biology performed to date within the same population-level framework to which population genetics is necessarily confined.

If transmission genetic studies across the species boundaries are virtually impossible, nothing would forbid, in principle, performing developmental genetic studies within the limit of one species or population; or, at least, to actively explore the developmental genetic basis of the morphological differences between very closely related species. Unfortunately, animals attractive from an evo-devo perspective, such as centipedes, do not have necessarily all the qualities of a good laboratory model species like the fruit-fly or the mouse. That is, their complete life cycle (say, from egg to egg) is tediously long and the problems of getting them mating in the lab have not been solved. The overlap between the favorites of evolutionary biology and the favorites of developmental biology is still very limited, but a few better-investigated examples show how fruitful an integrated evo-devo approach can be, and, more interesting in the present context, how harmless this is for the neo-Darwinian tradition in evolutionary biology.

Darwin's finches offer an excellent case in point. These little birds (13 species confined to the Galapagos Islands, plus one on Cocos Island) are a classic example of adaptive radiation within a group of fairly recent common ancestry, having been the subject of long-term field studies culminating in Grant's (1999) monograph. The most spectacular aspect of their adaptive radiation is the amazing diversity of their bills, which allows the varied species in this group to exploit very different food resources: insects, seeds, and fleshy fruits (one species actually can use a stick, firmly held with the bill, as a spear for collecting insect larvae living under tree bark or in logs). Recently, genes have been identified which affect the size and shape of these bills, and the major discovery has been that relatively minor heterochronic differences in the timing of the expression of one gene (*Bone morphogenetic protein 4*, or *Bmp4*) can account for the major differences of well-known adaptive value existing between different, closely related species of Darwin finches (Abzhanov et al., 2004).

A developmental biologist, thus, should be proud of having identified a gene whose expression has such a specific relevance for the phenotypic expression of a trait under strong selection (i.e., of great adaptive value). This does not seem to imply *per se* that this discovery changes the way an evolutionary biologist can see the history of adaptation of bill size and shape in this group of birds; this conclusion would be too hasty. In fact, with the discovery of the mechanism of gene control concerning bill development—and, especially, with demonstrating the large phenotypic effect of minor genetic differences—evo-devo has contributed something of fundamental interest to understanding evolution. It has thrown light, indeed, on the evolvability of a selectively important character, that is, on the nature and amount of genetic change that is correlated, in this case, with the phenotypic variation on which selection operates (Hendrikse et al., 2007).

From the Darwin's finches story, we can learn the general lesson that evo-devo is not simply the sum of two previously isolated research traditions (developmental

biology and evolutionary biology) but, rather, is an independent research field which asks specific questions that are relevant for developmental biology and evolutionary biology alike, and are empirically addressed by using the traditional tools of both these disciplines, but would be unanswerable within either of them in isolation. In the case of Darwin's finches, developmental biology informs us about the genes whose expression affects bill size and shape, while population genetics-based evolutionary biology tells us the effects of natural selection on the size and the shape of the bills and thus, indirectly, on the frequency of genes affecting this trait. The conceptual advance provided by an evo-devo perspective is to investigate *the same objects* (the same phenotypes and the same genes) from the point of view of both development and evolution, thus revealing something we could not know otherwise: the evolvability of the character under selection. By focusing on evolvability, evo-devo does not disrupt the neo-Darwinian view of evolution, as a process of change based on variation and selection (plus drift). Rather, it shows what can be gained by avoiding the myopic identification of variation with allelic differences only.

Interestingly, knowledge of evolvability helps us, also, in explaining the unusual invariance of some phenotypic traits, an otherwise intractable aspect of evolution. From a neo-Darwinian point of view, phenotypic invariance will generally translate into extremely strong stabilizing selection, but there are cases where this "explanation" is extremely unlikely. And a complete lack of genetic variation would not be easy to demonstrate beyond any doubt. This is exactly why an evolutionary biologist like John Maynard Smith (1960) became involved with segment numbers in centipedes and millipedes.

Characteristically, Maynard Smith did not focus on the macroevolutionary patterns of differences in segment numbers, which very commonly occur between different species or families, but on the complete lack of intraspecific variation in segment number found in many centipede and millipede species. This happens, for example, in some geophilomorph centipedes, where the number of leg-bearing segments is literally frozen, within each species, at not so small values as 45 or 49. Always odd numbers, as universally in centipedes, but in these species without the discontinuous variation (e.g., 39, 41, 43) more commonly found within each geophilomorph species. Maynard Smith was very puzzled by this uncommon level of developmental precision in the expression of a quantitative trait. At that time, developmental biology had nothing to offer besides the apparently reasonable guess that segments (whose number in this group is complete at the time of hatching) are probably generated, one by one, from a posterior, subterminal proliferating zone. But this was exactly what Maynard Smith challenged. How could a segment-producing process generate, say, 49 units one after the other, without ever missing the target? Is it reasonable to imagine the existence of a segment counting device of such a precision as to generate exactly 45 segments in *all* individuals of a given species, and exactly 49 in *all* individuals of another species?

Reasoning within a sound neo-Darwinian framework, Maynard Smith was not ready to acknowledge a complete lack of intraspecific variation for this quantitative trait; thus, he turned for a tentative explanation to the obvious missing link between genetic variation and phenotypic variation (in this case, invariance). The missing link was, of course, development, as "the expression of variation is structured by development" (Waddington, 1957, p. 294).

Maynard Smith was not a developmental biologist himself and, as noted, the developmental biology of the time had neither data nor models for explaining the lack of variation in centipede segment numbers. However, he advanced a tentative avenue toward an explanation. If intraspecific variation for genes potentially affecting segment number is very likely present, but the species is phenotypically uniform in this trait, then the developmental mechanisms responsible for segmentation must be quite robust. If so, it is unlikely that segments are produced serially, one by one, as this mechanism would require a counting device of unlikely precision. Maynard Smith suggested a different segmentation mechanism operating in two steps. At an early developmental stage the embryo is subdivided into a small, fixed number of primary segments. Then, at a later stage, each primary segment is subdivided into a fixed number of secondary, or final, segments. For example, there might be seven primary segments, each of which subsequently undergoes three runs of binary splitting. In this way, the embryo is eventually articulated into exactly $7 \times 2 \times 2 \times 2 = 56$ segmental units, thanks to a mechanism which is expected to be considerably more robust in relation to developmental noise that is a counting device targeted to stop segment production when exactly that number of segments have been generated.

Irrespective of the eventual explanatory success of this model in comparison to our current (still incomplete) awareness of segmentation processes in centipedes, Maynard Smith's theoretical exercise was really a walk into what we now call evo-devo. His question was originally framed in terms of intraspecific variation, that is, in classic neo-Darwinian style, but the empirical evidence at hand—i.e., the apparent lack of intraspecific variation for a quantitative trait—was clearly intractable without analyzing variation in developmental terms. If Maynard Smith, or other neo-Darwinians, failed to walk more frequently into the field of development, this was due to the kind of problems they chose to investigate, rather than to the lack of a reasonable and potentially productive interface between developmental biology and neo-Darwinian evolutionary biology.

5 Origins?

Besides evolvability, another major area has often been flagged as characteristic of the research program of evo-devo and also as a possible cause of a clash with the neo-Darwinian tradition in evolutionary biology. This area is the origination of evolutionary novelties.

To be sure, this problem is as old as evolutionary biology, having being addressed by Darwin himself in the *Origin* (1859). It also has a place in the neo-Darwinian tradition, but the way it was framed—for example, by Ernst Mayr (1960)—is regarded as inadequate by several representatives of present-day evolutionary developmental biology.

Characteristic of the Darwinian and neo-Darwinian tradition is a strong idea of continuity of evolutionary change, within which—as noted by Mayr (1960)—it is often difficult to say to what extent a structure is new, or just an old one revisited. In particular, major changes are often obtained by progressively adding new components to an already existing core, as might have occurred in the evolution of the eye. In this sense, as noted by Brigandt (2006), for neo-Darwinism a novelty is not really a structure that did not exist in a species' ancestors, but only a modified structure with

a qualitatively new function. On the contrary, according to Müller and Wagner (1991), a novelty is a structure that did not exist before; thus, the problem of its origin cannot be addressed in terms of function and adaptation.

In my opinion, the contrast between the neo-Darwinian approach and the evo-devo approach to evolutionary novelties does not go beyond a difference in what is put in focus: the Darwinian and neo-Darwinian approach stresses changes in function, faithful to the research program's general focus on adaptation, whereas evolutionary developmental biology introduces a structural component in the analysis of novelty.

Contrary to some evolutionary developmental biologists (e.g., Müller & Newman, 2005; Newman & Müller, 2000), however, I believe that evolutionary novelties stand out as completely new features only if we *arbitrarily* decide to select a given set of traits as a unit for which we cannot trace a global homology to the features present in the relevant ancestor we have chosen as term of comparison. Bird wings have no precedent as flying devices largely made of regular ranges of feathers, but the skeletal parts supporting these feathers are shared by birds with all tetrapod vertebrates that have retained the forelimbs.

I do not share the widespread perspective in comparative biology according to which in analyzing an organism's structure we can recognize a hierarchy of objectively identifiable homologues. Instead, I favour a factorial approach to homology (Minelli, 1998, 2003; Minelli & Fusco, 2005) according to which all novelties are seen as a mixture of conserved features and novel elements to be disentangled before submitting them to analysis.

The strongest version of the view that evolutionary developmental biology is uniquely entitled to explain the origin of novelties is the frequently expressed view that only evo-devo can explain the origin of body plans. The success of developmental genetics in identifying genes involved in the control of major body features such as the number of wings in the fruit-fly, or the alternative development of an insect appendage as an antenna, a mouthpart, or a leg, have fuelled the hope to eventually reduce the origin of the most conspicuous differences between different animal body plans (e.g., the vertebrate, the insect, the nematode worm) to a limited number of specific genetic or genomic changes.

There are two major problems, however, with this view. One problem is this obsession with origins, about which something has already been said before. The other is the very concept of body plan (or *Bauplan*, to use its original, nineteenth-century German name), which has been reintroduced in biology through widely used textbooks such as Brusca and Brusca's *Invertebrates* (1990), and has become quite fashionable among students of animal evolution (e.g., Valentine, 2008) and, specifically, of evolutionary developmental biology (e.g., Arthur, 1997; Carroll, 2005; Raff, 1996). This concept, however, is nothing but a typological abstraction of a set of a group's diagnostic characters and has no legitimate status at all in evolutionary biology (Breidbach & Ghiselin, 2007).

6 Conclusion

Summing up, I contend that the research program and results of evo-devo are in conflict with the tenets of neo-Darwinism only if we select, from either camp, some

specific sets of tenets that are, however, matters of a personal (and temporally restricted) position within these research traditions. Interestingly, Gilbert (2003) has noticed a strict parallel between the discussion over whether evo-devo is separate from the population genetic framework of the Modern Synthesis or if it fits into the Modern Synthesis and past controversies over whether genetic assimilation fits, or not, into the neo-Darwinian paradigm. As in the present case, there were then arguments for either view, and in the end a decision depended on whose opinions the comparison was based on.

Once more, a perusal of texts from the earlier times of the Modern Synthesis can be illuminating. As I flagged above, Julian Huxley's keen interest in the role of developmental biology in evolutionary biology elicited a very critical reaction of a member of Morgan's school who dismissed the relevance for evolutionary biology of the studies of gene expression. Characteristically, this was not a clash on theoretical or methodological principles, but an empirical argument employed to defend the self-consistency and the academic prestige of a school. Perhaps it was not Huxley's rate-genes that were perceived as a threat to an evolutionary biology increasingly rooted in population genetics, but the threat was potentially represented by the views of another scientist who at that time was suggesting that some genes do not have the small, additive effects of the genes the *Drosophila* geneticists were then studying in the lab, but could open, or re-open, the door to a saltationist view of evolution. To be sure, this scientist—Richard Goldschmidt, of course—had to be silenced. Goldschmidt's best known and most controversial book, *The Material Basis of Evolution*, was published in 1940. Huxley (1942) referred to it with praise in *Evolution: The Modern Synthesis*. But that was the last time, for a long while, that Goldschmidt's ideas were flagged as important for evolutionary biology. Characteristically, we had to wait for that until 1982, when Stephen Jay Gould issued a new edition of *The Material Basis of Evolution*. Also characteristically, Goldschmidt's name would eventually appear, in the late twentieth century's literature, when developmental genetics succeeded in the identification of major changes in a body structure, such as developing a leg where an antenna would normally be formed, as a consequence of a mutational change affecting the spatial expression of a Hox gene. Was this discovery actually the vindication of Goldschmidt's hopeful monsters? If so, evolutionary biology's fondness for these results would equate to this discipline's self-exclusion from the established tradition in evolutionary biology. No harm, however. Fruit-flies with antennae replaced by legs are desperately hopeless monsters, as are their well-known four-winged companions, the phenotypic expression of a mutation affecting the expression of another Hox gene. These monsters can only survive in the protected environment of the lab. Their fitness, a population geneticist would say, is actually zero. Too much was expected from a single mutation. Thus, let's try to dissect evolutionary history into a series of more gradual steps. Researches such as those on the genes controlling bill size and shape in Darwin's finches, and equivalent researches on the genetics of species-level differences among columbines (e.g., Kramer et al., 2007) and other flowering plants, are opening the avenue for a developmental analysis of the material basis of evolution. Thus: "The suggestion that neo-Darwinism is somehow incompatible with embryology makes no more sense than the notion that it is incompatible with genetics. . . . Continuing to synthesize seems perfectly reasonable, given the record of our predecessors" (Breidbach & Ghiselin, 2007, p. 169).

Understanding evolution requires evidence of evolvability, which is the most characteristic research target of evo-devo. By studying evolvability, the study of variation—together with natural selection, one of the two prongs of the neo-Darwinian approach to evolution—expands from the aspects accessible to transmission genetics to the aspects targeted by developmental genetics. As presciently shown by John Maynard Smith's paper on segmentation, exploring evolvability does not conflict with neo-Darwinian tradition. To the contrary, appreciation of the natural integration of the two approaches will, it is hoped, steer focus in evolutionary developmental biology toward the study of variation in developmental processes within the traditional neo-Darwinian arena, that is, at the species and population level. Relationships between disciplines are still open anyway; thus, I think that Gilbert and Burian's (2003) summary that evo-devo "is both a synthesis between evolutionary biology and developmental biology and an ongoing negotiation between these two disciplines" (p. 61) is still up-to-date.

Postscript: Counterpoint

I agree with Manfred Laubichler's tenet that the targets of evo-devo research are not identical to those of neo-Darwinian evolutionary biology, in particular because of evo-devo's focusing on the phenotype and, I would add, on evolvability. However, I would not describe as a challenge to an established paradigm a research program which brings in a new theoretical perspective and a novel class of explanations, as long as these perspectives and explanations are not in conflict with the former paradigm and, indeed, may well be described as extending or complementing it.

Furthermore, Laubichler characterizes evo-devo as a "counter-revolution." If this means abandoning the most extreme positions of recent neo-Darwinists like Richard Dawkins to re-discover problems and attitudes once shared by some of the architects of the Modern Synthesis like Julian Huxley, as I argue in my paper, this seems hardly to represent a real challenge to neo-Darwinism. Rather, this is a witness of the conspicuous evolution of perspectives and research agenda that has happened within neo-Darwinism since its inception.

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PART VII

WERE THE BASIC COMPONENTS OF THE HUMAN MIND SOLIDIFIED DURING THE PLEISTOCENE EPOCH?

Introduction

This section begins with a paper from Valerie Starratt and Todd Shackelford, two advocates of evolutionary psychology. All evolutionary psychologists posit that evolution is responsible not only for human physiology and anatomy, but also for certain human psychological and behavioral characteristics that evolved in our past to solve specific problems of survival. The time period that many evolutionary psychologists look to as being significant for the evolution of the mind is the Pleistocene Epoch (1.8 million years ago to 10,000 years ago), and this would make sense since mitochondrial DNA and fossil evidence indicate that this is when modern *Homo sapiens* evolved. Just as other traits developed functions in some specified evolutionary history, so, too, the human brain is envisioned as having evolved certain psychological *modules*, *intelligences*, or *domains* that, in the words of Leda Cosmides and John Tooby (1992), are "specialized for solving evolutionary long-enduring adaptive problems and . . . these mechanisms have content-specialized representational formats, procedures, cues, and so on" (p. 34). Some of these mental modules, like those associated with rudimentary phoneme and object recognition, are considered domain-specific, since they are devoted to solving one particular kind of adaptive

problem. Other modules—or, possibly, just one huge module—are considered domain-general, since they are devoted to solving any number of adaptive problems. Thus, Steven Pinker (1997) claims that the mind "is not a single organ but a system of organs, which we can think of as psychological faculties or mental modules . . . intelligent behavior is learned successfully because we have innate systems that do the learning" (p. 27)—such systems having evolved to deal with adaptive problems in our early hominin past.

Evolutionary psychology has generated testable results and an entire research program all its own, and Starratt and Shackelford explain some of this research in their paper. For example, people very readily can detect cheaters, at an early age even, and a sensible explanation for this has to do with the fact that members of our early hominin past had to be very good at this in their socially evolved groups during the Pleistocene. Thus, this trait became entrenched in our psyches. There are many other examples whereby evolutionary psychology has been explanatorily useful, including with respect to evolved landscape preferences, stepchild abuse by stepfathers being 40 times higher than by biological fathers, relationship-specific betrayal sensitivities, waist-to-hip ratio as being a marker of fertility and beauty, deception in mating tactics, superior female spatial location ability, and many others (see Buss, 2007).

However, because of brain-process and psychological modules interacting with environments for thousands—perhaps even millions—of years, evolutionary psychologists think that psychological modules have become specialized in their particular performances to deal with the various challenges posed by the environments in which our hominin ancestors *used to* live during the Pleistocene Epoch. What about the kinds of abilities that humans have *today*? Further, what about mental mechanisms that most assuredly were in place *prior to* the Pleistocene Epoch? In his paper included in this part, Stephen Downes is concerned with exactly these kinds of questions, and challenges this Stone-Age-mind-like explanation given by evolutionary psychologists. Downes claims that the

> full story of the evolution of the human mind will include components that account for our lineage's divergence from that of the rest of the great apes and components that account for our predictable psychological responses to aspects of large highly complex social groups. Components of this account will come from various times in our evolutionary history, including the Pleistocene, but also including times long before and long after this period.

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CHAPTER THIRTEEN

The Basic Components of the Human Mind Were Solidified During the Pleistocene Epoch

Valerie G. Starratt and Todd K. Shackelford

The basic components of the human mind, i.e., evolved psychological mechanisms, were solidified during the Pleistocene Epoch. The Pleistocene is the time period between 1.8 million years ago and 10,000 years ago that included the environmental selection pressures (both internal and external) that are responsible for the evolution of human psychological mechanisms. Although we share some psychological mechanisms with our pre-hominid ancestors—such as those that motivate predator avoidance behaviors—these mechanisms could not have "solidified" before the arrival of our hominid ancestors. Humans continue to be subject to natural selection. However, because the time between the end of the Pleistocene and today is such a small portion of human existence (one half of 1%), it is unlikely that evolution has altered human design greatly over the last 10,000 years.

1 Introduction

The basic components of the human mind were solidified during the Pleistocene Epoch. Before discussing this further, however, we will clarify our definition of the key terms. First, we interpret "the basic components of the human mind" to be the varied and numerous psychological mechanisms that evolved to solve specific adaptive problems. An adaptive problem is any hindrance to survival or reproduction that occurred repeatedly throughout our ancestral history. These adaptive problems included, but are not limited to, pressures to evade predators, pressures to capture prey, and pressures to out-compete same-sex rivals for access to the most desirable mates. One example of an adaptive problem is the need for nutritious food. Ancestral humans who could not successfully identify nutritious foods would have been at a disadvantage relative to those who had a diet of more calorically dense foods. Psychological mechanisms are the responses that function to solve adaptive problems

such as this. Psychological mechanisms include information-processing mechanisms and internal motivations that work in conjunction with internal and external stimuli to produce behavior. Continuing with the example of the need for nutritious food, the associated psychological mechanisms include the preference for high-calorie foods (i.e., foods containing relatively large amounts of fats and sugars). Individuals who preferred to eat foods with high fat and sugar contents gained survival and reproductive advantage over those who did not.

Second, we address the Pleistocene Epoch. This covers the time period from about 1.8 million years ago to a little more than 10,000 years ago. This time period is most relevant to the current discussion because of its association with the Environment of Evolutionary Adaptedness (EEA). The EEA is not a place or a time in history, but a statistical composite of the selection pressures (i.e., all environmental characteristics influencing the ability of individuals of a species to survive and reproduce) operating on the adaptations that characterize a species' ancestral past (Tooby & Cosmides, 1990). Each adaptation has its own specific EEA, but there is likely to be overlap in the EEAs of similar adaptations within the same organism. The Pleistocene is the period of time hypothesized to contain the EEAs of the majority of human-specific adaptations. The early Pleistocene was the period of time during which the genus *Homo*, of which modern humans are the only remaining species, first appeared. If humans appeared no earlier than 1.8 million years ago, then adaptations specific to the human mind could have evolved no earlier than that.

Third, we address the solidification of these psychological mechanisms. Although we argue that psychological mechanisms were solidified during the EEA, we do not believe that the evolution of human psychology is fully contained between 1.8 million years ago and 10,000 years ago. Many psychological adaptations, such as those that motivate predator avoidance, are likely to have evolved in our pre-hominid ancestors, then perhaps sharpened in our hominid ancestors to motivate avoidance of predators that targeted them. In much the same manner, we do not support the argument that evolution stopped 10,000 years ago. Humans continue to be subject to natural selection. However, that is not contrary to our argument that most of our psychological mechanisms evolved during the Pleistocene and continue to be expressed today. In the evolutionary history of humans, 10,000 years is a brief period of time—roughly one half of 1% of the 1.8 million years of human existence. Additionally, geneticists investigating human genetic diversity report that 80% of all genetic differences are among individuals within the same population. In contrast, variations among populations of different continents account for only about 10% of all genetic differences. This suggests that most genetic variation occurred before modern humans migrated out of Africa roughly 100,000 years ago (see Owens & King, 1999, for a brief overview). If the bulk of our species' genetic makeup has remained relatively constant over the last 100,000 years, it is not unreasonable to argue that our psychological design (which is built by our genes) has remained relatively constant over the last 10,000 years.

This argument should not be misinterpreted to mean that, because psychological mechanisms were solidified hundreds of thousands of years ago, modern human cognition mirrors that of our ancestors. For instance, our ancestral environment did not contain selection pressures for performing calculus or geometry. However, because humans today are capable of such higher-level analytical reasoning does not mean that there is a psychological mechanism devoted to geometry. Most likely the ability

to perform such reasoning results from a parasitization of the evolved mechanisms that provide the ability to perform other, evolutionarily relevant reasoning and cognitions, such as tracking prey through dangerous terrain or successfully maneuvering tricky social situations.

2 Evolution

Evolution by natural selection is the only known scientifically viable process capable of producing the complex construction of the human body and brain (see Dawkins, 1986). Natural selection is the process that acts on characteristics in a population in the presence of the following necessary circumstances: variation, heredity, and intra-species competition resulting in differential reproduction (Darwin, 1859, 1871; Mayr, 1982). First, a characteristic must exist in varied forms within the target population. Second, that characteristic must also be subject to heredity—have a genetic basis that can be transmitted from parent to offspring. The third specification is that the characteristic must be differentially beneficial, such that some variations better aid survival and reproduction compared to other variations. Individuals within the population who display the relatively more beneficial variations of that characteristic will out-reproduce those with other variations. Subsequently, the relatively more beneficial variations will be more likely to be spread throughout the species.

Darwin (1871) originally distinguished sexual selection from natural selection in an attempt to explain the existence of reliably developing characteristics in some species that appeared to hinder survival (most notably, the peacock's tail). Sexual selection is concerned with reproduction, rather than survival. There are two components to sexual selection: intrasexual competition and intersexual competition. *Intrasexual* competition involves competition among members of the same sex for access to the most desirable mates. In several deer species, for example, large antlers allow males to intimidate and physically compete against one another for social dominance, access to resources, and sexual access to the most desired females (e.g., Bowyer, 1986; Kucera, 1978). *Intersexual* competition, on the other hand, refers to differential mate choice of members of the opposite sex. For example, the large and vibrant peacock's tail is a hindrance to survival. It is an impediment when escaping predators, an obstacle when stalking prey, and physiologically costly to build and maintain. However, it is incomparably helpful when attempting to attract a mate. Peahens are most attracted to peacocks with the most impressive plumage and, so, grant sexual access to these high-quality peacocks over those with less impressive trains (e.g., Petrie & Halliday, 1994; Petrie, Halliday, & Sanders, 1991). Here we present natural selection and sexual selection as two separate entities, but today both processes are often categorized under the term natural selection.

Evolutionary psychologists often speak of natural selection as responsible for "designing" the mind. This can be an unfortunate short-hand, as it is often misinterpreted as implying that natural selection acts with intent. Evolution, however, has no intent. Genes cannot see into the future to determine what will be beneficial later. The selection pressures that act on organisms are not a unidirectional force pushing organisms from point A (amoeba) to point B (human). Rather, selection pressures are a conglomeration of all the forces acting on an organism that impact the organism's

survival or reproduction. These can include pressures to evade predators, pressures to capture prey, pressures to out-compete same sex rivals for access to the most desirable mates, or pressures from any number of other sources. Evolution is not deliberate in that it is not forcing organisms down a pre-determined path of species development. The genetic mutations that produce variations in the traits that they build occur randomly. It is just as likely that random mutation produces a gene coding for immunity to cancer as a gene coding for faster-growing fingernails. The process through which some mutations survive natural selection and become part of the genotype that builds adaptations, however, is non-random. An individual whose genes build adaptations that produce immunity to cancer gains a significant advantage in survival and reproduction. It is likely that an individual with quickly growing fingernails shares no such advantage. Only those random mutations which lead to beneficial adaptations are consistently selected for and are spread throughout the species.

There are three distinct products of evolution by natural selection: adaptations, by-products, and noise. An adaptation is an inherited characteristic that reliably develops within a species and functions to solve a particular adaptive problem (Buss, Haselton, Shackelford, Bleske, & Wakefield, 1998; Thornhill, 1997; Tooby & Cosmides, 1990). An example of a *physical adaptation* is the umbilical cord. It is species-typical (i.e., reliably developing in all members of a species) and serves the necessary function of transferring nutrients from a mother to her fetus. *By-products* do not serve a particular adaptive function, but exist as a direct result of an adaptation. An associated by-product of the aforementioned adaptation would be the belly button. It serves no function, but occurs as a direct result of the necessary umbilical cord. *Noise* is random variation that exists within a population. It does not aid in survival or reproduction and is not directly associated with an adaptation. The shape of the navel represents noise. The particular shape of one's navel serves no function and is not directly related to any adaptation.

3 Evolutionary Psychology

Evolutionary psychologists attempt to understand human behavior by identifying how humans lived during ancestral times and the adaptive problems they were likely to face. Critics of evolutionary psychology argue that it is impossible to know how early humans lived, so how can we possibly know what types of problems they faced? It is true that we cannot know all of the specifics of ancestral life. However, there are several aspects of ancestral life of which we can be certain. For instance, we can be certain that ancestral humans breathed oxygen and were subject to the laws of gravity. We can also be certain that ancestral women, and not men, bore children. This one fact alone underscores several theories of human behavior. One of the most influential theories to develop from this fact is the theory of parental investment (Trivers, 1972). Given the biology of human reproduction, women are required to invest significantly more in the production of offspring than are men. At the very least, a woman must devote nine months to gestation and often several years of lactation and constant care to ensure the survival of one child. A man, on the other hand, need invest little more than an ejaculate. Because of the relatively large parental investment required of a woman and the relatively small number of possible offspring produced

throughout her life, her best interest may lie in careful selection of a mate who is willing and able to provide resources for her and her offspring, thereby increasing the likelihood of each offspring's survival. As such, women should demonstrate a preference for high-status men with sufficient access to resources who appear willing to share those resources with her and her children. A man, on the other hand, is not limited by such restrictions. Should a man have sex with 100 different women, he has the possibility of siring 100 different offspring. Consequently, his best reproductive interest may lie in attracting as many mates as possible. This could be demonstrated by a willingness to engage in casual sex with a wide variety of women. There is a large body of evidence supporting the existence of these sex-differentiated mating strategies (Buss, 2003).

Evolutionary psychologists investigate psychological adaptations. Psychological adaptations, often referred to as evolved psychological mechanisms (EPMs), have the following characteristics:

1 An EPM exists as it does because it solved a recurrent adaptive problem.
2 An EPM processes only the specific stimuli relevant to the particular adaptive problem it evolved to solve.
3 An EPM makes the organism aware of the particular problem it is facing.
4 The input received by an EPM is transformed into output via decision rules.
5 The output produced by an EPM can be physiological activity, information that becomes input to another EPM, or manifest behavior.
6 The output is directed to the solution of the specific adaptive problem that EPM functions to solve (Buss, 2005).

In the venue of evolutionary psychology, adaptations do not refer to the behaviors themselves. Rather, the adaptations are the psychological mechanisms: the biases that motivate individuals to perform certain behaviors that, in turn, served as solutions to adaptive problems.

Consider, for a comparative example, the physical adaptation of the human eye. The human eye evolved to be sensitive to a particular type of stimuli: light waves that fall in the visual spectrum. The human eye does not attend to, or respond to, other types of stimuli, such as smells or even light waves that fall outside of the visual spectrum (e.g., infrared waves). Our eyes respond to light waves, and not smell, for much the same reason as our hearts pump blood instead of pumping blood *and* storing waste. One system performs one function. Our eyes respond to light waves instead of infrared waves because the stimuli most relevant to our survival and reproduction reside in that spectrum.

The human mind works in much the same way. There is a vast store of internal and external stimuli available to the mind. However, it would be impractical, if not impossible, for each part of one's mind to attend to all stimuli at once. Consequently, in much the same way as the human eye focuses solely on light waves, so do the individual components of the human mind focus on particular sets of stimuli. The psychological mechanism that generates fear, for instance, responds only to fear-relevant stimuli. If you come across a snake lying in your path, the stimuli associated with that snake will be processed by the psychological mechanism responsible for fear-related stimuli. This psychological mechanism, then, will motivate any of a number

of responses. Your physiological responses may include a change in heart rate, breathing pattern, and diversion of physiological resources from digestion to muscle contractions. Your psychological responses may include an urge to run away or an unwillingness to move. It is likely, however, that you do not feel the desire to mate with the snake. This is because snake-relevant information is processed by the psychological mechanism that evolved to solve problems associated with escaping organisms that could cause bodily harm, and *not* by the psychological mechanism that evolved to solve problems of mate selection.

But how do we know that fear is produced by an evolved psychological mechanism? Mineka and Öhman (2002) present four characteristics of fear that indicate it is produced by an evolved psychological mechanism. First, fear is most likely to be associated with stimuli that would have been ancestrally dangerous, such as snakes, spiders, and heights. This is not to imply that fear of a particular object or situation occurs in the absence of any learning. However, people appear to be more prepared to learn fear of ancestrally dangerous stimuli than evolutionarily novel stimuli. It is easier and quicker to condition fear in response to snakes, for instance, than to cars or damaged electrical outlets, even though all three items are considered dangerous in today's environment. Second, fear occurs automatically, that is, without the need for conscious processing. This automaticity, however, has only been demonstrated in response to fear-relevant stimuli. The psychological mechanism that produces fear is not automatically activated in response to kittens, but can be automatically activated by spiders. Third, fear appears to be disconnected from higher-level conscious thought. An individual who fears spiders may understand consciously that a picture of a spider can cause no harm. However, this does not prevent the activation of a fear response. Finally, neurological research has demonstrated that the neural mechanism responsible for fear is seated in the amygdale, an evolutionarily old part of the brain that is shared with other mammals. These neural mechanisms also appear separated from neural mechanisms associated with learning material that is unassociated with fear.

Fear also exhibits the characteristics Buss (2005) describes as being true of all psychological mechanisms:

- *Characteristic 1: An EPM exists as it does because it solved a recurrent adaptive problem.* The fear of snakes helped to solve the recurrent adaptive problem of avoiding an animal that is potentially harmful or lethal.
- *Characteristic 2: An EPM processes only the specific stimuli relevant to the particular adaptive problem it evolved to solve.* When you encounter a snake, the fear mechanism is devoted to processing the information relevant to the snake. This information may include the size and shape of the animal, the nature of its movements, and its distance from you. The color of the flowers on the bush next to the snake, however, is not information relevant to the problem at hand and, so, is not likely to be processed in the same manner.
- *Characteristic 3: An EPM makes the organism aware of the particular problem it is facing.* Fear of the snake draws your attention toward the snake. It would be unproductive to direct your attention to irrelevant stimuli, such as the color of those flowers, while still in the presence of potential dangers associated with the snake.

- *Characteristic 4: The input received by an EPM is transformed into output via decision rules*. Encountering a snake does not produce the same response regardless of the situation. Rather, the input of seeing the snake may pass through any one of a number of decision rules, such as "if the snake appears disturbed by your presence and looks as though it may strike, then run in the opposite direction" or "if the snake is very small and does not appear as though it can harm you, then continue along the path."
- *Characteristic 5: The output produced by an EPM can be physiological activity, information that becomes input to another EPM, or manifest behavior*. Physiological responses to seeing a snake may include a change in heart rate, breathing pattern, diversion of physiological resources from digestion to muscle contractions, or any number of other responses designed to facilitate either fight or flight from the situation. If the snake is deemed non-harmful, then the information may be passed to another EPM. For instance, if it is determined that the snake is not harmful, and you are hungry, then it may be deemed a good dinner. The relevant information would then be passed to the EPM designated to solve the adaptive problem of food acquisition.
- *Characteristic 6: The output is directed to the solution of the specific adaptive problem that EPM functions to solve*. Suppose that the decision rules associated with encountering a snake determined that a behavioral response is warranted. What is a more beneficial response, to run as fast as I can in the opposite direction, or to sneeze? Running in the opposite direction would function to remove me from the dangerous situation, thus solving my problem. Sneezing, on the other hand, most likely would do nothing to help my situation.

4 Ultimate vs. Proximate Causes of Behavior

Some critics argue that evolutionary psychology focuses too much on the ultimate causes of behavior, as opposed to the proximate causes of behavior. *Ultimate* causes of behavior are those defined by the adaptations addressing the adaptive problems facing our ancestors. For instance, an ultimate explanation for why you ate that double cheeseburger for lunch may be that your ancestors preferred foods that were rich in fat and, so, out-reproduced others who preferred less calorically dense foods. *Proximate* causes of behavior refer to the present environment in which the behavior occurs. Maybe you ate that cheeseburger because you were hungry, and you happened to walk by a restaurant that serves cheeseburgers. An evolutionary psychological perspective, however, would then beg the question of why that cheeseburger existed in the first place? That cheeseburger existed because at some point someone realized that people preferred to eat, and would spend money on, fatty foods over other available foods. Why do people prefer to eat fatty foods? Because over human evolutionary history, individuals who preferred high-calorie foods were more likely to survive and reproduce.

We do not argue that ultimate causes of behavior are the only causes of behavior. Rather, we argue that proximal causes *are not* the only causes of behavior. Ultimate causes of behavior are responsible for people's inherent biases. Proximal causes of

behavior serve as the catalysts that trigger those biases to motivate certain behaviors. If the only causes of behavior were ultimate causes, then (ridiculous) logic would follow that certain human behavior would occur in the absence of any environment. Suppose a man exists in a void: a place of nothingness where there is no environment with which he can interact. Would that man spend his days pantomiming behaviors such as eating and mating, even if there were no food to eat and no women with whom he could mate? If ultimate causes of behavior are the only causes of behavior, the answer would have to be an absurd yes.

The flip side of that argument is that proximate causes are the only causes of behavior. If evolution played no part in the development of adaptations and subsequent behaviors, then all human behaviors would be learned via socialization or trial and error, and all behavior would be motivated solely by the current environment and past personal experience. We are what we are because of what we are taught. But if this were true, how would it explain phenomena such as a two-month-old child's preference for attractive faces over less attractive faces (Langlois et al., 1987)? Is the first two months of life sufficient to learn and develop a preference for social conventions of beauty?

If neither of the above extremes appears sufficient to explain human behavior, then there must be some integral interplay between evolved mechanisms and contemporary environments. Again, consider the example of the human eye. The structure and capabilities of the eye itself are a result of the evolutionary history of the stimuli presented to it. Today, our eyes are sensitive to light waves because that is what was most beneficial to our ancestors. However, individually, our eyes see what they see based upon our own current environment. We see what is in front of us at the moment. Our retinas are not burned with the images presented to our ancestors. The same is true of psychological mechanisms. Different components of the human mind evolved to attend to particular stimuli that were especially relevant to solving particular problems facing our ancestors. However, the output of those mechanisms is not staunchly predetermined. Obviously, behavior is heavily reliant upon the current environment.

A related argument is that adaptations and the environment cannot be separated as distinct entities. Adaptations exist as they do as a direct result of past environments. The available stimuli supported the development of adaptations that attended to and solved problems associated with those stimuli. As a result, those stimuli became more salient to the organism and a larger factor in the surrounding environment. Thus, environments today exist as they do as a direct result of adaptive mechanisms. Environments and adaptive mechanisms are so heavily reliant upon each other that they cannot be rightly separated (Cronin, 2005).

It has been argued that an evolutionary psychological approach is ill-equipped to address the question of how multiple proximate causes can account for any given behavior (Downes, 2005). A proper understanding of the interplay between psychological mechanisms (ultimate causes of behavior) and the environment (proximal causes of behavior), however, belies this as a problem. Psychological mechanisms act as a set of decision rules for interpreting stimuli associated with an adaptive problem and motivating behavior according to what has been beneficial ancestrally in solving that problem. Let us address the particular example used by Downes (2005). A prominent adaptive problem is that of mate selection, or identifying and successfully attracting

a member of the opposite sex for the purposes of reproduction. Three separate hypotheses (in addition to several others) have been proposed to address the issue of how human males select high-quality mates: waist-to-hip ratio, fluctuating asymmetry, and chemical signaling.

Singh (1993) reported that body fat distribution in women, as measured by waist-to-hip ratio (WHR), is correlated significantly with youthfulness, reproductive status, and long-term health risk. Men seem to have an evolved mechanism for attending to this information and preferring predictable variations, as they report women with low WHR (0.7), compared to women with higher WHR (0.8 to 1.0), as more attractive, healthier, and of greater reproductive value. Singh argues that WHR serves as a cue to men in solving the adaptive problem of mate selection.

A second purported cue to mate selection is fluctuating asymmetry (FA). Bilateral symmetry is hypothesized to be a marker of low parasite load, resistance to environmental stressors, and overall "good genes" (e.g., Gangestad & Simpson, 2000; Thornhill & Møller, 1997). As such, recognition of, and preference for, potential mates with low FA should be reproductively beneficial. Grammer and Thornhill (1994), for example, reported that when presented with computer-generated faces manipulated to display varying levels of FA, men reported that the female faces demonstrating low FA were more attractive and sexy. So, FA also appears to serve the function of mate selection.

The third mate selection tactic is chemical signaling. Major histocompatibility (MHC) genes are important for immune system functioning and benefit from being paired with dissimilar MHC genes. As such, an individual whose mate is MHC-incompatible (i.e., had MHC genes different from his or her own) would be more likely to produce offspring with stronger immune systems and a higher likelihood of survival. Consequently, one would expect a preference for mates with incompatible MHC genes. Wedekind, Seebeck, Bettens, and Paepke (1995) reported just that. Individuals preferred the odors, a cue to MHC genes, of opposite-sex individuals with incompatible MHC genes.

Evolutionary psychologists have proposed three separate proximate causes—WHR, FA, and chemical signaling—to account for one set of behaviors, mate selection. The question has therefore been posed: How is one mechanism to account for three distinct causes of behavior? Surely an individual who could rely on more than one piece of information would be at an advantage over one who had to rely on a sole source. Consider the cue of MHC genes in men's mate selection. The psychological mechanism may employ a decision rule such as "If odor indicates that MHC is incompatible, then consider as potential mate." Now suppose that same system also could process information about female WHR, a separate cue to mate value. The decision rule may then be "If MHC is incompatible, but WHR is too high, then discount as potential mate." Both WHR and MHC compatibility could serve as cues to mate value. Singh (1993) reported a similar phenomenon in the investigation of WHR described above in including female body mass index (BMI) as a separate indicator of female health and fertility. In Singh's data, men preferred women with low WHR, but preferred women of normal weight over underweight or overweight women, regardless of WHR. It appears that both WHR and BMI are cues to selecting a mate, with BMI "trumping" WHR. It is not unreasonable to propose that such decision rules may affect human behavior in this manner. Consider the

following analogy: If the most disconcerting thing in your present environment is the ant crawling on your knee, then attend to the ant on your knee. If, however, while attending to the ant on your knee, a tiger lunges at you from behind a boulder, then attend to the tiger.

5 An Example of an Evolved Psychological Mechanism Solidified During the Pleistocene Epoch

Interpreting human behavior in terms of the adaptive problems those behaviors solve can add insight into behaviors that would otherwise be difficult to interpret via proximate causes alone. For instance, recent research in the field of evolutionary psychology has focused on sperm competition in humans. Sperm competition occurs when the sperm of two or more males concurrently occupy a female's reproductive tract. There is an extensive literature supporting sperm competition theory in non-human animals such as birds and insects. Recent research has also lent support to the theory of sperm competition in humans. Shackelford et al. (2002) provided the first empirical evidence of male psychological adaptations to sperm competition in humans. They reported that men at a greater risk of sperm competition (as measured by proportion of time spent apart from their partners since last sexual intercourse), compared to men at a lesser risk of sperm competition, display motivations that would have functioned to increase the probability of success in sperm competition. Specifically, these men reported that they found their partners to be more attractive, they expressed greater interest in copulating with their partners, they believed that other men found their partners more attractive, and they believed their partners were more sexually interested in them. Shackelford, Goetz, McKibbin, and Starratt (2007) reported that men at a greater risk of sperm competition, compared to men at a lesser risk of sperm competition, display motivations that would have functioned to increase the probability of success in sperm competition. Specifically, men who spent a greater proportion of time apart from their partners (compared to men who spent less time apart from their partners) reported greater sexual interest in their partners, greater distress in response to their partners' sexual rejection, and greater sexual persistence in response to their partners' sexual rejection.

The researchers suggest that this is because the men who spend a greater proportion of time apart from their partners are at an increased risk of partner infidelity and subsequent sperm competition and cuckoldry (investing unwittingly in offspring that they have not sired). Men at a greater risk for sperm competition who exhibit these behaviors (e.g., greater sexual interest in their partners) are more likely to have sex with their partners sooner, thus entering their sperm into competition with possible rival sperm. An alternative explanation would be that these men are more interested in having sex with their partners as a result of a general sexual frustration. Because both studies found these partner-directed motivations to be unrelated to the total time since the couple last had sex, however, this alternative hypothesis remains unsupported. At this point, we are unaware of any supported theory, other than sperm competition, that can parsimoniously account for these adaptive patterned behaviors (for a comprehensive review of human sperm competition, see Shackelford & Pound, 2006).

6 Summary

This chapter provides a brief overview of the role that evolution plays in current human behavior. Evolution by natural selection shaped the development of species-typical psychological mechanisms. Those psychological mechanisms attend to stimuli specific to the adaptive problems of our ancestors and motivate behaviors that function to solve those adaptive problems. The specific nature of these behaviors is inextricably linked to both the environments of our ancestors and our own current environment. The specific nature of the psychological mechanisms themselves, however, is the result of the environments of our ancestors, and has been influenced very little by novel environmental factors emerging within the last 10,000 years.

Postscript: Counterpoint

The basic components of the human mind, or evolved psychological mechanisms, evolved to solve the numerous and specific problems of survival and reproduction that faced our ancestors. Because these problems and associated solutions are species-specific, they could not have been solidified before the emergence of the human species in the early Pleistocene, roughly 1.8 million years ago. There is nothing special about the end of the Pleistocene that demands the end of human evolution. However, because the time between the end of the Pleistocene and today is such a small portion of human existence (one half of 1%), it is unlikely that evolutionary pressures have had much of an impact on human design over the last 10,000 years.

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CHAPTER FOURTEEN

The Basic Components of the Human Mind Were Not Solidified During the Pleistocene Epoch

Stephen M. Downes

I argue against the view that the basic components of the human mind evolved during the Pleistocene Epoch. I make a case that human evolution is a process that began before the Pleistocene, carried on throughout the Pleistocene, and has continued since the Pleistocene. I review arguments against the Pleistocene mind view by behavioral ecologists, assess the implications of recent work in gene/culture co-evolution and human genetics for the view, and conclude by discussing the evolution of the human hand.

