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Ambient temperature exposure and rapid infant weight gain

Neora Alterman [1,*] Daniel Nevo [2] Ronit Calderon-Margalit,[1] Iaroslav Youssim [1] Bar Weinstein,[2] Itai Kloog,[3] Michael Hauzer[4] and Raanan Raz [1]

[1]Braun School of Public Health and Community Medicine, Faculty of Medicine, The Hebrew University of Jerusalem, Jerusalem, Israel, [2]Department of Statistics and Operations Research, Tel Aviv University, Tel Aviv, Israel, [3]Department of Geography and Environmental Development, Ben-Gurion University of the Negev, Beer-Sheva, Israel and [4]Bonen Clinic, Haifa and Western Galilee District, Clalit Health Services, Haifa, Israel

*Corresponding author. Braun School of Public Health and Community Medicine, Faculty of Medicine, The Hebrew University of Jerusalem, Ein Karem, PO Box 12272, Jerusalem 91120, Israel. E-mail: neora.alterman@mail.huji.ac.il

Abstract

Background: Childhood obesity is a major public health concern, and the global rate is rising. Rapid infant weight gain is a risk factor for later overweight. Studies have linked prenatal ambient temperature exposure to fetal growth, and preliminary evidence suggests postnatal exposure may be associated with infant weight gain.

Methods: Using a population-based historical cohort study including 1 100 576 infants born 2011–2019, we assessed the relationship between prenatal and one-month postnatal ambient temperature exposure and rapid infant weight gain. We used a hybrid spatiotemporal model to assess temperatures at the family's recorded residence at birth. Repeated weight measurements between birth and 15 months were used to model the outcome using the SuperImposition by Translation and Rotation (SITAR) method. We employed generalized linear models and distributed lag models to estimate the association between prenatal and postnatal exposure and rapid infant weight gain, defined as the upper tertile of the SITAR growth velocity.

Results: Overall, higher ambient temperatures were associated with rapid infant weight gain. The cumulative adjusted relative risk for the highest exposure quintile during pregnancy compared with the lowest quintile was 1.33 [95% confidence interval (CI): 1.25, 1.40], and the corresponding association for the first postnatal month was 1.19 (95% CI: 1.15, 1.23). Exposure to high ambient temperature during early and mid-pregnancy, as well as the first postnatal month, was associated with rapid weight gain, while during late pregnancy, exposure to low temperatures was associated with this outcome.

Conclusions: Prenatal and postnatal ambient temperatures are associated with rapid infant weight gain.

Keywords: Climate change, rapid infant weight gain, infant growth, ambient temperature.

Key Messages

- The study examined whether prenatal or postnatal exposure to ambient temperature is associated with rapid infant weight gain, a risk factor for overweight and obesity.
- We found that exposure to higher temperatures during early and mid-pregnancy, as well as the first postnatal month, are associated with rapid infant weight gain.
- In the era of climate change, a possible effect of ambient temperature on early growth and potentially on later adiposity could have population-wide implications.

Introduction

Over the past five decades, the global prevalence of obesity among children and adolescents has escalated from less than 1% to 9%.[1,2] In Israel, obesity is documented in 9% of children and 13% of adolescents.[3] These high rates present a substantial public health concern, as obesity is a well-established risk factor for non-communicable diseases such as cardiovascular disease, type-2 diabetes, and certain types of cancer.[4] Human rapid infant weight gain is associated with childhood overweight and obesity,[5–8] and animal experiments have demonstrated the adverse effects of excess neonatal weight gain on adult weight and metabolic health.[9,10]

Epidemiological studies suggested an association between *in utero* exposure to ambient temperature and fetal and neonatal outcomes, such as fetal growth and preterm birth.[11–13] Increased temperatures are associated with low birthweight and small for gestational age in term babies,[14–16] but some

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studies have found that both temperature extremities may be associated with these outcomes.[17–19] Exposure to high temperatures in adulthood is also associated with obesity.[20–22] There is, therefore, considerable evidence of a possible effect of prenatal temperature exposure on intrauterine growth and some further evidence of an effect of later exposure on adiposity. Yet, there is a scarcity of studies focusing on exposure during infancy, the critical window during development when rapid weight gain is thought to have long-term implications on body composition.[5,8]

We have previously described a preliminary positive association between rapid infant weight gain and ambient temperature during the first year of life.[23] These earlier results were based on an outcome composed using only two weight measurements, and the exposure was based on clinic addresses and averaged over the entire first year. An additional limitation of the prior study was that prenatal temperature exposure was not examined, although the seasonal nature of ambient temperature induces a strong inter-correlation among temperature exposures across periods of early life development.

