Journal of Communication Disorders 57 (2015) 3–15

Contents lists available at ScienceDirect

Journal of Communication Disorders

Causal effects on child language development: A review of studies in communication sciences and disorders

Clare R. Rogers [a,*], Karissa L. Nulty [b], Mariana Aparicio Betancourt [a,c], Laura S. DeThorne [a]

[a] Department of Speech & Hearing Science, University of Illinois, 901 S. Sixth Street, Champaign, IL 61820, United States
[b] School of Molecular & Cellular Biology, University of Illinois, 393 Morrill Hall, 505 South Goodwin Avenue, Urbana, IL 61801, United States
[c] Neuroscience Program, Beckman Institute, 405 N. Mathews Avenue, Urbana, IL 61801, United States

ARTICLE INFO

Article history:
Received 4 November 2014
Received in revised form 1 May 2015
Accepted 29 June 2015
Available online 2 July 2015

Keywords:
Child language development
Causal factors
Environment
Genetics
Epigenetics
Developmental systems theory
Ecological developmental biology

ABSTRACT

We reviewed recent studies published across key journals within the field of communication sciences and disorders (CSD) to survey what causal influences on child language development were being considered. Specifically, we reviewed a total of 2921 abstracts published within the following journals between 2003 and 2013: Language, Speech, and Hearing Services in Schools (LSHSS); American Journal of Speech–Language Pathology (AJSLP); Journal of Speech, Language, and Hearing Research (JSLHR); Journal of Communication Disorders (JCD); and the International Journal of Language and Communication Disorders (IJLCD). Of the 346 eligible articles that addressed causal factors on child language development across the five journals, 11% were categorized as Genetic (37/346), 83% (287/346) were categorized as Environmental, and 6% (22/346) were categorized as Mixed. The bulk of studies addressing environmental influences focused on therapist intervention (154/296 = 52%), family/caregiver linguistic input (65/296 = 22%), or family/caregiver qualities (39/296 = 13%). A more in-depth review of all eligible studies published in 2013 ($n = 34$) revealed that family/caregiver qualities served as the most commonly controlled environmental factor (e.g., SES) and only 3 studies explicitly noted the possibility of gene–environment interplay. This review highlighted the need to expand the research base for the field of CSD to include a broader range of environmental influences on child language development (e.g., diet, toxin exposure, stress) and to consider more directly the complex and dynamic interplay between genetic and environmental effects.

Learning outcomes: : Readers will be able to highlight causal factors on child language development that have been studied over the past decade in CSD and recognize additional influences worthy of consideration. In addition, readers will become familiar with basic tenets of developmental systems theory, including the complex interplay between genetic and environmental factors that shapes child development.

* Corresponding author.
E-mail address: clarerogers93@gmail.com (C.R. Rogers).

http://dx.doi.org/10.1016/j.jcomdis.2015.06.004

1. Introduction

Given the key role of language in childhood development, and in the evolution of the human species as a whole, language has been studied by multiple disciplines for over a century. The field of communication sciences and disorders (CSD), itself a fairly young discipline that emerged in the 1900s, has drawn largely from fields such as linguistics and psychology for theories of language learning. Prominent theories found in introductory textbooks often include behaviorist accounts, which tend to emphasize the role played by the environment, primarily aspects of parent–child interaction (e.g., modeling, reinforcement, punishment, etc.), on child language development, and nativist/linguistic models, which tend to highlight innate predispositions to language learning (Gleason & Ratner, 2013; Hulit & Howard, 2006; Pence & Justice, 2012). Hirsh-Pasek and Golinkoff (1996) have referred to differences across such theories as ''Outside-In'' versus ''Inside-Out'' models of language development accordingly, depending on whether the emphasis tends to be outside or inside the child (i.e., environmental versus innate). Although there are additional theoretical foundations, such as cognitive and social-interactionist accounts that more directly acknowledge both intrinsic capacity and extrinsic influences on child language development, such theoretical models rarely emphasize environmental factors other than caregiver interactions and generally fail to emphasize the iterative dynamics of environmental and genetic influences over time (Evans, 2007).

Within the last decade, models in psychology and the emergent field of ecological developmental biology (eco-devo) have emphasized the complex interplay across genetic and environmental influences throughout the course of development, expanding concepts of the environment and challenging traditional delineations between genetic and environmental effects. Situated within the field of psychology, developmental systems theory (DST) has emerged as a particularly useful framework for highlighting the complex interplay across development (Griffiths & Tabery, 2013; Hood, Halpern, Greenberg, & Lerner, 2010; Robert, 2003; Sameroff, 2010). Proponents of DST have been critical of the traditional distinction between genetic and environmental effects that have been widely publicized through the nature versus nurture debate. In particular, DST highlights the essential reality that genes are not traits (Griffiths & Tabery, 2013). They live within cells, tissues, organs, and organisms – each of which provides environmental effects on gene expression. Specifically, the ability of environmental stimuli to selectively activate and silence specific genes through such processes as DNA methylation and histone modification is referred to as epigenetic effects (cf. Harper, 2010; Lam et al., 2012; Nestler, 2009). There is even evidence that some epigenetic markers are passed on to subsequent generations without alterations in the DNA sequence, broadening our concept of inheritance, and blurring traditional boundaries between genetic and environmental factors (Harper, 2010). The field of ecological developmental biology has emphasized the importance of such plasticity for development and survival, offering key examples such as the impact of maternal diet on body size in the agouti mouse and resource availability on the prevalence of cannibalism in the developing spadefood toad (Gilbert & Epel, 2009). Even the impact of teratogens, extrinsic agents such as drugs and chemicals associated with disruptions in embryonic development, are considered to be dependent on genotype. A review by Robinson, Fernald, and Clayton (2008) similarly highlighted the impact of social experience on gene expression in the brain; for example, the rate at which honeybees mature is influenced by the pheromones of older bees in the colony through inhibition of gene expression.

Although less substantiated within human research, a variety of environmental factors including social experiences, stress, diet, and toxin exposure have been associated with childhood developmental differences, often through specified epigenetic processes (Boyce, Sokolowski, & Robinson, 2012; Ellis & Garber, 2000; Evans, 2006; Hepp, 2011; Jaenisch & Bird, 2003; Oberlander et al., 2008; Smith, Kim, & Refsum, 2008; van Engeland et al., 2003). Additionally, epigenetic processes have been shown to influence early brain development and have been implicated in developmental disorders such as autism, Fragile X syndrome, Angelman syndrome, and Rett syndrome (Day & Sweatt, 2011; Fagiolini, Jensen, & Champagne, 2009; Gregory et al., 2009; Monteggia & Kavalali, 2009; Rodenhiser & Mann, 2006). In addition to recognized syndromes, the potential impact of epigenetic influences on language development and communication disorders more generally is just beginning to be recognized (e.g., Kraft & DeThorne, 2014; Rice, 2012).

Another emphasis of DST is the mutually coactive nature of causal influences over time. Thelen (2005) offers the illustrative metaphor of development as a mountain stream, highlighting that ''how a child behaves depends not only on the immediate current situation but also on his or her continuous short- and longer-term history of acting, the social situation, and the biological constraints he or she was born with. Every action has within it the traces of previous behavior. The child's behavior, in turn, sculpts his or her environment, creating new opportunities and constraints'' (pp. 259–260). Related concepts have been explored by Scarr and McCartney (1983) through delineation of specific mechanisms for gene–environment correlation: passive, active, and evocative (see also Plomin, DeFries, & Loehlin, 1977). Passive gene–environment correlation highlights that shared genes between child and parent likely shape the environments children are raised in (e.g., parents and children who share a genetic proclivity to language are likely to co-create a language-rich environment). Evocative gene–environment correlation (aka, 'child effects') suggests that children's genetic proclivities elicit differences in the environments they are raised in (e.g., children with genetic advantages in language learning are likely to be placed in more demanding educational environments). And finally, active gene–environment correlation underscores the tendency for children to seek out environments that are consistent with their own genetic proclivities (e.g., children who are predisposed to language learning are more apt to seek out opportunities to utilize those skills). As an illustrative example of potential evocative gene–environment correlation, DeThorne and Channell (2007) examined the language use of one examiner in her interactions with

29 different children with varying levels of language ability. Even though the examiner was naïve to the research purpose at the time the study was conducted, the linguistic complexity of her utterances correlated positively with the complexity of the children's productions, suggestive of child effects (see also Cramblit & Siegel, 1977; Paul & Elwood, 1991; Rescorla & Ratner, 1996). In addition, a behavioral genetic design by Dale, Tosto, Hayiou-Thomas, and Plomin (this issue) including over 8000 twin pairs demonstrated shared genetic influences on a self-reported measure of parental language stimulation and indices of child language development, thereby supporting the presence of gene–environment correlation.