1 Introduction

There are a number of competing hypotheses about human evolution. For example, *Homo habilis* and *Homo erectus* could have existed together, or one could have evolved from the other, and paleontological evidence may allow us to decide between these two hypotheses (see, e.g., Spoor et al., 2007). For most who work on the biology of human behavior, there is no question that human behavior is in some large part a product of evolution. But, there are competing hypotheses in this area as well. Some claim that human behavior is produced by a collection of psychological mechanisms, for the most part, and that these mechanisms are adaptations that arose in the Pleistocene Epoch (e.g., Buss, 2007; Tooby & Cosmides, 1992). The claim is important and testable (although, more difficult to test than the above-mentioned hypotheses about origins); but importantly, it is only one among many hypotheses about the evolutionary origins of human behavior. While I think that there may be components of our behavior that are best explained by appealing to processes or mechanisms that arose in the Pleistocene, I think that human behavior is a result of evolutionary processes both

much older and *more recent* than the Pleistocene. I also maintain that much of human behavior, and the mechanisms underlying it, could still be subject to evolutionary change. The aim of this paper is to provide some arguments for the view that the basic components of the human mind have a long evolutionary history that stretches up to the present, and that will carry on into the future. In supporting this position, I will also provide some arguments against the thesis that the basic components of the human mind are a product of the Pleistocene Epoch.

At the outset, I need to make clear what I am *not* claiming. I do not challenge the view that human behavior has evolutionary roots and that evolutionary biology can be usefully brought to bear in explaining large amounts of our behavior. Some who challenge sociobiology and evolutionary psychology in all their many guises simply reject the thesis that our behavior is a product of evolution. Sometimes this rejection is founded on methodological issues. For example, Richard Lewontin (1998) espouses a particularly strong skepticism about the possibility of explaining human behavior in evolutionary terms, on the grounds that the relevant hypotheses are hard to formulate in a useful way and even harder to test. Others reject the thesis on the grounds that it requires genetic determinism, a position that many in the social sciences reject. I do not think that providing evolutionary explanations of our behavior requires an adherence to genetic determinism. I also do not reject the thesis that some of our behavior may result from evolutionary processes that occurred during the Pleistocene Epoch. It follows directly from the thesis I defend—viz., human behavior is the product of evolutionary processes long before, throughout, and after the Pleistocene—that some of our behavior could be a result of mechanisms produced by evolutionary processes during the Pleistocene. The thesis I reject is that the Pleistocene was the epoch during which *all* our important and defining psychological characteristics evolved.

Next, we need to get clearer about what exactly the relevant thesis claims as well as what variants of the thesis arise in the literature. The thesis is a driving theoretical claim in the work of Evolutionary Psychologists. David Buller (2000, 2005) distinguishes *E*volutionary *P*sychologists from *e*volutionary *p*sychologists and others who work in the biology of human behavior, for example human behavioral ecologists. *E*volutionary *P*sychologists defend a specific set of theoretical claims about the evolution and structure of the human mind and, hence, about the origin and explanation of human behavior. According to these researchers (represented by Valerie Starratt and Todd Shackelford in this volume), human behavior is best understood as the product of a large collection of psychological mechanisms, often called modules, each of which is an adaptation that arose in response to environmental challenges during the Pleistocene Epoch (also see Buss, 2007; Carruthers, 2006; Tooby & Cosmides, 2005). The period during which these psychological adaptations arose is also often called the Environment of Evolutionary Adaptedness (EEA), a term introduced by John Bowlby (1969, 1973), and later extended and carefully characterized by Donald Symons (1979, 1992). Starratt and Shackelford (this volume) make the following useful distinction:

> The EEA is not a place or a time in history, but a statistical composite of the selection pressures (i.e., all environmental characteristics influencing the ability of individuals of a species to survive and reproduce) operating on the adaptations that characterize a species' ancestral past. . . . The Pleistocene is the period of time hypothesized to contain the EEAs of the majority of human-specific adaptations.

In this volume, what is at stake in the debate is the time during which crucial evolutionary changes occurred in the human lineage, and from here on I will refer to the Pleistocene rather than the EEA.

There are a number of alternative forms that the relevant thesis—viz., the human mind is a product of one particular evolutionary period, the Pleistocene—can take:

1 A general version, whereby *all* components of the human mind are products of the Pleistocene. This version is clearly false and is agreed to be false by nearly all those working in the biology of human behavior, including Evolutionary Psychologists.
2 A trivial version, whereby all components of the human mind that arose as adaptive responses to challenges our human ancestors faced in the Pleistocene evolved in the Pleistocene. This is likely true, but uninformative and question begging.
3 A basic components version, whereby the key distinguishing components of the human mind, or the uniquely human components of the mind, arose in the Pleistocene. Suitably refined, this latter thesis is up for grabs empirically, and is the thesis rejected here.

In what follows, I introduce the relevant thesis and review human evolutionary history. I go on to review William Irons' arguments against the Pleistocene mind thesis, and then add some new examples and some new theoretical approaches to strengthen his arguments. Finally, I introduce an analogy that helps to reinforce the positive thesis of this paper, which is that human psychology is a product of evolutionary processes that took place long before, during, and after the Pleistocene, and will likely continue into the future.

2 The Pleistocene Mind Thesis and Our Evolutionary History

Here is Symons' (1979) account of the Pleistocene mind thesis:

> Large-brained hominids with advanced tool technologies have existed for more than one million years. . . . For over 99% of this period humans lived in small nomadic groups without domesticated plants or animals. This hunting and gathering way of life is the only stable, persistent adaptation humans have ever achieved . . . it is generally agreed that insufficient time has elapsed since the invention of agriculture 10,000 years ago for significant change to have occurred in human gene pools. . . . Humans can thus be said to be genetically adapted to a hunting and gathering way of life. (p. 35)

Here is another nice clear statement of the thesis at stake, from John Tooby and Leda Cosmides (2005):

> [T]he evolved structure of the human mind is adapted to the way of life of Pleistocene hunter gatherers and not necessarily to our modern circumstances. . . . The few thousand year since the scattered appearance of agriculture is only a small stretch in evolutionary terms, less than 1% of the two million years our ancestors spent as Pleistocene hunter-gatherers. (p. 5)

Both authors locate evolutionary change in our lineage in the Pleistocene, and both indicate that evolution is unlikely to have occurred *since* this period, for slightly different reasons.

At this point, it is worth briefly reviewing a little of the relevant evolutionary history at stake. The Cenozoic era, starting around 65 million years ago (mya), is divided into two periods: the Tertiary and Quaternary. The later Quaternary period is divided into two epochs: the Pleistocene, which runs from 1.8 (mya) to 10,000 years ago and is further subdivided into the Lower (.5-.25 mya), Middle (.25-.06 mya), and Upper (.06-.01 mya) Paleolithic, and the Holocene, which runs from .01 mya to the present. The first human ancestors, the Australopithecines, arose in the Pliocene, the last period of the tertiary quarter of the Cenozoic.

The standard story of human evolution as evidenced by the fossil record has the following highlights: at around 4.0 mya, bipedalism arises in the hominid lineage; at 2.0 mya widespread use of stone tools is found; at 1.6 mya *Homo Erectus* arises (*Homo habilis* is also dated to around this period);[1] at 1.5 mya some argue that *Homo erectus* uses fire; at .23 mya conservative paleontologists agree fire is controlled; at around .2 mya *Homo sapiens* arises; at .02 mya mil *Homo sapiens* are painting cave walls; at 0.01 mya the first permanent human settlements are formed; at 0.006 mya the first writing is produced. The evolution of brain size throughout this period is also instructive: From 3–4 mya *Australopithecus afarensis* exist with a cranial volume of around 450 cc; the later *Australopithecus africanus*, 3–2 mya, have a cranial volume of 500 cc; *Homo habilis*, 2.5–1.5 mya, have an increased cranial volume of 800 cc; *Homo erectus*, 2–.3 mya, have larger brains still inferred from their 900–1,200 cc cranial volume; and finally *Homo sapiens* have a cranial volume of 1,500 cc measured in specimens from .12 mya.

All the above changes in the human lineage are evolutionary changes of interest, and some can certainly be used to infer changes in our ancestors' psychology during this period. Evolutionary Psychologists emphasize the period from some time after the time stone tools emerged to the point at which the first settlements were developed as *the* crucial period of human evolutionary history from the point of view of the evolution of our psychological traits. What will be emphasized in the next two sections is that, although some important evolutionary events happened during this period, other periods of evolutionary history are just as important for our understanding of how human psychology arose.

3 Behavioral Ecologists' Response to the Stone Age Mind Thesis

William Irons (1998) presents several arguments against the thesis that the human mind is a product of evolutionary changes that occurred *only* during the Pleistocene Epoch. Irons' response is typical of others in his field of behavioral ecology[2] and, in part, reflects important methodological and theoretical differences between behavioral ecologists and Evolutionary Psychologists (cf. Downes, 2001).[3] First, Irons argues that Tooby and Cosmides' (1992) claim that our ancestors spent two million years as hunter gatherers is misleading. The paleontological evidence (briefly summarized above) reveals a large number of hominid species appearing and disappearing during the Pleistocene.

By the end of this period, *Homo sapiens* are established with their respective way of life, but this way of life may have varied greatly from that of other hominids during this period. Irons (1998) concludes that "saying that human beings were Pleistocene hunter-gatherers for one or two million years creates a false picture of stasis during this period" (p. 195). His next argument concludes that it is mistaken to assume that no evolutionary change could have happened in the 10,000 years of the Holocene leading up to the present. I will pursue this argument, in detail, in the next section and expand it to take into account some theoretical thinking that is different from Irons'.

There is a key difference between Irons' approach and that of Evolutionary Psychologists. Behavioral ecologists seem committed to the view that present fitnesses "caused the origin of an adaptation" (Irons, 1998, p. 196). In contrast, Evolutionary Psychologists argue that their approach, via the concept of the EEA, maintains a more reasonable stance toward accounting for adaptation: adaptations arise, via natural selection, in response to specific environments at specific times. The environment that early humans were exposed to during the Pleistocene was the environment that fueled the evolutionary changes that we now see reflected in our current suite of psychological mechanisms. The behavioral ecology approach, so this line of criticism goes, is committed to considering the reproductive consequences of current behavior and factoring this into an account of the adaptiveness of the behavior. This criticism could simply hinge on the distinction between traits that are *adaptations*, or products of natural selection, and traits that are *adaptive*, or those that fit the organism well to a specific feature of its environment (cf. Sober, 2000, p. 85). This distinction is part of what the critics have in mind.

The other dimension to the disagreement is that Irons, along with other behavioral ecologists, sees current environments as providing important explanatory insight into various behavioral, as well as morphological, traits. Irons counters by explaining that an adaptation need not be the same thing as providing the causal story of its origin. As a result, "if the form of an adaptation is stable for a very long time and the environment to which it is adjusted is stable, then measures of current fitness can play a logical role in explaining the maintenance of this adaptation" (Irons, 1998, p. 196). This is not all that Irons and behavioral ecologists are after. They clearly hold the view that evolution in human traits occurred before the Pleistocene, during the Pleistocene, and after the Pleistocene. Their methodology, which includes an emphasis on relations between behavioral traits and current fitness, permits both hypotheses about the stability of traits since the Pleistocene and hypotheses about subsequent evolutionary changes among humans.

Evolutionary Psychologists are committed to the view that all the important evolutionary changes that resulted in the suite of behaviors (and their underlying psychological mechanisms) that we now have occurred during the Pleistocene. The behavioral ecologists' response is that this position limits Evolutionary Psychologists' range of hypotheses about the evolution of human traits.

4 An Example and Some Alternate Theoretical Directions

In the previous section, I pointed out that Irons claims that evolution could have taken place during the last 10,000 years, and, of course, if this is right, then the

Pleistocene mind thesis could be undermined. He supports this claim with evidence from evolutionary changes in other organisms that have happened in much shorter time spans than this. But evolutionary rates in other animals need not be applicable to humans, and whether or not such rates of evolutionary change occur in nature is an empirical issue. The view that evolution occurred in humans in the last 10,000 years gains a great deal of support from work cited by Richerson and Boyd (2005) concerning human lactose tolerance. I first review this example, and then look at some of the implications of the example for the view that our minds are a product of the Pleistocene Epoch.[4]

The brief sketch of human evolution above reveals that permanent human settlements, evidenced by permanent dwelling structures and domesticated animals and plants, arose 11,000 to 10,000 years ago. Richerson and Boyd argue that during the period since the domestication of animals, human lactose tolerance evolved. The way in which this occurred on their view was via gene/culture co-evolution. The actions of humans in domesticating animals and living with them produced a new selection pressure, and that cultural selection pressure contributed to the increased fitness of individuals who could digest lactose. This trait emerged in humans after the Pleistocene Epoch.

First, we should note that lactose tolerance is not a psychological mechanism, but is a clear case of a human specific evolutionary change that has occurred recently. So while the example is not a case of a human psychological mechanism arising after the Pleistocene, it is a case of evolutionary change occurring at a more rapid rate than that assumed by Evolutionary Psychologists and the relevant change occurs in the human lineage. Second, it is important to understand the mode of evolutionary explanation that Peter Richerson and Robert Boyd explicitly adopt: gene/culture co-evolution. In the previous section, we saw that behavioral ecologists emphasize different aspects of evolutionary explanation than Evolutionary Psychologists and that this, in part, leads to the disagreement between them over the evolutionary relevance of the Pleistocene Epoch. Analogously, Richerson and Boyd's (2005) commitment to a different theoretical framework leads them to emphasize different explanatory aspects of evolutionary theory.

In arguing against the approach of Evolutionary Psychologists, they maintain: "[C]ulturally evolved traits affect the relative fitness of different genotypes in many ways" (p. 193), and this leads them to oppose the view that "cultural evolution is molded by our evolved psychology, but not the reverse" (p. 194). The target here is the evolution of specific human forms of cooperation and moral norms. Boyd and Richerson argue that Evolutionary Psychologists, via the Pleistocene Epoch thesis, are committed to a view that human forms of cooperation and moral norms arise as a result of psychological mechanisms that were adaptations in earlier humans, which arose as a response to the challenges of small-group living in the Pleistocene environment. Here is Tooby and Cosmides' (1992) version of that point:

> [T]hings that are cultural in the sense of being organized, contentful, and shared among individuals may be explained in a number of different ways. Within-group commonalities may have been evoked by common circumstances impacting universal architectures. An even larger proportion of organized, contentful, and shared phenomena may be explained as the expression of our universal psychological and physiological architectures in interaction with the recurrent structure of the social or non-social world. (pp. 117–118)

In contrast, Richerson and Boyd's view opens up the possibility that, as various new human cultural formations arose, they played an important role in producing subsequent changes in human psychological makeup via gene/culture co-evolution.

A related line of evolutionary thinking to Richerson and Boyd's gene/culture co-evolution view is niche construction (also see Laland & Brown, 2002; Laland, Odling-Smee, & Feldman, 2000). Rather than focusing exclusively on relations between human cultural products and subsequent evolutionary change, niche constructionists point to the way in which many organisms actively construct aspects of their own niches and, as a result, change the selective environment that impacts upon them (examples are abundant, but include nest building, dam building, and the building of colony dwelling structures). In Kim Sterelny's (2003) hands, human downstream niche construction provides numerous ways in which to impact upon evolutionary change in the human lineage, specifically with respect to the production of novel behavioral repertoires. Gene/culture co-evolution theorists and niche constructionists are more inclined to think of the Holocene as the period during which the most and the most rapid evolutionary change occurred in the human lineage, as opposed to the view that no evolutionary change occurred in this period.[5] As a result, these theorists reject the thesis that the human mind is a product of evolutionary processes that occurred during the Pleistocene.

5 An Instructive Analogy: The Evolution of the Human Hand

There are many obvious reasons why we focus a great deal of attention on the evolution of human psychological traits. The wide variety of complex cognitive capacities that our minds support are, in large part, responsible for the huge array of unique behaviors we have come up with in our massively varied and challenging environments. But, there are other traits that enable us to get ahead in our complex world. If we were brains in vats, we wouldn't be doing much running, throwing, fishing, sewing, and even playing chess. I contend that the way in which we approach the explanation of the evolution of human behavior and human mental capacities should be consistent with the way in which we approach the evolution of any trait or cluster of traits. A brief reflection on an account of the evolution of the human hand illustrates the points that I want to make here.

Our hands allow us to perform many tasks that our nearest primate relatives are unable to do: sewing, making fish hooks and fishing nets, tying knots, playing instruments, rolling cigarettes, etc. Our hands evolved from mammal forefeet, and their basic structure owes a lot to this ancestry. The hand did not evolve in response to a particular environmental stimulus at any particular time. Rather, various selection pressures, including bipedalism, the occupation of niches with widely varying food resources, and our own niche construction, led to the musculature and bone structure that supports the range of activities for which human hands can be used. One hypothesis about a component of human hand evolution is that it is driven, in large part, by the activities of throwing and clubbing. The idea is that throwing and clubbing pre-date stone tool use, and the throwing and clubbing grips are present in all humans from *Australopithecus* onwards but in no ape lineages (Young, 2003). This account,

if correct, takes care of some of the specific morphological and functional attributes of the hand, but by no means all. If we had hands adapted for clubbing and nothing more, we would have a hard time repairing fishing nets with a small bone needle (or making the needle in the first place). The throwing and clubbing account does, however, present a useful hypothesis about an important part of our evolutionary history, the point at which our lineage diverged from that of the rest of the great apes.

And this account also illustrates the point that, in giving an evolutionary account of the suite of traits an organism has, many periods of evolutionary history are relevant; some in quite deep evolutionary time, and some in very recent evolutionary history. There is no *one* evolutionary period in which a set of adaptive problems arose that hands had to face, and I would argue, analogously, that there is no *one* evolutionary period in which the suite of adaptive problems arose that shaped our minds.

Obviously, our brains are far more complicated than our hands; but a similar story should be told about their evolution. Rather than one evolutionary period providing a source of specific selection pressures that produced local adaptive response in brain machinery, the brain's evolution was driven by bipedalism, increased and varied food resources, leading to more use of vision, competition between con-specifics, the need for strategic food gathering (e.g., coordinated hunting), larger group size, and more complex cooperation and coordination problems, including tracking dominance hierarchies, and so on. Crucially, also, the brain's evolution is determined, in part, by the evolution of the hand. The more fine-grained motor skills our hands are capable of, the more monitoring of these skills is required by the brain. The full story of the evolution of the human mind will include components that account for our lineage's divergence from that of the rest of the great apes and components that account for our predictable psychological responses to aspects of large highly complex social groups. Components of this account will come from various times in our evolutionary history, including the Pleistocene, but also including times long before and long after this period.

6 Conclusions

Here, I have not contended that evolution is *irrelevant* to our understanding of our mental capacities. Rather, I have presented some arguments that cast doubt on the view that our minds are a suite of adaptive mechanisms which arose *only* during the Pleistocene Epoch. The broader context for this conclusion is that a complete account of human evolution will be drawn from numerous research programs in the biology of human behavior. One such program is Evolutionary Psychology, but the contribution of this research program to understanding human evolution is limited by an adherence to the thesis that our minds are a product of the Pleistocene Epoch. Actually, more is gained by rejecting this thesis than by retaining it.

Postscript: Counterpoint

Evolution of any trait occurs only if there is available variation. Variation in beak shape in finches or variation in running speed in zebras, as long as this variation results in a variation in fitness, leads to selection of one of the variants over

others. The relevant variation must also be heritable. One important way in which variation can be heritable is when genetic variation underlies variation in traits. What distinguishes the position I support above from Starratt and Shackelford's is that I maintain that there is still a significant amount of genetic variation in humans. As a result of this, it seems reasonable to argue that there can have been recent evolutionary changes in human traits, including psychological traits. Starratt and Shackelford disagree. They say:

> Additionally, geneticists investigating human genetic diversity report that 80% of all genetic differences are among individuals within the same population. In contrast, variations among populations of different continents account for only about 10% of all genetic differences. This suggests that most genetic variation occurred before modern humans migrated out of Africa roughly 100,000 years ago (see Owens & King, 1999, for a brief overview). If the bulk of our species' genetic makeup has remained relatively constant over the last 100,000 years, it is not unreasonable to argue that our psychological design (which is built by our genes) has remained relatively constant over the last 10,000 years.

Who is right?

In a sense we are both right. John Hawks et al. (2007) report a fair amount of recent genetic variation, some of which they argue is adaptive. They do not have to demonstrate that all human genetic variation arose after the Pleistocene to show that there is potential for the evolution of new traits. Rather, they merely need to present evidence of significant genetic variation occurring in recent years. My argument above is that not all significant evolution of human psychological traits took place in the Pleistocene. The way this discussion impacts on the argument is that it helps establish that the claim that there was human evolution *after* the Pleistocene is a reasonable one to make. The research project of Evolutionary Psychology can be expanded in exciting ways if these new findings in genetics help us discover the evolutionary origins of some of our psychological traits.

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Notes

1 This gives rise to the dispute mentioned in the introduction and discussed in Spoor et al. (2007).
2 Sarah Hrdy (1999) provides similar criticisms of the EEA concept in her discussion of the evolution of human motherhood.
3 The presentation here closely follows the presentation in my earlier piece on this topic (Downes, 2001).
4 For many more examples of recent human evolution, see Hawks, Wang, Cochran, Harpending, & Moyzis (2007).
5 Hawks et al.'s (2007) data strongly support this line of thinking.

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PART VIII

DOES MEMETICS PROVIDE A USEFUL WAY OF UNDERSTANDING CULTURAL EVOLUTION?

Introduction

Richard Dawkins, also known as "Darwin's Rottweiler," is a famous living biologist who has a Ph.D. in zoology. His ideas have contributed to forming several of the basic topics found in the philosophy of biology as they exist today, and he has been an important source for popularizing and clarifying many of the principles surrounding evolutionary theory (Grafen & Ridley, 2006; Hall, 2005). For example, in his book titled *The Selfish Gene* (1976) he coined the term "meme" for any copied thought or behavior. The idea surrounding memes goes something like this: just as organic traits can evolve by principles such as variation, inheritance, population increase, struggle for existence, differential survival, differential production, and natural selection, so too, according to Dawkins, culturally learned thoughts and behaviors such as beliefs, melodies, catch-phrases, trial-and-error activities, and various other kinds of technology can analogously evolve by many of these same principles. This evolutionary (and revolutionary) idea has generated an entire research program in memetics (for example, *Journal of Memetics: Evolutionary Models of Information Transmission*: http://jom-emit.cfpm.org/) which has intersected with areas such as information theory, linguistics, behavioral psychology, and philosophy of mind.

But, does memetics provide a useful way of understanding cultural evolution? In the first paper in this part, Susan Blackmore answers yes to this question, arguing for memes as a kind of second replicator (for more on genes as replicators, see Part IV

of this book). She maintains: "Criticisms of memetics are often based on trying to draw too close an analogy between genes and memes: it is better to think of genes as an ancient replicator that is now copied extremely efficiently, and memes as a new replicator which is copied by a variety of more or less successful copying methods." For example, in Blackmore's opinion, Peter Richerson and Robert Boyd (2005) have drawn the analogy too closely, thus inappropriately concluding that memes do not exist or are useless.

Taking a different tack, in his paper contained in this part William Wimsatt responds to Blackmore by noting that there are many other factors that contribute to cultural evolution, including what he calls cultural scaffolding, and cultural infrastructure, as well as other learning and cognitive processes. Thus, Wimsatt wants to downplay memes and argue that they are not the primary drivers of cultural and technological evolution; rather, memes are parts of a larger multi-component cultural system. Blackmore responds that these other factors mentioned by Wimsatt, along with the memes, are "the inevitable co-evolution of a replicator with its copying machinery." In the end, both authors believe that there is not yet a thriving science of memetics; however, Blackmore definitely seems more optimistic that such a science is possible.

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CHAPTER FIFTEEN

Memetics Does Provide a Useful Way of Understanding Cultural Evolution

Susan Blackmore

Memetics is a theory of cultural evolution based on the idea that behaviors, skills, habits, stories, and technologies that are copied from person to person in culture act as a second replicator. That is, they are information that is copied with variation and selection, and they therefore sustain a new evolutionary process, both cooperating and competing with the old. Like genes, memes are selfish replicators that spread for their own benefit, not necessarily for the benefit of the people who copy them, or the culture they form a part of. Unlike genes, they are a new replicator that is copied with varying degrees of fidelity and by various more or less reliable processes, including behavioral imitation, teaching, and many new methods of copying using modern technology.

Criticisms of memetics are often based on trying to draw too close an analogy between genes and memes: it is better to think of genes as an ancient replicator that is now copied extremely efficiently, and memes as a new replicator which is copied by a variety of more or less successful copying methods. I discuss ways of defining and categorzing memes, difficulties with units and notions of "memotypes," sources of variation, and questions of whether memes are really reproduced or replicated. The major difference between memetics and other theories of cultural evolution is that for other theories cultural traits are usually treated as aspects of the human phenotype, with the genes having ultimate control. In memetics, they are treated as a new replicator that uses the vehicles constructed by the old replicator (genes) as their copying machinery. Memes need not be subservient to genes, but evolve alongside them, using their vehicles, whether symbiotically or parasitically. The important point is that memes evolve for the sake of memes, just as genes evolve for the sake of genes.

I discuss viral memes such as chain letters and religions, and the evolution of parasitic memes; and consider how the co-evolution of memes and genes has shaped human evolution. I argue that humans became different from all other species when they began to imitate and hence let loose the second replicator, memes. From then on the process of memetic drive, by which evolving memes drive genes to produce brains that are better at copying those memes, was responsible for the design of the human brain, language, and cultural capacities. With modern information technology we are on the brink of letting loose a third-level replicator which will also spread selfishly, using us, and our culture, to do so.

1 Introduction

Imagine a planet on which a self-replicating molecule appears, is copied with variation and selection, and evolves to build itself living creatures that protect and propagate it; those creatures eventually spread all over the planet, changing its atmosphere and environment as they do so. Now, imagine that one of those creatures begins to copy behaviors from one individual to another, and this information gets copied with variation and selection. In other words, a second kind of replicator emerges. This transforms the original creatures to make them better at protecting and propagating the second kind of replicator, and goes on to build cultural objects that eventually spread all over the planet, changing its atmosphere and environment all over again.

This is the vision that memetics provides: that human beings, alone on earth, are the product of two replicators, not one. In consequence, they are *meme machines* as well as gene machines, and culture uses them as its replication machinery. Language, music, and technology are all parasites, often turned symbiotic, that evolved for their own sake, not for the sake of individual humans or their genes.

This vision is different from most theories of cultural evolution or gene/culture co-evolution, which generally treat genes as the final arbiter and culture as an adaptation or an aspect of the human phenotype. Whether this new vision of human and cultural evolution is valuable or not is the question for this debate.

2 Origins of the Meme *Meme*

The term *meme* was coined by Richard Dawkins in his 1976 book *The Selfish Gene*. His intention was not to create an analogy with genes but, rather, to illustrate the principle of universal Darwinism and his contention that "Darwinism is too big a theory to be confined to the narrow context of the gene" (p. 191). *Universal Darwinism* is the general principle that whenever you have information that is copied with variation and selection, then you must get evolution. Darwin (1871) applied this process to languages as well as evolving organisms, and universal Darwinism has a long history (Plotkin, 1993). The process has also been described as "blind variation with selective retention," a phrase used by Campbell (1960) to describe the evolution of knowledge or "evolutionary epistemology" and now used mainly in cybernetics. The three steps—viz., variation, selection, and heredity—can be understood as a simple algorithmic process, the evolutionary algorithm, or, in Dennett's (1995) words, "a scheme for creating Design out of Chaos without the aid of Mind" (p. 50).

Dawkins called the information that is copied in such a process the *replicator* and contrasted this with *vehicles* (such as living organisms) that carry the replicators around, protecting and propagating them. In a slightly different scheme, Hull (1988) refers to replicators, interactors, and lineages. Dawkins (1976) wanted "to claim almost limitless power for slightly inaccurate self-replicating entities, once they arise anywhere in the universe" (p. 322). His point was that genes are just one example of a replicator, and the general principle is more important than the specifics of the way genes have evolved on earth. This is why, at the end of the book, he asked his now famous question, "do we have to go to distant worlds to find other kinds of replicator and

other, consequent, kinds of evolution?" (p. 192). His answer was, of course, no. Staring us in the face, drifting about in its "primeval soup" of culture, is a new replicator: tunes, ideas, catch-phrases, fashions, and ways of making things. These show heredity (they are copied from person to person), variation (for example, through errors in transmission or memory, and through recombination), and they are selected (people choose what to copy and from whom). So they must be replicators. He wanted a name that would sound a bit like gene while conveying the central meaning of "that which is imitated," so, from the Greek *mimeme* (something imitated), he took the abbreviation *meme*.

The term *meme*, itself, has been a successful meme, spreading widely, and being accepted into the *Oxford English Dictionary* in 1997. The science of memetics, however, has not grown correspondingly. The reasons for this are not clear. Memetics has some potential advantages over other theories: for example, in how it deals with informational viruses such as email viruses, cults, and religions, and in its novel approach to the evolution of language, the big brain, and the co-evolution of genes and culture. Nevertheless, there is no thriving science of memetics, neither are there specialist journals or conferences. Perhaps memetics is just plain wrong; perhaps it is currently too difficult to test its predictions; or perhaps its implications are too scary or unappealing for people to accept. Certainly part of the problem is that memetics is frequently misunderstood. I shall, therefore, begin by discussing some of the most common confusions before turning to the value of memetics itself.

3 Do Memes Exist?

This question, although often asked, reveals a misunderstanding of the concept of a meme. McGrath (2005) complains that there is "no direct evidence for the existence of 'memes' themselves" (p. 121), and Aunger (2000) states that "their existence has yet to be proven" (p. 7). Wimsatt (1999) claims they are "both misnamed and mischaracterized, and perhaps even . . . do not exist" (p. 280). Yet, as Laland and Odling-Smee (2000) point out, "the pertinent question is not whether memes exist, as suggested by Aunger . . . but whether they are a useful theoretical expedient" (p. 121).

To see why the existence question is misplaced it is only necessary to remember how the idea was first conceived, that is, to think of skills, habits, words, or stories as replicators. There is nothing hypothetical about teenagers listening to iPods, wearing pre-torn jeans, or piercing their eyebrows after seeing others do the same. There is no question about the existence of money, railways, bicycles, telephones, furniture, skyscrapers, holiday brochures, football, or the days of the week. They are all information encoded in some kind of matter and energy, and they can all be copied or not. The core definition of memes is "that which is imitated" or that which is copied. It may be difficult to pin down just what has been copied but, whatever it is, that "something" is, by definition, a meme.

Accepting that memes exist does not, of course, mean that memetics is a good idea. To give a fanciful equivalent, imagine that I decided to take the class of all city streets that have plane (sycamore) trees in them and call them *plemes*. I could then go around counting the number of plemes in Bristol, or London, or the whole world. I could investigate whether there are more houses, or more cats, or more deaths in plemes,

as opposed to other streets. I doubt I would find out anything interesting, that is, unless there is truly something peculiar about that beautiful bark, or a poison in the leaves. My guess is that the science of plemetics would be a complete waste of time: not because plemes don't exist—they do—but because dividing the world up this way reveals no general principles, gives rise to no new insights, and is completely pointless.

Memetics might be like this, completely pointless, or it might not. So the existence question is a distraction. Memes exist. The interesting questions concern just what we should and should not count as memes, whether they really behave as replicators, whether they are inside or outside brains or both, how close the analogy with genes proves to be, and whether memetics gives rise to any useful theoretical or practical progress. Those are the questions I shall briefly consider here.

4 Trouble with Analogies and Units

It should be clear from the origin of the term *meme* that it was not primarily conceived as a gene analogue. Yet, this is often falsely assumed and then used as an argument against memetics. For example, Gould dismissed memes as "a useless analogy" and Midgley claims them to be "a meaningless metaphor" (Blackmore, 1999). McGrath (2005) goes further and claims that the "case for the existence of the 'meme' rests on the questionable assumption of a direct analogy with the gene, which proves incapable of bearing the theoretical weight that is placed upon it" (p. 121). Further, Richerson and Boyd (2005) argue against memetics on the grounds that "the best evidence suggests that cultural variants are only loosely analogous to genes" (p. 60).

Such criticisms are common, the argument often going something like this: memes were invented by analogy with genes, genes are real physical entities made of DNA, and no equivalent physical entity can be found for memes; therefore, the notion of memes is false (or is useless, or memes don't exist). But this misses the point of Dawkins' reason for inventing the term, which was to apply universal Darwinism and treat cultural information as a replicator. When understood this way, it becomes clear that analogies between genes and memes are secondary, and must be treated with care because the two replicators are different in significant ways.

So I prefer to see it this way: genes are replicators, and we know a great deal about how they work. Memes are replicators, and we know rather little about how they work. This means that some analogies will be fruitful because memes and genes are both replicators and, therefore, will show some profound similarities, but other analogies will be false because, in many ways, the two replicators are so different. For example, they use entirely different copying mechanisms, their sources of variation are quite different, and they differ in fidelity, storage, and in many other ways. Also, one replicator has been evolving along with its replication machinery for approximately four billion years and has settled down to a very high fidelity system with (often) separated germ-line and phenotypes; the other has been around for at most a few million years and is, as Dawkins (1976) put it, "still in its infancy, still drifting clumsily about in its primeval soup" (p. 192).

I, therefore, agree with McGrath (2005) that "analogies can be dangerously misleading." (p. 132) and with Wimsatt (1999), who points out many dissimilarities between genes and memes, and their relevance for attempts to model cultural evolution. These

differences mean that we must treat gene–meme analogies with great care, using them where appropriate to build new hypotheses or explore possibilities, but not relying on them as a guide to how memes work.

Another source of confusion is the problem of units. Memes are often described as "units of culture" or "units of imitation" (although some definitions avoid the word by talking about "elements of culture," "contagious ideas," or "cultural instructions"). Some critics have made the problem of units into a major objection to the whole of memetics. For example, Midgley (2000) complains that culture cannot be neatly divided up into units, and nothing is to be gained by "atomizing thought," since "thought is not granular" (p. 67). Similarly, Jablonka and Lamb (2005) argue that, in cultural evolution, there are no "discrete unchanging units with unchanging boundaries that can be followed from one generation to the next" (p. 212), as though that disqualified the concept of memes.

Even stronger are the objections made by Richerson and Boyd (2005) to the idea that "culture must be divisible into tiny, independent gene-like bits that are faithfully replicated" or "tiny snippets of information" (p. 60). However, in defining memes as "discrete, faithfully replicating, gene-like bits of information" (p. 6), they are departing far from the original definition. Some memes are discrete and some are not; some replicate with very high fidelity (e.g., printed text) and some do not (e.g., dance steps); some memes are like genes in some ways and not in others. Going back to the original idea of memes as information that is copied, or "that which is imitated," helps to avoid these unrealistic claims.

To some extent, it is true that proponents of memes describe them as units, but this is at least partly because it is so much easier to talk about entities than to talk about "that which is imitated," in the abstract. Just try rewording some simple sentences about memes without inadvertently implying units: "Fax machines copy memes more accurately than telephones do." "Soduko spread rapidly across Northern Europe," or "The meme for texting is doing better in Britain than in the USA." All these make sense, and may seem to imply units, but in no case need there be precise units, let alone "discrete unchanging units with unchanging boundaries" or "independent gene-like bits."

Dennett (1995) tackles the problem by defining the units of memes as "the smallest elements that replicate themselves with reliability and fecundity" (p. 344), or as a cultural item "with enough Design to be worth saving—or stealing or replicating." (p. 143). In art, for example, a whole gallery of works is too large a unit for selection pressures to work on; but a blob of pink paint is too small to be selected, enjoyed, disliked, photographed, or thrown away. The single painting is the natural unit, as when we remember Picasso's *Guernica* or buy posters of Monet's *Water Lilies*. Styles of painting, such as Cubism or Impressionism, can also be copied and can, therefore, count as memes; but they can hardly be divided up into units and would be much harder to track than an individual painting. Nonetheless, this does not mean the concept is useless. Similarly, with written text, a single word is too short to copyright and an entire library too long, but we can and do copyright three-word advertising jingles and 300,000-word books. Either can count as memes because either can be copied with sufficient reliability and fecundity for selection to operate.

The same applies to music. Everyone's favorite example is the first four notes of Beethoven's Fifth Symphony, which have spread as though they have an independent

existence, all across the globe, in cultures where the whole symphony is scarcely heard. Wilson (1999) asks:

> What do we gain by thinking of the first four notes of Beethoven's fifth symphony as a powerful meme? . . . The ability to define fitness independently of what evolves saves the concept of natural selection from being a tautology. For the meme concept to escape the same problem, we must define cultural fitness independently of what evolves. If the first four notes of Beethoven's Fifth is a powerful meme only because it is common, we have achieved no insight. (p. 206)

I agree. So what is fitness for a short melody? It is the ability to survive and reproduce, which, in terms of music, means being copied, stored, and reproduced more frequently than other melodies. This ability certainly ought to be measurable independently of how common the melody is.

Inspired by Wilson's criticism, Larry Bull, Nick Rose, and I tried to find out. We chose the first four notes of three other well-known symphonies as comparison, and tested children aged between four and eight hoping to catch them before they were familiar with this music. They could press four keys to play any of the tunes in any order and as often as they liked until they got bored, and we asked them which they liked best and which they had heard before. We predicted that they would play the Beethoven more often than any other (thus, giving it more chances of being copied), even if they did not like it best. The experiment entirely failed because we could not find children old enough to do the task but naïve to the music. Indeed one five-year-old said he knew all of the tunes because he'd been to see *Peter and the Wolf*.

Nevertheless, the principle remains that, as happens in biology, it should be possible to measure the ability of memes to survive and reproduce independently of how common they are, to understand their behavior, and to predict how they will perform in new environments. Studies of this kind have been done, for example, in such widely different areas as the competition between different chemical terms in Western science and in China (Wright, 2000), the survival of replicating text in the Internet (Pocklington & Best, 1997), and the competition between different kinds of notation in music (Gersh, 2007). Although not using the concept of memes, Wimsatt (2006) has explored the evolution of Punnett squares as a technique for visualizing problems in genetics. He treats the squares as model organisms for studying cultural evolution, and shows how the way they exploit human cognitive capacities helps them to attain high scientific fitness and rapid evolution. There are also many studies of social contagion, including suicide contagion and even the spread of obesity through social networks (Christakis & Fowler, 2007). Such studies show that the problems of defining units and drawing analogies do not prevent effective research.

5 What is a Meme?

The literature is full of arguments about what counts as a meme, as well as lists of examples, including ideas, behaviors, objects, and brain states. Dawkins (1976, 1982, 1993, 1999) mentions habits, skills, and stories, and Dennett (1995) lists "arch, wheel, wearing clothes, vendetta, right triangle, alphabet, calendar, the *Odyssey*, calculus, chess,

perspective drawing, evolution by natural selection, impressionism, 'Greensleeves', and deconstructionism" (p. 344). Wimsatt (this volume) lists as "meme-like things", artifacts, practices, and ideas which are taught, learned, constructed or imitated. He adds five further things needed for a theory of developmental cultural evolution: individuals, institutions, organizations, structures, and finally the scaffolding that includes the entire infrastructure of our technological civilization.

Can a useful theory really be built out of such a curious assemblage? I agree with Dennett that it can. The way to see what unites its constituents is to return to the origin of the term *meme* as "that which is imitated" or, more generally, "that which is copied." An easy way to decide whether something is a meme or not is to ask whether it was copied from, or to, someone else or was learned or created some other way. If it was copied from person to person, or person to book, or computer or artifact, then it's a meme; otherwise, it's not. In this light, Dennett's examples make sense. All of them (with the possible exception of wearing clothes) are words, concepts, technologies, skills, or stories that are copied from person to person. They have all evolved over varying lengths of time and are survivors in the competition to be copied and stored.

I mention wearing clothes as a possible exception because different people might independently discover this useful ability, rather than copy it from each other. In that case, it would not count as a meme because no information has been copied from person to person. Alternatively, they might pick up the general idea, but then use totally different things as clothing. Culture is full of examples of this kind, such as the famous case of Sequoyah, the Cherokee Indian who reputedly invented the Cherokee syllabary after seeing Europeans reading and writing (see, for example, Bender, 2007). In this case, trying to pin down the meme (or what exactly was copied) is probably impossible, but the principle remains clear that Sequoyah created his writing system because he had seen a previous one, and so writing spread through many more people than it otherwise would, having effects on a whole society as it did so.

To give more modern examples, many learned skills entail a complex mixture of individual learning and memetic transmission. Riding a bike means watching someone else do it, but then one has to learn the physical skills of balance, steering, and braking for oneself through operant conditioning. The same can be said of driving a car, gardening, cooking, surfing, using a word processor, or painting a portrait. None of these things can be done without the evolved scaffolding provided by the complex society in which we live (Wimsatt, 1999). The bike is itself a meme (or complex of memes), copied by diverse processes involving raw materials, technology, other people, and factory processes; riding a bike is a meme, copied by one person seeing another doing it and wanting to do the same, but the physical skill is acquired by individual practice and is not a meme.

Does this differ from other theories of cultural evolution, and is there any point in thinking memetically here? The difference is that most theories of cultural evolution treat cultural traits as aspects of the human phenotype that are ultimately under the control of genetic evolution, while memetics treats all of the copied information and its products as selfish replicators, spreading for their own sake. Memes are unlike genes in many ways, but are like them in being information that replicates selfishly, using the resources of human individuals and society to do so. The bikes, cars, gardens, kitchens, surf boards, computers, and paint brushes are all there because

they have won in the competition to get humans to copy them. They, in turn, provide the selective environment in which more memes evolve.

This relates to an interesting debate in memetics over the extent to which memes are really copied as opposed to being reconstructed by the imitator. Jablonka (1999) points to the distinction between reproduction and replication of behaviors, and Sperber (2000) argues that a "fundamental objection to the meme model" is that most cultural items are "re-produced" in the sense of being produced many times, but are not "reproduced" in the sense of being copied from one another (p. 164).

Sperber gives the example of two drawings, one a more or less random scribble, the other a slightly wonky-five pointed star. What happens when people copy them? The first is hard to copy and results in a chain of copies in which each one differs slightly from the previous one. The second is easy to copy because people recognize it as a familiar shape and can re-produce (i.e., produce again) a shape they have seen and drawn many times before. As Dawkins (1999) points out, you could ask an independent observer to try to put the copies into the order in which they were made. In the first case, they would be likely to succeed, in the second case they would not. The second case is "self-normalizing." Sperber sees this as a serious objection to memetics because the drawing is not actually copied but is "re-produced," whereas Dawkins sees it as an example of the increasing fidelity of transmission as people learn to draw and to think about familiar shapes.

6 A New Replicator or Culture on a Leash?

Questions about reproduction and copying are important because of the central claim of memetics that imitation creates a new replicator (i.e., information copied with variation and selection). It may help to think in terms of a continuum. At one extreme are events that are pure re-production. Sperber gives the example of laughter, which is not a meme. Laughing when someone else laughs, like other examples of contagion (e.g., yawning or smiling), involves no copying. The second person's laugh is triggered by hearing the first, and is their own laugh, not a copy of the way the first person laughed (that is, unless they are deliberately mimicking for fun). Contagion occurs in other species, too, and can be useful, as when one individual picks up a warning cry from another, or flees because another does. This is entirely triggered re-production, not meme replication.

Then, there are examples in which a combination of individual learning and social learning (but not true imitation) end up in cultural transmission that is not memetic. The classic example, here, is the pecking of milk bottle tops by tits in England. This novel behavior was tracked from its beginnings, in several independent locations, in the 1940s (Fisher & Hinde, 1949). Although the behavior clearly spread from one bird to another, this is thought to have been by stimulus enhancement, and not true imitation (Sherry & Galef, 1984). That is, one bird discovers, by luck or accident, that there is cream underneath the foil bottle top. This bird then leaves a jagged, shiny foil top which is highly visible to other birds, so they come to investigate, thus learning that foil tops are associated with cream. They then land on unbroken foil tops and do what tits always do: peck, and find more cream. And, so, the behavior spreads. Is this a meme? No, because there is no copying with variation and selection and,

so, no replicator and no cultural ratchet effect (Tomasello, 1999). The tits do only what they always do: peck for food. All they have learned is to associate foil tops with food.

At the other extreme, consider learning to dance. At first everything is unfamiliar: the steps, the rhythms, the precise sequence of movements all have to be watched carefully and then reproduced. Trial, error, self-assessment, and hard work all result in the student being able to dance the same steps as the teacher. By the time the student is an accomplished dancer, new dances can be learned, easily. This becomes more like the five-pointed star than the random scribble. The student has learned all sorts of routines that can be mixed and matched to make new dances. By this stage, the process may look quite different, but, if anything, it more clearly reveals the principle of replicators at work.

Richerson and Boyd (2005) disagree, stating emphatically that "cultural variants are not replicators" (p. 82), thus effectively rejecting memetics. Part of their argument is that the peculiarities of biased transmission, behavioral attractors, and error-prone imitation are reasons to reject the idea of memes as replicators. However, I suggest that these only show how complex memetic transmission must be. Their other contention is that copying must be perfect for a replicator to count as such, and imitation does not produce perfect copies. But this makes no sense. If copying were perfect, there would be no variation and no evolution. More interesting is to ask how high the fidelity has to be for an evolutionary process to get off the ground. If human imitation is good enough to support cumulative evolution, then we should be justified in treating memes as replicators. And, clearly, whatever its shortcomings, it is.