This nationwide population-based historical cohort study aims to expand the knowledge of the potential effect of temperature on infant weight gain with an advanced growth modelling approach. It uses a larger and updated dataset with multiple growth measurements, including data on ambient temperature at the individual address level during pregnancy and the first month of life.

Methods

Study population

We conducted a historical cohort study using data from Israel's national network of maternal and child health clinics. These clinics provide free vaccinations and development screening services by certified nurses and doctors for all children aged birth to 6 years.

Our study utilized data about 1 295 893 infants visiting one of 526 out of 926 clinics that transfer data to the Ministry of Health[24] (Supplementary Figure S1, available as Supplementary data at *IJE* online). These include clinics managed by the Ministry, local municipalities and public health maintenance organizations, all of which provide similar services. Our data covers approximately 80% of the infants born in Israel between 2011 and 2019.[25] The population for analyses did not include 31 678 infants without a linked address, 159 990 missing birthweight that were excluded since birth is an important point in the infant's growth trajectory, and 3649 infants missing gestational age at birth for which the exposure could not be determined. In total we excluded 15.1% of the source population (Supplementary Figure S1, available as Supplementary data at *IJE* online). A comparison between the source population and the population for analyses presented generally similar characteristics (Supplementary Table S1, available as Supplementary data at *IJE* online).

Outcome

Validated weight measurements obtained by nurses according to a standard protocol were used to model infant growth curves and derive weight gain velocity.[26,27] This measure is a risk factor for later adiposity[8] and is often used as a growth indicator without incorporating information about length when length may be less reliable (such as in routinely collected data). We incorporated all weight measurements taken from birth to 15 months to capture latecomers to the age one-year recommended visit. We employed the SuperImposition by Translation and Rotation (SITAR) method,[28] a shape-invariant, non-linear mixed-effects model that represents the average growth curve in the population while including random-effect parameters that portray individual deviation from the average curve. The size parameter represents the vertical shift of an individual's growth curve relative to the population mean, and the growth velocity parameter indicates its steepness. Positive values indicate larger size (weight) or faster growth (weight gain).[28,29]

Using the *sitar R* package,[30] we applied the SITAR method to the source population ($n = 1\ 295\ 893$; Supplementary Figure S1, available as Supplementary data at *IJE* online) with an average of 8.4 weight measurements per infant (standard deviation 2.9). The modelling process was described in detail previously.[31] Briefly, we computed the average growth curve as a natural cubic spline function with four degrees of freedom and incorporated individual size and velocity parameters. We defined rapid weight gain as being in the upper tertile of SITAR velocity within the population for analysis. This measure has high agreement with the traditional measure of weight gain using age- and sex-standardized *z*-score difference >0.67.[31]

Exposure

Ambient temperature was assessed through a hybrid model that generates daily 1 × 1-km mean temperature estimates. The model, described in detail previously[32] uses remote-sensing satellite measures validated against meteorological stations with high model performance ($R^2 = 0.965$–0.968).[32] Mean temperatures in the areas covered by the model are presented in Supplementary Figure S2 (available as Supplementary data at *IJE* online).

We next geocoded the family's residential address at birth recorded by clinic nurses. Mean temperature at that location was calculated for several periods: each trimester of pregnancy (gestational weeks 1–13, 14–26 and 27–birth) and the first four postnatal weeks, i.e. first month. We further calculated weekly averages during each of 1–37 gestational weeks and during each of the first four postnatal weeks. The length of the postnatal exposure period was selected based on the average timing of infant peak weight gain velocity measured through the SITAR model, which is estimated to occur at 4–6 weeks of age.[31,33,34] Lastly, we categorized all exposure estimates into quintiles (<15.4, 15.4–19.4, 19.4–23.6, 23.6–26.7 and >26.7 °C).

Statistical analysis

To examine the association between mean exposure during each pregnancy trimester or the first month of life we used the 'Modified Poisson Regression Method' appropriate for common outcomes.[35] We estimated relative risks (RR) and 95% confidence intervals (CIs), fitting generalized linear models with a Poisson distribution and log link with robust standard errors using the lowest exposure quintile as reference.

As our primary analysis, we estimated the RRs of weekly exposures and rapid weight gain using distributed lag non-linear models with the *dlnm* R package.[36,37] This method estimates the association between exposure at each period (i.e. lags, in this case, prenatal or postnatal weeks) with the outcome, while

accounting for exposure during all other periods. It enables to flexibly identify critical windows without the restrain of predefined periods such as pregnancy trimesters.[38]

When fitting distributed lag models, we modelled the time-response dimension using natural splines with three degrees of freedom and the exposure-response dimension with temperature quintiles. We additionally modelled temperature as a continuous exposure, in which both the exposure-response and the time-response functions were modelled with natural splines with three degrees of freedom and using the minimum temperature in our sample (0 °C) as the reference. The degrees of freedom chosen were such that allowed a non-linear association but avoided over-fitting (Supplementary Figures S3 and S4, available as Supplementary data at *IJE* online). For exposure-response curves that appeared linear, we further estimated the RR per increase in temperature inter-quartile range.