A final complexity we will highlight from developmental frameworks offered by DST and the field of eco-devo is that phenotypic variations often confer adaptive advantages for the contexts in which they emerge (Gottlieb, 2010). One example in which phenotypic variations appear to confer advantage within the contexts in which they emerge comes from work by Meaney (2010) in relation to parenting practices and the development of anxiety-related behavior in rodent pups. Stressful environments lead to less maternal nurturing, which in turn leads to stable changes in the gene expression underlying the pups' behavioral and neuroendocrine responses to stress. Pups who have received more maternal licking and grooming tend to show a reduced stress response. The key component of this for our current point is that either parenting style (high or low nurturing) could be viewed as conferring adaptive value for the conditions in which they evolved. Although a heightened stress response creates a physiology burden on well-being, it can confer an adaptive advantage in dangerous environments. In essence, such a mechanism may provide an adaptive function for parental effects in preparing offspring for the environmental demands they are born into (see Meaney, p. 64). Such work may offer insight into differences in parenting practices associated with lower-income communities with higher rates of violence and stress (e.g., Hoff, Laursen, & Tardif, 2002; Kotchick & Forehand, 2002).

Given the functional significance and contextual variability of human language, it seems a likely candidate for complex causal mechanisms, including epigenetic influences. Consider the immense variability in children's developmental contexts, including cultural and individual differences in geographic region, diet, parenting practices, educational opportunities, number of languages, dialect, and family structure. Children learn language successfully under a vast diversity of conditions, and evolutionary pressures are likely to favor plasticity in the development of such a critical and adaptive ability. Of course, such developmental plasticity may lead to a disadvantageous trait when environments change after the period of adaptability is past (cf. Boyce & Ellis, 2005; Gluckman, Hanson, Spencer, & Bateson, 2005). The developmental windows associated with phenotypic plasticity are consistent with the concept of critical periods that have been discussed in relation to language learning in young children and may help explain why it can be more difficult to learn a second language after adolescence (Johnson & Newport, 1989; Newport & Supalla, 1987; Weikum, Oberlander, Hensch, & Werker, 2012).

Examples of developmental plasticity highlight the oversimplicity of valuing one genotypic or environmental influence over another (e.g., more or less parental nurturing) without consideration of the context in which individuals are expected to thrive, such as different cultural-linguistic communities. Accordingly, increased attention is being paid to the potential adaptive developmental responses to such factors as stress, heat, malnutrition, and early microbe exposure on child development (e.g., Gluckman et al., 2005; McDade, 2012; McEwen, 2012; Zeisel, 2009). On a related note, the same environmental stimuli can have substantially different effects depending on the allelic variation of the individual, a concept referred to as gene–environment interaction (GxE; cf. Tabery & Griffiths, 2010). Consequently, differences in one's genotype can moderate the effects of environmental influences on endophenotypes and ultimately on phenotypic expression. Gene–environment interaction has been studied in a wide range of human domains, with allelic variation found to moderate relationships between such variables as (a) childhood maltreatment and onset of major depression, (b) adolescent cannabis use and development of psychosis, and (c) alcohol consumption and addictive cravings (Caspi & Moffitt, 2006). In another example by Caspi et al. (2007), IQ advantages were associated with early breastfeeding but only for children who carried a specified allelic variation in the *FADS2* gene. For children with different alleles of the *FADS2* gene, breastfeeding appeared to confer neither advantage nor disadvantage. On the general topic of GxE, some investigators have hypothesized that children with certain genotypes may be particularly susceptible or sensitive to environmental influences, thereby suggesting that what children inherit is differences in their developmental plasticity. Such plasticity may offer a potential risk and/or protective factor depending on the circumstances they are reared in (Belsky & Pluess, 2009; Boyce & Ellis, 2005). In sum, the adaptive value of phenotypic variation, environmental factors, and genotypic variations are all dependent on contextual factors.

Together, the developmental systems theory and field of eco-devo offer three key tenants for the study of childhood development that may have particular relevance for CSD. First, environmental effects are construed more broadly than caregiver interactions to include such factors as diet, toxins, and stress. Second, the boundary between genetic and environmental effects is illusive as genes and environmental influences work interactively over time beginning before birth (e.g., Rosenblatt, 2010). Finally, context matters, and the benefit of any particular trait, or the genetic and environmental influences that impinge upon it, cannot be considered in isolation. Despite the prominence of such tenets in other literatures, it is unclear to what extent such complexity has permeated the study of child language development in CSD. Consequently, the present review addressed the following explicit questions:

1. What causal influences on child language development have been explicitly studied within key CSD literature over the last 11 years?

2. What causal influences are being controlled for and discussed within CSD journal articles from 2013 and to what extent is the possibility of gene–environment interplay being acknowledged?

2. Methods

In order to capture a breadth of recent work within the field of CSD, our review initially focused on journals published between 2003 and 2013 by the American Speech–Language–Hearing Association and then expanded to include two journals published outside the United States that were selected for review based on investigator familiarity and higher impact factors. In total, we focused on five journals to represent the child language literature in CSD: American Journal of Speech–Language Pathology (AJSLP); Journal of Speech, Language, and Hearing Research (JSLHR); Language, Speech, and Hearing Services in Schools (LSHSS); International Journal of Language and Communication Disorders (IJLCD); and Journal of Communication Disorders (JCD). The review focused on all 2921 abstracts published in these five journals between 2003 and 2013.

To address of our first research question, abstracts were reviewed by our team of four investigators, which together offered representation from speech and hearing science, neuroscience, and molecular and cellular biology. Abstracts were considered as eligible studies if they examined the influence of genetic or environmental influences on children's intrinsic language proficiency. Consistent with classic definitions in the field (Nippold, 2000; Paul, 2007; Pence & Justice, 2012), child language was operationalized as a measure of expressive and/or receptive language, across spoken and written modalities, across domains of semantics, pragmatics, phonology, and morphosyntax in children under 18 years of age.[1] Causal effects were conceptualized based on etiological influences, genetic and environmental, as noted in levels of causation in Bishop and Snowling (Bishop & Snowling, 2004, Fig. 2, p. 859). Environmental effects were defined as *extrinsic* to the child, including such influences as linguistic input, parental education, toxin exposure, nutrition, injury, and illness (cf., Conti-Ramsden, 1985; Hepp, 2011; Paul, 2007; Plomin & Bergeman, 1991; Scarr & McCartney, 1983; Stromswold, 2006; Thelen, 2005). The team excluded studies that examined the influence of children's intrinsic psychological constructs (e.g. working memory) on their own language development given that such intrinsic qualities are themselves presumably shaped by a cascade of genetic and environmental effects. Similarly, studies that described the language characteristics or differences related to a child's demographic characteristics (e.g., race, dialect) were also excluded from this review; however, the influence of broader family demographic information (e.g., SES, parent education) were included as relevant environmental variables. We also excluded studies that focused on group comparisons between children with and without language disorders of unspecified origin (e.g., SLI) given that etiology was not a focus of such investigations; however, if a study examined language outcomes in a participant group that was selected based on identified genetic differences (e.g., Down syndrome), it was deemed eligible as a study of genetic effects. Finally, studies of interventions on child language development, including reviews and meta-analyses, were generally included unless the study failed to provide adequate detail regarding the nature of the intervention, and duplications of the same study were excluded (e.g., one abstract from AJSLP was published in both English and Spanish).

Once identified as an eligible study that addressed causal effects on child language, the studies were delineated into the following categories: Genetic, Environmental, or Mixed, with Mixed representing a combination of genetic and environmental effects. The Mixed category included behavioral genetic studies given that such designs are intended to reflect both genetic and environmental influences (DeThorne & Hart, 2009; Stromswold, 1998). Ambiguous variables, such as family history, were not coded as genetic or environmental unless explicitly interpreted as such by the authors (e.g., Zubrick, Taylor, Rice, & Slegers, 2007). Given our particular interest in the breadth of environmental factors, the studies from both the Environmental and Mixed categories were further delineated into the types of environmental factors they examined. Emergent factors included (a) Therapist intervention, (b) Family/caregiver linguistic input, (c) Family/caregiver qualities, (d) other environmental effects, and (e) Multiple environmental (i.e. the studies that focused on two or more of the noted subcategories). Note that behavioral genetic studies from the Mixed category were not included in this analysis because they did not study explicit environmental factors that could be classified accordingly.

Fig. 1 provides an overview of the entire coding/categorization process. Development of the coding guidelines was an iterative process that began with a review of relevant literature on causal influences and the investigative team's joint review of abstracts from at least two journal issues. After initial coding guidelines were established, abstracts were reviewed by individual team members, and questions that arose during the process were discussed during weekly meetings. In cases where the abstract appeared unclear for the specified criteria, investigators referred to relevant sections of the article as a whole, and cases of ambiguity were always resolved via consensus amongst the investigators. Each time coding guidelines were revised, prior coding would be reviewed again and adjusted accordingly based on the revisions. The first author completed a second pass on all abstracts to ensure consistent application of the coding criteria.

To explicitly address our second research question, our team also completed a more in-depth review of all the eligible studies from the latest year, 2013. Specifically, the intent was to consider what causal influences were being considered across the entire article (i.e., methods, results, discussion) that would supplement our understanding of causal influences being studied as explicit independent/predictor variables. To this end, at least two members from the investigative team

[1] In the case of JSLHR where articles were pre-categorized within speech, language, and hearing sections, the abstracts in the sections of speech and hearing were automatically considered as ineligible.