Whether memes are replicators, or not, is a crucial question. Although Richerson and Boyd's theory is very close to memetics in some respects—for example, they occasionally use the term "selfish meme" and describe us, and our culture, as like obligate mutualists—they still maintain that "culture is an adaptation" and that "culture is on a leash, all right," even if the dog on the end is big and clever. This refers to Lumsden and Wilson's (1981) famous claim that "genetic natural selection operates in such a way as to keep culture on a leash" (p. 13).

Here, we see the fundamental difference between memetics and other theories of gene/culture co-evolution. For most theories, culture is an adaptation of benefit to human genes and, ultimately, kept under control by them (i.e., on a leash). For memetics, memes are not (and never were) an adaptation; they are a new replicator that was accidentally let loose by the human capacity for imitation. In 1976, Dawkins complained of his colleagues that "In the last analysis they wish always to go back to 'biological advantage' " (p. 193). This is what Richerson and Boyd wish to do and why their theory, although close, is fundamentally different from memetics.

7 Do Memes have Memotypes?

In asking what counts as a meme, I listed many widely differing examples, and these raise another important question: are memes inside brains, outside of brains, or both? For example, is the meme for "Cinderella" supposed to be the story itself, the spoken words, the written words, or their representation inside people's heads? Should we

take an analogy from genes and count the representation inside someone's head as the actual meme, and the written story as something like a meme phenotype, or vehicle, or interactor? Wimsatt (1999) asks: "How would we know when a meme corresponded to a gene? A chromosome? A gamete? A genotype? Or the gene pool or pools of a family? Or even of a whole ecosystem?" (p. 284). Or, are these all false analogies?

Proponents and critics have argued each way on this one, and no consensus has been reached. For example, in his original formulation, Dawkins made no distinction between memes and their vehicles. Later, in *The Extended Phenotype* (1982), he revised this to make "the distinction between the meme itself, as replicator, on the one hand, and its 'phenotypic effects' or 'meme products' on the other. A meme should be regarded as a unit of information residing in a brain" (p. 109). These two views are sometimes known as "Dawkins A" and "Dawkins B" (Gatherer, 1998).

Among followers of "Dawkins B" are those who distinguish between the actual meme and its products or vehicle, or, using Speel's (1997) terminology, they distinguish between the memotype and the phemotype. The first to do this, even before Dawkins' coining of the term "meme," was Cloak (1975), who distinguished the i-culture (cultural instructions inside people's heads) from the m-culture (the products of those instructions); the ultimate function of both being to maintain the i-culture. Delius (1989) and Aunger (2002) both propose that memes are some form of neural pattern inside the brain, and that their products or vehicles are outside of the brain. By contrast, Benzon (1996) argues precisely the opposite: that the replicators are outside brains and their vehicles inside.

This contentious issue has been used by some critics to explore the difficulties and limitations of memetics (e.g., Wimsatt, 1999), and by others to reject memetics altogether (e.g., Jablonka & Lamb, 2005). Calling them "the dreaded memes," Jablonka and Lamb (2005) argue that the "flaw stems from the distinction that is made between replicators (memes) and their vehicles (human brains, human artefacts, and humans themselves are all given this role)" (p. 208). However, not everyone makes this distinction. Dennett (1995) and I (Blackmore, 1999) do not. And Speel (1997) argues that "ideas, songs and norms reside in the brain somehow, as they are copied as units from human to human, but also from brains to books, or to physical air-waves . . . under the definition which makes physical memes replicators, brain-memes are also replicators" (p. 321).

Closely related to this issue are two other common objections: that memetic inheritance is Lamarckian (depending on the inheritance of acquired characteristics), and that cultural variation is directed (Aunger, 2000, Richerson & Boyd, 2005). For some critics, these two points seem to rule out memetics as a valid enterprise, while others are more concerned with whether culture can evolve if these two claims are true (Kronfeldner, 2007). I think the concern over Lamarckism is misplaced because the whole idea of calling memetic evolution "Lamarckian" rests on drawing an inappropriate memotype/phenotype distinction.

Hull (2000) argues that, since memes are replicators and not interactors, they are analogous to genes and not to phenotypic traits. So how you view such things as words, stories, or clothes fashions depends from where you look. From the perspective of genes, they are part of the phenotype, but from the meme's-eye view, they are memes. I think the whole problem stems from drawing too close an analogy between

genes and memes. In fact, these two replicators are different in important ways. Genes are neatly packaged inside bodies and passed on accurately down the germ-line, while their vehicles or interactors are not copied at all. For such a system, Weisman's law applies and there are good reasons why characteristics acquired by the phenotype are not passed on to the next generation (that is, leaving aside some forms of epigenetic effects). But most memes are not part of such a system and, so, the concept of Lamarckian inheritance simply does not apply (Blackmore, 1999).

I think that, as with so much of memetics, it helps to take a step back and look at the processes in a very general way. Genes have ended up with a system of genotypes and phenotypes because it is more effective to copy the instructions for making something than it is to copy the product itself. This is not only because you can make multiple copies from the same instructions (so increasing fecundity), but also because any accidents that befall the product—for example, in its construction or during its lifetime—are not passed on (thus, increasing fidelity). Dennett (1995) describes this as a system for retaining any "good tricks" that the evolutionary process stumbles upon, and Dawkins points out the design advantages of "going back to the drawing board" each time. By contrast, most memes have not yet evolved such a system (but, see below). Similarly, genes have evolved a system of blind variation which is more efficient than the varied sources of variation occurring among memes.

8 Old Genes, New Memes

All of the objections and confusions I have discussed here can be clarified by remembering that genes are old and memes are relatively new. Genes are part of an ancient system that has evolved over about four billion years into the amazingly complex and high-fidelity system that we see today, in which a phenomenal number of different kinds of creatures are all built from the recombination and mutation of information that is stored (almost entirely) in one single kind of molecule. Although we do not know precisely what the predecessor chemicals were, it is clear that DNA evolved from simpler systems into the modern system of genotypes and phenotypes, meiosis and mitosis, blind variation, and natural selection (Maynard Smith & Szathmáry, 1995). The whole process can be seen as a long and successful co-evolution between a replicator and its copying machinery.

By contrast, memes have been around for only a few million years. So, perhaps, it is not surprising that: much of the copying is of low fidelity; memes entail copying-the-product, rather than copying-the-instructions (Blackmore, 2001); generations are not clearly distinct; memetic transmission is piecemeal; and new copying machinery is appearing all the time. Yet, we might expect memetic evolution to be improving, and, indeed, it appears to be doing so.

For example, memetic equivalents of a germ-line and phenotypes have appeared in several domains. Take music, as an example. Originally, people would have listened to others singing or playing and then copied the sounds as best they could. Some people still do that, and, in this case, there is no replicator/vehicle distinction. But most music is now produced in recording studios, and then copied in factories onto various media, ending up listened to in people's homes or on mobile phones, MP3 players, and other personal music systems. The information on a CD or music

file is instructions for making sounds. When someone hears music they like, they don't copy the sounds directly, but go and buy or download the instructions from somewhere else. So this is all copy-the-instructions.

The same applies to cars, clothes, fridges, furniture, and almost all the household goods we take for granted. Few people make their own; instead they see ones they like and then go and buy similar, or even identical, ones. If lots of people buy a particular model of car, then the factory makes more from the production line it already had. It does not copy the cars already out on the streets.

Another change is the increase in fidelity and longevity created by the switch from analogue to digital storage. DNA is a digital system of very high fidelity. Human brains use both digital and analogue systems (e.g., information is carried by neurons either firing or not, but also as rates of firing, and the spatial summation of electrical potentials across membranes). Early memes were mostly analogue, but technological memes are far more often digitized, and increasingly so. Similarly, we may guess that culture is evolving toward systems that depend on blind variation, rather than directed variation. We can already see this happening in some computer software and on the Web, where software randomly recombines different memes to make new ones and then subjects them to selection. At the moment, most selection is still done by human beings, although processes of data mining and search engines are rapidly taking over some of this process. Eventually, we may suppose that all three processes—copying, variation, and selection—will be taken over by machinery outside of human brains. At this point, a third-level replicator will have emerged from the first two. These "technological memes" and their machinery may even deserve names of their own, such as "temes" and "teme machines" (Blackmore, 2009).

9 Religions, Cults, and Viral Information

In popular culture, memetics is best known for its treatment of viral information. As Dawkins originally pointed out, chain letters, pyramid schemes, and email viruses are all information that replicates for its own sake, rather than for the benefit of the people who do the copying. The structure of all of these viruses is the same: a copy-me instruction backed up with threats and promises. This, he argued, is precisely the structure of the major religions as well (Dawkins, 1993). Christianity and Islam, for example, have effective mechanisms for ensuring they keep infecting new hosts, many of which are familiar from biology (though, again, we must be careful with analogies). Many religions have mechanisms for ensuring that their central beliefs are passed on together in a package (e.g., confirmation classes or learning the catechism), and that the package is not picked apart. Threats and promises are used to ensure compliance (e.g., heaven, hell, or excommunication), and to prevent people from throwing off the infection (e.g., death sentences for apostates), and many other tricks are used such as piggy-backing beliefs on beautiful art and music, and persuading followers that they are superior, more altruistic, or more spiritual than non-believers (Blackmore, 1999).

Cults, religions, and alternative belief systems may be thought of as lying on a continuum from the purely viral, such as the most dangerous cults, to the symbiotic, and even beneficial. Where any particular example lies must depend on the effects

it has on the people who carry and propagate it, and on the groups who espouse it (Wilson, 2002). There is now increasing evidence of the harm that religions do to both individuals and societies (Harris, 2006, Paul, 2005); although also of some benefits, such as providing social cohesion and benefits to the group (Wilson, 2002).

If memetics is to be useful in the analysis of belief systems, it should be able to make predictions dependent on treating cults and religions as evolving systems parasitic on human carriers. For example, it should be able to predict which meme tricks religions need to use to survive in different circumstances, such as the difference between vertical transmission (as in Judaism and Catholicism, for example) and horizontal transmission (as in evangelical sects). Dawkins (1976) discusses the power of celibacy in ensuring that priests spend their time and energy promoting their memes rather than their genes, and the effectiveness of martyrdom as a gene-destroying meme-spreader. Lynch (1991) considers rules against masturbation as a way of promoting more offspring in vertically transmitted religions. An effective memetics should be able to understand these processes and predict the kinds of culture in which different systems will thrive.

10 Human Evolution

Memetics provides a completely different way of thinking about human evolution from other theories. The fundamental difference is that culture is seen not as an adaptation of benefit to early hominids and their genes, but as a parasitic second-level replicator that appeared when our ancestors became capable of imitation. From then on, the two replicators evolved together, the memes restructuring human bodies and brains to become better at propagating the very memes they copied. In this view, the turning point in human evolution is seen to be not the advent of tool use, or language, or symbolism (see, e.g., Deacon, 1997; Dunbar, 1996; Jablonka & Lamb, 2005), but, rather, the advent of imitation of sufficiently high fidelity that it inadvertently let loose memes: intelligence and language are seen as consequences of this transition, rather than its causes.

I have used the term *memetic drive* to describe the way this might have operated (Blackmore, 1999, 2001), and this process is different from those proposed in gene/culture co-evolution theories, in evolutionary psychology, or in theories based on sexual selection (Miller, 2000). The idea is that, once imitation became good enough, memes started spreading, possibly including ways of using fire, cooking food, wearing clothes, or making artifacts. These memes competed to be copied, with the winners affecting the selective environment such that people incapable of copying them would be at a biological disadvantage. This, alone, provides pressure for imitation ability to increase and, assuming that imitation requires brain space, pressure for increasing brain size.

However, the difference from other theories is that, in the memetic view, the direction taken by the evolution of the memes affects biological evolution as well, and this is true not only for useful memes such as lighting fires, but also for ones that are useless for survival such as wearing feathers in your hair. So, for example, if a fashion for performing ineffective rituals, wearing particular animal skins, or humming tunes took off, then people who could not copy these skills would lose out even

though the skills themselves were not adaptive. Many of the assumptions, here, are similar to those in other theories (e.g., Lumsden & Wilson, 1981; Richerson & Boyd, 2005), but the main difference is that memes are seen as replicators that evolve for their own sake, and the direction they happen to take then drags genetic evolution along.

A common example used in gene/culture co-evolution is the effect of dairying practices on genes for lactose tolerance (Durham, 1991). But this, like many such examples, is confined to a one-dimensional change. The equivalent, for memetic drive, would be if the evolution of cheeses and yoghurts—and eventually, TV dinners, microwaved chips, greasy hamburgers, or hot curries—drove complex changes in human digestive systems such that those systems showed traces of the history of the memetic evolution that caused them.

This is what I suggest happened with language, as well as the evolution of music, religious behaviors, and many other aspects of human nature. That is, over the past two or three million years, memes evolved by competing with each other, and the winners sculpted the direction of biological evolution to create brains that were not only larger, but also better adapted to copying the very memes which drove these changes. In the case of language, people began copying sounds; sounds of higher copying fidelity increased in the meme pool (for example, by processes of digitizing sound streams into discrete words, or linking sounds with seen objects); and, then, people who were good at copying those sounds thrived and passed on any genes conducive to that ability. In this way, brains gradually became better and better at copying the particular kinds of sounds that won in the memetic competition. In this view, language itself is seen as a parasite turned symbiont using human brains and vocal apparatus as its copying machinery. The same can be said of music, religion, and art: all these memetic systems have evolved in their own ways and, so, helped design human brains that are fond of music, ritual, and art.

One implication of this approach is that any system capable of imitating sounds and following gaze should give rise to its own language (Blackmore, 1999). Perhaps, surprisingly, this has turned out to be true for both simple imitating robots and autonomous agents (e.g., Steels, 2000, 2006). That is, language and reference can evolve without their being of any benefit to their carrier. In other research, simulations and computer modeling have explored the proposed interactions between the two replicators and the possible effects on brain size (Bull, Holland, & Blackmore, 2000; Higgs, 2000). Another prediction is that the parts of the human brain that maximally increased in size should also be those involved in imitation, and this has been confirmed by brain scanning studies (Iacoboni et al., 1999).

The human brain was the first meme machine, but now, other, far more effective meme machines are being created. Initial small steps toward increasing the longevity, fecundity, and fidelity of memes included the invention of writing, printing, roads, railways, telephones, and fax machines. All these developments can be seen as memes co-evolving with their copying machinery. Now the step to a third-level replicator can be discerned. We can look at it in the following way.

The first-level replicator (genes) constructed vehicles (Dawkins, 1976) or interactors (Hull, 1988) that protected and propagated them. One of these vehicles became capable of imitation and, so, let loose a second-level replicator (memes). That is, the vehicle from the previous level became the copying machinery for the next. A

similar step may be evident in the way that computer systems that began as vehicles for storing and manipulating memes are fast becoming capable of carrying out all three processes of copying, varying, and selecting information. This would make them true teme machines, and we should not expect them to be subservient to the old meme machines (Blackmore, 2009).

11 Consciousness, Creativity, and the Nature of Self

A common objection to memetics is that it undermines human autonomy and the creative power of consciousness, and treats the human self as a complex of memes without free will. These ideas follow naturally from the universal Darwinism on which memetics is based: that is, the idea that all design in the universe comes about through the evolutionary algorithm and is driven by replicator power. This means that human creativity emerges from the human capacity to store, vary, and select memes, rather than from some special creative spark, or power of consciousness (Blackmore, 2007). The human self may also be a construct of memetic competition, surviving because it protects and propagates memes, including the many memes that make up a person (Dennett, 1995). In this view, the self is not a continuously existing entity with consciousness and free will, but is a persistent illusion. This memetic view of human beings as the evolved creation of two replicators may be unsettling, but it has the advantage of uniting biological and human creativity into one, and providing new ways of understanding human nature, self, and consciousness.

12 Conclusion

These are just a few examples of a meme's-eye view on human and cultural evolution. I have outlined most of the major objections to memetics and shown why none of them effectively prevents memetics from being a viable scientific theory. In terms of the wider view of human nature and evolution, memetics clearly provides a novel way of looking at the world. But is it truer than existing models? The real test for memetics is whether its novel hypotheses and predictions can be tested and how they will fare in those tests. For that, it is still too early to say.

Postscript: Counterpoint

Wimsatt and I have many points of agreement. In particular, I agree (almost entirely) with his description of the three claims for memetics: first, that memes are replicators acting as endosymbiotic parasites (although I am not sure that they can truly be considered "autonomous" since they require humans or technology to copy them); second, that they are selfish and can manipulate their hosts for their own benefit; and, third, that they have evolved in efficacy and complexity and are responsible for the increase in human brain size and the elaboration of human culture.

We differ in that Wimsatt argues that "meme-like things" (MLTs) may fulfill the first of these, rarely fulfill the second, and that there are better ways of explaining

human and cultural evolution. He argues that memes are not the drivers of cultural and technological evolution but parts of a larger multi-component system. I cannot disagree that they are part of a larger system, or that cultural scaffolding, cultural infrastructure, learning, and cognitive processes are all important; the real question concerns the driving forces of that larger system. From a memetics point of view the driving force is selection acting on replicators, and since there are both genes and memes at play, these two replicators compete for resources, sometimes ending up in symbiotic relationships and sometimes in exploitative ones. The scaffolding, learning, and cognitive processes involved are all essential parts of the process (even if memeticists do not usually concentrate on them) in which memes are copied, varied, and selected. Memes are replicators in their own right subject to memetic selection, not merely aspects of the human phenotype subject only to biological selection.

Wimsatt presents a developmental theory of cultural evolution. Much of what he describes is compatible with memetics, but some of his predictions would be understood differently in a theory of memes. For example, he describes the infrastructure of roads, power supplies, and communication networks, and the standardization that is crucial to technological progress. I see these as the inevitable co-evolution of a replicator with its copying machinery. Roads and railways spread memes to far places and in the process spread the memes of building roads and railways. Similarly the Internet spreads content more widely and so encourages more use of the Internet. People adopt new technologies they like, apparently for their own benefit, with the result that they benefit the replication of those memes and meme machines.

As Wimsatt says, we do not yet have a thriving science of memetics, but I am sure that one is possible. Treating cultural information as a replicator will provide us with the best way of understanding human uniqueness, and the origins of the big brain, language, music and religions, and it will help us understand what is happening on a planet which is now acquiring a third replicator.

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CHAPTER SIXTEEN

Memetics Does Not Provide a Useful Way of Understanding Cultural Evolution

A Developmental Perspective

William C. Wimsatt

Are memes useful in the analysis of cultural evolution? Yes, in limited ways. But they don't contribute much to a deeper understanding of cultural change, and don't point in a useful direction for further theoretical development. If memes are conceptualized broadly and inclusively (so it is clear that they exist), they provide only a suggestive, but crude, tool indicating some features of the spread of cultural entities: primarily, the fact and consequences of horizontal transmission. They cannot, alone, provide the basis for a powerful theory of cultural evolution and change. After describing what memes can do, and the common assumptions shared by both memetic and non-memetic theories, I will discuss the shortcoming of a memetic approach to culture and outline the strategy for an alternative account. My basic contention is that the memetic approach cannot explain who acquires what memes—and in what order, as their acquisition is often order-dependent—nor can it give any account of the complex organization of culture. The alternative I propose can do both of these. In addition, my approach can explain a number of qualitatively distinct features of cultural and other complex organizational structures, as I make many distinctive predictions that are lacking in any of the alternative approaches.

1 Introduction

Are memes useful in the analysis of cultural evolution? Yes, in limited ways. But they don't contribute much to a deeper understanding of cultural change, and don't point in a useful direction for further theoretical development.[1] If memes are conceptualized broadly and inclusively (so it is clear that they exist), they provide only a suggestive, but crude, tool indicating some features of the spread of cultural entities: primarily, the fact and consequences of horizontal transmission. They

cannot, alone, provide the basis for a powerful theory of cultural evolution and change.[2] After describing what memes can do, and the common assumptions shared by both memetic and non-memetic theories, I will discuss the shortcoming of a memetic approach to culture and outline the strategy for an alternative account. My basic contention is that the memetic approach cannot explain who acquires what memes—and in what order, as their acquisition is often order-dependent—nor can it give any account of the complex organization of culture. The alternative I propose can do both of these. In addition, my approach can explain a number of qualitatively distinct features of cultural and other complex organizational structures, as I make many distinctive predictions that are lacking in any of the alternative approaches.

2 Some Commonalities

At some level, the existence of memes is obvious, and paradigmatically exemplified by the word itself, as Blackmore points out. Google-ing the word produced $3.29 \times 10^{**}8$ hits (on August 24, 2007); about 2.5 times as many as *gene*. (So, frequency of hits need not measure importance!) And the word *meme* is spreading into our discourse in other ways. There are now *bemes* (memes spread by bloggers) and *meme trackers* (sites and software, not people) that organize and cluster similar news stories. Search engines like Google can both measure the spread and diversity of memes (and, importantly, lead you to their contexts), but also, by bringing them to our attention, can find and make memes.[3] *Meme* is becoming so widely used by many of its defenders and the general public that any theory of cultural transmission that is not genetic may well come to be called a *memetic* theory. But this would be to trivialize the debate, which is not about naming. Before we look more closely at claims for memes, there are assumptions that meme theorists share with other students of cultural evolution. One must not assume that any theory sharing these assumptions is thereby "memetic."[4]

First, the biological underpinning. Human culture today is not driven by, or traceable to, genetics or explicable in terms of natural selection alone, but selection has played an important role in the evolution of different components of the capacity for culture. These include human sociality and social structure, bipedalism leading to the evolution of the hand and tool use, the beginnings of the language capacity, and the extended juvenile period that facilitated both brain growth and extended plasticity. We assume that these co-evolved with the capacity for, and production of, cultural behavior and artifacts. All of these are co-opted to facilitate role differentiation and extended sequential skill development with the emergence of an increasingly cumulative and diverse culture. (Sterelny 2009 has an illuminating discussion on the later stages of this evolution.)

Second, there are transmission processes operating other than through the germ-line. Non-genetic cultural transmission is real, rich, complex, and varied. Jablonka and Lamb (2005) systematically delineate three sets of non-genetic transmission processes—viz., epigenetic, behavioral, and symbolic—and classify further types within them. The latter two, at least, impinge upon, or constitute, the domain of culture.

Third, some of these processes at the social and cultural levels can achieve significant "robustness" and "dynamical autonomy" (Wimsatt, 2007) as "vicarious selectors—evolved substitute trial-and-error processes" (Campbell, 1974) that are relative to genetic processes.

Fourth, these autonomous processes can show positive feedbacks that may amplify, redirect, or even oppose primary biological selection processes, just as sexual selection can (Boyd & Richerson, 1985). These last three facts, together, generate processes claimed by memeticists, but also by most varieties of "dual-inheritance" theories.

Fifth, cultural transmission can become a "runaway" process primarily because culture can be transmitted horizontally (to non-relatives) as well as vertically (from biological parent to offspring). Many of its most interesting properties arise from horizontal transmission. But just as a fire can sweep across a forested area of diverse species, changing the niche and selection conditions for them all, cultural changes can have genetic consequences as well as cultural ones. Thus, the spread of herding among northern Europeans favored the evolutionarily rapid spread (within the last 8,000 years) of genes for extended lactose tolerance, but also the invention of cheeses, yoghurts, and kefirs (with lower lactose content) among other peoples lacking the relevant genes (Durham, 1991).

Sixth, I don't assume that cultural elements must be modular, though modularity increases evolvability, and can provide the basis for a combinatorial algebra for the construction of an array of things made with them. It is a powerful adaptation, both for things that are meme-like and cultural elements that don't meet the conditions for memes. Most modular cultural elements are not memes because they are not self-replicators, but parts of a larger reproductive complex.

As I see it, memetics claims three things: first, memes are autonomous replicators, which act as endosymbiotic parasites that are transmissible to other human beings; second, they are selfish like other parasites in that they can manipulate their hosts for their benefit, even in ways which would lower the host's welfare and biological fitness; third, they have undergone an evolution toward greater efficacy and complexity, and are responsible for our growth in cortical capacity, and for the increasing elaboration of our social and technological civilization, which, among other things, has given them other new transmission channels such as television and the Internet. I think that these claims are not required to explain brain evolution or the evolution of our technology, social structure, or culture. I believe that there are meme-like things (MLTs) that occasionally meet the first condition, rarely meet the second, and are better understood not as the primary drivers of cultural and technological evolution, but as parts of a larger multi-component process that better explains how their transmission and elaboration are mediated. Now, we must consider how these MLTs can (and cannot) be characterized. In the process, I will elaborate an alternative account.

3 Can a Memetic Approach to Cultural Change Work?

Can we conceptualize memes to put them on a firm theoretical footing? Two obvious ways look promising, at first, but seem to be dead ends, at least by themselves: the gene analogy and the viral analogy. A less obvious way is potentially quite promising, but demotes memes to one of several different kinds of elements in a richer and more complex theory. Such a theory would only very misleadingly be called *memetic*.

First, the tempting failures. Memeticists often talk about memes as if they were cultural genes or viruses. If described as self-replicating "selfish" entities that are like

either a gene or a virus (common assumptions), one might hope that genetics or epidemiology would suggest useful frameworks for building theory, viz., a memetics or memology, respectively. Blackmore (this volume) warns us against assuming analogies with genes or viruses, but if we are not to use such analogies, memeticists shouldn't either, and should provide us with a theory with some conceptual and predictive clout. The only reason to look for analogies is that they can give theoretical and predictive power. Either of these approaches could do this, if successful. Furthermore, if we can't get it there, we must seek a theoretical structure or construct it somewhere else, or accept that memetics is of very limited use. Without a strong guiding structure to articulate theory and guide application, it is only too easy to tell interesting "just-so" stories that are difficult to test. Such claims also do not differentiate, critically, between memetic and other theories of cultural evolution. I fear that this is the case for memetics as it stands. The alternative approach I propose does provide significant structure, predictive and explanatory power, and observable consequences.

4 Memetics and Genetics

Memeticists emphasize the importance of self-replication. But genetics does not get its rich theoretical power from the fact (or claim[5]) that genes are self-replicating. Gene replication remained a mystery to classical geneticists before 1953, and neo-Darwinism was developed without having an account of it. Genetics is a powerful component of evolutionary theory because genetic inheritance shows strong regularities that allow an immense variety of testable, and theoretically fruitful, predictions both for individual matings and for the selection of genes in populations. Genetics and population genetics texts are full of them.[6] As I have argued (Wimsatt, 1999), memes (or MLTs) show none of these structured patterns or any others of comparable predictive strength.

Complications for a gene-like interpretation for memes emerges from a closer look at these structural elements.

(1) Biological inheritance is normally obligately asexual or bisexual, and, for a given species, follows the same pattern, generation after generation.[7] Diploid organisms get one of each pair of chromosomes (and, thus, equal hereditary contributions) from each parent. Cultural inheritance for any trait can be derived from one to several "parents," which may make contributions of varying sizes, and with differing impacts, at different stages of ontogenetic development. The number of parents and the magnitudes of their contributions can vary from case to case and generation to generation. This variation, especially, undercuts the possibility of making population genetic-style models, in which such parameters are fixed and enter into recursion equations, define the model, and determine the behavior predictable from it.

(2) Genes occupy characteristic positions or *loci* in chromosomes. The two genes at corresponding positions in paired chromosomes are *allelic*. Different allelic variants are found in the breeding population. Genes at nearby loci in the same chromosome are inherited together with characteristic frequencies that decline with increasing distance. This association is called *linkage*, and is the basis for linkage mapping, which can determine the relative locations of all genes in the chromosome,

and predict new frequencies of association. The chromosomal source of prediction for inheritance in individuals and populations has no analogue for culture. There is no systematic linkage between cultural factors not caused by their functional co-adaptation. (There are other sources of patterned inheritance in co-adaptation, but this has not been systematically exploited by memetics. The developmental alternative I propose can do so.)

(3) Biological genomes are of a characteristic size, and arrangement, for a given species. Significant deviations from this arrangement are lethal, or can cause loss of genes and sterility in hybrids with other members of the breeding population.[8] This causes relative isolation and makes gene transfer from one species to another relatively rare. It also makes it fairly clear when two entities are con-specifics. There are no such similar constraints for cultural entities. Cross-lineage transfers are common, with few systematic constraints on what kinds of hybrids are possible. So species, gametes, chromosomes, and corresponding features of genetic and phylogenetic architecture become much more problematic. Phylogenies are much harder to disentangle without additional kinds of information (Lipo, O'Brien, Collard, & Shennan, 2006). The unfortunate fact for theory (though fortunate, perhaps, for our cultural adaptation) is that cultural inheritance is unconstrained in ways we would expect for genetic inheritance, a fact that is crucial for the predictive power of genetics and population genetics. The lack of *these* constraints, in part, contributes to quick-acting plasticity, adaptability, and tolerance for major change of cultural processes, but make it difficult to generate a memetic theory of co-inheritance, or of the growth of complex organization.

5 Memetics and Epidemiology

Could we exploit the host–parasite analogy further in search of a rich predictive structure? Thinking of memes as like viruses gives us many features that at first appear to support the memetic picture. These include horizontal transmission, parasite manipulation of host, and issues of infectivity (of viruses), differential infectibility (of hosts), and virulence. Some of the points of analogy seem rich,[9] but some crucial ones are missing.

A crucial difference is the number of distinct elements that an epidemiological process is supposed to handle. This might seem trivial, but it is not. Epidemiological models for the spread of diseases cover one or two diseases (with one equation per parasite and some simple assumptions about host susceptibility), and do not exploit the rich interactions between infective agents characteristic of cultural elements. By contrast, the average human "catches" on the order of 50,000 memes in a lifetime from a much larger array of MLTs available in the culture;[10] not in a long evolutionary history, but in the course of normal development. How this is possible demands explanation and receives none on the memetic approach. And writing down 50,000 epidemiological equations will not solve the problem.

The arrays of MLTs that different people catch differ, but show high internal order within persons and within group relationships. We can't learn ideas or acquire skills in just any order. Many MLTs are *constrained to be acquired in a developmental order* because later ones require the earlier ones to be intelligible, contextually relevant,

useful, or attractive. They thus show strong dependency relations for acquisition, affect which ones we are resistant to, and earlier MLTs modulate how later ones are interpreted.

Suppose that whether you could catch a given virus, and how it was expressed, was a complex function of what other viruses you had caught and in what order, and you were forced to deal with complex interactions of tens of thousands of virus types per individual. What would epidemiology look like then? This is crucial to understand learning and all modes of development: cognitive, social, and perceptual, and education processes from language learning to higher mathematics. Yet it is totally ignored in standard formulations of memetics. There is nothing in memetics to explain or to deal with the fact that, or ways in which, *cultural elements are structured*, either in their acquisition or in their action.

A second problem is that memetics ignores *the structured environments in which cultural elements are propagated*. Culturally induced social role and discipline-specific population structure[11] modulates the acquisition, maintenance, and elaboration of MLTs. (You do math problems in a math class as part of a math curriculum, and apply it later in a job for which you have been hired because you have the appropriate math competency.) Epidemiologists do worry about analogous details of population structure in looking at the transmission of disease and evaluating the possibility of epidemics, though memeticists have not. And the problem is much more complex for cultural MLTs than it is for viruses because of the far richer structure of the channels for exposure. Mathematical ideas from mainland China are inaccessible to a motivated, but monolingual, English speaker whether or not he or she is mathematically sophisticated. For some things, even knowing the language and having suitable training may not suffice (Nisbett, 2003). We need a theoretical framework that reflects and builds on these facts.

These two lacunae—viz., ignoring individual developmental structure and broader population structure and connectivity—are crippling. We live in cognitively and socially structured worlds (or, better, niches),[12] and any theory of cultural acquisition or change ignores these at its peril. The former tends to be the concern of developmental psychologists, educators, and practitioners of any specialty who must manage training; and the latter of sociologists and historians of science, religion, and a few other kinds of cultural lineages. I'll return to these when I present the alternative.

Some memeticists have pursued a third way, seeking necessary clarification from a search for a neurological basis for memes (e.g., Aunger, 2003). I believe that this is misdirected. Any such search must involve an articulated investigation at both conceptual and cognitive levels with the study of neural mechanisms, just like any other investigation in cognitive neuroscience (Bechtel, 2007). To understand MLT acquisition, we need to understand the relations between new MLTs and the existing MLT-complement first, in order to figure out what sorts of interactions we should be looking for at the neural level. Medical pathology and immunology provide rich sources for mechanisms of parasite–host interactions, but none for analogous parasite–parasite interactions, and it is the latter that demands attention for MLTs. And meme transmission is a different and commonly voluntary act, and should require whole other sets, respectively of cognitive and social interactions, to be coordinated with the study of the relevant articulated neural circuits and systems for us to understand.

So neither the genetic nor the viral analogies provide ways of getting a powerful predictive theory for memes, and we aren't even ready to start with cognitive

neuromemology. The issue is not whether memetics should have to look like one of these other sciences; I have already argued that it doesn't. The issue is whether we have *any* body of theory systematic enough to capture the major features of how MLTs are transmitted and acquired—as well as what makes them comprehensible and attractive—that reflects the rich population structure, context dependence, and relevance of some MLTs to what we already have that makes it worthwhile to assimilate (and, sometimes, to master and re-transmit) them. Memeticists puzzle over why memetics has seen little further theoretical development. The reason for this is because, with their focus on memes, they have ignored the many other systems they would need to study to produce a coordinated, coherent, adequate picture of the spread of memes. This broader theory would include some meme-like-things, but not in the dominant role they are supposed to have in memetics, and lacking the self-replication properties supposed for memes. This would not be a memetic theory.

6 The Myth of Self-replication

There is another problem with memes that has deep roots in the concept of a *self-replicator*, or *replicator* for short. This problem dates back to Richard Dawkins (1976) and the idea that strings of DNA are self-replicators. This is reductionistic mythology. The idea is that there is an informational core that contains all of the information necessary to remake itself and, also, a larger vehicle or interactor, and that the informational core is somehow self-replicating. *There is no such beast.* Von Neumann's self-reproducing automata had a machine and a tape that together made another machine + tape, but there were no tapes capable of remaking themselves. Similarly, over 200 enzymes are required for DNA replication. Of course, cells are self-reproducing as they go through a mitotic cell-cycle (well-documented by Moss, 2003), but this is a reproductive developmental cycle, *not* a copying event (see Griesemer, 2000; Wimsatt & Griesemer, 2007). There are modules that have like copies of them made, but they don't do it by themselves, and the capability to do so is always via a larger system. It is very easy to focus on the smaller duplicated parts, especially if they have a coding role in the system's activity in making other parts of the system; but we commit a serious functional localization fallacy if we act like the smaller part is doing this by itself. If we illegitimately project on the smaller part the ability to replicate or reproduce itself (by ignoring the role of the imbedding system in that production), it is only too easy to suppose it is the natural tendency of that part to evolve in ways to catalyze its own production, at the cost of the larger system or other such parts (thus, "selfish"). This can happen in special circumstances. Thus, we carry our load of transposons, which duplicate themselves throughout the genome (using cellular machinery), until it becomes too energetically costly to tolerate more. And cancer cells can throw off sufficient controls for runaway "selfish" reproduction to cause dissolution of the embedding system; but, thereby, terminate their lineage.[13] However, both of these kinds of events are normally under system-level controls to keep their effects bounded.

A better view of memes follows if they are not regarded (as genes have been) as the fundamental drivers of cultural and biological evolution, respectively, but viewed as modes or mechanisms of cultural hereditary transmission which can apply and

have significant effects when certain conditions are met. This is the course effectively argued by Sterelny (2006). Sterelny's analysis and this one are in many ways complementary. In his 2006 paper he does not emphasize culturally induced population structure, developmental dependencies, or social, cultural, and institutional context sufficiently, though he develops these significantly more in his most recent work (Sterelny, 2009).

With multiple hereditary channels, and multiple criteria which can be met in varying degrees for determining what counts as a hereditary channel, cultural evolution makes it only too easy to play fast and loose with calling things of which multiple similar or identical things are made "replicators" and act as if they may be "running the show." Griesemer and I (Wimsatt & Griesemer, 2007) try to show just how difficult it is to separate out relevant units of reproduction for culture. Generative entrenchment and modularity both play important roles in this endeavor, as does development (which is required for reproduction); but unconstrained "replicator" talk only muddies the water.

I suggest that one of the reasons why Blackmore (this volume) is able to find so many criteria for memes, and so many possible objects which could be memes on different interpretations, is the looseness that goes along with loose talk about replicators. The other reason why she is able to find (too) many plausible cases is that, on my account, there *are* meme-like-things (MLTs) that may be reproduced, and sometimes even copied, but, when that is the case, it is because of the existence of a larger system that produces them (usually along with many other things), and they are definitely not either self-replicating or "selfish."

Finally, the search for minimal memes (as proposed by Dennett, 1995) is not likely to be useful. In genetics, this search aided the determination of what kinds of things genes were, on the assumption that the structure of genes would explain their remarkable properties. The discovery of their double-helical structure and semi-conservative reproduction was genuinely explanatory as a way of explaining their stability at a time when the gene's stability appeared to contradict the known instability of very active molecules. Williams (1966) and Dawkins (1982) sought minimal units because they were more stable in the face of recombination, but this was more problematic: both ignored the fact that stability is a function of both selection and recombination (see Table 2 of Wimsatt, 1980, for computed tradeoffs), so the relationships involved in co-adapted complexes may be more evolutionarily stable than any of their parts, as recent work in evolutionary developmental biology has shown (Gilbert, Opitz, & Raff, 1996). The analogous strategy would be to look for meaningful adaptive complexes with modifiable components. But, since what is a meaningful cultural component, and how it is propagated, are both determined by the larger cognitive and cultural systems in which they are imbedded, finding a minimal memetic unit could not be expected to be particularly informative in any case.

7 An Alternative Approach

Can we do any better? Developmental constraints and the social structures in which we learn provide rich and theoretically salient sources of structure. This seems a productive place to start. Cultural elements are acquired by individuals throughout their

life cycles, in ways that show many determinable and predictable patterns, most commonly because these elements play roles in various sequentially acquired skills that depend on them. The orders and patterns of acquisition are not accidental, and have been crafted and modified to make them more efficient and effective. Some of these skills, such as language, basic self-care, and social functions, are acquired by all, and complement and scaffold the acquisition of other, more specialized skills. Some of these additional skills are common enough to a society that they become the subjects of curricula and are taught early in schools, and can, thereby, midwife major transitions in a society and culture, as when reading and basic numeracy became widespread competencies.

We acquire more differentiated skills, as alternatives, while training to adopt different social roles, careers, and life trajectories. These skills give competencies to acquire other skills, facilitated or scaffolded by institutional and organizational support structures and interactions. These dynamical support structures, themselves, are products of cultural evolution, and show developmental dependencies reflecting how these structures are produced and elaborated. One cannot understand how and when memes are acquired or even what can be an MLT (for an individual, or for a population) unless one understands how new potential MLTs interact with the cognitive developmental structure of individuals, and are propagated selectively through social and cultural institutions and organizations.

Consider the acquisition of mathematical skills. As we acquire basic cognitive skills for discrimination, counting and one-to-one matching, we learn how to generate new numbers, add and subtract, and multiply and divide. Some of this is by rote (e.g., multiplication tables), but increasingly it is anchored by learning how to follow rules in at first highly similar and then in more expansive ways. We learn manipulation of fractions, decimals, exponents, logarithms, natural numbers, signed numbers, rational numbers, real numbers, and complex numbers, all by extension of familiar ideas in new ways. Throughout all of this we are learning both geometry and—through word problems, and practical applications—how to interpret and use mathematics in an increasing range of natural situations that organize our perceptions and cognitive abilities. Knowing the calculus of several variables involves the instinctive use of dozens of component skills with most of them, in turn, dependent upon complex compound skills, which must be built up in sequence. How do we learn these? Through practice and problem sets, generating and solving thousands of problems along the way. We follow curricula, which may differ in fine details but each of which reflects a common strongly constrained developmental dependency of topics, the later of which are not even meaningful without having mastered and internalized the earlier ones.[14] The curricula, themselves, are products of a long historical development, the discussions of professionalized experts, and are modified or differentiated to respond to the needs of various user communities. Thus, the math sequence in college, even for very talented students, will differ—sometimes in major, sometimes in fairly subtle, ways—for students headed to become a professional mathematicians, biologists, physical scientists, social scientists, computer scientists, or economists. And the skills may further be differentiated as individuals develop in their profession by whether they use Mathematica, Spreadsheets, Matlab, various forms of statistical packages for complex data analysis, or agent-based simulations. Nearly three-quarters of a century ago in their classic genetics text, Sturtevant and Beadle (1939) noted both

the sequential dependence and the need for practice that students in the quantitative sciences will find so familiar:

> Genetics is a quantitative subject . . . it deals with ratios, with measurements, and with the geometrical relationships of chromosomes . . . it is a mathematically formulated subject that is logically complete and self-contained. We have attempted to treat the subject in a way suggested by these considerations—namely as a logical development in which each step depends upon the preceding ones. This book should be read from the beginning, like a textbook of mathematics or physics, rather than in an arbitrarily chosen order. . . . Genetics also resembles other mathematically developed subjects, in that facility in the use and understanding of its principles comes only from using them. The problems at the end of each chapter are designed to give this practice. It is important that they actually be solved. (p. 11)

8 Differential Dependency and Generative Entrenchment as Bases for a Theory of Evolutionary Change

Every complex machine has dependencies among its structural elements and dynamical dependencies in its operation. But these dependencies are not all equally great or important. Any interesting machine has *differential* dependencies for its different parts. This is an extremely general and robust truth for complex systems (Wimsatt, 2001). Changes in these dependencies, their modulation, and control provide the basis for differentiation and creation of different functional roles both within and between all complex systems, including cultures and the micro-cultures that make up different kinds of social roles. These changes are the means for the differentiation and elaboration of cumulative culture. This applies to even moderately adaptive systems of all kinds, whether they are biological, cultural, technological, or social ones.

Analysis of dependencies in developing structures is an extremely general way of characterizing structures. It also is crucial for assessing relative probabilities of change for different parts of that system. This method of analysis is no less general than population genetics; indeed, it is more general because it can be applied to the generative structures directly without knowledge of or even the presence of a genetic structure. Since relative dependencies have fitness consequences when there are disruptive mutations, dependency structures can also be used to build population genetic models. So this approach can be used with genetics, or without. It was originally used to model the evolution of gene control networks (Schank & Wimsatt, 1988, 2000; Wimsatt & Schank, 1988, 2004).

Differential dependencies affect the relative ease of making changes to and in an evolutionary process, the relative frequency of changes in the structures that show them. Deeper changes are more strongly selected against since they affect more things, are more work to change, and are more likely to cause serious malfunction in one or more of their larger number of downstream consequences. This has implications for their evolutionary rates. Simulations show that deeply entrenched elements are highly conserved, and that degree of entrenchment should be a good predictor of degree of conservatism. This principle is widely used in evolutionary developmental

biology in figuring out how developmental programs work and have evolved (e.g., Davidson & Erwin, 2006). Highly conserved genes and gene control circuits, phylogenetically, are widely distributed and relatively ancient. It is assumed that they play deep roles in generating other developmental structures. The systematic study of which genes affect the expression of others is used with this relative conservatism to infer the dependency structure of developmental circuits, and from that, how they work.

Differential dependencies can be used on genetic circuits, but they also can be applied to give an account of organization for an alternative conceptualization of evolution and prediction of differential evolutionary rates. Genes are not essential to such inferences. This makes the theory of dependencies applicable to culture and even more readily to technology, where the dependencies are more strikingly obvious. I suggest that if MLTs are analyzed and related in terms of the dependency structures for their acquisition, this gives a crucial part of what is needed to determine their evolutionary dynamics. If one has, in addition, the culturally induced population structure of the curricula of disciplines, the recruitment and training of companies and industries, the conceptual and normative slant of religions, ethnicities, and other affiliations that determine diffusion and acceptability of practices, ideas, and the use of artifacts, then the theory should apply there as well.

In this theory, the dependency structures for individuals determine the internal architectures of cultural genomes, an "endogenetics" (or, for culture, an endo-memetics). The culturally induced sociological structures correspond to population structure, and give an "exo-genetics" (or exo-memetics). From the perspective of an individual, culture has both an internal and an external complexity and organization, and, ideally, these are tuned to each other, so that individuals are embedded in the right social structures to learn what they need to know to fill social roles efficiently and effectively throughout their lifetimes. (Parents play a significant role in steering children either into their or into other productive occupations.) Here, memes play a very different kind of role, and it no longer seems appropriate to describe the theory as *memetic*.

9 Elements of a Developmental Theory of Cultural Evolution

If we look back at the kinds of elements we have discussed, raised, or presumed in this discussion, we find at least five kinds of things required for our theoretical account. First there are units, of which there are two types:

(1) *Meme*-like things (MLTs) that are units. Examples include artifacts, practices, ideas which are taught, learned, constructed, or imitated. These include both ideational and material things and are, themselves, capable of being chunked or black-boxed hierarchically; thus, they constitute multiple levels of organization. They may be chunked either within an individual's cognition and capabilities, or by an organization or profession, which puts together a team of individuals that collectively have the necessary capabilities.

(2) *Individuals* who are units. Examples include entities who develop, are socialized, and trained over time (in multiple contexts), and whose earlier training affects their capabilities, exposure, and receptivity.