Covariate selection was guided by directed acyclic graphs (DAGs, Supplementary Figures S5 and S6, available as Supplementary data at *IJE* online) using dagitty.net,[39] and the final set included population group (Jewish/Arab/other/missing), subdistrict, month and year of birth and the socio-economic index of the residence.[40] The latter was categorized into sextiles with an additional category for missing values. Since the DAGs suggested the possibility of confounding the effect of postnatal exposure by prenatal exposure and vice versa, we included all exposure periods in the same regression models.[38] All analyses were conducted in R version 4.2.1.[41]

Sensitivity analyses

We examined splines with several degrees of freedom for our exposure-response and lag-response models and ran models with additional adjustments for maternal country of birth and recent immigration status (<10 years) (as suggested by the DAGs). We further excluded preterm infants and those born with low birthweight as they typically experience 'catch-up' growth.[42] Lastly, we ran a linear regression using a similar model with a continuous outcome measure to examine weight gain velocity across the entire growth velocity scale.

Results

The analyzed population included 1 100 576 infants (Table 1). There was a higher proportion of rapid weight gain among infants born to families from higher socioeconomic classes, Muslim families, males, firstborns, and those born preterm (<37 weeks), early term (37–38 weeks) or with lower birthweight.

The mean weekly temperature exposure was 21.0 °C (inter-quartile range 9.8 °C; Supplementary Figure S7, available as Supplementary data at *IJE* online). A very weak correlation was found between the exposures during the second and third trimesters (Pearson's $r = 0.08$), and the strongest correlation was found between the first and third trimesters (Pearson's $r = -0.80$). Supplementary Table S2 and Supplementary Figure S8 (available as Supplementary data at *IJE* online) show the correlations among all exposure period pairs.

Table 1. Characteristics of study infants with and without rapid weight gain, Israel 2011–2019.

Characteristic		Non-rapid weight gain ($n = 733\,718$)	Rapid weight gain[a] ($n = 366\,858$)	Total ($n = 1\,100\,576$)
Socioeconomic index sextile	First (lowest)	17.7	12.1	15.8
	Second	17.8	14.3	16.6
	Third	14.9	17.4	15.7
	Fourth	15.2	17.2	15.9
	Fifth	14.9	17.3	15.7
	Sixth (highest)	15.3	17.3	16.0
	Missing	4.3	4.4	4.3
Population group	Jewish	64.1	57.4	61.8
	Muslim	18.6	24.4	20.5
	Other	4.9	5.7	5.2
	Missing	12.4	12.5	12.4
Sex	Male	49.1	55.9	51.4
	Female	50.9	44.1	48.6
Gestational age (week)	Preterm (<37)	1.1	20.0	7.4
	Early term (37–38)	19.0	36.7	24.9
	Full term (39–40)	61.2	37.1	53.1
	Late term (41)	16.0	5.3	12.4
	Post term (≥42)	2.8	0.8	2.1
Birthweight (kg)	<2.5	0.5	21.8	7.6
	2.5–3.5	59.3	71.9	63.5
	3.5–4.0	32.5	5.9	23.7
	>4.0	7.6	0.3	5.2
Parity	1	23.5	36.0	27.7
	2	22.3	21.2	21.9
	3	14.8	12.6	14.0
	4	7.5	5.8	6.9
	≥5	10.0	5.6	8.5
	Missing	21.9	18.8	20.9
Season of birth	Mar–May	23.9	23.7	23.8
	Jun–Aug	25.6	25.4	25.5
	Sep–Nov	26.2	25.9	26.1
	Dec–Feb	24.4	25.0	24.6

[a] Rapid weight gain is defined as the highest tertile of the SuperImposition by Translation and Rotation (SITAR) weight gain velocity parameter.