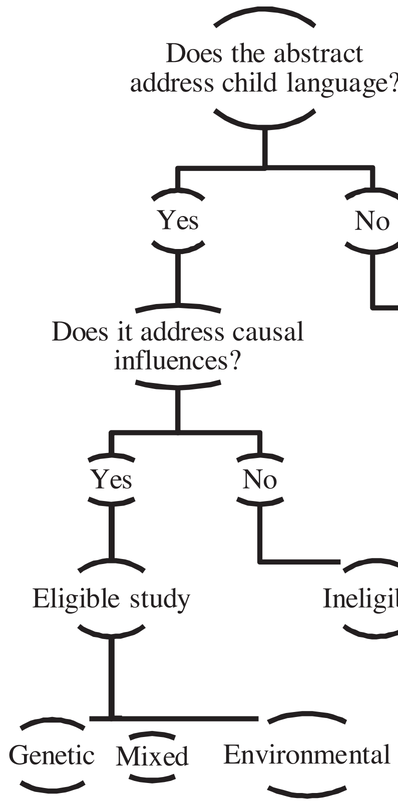

Fig. 1. Overview of the review process for the 2921 abstracts published in 2003–2013 across select journals in communication sciences and disorders.

independently read the full length of each of the 34 eligible studies from 2013, specifically noting any genetic or environmental influences that were (a) explicitly controlled for in the design or the analyses, including variables that were used to match experimental groups, or (b) were speculated about in the methods, results, or discussion sections. In addition, we specifically noted any studies that made explicit mention of genetic and environmental influences working in concert on child language development. Results were reviewed by the investigative team and discrepancies between reviewers were discussed and resolved by consensus.

3. Results

3.1. Review of published abstracts from 2003 to 2013

Table 1 provides a summary of our abstract review, delineated by journal. Of the 2921 abstracts reviewed, approximately 12% (346) addressed causal influences on child language development with the specific percentage varying across journals from 8% in JCD to 20% in LSHSS. The 346 total articles addressing causal factors were further delineated as follows: 11% (37/346) were categorized as Genetic, 83% (287/346) as Environmental, and the remaining 6% (22/346) as Mixed. Across the Genetic and Mixed categories, studies of genetic factors across all journals included 13 behavioral genetic studies; 4 family aggregation/family history studies; and 42 studies focused on characteristics affiliated with specified genetic conditions such as FOXP2 translocation, Down syndrome, Prader–Willi syndrome, and 22q11 deletion syndrome. Within the Mixed category, in addition to the 13 behavioral genetic studies that inherently examined the extent of both genetic and environmental effects, nine designated genetic studies explicitly examined environmental effects as well. As an example of studies in the Mixed category other than behavioral genetic designs, Bird et al. (2005) in AJSLP examined the potential impact of bilingual input on language development in children with Down syndrome, focusing on participants with a known genetic difference paired with the study of familial linguistic input (i.e., an environmental variable).

Finally, 83% (287/346) of all the eligible studies qualified as Environmental, with a range of 70–96% across individual journals. Given the focus of this special issue, particular attention was given to the nature of the environmental factors under examination, both from the studies in the Environmental category ($n = 288$) and the studies in the Mixed category that studied both genetic and explicit environmental variables ($n = 8$).[2] Emergent factors included therapist interventions, family/

[2] Behavioral genetic studies ($n = 13$) were excluded from this level of analysis because they did not examine specified environmental factors.

Table 1
Summary of results from the review of published abstracts as delineated by journal.

	Total abstracts	Eligible abstracts[a]	# Eligible abstract in each category ($n = 346$)		
			Genetic	Environmental	Mixed
AJSLP	380	56 (15%)	5 (9%)	50 (89%)	1 (2%)
JSLHR	1277	110 (9%)	6 (5%)	90 (82%)	14 (13%)
LSHSS	377	77 (20%)	1 (1%)	73 (95%)	3 (4%)
JCD	393	33 (8%)	8 (24%)	23 (70%)	2 (6%)
IJLCD	494	70 (14%)	17 (24%)	51 (73%)	2 (3%)
Total	2921	346 (12%)	37 (11%)	287 (83%)	22 (6%)

Note: AJSLP = American Journal of Speech–Language Pathology; JSLHR = Journal of Speech, Language, and Hearing Research; LSHSS = Language, Speech, and Hearing Services in Schools; JCD = Journal of Communication Disorders; IJLCD = International Journal of Language and Communication Disorders.
[a] Eligible abstracts refer to those that addressed causal factors on children's language development.

caregiver linguistic input, family/caregiver qualities, other environmental, and multiple environmental; results for this analysis are summarized by journal in Table 2 and collectively in Fig. 2. Over half (154/296) of the studies of environmental factors qualified under therapist intervention. Example intervention strategies included slowed speech rate, increased frequency of talk (either in general or of specific forms), enhanced linguistic complexity of input, and enhanced multimodality (e.g., access to AAC). An additional 22% (65/296) focused directly on differences in family/caregiver linguistic input, most often from a parent but sometimes a teacher. Differences in linguistic input were operationalized largely through differences in frequency of talk, either in general or of specific forms; use of elaborations; and responsivity to the child. An additional 13% (39/296) of studies focused on Family/caregiver qualities, as opposed to their behaviors; such qualities included socioeconomic status, culture, and family placement (e.g., international adoption, institution, foster care). A rather small 5% (16/296) of causal studies focused on factors outside of therapist intervention and family/caregiver linguistic input, and family/caregiver qualities; such studies are delineated in Fig. 2 as Other environmental. Finally, 7% (22/296) of studies represented environmental elements from more than one category (e.g., therapist intervention and caregiver linguistic input), thereby delineated as 'Multiple environmental' in Fig. 2. Given our particular interest in Other environmental influences, we summarized the relevant variables from all studies across Environmental and Mixed categories that considered Other environmental influences in Table 3. There were a total of 23 such studies (1 from Mixed + 16 from Environmental – Other + 6 Environmental – Multiple) with variables including prenatal drug exposure, ambient noise level, pre-term birth, traumatic brain injury (TBI), assessment method, geographic location, human immunodeficiency virus (HIV), and perceptual load.

Table 2
Frequency of environmental factors studied between 2003 and 2013 as delineated by journal ($n = 296$; from both the Environmental and the Mixed categories).[a]

	Therapist interventions	Family/caregiver linguistic input	Family/caregiver qualities	Other environmental variables	Multiple environmental
AJSLP	34 (67%)	8 (16%)	4 (8%)	1 (2%)	4 (8%)
JSLHR	42 (45%)	17 (18%)	23 (24%)	6 (6%)	6 (6%)
LSHSS	37 (49%)	23 (31%)	6 (8%)	3 (4%)	6 (8%)
JCD	14 (58%)	4 (17%)	0 (0%)	4 (17%)	2 (8%)
IJLCD	27 (52%)	13 (25%)	6 (12%)	2 (4%)	4 (8%)
Total	154 (52%)	65 (22%)	39 (13%)	16 (5%)	22 (7%)

Note: AJSLP = American Journal of Speech–Language Pathology; JSLHR = Journal of Speech, Language, and Hearing Research; LSHSS = Language, Speech, and Hearing Services in Schools; JCD = Journal of Communication Disorders; IJLCD = International Journal of Language and Communication Disorders.
[a] Excludes behavioral genetic studies from the Mixed category that do not study specified environmental variables.

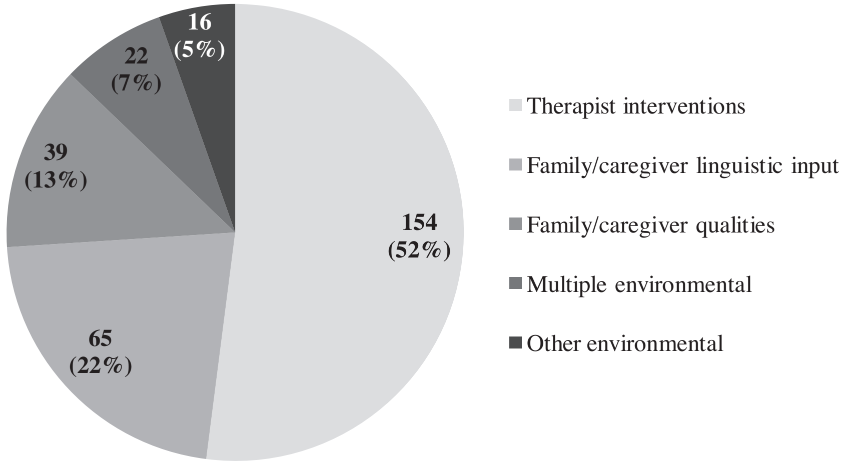

Fig. 2. Frequency of studies explicitly examining environmental influences on child language across select journals delineated by environmental factor (total $n = 296$, taken across both the Environmental and Mixed categories). Excludes behavioral genetic studies from the Mixed category that do not study specified environmental variables.