Second, there are the built parts of the human cognitive, normative, and affective environment that scaffolds acquisition and performance of knowledge and skills, and coordinates their acquisition. There are three types of these:

(3) *Institutions* that are like MLTs, but at social/group level, containing normative rules or frameworks that guide behavior. Examples include social norms of behavior, legal codes, certification exams, and transition rituals like confirmation, bar/bat mitzvah, and graduations.

(4) *Organizations* or self-maintaining groups of individuals, self-organized for some purpose. These are like individuals, but at a social/group level, examples of which include interest groups, firms, nations, and disciplines.

(5) *Structures* or artifacts providing physical infrastructure maintained on transgenerational time scales providing "public goods" to the groups practicing the differentiated activity. Government bodies are hybrids of all three, as are most other complex cultural constructs.

To understand the articulation of these kinds of cultural elements, we must note that many are specifically designed to aid the construction or development of competencies among individuals and organizations. Griesemer and I call this *scaffolding*, and distinguish agent-scaffolding, artifact scaffolding, and infrastructural scaffolding (Wimsatt & Griesemer, 2007). Thus, there is one more kind of thing (having three sub-types) that we must discuss.

(6) *Scaffolding* refers to structure-like dynamical interactions with performing individuals that are means through which other structures or competencies are constructed or acquired by individuals or organizations. Thus, for example, chaperone molecules scaffold the right configuration for folding proteins, and the cell scaffolds gene replication and expression so fully that one wonders whether the relevant reproductive unit is the cell rather than the gene or genome. One can see parallels here with the richness of the scaffolding provided by the arranged contexts of the enculturated socialized human:

(6a) *Scaffolding for individuals*, examples of which include family structure, schools, curricula, disciplines, professional societies, church, work-organization, interest-groups, governmental units, laws.

(6b) *Scaffolding for organizations*, examples of which include (taking businesses as kinds of organizations) articles of incorporation, corporate law, manufacturers' organizations, chambers of commerce, and distribution networks for manufactured parts in the business world. Other kinds of organizations would have other kinds of scaffolding appropriate to them.

(6c) *Infrastructural scaffolding*—a particularly important kind of scaffolding of such broad applicability that it may be difficult to say what particular individuals or organizations and what competencies it is designed for. These are infrastructures, and our technological civilization has many such systems: roads, sea, rail, and air networks, shopping centers, truck farming, gas, water, power, telephone, distribution warehouses and networks, public transport, the Ethernet. Because it facilitates so many diverse kinds of things, this kind of scaffolding, arguably, is maximally entrenched.

Because cultural heredity in its fully fledged modern form involves the articulated interaction of all of these kinds of things, we can see that the meme-like things are just the tip of the iceberg. To be a meme for that individual at that developmental stage located in that social and cultural nexus, a large number of relationships conferring understanding, motivation, and opportunity to propagate or acquire that meme must be satisfied. Memetics is satisfied with far too little to provide the basis for a theory that could be either explanatory or predictive.

10 New Predictions of This Theory

Now, we can make several predictions from a theory articulating cultural diffusion and change in terms of the dependencies of the propagated elements:

(1) Features earlier in development that are more generatively entrenched should tend to be more evolutionarily conservative, yielding von Baer's "laws"—roughly, that earlier developmental stages tend to look more alike than later stages (Gould, 1977). And this predicts life *cycles*: successive generations must start in places like their parents, however much they diverge later, until reproduction "closes" and restarts the cycle (Wimsatt, 2001).

(2) New population genetic models of entrenchment (Schank & Wimsatt, 1988) give purchase on "complexity catastrophes" and the evolution of modularity (Wimsatt & Schank, 1988, 2004), which are also suggestive for culture.

Within cognition and culture other new phenomena arise through generative entrenchment:

(3) Prediction (1) has implications for cross-cultural invariants of cognitive development and language learning of the sort studied since Piaget (1954), and other biological determinants that are preconditions for human cognition and culture. A new entrenchment account of phenomena spoken of as "innate" captures the maximal consistent subset of criteria for innateness and predicts new ones (Wimsatt, 1986, 2002). We can understand the deleterious effects of early deprivation, the role of early development, the generative role of innate features, and the universality claimed for innate traits. We can do so in ways consistent with new perspectives in evolutionary developmental biology, and avoids the static ideas of genetic determination afflicting traditional accounts.

(4) For culture, as things get more deeply entrenched, we resist changing them, suggesting how things become conventional, standardized, and acquire a normative loading.

(5) Standardization is crucial to technological progress when further developments require common components, a more general feature of the truly *cumulative* culture sought by Richerson and Boyd (2005); a coordination game then drives the standards to fixation; standardized components then can become a combinatorial algebra for designing and making a diversity of other biological or cultural entities, such as genes, proteins, cells, words, sentences, machines, or other adaptations.

(6) In literary theory, Turner (1991) employs entrenchment to explain differences between literal and figurative meaning.

(7) Differences between biology and culture in how we can generate, and deal with, deeply entrenched changes are crucial to the rapidity and sometimes revolutionary character of cultural change (Wimsatt & Griesemer, 2007).

(8) Some further observations about change in complex cultural structures: (small) things with no generative structure are *paradigmatic "naked memes"* and permit the highest horizontal transmission rates consistent with the channel characteristics. Meme theorists seem to like these *r*-selective cases the most. They reflect, in themselves, the least structure, and thus *most anger social scientists when offered as examples of "culture."* These are, however, special kinds of degenerate case, and are not revealing of the broader cultural processes that make them possible.

(9) *The more complex the cultural trait, the more slowly it should spread*; it is more complex to learn and to teach (*internal complexity*).

(10) *Traits entirely new to the culture may be harder to interface with other cultural elements*, so that no one knows much of what they need to master it, or it may conflict with other cultural values.

(11) *Traits entirely new should show a longer lag (and possibly intermediate states) before spreading*; they take time to teach, learn, adapt, (simplify?), and recontextualize (*relational complexity*). And they will be *learned by a smaller number of individuals who specialize for the new trait unless it becomes "socially required." The chance of "falling off the track" will increase* even for those trying to complete the trait.

(12) One may therefore find *successive waves of adoption of parts or simplifications of it which are self-sustaining, and may ultimately elaborate (speciate) along new tracks and compete with and block spread of the original trait.* (Similar processes may have played a role in the spread and diversification of functions of written languages (Sanders, 2006) and the emergence of language dialects (Mufwene, 2008).

Now, consider aspects of gene evolution relevant to the acquisition of culture:

(12) *If a cultural trait has a structure which is gene evolved, complex, and important, more scaffolding should evolve* (either structurally transgenerational supports or co-adapted and ontogenetically acquired competencies) *to facilitate learning it.*

And (13), if *the scaffolding* can be used to learn other things, *it may become more entrenched than the original, an exaptive elaboration*, as with mathematics or writing.

(14) *Evolution structures the organization of developmental programs* (dispositions of resources) *so that assembly of GE structures will become* (and look) increasingly *self-organizing and self-maintaining* reliably across the range of environments normally encountered. (So gene evolution and so-called "self-organization" should interact richly.)

Finally (15), judgments of *"importance" of most cultural traits will derive largely from their generative entrenchment in the production of other cultural traits.* This judgment should be robust, in that it should be shared both by participants in that culture and by external students of it.

11 Conclusion

By now, I hope to have shown that, and why, memetics has stalled in its development and is an inadequate basis for a theory of cultural evolution. Indeed, I should

have shown something stronger: that no theory without a rich account of the role of individual and institutional development can provide an adequate account. Thus, although Boyd and Richerson (1985, 2005; Richerson & Boyd, 2005) have made a powerful beginning—one that helps to define the landscape for all subsequent theory—they cannot finish the job or characterize complex cumulative culture without incorporating developmental components. And niche-construction theory (Odling-Smee, Laland, & Feldman, 2003) makes an important move in the right direction (both toward development and toward recognizing the importance of scaffolding); but both are developed in such a black-box fashion that they are presently inadequate to this task. Furthermore, I have sketched some of the new phenomena that can be addressed by a theory that includes individual and institutional development, and outlined many new predictions suggested by this account. (Further details can be found in Wimsatt, 2001, 2002; Wimsatt & Griesemer, 2007; Wimsatt & Schank, 2004, and the papers referred to there.)

Postscript: Counterpoint

Blackmore's discussion might seem to suggest that the account presented here is merely an elaboration of meme theory. I disagree. I deny that MLTs are characteristically either selfish or capable of self-reproduction. And the theory of which they are a part draws its main predictive power from the analyses of dependencies in complex systems. I have taken pains to discuss some of the ways memetics shares features with this alternative theory, in virtue of the fact that both emphasize cultural transmission and evolution, but I hope it is apparent that the points of convergence are far outweighed by their differences.

But there is more. For the account I propose also suggests limiting cases where memetics should work, as virus-like horizontal transmission, although without attributing the power for self-reproduction or selfish behavior to memes. If meme acquisition and propagation requires all of these conditions on individual development and prior knowledge and values, on institutional and organizational infrastructure and scaffolding, then we can also specify limiting cases when we can predict outcomes without attending to all of this detail because the population and social and cultural structures are sufficiently common and homogeneous that they drop out of the predictive equation. Pick something that depends only upon shared language, culture and values and such basic knowledge that everyone alike is infectable, something sufficiently uncontroversial that it is equally attractive to all, and something sufficiently simple that its transmission can be treated as a single-stage process. Then meme theory works pretty well. But then it no longer differs from the predictions that might be made by other theories.

Notes

1 A possible exception would be the growing domain of Internet viruses, Trojan horses, and the like, all discussed by Aunger (2003). They have well-described adaptations for replication, but do not evolve by themselves. These interesting cases fit neither the memetic

nor the genetic paradigms, but are products of our technology. Spontaneously evolving artificial life deserves further attention, and it evolves in an electronic substrate in software-scaffolded niches.

2 Susan Blackmore urges that "memetics" also supports a "general selection paradigm." But the paradigm does not need memetics, and was advocated by others before Dawkins (1976). Campbell (1965) argued the point even more broadly, starting (in 1956) with parallels between selection and trial-and-error learning, extended it to culture in 1965 and, by 1974, included 10 distinct levels of "vicarious selectors," several of them at the cultural level. Lewontin's (1970) elegant elaboration of "Darwin's principles" had a formative influence in recognizing multiple units of selection (Wimsatt, 1980). Campbell (1965) puts the selectionist paradigm most broadly: "Any case of fit between a (complex) system and its environment should be explained by selection" (p. 20).

3 *Google* is still more frequent than *meme*. Even *google-ing* got 5 × 10**6 hits.

4 Some earlier theorists (e.g., Lumsden & Wilson, 1981) sought to see cultural differences as due to genetic differences. Most theorists (e.g., Boyd & Richerson, 1985; Cavalli-Sforza & Feldman, 1981; Durham, 1991; Richerson & Boyd, 2005) have focused on a "dual-inheritance" gene/culture co-evolution model. We share with meme theorists a concentration on the vast majority of cultural processes for which genetic differences are without known significant effects. These views can all be seen as lying on a continuum. At the earliest stages of acquisition of the cultural capacity, genetics (plus epigenetics) would have played the most obvious role. As culture came to play a larger role, behavioral transmission (Jablonka & Lamb's third channel) and gene/culture co-evolution should be more obvious. And in the most recent stages, quasi-independent cultural change has dominated. In part, this is so because of the increasing rates of communication and change made possible in the last two millennia, but also change in the West to a culture that values change over stability and has pushed, through war, colonization, and commerce, a rapidly escalating technological evolution.

5 Despite claims to the contrary, genes are not *self*-replicating, except as part of a larger system, the cell (Moss, 2003).

6 See, for example, Crow & Kimura (1970) and Sturtevant & Beadle (1939), respectively. Both are developed essentially using a classical genetic perspective.

7 As with virtually everything else in biology, there are exceptions. Thus, aphids have about 20 asexual generations per year (so that they don't need mates early in the season when they are rare), followed by one sexual generation before winter (to reap the benefits of recombination). And haplodiploidy is common in the social insects.

8 There can be glacially rare exceptions to this, too. Thus, there are apparent cases of genome doublings. Genes only extremely rarely migrate from one species to another (and usually do so via viral or bacterial transfer), though even rare migrations can be significant on a macro-evolutionary time scale.

9 Dan Sperber's "cultural epidemiology" (1996) is probably the most promising of these variants. He considers aspects of cognition that bias receptivity or transmission of ideas, leading possibly to biased, failed, or transformed transmission. (Note that this is a property not of MLTs, but of the cognitive system, reflecting its design aims and constraints, so it counters the "selfish meme" account.) Christophe Heintz's lecture "Mathematical Cognition and History: A Case Study on the Notion of Infinitesimals" (2009) argues that these kinds of factors played a role in motivating the replacement of infinitesimals by limits in the development of the calculus. Sperber's idea also relates naturally to the suggestion below (section 10, point 12) that variants of an MLT that are simplified or transformed to be more culturally coherent may be transmitted more rapidly and even compete with the original. The Relevance account of Sperber and Wilson (1986) may also be useful in understanding the microprocesses of sequential skill acquisition. But one would still need the

structured cognitive content and scaffolding architecture proposed here to understand the acquisition and relevant use of the enormous number of MLTs to be acquired in a lifetime.

10 It is unclear how to quantify how many distinct cultural elements one acquires, but for crude "order of magnitude" estimates, consider the vocabularies we master or the number of chess positions recognizable by a chess master. Both of these are of about this size (Simon & Chase, 1973). This is clearly an underestimate when one considers the many different kinds of things we know how to do, and when.

11 I don't here mean to ignore the many other crucial individuators of social boundaries—e.g., ethnicity, religion, social class, nationality, and race—all of which mediate the maintenance and propagation of MLTs.

12 Postmodernist metaphors have led to a free reification of worlds, when niches are a better fit. This is exploited by the new "niche construction" theory of Odling-Smee et al. (2003), though one must recognize that cultural niches and units are orders of magnitude more complex, and require a distinct strategy of approach (Wimsatt & Griesemer, 2007).

13 Grosberg & Strathmann (1998) speculate that the termination of cancerous lineages is the reasons why metazoans go through a single-cell stage.

14 The rich co-evolution of teaching and learning methods, the relevant institutional support structures, and the development of mathematical physics at Cambridge from 1750 to 1930 is elaborated in Warwick's fascinating (2003) study, and summarized in Wimsatt & Griesemer (2007).

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PART IX

CAN THE BIOLOGICAL SCIENCES ACT AS A GROUND FOR ETHICS?

Introduction

Near the close of the twentieth century, the principle of natural selection continued to play a key factor in explaining not only animal and human anatomy and physiology, but also psychology and behavior. Edward Wilson wrote a groundbreaking book titled *Sociobiology: The New Synthesis* (1975), where he attempted to use natural selection to explain many animal and human traits, including altruistic behavior. This work would help facilitate the emergence of a few of the topics in the philosophy of biology as they exist today, including *evolutionary ethics*.

Throughout his book, Wilson argues for the evolution of cooperation and altruism in animals via natural selection, which, although it, on the face of it, seems counterintuitive to the idea that an individual is naturally selected because of its own fortunate survival traits, still seems sensible to most people. For example, one would think that an animal like a worker bee would *not* have evolved a stinger that ends its life when used; however, since stinging some predator or intruder assists in saving the entire hive (and its own species), then we can see how the worker bee has made it through the evolutionary sieve. The same goes for many other species, especially those group or cooperative species that form tight, interconnected networks.

Now, here's what many considered to be the rub (and, possibly, the genius). Wilson suggests that *human*, ethical, altruistic behavior should be considered as "biologicized" (his term) since we humans essentially do the exact same kinds of things as animals, at a basic evolutionary biological level. So, consider the possibility that the *only* or *real* reason why a mom takes care of her child, or Mother Theresa helped

AIDS victims, or a Good Samaritan comes to your aid, or you don't just randomly take whatever you want from people whenever you want to, etc., is *not* because of a fully conscious, rational choice but, rather, because of basic, biologically based, kin selection. Nowadays, this may not strike us as shocking, but back in the 1970s it downright angered a lot of people (in fact, at a conference in 1978, someone actually threw water on Wilson, accusing him of being racist; Thacker, 2001). Since Wilson's publication, several thinkers have used plenty of ink debating (1) whether ethics is biologically based, and (2) the extent to which ethics is affected by our biology as well as our conscious, rational decisions to act in social settings through the erection of norms, values, moral principles, and civil laws.

Michael Ruse (1986, 1995) has been a well-known proponent of ethics as being biologically based, and has had his share of responders (see Ayala, 1995; Maienschein & Ruse, 1999; Nitecki & Nitecki, 1993). In fact, a paper by Ruse is the first one in this part. "Morality is an adaptation like hands, teeth, penises, and vaginas," claims Ruse, but he also maintains that culture plays a factor in the evolution of morality. Yet, it seems as if all aspects of humanity, even culture itself, are reducible to the workings of natural selection for Ruse. Ultimately, Ruse wants to argue that a kind of Humean, sympathetic, utilitarian cooperation acts as the basis for all of morality (even saying that he "takes this as a compliment, and not a criticism"), and this cooperation is the *direct* effect of natural selection.

In the second paper that comprises this part, Francisco Ayala argues against Ruse both (1) that all there is to morality is *merely* cooperation and (2) morality is the direct effect of natural selection. In opposition to Ruse, who views "humans (with respect to morality) as absolutely and completely part of the animal world," Ayala envisions morality as brought about through conscious intelligence and, hence, it has its basis in the realm of culture and not biology. According to Ayala: "Ethical behavior came about in evolution not because it is adaptive in itself, but as a necessary consequence of humanity's eminent intellectual abilities, which are an attribute directly promoted by natural selection. That is, morality evolved as an exaptation, not as an adaptation." Of course, according to both Ruse and Ayala, humans are animals subject to the same principles of evolution as any organism. However, Ruse emphasizes that reciprocal altruism, kin selection, and other forms of cooperation and morality have emerged directly via natural selection and comprise a part of culture as adaptations, while Ayala argues for reciprocal altruism and the like as emerging *in* culture indirectly through natural selection as exaptative by-products. There is a big difference between humans and animals that Ayala notes, which many would see as a central distinction: "[H]umans can *understand* the benefits of altruistic behavior (to the group and indirectly to them) and thus adopt altruism and protect it, by laws or otherwise, against selfish behavior that harms the social group."

This debate fascinates most people because we humans usually think that we are somehow different, or even better, than animals or any other organisms. Further, we think that our complex minds have enabled us to control or break free from our biological roots, to a certain extent. A well-developed morality in a civilized society—and *not* a *Lord of the Flies* scenario—is one way we may think we are different. Thus we are resistant to Ruse's claim, which may or may not be true, that "ethics is an illusion put in place by our genes to make us good social cooperators."

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CHAPTER SEVENTEEN

The Biological Sciences Can Act as a Ground for Ethics

Michael Ruse

This paper is interested in the relationship between evolutionary thinking and moral behavior and commitments, ethics. There is a traditional way of forging or conceiving of the relationship. This is traditional evolutionary ethics, known as Social Darwinism. Many think that this position is morally pernicious, a re-description of the worst aspects of modern, laissez-faire capitalism in fancy biological language. It is argued that, in fact, there is much more to be said for Social Darwinism than many think. In respects, it could be and was an enlightened position to take; but it flounders on the matter of justification. Universally, the appeal is to progress—evolution is progressive and, hence, morally we should aid its success. I argue, however, that this progressive nature of evolution is far from obvious and, hence, traditional Social Darwinism fails. There is another way to do things. This is to argue that the search for justification is mistaken. Ethics just is. It is an adaptation for humans living socially and has exactly the same status as other adaptations, like hands and teeth and genitalia. As such, ethics is something with no standing beyond what it is. However, if we all thought that this was so, we would stop being moral. So part of the experience of ethics is that it is more than it is. We think that it has an objective referent. In short, ethics is an illusion put in place by our genes to make us good social cooperators.

Ethics is an illusion put in place by natural selection to make us good cooperators.
Michael Ruse and Edward O. Wilson, "The Evolution of Morality" (1985)

1 Introduction

Not so long ago, evolutionary ethics was the philosophical equivalent of a bad smell. One knew that not only was it false, but somehow it was unclean—it was the sign that one had a tin ear for philosophy. First Henry Sidgwick (1874), and then G.E. Moore (1903, in *Principia Ethica*), had shown that evolutionary ethics simply will

not work; in Moore's language, it ignores or ploughs through the *naturalistic fallacy*. Or, to put matters in a more historical context, evolutionary ethics violates the distinction drawn by David Hume (1739–1740/1978) between *is* and *ought*. There are still those today who feel much as these earlier thinkers did. Richard Rorty, in perhaps the last thing he wrote before he died, was severely critical of an attempt by the Harvard evolutionary psychologist Marc Hauser (2006) to tie in ethics with our evolutionary past. Rorty (2006) wrote:

> We need, Hauser says, a "radical rethinking of our ideas on morality, which is based on the analogy to language." But the analogy seems fragile. [Noam] Chomsky has argued, powerfully if not conclusively, that simple trial-and-error imitation of adult speakers cannot explain the speed and confidence with which children learn to talk: some special, dedicated mechanism must be at work. But is a parallel argument available to Hauser? For one thing, moral codes are not assimilated with any special rapidity. For another, the grammaticality of a sentence is rarely a matter of doubt or controversy, whereas moral dilemmas pull us in opposite directions and leave us uncertain. (Is it O.K. to kill a perfectly healthy but morally despicable person if her harvested organs would save the lives of five admirable people who need transplants? Ten people? Dozens?)

Rorty allows that it may be possible "to update our moral software," but he doubts that biology is going to help any time soon.

My sense is that, thanks to evolution, we can do much more than Rorty thought possible. Perhaps the biologists should take things over. Can the biological sciences act as a ground for ethics? I give an emphatic "yes" to this question. Let us see how the case might be made.

2 Normative Ethics

In dealing philosophically with morality, there are always two levels to be discussed: *normative* or *substantive ethics*, which deals with what one ought to do ("love your neighbor as yourself"), and *meta-ethics*, which deals with why one ought to do what one ought to do ("God wants you to love your neighbor as yourself"). If one is trying to link evolution and normative ethics, then most obviously one will be trying to show that human ethical relationships are produced by evolution. Clearly, by its very nature, this is a naturalistic process, so let me stress now that my concern in this essay is with *methodological naturalism*—that is, trying to explain things scientifically—which, I take it, precludes supernatural events. Things must occur according to unbroken law. Miracles or interventions by the deity (or an Intelligent Designer) are just not allowed. I am saying nothing at all about *metaphysical naturalism*—whether science is the only thing that there is, or whether there is a world of God beyond science. At the end of this paper, I will have a few things to say about God and metaphysical realities beyond science; but nothing for now.

In substantive ethics, one is trying to show how people feel about moral statements. One is not judging the moral statements as such, although such an approach does not preclude any argumentation whatsoever about content. One could get into discussion about such issues as consistency, as well as the relevance of factual claims to moral issues. For instance, one might ask whether one is consistent in opposing

capital punishment yet, at the same time, allowing abortion on request. One might ask whether peace is more likely if one goes to war with Iran or if one tries other methods of containment. But, ultimately, I take it that one is in the business of description and scientific explanation. (More in a moment on this whole business of arguing for consistency. I agree with those people who think that this issue might be more complex than it looks, and that biology might have something to say on the issue.)

There has been much work done in the past 30 years trying to show how Darwinism *does* explain (in the sense of showing the origins of) normative ethics (e.g., Gibbard, 1990; Skyrms, 1998; Sober & Wilson, 1997; Wright, 1994). Although it is a dirty word in philosophical circles, the key breakthrough was the rise of *sociobiology* in the 1970s, with the various models of kin selection, reciprocal altruism, and the like, showing how Darwinian advantage could be gained by helping others; all a kind of enlightened self-interest on the part of the genes (Ruse, 1985, 1986). "You scratch my back and I'll scratch yours." Uncomfortable with the "selfish gene" approach, in recent years a number of holistic-type thinkers have been trying to promote an understanding of selection that emphasizes adaptations for the group (as against adaptations for the individual). I myself am not very keen on this way of seeing things, but here I will not dispute it. The main point is one of overlap. All are attempting to explain normative ethics as the result of evolutionary processes, and by this is meant that natural selection of some kind is the chief causal force. The late Stephen Jay Gould (2002) argued that perhaps mental attributes—and these would presumably include mental *moral* attributes—simply are what he called *spandrels*: by-products of the evolutionary process without any adaptive value. Although there are certainly philosophers who would be sympathetic to Gould's approach, the people who have tried to understand ethics in terms of evolution would all dispute this.

For at least two decades, I have been arguing for such a naturalistic, evolution-based approach to normative ethics (Ruse, 1986, 1996, 2001). Here, I do not intend to retread that material. Frankly, I think there is only so far that a philosopher like myself can take the discussion. I stand opposed to Rorty. A naturalistic approach means just that—one puts oneself in the hands of the scientists. These would include primatologists, students of comparative cultures, game theorists, evolutionary psychologists, economists perhaps, and others. All I will say here is that I find the results thus far very encouraging, although I am sure my critics would say that they would hardly expect me to find otherwise. I hope people will not be disappointed if I stress that I do not expect to find much difference between the findings of the biologists and the findings of the philosophers. Why should we? We love our neighbors as ourselves because, paradoxically, it is in our interests to do so. That is all there is to be said. That is how we feel.

If authority is needed to bolster my position, let me mention that the great social philosopher John Rawls (1971) seemed to think that this is how things work. As is well-known, his theory of *justice as fairness* is a form of social contract theory. How would we want society constituted—pay, medical care, and so forth—if we did not know (we were "behind the veil of ignorance") what role would be allotted to us? We could be female, born of rich parents, healthy, and beautiful; or male, born of poor parents, sick, and ugly. Rawls argues that we want society set up so whatever place we find ourselves in, we would benefit the most given the risks. We cannot just go for the female role because we might end up with the male role. Hence, we want a society that will look after the male as well as possible. This does not

necessarily mean that everyone will get the same. If we want good medical care, we might have to pay doctors twice the amount we pay professors. Rather, we want a society where the loser in birth's gamble gets as good a deal as possible.

Rawls (1971) admits fully that this all talks about hypotheticals. No one thinks that societies were set up by a gang of leaders and then the rules made mandatory. However, perhaps our genes did what our ancestors did not.

> In arguing for the greater stability of the principles of justice I have assumed that certain psychological laws are true, or approximately so. I shall not pursue the question of stability beyond this point. We may note however that one may ask how it is that human beings have acquired a nature described by these psychological principles. The theory of evolution would suggest that it is the outcome of natural selection; the capacity for a sense of justice and the moral feelings is an adaptation of mankind to its place in nature. As ethologists maintain, the behavior patterns of a species, and the psychological mechanisms of their acquisition, are just as much its characteristics as are the distinctive features of its bodily structures; and these patterns of behavior have an evolution exactly as organs and bones do. It seems clear that for members of a species which live in stable social groups, the ability to comply with fair cooperative arrangements and to develop the sentiments necessary to support them is highly advantageous, especially when individuals have a long life and are dependent on one another. These conditions guarantee innumerable occasions when mutual justice consistently adhered to is beneficial to all parties. (p. 440; in support of his position, Rawls footnotes Trivers on reciprocal altruism)

Incidentally, I am fully aware of the fact that biology and philosophy overlap at this point is in major part a function of the fact that they both stem from the same eighteenth-century ideas about competition, working together, and so forth. Darwin drew heavily on Thomas Robert Malthus, as well as Adam Smith and other great thinkers from the Scottish Enlightenment (Ruse, 1999). But I don't think we have a vicious circle here. If the philosophical and economic arguments did not work in biology, we would soon know. I see more of a kind of reinforcing feedback situation.

3 Biology Making the Decisions

Does the biology make no difference to the normative claims? Was Rorty worrying about a pseudo-question when he feared that biologists might make the moral decisions for us? I think biology can sometimes clarify things for us. Take the question of moral obligations to our closest loved ones (spouses, children, friends), as opposed to our moral obligations to total strangers. Some systems—perhaps utilitarianism, perhaps Christianity—suggest that the moral obligation lies equally with relative and with stranger. If, with the same amount of effort, you make the stranger happier than your own child—you can feed ten Africans for the price of one North American child—then that is the way you should go. Others, however, suggest that truly charity begins at home:

> "You find me, my dears," said Mrs. Jellyby, snuffing the two great office candles in tin candlesticks, which made the room taste strongly of hot tallow (the fire had gone out,

> and there was nothing in the grate but ashes, a bundle of wood, and a poker), "you find me, my dears, as usual, very busy; but that you will excuse. The African project at present employs my whole time. It involves me in correspondence with public bodies and with private individuals anxious for the welfare of their species all over the country. I am happy to say it is advancing. We hope by this time next year to have from a hundred and fifty to two hundred healthy families cultivating coffee and educating the natives of Borrioboola-Gha, on the left bank of the Niger." (p. 53)

This is from the great novel by Charles Dickens, *Bleak House* (1852–1853/2003), where he is highly critical of those who, like Mrs. Jellyby and her concern for the natives of Africa, neglect the sick and poor of their own land (Jo, the crossing sweeper), and even their own families (her children, Caddy and Peepy, for a start). David Hume (1739–1740/1978) had similar sentiments: "A man naturally loves his children better than his nephews, his nephews better than his cousins, his cousins better than strangers, where every thing else is equal. Hence arise our companion measures of duty, in preferring one to the other. Our sense of duty always follows the common and natural course of our passions" (pp. 483–484). All of this fits in very well with our biology, whereas helping strangers at the expense of family does not.

I am not sure that this is a case where biology would change our minds about moral thinking, but one might want to say that it helps to clarify difficult situations. It would help us to understand what we really think and want, as opposed to what we might think we want. Could biology actually make the decisions for us? Consider the following (precisely a version of that mentioned by Rorty). Suppose you saw a trolley, out of control, going down the track, about to kill five people, and could choose to set the points to send the cart onto a side-line and kill just one person. What would you do? You would probably switch the points. Now suppose you are on a bridge about to fall and you can save five of the six other people on the bridge by pushing off the sixth, fat one. Would you sacrifice him? I doubt you would do it. Why? Does evolution throw some light on this paradox, for formally the case is the same in both cases? People like Peter Singer (2005) suspect that our biologically evolved emotions might be significant here. Some leading brain researchers, to whom he refers, write as follows:

> We maintain that, from a psychological point of view, the crucial difference between the trolley dilemma and the footbridge dilemma lies in the latter's tendency to engage people's emotions in a way that the former does not. The thought of pushing someone to his death is, we propose, more emotionally salient than the thought of hitting a switch that will cause a trolley to produce similar consequences, and it is this emotional response that accounts for people's tendency to treat these cases differently. This hypothesis concerning these two cases suggests a more general hypothesis concerning moral judgment: Some moral dilemmas (those relevantly similar to the footbridge dilemma) engage emotional processing to a greater extent than others (those relevantly similar to the trolley dilemma), and these differences in emotional engagement affect people's judgments. (Greene, Sommerville, Nystrom, Darley, & Cohen, 2001, p. 2106)

This might be so because the trolley situation is something we (i.e., our ancestors) have encountered in the past, whereas the bridge situation is not something we have had experience of. At least, it may be that the emotions have been shaped in the

past to do what is in our best evolutionary interests, and this leads to the different responses. All of this suggests that rationality is not quite as nice and tidy as the logicians suggest, and that moral sentiments are more complex than philosophers and moralists have thought in the past. Rawls urges us to achieve "reflective equilibrium"—get our moral sentiments in a consistent whole—but Singer argues that this is simply not possible. And biology shows why.

For myself, I am not arguing this point in a definitive fashion, but I am suggesting that it might be a case where Rorty is wrong, and biology might indeed have a role in helping us with moral decisions. Or understanding why we think certain courses of action right and not others. Actually, I want to argue something rather stronger than this, but before I can do so I must turn to the purely philosophical part of the equation about morality, namely, that of justification. What of Darwinian meta-ethics?

4 Meta-ethics

There is still some hesitation by philosophers on this one. It is one thing to turn normative ethics over to the empiricists. It is quite another to think that the results of empirical science can truly answer questions that are so fundamentally philosophical—so dear to the hearts of those of us who stand in the tradition of Plato, Aquinas, and Kant. This ambivalence is shown in a recent piece by the well-known philosopher Philip Kitcher (2003). He asks the question: "So what exactly is the relationship between evolutionary theory and ethics?" Then he gives a preliminary answer:

> Let's start with a simple answer. There are many different projects relating evolutionary biology to ethics, some of which are perfectly sensible, others flawed. The hyper-Darwinian ambition is to show how our understanding of the history yields new basic moral principles. Somewhat less ambitiously, one might contend that Darwinism supports some distinctive metaethical view, that it shows, for example, that moral judgements cannot have truth-values or that moral knowledge is impossible. Much more modestly, we can see the evolutionary understanding of our species as relevant to the tracing of all aspects of human history, including the history of our morality and social systems. Finally, one might suppose that recognition of the kinship of life, coupled with moral principles we already hold, enables us to arrive at new derivative moral judgements—perhaps we come to understand ourselves as having obligations not to treat other animals in particular ways. The simple answer proposes that the first two of these ventures are illegitimate, while the latter two are well grounded. (pp. 411–412)

Kitcher argues that this simple answer is three-quarters right, only. The second part of the answer may well be false. "What is more problematic—and more interesting—is the claim about the irrelevance of Darwin for metaethics." All well and good. But do not get too excited. Before he is finished, Kitcher escapes making any definite decisions, concealed as he is in a cloud of apparently judicious hesitation about making any final judgments before all the facts are in:

> In outline, we can view morality as a human phenomenon that enters our history as a device for regulating the conflict between our sympathetic and selfish dispositions (where regulation plays a key role in the maintenance of our societies) and is further articulated

> through interactions among different social groups and members' reflections on those interactions. What status this assigns to our moral claims depends, I suggest, on the details of the story, and the details require much more research in evolutionary biology, anthropology, psychology and history than anyone has yet attempted. (p. 415)

Positions of this kind are not unknown in the philosophical community. If the science turns up trumps, I was there before you. And if not, then don't blame me. Run with the hare of naturalism, and hunt with the hounds of anti-naturalism—and blame science for your ambivalence.

Let me rush in where angels fear to tread. There is another philosophical tradition to ethics—that of Aristotle, Hobbes, and Hume, where the natural world is considered relevant, all the way down (or up). I believe we do now have enough material to make some judgments and decisions at the meta-ethical level, and in this discussion I am going to show you why I believe this. I agree that we do not have everything in that we would like at the normative level. All of the details—perhaps, even, the broad strokes—of the natural development of morality have not been explicated and explained. But as Kitcher himself agrees in the last quotation just given, we do have something. Biology—let us now agree for the sake of argument, natural selection—has played some significant role in making us moral beings. Morality is an adaptation like hands, teeth, penises, and vaginas. Obviously biology does not play the only role, and we must certainly allow culture some significant part also. How significant we can leave more or less open, between two false extremes—that everything is basically cultural (the blank slate hypothesis) and that everything is basically biological (the genetic determinism hypothesis). The point is that morality has come through human evolution, and it is adaptive.

5 Social Darwinism

Thinking now about meta-ethical issues, there is a traditional way of relating evolution and morality: that is, about issues centering on the justification or foundation of morality. (Why should I do that which I should do?) This is the way of the Social Darwinian (Richards, 1987; Ruse, 1996). Take as a paradigm the nineteenth-century philosopher Herbert Spencer. He argued from the way that things have been to the way that things ought to be. One ferrets out the nature of the evolutionary process—the mechanism or cause of evolution—and then one transfers it to the human realm (if this has not already been done), arguing that which holds as a matter of fact among organisms holds as a matter of obligation among humans (Ruse, 1986). Spencer (1851, 1857) himself started with the struggle for existence and the consequent selective effects: a connection which he made years after Darwin made the connection, but years before Darwin published. He then transferred to the human realm; not much to do here, actually, since Spencer speculated on selective effects showing themselves in the different natures and behaviors of the Irish and the Scots. He concluded that struggle and selection in society translates into extreme laissez-faire socioeconomics: the state should stay out of the way of people pursuing their own self-interests and should not at all attempt to regulate practices or redress imbalances or unfairnesses. Libertarian license, therefore, is not only the way that things are, but the way that they should be.

In fact, Spencer (1851) was far from convinced that mid-Victorian Britain was a laissez-faire society, but this is what he hoped fervently it would become.

> We must call those spurious philanthropists, who, to prevent present misery, would entail greater misery upon future generations. All defenders of a Poor Law must, however, be classed among such. That rigorous necessity which, when allowed to act on them, becomes so sharp a spur to the lazy and so strong a bridle to the random, these pauper's friends would repeal, because of the wailing it here and there produces. Blind to the fact that under the natural order of things, society is constantly excreting its unhealthy, imbecile, slow, vacillating, faithless members, these unthinking, though well-meaning, men advocate an interference which not only stops the purifying process but even increases the vitiation—absolutely encourages the multiplication of the reckless and incompetent by offering them an unfailing provision, and *discourages* the multiplication of the competent and provident by heightening the prospective difficulty of maintaining a family. (pp. 323–324)

Spencer could sound positively brutal about those who would help the unfortunate within society:

> Besides an habitual neglect of the fact that the quality of a society is physically lowered by the artificial preservation of its feeblest members, there is an habitual neglect of the fact that the quality of a society is lowered morally and intellectually, by the artificial preservation of those who are least able to take care of themselves. . . . For if the unworthy are helped to increase, by shielding them from that mortality which their unworthiness would naturally entail, the effect is to produce, generation after generation, a greater unworthiness. (cited in Richards, 1987, p. 303)

I should say that not everyone argued in this way from evolution. At times, Social Darwinism reminds one of Christianity. It tells us that we should love our neighbors as ourselves. To President George W. Bush, this translates out as invading Iraq. To the Quakers, it translates out as pacificism. Showing how evolution can serve different ends, take another ardent evolutionary ethicist in the Spencerian tradition, Julian Huxley (the grandson of Thomas Henry Huxley and the older brother of the novelist Aldous Huxley). He argued that evolution justifies an obsession with technology, science, and major public works. While Huxley (1934) was not uninterested in life at the personal level, it was the general domain which really excited him.

> All claims that the State has an intrinsically higher value than the individual are false. They turn out, on closer scrutiny, to be rationalizations or myths aimed at securing greater power or privilege for a limited group which controls the machinery of the State. On the other hand the individual is meaningless in isolation, and the possibilities of development and self-realization open to him are conditioned and limited by the nature of the social organization. The individual thus has duties and responsibilities as well as rights and privileges, or if you prefer it, finds certain outlets and satisfactions (such as devotion to a cause, or participation in a joint enterprise) only in relation to the type of society in which he lives. (pp. 138–139)

The key moral principle seems to have been for the need of planning in running the state and, above all, the application of scientific principles and results in such planning

and its implementation. You simply cannot (or should not) leave things to chance or intuition—the implication being that this is precisely where your average politician does leave things—but should bring the trained scientific mind to bear on life's problems.

Again and again, Huxley returned to this theme. For instance, in the book cited above, *If I Were Dictator* (1934), he stressed the need for science in the running of an efficient state and that such science would need to be of the social variety as well as physico-chemical and biological. During World War II, he wrote a highly laudatory essay on the Tennessee Valley Authority, that marvel of the Rooseveltian New Deal, whereby the federal government built and ran a massive system of river damming and irrigation in what had hitherto been one of the more desolate parts of the U.S. (Huxley, 1943). Then, after the war it was Huxley who insisted on the Science being added to UNESCO, and he wrote a vigorous polemic arguing that the organization had to be run on evolutionary lines—lines demanding lots of science (Huxley, 1948). So vigorous was his polemic, indeed, that he upset his masters and he was refused a full four-year term as director-general.

6 Progress

But how does one justify moves like those of Spencer and Huxley? It is here that Moore and others found the fallacy. Because things are this way, it does not follow that things should be this way. In fact, I myself agree with this criticism, but my experience is that Social Darwinians (these days, they tend not to be called by this name) find this criticism supremely unimpressive. My sometime co-author Edward O. Wilson points out that, while it is indeed true that one is going from "is" to "ought"—in his own case he is concerned to promote biodiversity, as one does when saving the Brazilian rainforests and, hence, goes from the premise that humans need biodiversity to the conclusion that we should promote biodiversity—this in itself hardly makes the inference fallacious (Wilson, 1984, 1992, 1994). In science, one is always going from talk of one kind to talk of another kind, and no one thinks this fallacious in itself. In gas theory, one goes from talk of molecules bouncing around a chamber at different speeds to talk of increases in pressure and temperature. Is this any more odd than going from "humans need the rainforests" to "we ought to preserve the rainforests"?

We need to dig further into the meta-ethics of Social Darwinism, and soon the real reason for the confidence becomes apparent. To a person, Social Darwinians—call them traditional evolutionary ethicists, if you prefer—are progressionists. They think that the course of evolution is upward, from the bad or the non-moral to the good and the moral and the worthy of value. Hence, to keep this progress going is in itself a good thing. Listen, for instance, to Herbert Spencer (1857). For him, evolution was a transition from the undifferentiated, or what he called the "homogeneous," to the completely mixed up, or what he called the "heterogeneous." Progress was not just a biological or a social phenomenon; it was an all-encompassing world philosophy.

> Now we propose in the first place to show, that this law of organic progress is the law of all progress. Whether it be in the development of the Earth, in the development of

> Life upon its surface, in the development of Society, of Government, of Manufactures, of Commerce, of Language, Literature, Science, Art, this same evolution of the simple into the complex, through successive differentiations, hold throughout. From the earliest traceable cosmical changes down to the latest results of civilization, we shall find that the transformation of the homogeneous into the heterogeneous, is that in which Progress essentially consists. (p. 31)

Likewise with later thinkers of this ilk. It is progress, and the need to keep it going, that is the foundation. Julian Huxley (1927) is quite clear on this:

> When we look at evolution as a whole, we find, among the many directions which it has taken, one which is characterized by introducing the evolving world-stuff to progressively higher levels of organization and so to new possibilities of being, action, and experience. This direction has culminated in the attainment of a state where the world-stuff (now moulded into human shape) finds that it experiences some of the new possibilities as having value in or for themselves; and further that among these it assigns higher and lower degrees of value, the higher values being those which are more intrinsically or more permanently satisfying, or involve a greater degree of perfection.
>
> The teleologically-minded would say that this trend embodies evolution's purpose. I do not feel that we should use the word purpose save where we know that a conscious aim is involved; but we can say that this is the *most desirable* direction of evolution, and accordingly that our ethical standards must fit into its dynamic framework. In other words, it is ethically right to aim at whatever will promote the increasingly full realization of increasingly higher values. (p. 137)

Likewise Edward O Wilson (1992):

> [T]he overall average across the history of life has moved from the simple and few to the more complex and numerous. During the past billion years, animals as a whole evolved upward in body size, feeding and defensive techniques, brain and behavioral complexity, social organization, and precision of environmental control—in each case farther from the nonliving state than their simpler antecedents did.

He concludes: "Progress, then, is a property of the evolution of life as a whole by almost any conceivable intuitive standard, including the acquisition of goals and intentions in the behavior of animals" (p. 187). The point is made.

And here I think is the reason to be dubious about the meta-ethics of Social Darwinism. Popular though it may be, the very idea of progress in evolution is clouded in problems. It is far from obvious either that natural selection promotes progress or that progress actually occurs, at least in any clear definable and quantifiable way. One can, of course, label humans as the pinnacle of being—I myself am inclined to do just this—but such an act is arbitrary, at least as applied to evolution. Why not label a dog the pinnacle of being or a buttercup? From a biological point of view, the AIDS virus is far more successful than the gorilla, but does anyone truly want to say that the former is superior in a moral or other value sense to the latter?

In a typically hyperbolic fashion, Stephen Jay Gould (1988) writes: "Progress is a noxious, culturally embedded, untestable, nonoperational, intractable idea that must be replaced if we wish to understand the patterns of history" (p. 319). With respect to human evolution, he writes:

> Since dinosaurs were not moving toward markedly larger brains, and since such a prospect may lie outside the capabilities of reptilian design . . . we must assume that consciousness would not have evolved on our planet if a cosmic catastrophe had not claimed the dinosaurs as victims. In an entirely literal sense, we owe our existence, as large and reasoning mammals, to our lucky stars. (Gould, 1989, p. 318)

Even if one thinks that this is perhaps a little extreme, there is surely enough truth to make one very wary about biological progress as a basis for one's moral code. Whatever one might say about the normative ethics of Social Darwinism—and although I am not very keen on laissez-faire, I am very keen on the rainforests and their preservation—meta-ethically, the justification seems shaky.

7 Ethical Skepticism

But can one do better? Can one overcome Kitcher's hesitation? I think one can. Remember that, for the sake of argument, we are agreeing—and I think Kitcher gives us this much—that we humans have built-in innately, or instinctively if you like, a capacity for working together socially. And this capacity manifests itself at the physical level as a moral sense. Hence morality or, rather, a moral sense is something which is hard-wired into humans—mediated and fashioned by culture. Morality has been put there by natural selection in order to get us to work together socially or to cooperate. This is not to say that we do not have freedom in any sense. It is not to say that we never disregard our moral sense, but rather that we do have the moral sense and we have the moral sense not by choice or decision, but because we are human. (Of course, there are going to be psychopaths without a moral sense, but in biology you know that there are going to be exceptions for every rule.) The claim, therefore, is that when humans find themselves in a position where cooperation might pay, morality kicks into place.

