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Maternal Stress and Early Childhood BMI Among US Children from the Environmental influences on Child Health Outcomes (ECHO) Program

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Author Contributions

CTW, CLB, MLC, and MM designed, analyzed, and interpreted the data and drafted the article. All authors revised and critically appraised the article for intellectual content and approve the final version.

Competing Interests Statement

No authors have conflicts of interest or competing interests relevant to this manuscript.

Consent Statement

Written informed consent was obtained for ECHO-wide Cohort Data Collection Protocol participation and for participation in specific cohorts.

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Abstract

Background: We aimed to understand the association between maternal stress in the first year of life and childhood body mass index (BMI) from 2 to 4 years of age in a large, prospective United States (U.S.)-based consortium of cohorts.

Methods: We used data from the Environmental influences on Child Health Outcomes (ECHO) program. The main exposure was maternal stress in the first year of life measured with the Perceived Stress Scale (PSS). The main outcome was the first childhood BMI percentile after age 2 until age 4 years. We used an adjusted linear mixed effects model to examine associations between BMI and PSS quartile.

Results: The mean BMI percentile in children was 59.8 (SD 30) measured at 3.0 years (SD 1) on average. In both crude models and models adjusted for maternal BMI, age, race, ethnicity, infant birth weight, and health insurance status, no linear associations were observed between maternal stress and child BMI.

Conclusions: Among 1694 maternal-infant dyads, we found no statistically significant relationships between maternal perceived stress in the first year of life and child BMI after 2 through 4 years.

INTRODUCTION

Obesity is a pervasive public health problem associated with significant costs and comorbidities.¹ Obesity problems and their comorbidities can start at an early age. A large number of children in the United States (U.S.) enter their school years already with obesity,² and children with obesity at 3 years of age have a 90% probability of having overweight or obesity as an adolescent.³ Despite a long list of well-documented risk factors for obesity that are present in the prenatal and early postnatal periods⁴ and an array of interventions

to reduce the incidence of obesity in early childhood,⁵ there remain significant gaps in understanding the mechanisms of obesity in children.

Maternal stress and stress reactivity play a role in childhood development and lifespan morbidity and mortality.⁶ Childhood obesity is one of the adverse health outcomes found to be associated with parental stress, yet the mechanism through which stress affects body mass index (BMI) in children is complex.^{7,8} The current body of literature shows that parental stress may determine parenting practices related to a child's diet quality, feeding behaviors, or sedentary behaviors.⁹ The majority of analyses assessing relationships between parental stress and obesity-related behaviors have been conducted in older, school-age children,⁷ and are cross-sectional in design. The psychosocial environment during infancy, of which one component is maternal stress, has been shown to affect adolescent BMI and intermediate cardiometabolic outcomes.¹⁰ However, there is a lack of longitudinal studies assessing maternal stress during infancy and subsequent childhood obesity.

Using a large U.S.-based sample, we analyzed the associations between maternal perceived stress and early childhood obesity. Given the existing literature that shows the influence of stress and psychosocial environment on obesity, we hypothesized that higher levels of maternal stress during a child's first year of postpartum life may be associated with higher BMI percentiles in early childhood, with differences by sex.

METHODS

Data were obtained from the Environmental influences on Child Health Outcomes (ECHO), a consortium of 69 pediatric cohort studies designed to investigate early life exposures and child health outcomes.^{11,12} The ECHO study protocol was approved by the local and/or single ECHO Institutional Review Board. Written informed consent was obtained for ECHO-wide Cohort Data Collection Protocol participation and for participation in specific cohorts. The present study population included biological mother-child dyads with available data on maternal perceived stress reported between the birth of the child and 1 year of age and child height and weight measured between 2 and 4 years of age. We restricted our population to singleton births and biological mothers with only one child enrolled in the ECHO-wide consortium. Our study sample consisted of 1694 biological mother-child dyads from 15 ECHO cohorts (Figure 1).