3.2. In-depth review of eligible studies from 2013

Related to our second question regarding what causal influences were being controlled for within journal articles from 2013, the results of the in-depth review of the 34 eligible studies published in 2013 are presented in Table 4. Given that all the control variables within the methods were related to environmental, rather than genetic influences, we categorized them according to the emergent factors from our abstract review (e.g., family/caregiver linguistic input, therapist interventions). Similar to the review of the independent variables, family/caregiver qualities emerged as a common control variable, most frequently through the construct of SES (8/34 eligible studies), but also as measured through parental demographics/intrinsic qualities, family background, and socio-demographic characteristics of the child. The nine studies that controlled Other environmental variables were most heavily weighted toward considering the influence of activity on child performance (5/34), with other variables being classroom placement, family placement, and drug exposure. The remaining controlled environmental variables related to aspects of the therapist intervention (5/34) and family/caregiver linguistic input (3/34).

Table 3
List of independent/predictor variables from studies that explicitly studied ‘other’ environmental factors from select journals 2003–2013.

Independent variable	Example research questions	Specified studies
Prenatal drug exposure	- How does fetal alcohol spectrum disorder impact language performance and social communication? - What are the long-term effects of prenatal cocaine exposure on the language development of 12-year-old children?	Coggins, Timler, and Olswang (2007)[a], Cone-Wesson (2005), Henry, Sloane, and Black-Pond (2007)[a], Hyter (2007)[a], Kjellmer and Olswang (2013), Lewis et al. (2013)[a], Olswang, Svensson, and Astley (2010); Rogers-Adkinson and Stuart (2007)[a]
Ambient noise level	- Does altering classroom noise levels influence attention and speech perception? - Does ambient frequency predict consonant development?	Alston and James-Roberts (2005)[a], Bradlow, Kraus, and Hayes (2003), Nelson, Kohnert, Sabur, and Shaw (2005), Riley and McGregor (2012), Stokes and Surendran (2005)
Pre-term birth	- What is the effect of pre-term birth on linguistic competencies and phonological working memory at preschool age? - Are there differences in oral narrative skills between children born pre-term compared with their full-term born peers?	Cattani et al. (2010), Crosbie, Holm, Wandschneider, and Hemsley (2011), Sansavini et al. (2007), Zubrick et al. (2007)[b]
Pediatric traumatic brain injury	- What is the effect of TBI on a child’s linguistic development?	Adams-Chapman (2009), Hay and Moran (2005)
Assessment method	- Does the assessment method impact estimates of prevalence of language impairments in families?	Conti-Ramsden, Simkin, and Pickles (2006)
Geographical location	- Do the types and frequency of conversational repairs used by African American children vary by geographic location?	Stockman, Karasinski, and Guillory (2008)
Human immunodeficiency virus	- How do the different stages of HIV disease impact communication?	McNeilly (2005)
Perceptual load	- Does the level of perceptual load influence ease of semantic access in younger and older children?	Jerger et al. (2013)

[a] Indicates studies from the Environmental category coded as studying ‘Multiple’ environmental factors.
[b] Indicates studies in the Mixed category that included ‘Other’ environmental factors as variables of interest.

Table 4
Summary of causal factors controlled through experimental design and analyses across the eligible studies published in 2013.

Category of controlled causal factors	Specified variables	# Eligible studies	Specified studies
Family/caregiver qualities (*n* = 10)[a]	SES (e.g. school, location, parental education, free/reduced lunch.	8	Carson, Gillon, and Boustead (2013), Haebig, McDuffie, and Weismer (2013), Van Herwegen, Dimitriou, and Rundblad (2013), Martin, Losh, Estigarribia, Sideris, and Roberts (2013), Restrepo, Morgan, and Thompson (2013), Smith-Lock, Leitao, Lambert, and Nickels (2013), Tadić, Pring, and Dale (2013), Thordardottir and Brandeker (2013)
	Caregiver demographics/intrinsic qualities (parental stress; maternal marital status, age, number of births, vocabulary, IQ)	2	Fey, Yoder, Warren, and Bredin-Oja (2013), Lewis et al. (2013)
	Family background (number of single parent in each group)	1	Van Herwegen et al. (2013)
	Socio-demographic characteristics of children (birth order, number of siblings)	1	Tadić et al. (2013)
Other (*n* = 9)[a]	Activity (task, materials, goals, stimuli)	5	Eaton and Ratner (2013), Fey et al. (2013), Mira and Schwanenflugel (2013), Smith-Lock et al. (2013), Storkel, Maekawa, and Aschenbrenner (2013)
	Classroom placement (mainstream, class size, access to professional development and administration procedures)	3	Baxendale et al. (2013), Kjellmer and Olswang (2013), Smith-Lock et al. (2013)
	Family placement (biological vs. adoptive care)	1	Lewis et al. (2013)
	Drug exposure (cigarette, alcohol, marijuana use)	1	Lewis et al. (2013)
Therapist intervention (*n* = 5)	Intervention details (type, dosage, received or not)	3	Bunta and Douglas (2013), Kaiser and Roberts (2013), Sepúlveda, López-Villaseñor, and Heinze (2013)
	Clinician/examiner linguistic Input	2	Baxendale et al. (2013), Eaton and Ratner (2013)
Family/caregiver linguistic input (*n* = 3)	Parent–child interaction and linguistic practices (e.g. amount of exposure to French over lifetime, age of onset of bilingualism, maternal verbosity)	3	Fey et al. (2013), Tadić et al. (2013), Thordardottir and Brandeker (2013)

[a] Indicates the number of studies that examined such factors, counting only once studies that included more than one specified variable within a category of controlled causal factors.

Also related to question two, the nature of causal influences mentioned in the discussion was largely similar in nature to the variables already discussed with some notable additions. Specifically, some authors speculated about the potential influence of such factors as the communicative practices of the clinician (e.g., King, Hengst, & DeThorne, 2013, pp.208–209), peer input/responsiveness (Brady, Thiemann-Bourque, Fleming, & Matthews, 2013, p.1607), expectations of others (e.g., Dimitropoulos, Ferranti, & Lemler, 2013, pp.199–200), and genetic variation in the child (e.g., Dimitropoulos et al., 2013, pp.199–200). In addition, only 9% of the 2013 eligible studies (3/34) speculated about the potential interplay of both genetic and environmental effects. Specifically, Martin, Losh, Estigarribia, Sideris, and Roberts. (2013, p.335) noted that preferences for communication modality may differ based on child genotype; Lewis et al. (2013, p.1673) explicitly noted the influence of both genetic and environmental influences on children's verbal skills; and Dimitropoulos et al. (2013, pp.199–200) noted the potential impact of genetic subtype on the language abilities of children with Prader–Willi syndrome.

4. Discussion

The current review examined the causal influences of child language development as represented within five key journals within the field of Communication Sciences and Disorders. Of the 346 studies that addressed causal influences on child language development, the vast majority (83%) were categorized as Environmental, with only 17% directly examining some genetic effects, either in isolation or in concert with environmental factors. Of the 296 studies across both the Environmental and Mixed categories that explicitly examined specific environmental factors, most (87%, 258/296) focused on Therapist interventions, Family/caregiver linguistic input, and Family/caregiver qualities. Only 8% (23/296) addressed Other environmental variables such as prenatal drug exposure, ambient noise level, pre-term birth, traumatic brain injury (TBI), geographic location, and human immunodeficiency virus (HIV). Similarly, a more in-depth review of the eligible 2013 studies revealed that family/caregiver qualities served as the most commonly controlled environmental factor and only 3 studies explicitly noted the possibility of gene–environment interplay. The discussion that follows will focus on how key findings aligned with prominent theoretical frameworks in CSD and implications for future directions in the field.

In general, results from the present review aligned with prominent theoretical models of language development in the field of communication sciences and disorders in two key ways. First, the emphasis on environmental contributions, particularly therapist interventions and linguistic input from caregivers, suggested a current prominence of Outside-In models of child language development within the field. Differences in linguistic input were operationalized largely through differences in frequency of talk, either in general or of specific forms, the linguistic complexity of the input (e.g., grammatical vs. telegraphic), and the modality of presentation (e.g., access to AAC). Such findings are consistent with the current scope of practice for speech–language pathologists, which includes behavioral interventions to decrease disruptive behaviors and to increase communicative opportunities in a child's environment (cf. ASHA, 2007). Despite this focus, there was little explicit examination of how environmental differences across children may be correlated with their genetic differences, such as how children's inherent language proclivities may shape the interactions they have with caregivers (i.e., 'child effects'), although a few studies acknowledged this in their discussions (e.g., Iverson, Longobardi, Spampinato, & Caselli, 2006; Kjellmer & Olswang, 2013). In addition, there was little direct consideration of how differences in parenting practices might be linked to important socio-cultural variables such as race and ethnicity, which in turn may be linked to important differences in stress, nutrition, and toxin exposure.

Second, the reviewed literature mirrored prominent theoretical models in CSD in the tendency to focus primarily on genetic versus environmental effects, rather than emphasizing the complex interplay of such influences over time. Specifically, only 6% of the (22/346) eligible studies emerged as explicit studies of both genetic and environmental effects, with 13 of those being behavioral genetic studies which by design focus on partitioning genetic and environmental effects rather than addressing complex bidirectional influences (cf. Plomin, DeFries, McClearn, & McGuffin, 2008; Tabery & Griffiths, 2010). With that said, of the 59 studies examining genetic causal factors, 5 studies speculated about the relationship between more than one level of development, i.e. between genes and molecular or cellular levels (DeThorne, Petrill, Hayiou-Thomas, & Plomin, 2005; Persson et al., 2006), and between innate (heritable) language skills and environmental exposure (DeThorne et al., 2008; van Bysterveldt, Westerveld, Gillon, & Foster-Cohen, 2012; Zubrick et al., 2007). Similarly, of 34 studies from 2013 that were reviewed in-depth, 9% (3/34) noted the possibility of genetic and environmental influences working in concert.