This is not to say that we always will cooperate or be moral. We are influenced by many factors, including selfish and other sorts of desires. But morality is one of these factors and, overall, we humans do generally work together. Sometimes the morality backfires. I might go to the aid of a drowning child and drown myself. This is hardly in my self-interest. But, on balance, it is in my interests to have the feeling that I ought to help people in distress, particularly children in distress. This is both because I myself was at some stage of my life a child, and also because I myself will probably have or be having children. I want others to be prepared to make a risk on my behalf or on the behalf of my children.

Let it also be stressed that humans have a genuine sense of morality. It is the kind of morality that someone like Immanuel Kant (1788/1949) talks about. This is not a scientific position of pure ethical egoism in the sense that we are all selfish people just simply calculating for our own ends. We are, rather, people with a real moral sense, a feeling of right and wrong and obligation. Admittedly, at the causal level, this may well be brought about by individual selection maximizing our own reproductive ends. But the point is that, although humans are produced by selfish genes, selfish genes do not necessarily produce selfish people. In fact, selfish people in the literal sense tend to get pushed out of the group or ostracized pretty quickly. They

are simply not playing the game. In a way, therefore, we have a kind of social contract. But note that it is not a social contract brought about, in the long-distant past, by a group of grey-bearded old men sitting around a camp fire. It is rather a social contract brought on by our *biology*, that is to say, by our genes as fashioned and selected by natural selection. Remember Rawls on this point.

This, then, is the Darwinian perspective on the evolution and current nature of morality. Let us now see how this plays out when we try to put things into a philosophical context. What kind of meta-ethical justification can one give for such claims as that one ought to be kind to children, and that one ought to favor one's own family over those of others? I would argue, paradoxically but truthfully, that ultimately there is no justification which can be given! That is to say, I argue that at some level one is driven to a kind of moral skepticism: a skepticism, please note, about foundations rather than about substantive dictates. What I am saying therefore is that, properly understood, the Darwinian approach to ethics leads one to a kind of moral non-realism (Ruse, 1986).

In this respect, the Darwinian meta-ethics I am putting forward in this paper differs very dramatically from traditional Darwinian meta-ethics, that of Social Darwinism. There, the foundational appeal is to the very fact of evolution. People like Herbert Spencer and Edward O. Wilson argue that one ought to do certain things because by so doing one is promoting the welfare of evolution itself. Specifically, one is promoting human beings as the apotheosis of the evolutionary process—a move we have seen condemned by philosophers as a gross instance of the naturalistic fallacy, or as a flagrant violation of Hume's Law (that which denies that one can move legitimately from the way that things are to the way that things ought to be). My kind of evolutionary meta-ethics agrees with the philosopher that the naturalistic fallacy is a fallacy and so, also, is the violation of Hume's Law. My kind of evolutionary meta-ethics also agrees that Social Darwinism is guilty as charged. But my kind of evolutionary meta-ethics takes this failure as a springboard of strength to its own position. The Darwinian meta-ethics of this paper avoids fallacy, not so much by denying that fallacy is a fallacy, but by doing an end run around it, as it were. There is no fallacious appeal to evolution as foundations because there are no foundations to appeal to!

Although I am arguing that morality does exist, if without foundations, do note that my position has a real bite that other positions do not have. Go back to the researchers on psychological reasons for our taking different paths on the trolley versus the bridge situation. They ran experiments that showed that it is truly the case that we use different parts of the brain to make the two different assessments. Emotion is involved and not just some disinterested reason. They conclude:

> The trolley and footbridge dilemmas emerged as pieces of a puzzle for moral philosophers: Why is it acceptable to sacrifice one person to save five others in the trolley dilemma but not in the footbridge dilemma? Here we consider these dilemmas as pieces of a psychological puzzle: How do people manage to conclude that it is acceptable to sacrifice one for the sake of five in the one case but not the other? We maintain that emotional response is likely to be the crucial difference between these two cases. But this is an answer to the psychological puzzle, not the philosophical one. Our conclusion, therefore, is descriptive rather than prescriptive. We do not claim to have shown any actions or judgments to be morally right or wrong. (Greene et al., 2001, p. 2107)

Actually, they go on to say that the story is a little more complex than this because, in some sense, reason can get involved along with the emotions. But the point I want to make is clear. As a moral skeptic, I argue that there is no higher court of appeal than the emotions. To quote David Hume (1739–1740/1978): "Reason is, and ought only to be, the slave of the passions" (2.3.3.4). This is what right and wrong is all about. At this point the descriptive and prescriptive come together. Not because the former justifies the latter, but because the former leads us causally to have the latter and there is nothing more. (At least, there is nothing more in a naturalistic world. See below for some religious qualifications.) The one decision is right and the other wrong, and that is all there is to it. It is all a bit like baseball. After three outs, your share of the innings is over. That is it. There is no higher appeal. Of course, in baseball you can decide not to play, or to play tennis instead. In life, things are not so easy, and those of us who do try to opt out—like Raskolnikov in *Crime and Punishment*—tend (if we are not sociopaths) to find ourselves being tugged back in. Our psychology trumps what our reason might tell us is really what is happening. Hume (1739–1740/1978) again:

> The intense view of these manifold contradictions and imperfections in human reason has so wrought upon me, and heated my brain, that I am ready to reject all belief and reasoning, and can look upon no opinion even as more probable or likely than another. Where am I, or what? From what causes do I derive my existence, and to what condition shall I return? Whose favour shall I court, and whose anger must I dread? What beings surround me? and on whom have I any influence, or who have any influence on me? I am confounded with all these questions, and begin to fancy myself in the most deplorable condition imaginable, environed with the deepest darkness, and utterly depraved of the use of every member and faculty. Most fortunately it happens that, since reason is incapable of dispelling these clouds, nature herself suffices to that purpose, and cures me of this philosophical melancholy and delirium, either by relaxing this bent of mind, or by some avocation, and lively impression of my senses, which obliterate all these chimeras. I dine, play a game of backgammon, I converse, and am merry with my friends; and when, after three or four hours amusement, I would return to these speculations, they appear so cold, and strained, and ridiculous, that I cannot find in my heart to enter into them any farther. (p. 269)

8 Objectification

To be blunt, my Darwinism says that substantive morality is a kind of illusion, put in place by our genes, in order to make us good social cooperators (Ruse & Wilson, 1985, 1986). I would add that the reason why the illusion is such a successful adaptation is that not only do we believe in substantive morality, but we also believe that substantive morality does have an objective foundation. An important part of the phenomenological experience of substantive ethics is not just that we feel that we that ought to do the right and proper thing, but that we feel that we ought to do the right and proper thing because it truly is the right and proper thing. As John Mackie (1979) argued before me, an important part of the moral experience is that we objectify our substantive ethics. There are, in fact, no foundations, but we believe that in some sense there are.

There is a good biological reason why we do this. If, with the emotivists, we thought that morality was just simply a question of emotions without any sanction or justification behind them, then pretty quickly morality would collapse into futility. I might dislike you stealing my money, but ultimately why should you not do so? It is just a question of feelings. But in actual fact, the reason why I dislike you stealing my money is not simply because I do not like to see my money go, but because I think that you have done wrong. You really and truly have done wrong in some objective sense. This gives me and others the authority to criticize you. Substantive morality stays in place as an effective illusion because we think that it is no illusion but the real thing. Thus, I am arguing that the epistemological foundation of evolutionary ethics is a kind of moral non-realism, but that it is an important part of evolutionary ethics that we think it is a kind of moral realism.

This is my counter to the worries expressed by people like Alex Rosenberg (2003), who point out that the kind of position that I endorse is close to the twentieth-century moral philosophy of emotivism—where ethical claims are simply emotive utterances—and who point out, also, that emotivism is clearly false. Killing babies is wrong is not just an emotive cry, but a claim about something's being truly really wrong. For me, substantive ethics is only emotion, but it means more than that. Ethics is subjective, but its meaning is objective.

9 Spiritualism

In a way, what has been given thus far is just a statement rather than a proof. What justification can I offer for my claim that evolution points toward ethical skepticism (about foundations)? Why should one not say that there truly is a moral reality underlying morality at the substantive level, and that our biology has led us to it? After all, we would surely want to say that we are aware of the speeding train bearing down on us because of our biology, but this in no sense denies the reality of the speeding train (Nozick, 1981). Why should we not say, in a like fashion, that we are aware of right and wrong because ultimately there is an objective right and wrong lying behind moral intuitions?

However, things are rather different in the moral case from the speeding-train case. A more insightful analogy can be drawn from spiritualism. In World War I, when so many young men were killed, the bereaved—the parents, the wives, the sweethearts, on both sides of the trenches—often went to spiritualists, hoping to get back in touch with the departed dead. And indeed they would get back in touch. They would hear the messages come through the Ouija boards or whatever assuring them of the happiness of the now deceased. Hence, the people who went to spiritualists would go away comforted. Now, how do we explain this sort of thing? Cases of fraud aside, we would say that people were not listening to the late departed, but rather were hearing voices created by their own imaginations which were, in some sense, helping them to compensate for their loss. What we have here is some kind of individual illusion brought about by powerful social circumstances. Almost no one would think that the late Private Higgins was really speaking to his mum and dad. Indeed, there are notorious cases where people were reported killed and then found not to be dead. How embarrassing it would be to have heard the late departed assure you of his

well-being, and then to find out that the late departed was in fact lying injured in a military field hospital.

In the spiritualism case, once we have got the causal explanation as to why people hear as they do, we recognize that there is no further call for ultimate foundations. I would argue that the biological case is very similar: that there are strong biological reasons for cooperation. Naturally, we are going to be selfish people, but as cooperators we need some way to break through this selfishness; and so our biology has given us morality in order to help us do it. Once again, I stress that this is not to say that we are always going to be moral people; in fact, we are an ambivalent mixture of good and bad, as the Christian well knows (Ruse, 2001). It is to say that we do have genuine moral sentiments which we think are objective, and that these were put in place by biology. Once we recognize this, we see the sentiments as illusory—although, because we objectify, it is very difficult to recognize this fact. That is why I am fairly confident that my having told you of this fact will not mean that you will go off and rape and pillage because you now know that there is no objective morality. The truth does not always set you free.

10 Progress Again

But, still, you might protest that this does not mean that there is no objective morality behind all of this: either an objective morality of a Platonic ilk which actually exists out there, or an objective morality of the Kantian form which is a kind of necessary condition for rational beings getting along. Here, however, the Darwinian can come back with a further argument, namely, one based on the doubts expressed earlier about biological progress. There is no natural climb upward from the blob to the human, from the monad to the man, as people used to say in the nineteenth century. Rather, evolution is a directionless process, going nowhere rather slowly (McShea, 1991; Ruse, 1993). What this means, in this particular context, is that there is really no reason why humans might not have evolved in a very different sort of way, without the kind of moral sentiments that we have. From the Darwinian perspective, there is no ontological compulsion about moral thinking.

It is true that, as Kant stressed, it may possibly be that social animals may necessarily have to have certain formal rules of behavior. But it is not necessarily the case that these formal rules of behavior have to incorporate what we would understand as common-sense (substantive) morality. In particular, we might well have evolved as beings with what I like to call the "John Foster Dulles system of morality," so named after Eisenhower's Secretary of State during the Cold War in the 1950s. Dulles hated the Russians, and he knew that the Russians hated him. He felt he had a moral obligation to hate the Russians because if he did not, everything would come tumbling down. But because there was this mutual dislike, of a real obligation-based kind, there was in fact a level of cooperation and harmony. The world did not break down into war and destruction. As a Darwinian, it is plausible to suggest that humans might have evolved with the John Foster Dulles kind of morality, where the highest ethical calling would not be love your neighbor, but hate your neighbor. But remember that your neighbor hates you and, so, you had better not harm him or her because they are going to come straight back at you and do the same.

Now, at the very least, this means that we have the possibility not only of our own (substantive) morality but of an alternative, very different kind of morality: a morality which may have the same formal structure, but which certainly has a different content. The question now is, if there is an objective foundation to substantive morality, which of the two is right? At a minimum, we are left with the possibility that we humans now might be behaving in the way that we do but that, in fact, what is objective morality is something quite else from what we believe. We believe what we do because of our biology, and we believe that because of our biology our substantive morality is objectively justified. But the true objective morality is something other from what we have.

Obviously, this is a sheer contradiction to what most people mean by objective morality. What most people mean by objective morality incorporates the fact that it is going to be self-revealing to human beings. Not necessarily to all human beings but—like Descartes' clear and distinct ideas—certainly self-revealing to all decent human beings who work hard at it. So, given Darwinism, we have a refutation of the existence of such a morality. Darwinian evolutionary biology is non-progressive, pointing away from the possibility of our knowing objective morality. We might be completely deceived, and since objective morality could never allow this, it cannot exist. For this reason, I argue strongly that Darwinian evolutionary theory leads one to a moral skepticism, a kind of moral non-realism.

Remember, incidentally, what was stressed at the beginning of this whole discussion: all of my arguments are based on an assumption of naturalism, methodological naturalism (meaning that science can make no appeal to the supernatural) that is. Is my position necessarily atheistic, denying the existence of God or of morality being something that He decrees? In other words, am I also committed to metaphysical naturalism (meaning that there is no supernatural)? I would say not. It is quite open for the believer to take my position and say that this is the way that God creates morality—not by laying down divine laws by fiat, rather like the laws of mathematics, but by making morality emerge from human nature. In fact, of course, my position paves the way perfectly for a natural law theory of morality, where it is precisely the case that morality is a matter of human nature rather than simply dictates from above (Arnhart, 2005; Ruse, 2009). This gets around the problem with crude divine command theories of morality, which are open to questions like: "Could God have made it okay to rape little old ladies, if he had wanted to?" The answer is: "No, not if he created humans as he did. Our human nature dictates that rape is wrong." I would say that here we are close to the Leibnizian solution to physical evil. Pain from burning is part of the overall human design. Likewise, the moral emotions that we have are part of the human design.

I do recognize that, as so often happens, you cannot quite leave things here. You close off one problem and another emerges. I have argued strongly against progress, but if one is a Christian then one cannot allow that the appearance of humans—or human-like beings, with intelligence and a moral sense—is just contingent. For Christians, humans are part of God's plan. Here I think you need a theological rather than a scientific solution. Don't try to smuggle progress back in. Rather, be Augustinian. God stands outside time. For him, the thought of creation, the act of creation, and the product of creation are as one. Humans could evolve. We know that because they have! I don't know how likely it was: In every universe? In one universe in ten? One

in a million? Or more? The point is that it does not matter to God, whether he creates a billion universes at once or a billion end on end (Ruse, 2010). Time is irrelevant to him. We would emerge somewhere down the line. And we did! It can be just as contingent a process as Gould insists. In fact, Gould himself has gone so far as to say that he thinks human-like beings might emerge in this universe even if we had not done so: "I can present a good argument from 'evolutionary theory' against the repetition of anything like a human body elsewhere; I cannot extend it to the general proposition that intelligence in some form might pervade the universe" (Gould, 1987, p. 407).

So you don't need progress, but you can get humans and their morality, and that is what the Christian needs. Indeed, if I were a Christian, I would just love the conclusions of this paper.

11 Conclusion

This, then, is my counter to folk like Philip Kitcher, and also why I think that the biological sciences can act as a ground for ethics. Should you point out that, far from being very original, my whole position starts to sound very much like that of David Hume, who likewise thought that morality was a matter of psychology rather than a reflection of non-natural objective properties, I shall take this as a compliment, not a criticism. It is indeed true that I regard my position as that of David Hume—brought up to date via the science of Charles Darwin. What better mentors could one have than them?

Postscript: Counterpoint

In a way, Francisco J. Ayala and I are so close together. In a way, Francisco J. Ayala and I are so far apart!

How are we together? We are both ardently committed Darwinian evolutionists and we both believe absolutely and completely that humans are part of this picture. God does not come into it. This does not mean that God does or does not exist. Whatever our personal religious beliefs, Ayala and I respect the integrity of someone who does believe. What it means is that God plays no direct role in the making of humans. It may be his process, but it is a process and not a direct miracle. Again we are together in thinking that morality, ethics, can and must be given a naturalistic explanation. For us, there is no climbing up the mountain and getting the truth on tablets of stone. And finally we are together in thinking that morality is a product of our evolutionarily acquired abilities. We are both evolutionary ethicists.

How are we apart? I want to make morality a direct product of biology, something that came about directly because of natural selection. It is, in the language of the biologists, an adaptation. It is an adaptation like hands and teeth and penises and vaginas. Or rather it is a social adaptation like the pheromone trails that the leaf-cutter ants leave to guide their nest-mates to the bounty. No less, but certainly no more. (Lots more, actually, but still in the world of adaptation.) Ayala wants to

make it all indirect. In the language of the biologists, morality is an exaptation. Morality is not akin to reciprocal altruism or kin selection (or the products thereof) but is something brought about by intelligence (which is a selection-produced adaptation) and hence in the realm of culture and not biology. In other words, morality comes only on the back of real adaptations, and of course might not be or might be quite otherwise.

I see humans (with respect to morality) as absolutely and completely part of the animal world. Ayala sees humans (with respect to morality) as transcending the animal world. It would be easy to sneer and say that this is just what you might expect from a former Catholic priest. But I am not sure that the connection holds. Why am I, a formerly very intense Quaker, not inclined like him? It is probably better to leave the psychology out of it. In any case, I don't think that my position is any less religious than his, meaning that if one were a Christian and wanted to say that God stands behind everything, I don't see why God should not stand behind my position rather than Ayala's. But that perhaps is a topic for another discussion. For now, let me say that I am fascinated by our differences and I hope our readers are, too.

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CHAPTER EIGHTEEN

What the Biological Sciences Can and Cannot Contribute to Ethics

Francisco J. Ayala

The question whether ethical behavior is biologically determined may refer either to the *capacity* for ethics (i.e., the proclivity to judge human actions as either right or wrong), or to the moral *norms* accepted by human beings for guiding their actions. I herein propose: (1) that the capacity for ethics is a necessary attribute of human nature; and (2) that moral norms are products of cultural evolution, not of biological evolution. Humans exhibit ethical behavior by nature because their biological makeup determines the presence of three necessary conditions for ethical behavior: (i) the ability to anticipate the consequences of one's own actions; (ii) the ability to make value judgments; and (iii) the ability to choose between alternative courses of action. Ethical behavior came about in evolution not because it is adaptive in itself, but as a necessary consequence of humans' eminent intellectual abilities, which are an attribute directly promoted by natural selection. That is, morally evolved as an exaptation, not as an adaptation. Since Darwin's time there have been evolutionists proposing that the norms of morality are derived from biological evolution. Sociobiologists represent the most recent and most subtle version of that proposal. The sociobiologists' argument is that human ethical norms are sociocultural correlates of behaviors fostered by biological evolution. I argue that such proposals are misguided and do not escape the naturalistic fallacy. The isomorphism between the behaviors promoted by natural selection and those sanctioned by moral norms exists only with respect to the consequences of the behaviors; the underlying causations are completely disparate.

I fully subscribe to the judgment of those writers who maintain that of all the differences between man and the lower animals, the moral sense or conscience is by far the most important.
Charles Darwin, *The Descent of Man* (1871)

1 Introduction

I will define moral behavior for the present purposes as the actions of a person who takes into account in a sympathetic way the impact the actions have on others.[1] Altruism may be defined in a similar way as, for example, "unselfish regard for or devotion to the welfare of others" (*Webster's New Collegiate Dictionary*, 10th ed.). Altruism, however, is usually taken to imply some cost to the altruist for the benefit of others,[2] and this is the sense in which "altruism" will be used here. I will use the term "ethical behavior" as a synonym of "moral behavior," and "morality" and "ethics" as synonyms of each other, except when explicitly noted or contextually obvious that they are used with a somewhat different meaning. Some authors use "morality" or "virtue ethics" in a broader sense that would include good feelings in regard to others and exclude inappropriate thoughts or desires, such as entertaining sexual desires for somebody else's wife or wishes that something harmful would happen to others. So long as these thoughts or desires are not transformed into actions, they will not be included in my use of "morality." Actions that may be thought to be evil or sinful in some moral systems, such as masturbation, will not be included either in my use of "morality," so long as the actions have no consequences for others.

2 Theories of Morality

People have moral values: that is, they accept standards according to which their conduct is judged either right or wrong, good or evil. The particular norms by which moral actions are judged vary to some extent from individual to individual, and from culture to culture (although some norms, like not to kill, not to steal, and to honor one's parents, are widespread and perhaps universal), but value judgments concerning human behavior are passed in all cultures. This universality raises the questions whether the moral sense is part of human nature, one more dimension of our biological makeup; and whether ethical values may be the product of biological evolution, rather than being given by religious and cultural traditions.

There are many theories concerned with the rational grounds for morality, such as deductive theories that seek to discover the axioms or fundamental principles that determine what is morally correct on the basis of direct moral intuition; or theories like logical positivism or existentialism that negate rational foundations of morality, reducing moral principles to emotional decisions or to other irrational grounds. After the publication of Darwin's theory of evolution by natural selection, several philosophers as well as biologists attempted to find in the evolutionary process the justification for moral behavior.

Aristotle and other philosophers of classical Greece and Rome, as well as many other philosophers throughout the centuries, held that humans hold moral values by nature. A human is not only *Homo sapiens*, but also *Homo moralis*. But biological evolution brings about two important issues: timing and causation. We do not attribute ethical behavior to animals (surely, not to all animals and not to the same extent as to humans, in any case). Therefore, evolution raises distinctive questions about the origins and tenets of moral behavior. When did ethical behavior come about in human evolution? Did modern humans have an ethical sense from the beginning? Did Neanderthals hold moral values? What

about *H. erectus* and *H. habilis*? And how did the moral sense evolve? Was it directly promoted by natural selection? Or did it come about as a by-product of some other attribute (such as rationality) that was the direct target of selection? Alternatively, is the moral sense an outcome of cultural evolution rather than of biological evolution?

3 Darwin and the Moral Sense

Two years after returning from his trip in the HMS *Beagle* (1826–1831), Darwin gathered contemporaneous literature on human moral behavior, including such works as William Paley's *The Principles of Moral and Political Philosophy* (1785), which he had encountered earlier while a student at the University of Cambridge, and the multivolume *Illustrations of Political Economy* by Harriet Martineau, published more recently, in 1832–1834. These two authors, like other philosophers of the time, maintained that morality was a conventional attribute of humankind, rather than a naturally determined human attribute, using an argument often exploited in our days: the diversity of moral codes.

The proliferation of ethnographic voyages had brought to light the great variety of moral customs and rules. This is something Darwin had observed in South American Indians. But this apparent dispersion had not distracted him. He would eventually develop a more complex and subtle theory of the moral sense than those contemporaneous authors; a theory that, implicitly at least, recognized moral behavior as a biologically determined human universal but with culturally evolved differences. For Darwin, the ethnographic diversity of moral customs and rules came about as an adaptive response to the environmental and historical conditions, unique in every different place, without necessarily implying that morality was an acquired, rather than natural, human trait.

A variable adaptive response could very well derive from some fundamental capacity, a common substrate, unique for the whole human race, but capable of becoming expressed in diverse directions. Darwin did not attribute the universality of morality to supernatural origin, but rather saw it as a product of evolution by natural selection. The presence of a universal and common foundation, endowing humans with an ethical capacity, was for Darwin compatible with different cultures manifesting different stages of moral evolution and with different sets of moral norms.

In *The Descent of Man*, Darwin writes:

> Brehm encountered in Abyssinia a great troop of baboons who were crossing a valley: some had ascended the opposite mountain, and some were still on the valley: the latter were attacked by the dogs, but the old males immediately hurried down from the rocks, and with mouths widely opened, roared so fearfully, that the dogs precipitately retreated. They were again encouraged to the attack; but by this time all the baboons had reascended the heights, excepting a young one, about six months old, who, loudly calling for aid, climbed on a block of rock and was surrounded. Now one of the largest males, a true hero, came down again from the mountain, slowly went to the young one, coaxed him, and triumphantly led him away—the dogs being too much astonished to make an attack. (p. 124)

This is just one of the many examples given by Darwin of animals that help a distressed group member. However, in this particular case, Darwin uses a word that deserves

attention: the baboon that comes down from the mountain is called "a true hero." Heroism is an ethical concept. Is Darwin using it in this sense or only metaphorically?

Darwin belongs to an intellectual tradition, originating in the Scottish Enlightenment of the eighteenth and nineteenth centuries, which uses the moral sense as a behavior that, based on sympathy, leads human ethical choice. In his account of the evolution of cooperative behavior, Darwin states that an animal, with well-defined social instincts—like parental and filial affections—"would inevitably acquire a moral sense or conscience, as soon as its intellectual powers had become as well developed, or nearly as well developed, as in man" (Darwin, 1871, pp. 68–69). This is a hypothetical issue, because no other animal has ever reached the level of human mental faculties, language included. But this is an important statement, because Darwin is affirming that the moral sense, or conscience, is a necessary consequence of high intellectual powers, such as exist in modern humans. Therefore, if our intelligence is an outcome of natural selection, so would be the moral sense. Darwin's statement further implies that the moral sense is not by itself directly conscripted by natural selection, but only indirectly as a consequence of high intellectual powers.

4 Moral Behavior vs. Moral Norms

Darwin also states that even if some animal could achieve a human-equivalent degree of development of its intellectual faculties, we cannot conclude that it would also acquire exactly the same moral sense as ours. "I do not wish to maintain that any strictly social animal, if its intellectual faculties were to become as active and as highly developed as in man, would acquire the same moral sense as ours. . . . [T]hey might have a sense of right and wrong, though led by it to follow widely different lines of conduct" (Darwin, 1871, p. 70). These statements imply that, according to Darwin, having a moral sense does not by itself determine what the moral norms would be: which sorts of actions might by sanctioned by the norms and which ones would be condemned.

This distinction is important. Indeed, it is a distinction central to my thesis herein. Much of the historical controversy, particularly between scientists and philosophers, as to whether the moral sense is or not biologically determined has arisen owing to a failure to make the distinction. Scientists often affirm that morality is a human biological attribute because they are thinking of the predisposition to pass moral judgment: that is, to judge some actions as good and others as evil. Some philosophers argue that morality is not biologically determined, but rather comes from cultural traditions or from religious beliefs, because they are thinking about moral codes, the sets of norms that determine which actions are judged to be good and which are evil. They point out that moral codes vary from culture to culture and, therefore, are not biologically predetermined.

I consider this distinction fundamental. Thus, I'll argue that the question of whether ethical behavior is biologically determined may refer to either one of the following two issues. First, is the capacity for ethics—the proclivity to judge human actions as either right or wrong—determined by the biological nature of human beings? Second, are the systems or codes of ethical norms accepted by human beings biologically determined? A similar distinction can be made with respect to language. The

question whether the capacity for symbolic creative language is determined by our biological nature is different from the question whether the particular language we speak—English, Spanish, Chinese, etc.—is biologically determined, which in the case of language obviously it is not.

The distinction between the *inclination* to judge certain sorts of actions as either morally good or evil and the *norms* according to which we determine which actions are good and which actions are evil, has affinity with the distinction made by moral philosophers between *meta-ethics* and *normative* ethics. The subject of meta-ethics is why we ought to do what we ought to do, while normative ethics tells us what we ought to do. I will propose that the moral evaluation of actions emerges from human rationality, or, in Darwin's terms, from our highly developed intellectual powers. Our high intelligence allows us to anticipate the consequences of our actions with respect to other people and, thus, to judge them as good or evil in terms of their consequences for others. But I will propose that the norms according to which we decide which actions are good and which actions are evil are largely culturally determined, although conditioned by biological predispositions.

5 Darwinian Aftermath

Herbert Spencer (1820–1903) was among the first philosophers seeking to find the grounds of morality in biological evolution. In *The Principles of Ethics* (1893), Spencer seeks to discover values that have a natural foundation. Spencer argues that the theory of organic evolution implies certain ethical principles. Human conduct must be evaluated, like any biological activity whatsoever, according to whether it conforms to the life process; therefore, any acceptable moral code must be based on natural selection, the law of struggle for existence. According to Spencer, the most exalted form of conduct is that which leads to a greater duration, extension, and perfection of life; the morality of all human actions must be measured by that standard. Spencer proposes that, although exceptions exist, the general rule is that pleasure goes with that which is biologically useful, whereas pain marks what is biologically harmful. This is an outcome of natural selection; thus, while doing what brings them pleasure and avoiding what is painful, organisms improve their chances for survival. With respect to human behavior, we see that we derive pleasure from virtuous behavior and pain from evil actions, associations which indicate that the morality of human actions is also founded on biological nature.

Spencer proposes as the general rule of human behavior that anyone should be free to do anything that they want, so long as it does not interfere with the similar freedom to which others are entitled. The justification of this rule is found in organic evolution: the success of an individual, plant or animal, depends on its ability to obtain that which it needs. Consequently, Spencer reduces the role of the state to protecting the collective freedom of individuals so that they can do as they please. This laissez-faire form of government may seem ruthless, because individuals would seek their own welfare without any consideration for others' (except for respecting their freedom), but Spencer believes that it is consistent with traditional Christian values. It may be added that, although Spencer sets the grounds of morality on biological nature and on nothing else, he admits that certain moral norms go beyond

that which is biologically determined; these are rules formulated by society and accepted by tradition.

Social Darwinism, in Spencer's version or in some variant form, was fashionable in European and American circles during the latter part of the nineteenth century and the early years of the twentieth century, but it has few or no distinguished intellectual followers at present. Spencer's critics include the evolutionists Julian Huxley and C.H. Waddington, who, nevertheless, maintain that organic evolution provides grounds for a rational justification of ethical codes. For Huxley (1953; T. Huxley & Huxley, 1947), the standard of morality is the contribution that actions make to evolutionary progress, which goes from less to more "advanced" organisms. For Waddington (1960), the morality of actions must be evaluated by their contribution to human evolution.

Huxley and Waddington's views are based on value judgments about what is or is not progressive in evolution. But, contrary to Huxley's claim, there is nothing objective in the evolutionary process itself (i.e., outside human considerations; see Ayala, 1982, 1987) that makes the success of bacteria, which have persisted as such for more than two billion years and which consist of a huge diversity of species and astronomic numbers of individuals, less desirable than that of the vertebrates, even though the latter are more complex. The same objection can be raised against Waddington's human evolution standard of biological progress. Are the insects, of which more than one million species exist, less desirable or less successful from a purely biological perspective than humans or any other mammal species? Waddington fails to demonstrate why the promotion of human biological evolution by itself should be the standard to measure what is morally good.

More recently, numerous philosophers as well as scientists have sought to give accounts of moral behavior as an evolutionary outcome (e.g., Blackmore, 1999; Hauser, 2006; Maienschein & Ruse, 1999; Ruse, 1995; Sober & Wilson, 1998). Particularly notable are the contributions of Edward O. Wilson (1975, 1978, 1998), founder of sociobiology as an independent discipline engaged in discovering the biological foundations of all social behavior. Wilson and other sociobiologists, as well as the derivative subdisciplines of evolutionary psychology (e.g., Barkow, Cosmides, & Tooby, 1992) and memetics (Blackmore, 1999), have sought to solve the naturalistic fallacy by turning it on its head. They assert that moral behavior does not exist as something distinct from biological, or biologically determined, behavior. As Ruse and Wilson (1985) have asserted, "Ethics is an *illusion* [italics added] put in place by natural selection to make us good cooperators" (p. 50). I shall return later to these sociobiological and related proposals.

6 Moral Behavior as Rational Behavior

The first proposition I will defend here is that humans, because of their high intellectual powers, are necessarily inclined to make moral judgments and to accept ethical values: that is, to evaluate certain kinds of actions as either right or wrong. The claim I make is that moral behavior is a necessary outcome of the biological makeup of humans, a product of their evolution. This view would fall within the meta-ethical theories known as deontological or rational. It is the exalted degree of

rationality that we humans have achieved that makes us moral beings. Humans are *Homo moralis* because they are *Homo rationalis*.

This thesis does not imply that the norms of morality are also biologically determined or that they are unambiguous consequences of our rationality. Independent of whether or not humans have a biologically determined moral sense, it remains to be ascertained whether particular moral prescriptions are in fact determined by the biological nature of humans, or whether they are products of cultural evolution, be these chosen by society or established by religious beliefs, or even selected according to individual preferences. Even if we were to conclude that people cannot avoid having moral standards of conduct, it might be that the choice of the particular standards used for judgment would be arbitrary or a product of cultural evolution. The need for having moral values does not necessarily tell us what the moral values should be, like the capacity for language does not determine which language we shall speak.

I will first argue that humans are ethical beings by their biological nature: that humans evaluate their behavior as either right or wrong, moral or immoral, as a consequence of their eminent intellectual capacities, which include self-awareness and abstract thinking. These intellectual capacities are products of the evolutionary process, but they are distinctively human. Thus, I will assert that ethical behavior is not causally related to the social behavior of animals, including kin selection and the so-called "reciprocal altruism."

A second argument that I will put forward is that the moral norms according to which we evaluate particular actions as either morally good or morally bad (as well as the grounds that may be used to justify the moral norms) are products of cultural evolution, not of biological evolution. The norms of morality belong, in this respect, to the same category of phenomena as political and religious institutions, or the arts, sciences, and technology, as well as the particular languages we speak. The moral codes, like these other products of human culture, are often consistent with the biological predispositions of the human species, and of other animals. But many moral norms are formulated independently of biological necessity or predisposition, simply because they don't have necessary biological consequences. Biological welfare (survival and reproduction) is not determinant of all ethical norms in any given society or culture.

Moral codes, like any other cultural system, depend on the existence of human biological nature and must be consistent with it in the sense that they could not counteract it without promoting their own demise. Moreover, the acceptance and persistence of moral norms is facilitated whenever they are consistent with biologically conditioned human behaviors. But the moral norms are independent of such behaviors in the sense that some norms may not favor, and may hinder, the survival and reproduction of the individual and its genes, which processes are the targets of biological evolution. Discrepancies between accepted moral rules and biological survival are, however, necessarily limited in scope or would otherwise lead to the extinction of the groups accepting such discrepant rules.

7 Biology to Ethics

I will now refer to the moral sense in its strict meaning as the evaluation of some actions as virtuous, or morally good, and others as evil, or morally bad. Morality in

this sense is the urge or predisposition to judge human actions as either right or wrong in terms of their consequences for other human beings. In this sense, humans are moral beings by nature because their biological constitution determines the presence in them of the three necessary conditions for ethical behavior. These conditions are: (i) the ability to anticipate the consequences of one's own actions; (ii) the ability to make value judgments; and (iii) the ability to choose between alternative courses of action. These abilities exist as a consequence of the eminent intellectual capacity of human beings.[3]

The ability to anticipate the consequences of one's own actions is the most fundamental of the three conditions required for ethical behavior. Only if I can anticipate that pulling the trigger will shoot the bullet, which in turn will strike and kill my enemy, can the action of pulling the trigger be evaluated as nefarious. Pulling a trigger is not in itself a moral action; it becomes so by virtue of its relevant consequences. My action has an ethical dimension only if I do anticipate these consequences.

The ability to anticipate the consequences of one's actions is closely related to the ability to establish the connection between means and ends: that is, of seeing a means precisely as a means, as something that serves a particular end or purpose. This ability to establish the connection between means and their ends requires the ability to anticipate the future and to form mental images of realities not present or not yet in existence.

The ability to establish the connection between means and ends happens to be the fundamental intellectual capacity that has made possible the development of human culture and technology. An evolutionary scenario, seemingly the best hypothesis available, proposes that the remote evolutionary roots of this capacity to connect means with ends may be found in the evolution of bipedalism, which transformed the anterior limbs of our ancestors from organs of locomotion into organs of manipulation. The hands thereby gradually became organs adept for the construction and use of objects for hunting and other activities that improved survival and reproduction: that is, which increased the reproductive fitness of their carriers. The construction of tools depends not only on manual dexterity, but on perceiving them precisely as tools, as objects that help to perform certain actions: that is, as means that serve certain ends or purposes—a knife for cutting, an arrow for hunting, an animal skin for protecting the body from the cold. According to this evolutionary scenario, natural selection promoted the intellectual capacity of our bipedal ancestors because increased intelligence facilitated the perception of tools as tools, and therefore their construction and use, with the ensuing amelioration of biological survival and reproduction.

The development of the intellectual abilities of our ancestors took place over several million years, gradually increasing the ability to connect means with their ends and, hence, the possibility of making ever more complex tools serving more remote purposes. According to the hypothesis I am proposing, the ability to anticipate the future, essential for ethical behavior, is therefore closely associated with the development of the ability to construct tools, an ability that has produced the advanced technologies of modern societies and that is largely responsible for the success of humans as a biological species.

The second condition for the existence of ethical behavior is the ability to make value judgments, to perceive certain objects or deeds as more desirable than others.

Only if I can see the death of my enemy as preferable to his survival (or vice versa) can the action leading to his demise be thought of as moral. If the consequences of alternative actions are neutral with respect to value, an action cannot be characterized as ethical. Values are of many sorts: not only ethical, but also aesthetic, economic, gastronomic, political, and so on. But in all cases, the ability to make value judgments depends on the capacity for abstraction: that is, on the capacity to perceive actions or objects as members of general classes. This makes it possible to compare objects or actions with one another and to perceive some as more desirable than others. The capacity for abstraction requires an advanced intelligence such as it exists in humans and apparently in them alone.

I will note at this point that the model that I am advancing here does not necessarily imply the ethical theory known as utilitarianism (or, more generally, consequentialism). According to so-called "act consequentialism," the rightness of an action is determined by the value of its consequences, so that the morally best action in a particular situation is the one the consequences of which would have the most benefit to others. I am proposing that the morality of an action depends on our ability (1) to anticipate the consequences of our actions, and (2) to pass value judgments. But I am not asserting that the morality of actions is exclusively measured in terms of how beneficial their consequences will be to others.

The third condition necessary for ethical behavior is the ability to choose between alternative courses of actions. Pulling the trigger can be a moral action only if you have the option not to pull it. A necessary action beyond conscious control is not a moral action: the circulation of the blood or the process of food digestion is not a moral action. Whether there is free will is a question much discussed by philosophers, and the arguments are long and involved.[4] Here, I will advance two considerations that are common-sense evidence of the existence of free will. One is personal experience, which indicates that the possibility to choose between alternatives is genuine rather than only apparent. The second consideration is that when we confront a given situation that requires action on our part, we are able mentally to explore alternative courses of action, thereby extending the field within which we can exercise our free will. In any case, if there were no free will, there would be no ethical behavior; morality would only be an illusion. A point to be made, however, is that free will is dependent on the existence of a well-developed intelligence, which makes it possible to explore alternative courses of action and to choose one or another in view of the anticipated consequences.

8 Adaptation vs. Exaptation

I will now consider explicitly two issues that are largely implicit in the previous section. I have proposed that the moral sense emerges as a necessary implication of our high intellectual powers, which allow us to anticipate the consequences of our actions and evaluate such consequences. But is it the case that the moral sense may have been promoted by natural selection in itself and not only indirectly as a necessary consequence of our exalted intelligence? The question in evolutionary terms is whether the moral sense is an adaptation or, rather, an exaptation. Evolutionary biologists define exaptations as features of organisms that evolved because they served

some function, but are later co-opted to serve a different function, which was not originally the target of natural selection. The new function may replace the older function or coexist together with it. Feathers seem to have evolved first for conserving temperature, but were later co-opted in birds for flying. The beating of the human heart is an exaptation used by doctors to diagnose the state of health, although this is not why it evolved in our ancestors. The issue at hand is whether moral behavior was directly promoted by natural selection or rather whether it is a consequence of our exalted intelligence, which was the target of natural selection, because it made possible the construction of better tools. Art, literature, religion, and many other human cultural activities might also be seen as exaptations that came about as consequences of high intelligence and tool making.

The second issue is whether some animals, apes or other non-human primates, for example, may have a moral sense, however incipient, either as directly promoted by natural selection or as a consequence of their own intelligence.

The position that I'll argue here is that the human moral sense is an exaptation, not an adaptation. The moral sense consists of *judging* certain actions as either right or wrong; not of choosing and carrying out some actions rather than others, or evaluating them with respect to their practical consequences. It seems unlikely that making moral judgments would promote the reproductive fitness of those judging an action as good or evil. Nor does it seem likely that there might be some form of "incipient" ethical behavior that would then be further promoted by natural selection. The three necessary conditions for there being ethical behavior are manifestations of advanced intellectual abilities.

It, indeed, rather seems that the target of natural selection was the development of the advanced intellectual capacities. This was favored by natural selection because the construction and use of tools improved the strategic position of our biped ancestors. In the account I am advancing here, once bipedalism evolved and after tool using and tool making became practical, those individuals more effective in these functions had a greater probability of biological success. The biological advantage provided by the design and use of tools persisted long enough so that intellectual abilities continued to increase, eventually yielding the eminent development of intelligence that is characteristic of *Homo sapiens*.

A related question is whether morality would benefit a social group within which it is practiced, and, indirectly, individuals as members of the group. This seems likely to be the case if indeed moral judgment would influence individuals to behave in ways that increase cooperation, or benefit the welfare of the social group in some way: for example, by reducing crime or protecting private property. This brings about the issue of whether there is in humans "group selection," and the related issues of kin selection and inclusive fitness, which I will discuss below.

Group selection based on altruistic behavior is generally not an evolutionary stable strategy (ESS). This is because mutations that favor selfish over altruistic behavior will be favored by natural selection within a given population, so that selfish alleles will drive out altruistic alleles. Of course, it may be the case that populations with a preponderance of altruistic alleles will survive and spread better than populations consisting of selfish alleles. This would be group selection. But typically there are many more individual organisms than there are populations; and individuals are born, procreate, and die at rates much higher than populations. Thus, the rate of

multiplication of selfish individuals over altruists is likely to be much higher than the rate at which altruistic populations multiply relative to predominantly selfish populations.

There is, however, an important difference between animals and humans that is relevant in this respect. Namely, the fitness advantage of selfish over altruistic behavior does not apply to humans, because humans can *understand* the benefits of altruistic behavior (to the group and indirectly to them) and thus adopt altruism and protect it, by laws or otherwise, against selfish behavior that harms the social group. As Darwin wrote in *The Descent of Man*:

> It must not be forgotten that, although a high standard of morality gives but a slight or no advantage to each individual man and his children over the other men of the same tribe, yet that an advancement in the standard of morality and an increase in the number of well-endowed men will certainly give an immense advantage to one tribe over another. (Darwin, 1871, p. 159)

The theory of sociobiology advances a ready answer to the second question raised above, whether morality occurs in other animals, even if only as a rudiment. The theory of kin selection, sociobiologists argue, explains altruistic behavior, to the extent that it exists in other animals as well as in humans. I will propose, however, that moral behavior properly so does not exist, even incipiently, in non-human animals. The reason is that the three conditions required for ethical behavior depend on an advanced intelligence—which includes the capacities for free will, abstract thought, and anticipation of the future—such as it exists in *H. sapiens* and not in any other living species. It is the case that certain animals exhibit behaviors analogous with those resulting from ethical actions in humans, such as the loyalty of dogs or the appearance of compunction when they are punished. But such behaviors are either genetically determined or elicited by training (conditioned responses). Genetic determination and not moral evaluation is also what is involved in the altruistic behavior of social insects and other animals. I will argue below that biological altruism (altruism_b) and moral altruism (altruism_m) have disparate causes: kin selection in altruism_b, regard for others in altruism_m.

The capacity for ethics is an outcome of gradual evolution, but it is an attribute that only exists when the underlying attributes (i.e., the intellectual capacities) reach an advanced degree. The necessary conditions for ethical behavior only come about after the crossing of an evolutionary threshold. The approach is gradual, but the conditions only appear when a degree of intelligence is reached such that the formation of abstract concepts and the anticipation of the future are possible, even though we may not be able to determine when the threshold was crossed. Thresholds occur in other evolutionary developments—for example, in the origins of life, multicellularity, and sexual reproduction—as well as in the evolution of abstract thinking and self-awareness. Thresholds also occur in the physical world: for example, water heats gradually, but at 100°C boiling begins and the transition from liquid to gas starts suddenly. Surely, human intellectual capacities came about by gradual evolution. But when looking at the world of life as it exists today, it would seem that there is a radical breach between human intelligence and that of other animals. The rudimentary cultures that exist in chimpanzees (Whiten et al., 1999; Whiten, Horner, & de Waal, 2005) do not imply advanced intelligence as it is required for moral behavior.

The question remains: when did morality emerge in the human lineage? Did *Homo habilis* or *Homo erectus* have morality? What about the Neanderthals, *Homo neanderthalensis*? When in hominid evolution morality emerged is difficult to determine. It may very well be that the advanced degree of rationality required for moral behavior may only have been reached at the time when creative language came about, and perhaps in dependence with the development of creative language. When creative language may have come about in human evolution is, however, a question that transcends the scope of this essay.[5]

9 Whence Moral Codes?

I have distinguished between moral behavior—judging some actions as good, others as evil—and moral codes—the precepts or norms according to which actions are judged. Moral behavior, I have proposed, is a biological attribute of *H. sapiens*, because it is a necessary consequence of our biological makeup, namely, our high intelligence. But moral codes, I argue, are products not of biological evolution, but of cultural evolution.