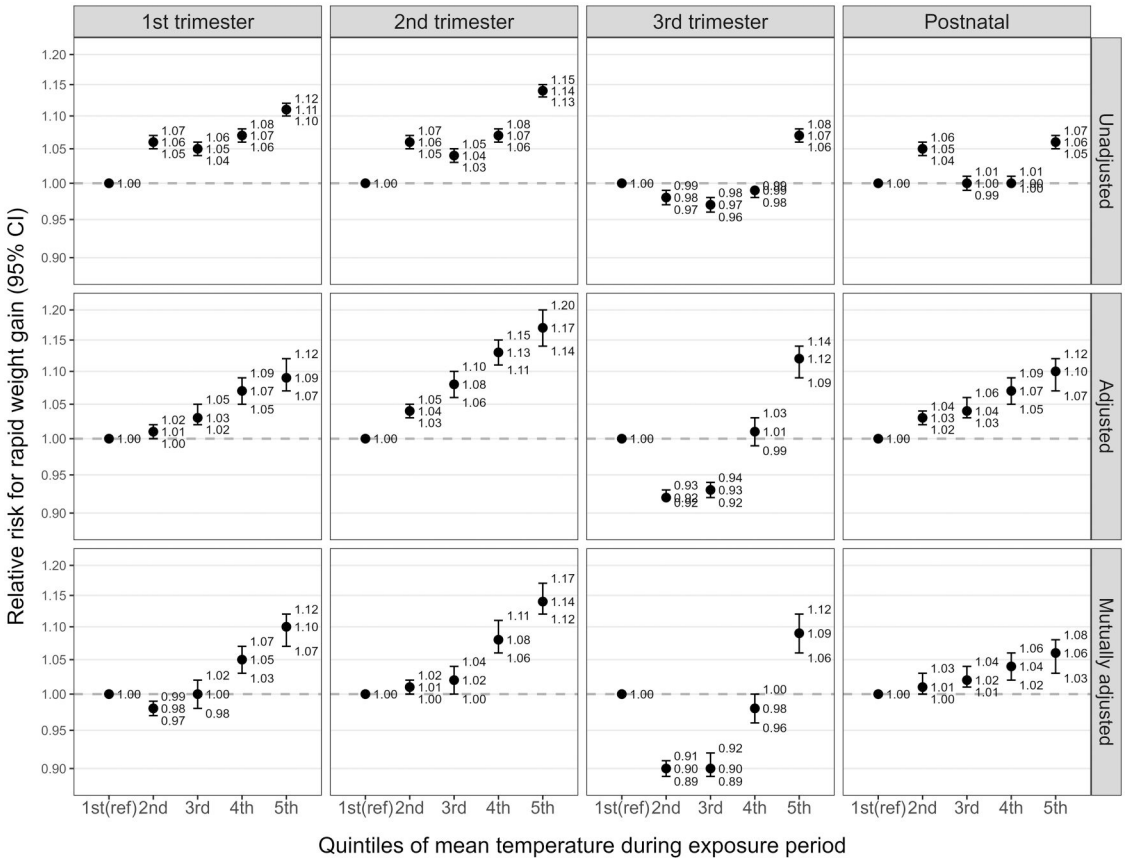

Figure 1. The association between quintiles of mean ambient temperature (<15.4, 15.4–19.4, 19.4–23.6, 23.6–26.7, >26.7 °C) during the three trimesters of pregnancy and first postnatal month with rapid infant weight defined as the upper tertile of SITAR weight gain velocity—unadjusted, adjusted and mutually adjusted models. Associations are presented as the relative risk (95% confidence interval) during each exposure period per each quintile of ambient temperature compared to the first quintile. Adjusted models include socioeconomic index sextile, population group, subdistrict, year and month of birth. Mutually adjusted models are adjusted for all listed confounders as well as for mean weekly temperature exposure during the alternate period—prenatal or postnatal. $n = 1\ 099\ 445$ Israel, 2011–2019

After accounting for potential confounders, the adjusted associations between quintiles of mean temperature during each trimester or the first postnatal month and rapid weight gain were all positive (Figure 1, middle row). The associations were slightly reduced in models that included all exposure periods (i.e. mutually adjusted; Figure 1, bottom row). The adjusted relative risks (aRR) when exposed to the highest temperature quintile compared with the lowest in the first trimester, the second, the third, and the first postnatal month were 1.10 (95% CI 1.07, 1.12), 1.14 (95% CI 1.12, 1.17), 1.09 (95% CI 1.06, 1.12) and 1.06 (95% CI 1.03, 1.08), respectively. The associations were mostly positive—either monotonous or J-shaped (Figure 1, bottom row).

Using a distributed lag model with weekly exposures, we found that the cumulative adjusted association for prenatal exposure was weak positive for the second and third quintiles but stronger for the fourth and fifth, reaching an aRR of 1.46 (95% CI 1.38, 1.54) for the fifth exposure quintile compared with the first (Figure 2a, middle). Further adjustment for postnatal exposure weakened the association slightly (aRR = 1.33, 95% CI 1.25, 1.40) (Figure 2a, right). The adjusted association for the postnatal period was positive monotonous, with an aRR of 1.15 (95% CI 1.11, 1.18) for the highest exposure quintile (Figure 2b, middle), and the association strengthened slightly when further adjusting for the prenatal exposure (aRR = 1.19, 95% CI 1.15, 1.23) (Figure 2b, right).