Maternal Perceived Stress

The Perceived Stress Scale (PSS) is a widely used self-report instrument that measures the perception of stress.¹³ Maternal stress scores were gathered using either the 14-item, 10-item, or 4-item PSS forms, each containing items rated on a 5-point Likert scale. We included any measure of stress prior to 1 year of age (mean 7.2 months, standard deviation [SD] 4.0) to focus on the exposure to stress during infancy and obesity at 2 years. Item response theory was used to harmonize the PSS to a T-score metric with a mean of 50 and an SD of 10. For mothers with multiple PSS observations, we selected the earliest observation between the birth of the child and 1 year of age, to focus on the earliest postnatal exposure to maternal stress. The PSS T-scores were further categorized into quartiles of the study population: 1st quartile [22.4, 33.5], 2nd quartile (33.5, 42.3], 3rd quartile (42.3, 50.8],

and 4th quartile (50.8, 82.0]. Additionally, continuous and binary PSS scores based on the median of the study population were investigated in sensitivity analyses.

Child Body Mass Index

Measurement of weight and length/height was completed according to ECHO guidelines: stadiometers and height adjustment rulers were used to measure standing height and scales with precision of ± 0.10 kilogram (kg) were used to measure weight. All units were converted to metric units (kg or centimeter [cm]). Weights and heights outside the allowable range of 0.126–250.00 kg or 25.00–250 cm were set to missing. The small number of participants ($n=13$) who had recumbent height measured after 2 years of age were measured via infantometer or length measuring board with 0.1 cm markings. All data were transferred/uploaded using ECHO Data collection forms. Children's BMI (weight in kg/height in meters [m] squared) was calculated using height and weight measurements from cohort study visits. Furthermore, BMI percentiles for age and sex were calculated based on U.S. Centers for Disease Control and Prevention (CDC) growth charts.¹⁴ Implausible values determined by established CDC cutoffs were excluded ($n=14$). When multiple BMI values were available, we used the average of BMI percentiles between 2 and 4 years of life for analyses.

Maternal and Child Characteristics

Biological mothers' pre-pregnancy height and weight were obtained by each cohort from measurements taken at the cohort study visit, medical record abstractions, or self-reported measurements. Per ECHO data harmonization, BMI was collected as early as 13 months prior to conception through the end of the first trimester, prioritizing the BMI closest to conception. These height and weight measurements were used to calculate pre-pregnancy BMI (kg/m^2). Child's birthweight (BW, kg), gestational age (GA) at birth (in completed weeks), and sex assigned at birth were obtained from medical records and/or parent report and were used to calculate child's BW-for-GA sex-specific z-score.¹⁵ Mother's age at delivery in years was calculated from maternal year of birth and child year of birth. Self-reported maternal race was categorized as Black, White, and Other Race. Other Race included American Indian/Native American, Asian, Native Hawaiian or Pacific Islander, Multiple Race, and Other Race. Ethnicity was defined as Hispanic or Non-Hispanic, and child sex at birth was defined as male, female, or ambiguous. Health insurance type was categorized as no health insurance; publicly subsidized insurance based on the income or disability of the biological mother, including Medicaid, Medical Assistance, CHIP, or any kind of state or government assistance plan; and private insurance, including employer-provided insurance, marketplace, TRICARE, VA, and Indian Health Services.

Statistical Analysis

Means and SDs were calculated for the continuous variables (child BMI percentile, maternal pre-pregnancy BMI, and maternal age at delivery) for the study population and each PSS quartile. The number of participants and percentages were reported for categorical variables (child sex, maternal ethnicity, maternal race, and insurance) for the study population and each PSS quartile. To evaluate unadjusted associations between PSS quartiles and continuous variables, one-way ANOVA tests were utilized. Pearson χ^2 tests were performed to determine the unadjusted associations between PSS quartiles and categorical variables.