Considering results from this review, we recommend that CSD's theoretical framework be shifted in order to embrace broader concepts of the environment and to acknowledge the complex interplay of genetic and environmental influences within development. Questions regarding how language development takes shape are not rhetorical. How we conceptualize causality influences the interventions we provide as a practice-based field. For example, a relatively exclusive focus on linguistic input implies to therapists and parents that they should be able to prevent and remediate language disorders if they only provide the "right" type and amount of linguistic input, when the reality is much more complex. It is possible that controlling for demographic factors, such as SES, is currently the field's mechanism for acknowledging broader environmental influences on child language development. However, if so, the rationale remains largely unarticulated, and still does not address the fact that other factors merit direct examination. Specific to broadening our concept of environmental influences, research across other fields has highlighted the importance of such influences as diet, stress, and toxin exposure. Although not within the journals reviewed here, relevant studies are published elsewhere on these influences, such as links between autism and exposure to air pollution and pesticides (Roberts et al., 2013; Shelton, Hertz-Picciotto, & Pessah, 2012).

In addition, consideration of other environmental factors, such as nutrition, may be more prevalent for other types of communication disorders such as stuttering (e.g., Mahurin-Smith & Ambrose, 2013).

In addition to expanding our concept of the environment, the second recommendation is the need to recognize the complex interplay of genetic and environmental effects. Few studies from our review mentioned the possibility of gene–environment correlation or interaction, and none explicitly studied it. Given the emerging evidence of such complexities in other disciplines (e.g., Boyce et al., 2012; Ellis & Garber, 2000; Evans, 2006; Hepp, 2011; Jaenisch & Bird, 2003; Oberlander et al., 2008; Smith et al., 2008; van Engeland et al., 2003), it is important to consider the likelihood that the effects of any environmental variable are not going to be consistent, in nature or magnitude, across all individuals due to the complex interplay across multiple levels of developmental systems, from genes to sociocultural communities. It is quite possible that the same environmental influence might be beneficial to some and harmful to others. See for example the work by Dotterer, Iruka, and Pungello (2012) that documents differential associations between maternal interaction style and child outcomes dependent on race. Neither genes nor individual environmental factors operate in a vacuum. In fact, findings of epigenetic effects blur the traditional distinction between the two, and gene–environment interactions highlight the reality that environmental variables can have very different effects depending on one's genotype. Even though relevant findings are sparse in relation to child development, particularly *replicated* effects, it is naïve to believe that the complexities of epigenetic effects and GxE interaction do not shape the intricate evolutionary phenomenon of child language development. In fact, Halpern (2010) claims that such complexity in development is what makes replication of reductionist experimental paradigms so difficult, if not impossible. Such complexity highlights the value in studying individual developmental trajectories, preferably through a variety of scientific paradigms. At the same time that molecular genetic paradigms are providing increasingly sophisticated means to examine epigenetic effects (Kraft & DeThorne, 2014), longitudinal case studies offer an opportunity to document the emergence and development of behavior "in the wild," including the multitude of factors likely to impinge upon it. At the very least, both quantitative and qualitative studies should be moving toward the inclusion of more contextual information in the study of child language development, incorporating information across levels from individual biology to social–cultural community (see for example Baxendale, Lockton, Adams, & Gaile, 2013 or Zubrick et al., 2007).

Despite the contribution of the present review, it is important to keep in mind that we focused on a subset of prominent journals within CSD that may not be fully representative of the field as a whole. In addition, the review of studies from 2003 to 2012 focused primarily on abstract reviews with full consideration of the article only as deemed necessary. Given that a study's purpose and key variables are typically critical elements of the abstract, we considered this relatively superficial analysis adequate for our purpose. In addition, the results from our abstract review were supported by the in-depth review of all eligible articles from 2013, which served as a means to confirm our categorization of the abstracts and to examine causal factors from the perspective of methodological/statistical control variables within the analyses and interpretation within the discussion.

5. Conclusions

In sum, the current review highlights the need to broaden the research base for the field of Communication Sciences & Disorders both nationally and internationally. Although it makes sense that journals within CSD tend to focus on causal influences most directly linked to current scope of practice within speech–language pathology (cf. ASHA, 2007), we recommend a bidirectional relationship between research and practice. How we conceptualize causality shapes clinical interventions and interventions shape our scientific understanding. Focusing on linguistic input at the expense of other factors is likely to restrict the positive impact our field can have in understanding child development and associated treatments and interventions. Future research should include a broader range of causal influences on child language development, including such factors as diet, toxins, and stress. In addition, an integrative approach sensitive to the complex and dynamic interplay between genetic and environmental influences over time that encompasses multiple levels of development (e.g. molecular, cellular, cultural and historical) is warranted.

Acknowledgements

We would like to specifically thank Kathy Dejmek and Paulina Mitra for their assistance with this project. Results were presented in part at the Undergraduate Research Symposium in 2014 at the University of Illinois.

Appendix A. Continuing education questions

1. Traditionally, theories in communication sciences and disorders have emphasized which of the following environmental influences on child language development?
   a. Parent interaction
   b. Prenatal nutrition
   c. Exposure to toxins
   d. Infection
2. Together, developmental systems theory and the field of ecological developmental biology emphasize which of the following points?
   a. The complex interplay between genetic and environmental factors over time
   b. The value of a given phenotype depends on context
   c. Environment is construed broadly from cellular contents to sociocultural communities
   d. All the above
3. Which of the following statements is true of epigenetic effects?
   a. The effects are limited to a single generation
   b. Such effects have been demonstrated only in nonhuman species
   c. They involve changes in an organism's DNA sequence
   d. They are defined as environmental influences on gene expression
4. Of the journal abstracts reviewed by the authors over the last decade, the majority of causal studies directly addressed genetic effects on child language development.
   a. True
   b. False
5. The authors propose a shift in the field of CSD's theoretical framework to:
   e. Outside-in and inside-out models of language development
   f. A cognitive and social-interactionist framework of development
   g. A dynamic multilevel framework of development
   h. A nature–nurture developmental model

References

Adams-Chapman, I. (2009). Insults to the developing brain and impact on neurodevelopmental outcome. *Journal of Communication Disorders, 42*(4), 256–262. http://dx.doi.org/10.1016/j.jcomdis.2009.03.010