When examining weekly associations, we found that temperature exposure during early and mid-pregnancy was positively associated with rapid weight gain (Figure 3 and Supplementary Figure S9, available as Supplementary data at *IJE* online). The association between temperature and the outcome turned negative at later gestational weeks. The average postnatal weekly association was generally much stronger than the average prenatal weekly association (Supplementary Figure S9, available as Supplementary data at *IJE* online).

When modelling temperature as a continuous variable, we found that prenatal temperature exposure has an S-shaped cumulative association with rapid weight gain, with the minimal risk at a weekly average of approximately 15 °C and the maximal risk at approximately 29 °C (Figure 4a). This pattern was found for exposure throughout most of the pregnancy length, but towards late pregnancy, a negative association between ambient temperature and rapid weight gain was found (Supplementary Figure S11a, available as Supplementary data at *IJE* online). For postnatal exposure, the cumulative association with rapid weight gain was monotonous positive (Figure 4b and Supplementary Figure S11b, available as Supplementary data at *IJE* online). For every 9.8 °C increase in postnatal temperature (i.e. interquartile range), there was a cumulative higher risk for rapid weight gain of aRR = 1.12 (95% CI 1.10, 1.14).

A sensitivity analysis of additional adjustment for recent immigration and maternal country of birth did not materially