It must, first, be stated that moral codes, like any other cultural systems, cannot survive for long if they run in outright contrast to our biology. The norms of morality must be consistent with biological nature, because ethics can only exist in human individuals and in human societies. One might therefore also expect, and it is the case, that accepted norms of morality will promote behaviors that increase the biological fitness of those who behave according to them, such as child care. But the correlation between moral norms and biological fitness is neither necessary nor indeed always the case: some moral precepts common in human societies have little or nothing to do with biological fitness and some moral precepts are contrary to fitness interest.

Before going any further, it seems worthwhile to consider briefly the proposition that the justification of the codes of morality derives from religious convictions and only from them. There is no necessary, or *logical*, connection between religious faith and moral principles, although there usually is a motivational or psychological connection. Religious beliefs do explain why people accept particular ethical norms, because they are motivated to do so by their religious convictions. But in following the moral dictates of one's religion, one is not rationally justifying the moral norms that one accepts. It may, of course, be possible to develop such rational justification: for example, when a set of religious beliefs contains propositions about human nature and the world, from which a variety of ethical norms can be logically derived. Indeed, religious authors, including, for example, Christian theologians, do often propose to justify their ethics on rational foundations concerning human nature. But in this case, the logical justification of the ethical norms does not come from religious faith as such, but from a particular conception of the world; it is the result of philosophical analysis grounded on certain premises.

It may well be that the motivational connection between religious beliefs and ethical norms is the decisive one for the religious believer. But this is true in general: most people, religious or not, accept a particular moral code for social reasons, without trying to justify it rationally by means of a theory from which the moral norms can be logically derived. They accept the moral codes that prevail in their

societies, because they have learned such norms from parents, school, or other authorities. The question therefore remains: how do moral codes come about?

The short answer is, as already stated, that moral codes are products of cultural evolution, a distinctive human mode of evolution that has surpassed the biological mode, because it is a more effective form of adaptation; it is faster than biological evolution and it can be directed. Cultural evolution is based on cultural heredity, which is Lamarckian, rather than Mendelian, so that acquired characteristics are transmitted. Most important, cultural heredity does not depend on biological inheritance, from parents to children, but is transmitted also horizontally and without biological bounds. A cultural mutation, an invention (think of the laptop computer, the cell phone, or rock music), can be extended to millions and millions of individuals in less than one generation.

Darwin's Chapter V of *The Descent of Man* (1871) is entitled, "On the Development of the Intellectual and Moral Faculties during Primeval and Civilized Times." There, he writes:

> There can be no doubt that a tribe including many members who, from possessing in a high degree the spirit of patriotism, fidelity, obedience, courage, and sympathy, were always ready to give aid to each other and to sacrifice themselves for the common good, would be victorious over most other tribes; and this would be natural selection. At all times throughout the world tribes have supplanted other tribes; and as morality is one element in their success, the standard of morality and the number of well-endowed men will thus everywhere tend to rise and increase. (pp. 159–160)

Darwin is making two important assertions. First, morality may contribute to the success of some tribes over others, which is natural selection in the form of group selection. Second, the standards of morality will tend to improve over human history, because the higher the standards of a tribe, the more likely the success of the tribe. This assertion depends on which standards are thought to be "higher" than others. If the higher standards are defined by their contribution to the success of the tribe, then the assertion is circular. But Darwin asserts that there are some particular standards that, in his view, would contribute to tribal success: patriotism, fidelity, obedience, courage, and sympathy.

10 Sociobiology's Account of Moral Behavior

Darwin was puzzled by the social organization and behavior of hymenopterans: bees, wasps, ants, and termites. Consider Meliponinae bees, with hundreds of species across the tropics. These stingless bees have typically single-queen colonies with hundreds to thousands of workers. The queen generally mates only once. The worker bees toil, building the hive and feeding and caring for the eggs and larvae, even though they themselves are sterile and only the queen produces progeny. Assume that in some ancestral hive, a gene arises that prompts worker bees to behave as they now do. It would seem that such a gene would not be passed on to the following generation because such worker bees do not reproduce. But such inference would be erroneous.

Meliponinae bees, like other hymenopterans, have a haplo-diploid system of reproduction. Queen bees produce two kinds of eggs: some are unfertilized and develop into males (which are therefore haploid: i.e., they carry only one set of genes); others are fertilized (hence, are diploid: i.e., they carry two sets of genes) and develop into worker bees and occasionally into a queen. W.D. Hamilton (1964) demonstrated that with such a reproductive system the queen's daughters share in three-quarters of their genes among them, whereas the queen's daughters and their mother share in only one-half of their genes. Hence, the worker-bee genes are more effectively propagated by workers caring for their sisters than if they would produce and care for their own daughters. Natural selection can thus explain the existence in social insects of sterile castes, which exhibit a most extreme form of apparently altruistic behavior by dedicating their life to care for the progeny of another individual, the queen.

Hamilton's discovery solved the mystery that had puzzled Darwin and had continued puzzling specialists in hymenopteran biology and other evolutionists for somewhat more than a century. In 1975, the notable Harvard ant specialist Edward O. Wilson published *Sociobiology: the New Synthesis*, a treatise appropriately considered the founding document of the new discipline of sociobiology. The last chapter of the book was concerned with the social organization of human societies, with the telling title "Man: From Sociobiology to Sociology," and with sections dedicated to "Culture, Ritual, and Religion" and "Ethics." The first sentence of the "Ethics" section startled many readers: "Scientists and humanists should consider together the possibility that the time has come for ethics to be removed temporarily from the hands of the philosophers and biologicized" (p. 562). Wilson (1975, 1978, 1998), like other sociobiologists (Alexander, 1979; Barash, 1977; see also Kitcher, 1985; Ruse, 2000, 2006; Sober & Wilson, 1998), sees that sociobiology may provide the key for finding a naturalistic basis for ethics.

According to Wilson (1975): "The requirement for an evolutionary approach to ethics is self-evident. It should also be clear, for example, that no single set of moral standards can be applied to all human populations, let alone all sex-age classes within each population. To impose a uniform code is therefore to create complex, intractable moral dilemmas" (p. 564). Moral pluralism is, for Wilson, "innate." It seems, therefore, that, according to Wilson, biology helps us at the very least to decide that certain moral codes (e.g., all those pretending to be universally applicable) are incompatible with human nature and therefore unacceptable.

However, Wilson (1978) goes further when he writes: "Human behavior—like the deepest capacities for emotional response which drive and guide it—is the circuitous technique by which human genetic material has been and will be kept intact. *Morality has no other demonstratable ultimate function* [italics added]" (p. 167). How is one to interpret this statement? It is possible that Wilson is simply giving the reason why ethical behavior exists at all; his proposition would be that humans are prompted to evaluate morally their actions as a means to preserve their genes, their biological nature. But this proposition is, in my view, erroneous. Human beings are by nature ethical beings in the sense I have expounded: they judge morally their actions because of their innate ability for anticipating the consequences of their actions, for formulating value judgments, and for free choice. Human beings exhibit ethical behavior by nature and necessity, rather than because such behavior would help to preserve their genes or serve any other purpose.

Wilson's statement may alternatively be read as a justification of human moral codes: the function of these would be to preserve human genes. But this would entail the naturalistic fallacy[6] and, worse yet, would seem to justify a morality that most people detest. If the preservation of human genes (be those of the individual or of the species) is the purpose that moral norms serve, Spencer's Social Darwinism would seem right; racism or even genocide could be justified as morally correct, if they were perceived as the means to preserve those genes thought to be good or desirable and to eliminate those thought to be bad or undesirable. Surely, however, Wilson is not intending to justify racism or genocide.

I believe that what Wilson and other sociobiologists are saying is something else, something of great philosophical import that has been stated, with characteristic verve and clarity, by Michael Ruse in the companion paper (as well as elsewhere: Ruse & Wilson, 1985): "To be blunt, my Darwinism says that substantive morality is a kind of *illusion* [italics added], put in place by our genes, in order to make us good social cooperators." Ruse (in the companion paper) proceeds to explain why the illusion of ethics is a powerful adaptation:

> I would add that the reason why the illusion is such a successful adaptation is that not only do we believe in substantive morality, but we also believe that substantive morality does have an objective foundation. An important part of the phenomenological experience of substantive ethics is not just that we feel that we ought to do the right and proper thing, but that we feel that we ought to do the right and proper thing because it truly is the right and proper thing.

The deceit perpetrated on us by our genes is complete: "There are in fact no foundations, but we believe that there are in some sense foundations."

How come that "selfish genes" move us to act altruistically and otherwise behave in ways that seem morally right? The answer comes, according to sociobiologists, from the theory of kin selection that explains the altruism of haplo-diploid insects and much more, as well as from other related theoretical constructs such as inclusive fitness and reciprocal altruism. The sociobiologists' argument concerning normative ethics is not that the norms of morality can be grounded in biological evolution, but rather that evolution predisposes us to accept certain moral norms, namely, those that are consistent with the "objectives" of natural selection. It is because of this predisposition that human moral codes sanction patterns of behavior similar to those encountered in the social behavior of animals. According to sociobiologists, the commandment to honor one's parents, the incest taboo, the greater blame usually attributed to the wife's adultery than to the husband's, and the banning or restriction of divorce are among the numerous ethical precepts and practices that endorse behaviors that are promoted by natural selection. The sociobiologists reiterate their conviction that science and ethics belong to separate logical realms; that one may not infer what is morally right or wrong from a determination of how things are or are not in nature. The sociobiologists avoid the naturalistic fallacy by the drastic move of denying that ethical behavior exists as an activity with different causation than any other activities or traits simply determined by our genes. Ethical behavior is simply an expression of our genes and a direct consequence of natural selection as it adapts humans, as well as other organisms, to their environments.

Evolutionists had for years struggled to find an explanation for the apparently altruistic behavior of animals. When a predator attacks a herd of zebras, adult males attempt to protect the young in the herd, even if they are not their progeny, rather than fleeing. When a prairie dog sights a coyote, it will warn other members of the colony with an alarm call, even though by drawing attention to itself this increases its own risk. Darwin tells the story of adult baboons protecting the young. Examples of altruistic behaviors of this kind can be multiplied. But to speak of animal altruism is to claim not that explicit feelings of devotion or regard are present in them, but rather that animals act for the welfare of others at their own risk just as humans are expected to do when behaving altruistically.

The problem is precisely how to justify such behaviors in terms of natural selection. Assume, for illustration, that in a certain species there are two alternative forms of a gene (two alleles), of which one but not the other promotes altruistic behavior. Individuals possessing the altruistic allele will risk their life for the benefit of others, whereas those possessing the non-altruistic allele will benefit from altruistic behavior without risking themselves. Possessors of the altruistic allele will be more likely to die or fail to reproduce, and the allele for altruism will therefore be eliminated more often than the non-altruistic allele. Eventually, after some generations, the altruistic allele will be completely replaced by the non-altruistic one. But then, how is it that altruistic behaviors are common in animals without the benefit of ethical motivation? The explanation comes from the theory of kin selection.

To ascertain the consequences of natural selection it is necessary to take into account a gene's effects not only on a particular individual but also on all individuals possessing that gene, as in the explanation of the social organization of bees and other hymenopterans. When considering altruistic behavior, one must take into account not only the risks for the altruistic individual, but also the benefits for other possessors of the same allele. Zebras live in herds where individuals are blood relatives. This is also the case for baboon troops. A gene prompting adults to protect the defenseless young would be favored by natural selection if the benefit (in terms of saved individuals that are carriers of that gene) is greater than the cost (due to the increased risk of the protectors). An individual that lacks the altruistic gene and carries instead a non-altruistic one will not risk its life, but the non-altruistic gene is partially eradicated with the death of each defenseless relative.

It follows from this line of reasoning that the more closely related the members of a herd, troop, or animal group are, the more altruistic behavior should be present. This seems to be generally the case. Consider parental care. Parental care is most obvious in the genetic benefits it entails. Parents feed and protect their young because each child has half the genes of each parent: the genes are protecting themselves, as it were, when they prompt a parent to care for its young. That is why parental care is widespread among animals.

Sociobiologists point out that many of the moral norms commonly accepted in human societies sanction behaviors also promoted by natural selection, such as the commandment to honor one's parents and the incest taboo, as pointed out above. Once again, the sociobiologists' argument is that human ethical norms are sociocultural correlates of behaviors fostered by biological evolution. Ethical norms protect such evolution-determined behaviors as well as being specified by them.

I, however, believe that the sociobiologists' arguments to that effect are misguided. Consider altruism as an example. Altruism in the biological sense ($altruism_b$) is defined in terms of the population genetic consequences of a certain behavior. $Altruism_b$ is explained by the fact that genes prompting such behavior are actually favored by natural selection (when inclusive fitness is taken into account), even though the fitness of the behaving individual is decreased. But altruism in the moral sense ($altruism_m$) is explained in terms of motivations: a person chooses to risk his/her own life (or incur some kind of cost) for the benefit of somebody else. The similarity between $altruism_b$ and $altruism_m$ is only with respect to the consequences: an individual's chances are improved by the behavior of another individual who incurs a risk or cost. The underlying causations are completely disparate: the ensuing genetic benefits in $altruism_b$; regard for others in $altruism_m$. As Darwin put it, the behavior of a baboon and a human are similar in that they both save an infant (from the dogs or from drowning), but they differ in that humans carry out an assessment, which baboons do not. As I have argued above, humans make moral judgments as a necessary consequence of their eminent intellectual abilities. Their judgments, as well as the moral norms on which they are based, are not always accompanied by biological gain.

Parental care is a behavior generally favored by natural selection, which may also be present in virtually all codes of morality, from primitive to more advanced societies. There are other human behaviors sanctioned by moral norms that have biological correlates favored by natural selection. One example is monogamy, which occurs in some animal species but not in many others. It is also sanctioned in many human cultures, but surely not in all. Polygamy is sanctioned in some current human cultures and surely was more so in the past. Food sharing outside the mother–offspring unit rarely occurs in primates, with the exception of chimpanzees and capuchin monkeys, although even in chimpanzees food sharing is highly selective and often associated with reciprocity. A more common form of mutual aid among primates is coalition formation; alliances are formed in fighting other conspecifics, although these alliances are labile, with partners readily changing partners.

One interesting behavior, associated with a sense of justice, or equal pay for equal work, has been described by Sarah Brosnan and Frans de Waal (2003; see also de Waal, 1996) in the brown capuchin monkey, *Cebus paella*. Monkeys responded negatively to unequal rewards in exchanges with a human experimenter. Monkeys refused to participate in an exchange when they witnessed that a conspecific had obtained a more attractive reward for equal effort.

Is the capuchin behavior phylogenetically related to the human virtue of justice? This seems unlikely, since similar behavioral patterns have not been observed in other primates, including apes, phylogenetically closer to humans. Cannibalism is practiced by chimps, as well as by human cultures of the past. Do we have a phylogenetically acquired predisposition to cannibalism as a morally acceptable behavior? This seems unlikely. Moral codes arise in human societies by cultural evolution. Those moral codes tend to be widespread that lead to successful societies.

Since time immemorial, human societies have experimented with moral systems. Some have succeeded and spread widely through humankind, like the Ten Commandments, although other moral systems persist in different human societies. Many moral systems of the past have surely become extinct because they were replaced

or because the societies that held them became extinct. The moral systems that currently exist in humankind are those that were favored by cultural evolution. They were propagated within particular societies for reasons that might be difficult to fathom, but that surely must have included the perception by individuals that a particular moral system was beneficial for them, at least to the extent that it was beneficial for their society by promoting social stability and success. Acceptance of some precepts in many societies is reinforced by civil authority (e.g., those who kill or commit adultery will be punished) and by religious beliefs (God is watching and you'll go to hell if you misbehave). Legal and political systems, as well as belief systems, are themselves outcomes of cultural evolution.

Postscript: Counterpoint

According to Edward O. Wilson (1992), progress "is a property of the evolution of life as a whole by almost any conceivable intuitive standard, including the acquisition of goals and intentions in the behavior of animals" (p. 187).

Herbert Spencer (1851) was perhaps the first philosopher seeking to find the grounds of morality in biology. Spencer argued that the theory of evolution implies certain ethical principles: any acceptable moral code must be based on natural selection, the law of struggle for existence. The morality of human actions must be measured by their contribution to the greater duration, extension, and perfection of life: that is, as they conform to the progress of life, because this is the goal of evolution as promoted by natural selection. Julian Huxley (1927, 1953) did not quite endorse Spencer's view of evolution as progressive on the whole, but there are privileged lines of evolutionary progress as it occurs, for example, in the evolution toward higher levels of organization (more complex organisms), from amoeba to animals and from fish to mammals and, ultimately, humans.

I join Ruse (1995 and this volume) in his unambiguous rejection of any efforts to justify ethical values on the supposedly progressive character of the evolutionary process. "It is far from obvious," he writes in his companion paper, "either that natural selection promotes progress or that progress actually occurs in any clear definable and quantifiable way." I would add that this is the case because the concept of progress contains two elements: one descriptive—that directional change has occurred; the other axiological—that the change represents an improvement or betterment. The notion of progress requires that a value judgment be made of what is better and what is worse, or what is higher and what is lower, according to some axiological standard (Ayala, 1974).

Ruse's next move amounts, however, to throwing the baby out with the bathwater: morality does not exist in objective reality, except as a mirage placed in our genes by natural selection so that we may become good cooperators (Ruse & Wilson, 1985, 1986). This Ruse had stated before, but he now goes further. On top of the illusion that there is right and wrong is the additional mirage, "put in place by biology," that morality has an objective foundation. It is "not just that we feel that we ought to do the right and proper thing, but that we feel that . . . it truly is the right and proper thing."

I rather see it that morality exists in objective reality because our exalted intelligence allows us to anticipate the consequence of our actions in regard to others and

to evaluate the actions in terms of these consequences. Such are the biological foundations of moral behavior. Morality is an adaptation that contributes to the biological success of our species, but it is an exaptation, rather than an adaptation, because it was not directly promoted by natural selection. The target of natural selection was rather exalted intelligence, because tool making improved the fitness of our Pleistocene ancestors. Human societies have experimented over millennia with different moral systems. Those that persist in modern humankind are those that proved successful within the societies in which they exist. Moral codes are products of cultural evolution, not direct outcomes of natural selection.

Notes

1 This is similar to the definition of David Copp (2006): "[W]e can take a person's moral beliefs to be the beliefs she has about how to live her life when she takes into account in a sympathetic way the impact of her life and decisions on others" (p. 4).

2 *Merriam Webster's Collegiate Dictionary*, 10th edition, gives a second definition of altruism as "behavior by an animal that is not beneficial to or may be harmful to itself but that benefits others of its species."

3 Notice that, as I will state later, I am not taking the position known as "utilitarianism," because I am not claiming that maximizing the benefits to others, and to as many others as possible, is the ultimate standard by which the morality of actions should be determined.

4 For a brief but insightful discussion of free will in the context of evolution, see Ruse (2006, Ch. 12).

5 For the evolutionary model of the evolution of language that I favor, see Cela-Conde & Ayala (2007, pp. 339–353).

6 The "naturalistic fallacy" (Moore, 1903) consists in identifying what "is" with what "ought to be." This error was pointed out already by Hume (1740/1978):

> In every system of morality which I have hitherto met with I have always remarked that the author proceeds for some time in the ordinary way of reasoning . . . when of a sudden I am surprised to find, that instead of the usual copulations of propositions, is and is not, I meet with no proposition that is not connected with an ought or ought not. This change is imperceptible; but is, however, of the last consequence. For as this ought or ought not express some new relation or affirmation, it is necessary that it should be observed and explained; and at the same time a reason should be given, for what seems altogether inconceivable, how this new relation can be a deduction from others, which are entirely different from it. (p. 469)

The naturalistic fallacy occurs whenever inferences using the terms "ought" or "ought not" are derived from premises that do not include such terms but are rather formulated using the connections "is" or "is not." An argument cannot be logically valid unless the conclusions only contain terms that are also present in the premises. In order to proceed logically from that which "is" to what "ought to be," it is necessary to include a premise that justifies the transition between the two expressions. But this transition is what is at stake, and one would need a previous premise to justify the validity of the one making the transition, and so on in a regression *ad infinitum*. In other words, from the fact that something is the case, it does not follow that it ought to be so in the ethical sense; is and ought belong to disparate logical categories. Because evolution has proceeded in a particular way, it does not follow that that course is morally right or desirable. The justification of

ethical norms on biological evolution, or on any other natural process, can only be achieved by introducing value judgments, human choices that prefer one rather than other object or process. Biological nature is in itself morally neutral.

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PART X

IS THERE A PLACE FOR INTELLIGENT DESIGN IN THE PHILOSOPHY OF BIOLOGY?

Introduction

The topic of whether some god exists excites almost anyone, and most people on the planet are theists of one sort or another. One argument that has compelled people to believe in the divine—probably since the dawn of consciousness—has to do with the incredible order and complexity that various parts, processes, and principles of nature exhibit. A quick look at any cell biology textbook shows the awesome intricacies and complexities present at the microscopic level, while a quick look at any cosmology textbook demonstrates similar intricacies and complexities at the macroscopic level. For many people, this order and complexity exhibited in nature must ultimately be explained by virtue of some kind of divine mind that has designed nature this way. This is all the more true for living things, as they seem to be even *more* ordered and *more* complex than other natural phenomena like tornadoes and chemical bonds. Through the voice of one of his characters named Cleanthes in his *Dialogues Concerning Natural Religion*, the famous philosopher David Hume (1779/1947) notes the analogy between human design and divine design:

> Look around the world: Contemplate the whole and every part of it: You will find it to be nothing but one great machine, subdivided into an infinite number of lesser machines. . . . All these various machines, and even their most minute parts, are adjusted to each other with an accuracy, which ravishes into admiration all men, who have ever

> contemplated them. The curious adapting of means to ends, throughout all nature, resembles exactly, though it much exceeds the productions of human contrivance; of human design, thought, wisdom, and intelligence. Since, therefore the effects resemble each other, we are led to infer, by all the rules of analogy, that the causes also resemble; and that the Author of nature is somewhat similar to the mind of man; though possessed of much larger faculties, proportioned to the grandeur of the work, which he has executed. (p. 143)

In Hume's passage above, the Author of nature is also the *designer* of nature.

Evolutionary theory has challenged the design argument for God's existence, essentially because the order and complexity exhibited by living things can be explained through basic naturalistic principles such as variation, inheritance, population increase, struggle for existence, differential survival, differential production, and natural selection. Thus, God is no longer needed; although, this does not mean *necessarily*, a priori, that some kind of a god cannot be made compatible with evolutionary theory (see, for example, Ayala, 2007a; Ruse, 2000).

Since the publication of Darwin's *Origin*, this "no need for the God hypothesis" has caused religious people to consider evolution as tantamount to *evil*-ution. A movement called *creationism* has emerged, especially in the past 40 years or so. Creationists not only believe that the universe was designed and created by an all-powerful, intelligent being, but they also believe that this idea should be taught alongside the Big Bang and evolutionary theory in biology classrooms. Most creationists are anti-evolutionists of one stripe or another, usually as a result of misunderstandings and misinterpretations of science.

The idea of creationism being taught in a science classroom has struck most biologists and philosophers of biology as absolutely ludicrous given that (1) a god is beyond the purview of publicly testable empirical knowledge (for example, do we have any scientific evidence of a god of any kind, and how could we anyway if God is, by definition, immaterial?), and that (2) there is not even any kind of clear, consistent research program that exists for creationists (for example, which god of the multitude of creation myths on the planet are we testing for, and how do you even begin to set up such a test?). The reader is encouraged to look at the Further Reading in Philosophy of Science material at the end of the general introduction for more on the nature and methodology of science. Further, consider John E. Jones' concluding claims in the *Kitzmiller* v. *Dover* trial (December 20, 2005), where Jones ruled that intelligent design should not be taught in Pennsylvania's Middle District public school science classrooms:

> To be sure, Darwin's theory of evolution is imperfect. However, the fact that a scientific theory cannot yet render an explanation on every point should not be used as a pretext to thrust an untestable alternative hypothesis grounded in religion into the science classroom or to misrepresent well-established scientific propositions. The citizens of the Dover area were poorly served by the members of the Board who voted for the ID [intelligent design] Policy. It is ironic that several of these individuals, who so staunchly and proudly touted their religious convictions in public, would time and again lie to cover their tracks and disguise the real purpose behind the ID Policy. (pp. 136–137)

Modern-day creationists are known as *intelligent design creationists*, and they have attempted to resurrect a version of the design argument by pointing out that certain

features of nature are irreducibly complex. Irreducible complexity essentially is the idea that certain features of nature must be explained by the intervention of some divine (or divine-like) mind because these features are too complex to have come into existence in some other kind of fashion (for example, step-by-step, trial-and-error, partly working to better working, etc.). Michael Behe (1996) is an intelligent design creationist who has argued that the parts and processes associated with the bacterial flagellum are irreducibly complex, leading us to the conclusion that it must have been designed by some mind. Behe uses the analogy of the typical spring-loaded bar mousetrap with its numerous parts: the mousetrap must come together *wholesale*; otherwise, it would not work *piecemeal*. So too, there are natural parts and processes that must come together wholesale as irreducibly complex through the influence of some designer god; otherwise, they would not work piecemealed together through evolutionary tinkering. Many biologists and philosophers of biology have shown that Behe is as mistaken about the irreducible complexity of the bacterial flagellum as he is about any and all design inferences. In fact, the bacterial flagellum has been demonstrated to be *reducibly* complex (and, in some cases, simple), and thinkers over the past few years—like John McDonald (2008)—have delighted in building mousetraps that can work with one or two simple parts.

In the first paper included in this part, although he claims not to be a proponent of intelligent design (ID), Del Ratzsch argues that the normal arguments against ID probably do not work as well as people think they do. He also argues that, since science itself claims to be fallibilistic, it should not rule out ID-like explanations a priori, but should be open to such possibilities. It may be that, in the end, science needs to adjust and broaden its scope. After all, claims Ratzsch, science already countenances a concept like teleology (also see Part II in this book), which does not seem to be as "scientific" as other standard, basic reductionist concepts.

In his paper, Francisco Ayala counters Ratzsch's optimism that anything like ID could ever be considered scientific with a basic argument: a legitimate scientific theory or hypothesis requires (a) testability and falsifiability through empirical observation and experiment, as well as (b) results, predictions, and explanations. ID fails the requirements for (a) and (b). Therefore, ID is not science, nor should it even be considered as a candidate some day. Further, he argues that teleology does have a legitimate place in the biological sciences without having to invoke an ID-like god: "The design of organisms comes about not by intelligent design, but by the interaction of mutation and natural selection, in a process that is creative through the interaction of chance and necessity."

Some biologists and philosophers of biology think that intelligent design creationism should not even be addressed because it is a waste of time and ink. Further, addressing creationism might mean that it is something worthy of being addressed. However, given that there are religious zealots who want religion to trump science in the classroom, or religion to trump rights in the political sphere, it is important for people to be made aware of creationism, its flawed thinking, and surprisingly large following. Along with Robert Pennock (2002), we must ask ourselves: "Are parents doing an educational disservice to their children in teaching them creationism?" (p. 114). This is an important question for biologists and philosophers of biology to ponder. The National Center for Science Education (NCSE, http://www. ncseweb.org/) is a good resource for educators and other thinkers because not only is it devoted

to the defense of the teaching of evolution in public schools, it also contains basic explanatory and clarifying material surrounding Darwinism and evolution, as well as links to other valuable resources in biology and philosophy of biology.

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CHAPTER NINETEEN

There Is a Place for Intelligent Design in the Philosophy of Biology

Intelligent Design in (Philosophy of) Biology: Some Legitimate Roles

Del Ratzsch

The status (positive or negative) of the concept of *intelligent design* (ID) in the sciences is not nearly the straightforward matter it is often presented as being. Although I am not a defender of the theories, positions, and claims of the present ID movement, in what follows I argue, among other things, that (a) the standard arguments against the legitimacy of the concept of *intelligent design* in science do not work as advertised; that (b) the potential scope of the scientific legitimacy of such concepts is surprisingly large; and that (c) some among a variety of factors both inside and outside biology render biology not only resistant to reduction attempts but actually hospitable to teleological concepts, including *design*. Indeed, those factors may make such concepts or their connections to deeper teleological matters ineradicable from biology.

1 Introduction

The question of whether the concept of *intelligent design*—particularly, *supernatural* intelligent design—is necessary, useful, or even permissible within natural science has generated heated debate in recent years. Ranged on the intelligent design side are, for example, Phillip Johnson, Michael Behe, and William Dembski, while on the other one finds people like Richard Dawkins, Eugenie Scott, and Kenneth Miller (see the papers in Dembski & Ruse, 2004; Pennock, 2001a). There is little dispute that the generic concept of *design* has played useful roles within science (especially biology). With the concept of *intelligent* design, the situation is more complex, but is in some

contexts uncontroversial. In archeology, in SETI searches, and other contexts, intelligent design is unproblematically put to descriptive and explanatory use. But the real flashpoint concerns deployment of full-blown intelligent design concepts within natural science. I will argue that, although application of such concepts perhaps cannot be extended scientifically as far as current intelligent design (ID) advocates claim, the range of application can, in principle, be extended significantly further than often recognized. I will then show that not only are robust design concepts already inextricably woven through biology, but that there are suggestive reasons for thinking that these design concepts play a good—even crucial—role here. The question then may not be, "*Is* there a place for intelligent design in biology?" but, rather, "Exactly what and how essential is that place?"

2 A Brief Historical Note

Given the widespread conviction that this whole matter is a vicious scientific nonstarter, we must begin further back, and the history of science, itself, would suggest that this issue might not be completely simple. Some historians of science—for example, Reijer Hooykaas (2000)—have argued that the Western, European Christian intellectual context provided conceptual resources essential to the existence and rise of modern science. Historically, a conviction that the structure of the cosmos was deliberately designed underwrote key foundational presuppositions of modern science; for example, that the cosmos is uniform, predictable, law-governed, ultimately intelligible, and so forth. Various historians have argued that it was not coincidence that modern theoretical science arose only once, within the context of a theological doctrine of creation according to which the cosmos was a supernaturally designed artifact.[1]

And that fact is not of merely historical significance. The above worldview *structure* persists. Physicist Paul Davies (1995) remarks that science "began as an outgrowth of theology, and all scientists, whether atheists or theists . . . accept an essentially theological worldview" (p. 138). If Davies is right about the deep structure of the scientific picture, then subtle design ideas are woven deeply into even current scientific content (consider Cummins & Roth's arguments against function, in this volume). It is worth noting that the history of design in science—viz., its role, its apparent mid-nineteenth-century demise, the driving causes of that change, the role which evolution played, etc.—is not at all as it is widely advertised (Ratzsch, 2005a). And historically, design concepts have inarguably been fruitful *heuristically* in science, and especially so in biology. That usefulness, and the accompanying tendency to slip into actual design thinking, has been so pervasive that Nobel laureate Francis Crick (1998) felt compelled to warn fellow biologists: "Biologists must constantly keep in mind that what they see was not designed, but rather evolved" (p. 138).

3 Failed Shortcuts

But many argue that whatever the history (and present temptations), the idea of supernatural intelligent design cannot (now) be legitimately employed in either scientific descriptions or explanations. For instance, Eugenie Scott (1990): "To be dealt with

scientifically, 'intelligence' must also be natural, because *all* science is natural. . . . Any theory with a supernatural foundation is not scientific" (pp. 16–18).

Although some claim that science is committed to the view that the natural realm constitutes the whole of reality (*metaphysical* or *philosophical naturalism*), the nearly consensus position is that natural science is committed only to *methodological naturalism*, typically construed as follows:

> Philosophical naturalism may or may not be correct (science itself simply takes no position), but since science cannot deal with the supernatural, it is an essential methodological principle of science that science must proceed *as if* philosophical naturalism is correct.[2]

Supports for such views typically fall into several categories: conceptual, criteriological, pragmatic, and empirical. But, as we will see, the typically advanced supports do not close the case.

3.1 Conceptual support

According to many, confinement of science to the natural is part of the very essence of science. Thus, Michael Ruse (2001): "*[Q]ua* science, ***that is*** [boldface mine] *qua an enterprise formed through the practice of methodological naturalism . . .*" (pp. 365–366). Quite a number of others make restriction of natural science to the natural realm a matter of the very definition of *science* itself (see, e.g., Murphy, 1993, p. 33).

But one must be careful resting one's case on definitions or favored concepts of just what science *must* be, for the simple reasons that (a) no one actually has a completely viable definition, or definitive conception, of science, and (b) definitions and conceptions of science appealed to in the past (often by scientists themselves) have a track record of being subsequently overturned (often by scientists themselves), leading some to deny that there is any unchanging essence of science (see the examples given in Hung, 1996). Besides, definitions are *human* constructions and, moreover, are not themselves the results of science. Exactly why human definitions should be inviolably normative for attempts to understand the physical cosmos around us is unclear. The cosmos is not obliged to conform to our philosophical preferences and edicts.

3.2 Criteriological support

Design theories, it is claimed, are non-predictive, unfalsifiable, untestable, and empirically empty; thus, they lie somewhere between non-scientific, useless, and fraudulent. Although such claims are not without some plausibility, criticisms on these broad counts are not straightforward. For example, predictiveness is a tricky matter. It is widely recognized that even the most upright scientific theory makes no predictions in isolation, but only in conjunction with other inferential resources: boundary conditions, auxiliary hypotheses, instrumentation theories, and so forth. Furthermore, different scientifically essential principles operate at different levels in a conceptual hierarchy within science, at different degrees of removal from the empirical trenches.

What precise connection a conceptual component of science should have to empirical predictions, in part, is a function of the role which that component plays in science, and the level at which it operates. Of course, even overarching high-level (or deep underlying) principles must be linked to *some* payoff in the broader scientific picture; but that payoff does not reduce to something so simple as particular, identifiable empirical predictions. Many believe that evolutionary theory itself has a quite modest (even thin) predictive track record. Others, of course, dispute that perception (e.g., Ayala in this volume). But even if the claim is true, that does not make evolution non-scientific, or even less scientific, in the slightest. Evolution is widely seen as having other (and outweighing) scientific epistemic virtues in spades: for example, explanatory and unifying power.

And design theories are not *inherently* non-predictive. If we know that some subsystem of some object is deliberately designed for some purpose or function, we can often predict other things concerning, not only that subsystem, but also the existence and characteristics of correlated entities or other subsystems. The specific theories associated with contemporary design advocates may make no requisite predictions, but, if so, that *may* be simply a failure of their specific theories, not necessarily a problem of principle.

Falsifiability is equally tricky. Given that theories, by themselves, never generate predictions, failure of predictions never *strictly* implicates merely one of the components (theory, auxiliary hypotheses, etc.) involved in deriving the prediction. It does, of course, indicate a glitch somewhere in the combined derivation system, but precise responsibility has to be assigned on other (and complex) grounds. Similar cautions apply to the other criticisms in this category as well.

3.3 Pragmatic support

A widely advanced criticism is that non-natural concepts are destructive of good scientific order and effort. Warnings against allowing such concepts into science are found from the seventeenth century (e.g., Christian scientists such as Bacon and Boyle) to the present. Robert Pennock (2001b) has claimed that "once such supernatural explanations are permitted they could be used in chemistry and physics as easily as Creationists have used them in biology and geology. Indeed, all empirical investigation beyond the purely descriptive could cease, for scientists would have a ready-made answer for everything" (p. 90).

The proposed risk, then, is that *if* one were allowed to appeal to intelligent design, supernatural agency, gaps in nature's causal economy, and the like, in science, then natural scientists would succumb to the always-present temptation to take that easy way out—"it must be designed"—when confronting seemingly intractable theoretical problems. They would be tempted, thus, to give up on the hard problems too soon.

That is indeed a legitimate worry, and methodological naturalist restrictions as a *pragmatic strategy* may well be appropriate. But there is a corresponding worry: the risk of refusing to recognize when it *is* time to quit. Historically, science has often abandoned previously pursued projects (perpetual motion, for instance) and, in many cases, recognition of when it was time to drop pursuit was to the ultimate benefit of science. But imposing doctrinaire methodological naturalism as a *policy* amounts to a refusal ever, in principle, to recognize that purely naturalistic resources for explaining

and understanding phenomena in the cosmos around us might be inadequate; regardless of what we find next week, next year, or next millennium (see Watson & Arp, 2008). But *if* it should turn out that some things in nature *are* deliberately designed and can only be correctly understood in design terms, then a human edict that deliberate design is a scientifically forbidden concept will inevitably drive our scientific investigation, in that area, into either error or failure.

3.4 Empirical support

Many biologists take design explanations to have been already empirically falsified and, thus, to be *a fortiori* falsifiable.[3] From Darwin to the present, arguments that an omniscient, benevolent God would not have designed and created a world with the features of *this* one—e.g., apparently gratuitous suffering, non-optimal design—have been popular weapons against design contentions. Such arguments do have philosophical weight, as the religious community's centuries-old grappling with the traditional argument from evil attests. But note that these allegedly *empirical* evidences against design are based on *theological* presuppositions concerning what God would or would not do.

That doesn't seem to be a straightforwardly *scientific* matter, much less an empirical issue, and for various reasons such "scientific" cases are thoroughly problematic (Ratzsch, 1998, 2001). For now, I merely point out that these implicitly theological cases fit oddly within a critical community which frequently rejects design on the grounds that if God exists, he is totally beyond our ability to comprehend and anticipate, and that for *that* very reason design theories cannot generate any predictions, or even legitimate explanations, at all.

The upshot of the foregoing is that the question of whether or not there might be a role for design in science—and, specifically biology—cannot be settled in the easy, shortcut manner which much contemporary discussion assumes. We must look deeper.

4 Looking Deeper

Historically, the concept of design has played a sometimes significant role in the course and progress of science. Furthermore, some of that content may still lurk, and function, in various scientific contexts. The question then arises, "How far within science can the general concepts of deliberate design, intent, purpose, etc., extend?" We begin with a thought experiment.

Upon first landing on Mars, if we were to discover an undeniable artifact (e.g., a Martian oscilloscope or bulldozer), no one would think it improper or unscientific to identify it as a product of deliberate design. In general, anything plausibly artifactual can be unproblematically described and explained as deliberately intelligently designed; whether or not we can say much about *what* the design is, *who* or what its designer might be, or *how* it was produced. (There are, of course, a thicket of necessary qualifications; Ratzsch, 2001) Recognition, description, and explanation with reference to design, in such cases, can be conceptually fruitful in scientifically recognizable and legitimate ways.

Recognition of the legitimacy of design principles in the case of artifacts has some surprising implications. For instance, Crick and Orgel (1973), Frederick Hoyle (1983), and others have argued that life as we know it could not have arisen by natural processes under early earth conditions in the time available, and have claimed (controversially) that life migrated to, or was planted on, earth from elsewhere (a doctrine known now as *panspermia*). It is, at least, *possible* that life as we know it, in fact, was deliberately engineered for earth conditions by some advanced civilization. (Let me be perfectly clear: I do not know of any evidence whatsoever that there are aliens; the point here is purely conceptual.) If life as *we* know it was deliberately designed for earth conditions, then life as we know it would be an artifact, and the concept of intelligent design would legitimately apply to it. Interestingly enough, the day-to-day lab or field work of the vast majority of biologists would be unaffected. (That fact should trouble many current ID advocates.)

There might not be the slightest evidence for life having been so designed, but the point is that if were true, *recognizing* that fact (however recognition might be achieved)—and attempting to investigate the parameters of the design, of production techniques, and a host of other relevant matters—would be a perfectly legitimate *scientific* pursuit, essentially constituting a form of biological archeology, or reverse engineering. A key legitimating factor here, according to many, would be the fact that the designers in question would be *natural* beings; albeit, not from Earth.

That general principle, potentially, extends further than biology. Andrei Linde (2004, 2007) has suggested that it might be possible for technologically advanced civilizations to generate artificial bubble universes with deliberately controlled parameters of laws, constants, boundary conditions, and so forth. If intelligent beings emerged *within* such a constructed universe (perhaps as a deliberately intended product of the chosen parameters), then those bubble inhabitants might scientifically investigate their "universe" and *might* even conclude (quite correctly) that their universe (their nature) was an artifact, a product of deliberate intelligent design. Linde even toys with the idea of the producers of the bubble universe *communicating* with the intelligences evolving within it through careful structuring of the relevant parameters. (More specifically biological, Paul Davies [1995] raises the possibility, in principle, of an alien message in so-called "junk DNA.") Here, the whole concept of what does or does not constitute nature begins to blur; the bubble universe being an artifact, but nonetheless the only nature the inhabitants know. Intriguingly, Newton explicitly identified things like the eye as simultaneously artificial *and* part of nature. Such blurrings also blur any stipulated line beyond which the concept of intelligent design is supposed to be illegitimate in natural science.

It should be evident that the *identity* of any designer is irrelevant to the above considerations. Forgotten human biological engineers, technologically advanced aliens, whatever—it makes no *necessary* ultimate difference to the genuinely scientific investigability of the object in question. And that holds true for *any* designer, supernatural as well. A domain exhibiting the requisite properties to be a domain of scientific investigation can support such investigation *regardless* of how it came to have those characteristics. Suppose the Martian bulldozer, in fact, had been designed and miraculously created by a supernatural being for whatever reason, humanly scrutable or not. That would have no bearing upon whether or not any competent investigator should be able to spot its designedness (independent of any notions—right,

wrong, or lunatic—of the nature of the designer), upon the investigator's ability to sort out and understand the principles of operation, etc. Exactly the same goes for life.

Of course, there are complications. ID theories are ultimately *agent* theories, and a properly scientific deployment of such theories would seem to require *some* grasp of the relevant agent's interests, purposes, cognitive processes, etc. For human agents in relatively normal states and circumstances, we can usually make reliable judgments of intention; but beyond that, things get dicier. Step minimally outside the human realm to terrestrial animals, for example, and we can make some judgments, but with less warranted confidence. With advanced aliens, we could probably make sensible cases for their intentions regarding some specific artifacts and behaviors, but for other intentions we likely wouldn't (couldn't?) have a clue.

With supernatural designers, things get even trickier. How could we hope to understand the intentions, plans, and purposes of a supernatural mind? Some religious believers have argued that we have some hope here, because the doctrine that humans were created in God's image can be taken to mean that the minds with which we try to understand nature reflect, in some degree, the mind whose thoughts are structured into nature. That idea is explicit in Kepler and Galileo, for example.[4]

Thus, it seems clear that not only do the easy illegitimacy arguments fail, but also the potential range of design legitimacy is surprisingly large.

5 The Richness of Biology

Now, I want to explore some implications of the unique character of biology itself. There is a longstanding dispute over whether there is a single canonical structure to which all legitimate sciences must conform. Some decades back, the accepted answer was affirmative, physics being the chosen exemplar of that structure. Now, most believe that that proposed structure was more Procrustean bed than viable norm, biology being a (the) primary case in point (see, e.g., Dupré, 1993).

5.1 Reduction

One hint that there is something irreducibly unique about specifically biological science is the track record of failed attempts to reduce biology to physics (or physics plus chemistry). Why such failure?

Practical hurdles

Some have suspected that this failure is merely practical, given that biology deals with complexities of unmanageable orders. Even if classical reduction were possible, in principle, it is unlikely that *we* could handle the reductive derivations. For instance, suppose that one wished to derive phenotypic generalizations from principles involving molecular genetics. Given what David Hull (1974) has called the *many-many properties problem*—i.e., that typically a variety of phenotypic characteristics can result from, or be affected by, the same genetic substructures (in the context of different activation orders and processes, different epigenetic environments, etc.) and that different genetic substructures can (in variant circumstances) give rise to similar

phenotypic phenomena—full-blown reduction attempts rapidly get out of hand. (And, of course, molecular genetics involves a biological starting point already, so this would be only a partial reduction. Attempts to derive distinctively biological principles from first principles of physics would involve complexities many orders of magnitude worse.) The required bridge principles would be, as Alexander Rosenberg (1989) put it, "hideously complex, each side of which is a disjunction over a vast number of further disjunctions" (p. 246).

Further, even if reduction were in principle possible, in order for *us* to get any grip on a number of key biological issues in some areas we would need some more course-grained, larger-level conceptual units (LCUs), viz., primitive concepts of organs, organisms, species, and so forth (perhaps, akin to what Eleanor Rosch [1973] calls "basic level categories"). Practical hurdles do not, of course, entail actual irreducibility. But other reasons go deeper.

Structural mismatches

A related reason is that biology—as we humans are limited to studying it—may not involve laws as we have traditionally thought of sciences as doing. The inerradicable course-grained, larger-level biological conceptual units simply do not map into any standard law-structure; hence, the notorious fact that virtually any general biological claim has exceptions, typically supports only very general predictions (if any), and so forth (for more on this, see the chapters in Part I of this volume). There are no identifiable laws taking those LCUs as basic conceptual components. Unless we revert to biological essentialism, the concept *liver*—let alone *seaslug*—just does not carve out a basic, law-supporting natural kind like, say, *electron* does.