We employed a linear mixed effects model to examine the adjusted associations between PSS quartiles and child BMI percentiles at 2–4 years of age with random intercepts for cohort membership.¹⁶ Analyses were adjusted for pre-pregnancy BMI (continuous), BW-for-GA z-score (continuous), maternal race and ethnicity, maternal age at delivery (continuous), and health insurance status. To investigate possible effect modification by child sex, we stratified the adjusted model by child sex. As only 6% of the infants in our sample were born prior to 37 weeks gestation, we elected not to stratify analyses by preterm status, however, we did run a sensitivity analysis excluding preterm infants from the sample. Missing data on maternal race (16%), ethnicity (13%), maternal age at delivery (6%), pre-pregnancy BMI (25%), health insurance status (21%), and BW-for-GA z-score (2%) were imputed using the Multivariate Imputation by Chained Imputation (MICE) package. Results were pooled after 10 iterations.¹⁷ All analyses were performed using the R statistical software package, version 4.1.0 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Table 1 shows the distribution of maternal and child characteristics for the study population and by PSS quartile (a higher PSS score and a higher quartile indicate higher perceived stress). Overall, the mean PSS T-score was 43.1 (SD 11, median 42.3). The mean BMI percentile for children 2–4 years of age was 59.8 (SD 30), and the mean age at BMI observation was 3.0 years (SD 1), from an average of 1.33 (SD 1) observations per child. The mean BW-for-GA z-score was –0.03 (SD 1), and 53% of the children were male. The majority (71%) of the mothers were of Non-Hispanic ethnicity. Overall, 28% were Black; 41% were White; and 15% identified as Other Races. Approximately 50% of mothers reported having no health insurance or having public insurance, and 9% reported less than a high school education. The mean pre-pregnancy BMI was 27.6 kg/m² (SD 7), and the mean maternal age at delivery was 28.9 years (SD 6).

Child BMI percentiles at 2–4 years of age were not significantly different across PSS quartiles (ANOVA $p=0.57$, Table 1). We found no differences in weight-for-age z-score changes in the first year of life and overall PSS quartiles (data not shown). However, significant associations were observed between PSS quartiles and maternal ethnicity, maternal race, and insurance status (chi-squared $p=0.01$, <0.001 , and 0.01 , respectively). The proportion of first quartile PSS scores reported by Hispanic mothers was 11%, which doubled to encompass 23% of fourth quartile scores. Similarly, the proportion of Black mothers increased as PSS quartiles increased. Those with public insurance comprised approximately 50% of each PSS quartile, while those with private insurance comprised a higher percentage (35%) of first quartile scores and a lower percentage (22%) of fourth quartile scores. No significant associations were found between PSS quartiles and child sex, BW-for-GA z-score, pre-pregnancy BMI, and maternal age at delivery.

No linear associations were observed between maternal PSS quartiles and child BMI percentiles at 2–4 years of age (Table 2). However, as expected, increases in pre-pregnancy BMI ($\beta=0.89$, 95% CI: 0.68, 1.09) and BW-for-GA z-scores ($\beta=3.91$, 95% CI: 2.59, 5.24) were significantly associated with increasing child BMI percentiles at 2–4 years of age. Additionally, children born to Hispanic mothers, compared with Non-Hispanic mothers, had

a higher BMI percentile at 2–4 years ($\beta=8.85$, 95% CI: 4.61, 13.10). No significant trends were observed in the child sex-stratified models, although a marginally significant difference was found between the 4th and 1st quartile among females.

As sensitivity analyses, the models were run using continuous PSS and binary PSS T-scores based on the median of the sample (43.2). No significant association was observed between continuous or binary maternal PSS T-scores assessed during infancy and obesity in early childhood. There was no significant variation in the outcome due to between-cohort variation (ICC 0.01 – 0.02; Supplementary Figures 1–3). Analyzing the association with overweight/obesity (i.e., BMI percentile $\geq 85\%$) in logistic regression did not change our results (data not shown). Additionally, exclusion of the 99 infants born preterm did not change our results (data not shown).

DISCUSSION

In a large, longitudinal sample of children from multiple diverse cohorts in the U.S., maternal perceived stress during infancy was not predictive of the child's BMI percentile at 2–4 years of age. Although secondary to our primary question, our analysis did suggest positive relationships between BW, maternal pre-pregnancy BMI, and Hispanic ethnicity with child BMI, which are consistent with the existing literature.^{18,19}

Cross-sectional studies have shown higher maternal stress is associated with higher child BMI, yet the longitudinal effects of maternal stress on child BMI remain less clear. One meta-analysis of studies assessing maternal stress and child obesity included 14 cross-sectional and five longitudinal studies across five countries. Although there was a significant, positive association between maternal stress and child obesity in pooled cross-sectional studies, the pooled effect size from five longitudinal studies was not statistically significant.⁸ Of the longitudinal analyses, three measured maternal stress when the child was 1–4 years of age with two showing statistically-significant effects between maternal stress and child obesity at 5 years of age.^{20,21} Two studies measured maternal stress during the first year of life, and neither study showed an association with BMI $>85\%$ in the first 18 months of life or at 7 years, respectively.^{22,23} These studies, consistent with our findings, suggest that maternal stress during infancy may not have consistent effects on child BMI over time.