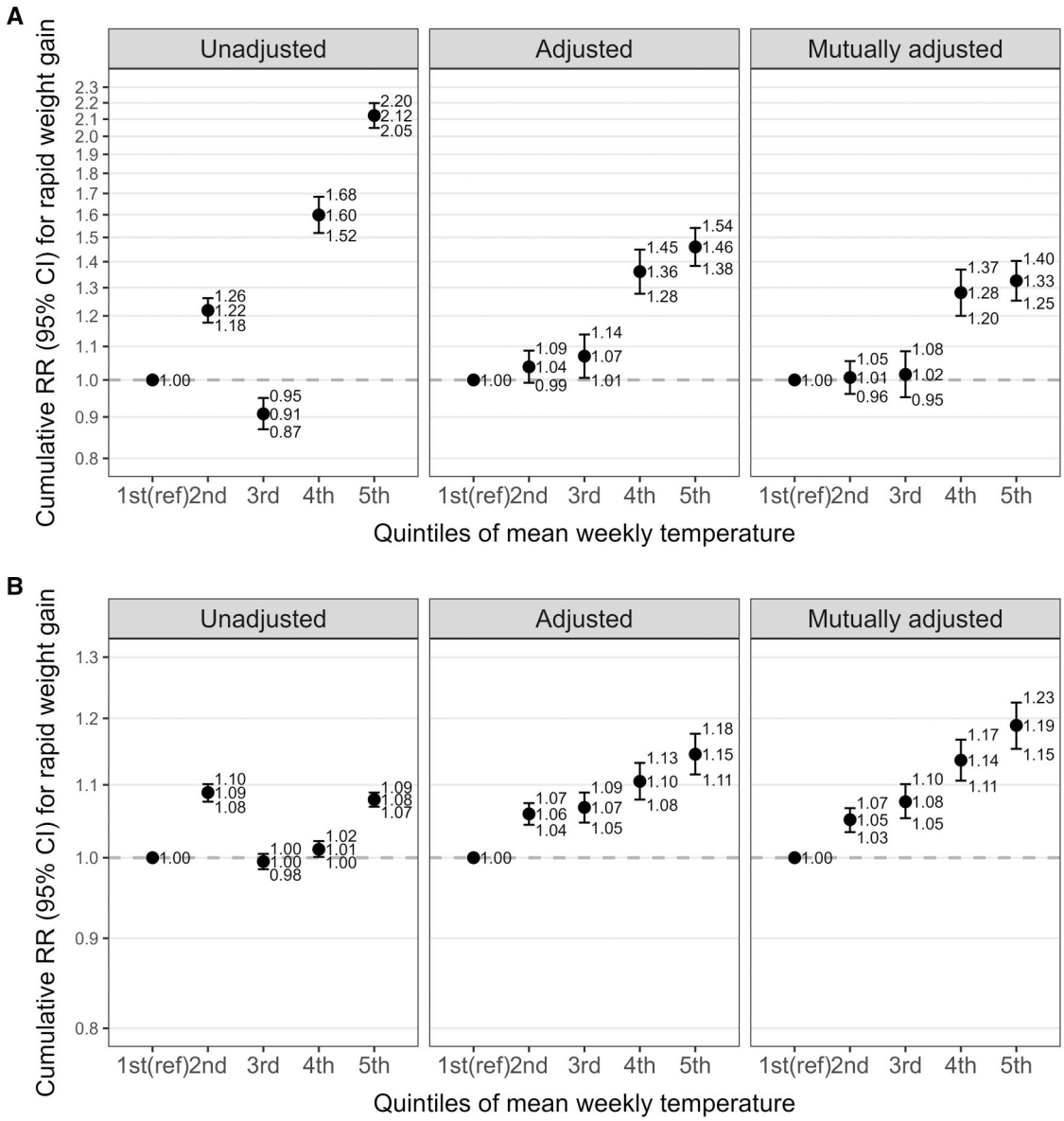

Figure 2. Cumulative association between quintiles of prenatal or postnatal mean weekly ambient temperature and rapid infant weight gain defined as the upper tertile of SITAR weight gain velocity—unadjusted, adjusted and mutually adjusted models. Associations are presented as the cumulative relative risk (95% confidence interval) during the entire exposure period per each quintile of weekly ambient temperature compared to the first quintile—(a) Prenatal and (b) Postnatal. Note different *Y* scales. Adjusted models include socioeconomic index sextile, population group, subdistrict, year and month of birth. Mutually adjusted models are adjusted for all listed confounders as well as for mean weekly temperature exposure during the alternate period—prenatal or postnatal. $n = 1\,100\,576$, Israel, 2011–2019

change the results (Supplementary Figure S12, available as Supplementary data at *IJE* online). Limiting the sample to infants born at term with birthweight >2.5 kg resulted in somewhat stronger associations, especially for the prenatal exposure (Supplementary Figure S13, available as Supplementary data at *IJE* online) with positive associations during early and mid-pregnancy and null towards the end (Supplementary Figure S14a, available as Supplementary data at *IJE* online). Finally, examining weight gain velocity as a continuous outcome was consistent with the primary models (Supplementary Figure S15, available as Supplementary data at *IJE* online).

Discussion

Our study shows that ambient temperature exposure during pregnancy and the first month of life is positively associated with rapid infant weight gain. Findings remained similar after mutual adjustment for the alternate exposure period. When exploring the critical periods of risk, we found that exposure to higher temperature during early and mid-pregnancy, as well as the first four postnatal weeks are associated with a higher risk of rapid infant weight gain. Exposure to higher temperatures during late pregnancy is associated with a lower risk for this outcome.

Interpretation

The majority of the evidence available to date about temperature and early growth has been focused on prenatal exposure and neonatal outcomes. Most studies found high temperatures associated with low birthweight and small for gestational age.[11] Since infants born smaller are predisposed to rapid weight gain,[43] the current study findings regarding prenatal

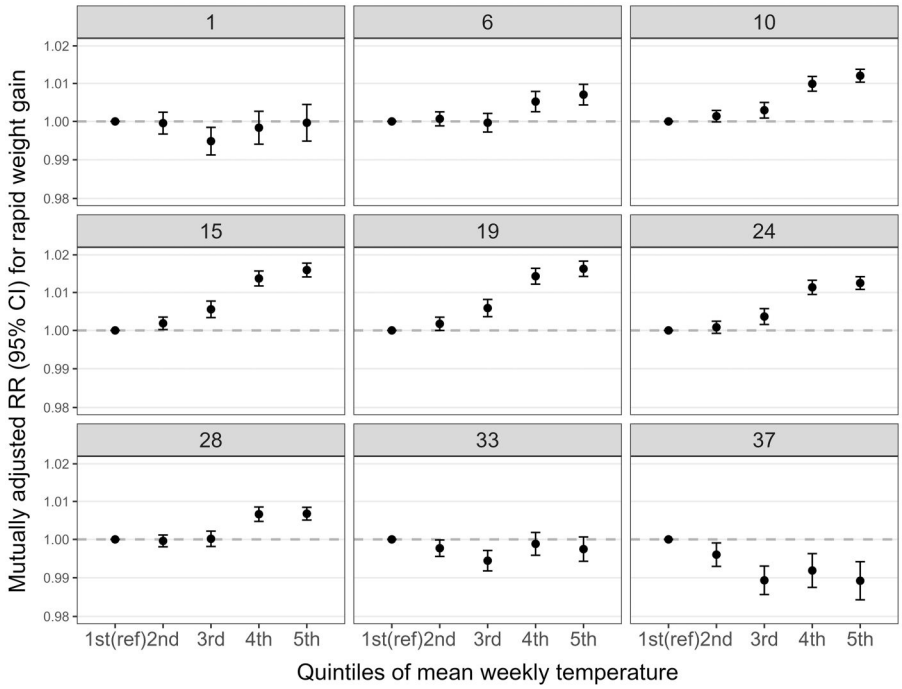

Figure 3. The association between quintiles of mean weekly temperature and rapid infant weight gain defined as the upper tertile of SITAR weight gain velocity. Associations are presented as the mutually adjusted relative risk (RR) with 95% confidence interval per quintile of weekly temperature compared to the first quintile at gestational weeks dispersed at equal intervals throughout pregnancy. Mutually adjusted models include socioeconomic index sextile, population group, subdistrict, year and month of birth as well as mean weekly postnatal temperature exposure. $n = 1\,100\,576$, Israel, 2011–2019