That lack of fit between many apparently essential and fundamental biological principles and the generally accepted character of law suggests that various, purported, *ideal* structures and constructions of science, scientific methods, scientific conceptual resources, etc., may not rigorously apply to biology as we humans are constrained to doing it. That does *not*, of course, cast any sort of cloud on biology as a science. It simply points up a characterizing uniqueness of biology.

An ongoing dispute within philosophy of biology (and biology) illustrates one facet of this situation nicely. The notion of a *species* was central to understanding evolution (recall Darwin's *Origin of Species*) as well as some other key matters. Yet, there is quite a history of disputes over what a species actually is, including claims that: a species is an *individual* and, hence, not a proper conceptual constituent of genuine law at all; every individual organism is its own species; species concepts (whatever they come to) no longer have any remaining relevance to contemporary biology; there isn't even any such thing as a species (see the chapters on species in this volume; also the papers in Claridge, Dawah, & Wilson, 1997; Coyne & Orr, 2004). It is not surprising that Marjorie Grene (1989) speaks of the *species* concept as exhibiting a "resistance to lawhood" (p. 71).

Much relevant biological action takes place via, and to, units having no ontological status or stability in terms of the fundamental physics entities that compose them. Sherlock Holmes once claimed that from a single drop of water, a perfect reasoner could predict the possibility of a Niagara. Even if (implausibly) so, it is not obvious that anything less than a relevantly omniscient reasoner could deduce the possibility of a liver fluke from fundamental physics.

Deeper conceptual barriers

Others have thought that reasons for reductive failures lie even deeper. Ernst Mayr (1985) argued that the biological realm is hierarchical and that "systems at each hierarchical level . . . act as wholes (as if they were a homogenous entity), and their characteristics cannot (not even in theory) be deduced from the most complete knowledge of the components, taken separately, or in other partial combinations. . . . [N]ew characteristics . . . emerge that could not have been predicted from a knowledge of the components" (p. 58). And even more strongly, Mayr (1988) noted that the "conceptual framework of biology is entirely different from that of the physical sciences and cannot be reduced to it" (p. 18).

If strict reduction cannot fly—and if that failure includes not only theory irreducibility, but also irreproducibility (or non-constructability) of conceptual contents—then if *understanding* is supposed to be in terms of conceptual subcomponents (as is widely held), genuine biological understanding will require conceptual components beyond those available within the reducing science (physics and/or chemistry). So, to understand at least some biological phenomena, we *have* to employ concepts with a richer, or different, content than that constructible or derivable from the results of analysis of merely the constituents of larger-level biological units. That does *not* imply that the concepts on higher levels necessarily have some non-natural component. But nothing, so far forth, rules that out either.

5.2 Teleology and design: an extra dimension

Although sharing some fundamental constraints, methodologies, logics, and the like, biology on some levels is just *different* than physics, and has (nearly effortlessly) resisted reductive attempts. Pinpointing the definitive differences has not proven easy. But most attempts to isolate biology's uniqueness have circled around the notion of *teleology* and concomitant *design*. Historically, an array of derivative notions has filled some of the additional conceptual dimensions required within principles involving LCUs. Given the seeming goal-directedness of many biological phenomena, teleological explanations are both natural and plausible. Indeed, it has frequently been remarked that, prior to Darwin, teleological—even creationist—biological explanations were the most reasonable of the then-available alternatives (see, e.g., Ruse, 2004).

Some familiar discontents

The traditional concept of teleology carried implications of: divine design, plan, and purpose; intentional goal-driven processes sometimes involving backward causation; or mysterious vital processes, or entelechies. All such notions have come to be thought to fail essential scientific constraints. Yet, biological discourse is rife with such phrases as: serving the function of, for the sake of, for the purpose of, in order that, designed to do, and so forth. How might one capture what those phrases are intended to encompass and convey *without* inadvertently importing scientifically subversive content? Can biology (and biological discourse) be fully robust *and* simultaneously free of any of the "wrong" sort of teleological or design taint?

Although there are (of course!) technical disputes concerning details, most mainstream biologists and philosophers of biology believe that it can (for more on this,

see the chapters on function in this volume; also see Allen, Bekoff, & Lauder, 1998; Ariew, Cummins, & Perlman, 2002; Arp, 2006; Buller, 1999). Explaining the *operation* of the eye (and similar "organs of extreme perfection," to quote Darwin) seems to require no reference to anything beyond ordinary material processes (although involving wild complexity). Indeed, that holds true even of known products of human design. The goal-directed *operation* of something so intentional and purpose-generated as an automatic thermostat can be explained with no reference to intention, agency, and the like, as can the operation of so obviously a purposeful object as a bulldozer. Everything in the operation of both is explainable in terms of physical principles and processes. Similarly, the operation of the eye, though exquisitely complex, is not in principle mysterious.

When it comes to *existence*, however, things get more complicated. An oscilloscope, obviously, could come into existence without any direct agent input into the immediate production process; if, say, it were produced by some fully automated processes. Indeed, it might be brought into existence without any agent ever having *thought* about that specific oscilloscope, or even being aware of its specific existence, much less taking some direct productive role. One would, however, be hard pressed to make the case that an oscilloscope—let alone a bulldozer—could come into existence without some agency and deliberate intention factoring *somewhere* into the object's history. That somewhere might be well in the past, perhaps in the construction of the automated production machinery, or the machinery which produced the machinery which produced it, etc.

With biological matters (organisms, organs, complex behaviors, and the like) the situation is, again, more complicated. Obviously, one can hold that phenomena so complex as specific eyes are produced and can be explained without any direct agency, intention, and so forth. One can also plausibly hold that the general existence of eyes, as such, can be immediately explained in purely physical and natural (evolutionary) terms, involving no immediate agency, intention, or awareness. Furthermore, living organisms have even further layers of complexity, being not only (usually) adapt*ed*, but adapt*ive*. Adaptive systems, in a sense, are goal-directed in that there is (are) a (number of) stable equilibrium state(s) which the system gravitates back into from a limited (often large) number of types of perturbations. This is where the impression of *striving* comes in, the state preferentially maintained in the system being the "goal." But having a goal, in this sense, does not automatically imply that the goal is *immediately* pre-selected, deliberate, or anything of the sort. So even here, one can hold that there is no immediately injected operative design, intent, or purpose.

Many theists believe that, although the cosmos and much in it *is* deliberately designed, this fact need play no *direct* conceptual role in the everyday workings of science. Most (not all) ID advocates disagree.[5]

Of course, it is inarguably possible that such a goal/equilibrium point is pre-selected from beyond the system, or that the system or the deeper conditions and processes giving rise to it are pre-selected for exactly that characteristic. A key question here is whether in biology (as with oscilloscopes and bulldozers), if one really follows the causal, explanatory thread of seemingly designed phenomena in biology indefinitely back, at *some* point one really will come to ground on some agency and deliberate intention; in other words, whether we can have "proper scientific" teleology or design

(call it $design_1$) in the complete absence of the richer, intention-laden teleology or design (call it $design_2$). Can we have the Cheshire cat's smile without the cat? Intuitions and deep philosophical commitments obviously diverge at this point, with supremely confident vociferocity exhibited on all sides (see the papers in Pennock, 2001a).

I do not think that we know, at this point, whether the sort of goal-directedness we see in nature is ultimately possible in the absence of pre-selection at *some* deep background level. Currently, influential positive cases on this general topic, in large part, are bluster and faith, and currently influential negative cases, in large part, are faith and bluster. My own suspicion is that the answer to the above question is *no*, and, if that is the case, then harmless scientifically kosher $design_1$ ultimately rests upon the more robust intentional $design_2$. If the answer is indeed *no*, a second related question arises: Can the ultimately foundational $design_2$ be cleanly excluded from any *science* involving $design_1$? We shall discuss this more later, but following are a couple of suggestive lines of thought.

A popular intuition is that if $design_1$ is accounted for by some causal mechanism operating at a deeper/prior level, then, to that extent, the relevance/necessity of $design_2$ is removed or at least diminished (and perhaps with a few further levels entirely erodes/evaporates away). That certainly is not true in the oscilloscope or bulldozer case. Having an immediate physical explanation of the physical properties of an oscilloscope (e.g., a wholly automated production facility) would not in the slightest weaken the case for the oscilloscope being a product *ultimately* of $design_2$; it would merely push the location of the $design_2$ input back a level.

One intriguing indication that severing $design_1$ from $design_2$ may be problematic in purely natural scientific fields is the fact that those claiming that the evident $design_1$ of cosmic fine-tuning does not require any $design_2$ have been driven to ontologically extravagant and empirically tenuous (or empty) *many-universe* or holographic theories in their attempts to save their position. Worse yet, even granting such theories does not automatically deliver that separation (Ratzsch, 2005b).

Whether finding causal mechanisms that produce $design_1$ does or does not attenuate the case for $design_2$ depends wholly on the specific character of the $design_1$ and what connection it has to $design_2$. In the specifically biological case (e.g., the eye), I don't think that, at this point, anyone actually knows. But the above presupposition is simply not a general truth, and having a natural mechanism for generating $design_1$ has no necessary bearing on the question of whether $design_2$ has played—or *must* play—some role somewhere.

Gaps and God-of-the-gaps theories

There is one further important implication here. The flip side of the above presupposition is the contention that if $design_2$ *is* relevant to the production of some phenomenon, then it must be via some gap in the chain of natural causation leading to that phenomenon. It may indeed be that agent causation must factor into that causal history *somewhere*, but it does not at all follow that *that* agent activity must factor into the foreground of the relevant causal history. As noted earlier, the agent causal input might, in principle, be at nearly any arbitrary level removed from the surface. Similarly in the biological case, evidence of deliberate, intentional design in some phenomenon does not imply, in the slightest, that the (perhaps supernatural) agent's causal input was anywhere near the surface of the causal history. In fact, it

might have been in the selection and structuring of the initial conditions, parameters, and laws of nature at the initial founding of nature itself billions of years ago.[6] If so, demands that design theories produce specific predictions at the surface empirical level will simply be misguided.

Historically, even prior to Darwin, the location of deliberate design activity in nature had shifted back to logically and historically prior levels, into laws and the like (see, e.g., Arp, 1999, 2002). Darwin himself placed the following quotation from William Whewell on the flyleaf of the first edition of the *Origin*: "But with regard to the material world, we can at least go so far as this—we can perceive that events are brought about not by insulated interpositions of Divine power, exerted in each particular case, but by the establishment of general laws." Darwin endorsed that position in his personal correspondence and notebooks, and many other biologists have held—and hold—similar positions. In fact, that position was standard among natural theologians in the early nineteenth century: for example, Paley and others explicitly built this point into their natural theology arguments, arguments not remotely so naïve as generally assumed (see the chapter on creationism in Sober, 2000). That itself attests to the fact that design theories are not, inevitably, gap theories. Further, that means that natural scientific explanations and deeper design explanations are not mutually inconsistent, as so many also assume. For instance, Mayr (1988) argued that his term *program* is *not* teleological because a program is material and exists prior to initiation of the relevant process, and is, thus, "consistent with a causal explanation" (p. 48).

But there is one additional matter worth considering. What if, in fact, there *were* causal gaps in nature which were bridged by supernatural agent activity? If there were gaps—and, surely, no one can deny that that is a *factual* (even empirical?) question—then any science forbidden by human philosophical or methodological edicts to ignore such gaps on principle, regardless of any empirical evidence which may surface, is headed for difficulties. We must walk cautiously here.

6 The Persistence of Design

Despite no longer being in the biological explanatory foreground, full-blown teleological notions have been astonishingly difficult to leave behind. As Timothy Lenoir (1982) observed: "Teleological thinking has been steadfastly resisted by modern biology. And yet, in nearly every area of research biologists are hard pressed to find language that does not impute purposiveness to living forms" (p. ix). Why is this? Why, despite decades of steadfast resistance and stern warnings from Nobel laureates, do biologists still find themselves hard-pressed here? There are four interwoven conceptual dimensions.

First, we apparently *cannot* do (especially theoretical) science without metaphor-based concepts, as the work of Mary Hesse, Mary Midgley, Michael Ruse, and many others makes clear: "I do not see that metaphor ever could or (more importantly should) be eliminated from science, from science that is of the most vigorous and important kind" (Ruse, 2003, p. 126). And such science includes evolutionary biology.

Second, as explored above, we apparently *cannot* capture essential biological conceptual content (involving, for example, LCUs) without essential metaphoric content bearing teleological and design-laced substance (see Arp, 2008a, Ch. 2; Arp, 2008b).

(Later in the above article Ruse considers, but leaves undecided, the possibility that teleological thinking always goes not merely with science, but with *rationality*.)

Third, one intriguing possibility has to do with innate human cognitive structures. Recent research indicates that our cognition is design-oriented, having a pronounced design interpretive tilt built into it (Ratzsch, 2006). We have what Justin Barrett (2004) has dubbed a *hypersensitive agent detection device* (HADD), a natural tendency to see purpose and intent behind a wide variety of natural phenomena. There are various, more or less, plausible hypothesized evolutionary explanations for our having such a device. But the presence of an HADD and the correctness of some natural explanation for it do not imply that the device is fundamentally misleading, any more than evolutionary explanations of our eyes or our reason imply that they are fundamentally unreliable. Nor do such explanations, in themselves, imply that such faculties were not part of some deep deliberate plan.

But if such a device is cognitively innate, we perhaps cannot avoid design conceptions in biology. And if we cannot avoid *doing* biology from a subtly implicit intelligent design perspective, attempts to purge biology of intelligent design motifs are *de facto* futile.

7 Real Design?

A fourth possible reason why design ideas are so difficult to excise from biology might be that (at least some) biological phenomena are, in fact, deliberately designed (directly or indirectly). For the moment, suppose we assume (as, basically, all religious believers do) that $design_2$ did play some ultimate role (at least initially) in the structure, character, and operation of nature. And suppose that $design_1$ (if any) must ultimately rest on, or require, $design_2$. Given that dependence, could aspects or undertones of $design_2$ be prevented from seeping into any science which incorporates $design_1$, as biology inarguably does? Could one claim that $design_2$ is relevant only to some non-scientific conceptual level (prior foundations or philosophical implications)? Even if (a) biology must employ $design_1$ or some equivalent, *and* (b) $design_1$ is dependent ultimately upon $design_2$, could a barrier impermeable to agency, deliberate intent, purpose, and other aspects of $design_2$ be raised between that deeper $designed_2$ level and science proper?

Methodological naturalism implies that it could, and should; and those are non-trivial philosophical claims. Here again, I think that the exact dimensions of the truth are unclear. But it is worth noting that every historical attempt to erect arbitrary demarcation boundaries, or to specify things which could never in principle play any conceptual role in science, have been unsuccessful, often dismally so (see Ratzsch, 2000, Chs. 2 and 3). Simply stipulating separation by definition won't do the job, for reasons noted earlier. Furthermore, to the (significant) extent that metaphor is indispensable in capturing LCUs (e.g., *selection*), substantive conceptual undertones will be constitutive parts of various even theoretical principles.

It is, again, *possible* both that (a) nature and various things in it *are* deliberately designed and that (b) the role which $design_2$ plays is such that an accurate understanding of some things intuitively taken to be part of nature cannot be achieved apart from that recognition. It is possible that our HADD delivers truth more frequently

than philosophical naturalists, methodological naturalists, or reductionists recognize. It is, thus, possible that the subtle shaping of our concepts by our in-built HADD cognitive design-tilt is part of the reason our cognition resonates in productive ways with the biological realm, and why our pursuit of biological understanding is so often successful. That would suggest that this tilt may well be a source of some of the additional (non-reducible) conceptual content which biology requires beyond that which, for example, physics and chemistry can provide. Thus, if our innate cognitive structures do supply design-tilted content, that may be neither avoidable nor regrettable. Indeed if nature *is* designed, it may be scientifically crucial.

The prospect of design$_2$ in science potentially raises the hoary God-of-the-gaps specter but, here again, nature is not particularly obliged to pay our philosophical preferences, fears, fads, and edicts any mind whatsoever. Whether or not nature, or some things in it, is designed$_2$ is simply a matter of fact independent of us, and not subject to human policy. If the cosmos really *is* a product of design$_2$ and if we develop a design$_1$ science in isolation from any constraints that design$_2$ may impose, then our science risks being mistaken in ways which it cannot self-correct short of reorganizing around some form of design$_1$ which embodies the relevant "forbidden" facets of design$_2$: intention, purpose, plan, and very deliberate design.

It may be that there is no room for design$_2$ in science, then, only if there is no room for the relevant naturalistically unpalatable *facts*. Many would claim that, at this point, we simply do not know whether or not nature or parts of it are designed$_2$. But if we don't, we must be very wary of stipulating what conceptual resources are forbidden to science, or humanly stipulating boundaries for science. Or, if we choose to stipulate such prohibitions and boundaries for science, we then must be wary of claiming that what science produces operating within those prohibitions and boundaries is, in fact, truth. If the truth of relevant matters lies outside the boundaries we stipulate, then there is no guarantee that a science confined within those boundaries can get at it, or even suspect its existence. There may be no humanly available methods by which those truths can be got at, or the methods we choose to classify as legitimately scientific may be incompetent to capture those truths. But if so, then again, we are simply not in a position to claim that whatever our methods deliver will be truth. If there are genuine gaps, or if there are design$_2$ ramifications for our science, then our restricted methods may well not pick those up either. And our science, then, would fall victim to our own philosophical, methodological, and theoretical human prejudices.

8 Conclusion

Biologists need richer conceptual resources than the physical sciences have been able to generate. Historically, design-linked ideas have provided such resources and those resources have been, and continue to be, scientifically fruitful in biology. And, despite strenuous efforts, biology has been unable to eliminate such ideas, whether as subtle undertones, persistent structures, or scientifically essential metaphors. Perhaps we should take that failure seriously, as telling us something not only about whether or where we should or should not impose (sometimes philosophically spawned) constraints on science, but even about reality.

Declaring the absence of any role for design$_2$ in biology, in principle, means that we must rein in our claims concerning the epistemological status of our scientific results. Further, if (as critics often claim) we have no reliable means for identifying the presence, absence, or implications of supernatural design$_2$, then in many cases we will simply not know whether our own prohibitions and stipulated boundaries are leading us scientifically astray or not.

The more fruitful route would be to adopt skepticism about surface design$_2$ claims or theories within science, as a provisional (but defeasible) *strategy*. After all, that skepticism has been scientifically fruitful and there remains a real possibility of getting carried away in counterproductive design directions. But the defeasibility leaves a small window for design$_2$ recognition and acknowledgment, should nature insistently confront us with empirical data best interpreted as pointing in such directions.

I do not know what such data might look like. And I do not believe that the current ID movement has given us any compelling cases in the biological context for thinking that design$_2$ is an essential explanatory resource at some surface biological level. But keep in mind that the ID movement's form of design advocacy does not exhaust the possible range of substantive design positions, nor does surface-level deployment exhaust the possible applications of design ideas.

And it must not be forgotten that design-colored concepts already underlie, and give an apparently irremovable design hue to, some areas of biology. It must not be forgotten that *that* very presence may well underpin some of the success of biology. And we must not forget that nature has shattered our fondest philosophical, methodological, empirical, and theoretical rules before: think, for example, of heliocentrism, evolution, relativity, and quantum mechanics. We had best be prepared to allow nature to do so again, to again correct our science, if necessary. This observation by Andrei Linde (2004) is well worth pondering:

> A healthy scientific conservatism usually forces us to disregard all metaphysical subjects that seem unrelated to our search. However, in order to make sure that this conservatism is really healthy, from time to time one should take a risk to abandon some of the standard assumptions. This may allow us either to reaffirm our previous position, or to find some possible limitation of our earlier point of view. (p. 449)

Postscript: Counterpoint

There is much in Professor Ayala's paper which is informative, insightful, and which I do not wish to dispute. But we do have our disagreements. We have different views of the logical character of Paley's argument and the effects of Darwin on it (see my Ratzsch, 2005a). We have divergent takes on ID, as I do not believe that all ID arguments are gap arguments, arguments for supernatural intervention, arguments from ignorance, or false dichotomies, and the like. And not all ID advocates are opposed to evolutionary theories, theories of common descent, etc. Not all see intelligent design and evolution as mutually exclusive (for more on all these issues, see Ratzsch, 2001, as well as the current paper). And a number of ID advocates—including the primary ID think-tank, the Discovery Institute—do not support requiring inclusion of ID in public school science curricula, much less employ ID as a disingenuous means of

circumventing the U.S. Constitution.[7] Some perhaps do, but theories (ID, Darwinism, whatever) should not be judged in terms of their more confused and/or more shrill advocates.

None of that is to say that the ID movement has made its case (I don't take it to have done so); merely that nothing about ID, as such, commits it to the foregoing positions.

My deeper disagreement involves Professor Ayala's general case for denying scientific legitimacy to ID theories. He does so on the basis of stipulated demarcation criteria, but I take his specific criteria to be seriously flawed. (Of course, failure to establish illegitimacy does nothing toward establishing legitimacy.) Ayala's basic argument is this:

> Premise (1): To be legitimately scientific, a theory (hypothesis, etc.) must be testable/falsifiable via empirical observation or experiment (and must produce results/explanations).
>
> Premise (2): ID is not (cannot be) testable/falsifiable via empirical observation or experiment (and has produced no results/explanations either).

Thus,

> Conclusion (3): ID is not (cannot be) legitimately scientific.

Many ID opponents find this argument persuasive, and dismiss ID as non-science on that basis. Obviously *given* (1) and (2), (3) is inescapable. But I do not propose to give either (1) or (2). Both seem to me to be mistaken.

Premise (1)

Ayala provides little support for (1), citing only Popper's theorizings about science in *The Logic of Scientific Discovery*. The three-quarters of a century since its first publication have not, however, been kind either to Popper's demarcation project, as such, or to his specific proposed solution to it.[8]

But that aside, neither prediction nor testing/testability is as direct and straightforward as several of Ayala's remarks suggest. It is commonplace that theories and hypotheses neither generate predictions, nor are they testable in isolation; that is the familiar *Quine–Duhem thesis* (see Gillies, 1998). In theoretical contexts, there is no straight correlation between theoretical entities/processes/principles and specific bits of observation. There is a complex ramified web of theoretical commitments, prior data interpretations, multiple auxiliary theories of varying levels, boundary conditions, and a host of other (sometimes inescapably human) matters, and although that web must make *some* contact with observation and experience, empirical tweaks along the model/observation interface do not bear merely upon some single component of the web. Similarly, no scientific theory generates empirical predictions in isolation, and none is ever brought into direct contact with uninterpreted, unmediated reality. Hence, none is ever directly tested—much less rigorously falsified—by such contact.

Further, what can or cannot go into the overall mix that generates the predictions via which empirical testing is done is a very murky matter. The simple fact is that

no one really knows. One can (and some do) hold that even some *metaphysical* principles can, do, or indeed *must* factor into such mixes (e.g., Hooker, 1987).

Beyond that, the construction of a structure/interpretation which allows for abstruse theory to come into even glancing contact with empirical observation and experiment often takes nearly as much human creativity as does the initial theory construction itself. There are often "seasons" in the lives of theoretical hypotheses and, early on, it may not even be *known* how (or even whether) one might bring the theory into empirical harm's way. And, early on, it may be equally difficult seeing exactly how to deploy the theory productively. But the theory may nonetheless be judged by scientists to be worth provisional pursuit. The theory may be particularly elegant, it may offer a gorgeous explanation of some otherwise recalcitrant phenomenon, or it may exhibit other extra-empirical theoretical virtues, as was, to some extent, the case with Darwin's own theory.

A theory/hypothesis must indeed (as part of a complex web) at *some* point, in *some* way, experience *some* telling empirical contact with the world, and thereby be at *some* genuine empirical risk. That is the kernel of truth in premise (1) above. But among other complexifying factors, (a) there is no rule concerning when that point has been reached, (b) there are no exceptionless criteria for exactly what kind of contact is required, (c) there is no precise specification of what is or isn't appropriately *telling* contact, (d) there are no a priori principles for what does or does not constitutes appropriate risk, and, again, (e) there is no known way of stipulating what can or cannot factor into that mix. Indeed, (f) there are not even set criteria for what does or does not constitute fatally *failing* such a test. All these matters are, in *some* degree, judgment calls: issues concerning which scientists frequently disagree, and matters on which the scientific community has changed its mind(s) multiple times over its history.

So although the apparently sharp cutting edge of premise (1) makes it attractive as a weapon, wielding it to specific effect is a much more delicate and unpredictable matter than one might initially suspect.

Premise (2)

Making a case for premise (2) requires showing that there is something about ID theories which prevents them from being legitimate parts of any complex web capable of making requisite contact with the empirical realm. Making that case looks difficult. As noted, it is simply not known what principles can or cannot ever function in such webs, and every major attempt has notoriously failed.

In addition to the well-known difficulties with falsifiability (and testability) principles, there are indispensable underlying principles of science, itself, which are themselves neither empirically falsifiable nor even testable. They are, rather, presupposed conditions for the existence of scientific testing itself. Uniformity principles, for instance, fall within this category. Science cannot function without such principles, they partially constitute every testing situation, and empirical testing is basically irrelevant to their legitimacy.

And the mere fact (*were* it a fact) that ID theories appeal to the supernatural would provide no straightforward case for premise (2) either. Even so unsympathetic a critic as Philip Kitcher (1983) noted in *Abusing Science*: "Even postulating an unobserved

Creator need be no more unscientific than postulating unobservable particles. *What matters is the character of the proposals and the ways in which they are articulated and defended* [emphasis added]" (p. 125).

The other major matter Ayala cites are ID references to the *inscrutability* of divine motives. Such appeals, Ayala argues, are deployed as impenetrable shields behind which ID theories hide from empirical assault; after all, who knows what designs serving what multifarious purposes a supernatural being might favor?

That seems right, as we very likely *couldn't* either fathom, or predict, all the motives, intentions, and the like of supernatural beings. However, ID advocates need not (if they do) claim that their theories reveal/presuppose very much about designer *motives*. The relevant claim is that science can (sometimes) identify the presence of *design*. *Motive* is a different issue—we might legitimately identify some Martian object as *designed* without having much clue as to the designer's *motives* (purposes, intents, methods, etc.). Those are different questions, and failure on the one says little about prospects of the other.

Thus, I think that the proposed premise (1) isn't quite right, and that the case for ID inevitably failing that criterion in premise (2) is not quite made either.

So, regardless of how much damage Ayala's criticisms may have inflicted on any purported success of specific ID theories (and again, I do not contend that ID has established much), I do not believe that Ayala has materially damaged the scientific legitimacy of the intelligent design *project* as such. So far as we can say, at this point, the possibility of a legitimate role for a robust sense of even *intelligent* design in biology remains.

Notes

1 Various authors (e.g., A.N. Whitehead, Stanley Jaki) have been read this way. Among the early examples is M.B. Foster (1934):

> [T]he conclusion follows that only a created nature is proper object of an empirical science. . . . Modern natural science could begin only when the modern presuppositions about nature displaced the Greek . . . but this displacement itself was possible only when the Christian conception of God had displaced the Pagan, as the object (not merely of unreasoning belief, but) of systematic understanding. (p. 465).

2 One representative example of this type is Scott (1993, p. 43).

3 The subtitle of Richard Dawkins' *The Blind Watchmaker* (1996) is "Why the evidence of evolution reveals a universe without design."

4 Here is just one example. In a 1599 letter to Johannes Georg Herwart von Hohenburg, Kepler wrote:

> Those [laws] are within the grasp of the human mind. God wanted us to recognize them by creating us after his own image so that we could share in his own thoughts . . . and, if piety allows us to say so, our understanding is in this respect of the same kind as the divine, at least as far as we are able to grasp something of it in our mortal life. (cited in Frankenberry, 2008, p. 45)

5 Examples abound of theists who believe that the cosmos and much in it is deliberately designed, but Hearn's *Being a Christian in Science* (1997)—which was published under the

auspices of the American Scientific Affiliation, a group of Christian professional scientists having several thousand members—is representative. For an example of a theist who believes that God plays no direct conceptual role in the everyday workings of science, see, e.g., Behe (2004):

> [Some critics of ID do not] think that guidance is necessary in evolution, but if it were (as I believe), then a route would be open for a subtle God to design life without overriding natural law. If quantum events such as radioactive decay are not governed by causal laws, then it breaks no law of nature to influence such events. As a theist . . . that seems perfectly possible to me. I would add, however, that such a process would amount to Intelligent Design. . . . Further, while we might not be able to detect quantum manipulations, we may nevertheless be able to conclude confidently that the final structure was designed. (p. 358)

6 Michael Behe (2006), for instance, takes this to be a legitimate ID position. Other ID advocates endorse "front-loading," according to which basic design specifications for subsequent evolutionary developments were built into the genetics of primordial cells. Front-loading is defended in Gene (2007).

7 For example, the following appears on the Discovery Institute website (http://www.discovery.org/csc/topQuestions.php):

> Discovery Institute's science education policy has been consistent and clear. We strongly believe that teaching about intelligent design is constitutionally permissible, but we think mandatory inclusion of intelligent design in public school curricula is ill-advised. Instead, we recommend that schools require only that the scientific evidence for and against neo-Darwinism be taught, while not infringing on the academic freedom of teachers to present appropriate information about intelligent design if they choose.

8 For good general critiques see Newton-Smith (1981) and Thornton (2006).

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CHAPTER TWENTY

There Is No Place for Intelligent Design in the Philosophy of Biology

Intelligent Design Is Not Science

Francisco J. Ayala

The argument from design has two parts. In one familiar form it asserts, first, that organisms evince to have been designed; second, that only God could account for the design. The argument from design was advanced, in a variety of forms, in Classical Greece and early Christianity. In the thirteenth century, it was proposed by Thomas Aquinas as one of five arguments to demonstrate the existence of God, and received much elaboration during the ensuing centuries, but it was mercilessly criticized by David Hume. Its most extensive formulation is due to William Paley in his *Natural Theology* (1802). The eye—as well as all sorts of organs, organisms, and their interactions—manifests to be the outcome of design and not of chance, thus shows to have been created by God.

Darwin's (1859) theory of evolution by natural selection disposed of Paley's arguments: the adaptations of organisms are outcomes not of chance, but of a process that, over time, causes the gradual accumulation of features beneficial to organisms, whenever these features increase the organisms' chances of surviving and reproducing.

In the 1990s, the design argument was revived in the United States by several authors. The flagellum used by bacteria for swimming and the immune system of mammals, as well as some improbability calculations, were advanced as evidence of "intelligent design" (ID), on the grounds that natural processes could not account for the phenomena to be explained. Scientists have refuted these arguments with extensive evidence. ID is not a scientifically acceptable proposal, because it cannot be empirically tested, nor has it produced any scientific results (Ayala, 2007).

There is "design" in the living world: eyes are designed for seeing, wings for flying, and kidneys for regulating the composition of the blood. The design of organisms comes about not by intelligent design, but by the interaction of mutation and natural selection, in a process that is creative through the interaction of chance and necessity.

1 Introduction: The Design Argument

The argument from design is a two-tined argument. The first prong, as formulated for example by the English author William Paley (1743–1805), asserts that organisms, in their wholes, in their parts, and in their relations to one another and to the environment, appear to have been designed for serving certain functions and to fulfill certain ways of life. The second prong of the argument affirms that only God, an omnipotent and omniscient creator, could account for the diversity, perfection, and functionality of living organisms and their parts.

The argument from design has been repeatedly advanced through history, but it has been formulated in different versions, with variable scope for each of the two prongs. The first prong comes, importantly, in at least two flavors. One version refers to the order and harmony of the universe as a whole; as for example, in St. Augustine (1998): "The world itself, by the perfect order of its changes and motions and by the great beauty of all things visible" (pp. 452–453); or in St. Thomas Aquinas (1905): "It is impossible for contrary and discordant things to fall into one harmonious order except under some guidance, assigning to each and all parts a tendency to a fixed end. But in the world we see things of different natures falling into harmonious order" (p. 12). The second version of the first prong refers to the living world, the intricate organized complexity of organisms, as formulated, among others, by William Paley (1802) and the modern proponents of intelligent design (ID).

The second prong of the argument from design has been formulated in, at least, three important versions. One formulation of the Designer appears in Classical Greece, including Plato (1997a), who postulates the existence of a Demiurge, a creator of the universe's order, who is a universal and impersonal ordering principle, rather than the personalized Judeo-Christian God. Plato's Demiurge is an orderer of the world who accounts for the world's rationality, but not necessarily for its creation. A second version of the Designer is the familiar one of the Judeo-Christian God, as formulated by Paley and other Christian philosophers and theologians (e.g., Aquinas, 1905, 1964; Swinburne, 1994), who is a "person," the creator and steward of the universe, who creates a world from nothing and is omniscient, omnipotent, omnibenevolent, and is provident for humans.[1]

Proponents of ID have in recent years formulated a third version of the second prong of the argument: an unidentified Designer who may account for the order and complexity of the universe, or who may simply intervene from time to time in the universe so as to design organisms and their parts, because the complexity of organisms, it is claimed, cannot be accounted for by natural processes. According to ID proponents, this intelligent designer could be, but need not be, God. The intelligent designer could be an alien from outer space or some other creature, such as a "time-traveling cell biologist," with amazing powers to account for the universe's design. Explicit reference to God is avoided, so that the "theory" of ID can be taught in the public schools as an alternative to the theory of evolution without incurring conflict with the U.S. Constitution, which forbids the endorsement of any religious beliefs in public institutions.

2 The Design Argument in Antiquity

Anaxagoras of Clazomenae (ca. 500–428 BCE) was among the early Presocratic Greek philosophers who formulated versions of the argument from design. Anaxagoras primarily concerned himself with astronomical and meteorological questions, but also addressed biological doctrines. He postulated a Mind that accounts for order in the world: "All things that were to be . . . those that were and those that are now and those that shall be. . . . Mind arranged them all, including this rotation in which are now rotating the stars, the sun and moon" (Kirk, Raven, & Schofield, 1983, p. 363).

Other Presocratic Greek philosophers who saw the presence of a Mind or "ordering principle" in the harmony of the cosmos and the Earth include Diogenes of Apollonia, a near contemporary of Anaxagoras:

> Men and the other living creatures live by means of air through breathing it. And this is for them both soul [i.e., life principle] and intelligence. . . . And it seems to me that that which has intelligence is what men call air, and that all men are steered by this and that it has power over all things. For this very thing seems to me to be a god and to have reached everywhere and to dispose all things and to be in everything. (Kirk et al., 1983, p. 442)

The argument from design is attributed to Socrates (470–399 BCE) by Xenophon. In the *Phaedo*, Plato (427–347 BCE) puts the argument in Socrates' mouth: "I have heard someone reading, as he said, from a book by Anaxagoras, and saying that it is Mind that directs and is cause of everything and placed each thing severally as it was best that it should be" (Plato, 1997b, 97c, p. 84). In the *Timaeus*, Plato attributes creative powers to this Mind, which does not create by making something out of nothing, but accounts for rational order in the world and for the configuration of organisms:

> Prior to the coming to be of time, the universe had already been made to resemble in various respects the model in whose likeness the god was making it, but the resemblance still fell short in that it didn't yet contain all the living things that were to have come to be within it. This he [Mind] went on to perform. (Plato, 1997a, 39e, p. 1243)

Among the ancient Romans, it was particularly Marcus Tullius Cicero (106–43 BCE), the great statesman and orator, who argued that the purposeful complexity of the living world, such as we see in the eye, could not come about by chance, or without guidance. The design argument was, however, dismissed by Lucretius (99–55 BCE): "This world was made naturally, and without Design, and the Seeds of Things of their own accord jostling together by Variety of Motions, rashly sometimes, in vain often, and to no purpose" (Lucrecius, 1743, p. 185).

3 Christian Authors

The argument from design was advanced in the early centuries of the Christian era on the basis of the overall harmony and perfection of the universe. Augustine (354–430),

as quoted above, affirms that the "world itself, by the perfect order of its changes and motions and by the great beauty of all things visible, proclaims . . . that it has been created, and also that it could not have been made other than by a God ineffable and invisible in greatness, and . . . in beauty" (Augustine, 1998, pp. 452–453). According to St. Thomas Aquinas, the argument from design had also been proposed by St. John of Damascus (675–749 CE).

Aquinas formulated the argument from design as the fifth way to demonstrate the existence of God. Aquinas distinguished between truths, such as the Incarnation and the Trinity, that can be known only by divine revelation and truths accessible by human reason, which include God's existence. In his *Summa Theologiae*, Aquinas advances five ways to demonstrate, by natural reason, that God exists. The fifth way derives from the orderliness and designed purposefulness of the universe, which evince that it has been created by a Supreme Intelligence: "Some intelligent being exists by which all natural things are directed to their end; and this being we call God" (Aquinas, 1964, I, 2, 3).[2]

The most forceful and elaborate formulation of the argument from design, before William Paley, was *The Wisdom of God Manifested in the Works of Creation* (1691) by the English clergyman and naturalist John Ray (1627–1705). Ray regarded as incontrovertible evidence of God's wisdom that all components of the universe—the stars and the planets, as well as all organisms—are so wisely contrived from the beginning and perfect in their operation. The "most convincing argument of the Existence of a Deity," writes Ray (1691), "is the admirable Art and Wisdom that discovers itself in the Make of the Constitution, the Order and Disposition, the Ends and uses of all the parts and members of this stately fabric of Heaven and Earth" (p. 33).

The design argument was advanced, in greater or lesser detail, by a number of authors in the seventeenth and eighteenth centuries (see, e.g., Arp, 1999; Klocker, 1968). John Ray's contemporary Henry More (1614–1687) saw evidence of God's design in the succession of day and night and of the seasons: "I say that the Phenomena of Day and Night, Winter and Summer, Spring-time and Harvest . . . are signs and tokens unto us that there is a God . . . things are so framed that they naturally imply a Principle of Wisdom and Counsel in the Author of them. And if there be such an Author of external Nature, there is a God" (More, 1662, p. 38). Robert Hooke (1635–1703), a physicist and eventual Secretary of the Royal Society, formulated the watchmaker analogy: God had furnished each plant and animal "with all kinds of contrivances necessary for its own existence and propagation . . . as a Clock-maker might make a Set of Chimes to be a part of a Clock" (Hooke, 1665, p. 124). The clock analogy, among other analogies such as temples, palaces, and ships, was also used by Thomas Burnet (1635–1703) in his *Sacred Theory of the Earth* (1651/1965), and it would become common among British natural theologians of the time.

On the Continent, the Dutch philosopher and theologian Bernard Nieuwentijdt (1654–1718) developed, at length, the argument from design in his three-volume treatise, *The Religious Philosopher*, where, in the Preface, he introduces the watchmaker analogy (Nieuwentijdt, 1718/2007). Voltaire (1694–1778), like other philosophers of the Enlightenment, accepted the argument from design. Voltaire asserted that in the same way as the existence of a watch proves the existence of a watchmaker, the design and purpose evident in nature prove that the universe was created by a Supreme Intelligence (Voltaire, 1967, pp. 262–270).

4 Hume's Onslaught

The great Scottish philosopher David Hume (1711–1776) wrote a multi-pronged attack against the design argument in his posthumously published *Dialogues Concerning Natural Religion* (1779)., the dialogues in question being with an imaginary interlocutor, Cleanthes. First, Hume denies the first prong of the design argument, namely, that the Universe gives evidence of design, just as a house does: "If we see a house, Cleanthes, we conclude, with the greatest certainty, that it had an architect or builder, because this is precisely that species of effect, which we have experienced to proceed from that species of cause. But surely you will not affirm, that the universe bears such a resemblance to a house" (Hume, 1779/2006, p. 23).

Indeed, the world is more like a vegetable or an animal, rather than a machine, writes Hume, in which case we may have infinite regress, one world generated by a previous one, which in turn would have been generated by another previous world, and so on: "The world plainly resembles more an animal or a vegetable, that it does a watch or a knitting-loom. Its cause, therefore, it is more probable, resembles the cause of the former" (Hume, 1779/2006, p. 62). An additional argument against the world giving evidence of design is that the world may contain, in itself, an ordering principle or power: "[O]rder, arrangement, or the adjustment of final causes, is not, of itself, any proof of design; but only so far as it has been experienced to proceed from that principle. For aught we can know *a priori*, matter may contain the source or spring of order originally within itself, as well as mind does" (Hume, 1779/2006, p. 25).

Hume further points out that the inference from design to a designer would not be warranted in this case, because it is based on the observation of a single instantiation —namely, the existence of the world—lacking the repetition of observations that warrant valid inference. The inference to a designer is, therefore, based only on an analogy, and a weak analogy at that:

> When two species of objects have always been observed to be conjoined together, I can *infer*, by custom, the existence of one wherever I *see* the existence of the other; and this I call an argument from experience. But how this argument can have place where the objects, as in the present case, are single, individual, without parallel or specific resemblance, may be difficult to explain. . . . That a stone will fall, that a fire will burn, that the earth has solidity, we have observed a thousand and a thousand times; and when any new instance of this nature is presented, we draw without hesitation the accustomed inference. The exact similarity of the cases gives us a perfect assurance of a similar event; and a stronger evidence is never desired nor sought after. But wherever you depart, in the least, from the similarity of the cases, you diminish proportionately evidence; and may at last bring it to a very weak *analogy*, which is confessedly liable to error and uncertainty. (Hume, 1779/2006, pp. 30, 23)

The design argument, says Hume, is anthropomorphic and, as such, it diminishes a proper conception of God, reducing it to the human level: the design argument, "may it not render us presumptuous, by making us imagine we comprehend the Deity and have some adequate idea of his nature and attributes? . . . by representing the Deity as so intelligible and comprehensible, and so similar to a human mind, we are guilty of the grossest and most narrow partiality, and make ourselves the model of the whole universe" (Hume, 1779/2006, pp. 37–38; also see Arp, 1998).

5 William Paley's *Natural Theology*

William Paley, one of the most influential English authors of his time, argued forcefully in his *Natural Theology* (1802) that the complex and precise design of organisms and their parts could be accounted for only as the deed of an Intelligent and Omnipotent "Designer." The design of organisms, he argued, was incontrovertible evidence of the existence of the Creator.

Paley was an influential writer of works on Christian philosophy, ethics, and theology, such as *The Principles of Moral and Political Philosophy* (1785) and *A View of the Evidences of Christianity* (1794). With *Natural Theology*, Paley sought to update Ray's *Wisdom of God Manifested in the Works of the Creation* (1691). But Paley could now carry the argument much further than Ray, by taking advantage of one century of additional biological knowledge. Paley's (1802) keystone claim is that there "cannot be design without a designer; contrivance, without a contriver; order, without choice; . . . means suitable to an end, and executing their office in accomplishing that end, without the end ever having been contemplated" (pp. 15–16).

Natural Theology is a sustained argument for the existence of God based on the obvious design of humans and their organs, as well as the design of all sorts of organisms, considered by themselves and in their relations to one another and to their environment. The argument has two parts: first, that organisms give evidence of being designed; second, that only an omnipotent God could account for the perfection, multitude, and diversity of the design.

Paley's first analogical example in *Natural Theology* is the human eye. Early in Chapter 3, Paley points out that the eye and the telescope "are made upon the same principles; both being adjusted to the laws by which the transmission and refraction of rays of light are regulated" (p. 20). Specifically, there is a precise resemblance between the lenses of a telescope and "the humors of the eye" in their figure, their position, and the ability of converging the rays of light at a precise distance from the lens—on the retina, in the case of the eye.

Paley makes two remarkable observations, which enhance the complex and precise design of the eye. The first observation is that rays of light should be refracted by a more convex surface when transmitted through water than when passing out of air into the eye. Accordingly, "the eye of a fish, in that part of it called the crystalline lens, is much rounder than the eye of terrestrial animals. What plainer manifestation of design can there be than this difference? What could a mathematical instrument maker have done more to show his knowledge of [t]his principle . . . ?" (p. 20).

The second remarkable observation made by Paley in support of his argument is dioptric distortion:

> Pencils of light, in passing through glass lenses, are separated into different colors, thereby tinging the object, especially the edges of it, as if it were viewed through a prism. To correct this inconvenience has been long a desideratum in the art. At last it came into the mind of a sagacious optician, to inquire how this matter was managed in the eye, in which there was exactly the same difficulty to contend with as in the telescope. His observation taught him that in the eye the evil was cured by combining lenses composed of different substances, that is, of substances which possessed different refracting powers. (pp. 22–23)

The telescope maker, accordingly, corrected the dioptric distortion "by imitating, in glasses made from different materials, the effects of the different humors through which the rays of light pass before they reach the bottom of the eye. Could this be in the eye without purpose, which suggested to the optician the only effectual means of attaining that purpose?" (p. 23).

Paley summarizes his argument by asserting the complex functional anatomy of the eye. The eye consists, "first, of a series of transparent lenses—very different, by the by, even in their substance, from the opaque materials of which the rest of the body is, in general at least, composed." Second, the eye has the retina, which, as Paley points out, is the only membrane in the body that is black, spread out behind the lenses, so as to receive the image formed by pencils of light transmitted through them, and "placed at the precise geometrical distance at which, and at which alone, a distinct image could be formed, namely, at the concourse of the refracted rays." Third, he writes, the eye possesses "a large nerve communicating between this membrane [the retina] and the brain; without which, the action of light upon the membrane, however modified by the organ, would be lost to the purposes of sensation" (p. 48).