Although we found no differences in sex-stratified models, a previous study using data from a German prospective birth cohort found that maternal perceived stress was associated with BMI in preschool-aged girls but not boys.²⁴ However, 79% of the parents in that cohort had no formal education beyond high school, whereas 37% in our sample had at least some college education. Another study that used a single-question assessment of stress in children with overweight or obesity enrolled in a randomized trial demonstrated significant associations between maternal stress and child BMI only when stratified by race, ethnicity, and household income.²⁵ Higher maternal stress was associated with higher child BMI only among Non-Hispanic Black children and in families with an annual household income less than \$50,000. Taken together, these findings^{24,25} suggest that sociodemographic factors may influence associations between maternal stress and offspring obesity.

A composite scale of perceived stress may not reflect phenomena that directly affect causal factors related to obesity, such as traumatic events²⁶ or structural racism. In one study, specific “stressors” of traffic, residential noise, and poor living conditions were significantly associated with overall maternal perceived stress, but specific stressors were not directly associated with child BMI. The field would benefit from a deeper investigation into the complexities of how perceived stress and specific stressors differ in their effects on offspring health, both for parents and their children. Some evidence suggests that maternal stress influences offspring BMI through multiple mechanisms, including feeding behaviors, maternal-infant attachment, and other diet-related factors.^{9,27,28}

Maternal stress has been associated with infant weight changes during the first year of life. In one analysis that independently assessed perceived stress during pregnancy, depressive symptoms, and exposure to stressful events, only exposure to stressful events was associated with a higher odds of rapid infant weight gain (greater than a 0.67 increase in weight-for-age z-score from birth to 6 months) in infants. The study did not include sufficient data to examine the mechanisms underlying rapid infant weight gain.²⁶ Another analysis of a longitudinal birth cohort examined relationships between maternal perceived stress at 3 months and rapid infancy weight gain, defined as greater than a 0.67 increase in the infant’s weight-for-age z-score between 3 and 12 months of age. Although rapid weight gain was highly prevalent in the sample (47% of infants), it was not associated with maternal stress.²⁹ When specific diet-related behaviors were examined in a population of women receiving assistance from the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC), higher maternal stress was associated with higher infant energy intake, earlier introduction of solid foods, and adding cereal to the infant’s bottle.²⁷

There is mixed evidence for relationships between timing of stress, infancy weight gain, and obesity, with considerable overlap between maternal mental health, stress, and parent perceptions of infant behavior. Stress prior to conception and birth may exert independent effects on subsequent child health. Prenatal stress and stress prior to conception have consistent associations with lower BW.³⁰ Higher pregnancy perceived stress has been shown to be predictive of preterm birth^{30,31} and lower birth weight,^{30,32} both of which have been associated with poor child health outcomes, including insulin resistance and type 2 diabetes.³³ Rapid infant weight gain is more common among infants born at lower BWs as regression to the mean and “catch up” growth are well-described phenomena; however, the interplay of GA, weight and length changes, and actual measured adiposity remains an area in need of further study.^{34,35}

Our analysis has some limitations, including only using maternal, not paternal or other caregiver, perceived stress and combining perceived stress measures throughout the first year of life. It is possible that perceived stress changes throughout the first year, such that pooling perceived stress across the first year could obscure significant relationships with more specific periods of time during infancy. However, others have found that perceived stress is stable across the first year of life.²⁹ Although our treatment of the perceived stress measures may not be directly comparable to other studies that use the raw scores of the PSS, use of item response theory during harmonization allows us to assess a large sample while maintaining validity. The first quartile of our T-scores is roughly equivalent to a PSS-10