temperature exposure are consistent with prior literature. Our results regarding postnatal temperature exposure strengthen, refine and expand our earlier exploratory study, in which the average temperature at the clinic address during the first year of life was positively associated with rapid infant weight gain between birth and age 1 year.[23]

When exploring critical periods of risk using average trimester exposures, all three trimesters were associated with the outcome. However, distributed lag models with weekly exposures revealed a more nuanced picture,[38] suggesting a critical window for high temperatures during early and mid-pregnancy, while in late pregnancy the risk for rapid weight gain may be related to lower temperature. This finding is aligned with previous studies which have described an effect of both warm and cold temperatures on intrauterine growth.[16–18] When we focused solely on term-born and normal or higher birthweight infants, only warmer temperatures were highlighted as risk factors, consistent with prior literature.[14–16]

The average association per week of exposure during the first postnatal month was stronger than that arising from each gestational week, suggesting that exposure during infancy might be more potent. A similar finding was previously observed for air pollution exposure.[31] Although the postnatal window we had examined was short, there is evidence of its biological and clinical importance: some studies have shown that the critical period during infancy for rapid weight gain to potentially affect adiposity later in life might be as early as the first postnatal week.[44–46]

Several causal mechanisms, both biological and behavioural, might drive the association. Brown adipose tissue is a thermogenic tissue that uses energy to produce heat in order to maintain body temperature.[47] Its function is most important in the neonate, who is exposed to the cold conditions of the extrauterine environment for the first time; indeed, its activation and proliferation are induced by cold exposure.[47] Animal and human studies suggest a negative association between brown adipose tissue activity and body mass index.[48] Ambient temperature may influence practices such as air-conditioning or calefaction use, time spent outdoors or choice of infant clothing, which affect the actual temperature the infant is exposed to, and its subsequent biological effect. Temperature might also influence maternal health behaviour and parental feeding patterns which may affect subsequent weight gain.[49] Any behaviour prompted by ambient temperature should be considered part of the causal effects of temperature.[50]

Strengths

The major strength of our study lies in being based on a public health service that covers the entire Israeli infant population, benefiting from high-quality weight measurements taken according to a detailed procedural protocol. The considerable sample size enables high statistical power necessary for detecting weak associations from exposures as short as a week. Moreover, a large sample size with high power is essential in studies assessing the exposure based on residential address rather than personal exposure, since it partially compensates for attrition of power due to the inevitable random exposure error. The exposure was assessed using a high-resolution spatiotemporal model and the outcome was modelled based on all available weight measurements using the advanced SITAR method. Lastly, we used mutually adjusted distributed lag models to account for all exposure periods and detect critical ones.

Limitations

Our study has several limitations. Not all maternal and child health clinics in Israel are included in the source population,