Alston, E., & James-Roberts, I. S. (2005). Home environments of 10-month-old infants selected by the WILSTAAR screen for pre-language difficulties. *International Journal of Language & Communication Disorders, 40*(2), 123–136. http://dx.doi.org/10.1080/13682820400006861
American Speech–Language–Hearing Association (2007). *Scope of practice in speech–language pathology [Scope of Practice]* http://dx.doi.org/10.1044/policy.SP2007-00283 Retrieved from www.asha.org/policy
Baxendale, J., Lockton, E., Adams, C., & Gaile, J. (2013). Parent and teacher perceptions of participation and outcomes in an intensive communication intervention for children with pragmatic language impairment. *International Journal of Language & Communication Disorders, 48*(1), 41–53. http://dx.doi.org/10.1111/j.1460-6984.2012.00202.x
Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885. http://dx.doi.org/10.1037/a0017376
Bird, E. K. R., Cleave, P., Trudeau, N., Thordardottir, E., Sutton, A., & Thorpe, A. (2005). The language abilities of bilingual children with Down syndrome. *American Journal of Speech–Language Pathology, 14*(3), 187–199. http://dx.doi.org/10.1044/1058-0360(2005/019)
Bishop, D. V., & Snowling, M. J. (2004). Developmental dyslexia and specific language impairment: Same or different? *Psychological Bulletin, 130*(6), 858. http://dx.doi.org/10.1037/0033-2909.130.6.858
Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context. I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17*(02), 271–301. http://dx.doi.org/10.1017/S0954579405050145
Boyce, W. T., Sokolowski, M. B., & Robinson, G. E. (2012). Toward a new biology of social adversity. *Proceedings of the National Academy of Sciences of the United States of America, 109*(Suppl. 2), 17143–17148. http://dx.doi.org/10.1073/pnas.1121264109
Bradlow, A. R., Kraus, N., & Hayes, E. (2003). Speaking clearly for children with learning disabilities sentence perception in noise. *Journal of Speech, Language, and Hearing Research, 46*(1), 80–97. http://dx.doi.org/10.1044/1092-4388(2003/007)
Brady, N. C., Thiemann-Bourque, K., Fleming, K., & Matthews, K. (2013). Predicting language outcomes for children learning augmentative and alternative communication: Child and environmental factors. *Journal of Speech, Language, and Hearing Research, 56*(5), 1595–1612. http://dx.doi.org/10.1044/1092-4388(2013/12-0102)
Bunta, F., & Douglas, M. (2013). The effects of dual-language support on the language skills of bilingual children with hearing loss who use listening devices relative to their monolingual peers. *Language, Speech, and Hearing Services in Schools, 44*(3), 281–290. http://dx.doi.org/10.1044/0161-1461(2013/12-0073)
Carson, K. L., Gillon, G. T., & Boustead, T. M. (2013). Classroom phonological awareness instruction and literacy outcomes in the first year of school. *Language, Speech, and Hearing Services in Schools, 44*(2), 147–160. http://dx.doi.org/10.1044/0161-1461(2012/11-0061)
Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: Joining forces with neuroscience. *Nature Reviews Neuroscience, 7*(7), 583–590. http://dx.doi.org/10.1038/nrn1925
Caspi, A., Williams, B., Kim-Cohen, J., Craig, I. W., Milne, B. J., Poulton, R., et al. (2007). Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. *Proceedings of the National Academy of Sciences of the United States of America, 104*(47), 18860–18865. http://dx.doi.org/10.1073/pnas.0704292104
Cattani, A., Bonifacio, S., Fertz, M., Iverson, J. M., Zocconi, E., & Caselli, M. C. (2010). Communicative and linguistic development in preterm children: A longitudinal study from 12 to 24 months. *International Journal of Language & Communication Disorders, 45*(2), 162–173. http://dx.doi.org/10.3109/13682820902818870
Coggins, T. E., Timler, G. R., & Olswang, L. B. (2007). A state of double jeopardy: Impact of prenatal alcohol exposure and adverse environments on the social communicative abilities of school-age children with fetal alcohol spectrum disorder. *Language, Speech, and Hearing Services in Schools, 38*(2), 117–127. http://dx.doi.org/10.1044/0161-1461(2007/012)
Cone-Wesson, B. (2005). Prenatal alcohol and cocaine exposure: Influences on cognition, speech, language, and hearing. *Journal of Communication Disorders, 38*(4), 279–302. http://dx.doi.org/10.1016/j.jcomdis.2005.02.004
Conti-Ramsden, G. (1985). Mothers in dialogue with language-impaired children. *Topics in Language Disorders, 5*(2), 58–68. Retrieved from http://journals.lww.com/topicsinlanguagedisorders/pages/default.aspx
Conti-Ramsden, G., Simkin, Z., & Pickles, A. (2006). Estimating familial loading in SLI: A comparison of direct assessment versus parental interview. *Journal of Speech, Language, and Hearing Research, 49*(1), 88–101. http://dx.doi.org/10.1044/1092-4388(2006/007)
Cramblit, N. S., & Siegel, G. M. (1977). The verbal environment of a language-impaired child. *Journal of Speech and Hearing Disorders, 42*(4), 474–482. http://dx.doi.org/10.1044/jshd.4204.474
Crosbie, S., Holm, A., Wandschneider, S., & Hemsley, G. (2011). Narrative skills of children born preterm. *International Journal of Language & Communication Disorders, 46*(1), 83–94. http://dx.doi.org/10.3109/13682821003624998
Dale, P. S., Tosto, M. G., Hayiou-Thomas, M. E., & Plomin, R. (2015). Why does parental language input predict child language development? A twin study of gene–environment correlation. *Journal of Communication Disorders, 57*, 106–117.
Day, J. J., & Sweatt, J. D. (2011). Epigenetic mechanisms in cognition. *Neuron, 70*(5), 813–829. http://dx.doi.org/10.1016/j.neuron.2011.05.019
DeThorne, L. S., & Channell, R. W. (2007). Clinician–child interactions: Adjustments in linguistic complexity. *American Journal of Speech–Language Pathology, 16*(2), 119–127. http://dx.doi.org/10.1044/1058-0360(2007/016)
DeThorne, L. S., & Hart, S. A. (2009). Use of the twin design to examine evocative gene–environment effects within a conversational context. *International Journal of Developmental Science, 3*(2), 175–194. http://dx.doi.org/10.3233/DEV-2009-3207
DeThorne, L. S., Petrill, S. A., Hart, S. A., Channell, R. W., Campbell, R. J., Deater-Deckard, K., et al. (2008). Genetic effects on children's conversational language use. *Journal of Speech, Language, and Hearing Research, 51*(2), 423–435. http://dx.doi.org/10.1044/1092-4388(2008/031)
DeThorne, L. S., Petrill, S. A., Hayiou-Thomas, M. E., & Plomin, R. (2005). Low expressive vocabulary higher heritability as a function of more severe cases. *Journal of Speech, Language, and Hearing Research, 48*(4), 792–804. http://dx.doi.org/10.1044/1092-4388(2005/055)
Dimitropoulos, A., Ferranti, A., & Lemler, M. (2013). Expressive and receptive language in Prader–Willi syndrome: Report on genetic subtype differences. *Journal of Communication Disorders, 46*(2), 193–201. http://dx.doi.org/10.1016/j.jcomdis.2012.12.001
Dotterer, A. M., Iruka, I. U., & Pungello, E. (2012). Parenting, race, and socioeconomic status: Links to school readiness. *Family Relations, 61*(4), 657–670.
Eaton, C. T., & Ratner, N. B. (2013). Rate and phonological variation in preschool children: Effects of modeling and directed influence. *Journal of Speech, Language, and Hearing Research, 56*(6), 1751–1763. http://dx.doi.org/10.1044/1092-4388(2013/12-0171)
Ellis, B. J., & Garber, J. (2000). Psychosocial antecedents of variation in girls' pubertal timing: Maternal depression, stepfather presence, and marital and family stress. *Child Development, 71*(2), 485–501.
Evans, G. W. (2006). Child development and the physical environment. *Annual Review of Psychology, 57*, 423–451. http://dx.doi.org/10.1146/annurev.psych.57.102904.190057
Evans, J. L. (2007). The emergence of language: A dynamical systems account. In *Blackwell handbook of language development* (pp. 128–147).
Fagiolini, M., Jensen, C. L., & Champagne, F. A. (2009). Epigenetic influences on brain development and plasticity. *Current Opinion in Neurobiology, 19*(2), 207–212. http://dx.doi.org/10.1016/j.conb.2009.05.009
Fey, M. E., Yoder, P. J., Warren, S. F., & Bredin-Oja, S. L. (2013). Is more better? Milieu communication teaching in toddlers with intellectual disabilities. *Journal of Speech, Language, and Hearing Research, 56*(2), 679–693. http://dx.doi.org/10.1044/1092-4388(2012/12-0061)
Gilbert, S. F., & Epel, D. (2009). *Ecological developmental biology: Integrating epigenetics, medicine, and evolution*. Sunderland: Sinauer Associates.
Gleason, J. B., & Ratner, N. B. (2013). *The development of language* (8th ed.). Upper Saddle River, NJ: Pearson Education.
Gluckman, P. D., Hanson, M. A., Spencer, H. G., & Bateson, P. (2005). Environmental influences during development and their later consequences for health and disease: Implications for the interpretation of empirical studies. *Proceedings of the Royal Society B: Biological Sciences, 272*(1564), 671–677. http://dx.doi.org/10.1098/rspb.2004.3001

Gottlieb, G. (2010). Normally occurring environmental and behavioral influences on gene activity: From central dogma to probabilistic epigenesis. In K. E. Hood, C. T. Halpern, G. Greenberg, & R. M. Lerner (Eds.), *Handbook of developmental science, behavior, and genetics* (pp. 13–38). Malden, MA: Wiley-Blackwell http://dx.doi.org/10.1002/9781444327632.fmatter

Gregory, S. G., Connelly, J. J., Towers, A. J., Johnson, J., Biscocho, D., Markunas, C. A., et al. (2009). Genomic and epigenetic evidence for oxytocin receptor deficiency in autism. *BMC Medicine, 7*(1), 62. http://dx.doi.org/10.1186/1741-7015-7-62

Griffiths, P. E., & Tabery, J. (2013). Developmental systems theory: What does it explain, and how does it explain it? *Advances in Child Development and Behavior, 44*, 65–94.

Haebig, E., McDuffie, A., & Weismer, S. E. (2013). The contribution of two categories of parent verbal responsiveness to later language for toddlers and preschoolers on the autism spectrum. *American Journal of Speech–Language Pathology, 22*(1), 57–70. http://dx.doi.org/10.1044/1058-0360(2012/11-0004)

Halpern, C. T. (2010). The significance of non-replication of gene–phenotype associations. In K. E. Hood, C. T. Halpern, G. Greenberg, & R. M. Lerner (Eds.), *Handbook of developmental science, behavior, and genetics* (pp. 466–490). Malden, MA: Wiley-Blackwell http://dx.doi.org/10.1002/9781444327632.fmatter

Harper, L. V. (2010). Trans-generational epigenetic inheritance. In *Handbook of developmental science, behavior, and genetics* (pp. 434–465) http://dx.doi.org/10.1002/9781444327632.ch14

Hay, E., & Moran, C. (2005). Discourse formulation in children with closed head injury. *American Journal of Speech–Language Pathology, 14*(4), 324–336. http://dx.doi.org/10.1044/1058-0360(2005/031)

Henry, J., Sloane, M., & Black-Pond, C. (2007). Neurobiology and neurodevelopmental impact of childhood traumatic stress and prenatal alcohol exposure. *Language, Speech, and Hearing Services in Schools, 38*(2), 99–108. http://dx.doi.org/10.1044/0161-1461(2007/010)

Hepp, N. (2011). Protecting children from toxicants. *The ASHA Leader,* (November). Retrieved from http://www.asha.org/Publications/leader/2011/111122/Protecting-Children-From-Toxicants.htm

Hirsh-Pasek, K., & Golinkoff, R. M. (1996). *The origins of grammar: Evidence from early language comprehension*. Cambridge, MA: MIT Press.