sum score of 14, which is similar to a “low stress/high stress” cutoff reported previously.³⁶ We did not assess differences related to preterm birth, as only 6% of infants in our sample were born preterm. We excluded multiple-gestation births, understanding that the home environment when caring for more than one infant may be significantly different in terms of parental stress; however, we know this particular population deserves focus. In our sample, excluding multiple gestations only reduced our analytic sample by 23 dyads. Excluding dyads with complete data necessary for our analyses may have biased our results toward the null if mothers with higher levels of stress or mothers with children more likely to have elevated BMI were also more likely to have incomplete data or dropped out of cohorts. In particular, excluding sibling pairs and multiple-gestation pregnancies may have biased our results toward the null if there is a true relationship between higher maternal stress and higher BMI and if multiple infants and young children are related to higher maternal stress. We expected that earlier exposure to higher stress would influence the earliest possible categorization of obesity (2 years); however, it is possible that the effects of stress may not be evident until later ages.⁸ The complex nature of multiple exposures over time should be explored in samples with repeated measures of stress, either as composite scales or specific stressors.

Although we found no significant associations between quartiles of maternal stress and childhood BMI, our analysis benefited from a large sample with maternal stress collected across the first year of life that preceded the child’s BMI after 2 years of age. Our large, longitudinal sample and the ability to capture BMI as early as 2 years of age complements the existing literature, much of which has only examined cross-sectional relationships.⁸ Given that 90% of children with obesity at 3 years of age go on to have overweight or obesity in adolescence,³ it is critical to examine modifiable risk factors within the early postnatal years. However, it will be important to continue to examine child BMI within the ECHO consortium as children age. Additionally, ongoing examination of the mechanisms of stress, including the timing of maternal stress, and the influence of specific traumatic events or isolated exposures on childhood growth, feeding, and the child’s own stress,³⁷ will continue to highlight areas for intervention to prevent obesity and improve intergenerational health.

CONCLUSION

Using data from 1694 maternal-infant dyads among 15 cohorts in the ECHO program, we found no statistically significant relationships between maternal perceived stress ascertained in the first year of the infant’s life and child BMI between 2 and 4 years of age despite adjusting for maternal age and pre-pregnancy BMI, BW-for-GA z-score, maternal race, maternal ethnicity, and health insurance. Given the mixed evidence that parental stress affects child obesity-related behaviors or child BMI, subsequent investigation should measure effects of stress on BMI as children age and distinguish the influence of specific stressors and of sociodemographic factors from global measures of perceived stress, focusing on modifiable components that influence childhood obesity.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability Statement

De-identified data from the ECHO Program are available through NICHD’s Data and Specimen Hub (DASH) (<https://dash.nichd.nih.gov>). DASH is a centralized resource that allows researchers to access data from various studies via a controlled-access mechanism. Researchers can now request access to these data by creating a DASH account and submitting a Data Request Form. The NICHD DASH Data Access Committee will review the request and provide a response in approximately two to three weeks. Once granted access, researchers will be able to use the data for three years. See the DASH Tutorial for more detailed information on the process (<https://dash.nichd.nih.gov/resource/tutorial>).

REFERENCES

1. Finkelstein EA, Trogon JG, Cohen JW & Dietz W Annual Medical Spending Attributable to Obesity: Payer-and Service-Specific Estimates. *Health Aff (Millwood)* 28, w822–831 (2009). [PubMed: 19635784]
2. Cunningham SA, Kramer MR & Narayan KM Incidence of Childhood Obesity in the United States. *N Engl J Med* 370, 403–411 (2014). [PubMed: 24476431]