Natural Theology has chapters dedicated to the human frame, which displays a precise mechanical arrangement of bones, cartilage, and joints; to the circulation of the blood and the disposition of blood vessels; to the comparative anatomy of humans and animals; to the digestive tract, kidneys, urethra, and bladder; to the wings of birds and the fins of fish; and much more. For 352 pages, *Natural Theology* conveys Paley's expertise: extensive and accurate biological knowledge, as detailed and precise as was available in the year 1802. After detailing the precise organization and exquisite functionality of each biological entity, relationship, or process, Paley draws again and again the same conclusion: only an omniscient and omnipotent Deity could account for these marvels of mechanical perfection, purpose, and functionality, and for the enormous diversity of inventions that they entail.

The strength of the argument against chance derives, Paley tells us, from what he names "relation," a notion akin to what Michael Behe (1996), a modern proponent of ID, has named "irreducible complexity." This is how Paley formulates the argument for irreducible complexity:

> When several different parts contribute to one effect, or, which is the same thing, when an effect is produced by the joint action of different instruments, the fitness of such parts or instruments to one another for the purpose of producing, by their united action, the effect, is what I call *relation*; and wherever this is observed in the works of nature or of man, it appears to me to carry along with it decisive evidence of understanding, intention, art. (Paley, 1802, pp. 175–176)

The outcomes of chance do not exhibit relation among the parts or, as we might say, they do not display organized complexity. Paley writes that: "The feet of the mole are made for digging; the neck, nose, eyes, ears, and skin, are peculiarly adapted to an under-ground life. [In a word,] this is what I call relation" (p. 183).

Throughout *Natural Theology*, Paley displays extensive and profound biological knowledge. He discusses the fish's air bladder, the viper's fang, the heron's claw, the camel's stomach, the woodpecker's tongue, the elephant's proboscis, the bat's

wing hook, the spider's web, insects' compound eyes and metamorphosis, the glowworm, univalve and bivalve mollusks, seed dispersal, and on and on, with as much accuracy and detail as known to the best biologists of his time. The complex disposition and purposeful function of the organisms' components reveal, in each case, an intelligent designer; and the diversity, richness, and pervasiveness of the designs show that only the omnipotent Creator could be this intelligent designer.

6 The Bridgewater Treatises

In 1829, nearly three decades after the publication of *Natural Theology*, Francis Henry Egerton (1756–1829), the eighth Earl of Bridgewater, bequeathed the sum of eight thousand pounds sterling with instructions to the Royal Society that it commission eight treatises that would promote natural theology by setting forth "The Power, Wisdom and Goodness of God as manifested in the Creation." Eight treatises were published in the 1830s, several of which artfully incorporate the best science of the time and had considerable influence on the public and among scientists. One additional treatise, never completed, was authored by the notable mathematician and pioneer in the field of calculating machines, Charles Babbage: *The Ninth Bridgewater Treatise: A Fragment* (1838), where he seeks to show how mathematics may be used to bolster religious belief. He advances the unexpected proposition that it "is more probable that any law, at the knowledge of which we have arrived by observation, shall be the subject to one of those violations, which, according to Hume's definition, constitutes a miracle, than that it should not be so subjected" (Jones & Cohen, 1963, pp. 69–70).

One of the Bridgewater treatises, *The Hand, Its Mechanisms and Vital Endowments as Evincing Design* (1833), was written by Sir Charles Bell, a distinguished anatomist and surgeon, famous for his neurological discoveries, who became professor of surgery in 1836 at the University of Edinburgh. Bell follows Paley's manner of argument, examining in considerable detail the wondrously useful design of the human hand, but also the perfection of design of the forelimb used for different purposes in different animals, serving in each case the particular needs and habits of its owner: the human's arm for handling objects, the dog's leg for running, and the bird's wing for flying. "Nothing less than the Power, which originally created, is equal to the effecting of those changes on animals, which are to adapt them to their conditions" (Bell, 1833, p. 133).

William Buckland, Professor of Geology at Oxford University, notes in *Geology and Mineralogy* (1836) the world distribution of coal and mineral ores, and proceeds to point out that they had been deposited in a remote part, yet obviously with the forethought of serving the larger human populations that would come about much later. This attribution to the Creator is particularly noteworthy because Buckland in two earlier treatises, *Vindiciae Geologicae* (1820) and *Reliquiae Diluvianae* (1823), had explained sedimentation, fossil deposits, and rock formation as natural processes, without invoking the direct intervention of God (Roberts, 2004, p. 282). Later, another geologist, Hugh Miller in *The Testimony of the Rocks* (1858), would formulate what I call the *argument from beauty*, which allows that it is not only the perfection

of design, but also the beauty of natural structures found in rock formations and in mountains and rivers that manifests the intervention of the Creator.

7 Intelligent Design: A Political Movement

The publication in 1859 of Darwin's *On the Origin of Species* very much disposed of natural theology as an attempt to prove the existence of God based on the argument from design. Nevertheless, in the 1990s several authors, notably biochemist Michael Behe (1996), theorist William Dembski (1995, 2002), and law professor Phillip Johnson (1993, 2002), among others, revived the argument from design (see Ayala, 2006, 2007; Dembski & Ruse, 2004; Pennock, 2001; Scott, 2005; Young & Edis, 2004). Often, however, these authors sought to hide their real agenda—and, thus, typically avoided explicit reference to God, so that the "theory" of intelligent design could be taught in the public schools, as an alternative to the theory of evolution, without incurring conflict with the U.S. Constitution, which forbids the endorsement of any religious beliefs in public institutions. These modern proponents, at times, claim that the intelligent designer need not be God, but could, as suggested above, be a space alien or some other intelligent superpower unknown to us (Scott, 2005). The folly of this pretense is apparent to anyone who takes the time to consider the issue seriously. It is nothing but a vulgar charade.[3]

Proponents of ID call for an intelligent designer to explain the supposed irreducible complexity in organisms. An *irreducibly complex system* is defined by Behe (1996) as an entity "composed of several well-matched, interacting parts that contribute to the basic function, wherein the removal of any one of the parts causes the system to effectively cease functioning" (p. 39). The claim is that irreducibly complex systems cannot be the outcome of evolution, which proceeds by small steps, slowly accumulating over thousands or millions of generations the components of complex systems. According to Behe:

> An irreducibly complex system cannot be produced directly . . . by slight, successive modifications of a precursor system, because any precursor to an irreducible complex system that is missing a part is by definition nonfunctional. . . . Since natural selection can only choose systems that are already working, then if a biological system cannot be produced gradually it would have to arise as an integrated unit, in one fell swoop, for natural selection to have anything to act on. (p. 72)

In other words, unless all parts of the eye come simultaneously into existence, the eye cannot function; it does not benefit a precursor organism to have just a retina, or a lens, if the other parts are lacking. The human eye, according to this argument, could not have evolved one small step at a time, in the piecemeal manner by which natural selection works.

Evolutionists have pointed out, again and again, with supporting evidence, that organs and other components of living beings are not irreducibly complex—they do not come about suddenly, or in one fell swoop (see, for example, Ayala, 2007; Brauer & Brumbaugh, 2001; Miller, 1999, 2004; Perakh, 2004a; Pennock, 2002). Evolutionists have shown that organs such as the human eye are not irreducible at all; rather, less

complex versions of the same systems have existed in the past, and some can be found in today's organisms as well.

8 Eyes to See

Eyes evolved gradually and achieved very different configurations in different organisms, all serving the function of vision. The simplest "organ" of vision occurs in some single-celled organisms that have enzymes or spots sensitive to light, which help them move toward the surface of their pond, where they feed on the algae growing there. Some multicellular animals exhibit light-sensitive spots on their epidermis. Further steps—deposition of pigment around the spot, configuration of cells into a cuplike shape, thickening of the epidermis leading to the development of a cornea, development of muscles to move the eyes and nerves to transmit optical signals to the brain—gradually led to the highly developed eyes of vertebrates and cephalopods (octopuses and squids) and to the compound eyes of insects.

A record of the major stages in the evolution of a complex eye has survived in living mollusks (clams, snails, and squids). The eye of octopuses and squids is as complex as the human eye, with cornea, iris, refractive lens, retina, vitreous internal substance, optic nerve, and muscle. Limpets (*Patella*) have about the simplest imaginable eye: just an eye spot consisting of a few pigmented cells with nerve fibers attached to them. Several intermediate stages are found in other living mollusks. One step in complexity above the limpet eye is found in slit-shell mollusks (*Pleurotomaria*), which have a cup-eye, one layer of pigmented cells curved like a cup with a wide opening through which light enters, with each pigmented cell in the back of the cup attached to a nerve fiber. More complex is the pinhole-lens eye found in *Nautilus*, a marine snail. The layer of pigmented cells is considerably more extensive than in slit-shell mollusks: the pigmented cells are covered towards the front with epithelium (skin) cells that are nearly closed except for a small opening ("pinhole") for passage of light, creating a cavity filled with water. *Murex*, another marine snail, has an eye with a primitive, refractive lens covered with epithelium cells (serving as a primitive cornea); the pigmented cells extend through the back of the eye cavity (thus serving as a retina) and the nerve fibers are collected into an optic nerve that goes to the brain. The most advanced mollusk eye is found in the octopus and the squid, an eye that, as mentioned, is just as complex and effective as the human eye. Moreover, the octopus' and squid's eyes lack the human blind spot, an imperfection due to the fact that the nerve fibers of the human eye are collected inside the eye cavity, so that the optic nerve has to cross the retina on its way to the brain; the nerve fibers and the optic nerve of the octopus eye are outside the eye cavity and travel to the brain without crossing the retina (Ayala, 2007; Blake & Truscianko, 1990; Cronly-Dillon, 1991; DeDuve, 1996; Horridge, 1987; Salvini-Plawen & Mayr, 1977).

The gradual process of natural selection adapting organs to functions occurs in a variety of ways, reflecting the haphazard characteristics of the evolutionary process, which are due to mutation, past history, and the vagaries of environments (Ayala, 1982; Ayala & Valentine, 1979; Berra, 1990; Mayr, 2001). In some cases, the changes of an organ amount to a shift of function, as in the evolution of the forelimbs of

vertebrates, which first evolved in amphibians as originally adapted for walking, but which are now used in birds for flying, in whales for swimming, and in humans for handling objects. Other cases, like the evolution of eyes, exemplify gradual advancement of the same function—seeing, in the case of eyes. In all cases, however, the process is impelled by natural selection's favoring through time individuals exhibiting functional advantages over others of the same species.

Examples of functional shifts are many and diverse. Some transitions, at first, may seem unlikely because of the difficulty in identifying which possible functions may have been served during the intermediate stages. These cases are eventually resolved with further research and, often, by the discovery of intermediate fossil forms or living organisms with intermediate stages of development, as in the case of mollusks' eyes. It is similarly the case with the bacterial flagellum, a favorite example of Michael Behe (1996, 2007). In different species of bacteria, there are different kinds of flagella, some simpler than the one described by Behe, others just different, even very different, as in the archaea, a very large bacteria-like group of organisms. Moreover, motility in many bacteria is accomplished without flagella at all. Still more, biochemists have shown that some flagellum components may have evolved from secretory systems, which are very similar to the flagellum, but lack some of the flagellum's components (see, for example, Liu & Ochman, 2007; Pallen & Matzke, 2006). The argument for the irreducible complexity of the flagellum is formulated, like other ID arguments, as an *argument from ignorance*: because one author does not know how a complex organ may have come about, it must be the case that it is irreducibly complex. This argument from ignorance dissolves as scientific knowledge advances, or when pre-existing scientific knowledge is taken into account (see below).

9 No "There" There

The proposition of ID as a scientific alternative to evolution brings to mind Gertrude Stein's quip about Oakland, her native city: "There is no there there." The call for an intelligent designer is predicated by ID proponents on the existence of irreducible complexity in organisms. Irreducible complexity, the claim goes, cannot come about by the stepwise manner of natural selection, the main process by which the theory of evolution accounts for adaptation. But the ID "explanation" is not a scientific hypothesis that can be tested by observation and experiment. Indeed, ID does not advance any explanation, but it amounts only to a negative claim: that the relevant evolutionary explanations are not satisfactory.

ID's lack of scientific cogency is unwittingly displayed by its proponents when confronted with the dysfunctions, imperfections, cruelty, even sadism of the living world. ID proponents affirm that these failings are not a valid argument against organisms being the creations of an intelligent designer. According to Behe (1996), the "argument from imperfection overlooks the possibility that the designer might have multiple motives, with engineering excellence oftentimes relegated to a secondary role. . . . The reasons that a designer would or would not do anything are virtually impossible to know unless the designer tells you specifically what those reasons are" (p. 223).

It is clear, then, that ID is not a scientific hypothesis because the claim just stated provides ID with an empirically impenetrable shield against any predictions, since we know not how "intelligent" or "perfect" a design should be, because the designer has not told us about it. ID, therefore, cannot be empirically tested (Ayala, 2008; National Academy of Sciences and Institute of Medicine, 2008; Pennock, 2002; Sober, 2007; Sonleitner, 2006). Science tests its hypotheses by observing whether or not predictions derived from them hold true in the observable world. A hypothesis that cannot be tested empirically—that is, by observation or experiment—is not scientific. ID as an explanation for the adaptations of organisms could be (natural) theology, as Paley would have it; but, whatever it is, it is not a scientific hypothesis.

There is a fundamental fallacy in the ID logic. The claim is that if evolution fails to explain some biological phenomenon, ID must be the correct explanation. This is a misunderstanding of the scientific process. If one explanation fails, it does not necessarily follow that some other proposed explanation is correct. Explanations must stand on their own evidence, not on the failure of their alternatives. Scientific explanations or hypotheses are creations of the mind, conjectures, imaginative exploits about the makeup and operation of the natural world. It is the imaginative preconception of what might be true in a particular case that guides observations and experiments designed to test whether a hypothesis is correct. The degree of acceptance of a hypothesis is related to the severity of the tests that it has passed (see Ayala, 1994; Giere, Bickle, & Mauldin, 2005; Gott & Duggan, 2003; Hung, 1996).

It is not sufficient for a theory to be accepted because some alternative theory has failed. Oxygen was not discovered simply because it was shown that phlogiston does not exist.[4] Nor is the periodic table of chemical elements accepted just because chemical substances react and yield a variety of components. Similarly, Darwin's theory of evolution by natural selection became generally accepted by scientists not because other evolutionary theories, such as Lamarck's, Bergson's, or Darwin's grandfather Erasmus', had failed the tests of science, but because it has sustained innumerable tests and has been fertile in yielding new knowledge (see, for example, Ghiselin, 1969; Quammen, 2007).

Theodosius Dobzhansky (1967), one of the greatest evolutionists of the twentieth century, has written:

> There are people . . . to whom the gaps in our understanding of nature are pleasing for a different reason. These people hope that the gaps will be permanent, and that what is unexplained will also remain inexplicable. By a curious twist of reasoning, what is unexplained is then assumed to be the realm of divine activity. The historical odds are all against the "God of the gaps" being able to retain these shelters in perpetuity. There is nothing, however, that can satisfy the type of mind which refuses to accept this testimony of historical experience. (p. 13)

Science is a complex enterprise that essentially consists of two interdependent episodes: one imaginative or *creative*, the other *critical*. To have an idea, advance a hypothesis, or suggest what might be true is a creative exercise, but scientific conjectures or hypotheses must also be subject to critical examination and empirical testing. Scientific knowledge may be characterized as a process of invention or discovery followed by validation or confirmation. One episode concerns the formulation

of new ideas, sometimes referred to as the *acquisition of knowledge*; the other concerns the validation of these new ideas, or the justification of knowledge.[5]

New ideas in science are advanced in the form of hypotheses. Hypotheses are mental constructs, imaginative exploits, that provide guidance as to what is worth observing and that encourage the scientist to seek observations that would corroborate or falsify the hypothesis. The tests to which scientific ideas are subjected include contrasting hypotheses with the world of experience in a manner that must leave open the possibility that anyone might reject any particular hypothesis if it leads to wrong predictions about the world of experience. The possibility of empirical falsification of a hypothesis is carried out by ascertaining whether or not precise predictions derived as logical consequences from the hypothesis agree with the state of affairs found in the empirical world. A hypothesis that cannot be subjected to the possibility of rejection by observation and experiment cannot be regarded as scientific. The possibility of empirical falsification of its hypotheses has been called by the philosopher Karl Popper (1959) the "criterion of demarcation" that sets scientific knowledge apart from other forms of knowledge. ID offers not any propositions to test, but makes only the lame claim that the theory of evolution by natural selection cannot account for the complexity of organisms.

10 Blood and Tears

The traditional favorite instantiation of irreducible complexity is the eye of humans and other vertebrates. I have explained in an earlier section that the complexity of the eye is not irreducible but, rather, may come about by steps, starting with the simplest of eyes, just one or a few pigmented cells sensitive to light. The evidence of how the complexity of the eye may arise by gradual steps is there for all to see in living mollusks. Michael Behe, a biochemist, the only proponent of ID who qualifies as a bona fide biologist, has argued other cases of supposed irreducible complexity: the bacterial flagellum, the blood clotting process, and the immune system. That these biological systems are not irreducible has been shown by numerous authors, including Barbara Forrest and Paul Gross (2004), Renyi Liu and Howard Ochman (2007), Kenneth Miller (2004), Ian Musgrave (2004), Mark J. Pallen and Nicholas J. Matzke (2006), Robert Pennock (2002), Mark Perakh (2004a, 2004b), David Ussery (2004), Matt Young and Taner Edis (2004), and others. The bacterial flagellum is, according to Behe, irreducibly complex because it consists of several parts so that, if any part is missing, the flagellum will not function. It could not, therefore, says Behe, have evolved gradually, one part at a time, because the function belongs to the whole; the separate parts cannot function by themselves. "Because the bacterial flagellum is necessarily composed of at least three parts—a paddle, a rotor, and a motor—it is irreducibly complex" (Behe, 1996, p. 72). This inference is, of course, erroneous.

The argument that the different components of the flagellum must have come about "in one fell swoop"—because the parts cannot function separately and, thus, could not have evolved independently—is reminiscent of Paley's argument about the eye. Of what possible use would the iris, cornea, lens, retina, or optic nerve be, one without the others? Yet, we know that component elements of the octopus eye can evolve

gradually, cumulatively, and that simple eyes, as they exist in a limpet, in slit-shell mollusks, and in marine snails, are functional.

The components of the bacterial flagellum have come about gradually in evolution, by genetic evolutionary changes that have been reconstructed in technical publications such as, for example, the articles by Pallen and Matzke (2006) and Liu and Ochman (2007). The components of the flagellum are encoded by gene clusters that may include, in some species, upwards of 50 genes. The number of genes, and the genes themselves, greatly vary among different groups of bacteria. Liu and Ochman (2007) have identified all the flagellar proteins in 41 species from 11 quite diverse groups of bacteria. Twenty-four of the genes encoding the flagellar proteins were already present in the remote common ancestor of all the bacterial species studied. The other genes have come about by duplication and evolution of pre-existing genes. Moreover, many of the core of 24 ancestral genes are also derived from a few pre-existing ones by successive gene duplications that gradually increased their number. The sequence similarity among all the flagellar genes in the 41 bacterial species has allowed Liu and Ochman to reconstruct the successive steps of addition and modification by which modern bacterial flagella have arisen.

An injured person bleeds for a short time until a clot forms, which soon hardens and the bleeding stops. Blood clotting is a very complex process with many interacting components. According to Behe (1996), "*no one on earth has the vaguest idea how the coagulation cascade came to be* [his italics]" (p. 97). As I have written elsewhere (Ayala, 2007, p. 151), this is a remarkable statement, particularly because of the numerous scientific papers about the evolution of the various components of the blood-clotting mechanism in vertebrates, including "The Evolution of Vertebrate Blood Coagulation: A case of Yin and Yang" by the eminent biochemist Russell F. Doolittle, published in 1993. Several of the authors cited above have provided accounts in layman's terms.

Behe (1996) has made the outlandish claim that there is "no publication in the scientific literature—in prestigious journals, specialty journals, or books—that describes how molecular evolution of any real, complex, biochemical system either did occur or even might have occurred" and, in particular, "the scientific literature has no answers to the origin of the immune system" (pp. 185, 138). In fact, however, examples of complex biochemical structures or systems that have arisen from simpler components are very, very numerous. One example concerns the phylogenetic enigma of snail hemoglobin. It turns out that the complex hemoglobin of the planorbid snail *Biomphalaria glabrata* has evolved from pulmonate myoglobin by a simple evolutionary mechanism that creates a high molecular mass respiratory protein from 78 similar globin domains (see Lieb et al., 2006). One example that involves the evolution of a particular protein from a single-cell ancestor to animal descendants appears in the same journal issue (Segawa et al., 2006). I suspect that, if I were to examine relevant scientific journals, I would find, each month, one or more examples showing biochemical structures that have evolved from simpler ones.

As a further response to Behe, it may suffice to quote Judge John Jones III in the *Kitzmiller* v. *Dover* decision:

> Professor Behe was questioned concerning his 1996 claim that science would never find an evolutionary explanation for the immune system. He was presented with fifty-eight

peer-reviewed publications, nine books, and several immunology textbook chapters about the evolution of the immune system; however, he simply insisted that this was still not sufficient evidence of evolution, and that it was not "good enough."

Judge Jones concludes: "We therefore find that Professor Behe's claim for irreducible complexity has been refuted in peer-reviewed research papers and has been rejected by the scientific community at large" (*Kitzmiller* v. *Dover*, 2005, p. 18).

11 Gambling to Non-existence

The mathematically trained William Dembski (1995, 2002) is another ID proponent who has used supposedly scientific arguments to demonstrate the irreducible complexity of organisms. According to Dembski, organisms exhibit "complex specified information," which is information that has a very low prior probability and, therefore, high information content. Dembski argues that mutation and natural selection are incapable of generating such highly improbable states of affairs. Consider the 30 proteins that make up the bacterial flagellum. Assuming that each protein has about 300 amino acids, he calculates that the probability of one such protein is 20^{-300}. After some refinements, he calculates that the probability of origination for the flagellum is 10^{-1170} (one divided by 1 followed by 1,170 zeroes; in order to get some idea of the magnitude of this number, consider that the number of atoms in the universe is estimated to be 10^{77}, or 1 followed by 77 zeroes). Dembski concludes that, even if one would take into account that life has existed on Earth for 3.5 billion years, the assembly of a functioning flagellum is impossibly improbable.

This numerological exercise is, however, irrelevant because Dembski does not take into account the role of natural selection and makes a number of erroneous assumptions, as pointed out in several of the works quoted above and others (see the papers in Dembski & Ruse, 2004; Pennock, 2001). Dembski, in his book *No Free Lunch* (2002), devotes many pages to the optimization of the "no free lunch" theorems of Wolpert and Macready (1997). Dembski's misunderstanding of the theorems and their implications have been pointed out, for example, by Mark Perakh (2004a, 2004b), and has been ridiculed by one of the theorems' authors, D.H. Wolpert, in his "William Dembski's Treatment of No Free Lunch Theorems is Written in Jell-O" (2003). The fundamental fallacies of Dembski's improbability calculations have been exposed in a recent insightful critique by the distinguished evolutionist Joe Felsenstein (2007), who has shown that there "can be no theorem saying that adaptive information is conserved and cannot be increased by natural selection. . . . Specified information, including complex specified information, can be generated by natural selection" (p. 24).

Dembski's numerological exercises suffer from several fatal flaws, including the assumption of a specified final outcome. This point has been amusingly made by developmental biologist Scott F. Gilbert and the Swarthmore College Evolution and Developmental Seminar (2007):

Some of Intelligent Design's most powerful arguments depend on a simple fallacy: the assumption of an end point. . . . [ID proponents claim that] it is impossible to evolve a particular protein because it has 100 amino acids and the chance of this occurring

> randomly is 1 in 20^{100}. . . . But such supporters of ID don't know a billionth of how impossible it is! Let's say that your mother ovulated 500 eggs during her life and that your father produced 2×10^{12} sperm. The chances of *you* being born, then, are 1 in 10^{15} . . . [and] the chances of your grandparents giving rise to you is 1 in 10^{45}. Another reason not to argue with the Intelligent Design people, then, is that, by their own logic, they cannot exist. (pp. 44–45)

It is worthwhile to point out, once again, the vacuity of the claims made by ID proponents. Their arguments consist of the tiresome repetition that something in the world of life cannot have come by natural processes because of its high improbability, or because no satisfactory scientific account exists of that something. Of course, Dembski and the other ID proponents exist. The playful argument about their nonexistence is based on the high improbability of mutations and other chance events, while ignoring natural selection, as they do, and assuming a pre-determined end point, as the ID proponents do as well.

ID proponents have anticipated that, in time, they would make scientific discoveries and even "breakthroughs." The science writer Gordy Slack (2007) has told how he first encountered in January 1998 Phillip Johnson, Professor of Law at the University of California, Berkeley, and often considered as the founding father of the ID movement. Slack and Johnson had lunch at the university's Faculty Club. In response to Slack's criticisms, Johnson advanced the following promise: "Give us five or ten years, and you'll see scientific breakthroughs biologists hadn't dreamed of before ID" (p. vii). Ten years have passed by since Johnson's promise was made. No scientific "breakthroughs" have been delivered, nor, indeed, any sort of scientific knowledge. Once again, there is no *there* there.

12 Natural Selection

Natural selection was proposed by Darwin (1859) primarily to account for the adaptive organization, or design, of living beings; it is a process that preserves and promotes adaptation. Evolutionary change through time and evolutionary diversification (multiplication of species) often ensues as by-products of natural selection, fostering the adaptation of organisms to their milieu. Evolutionary change is not directly promoted by natural selection and, therefore, it is not its necessary consequence. Indeed, some species remain unchanged for long periods of time, as Darwin noted. *Nautilus*, *Lingula*, and other so-called "living fossils" are Darwin's examples of organisms that have remained unchanged in their appearance for millions of years.

Evolution affects all aspects of an organism's life: morphology (form and structure), physiology (function), behavior, and ecology (interaction with the environment). Underlying these changes are changes in the hereditary materials. Hence, in genetic terms, evolution consists of changes in the organisms' hereditary makeup.

As a genetic process, evolution can be seen as a two-step process. First, hereditary variation arises by mutation; second, selection occurs by which useful variations increase in frequency and those that are less useful or injurious are eliminated over the generations. As Darwin (1859) saw it, individuals having useful variations "would

have the best chance of surviving and procreating their kind" (p. 81). As a consequence, useful variations increase in frequency over the generations, at the expense of those that are less useful or are injurious.

Natural selection is much more than a "purifying" process, for it is able to generate novelty by increasing the probability of otherwise extremely improbable genetic combinations. Natural selection, in combination with mutation, becomes, in this respect, a creative process. Moreover, it is a process that has been occurring for many millions of years, in many different evolutionary lineages, and in a multitude of species, each consisting of a large number of individuals. Evolution by mutation and natural selection has produced the enormous diversity of the living world with its wondrous adaptations.

Natural selection is an incremental process, operating over time and yielding organisms better able to survive and reproduce than others. Individuals of a given species differ from one another, at any one time, only in small ways: for example, the difference between bacteria that have or lack an enzyme able to synthesize the sugar lactose, or between moths that have light or dark wings. These differences typically involve one or only a few genes (Carroll, 2005), but they can make the difference between survival or death, as in the evolution of resistance to DDT in disease-transmitting mosquitoes or to antibiotics in people. Consider a different sort of example. Some pocket mice (*Chaetodipus intermedius*) live in rocky outcrops in Arizona. Light, sandy-colored mice are found in light-colored habitats, whereas dark (melanic) mice prevail in dark rocks formed from ancient flows of basaltic lava. The match between background and fur color protects the mice from avian and mammal predators that hunt guided largely by vision. Mutations in one single gene (coding for the melanocortin-1-receptor, represented as *MC1R*) account for the difference between light and dark pelage (Nachman, Hoekstra, & D'Agostino, 2003).

Adaptations that involve complex structures, functions, or behaviors involve numerous genes. Many familiar mammals, but not marsupials, have a placenta. Marsupials include the familiar kangaroo and other mammals native primarily to Australia and South America. Dogs, cats, mice, donkeys, and primates are placental. The placenta makes it possible to extend the time the developing embryo is kept inside the mother and thus make the newborn better prepared for independent survival. However, the placenta requires complex adaptations, such as the suppression of harmful immune interactions between mother and embryo, delivery of suitable nutrients and oxygen to the embryo, and the disposal of embryonic wastes. The mammalian placenta evolved more than 100 million years ago and proved a successful adaptation, contributing to the explosive diversification of placental mammals in the Old World and North America.

The placenta also has evolved in some fish groups, such as *Poeciliopsis*. Some *Poeciliopsis* species hatch eggs. The females supply the yolk in the egg, which furnishes nutrients to the developing embryo (as in chicken). Other *Poeciliopsis* species, however, have evolved a placenta through which the mother provides nutrients to the developing embryo. Molecular biology has made possible the reconstruction of the evolutionary history of *Poeciliopsis* species. A surprising result is that the placenta evolved independently three times in this fish group. The required complex adaptations accumulated in each case in less than 750,000 years (Reznick, Mateos, & Springer, 2002; see Avise, 2006).

Natural selection produces combinations of genes that would seem highly improbable because natural selection proceeds stepwise over long periods of time. Consider the evolution of the eye in humans and other vertebrates. Perception of light, and later vision, were important for the survival and reproductive success of their ancestors, because sunlight is a predominant feature of the environment. Accordingly, natural selection favored genes and gene combinations that increased the functional efficiency of the eye, according to the needs of different sorts of organisms. Such mutations gradually accumulated, eventually leading to the highly complex and efficient vertebrate eye.

Several hundred million generations separate modern animals from the early animals of the Cambrian geological period (542 million years ago). The number of mutations that can be tested, and those eventually selected, in millions of individual animals over millions of generations is difficult for a human mind to fathom, but we can readily understand that the accumulation of millions of small, functionally advantageous changes could yield remarkably complex and adaptive organs, such as the eye.

13 Natural Selection and Design

Natural selection, sorting out spontaneously arising mutations, is a creative process because it causes favorable mutations to combine and accumulate, yielding a great diversity of organisms over eons of time. But there are important features that distinguish the kind of "design" achieved by natural selection—namely, the adaptations of organisms—from the kind of design produced by an intelligent designer, an engineer.

An engineer has a preconception of what the design is supposed to achieve, and will select suitable materials and arrange them in a preconceived manner so that the design fulfills the intended function. On the contrary, natural selection does not operate according to some preordained plan. It is a purely natural process resulting from the interacting properties of physicochemical and biological entities. Natural selection is simply a consequence of the differential survival and reproduction of living beings. It has some appearance of purposefulness because it is conditioned by the environment: which organisms survive and reproduce more effectively depends on which variations they happen to possess that are useful or beneficial to them, in the place and at the time where they live.

Natural selection does not have foresight; it does not anticipate the environments of the future. Drastic environmental changes may introduce obstacles that are insuperable to organisms that were previously thriving. In fact, species extinction is a common outcome of the evolutionary process. The species existing today represent the balance between the origin of new species and their eventual extinction. The available inventory of living species describes nearly 2 million species, although at least 10 million are estimated to exist. But we know that perhaps more than 99 percent of all species that have ever lived on Earth have become extinct (Aitken, 1998; Eldredge, 1991).

Increased complexity is not a necessary consequence of natural selection, but it does emerge occasionally, when mutations that increase complexity are favored over mutations that do not. That complexity-increasing mutations do not necessarily accumulate over time is apparent in many evolutionary lineages. For example, the

longest living organisms on Earth are the microscopic bacteria, which have existed continuously on our planet for about 3.5 billion years. Yet, modern bacterial species appear to exhibit no greater complexity than their ancient ancestors. More complex organisms came about much later, without the elimination of their simpler relatives. Nevertheless, over the eons, multitudes of complex organisms have arisen on Earth. Some groups of complex organisms came into existence only recently (on the evolutionary scale). The primates appeared on Earth only 50 million years ago; our species, *Homo sapiens*, less than 200,000 years ago (Cela-Conde & Ayala, 2007; National Academy of Sciences and Institute of Medicine, 2008).

In evolution, there is no entity or person who is selecting adaptive combinations. These combinations select themselves because the organisms possessing them reproduce more effectively than do those with less adaptive variations. Therefore, natural selection does not strive to produce predetermined kinds of organisms, but only organisms that are adapted to their present environments. As pointed out, which characteristics will be selected depends on which variations happen to be present at a given time in a given place. This, in turn, depends on the random process of mutation, as well as on the previous history of the organisms (that is, on the genetic makeup they have as a consequence of their previous evolution). Natural selection is an opportunistic process. The variables determining the direction in which natural selection will proceed are the environment, the pre-existing constitution of the organisms, and the randomly arising mutations.

Thus, adaptation to a given habitat may occur in a variety of different ways. For example, many plants have adapted to a desert climate. Their fundamental adaptation is to the condition of dryness, which holds the danger of desiccation. During most of the year, and sometimes for several years in succession, there is no rain. Plants have adapted to the scarcity of water in different ways. Cacti have transformed their leaves into spines, and thus avoid the evaporation that occurs in the leaves; photosynthesis is performed on the surface of the stem instead. In addition, their stems have evolved into barrel-like structures that store a reserve of water. A second mode of adaptation occurs in desert plants that have no leaves during the dry season, but, after it rains, they burst into leaves and flowers and quickly produce seeds. A third mode of adaptation is that of desert ephemeral plants, which germinate from seeds, grow, flower, and produce seeds, all within the few weeks of the year when rainwater is available; at other times, the seeds lie quiescent in the soil.

The fossil record shows that life has evolved in a haphazard fashion. The radiations of some groups of organisms, the numerical and territorial expansions of other groups, the replacement of some kinds of organisms by other kinds, the occasional but irregular occurrence of trends toward increased size or other sorts of change, and the ever-present extinctions are best explained by natural selection of organisms subject to the vagaries of genetic mutation, environmental challenge, and past history. The scientific account of these events does not necessitate recourse to a preordained plan, whether imprinted from the beginning or through successive interventions by an omniscient and almighty Designer. Biological evolution differs from a painting or an artifact in that it is not the outcome of preconceived design. The design of organisms is not intelligent, but imperfect and, at times, outright dysfunctional.

The arguments of ID proponents that state the incredible improbability of chance events, such as mutation, in order to account for the adaptations of organisms are

irrelevant because evolution is not governed by random mutations. Rather, there is a natural process (namely, natural selection) that is not random, but oriented and able to generate order or "create." The traits that organisms acquire in their evolutionary histories are not fortuitous but, rather, determined by their functional utility to the organisms, designed, as it were, to serve their life needs. Natural selection preserves what is useful and eliminates what is harmful. Without hereditary mutations, evolution could not happen because there would be no variations that could be differentially conveyed from one to another generation. But without natural selection, the mutation process would yield disorganization and extinction because most mutations are disadvantageous. Mutation and selection have jointly driven the marvelous process that, starting from microscopic organisms, has yielded orchids, birds, and humans.

The theory of evolution conveys chance and necessity jointly enmeshed in the stuff of life; randomness and determinism interlocked in a natural process that has spurted the most complex, diverse, and beautiful entities that we know of in the universe: the organisms that populate the Earth, including humans who think and love, endowed with free will and creative powers, and able to analyze the process of evolution itself that brought them into existence.

Postscript: Counterpoint

The steering wheel of a car has been designed for turning; the human eye has been designed for seeing. Most of us would be willing to accept these two statements, but would probably balk if somebody claimed that a mountain has been designed for climbing. We might note that mountain slopes are there whether or not there is anybody to climb them, but steering wheels would never have been produced if it were not for the purpose they serve. Mountain slopes and steering wheels have in common that they are used for certain purposes, but differ because steering wheels, but not mountain slopes, have been specially created for the purpose they serve. This is what we mean when we say that steering wheels are "designed" for turning: the reason why steering wheels exist at all and exhibit certain features is that they have been designed for turning the car. This is not so with mountain slopes.

But what about eyes? Human eyes share something in common with steering wheels and something with mountain slopes. Human eyes like steering wheels have been "designed," because were it not for the function of seeing they serve, eyes would have never come to be; and the features exhibited by eyes specifically came to be in order to serve for seeing. But eyes share in common with mountain slopes that both came about by natural processes, the eyes by natural selection, the mountain slopes by geological movements and erosion. Steering wheels, on the contrary, are designed and produced by human engineers.

In *The Origin of Species* (1859) Darwin accumulated an impressive number of observations supporting the evolutionary origin of living organisms. Moreover, and most importantly, he advanced a causal explanation of evolutionary change—the theory of natural selection, which provides a natural account of the design of organisms, or, as we say in biology, their adaptations. Darwin accepted that organisms are adapted to live in their environments, and that their parts are adapted to the specific functions

they serve. Penguins are adapted to live in the cold, the wings of birds are made to fly, and the eye is made to see. Darwin accepted the facts of adaptation, but advanced a scientific hypothesis to account for the facts. It may count as Darwin's greatest accomplishment that he brought the design aspects of nature into the realm of science. The wonderful designs of myriad plants and animals could now be explained as the result of natural laws manifested in natural processes, without recourse to an external Designer or Creator.

Biologists need to account for the functional features of organisms, their "design," in terms of the goals or purposes they serve, which is accomplished by means of teleological hypotheses or teleological explanations. Physical scientists do not face similar demands. Inanimate objects and processes (other than those created by humans) are not teleological because they are not directed toward specific ends; they do not exist to serve certain purposes. The configuration of sodium chloride depends on the structure of sodium and chlorine, but it makes no sense to say that that structure is made up so as to serve a certain end. Similarly, the slopes of a mountain are the result of certain geological processes and weather erosion, but did not come about so as to serve a certain end, such as skiing.

I have elsewhere (Ayala, 1968, 1970, 1999) proposed, as a working definition, that "teleological explanations are those that account for the existence of a certain feature in a system by demonstrating the feature's contribution to a specific property or state of the system, in such a way that this contribution is *the reason why* the feature or behavior *exists at all*." Teleological explanations require that the feature or behavior being explained contributes to the existence or maintenance of a certain state or property of the system. But, the essential component of a teleological explanation is that the feature or behavior could not have come about were it not for the particular end or purpose it serves. The end, goal, or purpose served is, therefore, the explanatory reason for the existence of the feature or behavior and its distinctive characteristics. This is also the case for human artifacts: they are produced so that they serve a certain purpose—a knife for cutting, a car for transportation.

Teleological explanations are appropriate and, indeed, indispensable in biology, because we cannot have a full understanding of what the eye is without knowing that it serves the purpose of seeing; that its configuration is designed precisely for the purpose of seeing; and that if it were not because they serve that purpose, eyes would never have come into existence. But teleological explanations are neither necessary nor appropriate in the physical sciences. Teleological explanations are fully compatible with causal accounts, but they cannot be reduced to causal explanations, without loss of explanatory value, because the design, and the function or purpose of the design, are required in order to achieve a full explanation of the eye and why it came to be. Thus, I have argued that the biological sciences are irreducible, even in principle, to the physical sciences, at least as evolutionary biology is concerned (Ayala, 1968).

The related concepts of design, purpose, and teleology, as I have expounded them, fall under the category of what Del Ratzsch (companion paper) calls $design_1$. But he adds: "A key question here is whether in biology . . . if one follows the causal, explanatory thread of seemingly designed phenomena in biology indefinitely back, at *some* point one really will come to ground on some agency and deliberate intention." My answer is: "definitely not." What Darwin saw and what modern population genetics

and other evolutionary disciplines have further accomplished is a scientific explanation of design in the living world. I do not mean, of course, that we do have at hand a full scientific account, with every detail in place, of the adaptations or designs of organisms, but rather that we need not leave the realm of scientific explanation, as it is pursued and accomplished in evolutionary biology, in order to provide a scientific account of biological design.

Is there room for an account of the design of organisms as "being a product *ultimately* of design" (Ratzsch, this volume)? There is room, in my view, but not for a *scientific* account. As I have expounded elsewhere (Ayala, 2007, 2008), people of faith may see the presence of God in the world in a variety of ways that include the marvelous diversity and exquisite design of the living world. Some science-savvy theologians, such as John F. Haught (1998, 2008), Arthur Peacocke (1998), and John Polkinghorne (1989), have developed an evolutionary theology of creation. As, for example, Haught (2008) has asserted, "[O]ne of the great gifts evolution brings to theology is that it renders the notion of continuous creation more meaningful perhaps than at any other time in this history of Christianity. In a still unfolding world that includes the evolution of life, creation can now be understood, more realistically than before, as happening every moment" (p. 174).

Two more points, briefly. One is that the reason to bring in our ignorance of the intentions of the designer as a reason to exclude ID from science is Behe's claim. Behe seeks to bypass the unholy implications that the dysfunctions, imperfections, cruelty, and even sadism that pervade the living world would have if organisms were intentionally designed by the Creator. "The argument from imperfection," writes Behe (1996), "overlooks the possibility that the designer might have multiple motives. . . . The reasons that a designer would or would not do anything are virtually impossible to know unless the designer tells you specifically what those reasons are" (p. 223). I would agree with Ratzsch that this is a cop-out.

In his Postscript, Ratzsch brings in the complexities of empirical testing of hypotheses. But, as he asserts, "A theory/hypothesis must indeed . . . at *some* point in *some* way, experience *some* telling empirical contact with the world, and thereby be at *some* genuine empirical risk." Alas, ID fails in this respect as well.

Notes

1 David Hume, in his posthumously published *Dialogues Concerning Natural Religion* (1779/2006), summarizes the argument from design as follows:

> Look round the world: Contemplate the whole and every part of it: You will find it to be nothing but one great machine, subdivided into an infinite number of lesser machines, which again admit of subdivisions, to a degree beyond what human senses and faculties can trace and explain. All these various machines, and even their most minute parts, are adjusted to each other with an accuracy, which ravishes into admiration all men, who have ever contemplated them. The curious adapting of means to ends, throughout all nature, resembles exactly, though it much exceeds, the productions of human contrivance; of human design, thought, wisdom, and intelligence. Since therefore the effects resemble each other, we are led to infer, by all the rules of analogy, that the causes also resemble; and that the Author of Nature is somewhat similar to the mind of man; though possessed of much larger faculties, proportioned to the grandeur of the work, which he has executed. By this argument *a posteriori*, and by this

> argument alone do we prove at once the existence of a Deity, and his similarity to human mind and intelligence. (p. 22)

2 A bilingual edition in English and Latin, in 60 volumes, has been published by Blackfriars & McGraw-Hill. *Existence and Nature of God* I, 2–11, is Vol. 2 (1964). The argument from design was formulated in the Middle Ages not only by Christian theologians, but also by Islamic and Jewish writers. For example, al-Kasim ibn Ibrahim (785–860) uses the analogy of a painter, among other analogies, to argue for the existence of a Creator: "[W]hoever sees a painting knows certainly that there is someone who painted it. . . . This is also [for] this wonderful creation that is seen; whoever considers and thinks, knows certainly that it has a creator" (Abrahamov, 1986, p. 261). The Spanish Jewish philosopher Moses Maimonides (1135–1204) argued that the universe gave evidence of design and rejected the possibility that it would have come about as the result of natural laws:

> [A]ll things in the Universe are the result of design. . . . How, then, can any reasonable person imagine that the position, magnitude, and number of the stars, or the various courses of their spheres, are purposeless or the result of chance? . . . [I]t is extremely improbable that these things should be the necessary result of natural laws, and not that of design. (Maimonides, 1956, pp. 184 and 188)

3 See, for example, Ayala (2007, Ch. 8). Federal Judge John E. Jones III in his 2005 *Dover* decision wrote: "[A]lthough proponents of IDM [intelligent design movement] occasionally suggest that the designer could be a space alien or a time-traveling cell biologist, no serious alternative to God as a designer has been proposed by members of the IDM." Further: Professor Behe's "testimony at trial indicated that ID is only a scientific, as opposed to a religious, project for him; however, considerable evidence was introduced to refute this claim. . . . ID's religious nature is evident because it involves a supernatural designer . . . expert witness ID proponents confirmed that the existence of a supernatural designer is a hallmark of ID" (*Kitzmiller* v. *Dover Area School District*, 2005, slip opinion, p. 18; pp. 25, 28–29).

4 In the seventeenth century and beyond, most chemists accepted that every combustible substance, such as wood or coal, was in part composed of phlogiston. Burning was thought to be caused by the liberation of phlogiston, with the remaining substance left as ash. Between 1770 and 1790, Antoine Lavoisier, the founder of modern chemistry, studied the gain or loss of weight when various substances were burned, and he demonstrated that phlogiston does not exist; eventually, he demonstrated that oxygen, which had been recently discovered by Joseph Priestley, was always involved in combustion and he went on to propose a general theory of oxidation (see Donovan, 1996).

5 These two episodes have recently been described, particularly by molecular biologists, as *discovery-based science* and *hypothesis-driven science*, respectively. Discovery-based science, at times, is associated with "big science" and the work of interdisciplinary research teams, whereas hypothesis-driven science is said to be "small science," primarily carried out by individual investigators. In different proportions or degrees, science always involves both, observation and testing.

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