Hoff, E., Laursen, B., & Tardif, T. (2002). Socioeconomic status and parenting. In M. H. Bornstein (Ed.), *Handbook of parenting: Biology and ecology of parenting* (Vol. 2, pp. 231–252). USA: Lawrence Erlbaum Associates.

Hood, K. E., Halpern, C. T., Greenberg, G., & Lerner, R. M. (2010). Developmental systems, nature-nurture, and the role of genes in behavior and development: On the legacy of Gilbert Gottlieb. In K. E. Hood, C. T. Halpern, G. Greenberg, & R. M. Lerner (Eds.), *Handbook of developmental science, behavior, and genetics* (pp. 3–12). Malden, MA: Wiley-Blackwell http://dx.doi.org/10.1002/9781444327632.fmatter

Hulit, L. M., & Howard, M. R. (2006). Language acquisition: A theoretical journey. *Born to talk: An introduction to speech and language development* (4th ed., pp. 16–54). .

Hyter, Y. D. (2007). Prologue: Understanding children who have been affected by maltreatment and prenatal alcohol exposure. *Language, Speech, and Hearing Services in Schools, 38*(2), 93–98. http://dx.doi.org/10.1044/0161-1461(2007/009)

Iverson, J. M., Longobardi, E., Spampinato, K., & Caselli, M. C. (2006). Gesture and speech in maternal input to children with Down's syndrome. *International Journal of Language & Communication Disorders, 41*(3), 235–251. http://dx.doi.org/10.1080/13682820500312151

Jaenisch, R., & Bird, A. (2003). Epigenetic regulation of gene expression: How the genome integrates intrinsic and environmental signals. *Nature Genetics, 33*, 245–254. http://dx.doi.org/10.1038/ng1089

Jerger, S., Damian, M. F., Mills, C., Bartlett, J., Tye-Murray, N., & Abdi, H. (2013). Effect of perceptual load on semantic access by speech in children. *Journal of Speech, Language, and Hearing Research, 56*(2), 388–403. http://dx.doi.org/10.1044/1092-4388(2012/11-0186)

Johnson, J. S., & Newport, E. L. (1989). Critical period effects in second language learning: The influence of maturational state on the acquisition of English as a second language. *Cognitive Psychology, 21*(1), 60–99. http://dx.doi.org/10.1016/0010-0285(89)90003-0

Kaiser, A. P., & Roberts, M. Y. (2013). Parent-implemented enhanced milieu teaching with preschool children who have intellectual disabilities. *Journal of Speech, Language, and Hearing Research, 56*(1), 295–309. http://dx.doi.org/10.1044/1092-4388(2012/11-0231)

King, A. M., Hengst, J. A., & DeThorne, L. S. (2013). Severe speech sound disorders: An integrated multimodal intervention. *Language, Speech, and Hearing Services in Schools, 44*(2), 195–210. http://dx.doi.org/10.1044/0161-1461(2012/12-0023)

Kjellmer, L., & Olswang, L. B. (2013). Variability in classroom social communication: Performance of children with fetal alcohol spectrum disorders and typically developing peers. *Journal of Speech, Language, and Hearing Research, 56*(3), 982–993. http://dx.doi.org/10.1044/1092-4388(2012/11-0345)

Kotchick, B. A., & Forehand, R. (2002). Putting parenting in perspective: A discussion of the contextual factors that shape parenting practices. *Journal of Child and Family Studies, 11*(3), 255–269. http://dx.doi.org/10.1023/A:1016863921662

Kraft, S. J., & DeThorne, L. S. (2014). The brave new world of epigenetics: Embracing complexity in the study of speech and language disorders. *Current Developmental Disorders Reports, 1*(3), 207–214. http://dx.doi.org/10.1007/s40474-014-0024-4

Lam, L. L., Emberly, E., Fraser, H. B., Neumann, S. M., Chen, E., Miller, G. E., et al. (2012). Factors underlying variable DNA methylation in a human community cohort. *Proceedings of the National Academy of Sciences of the United States of America, 109*(Suppl. 2), 17253–17260. http://dx.doi.org/10.1073/pnas.1121249109

Lewis, B. A., Minnes, S., Short, E. J., Min, M. O., Wu, M., Lang, A., et al. (2013). Language outcomes at 12 years for children exposed prenatally to cocaine. *Journal of Speech, Language, and Hearing Research, 56*(5), 1662–1676. http://dx.doi.org/10.1044/1092-4388(2013/12-0119)

Mahurin-Smith, J., & Ambrose, N. G. (2013). Breastfeeding may protect against persistent stuttering. *Journal of Communication Disorders, 46*(4), 351–360. http://dx.doi.org/10.1016/j.jcomdis.2013.06.001

Martin, G. E., Losh, M., Estigarribia, B., Sideris, J., & Roberts, J. (2013). Longitudinal profiles of expressive vocabulary, syntax and pragmatic language in boys with fragile X syndrome or Down syndrome. *International Journal of Language & Communication Disorders, 48*(4), 432–443. http://dx.doi.org/10.1111/1460-6984.12019

McDade, T. W. (2012). Early environments and the ecology of inflammation. *Proceedings of the National Academy of Sciences of the United States of America, 109*(Suppl. 2), 17281–17288. http://dx.doi.org/10.1073/pnas.1202244109

McEwen, B. S. (2012). Brain on stress: How the social environment gets under the skin. *Proceedings of the National Academy of Sciences of the United States of America, 109*(Suppl. 2), 17180–17185. http://dx.doi.org/10.1073/pnas.1121254109

McNeilly, L. G. (2005). HIV and communication. *Journal of Communication Disorders, 38*(4), 303–310. http://dx.doi.org/10.1016/j.jcomdis.2005.02.005

Meaney, M. J. (2010). Epigenetics and the biological definition of gene × environment interactions. *Child Development, 81*(1), 41–79. http://dx.doi.org/10.1111/j.1467-8624.2009.01381.x

Mira, W. A., & Schwanenflugel, P. J. (2013). The impact of reading expressiveness on the listening comprehension of storybooks by prekindergarten children. *Language, Speech, and Hearing Services in Schools, 44*(2), 183–194. http://dx.doi.org/10.1044/0161-1461(2012/11-0073)

Monteggia, L. M., & Kavalali, E. T. (2009). Rett syndrome and the impact of MeCP2 associated transcriptional mechanisms on neurotransmission. *Biological Psychiatry, 65*(3), 204–210. http://dx.doi.org/10.1016/j.biopsych.2008.10.036

Nelson, P., Kohnert, K., Sabur, S., & Shaw, D. (2005). Classroom noise and children learning through a second language double jeopardy? *Language, Speech, and Hearing Services in Schools, 36*(3), 219–229. http://dx.doi.org/10.1044/0161-1461(2005/022)

Nestler, E. J. (2009). Epigenetic mechanisms in psychiatry. *Biological Psychiatry, 65*(3), 189–190. http://dx.doi.org/10.1016/j.biopsych.2008.10.030

Newport, E., & Supalla, T. (1987). A critical period effect in the acquisition of a primary language. *Science*. http://dx.doi.org/10.1016/0010-0285(89)90003-0

Nippold, M. A. (2000). Language development during the adolescent years: Aspects of pragmatics, syntax, and semantics. *Topics in Language Disorders, 20*(2), 15–28. Retrieved from http://journals.lww.com/topicsinlanguagedisorders/pages/default.aspx

Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A. M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics, 3*(2), 97–106. http://dx.doi.org/10.4161/epi.3.2.6034

Olswang, L. B., Svensson, L., & Astley, S. (2010). Observation of classroom social communication: Do children with fetal alcohol spectrum disorders spend their time differently than their typically developing peers? *Journal of Speech, Language, and Hearing Research, 53*(6), 1687–1703. http://dx.doi.org/10.1044/1092-4388(2010/09-0092)

Paul, R. (2007). Definitions and models of language disorders in children. In K. Falk (Ed.), *Language disorders from infancy through adolescence: Assessment & intervention* (2nd ed., pp. 3–20). St. Louis, MO Mosby.

Paul, R., & Elwood, T. J. (1991). Maternal linguistic input to toddlers with slow expressive language development. *Journal of Speech, Language, and Hearing Research, 34*(5), 982–988. http://dx.doi.org/10.1044/jshr.3405.982

Pence, K. L., & Justice, L. M. (2012). *Language development from theory to practice* (2nd ed.). Upper Saddle River, NJ: Pearson Education.