3. Geserick M et al. Acceleration of Bmi in Early Childhood and Risk of Sustained Obesity. *N Engl J Med* 379, 1303–1312 (2018). [PubMed: 30281992]
4. Woo Baidal JA et al. Risk Factors for Childhood Obesity in the First 1,000 Days: A Systematic Review. *Am J Prev Med* 50, 761–779 (2016). [PubMed: 26916261]
5. Johnson BJ et al. Unpacking the Behavioural Components and Delivery Features of Early Childhood Obesity Prevention Interventions in the Topchild Collaboration: A Systematic Review and Intervention Coding Protocol. *BMJ Open* 12, e048165 (2022).
6. Shonkoff JP et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. *Pediatrics* 129, e232–e246 (2012). [PubMed: 22201156]
7. Jang M, Owen B & Lauver DR Different Types of Parental Stress and Childhood Obesity: A Systematic Review of Observational Studies. *Obes Rev* 20, 1740–1758 (2019). [PubMed: 31475448]
8. Tate EB, Wood W, Liao Y & Dunton GF Do Stressed Mothers Have Heavier Children? A Meta-Analysis on the Relationship between Maternal Stress and Child Body Mass Index. *Obes Rev* 16, 351–361 (2015). [PubMed: 25879393]
9. O'Connor SG et al. Associations of Maternal Stress with Children's Weight-Related Behaviours: A Systematic Literature Review. *Obes Rev* 18, 514–525 (2017). [PubMed: 28296057]
10. Doom JR et al. Infant Psychosocial Environment Predicts Adolescent Cardiometabolic Risk: A Prospective Study. *The Journal of Pediatrics* 209, 85–91.e81 (2019). [PubMed: 30876752]
11. Romano ME, Buckley JP, Elliott AJ, Johnson CC & Paneth N Spr Perspectives: Scientific Opportunities in the Environmental Influences on Child Health Outcomes Program. *Pediatr Res*, 1–7 (2021).
12. Blaisdell CJ et al. The Nih Echo Program: Investigating How Early Environmental Influences Affect Child Health. *Pediatr Res*, 1–2 (2021).
13. Cohen S, Kamarck T & Mermelstein R A Global Measure of Perceived Stress. *J Health Soc Behav* 24, 385–396 (1983). [PubMed: 6668417]
14. Kuczumarski RJ Cdc Growth Charts: United States (US Department of Health and Human Services, Centers for Disease Control and ..., 2000).
15. Aris IM, Kleinman KP, Belfort MB, Kaimal A & Oken EA 2017 Us Reference for Singleton Birth Weight Percentiles Using Obstetric Estimates of Gestation. *Pediatrics* 144 (2019).
16. Bates D, Mächler M, Bolker B & Walker S Fitting Linear Mixed-Effects Models Using Lme4. arXiv preprint arXiv:14065823 (2014).
17. Van Buuren S & Groothuis-Oudshoorn K Mice: Multivariate Imputation by Chained Equations in R. *Journal of statistical software* 45, 1–67 (2011).
18. Taveras EM, Gillman MW, Kleinman KP, Rich-Edwards JW & Rifas-Shiman SL Reducing Racial/Ethnic Disparities in Childhood Obesity: The Role of Early Life Risk Factors. *JAMA Pediatr* 167, 731–738 (2013). [PubMed: 23733179]
19. Woo Baidal JA et al. Risk Factors for Childhood Obesity in the First 1,000 Days: A Systematic Review. *Am J Prev Med* 50, 761–779 (2016). [PubMed: 26916261]
20. Koch FS, Sepa A & Ludvigsson J Psychological Stress and Obesity. *J Pediatr* 153, 839–844 (2008). [PubMed: 18657829]
21. Suglia SF, Duarte CS, Chambers EC & Boynton-Jarrett R Cumulative Social Risk and Obesity in Early Childhood. *Pediatrics* 129, e1173–1179 (2012). [PubMed: 22508921]
22. Ajslev TA, Andersen CS, Ingstrup KG, Nohr EA & Sørensen TI Maternal Postpartum Distress and Childhood Overweight. *PLoS One* 5, e11136 (2010). [PubMed: 20614031]
23. Sowan NA & Stember ML Parental Risk Factors for Infant Obesity. *MCN Am J Matern Child Nurs* 25, 234–240; quiz 241 (2000). [PubMed: 10992735]
24. Leppert B et al. Early Maternal Perceived Stress and Children's Bmi: Longitudinal Impact and Influencing Factors. *BMC Public Health* 18, 1211 (2018). [PubMed: 30376822]
25. Baskind MJ et al. Parent-Perceived Stress and Its Association with Children's Weight and Obesity-Related Behaviors. *Prev Chronic Dis* 16, E39 (2019). [PubMed: 30925139]
26. Felder JN et al. Prenatal Maternal Objective and Subjective Stress Exposures and Rapid Infant Weight Gain. *J Pediatr* 222, 45–51 (2020). [PubMed: 32418816]