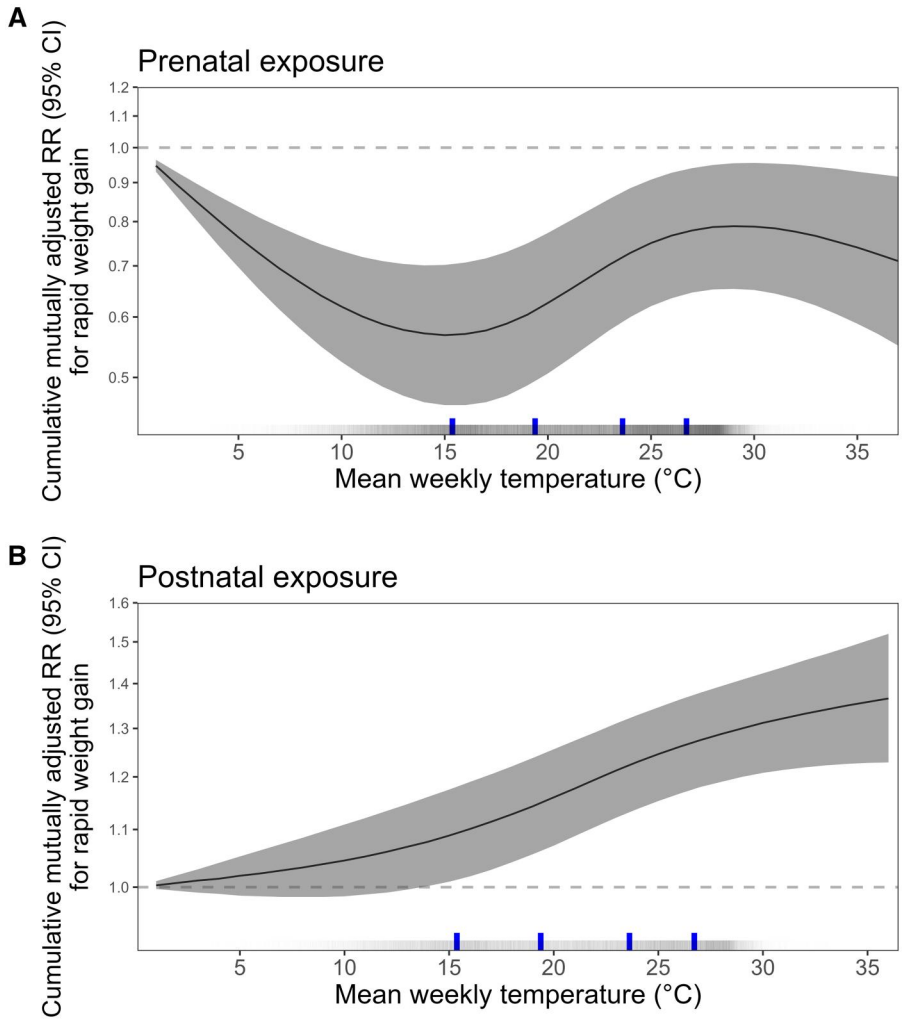

Figure 4. Cumulative association between prenatal or postnatal mean weekly ambient temperature and rapid infant weight gain defined as the upper tertile of SITAR weight gain velocity. Mutually adjusted associations are presented as the cumulative relative risk (RR) with 95% confidence interval during the entire exposure period per mean weekly ambient temperature compared to the minimum of 0 °C—(a) Prenatal and (b) Postnatal. Note different *Y* scales. Adjustments were made for socioeconomic index sextile, population group, subdistrict, year and month of birth as well as mean weekly temperature during the alternate period—prenatal or postnatal. Spikes indicate quintile cutoffs. Distribution of weekly temperature shown in bottom. $n = 1\ 100\ 576$, Israel, 2011–2019

leading to a coverage of approximately 80% of infants born during the study period. It is nevertheless unlikely that this had biased our estimations since all clinics provide similar services and virtually all Israeli children visit them during the first year of life.[51] A further limitation involves lack of information about address changes, which causes misclassification of the exposure. However, relocations are not expected to be differential with regards to the outcome and would therefore likely bias associations towards the null.[52]

The length of postnatal exposure we had examined was restricted to 4 weeks. This is because the SITAR growth velocity parameter peaks shortly after this point,[31] and longer exposure periods would have partially succeeded the outcome. It is possible that later temperature exposure also affects infant growth, but this should be assessed using a different method and in a way that does not defy the temporality principle when examining postnatal exposures and concurrent growth.

A further limitation arises from missing data about 15.1% of infants. However, the characteristics of infants included in the source population were not substantially different from those remaining in the cohort for analyses, thus reducing the plausibility of considerable selection bias. Live birth bias is another potential source of selection bias, but only for the prenatal temperature exposure analyses.[53] Lastly, like other studies, our analyses may suffer from residual confounding, for example, due to adjusting models for neighbourhood-based socioeconomic indices rather than personal measures.

Conclusion

Our findings suggest that overall, prenatal and postnatal exposures to higher ambient temperatures are associated with increased risk of rapid infant weight gain. Being a risk factor for later adiposity, there is need for additional evidence about the possible effect of temperature exposure on rapid infant growth. Future studies should examine various settings, implement specialized methods to evaluate whether these associations are likely to be causal, and focus on possible biological or behavioural mechanisms.

Ethics approval

The supreme ethics committee of the Israeli Ministry of Health approved the study.

Data availability

The authors are not permitted to share the data used in this study. Researchers interested in accessing the data may apply for data access to the Supreme Ethics Committee of the Israeli Ministry of Health.

Supplementary data

Supplementary data are available at *IJE* online.

Author contributions

All authors took part in planning the study. N.A. and I.Y. prepared and linked datasets and carried out analyses. D.N. and B.W. took part in data analysis. I.K. provided temperature data. M.H. and R.C.M. provided infant growth data. N. A. and R.R. prepared the initial drafts of the manuscript, and R.R. recruited funding and supervised the study. All authors gave feedback and reviewed the final manuscript.

Use of artificial intelligence (AI) tools

No AI tools were used in the process of conducting the study or preparing the manuscript.

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Conflict of interest

None declared.

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