Persson, C., Niklasson, L., Oskarsdóttir, S., Johansson, S., Jönsson, R., & Söderpalm, E. (2006). Language skills in 5–8-year-old children with 22q11 deletion syndrome. *International Journal of Language & Communication Disorders, 41*(3), 313–333. http://dx.doi.org/10.1080/13682820500361497

Plomin, R., & Bergeman, C. S. (1991). Nature and nurture. *Behavioral and Brain Sciences, 14*(03), 414–427. http://dx.doi.org/10.1017/S0140525X00070588

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 84*(2), 309. http://dx.doi.org/10.1037/0033-2909.84.2.309

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (Vol. 5). New York, NY: Worth Publishers.

Rescorla, L., & Ratner, N. B. (1996). Phonetic profiles of toddlers with specific expressive language impairment (SLI-E). *Journal of Speech, Language, & Hearing Research, 39*, 153–165. http://dx.doi.org/10.1044/jshr.3901.153

Restrepo, M. A., Morgan, G. P., & Thompson, M. S. (2013). The efficacy of a vocabulary intervention for dual-language learners with language impairment. *Journal of Speech, Language, and Hearing Research, 56*(2), 748–765. http://dx.doi.org/10.1044/1092-4388(2012/11-0173)

Rice, M. L. (2012). Toward epigenetic and gene regulation models of specific language impairment: Looking for links among growth, genes, and impairments. *Journal of Neurodevelopmental Disorders, 4*(1), 27. http://dx.doi.org/10.1186/1866-1955-4-27

Riley, K. G., & McGregor, K. K. (2012). Noise hampers children's expressive word learning. *Language, Speech, and Hearing Services in Schools, 43*(3), 325–337. http://dx.doi.org/10.1044/0161-1461(2012/11-0053)

Robert, J. S. (2003). Developmental systems theory. In B. K. Hall & W. M. Olson (Eds.), *Keywords & concepts in evolutionary developmental biology* (pp. 94–97). USA: President and Fellows of Harvard College.

Roberts, A. L., Lyall, K., Hart, J. E., Laden, F., Just, A. C., Bobb, J. F., et al. (2013). Perinatal air pollutant exposures and autism spectrum disorder in the children of nurses' health study II participants. *Environmental Health Perspectives, 121*(8), 978–984. http://dx.doi.org/10.1289/ehp.1206187

Robinson, G. E., Fernald, R. D., & Clayton, D. F. (2008). Genes and social behavior. *Science, 322*(5903), 896–900. http://dx.doi.org/10.1126/science.1159277

Rodenhiser, D., & Mann, M. (2006). Epigenetics and human disease: Translating basic biology into clinical applications. *Canadian Medical Association Journal, 174*(3), 341–348. http://dx.doi.org/10.1503/cmaj.050774

Rogers-Adkinson, D. L., & Stuart, S. K. (2007). Collaborative services: Children experiencing neglect and the side effects of prenatal alcohol exposure. *Language, Speech, and Hearing Services in Schools, 38*(2), 149–156. http://dx.doi.org/10.1044/0161-1461(2007/015)

Rosenblatt, J. S. (2010). Behavioral development during the mother–young interaction in placental mammals: The development of behavior in the relationship with the mother. In K. E. Hood, C. T. Halpern, G. Greenberg, & R. M. Lerner (Eds.), *Handbook of developmental science, behavior, and genetics* (pp. 205–233). Malden, MA: Wiley-Blackwell http://dx.doi.org/10.1002/9781444327632.fmatter

Sameroff, A. (2010). A unified theory of development: A dialectic integration of nature and nurture. *Child Development, 81*(1), 6–22. http://dx.doi.org/10.1111/j.1467-8624.2009.01378.x

Sansavini, A., Guarini, A., Alessandroni, R., Faldella, G., Giovanelli, G., & Salvioli, G. (2007). Are early grammatical and phonological working memory abilities affected by preterm birth? *Journal of Communication Disorders, 40*(3), 239–256. http://dx.doi.org/10.1016/j.jcomdis.2006.06.009

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Development,* 424–435. http://dx.doi.org/10.2307/1129703

Sepúlveda, E. M., López-Villaseñor, M. L., & Heinze, E. G. (2013). Can individuals with Down syndrome improve their grammar? *International Journal of Language & Communication Disorders, 48*(3), 343–349. http://dx.doi.org/10.1111/1460-6984.12002

Shelton, J. F., Hertz-Picciotto, I., & Pessah, I. N. (2012). Tipping the balance of autism risk: Potential mechanisms linking pesticides and autism. *Environmental Health Perspectives, 120*(7), 944. http://dx.doi.org/10.1289/ehp.1104553

Smith, A. D., Kim, Y. I., & Refsum, H. (2008). Is folic acid good for everyone? *The American Journal of Clinical Nutrition, 87*(3), 517–533. Retrieved from http://ajcn.nutrition.org/

Smith-Lock, K. M., Leitao, S., Lambert, L., & Nickels, L. (2013). Effective intervention for expressive grammar in children with specific language impairment. *International Journal of Language & Communication Disorders, 48*(3), 265–282. http://dx.doi.org/10.1111/1460-6984.12003

Stockman, I. J., Karasinski, L., & Guillory, B. (2008). The use of conversational repairs by African American preschoolers. *Language, Speech, and Hearing Services in Schools, 39*(4), 461–474. http://dx.doi.org/10.1044/0161-1461(2008/07-0095)

Stokes, S. F., & Surendran, D. (2005). Articulatory complexity, ambient frequency, and functional load as predictors of consonant development in children. *Journal of Speech, Language, and Hearing Research, 48*(3), 577–591. http://dx.doi.org/10.1044/1092-4388(2005/040)

Storkel, H. L., Maekawa, J., & Aschenbrenner, A. J. (2013). The effect of homonymy on learning correctly articulated versus misarticulated words. *Journal of Speech, Language, and Hearing Research, 56*(2), 694–707. http://dx.doi.org/10.1044/1092-4388(2012/12-0122)

Stromswold, K. (1998). Genetics of spoken language disorders. *Human Biology, 70*(2), 297–324. Retrieved from http://wsupress.wayne.edu/journals/detail/human-biology

Stromswold, K. (2006). Why aren't identical twins linguistically identical? Genetic, prenatal and postnatal factors. *Cognition, 101*(2), 333–384. http://dx.doi.org/10.1016/j.cognition.2006.04.007

Tabery, J., & Griffiths, P. E. (2010). Perspectives on behavioral genetics and developmental science. In *Handbook of developmental science, behavior, and genetics* (p. 41).

Tadić, V., Pring, L., & Dale, N. (2013). Story discourse and use of mental state language between mothers and school-aged children with and without visual impairment. *International Journal of Language & Communication Disorders, 48*(6), 679–688. http://dx.doi.org/10.1111/1460-6984.12040

Thelen, E. (2005). Dynamic systems theory and the complexity of change. *Psychoanalytic Dialogues, 15*(2), 255–283. http://dx.doi.org/10.1080/10481881509348831

Thordardottir, E., & Brandeker, M. (2013). The effect of bilingual exposure versus language impairment on nonword repetition and sentence imitation scores. *Journal of Communication Disorders, 46*(1), 1–16. http://dx.doi.org/10.1016/j.jcomdis.2012.08.002

van Bysterveldt, A. K., Westerveld, M. F., Gillon, G., & Foster-Cohen, S. (2012). Personal narrative skills of school-aged children with Down syndrome. *International Journal of Language & Communication Disorders, 47*(1), 95–105. http://dx.doi.org/10.1111/j.1460-6984.2011.00085.x

van Engeland, M., Weijenberg, M. P., Roemen, G. M., Brink, M., de Bruïne, A. P., Goldbohm, R. A., et al. (2003). Effects of dietary folate and alcohol intake on promoter methylation in sporadic colorectal cancer: The Netherlands cohort study on diet and cancer. *Cancer Research, 63*(12), 3133–3137. Retrieved from http://cancerres.aacrjournals.org/

Van Herwegen, J., Dimitriou, D., & Rundblad, G. (2013). Performance on verbal and low-verbal false belief tasks: Evidence from children with Williams syndrome. *Journal of Communication Disorders, 46*(5), 440–448. http://dx.doi.org/10.1016/j.jcomdis.2013.10.002

Weikum, W. M., Oberlander, T. F., Hensch, T. K., & Werker, J. F. (2012). Prenatal exposure to antidepressants and depressed maternal mood alter trajectory of infant speech perception. *Proceedings of the National Academy of Sciences of the United States of America, 109*(Suppl. 2), 17221–17227. http://dx.doi.org/10.1073/pnas.1121263109

Zeisel, S. H. (2009). Epigenetic mechanisms for nutrition determinants of later health outcomes. *The American Journal of Clinical Nutrition, 89*(5), 1488S–1493S. http://dx.doi.org/10.3945/ajcn.2009.27113B

Zubrick, S. R., Taylor, C. L., Rice, M. L., & Slegers, D. W. (2007). Late language emergence at 24 months: An epidemiological study of prevalence, predictors, and covariates. *Journal of Speech, Language, and Hearing Research, 50*(6), 1562–1592. http://dx.doi.org/10.1044/1092-4388(2007/106)