27. Hurley KM, Black MM, Merry BC & Caulfield LE Maternal Mental Health and Infant Dietary Patterns in a Statewide Sample of Maryland Wic Participants. *Maternal and Child Nutrition* 11, 229–239 (2015).
28. Anderson SE, Lemeshow S & Whitaker RC Maternal-Infant Relationship Quality and Risk of Obesity at Age 5.5 Years in a National Us Cohort. *BMC Pediatr* 14, 54 (2014). [PubMed: 24564412]
29. Fox K et al. Maternal Stress and Excessive Weight Gain in Infancy. *Int J Environ Res Public Health* 19 (2022).
30. Zhu P, Tao F, Hao J, Sun Y & Jiang X Prenatal Life Events Stress: Implications for Preterm Birth and Infant Birthweight. *Am J Obstet Gynecol* 203, 34.e31–38 (2010).
31. Kornfield SL, Riis VM, McCarthy C, Elovitz MA & Burris HH Maternal Perceived Stress and the Increased Risk of Preterm Birth in a Majority Non-Hispanic Black Pregnancy Cohort. *J Perinatol* 42, 708–713 (2022). [PubMed: 34400775]
32. Khashan AS et al. Second-Trimester Maternal Distress Increases the Risk of Small for Gestational Age. *Psychol Med* 44, 2799–2810 (2014). [PubMed: 25066370]
33. Martín-Calvo N, Goni L, Tur JA & Martínez JA Low Birth Weight and Small for Gestational Age Are Associated with Complications of Childhood and Adolescence Obesity: Systematic Review and Meta-Analysis. *Obes Rev* 23 Suppl 1, e13380 (2022). [PubMed: 34786817]
34. Lyons-Reid J, Albert BB, Kenealy T & Cutfield WS Birth Size and Rapid Infant Weight Gain- Where Does the Obesity Risk lie? *J Pediatr* 230, 238–243 (2021). [PubMed: 33157072]
35. Regnault N & Gillman MW Importance of Characterizing Growth Trajectories. *Ann Nutr Metab* 65, 110–113 (2014). [PubMed: 25413648]
36. Ling J, Xu D, Robbins LB & Meyer JS Does Hair Cortisol Really Reflect Perceived Stress? Findings from Low-Income Mother-Preschooler Dyads. *Psychoneuroendocrinology* 111, 104478 (2020). [PubMed: 31704637]
37. Dunton GF, Chu D, Naya CH, Belcher BR & Mason TB Longitudinal Associations of Maternal Stress and Child Stress with Child Body Mass Index Trajectory. *Pediatr Obes* 16, e12724 (2021). [PubMed: 32881324]

IMPACT

- Although existing literature suggests relationships between parental stress and childhood BMI, we found no linear associations between maternal stress in the first year of life and childhood BMI at 2 to 4 years of age among participants in ECHO cohorts.
- Higher maternal stress was significantly associated with Hispanic ethnicity, Black race, and public health insurance.
- Our analysis of a large, nationally representative sample challenges assumptions that maternal stress in the first year of life, as measured by a widely used scale, is associated with offspring BMI.

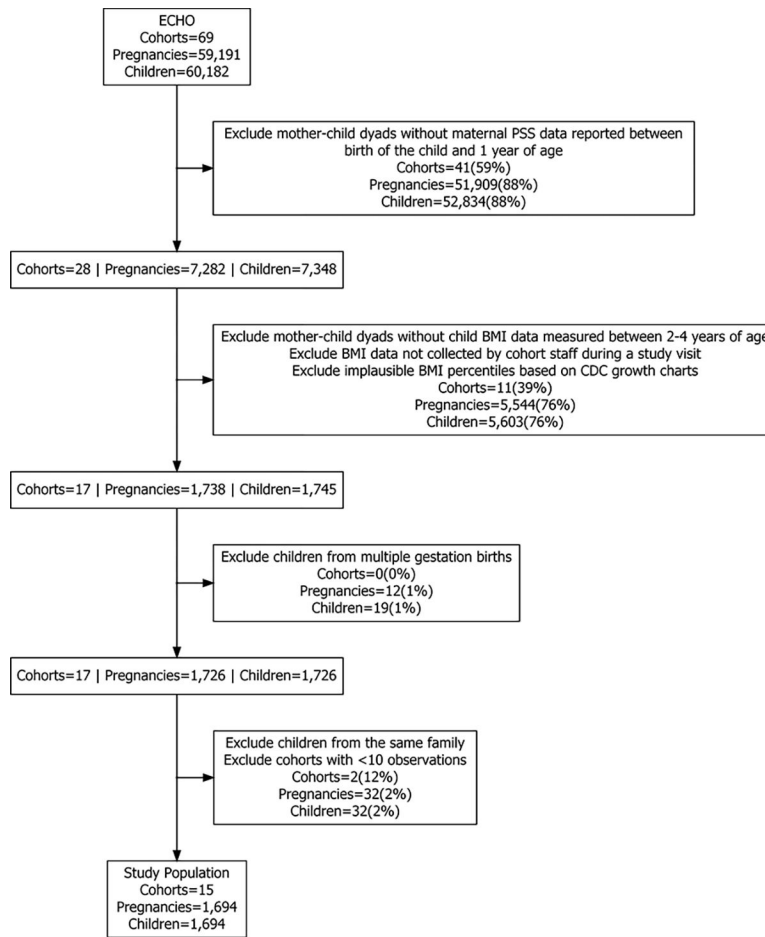


Figure 1.
ECHO cohorts inclusion in the analytical data set

Table 1.

Descriptive characteristics by maternal perceived stress T-score

	Perceived stress score (T-score)				Total (N=1,694)	P-value
	1st Quartile [22.4, 33.5] (n=447)	2nd Quartile (33.5, 42.3] (n=405)	3rd Quartile (42.3, 50.8] (n=419)	4th Quartile (50.8, 82] (n=423)		
Child BMI percentile, mean (SD)	61.2 (28)	60.1 (30)	58.8 (30)	58.8 (30)	59.8 (30)	0.57
Number of BMI observations per Child, mean (SD)	1.30 (1)	1.31 (1)	1.32 (1)	1.37 (1)	1.33 (1)	0.25
Child sex, N (%)						0.19
Male	236 (53%)	222 (55%)	233 (56%)	206 (49%)	897 (53%)	
Female	211 (47%)	183 (45%)	186 (44%)	217 (51%)	797 (47%)	
Maternal ethnicity, N (%)						0.01
Non-Hispanic	296 (66%)	286 (71%)	311 (74%)	304 (72%)	1,197 (71%)	
Hispanic	48 (11%)	65 (16%)	69 (16%)	96 (23%)	278 (16%)	
Missing	103 (23%)	54 (13%)	39 (9%)	23 (5%)	219 (13%)	
Maternal race, N (%)						<0.001
White	189 (42%)	182 (45%)	173 (41%)	148 (35%)	692 (41%)	
Black	94 (21%)	110 (27%)	135 (32%)	138 (33%)	477 (28%)	
Other Race	54 (12%)	48 (12%)	55 (13%)	94 (22%)	251 (15%)	
Missing	110 (25%)	65 (16%)	56 (13%)	43 (10%)	274 (16%)	
Maternal education, N (%)						0.39
Less than high school	21 (5%)	39 (10%)	36 (9%)	56 (13%)	152 (9%)	
High school degree or equivalent	41 (9%)	48 (12%)	54 (13%)	67 (16%)	210 (12%)	
Some college and above ^a	102 (23%)	164 (40%)	180 (43%)	183 (43%)	629 (37%)	
Missing	283 (63%)	154 (38%)	149 (36%)	117 (28%)	703 (41%)	
Insurance, N (%)						0.01
Private/Other ^b	158 (35%)	109 (27%)	104 (25%)	92 (22%)	463 (27%)	
Public/No ^c	223 (50%)	213 (53%)	223 (53%)	217 (51%)	876 (52%)	
Missing	66 (15%)	83 (20%)	92 (22%)	114 (27%)	355 (21%)	
Parity						0.32
Nulliparous	49 (11%)	84 (21%)	111 (26%)	111 (26%)	355 (21%)	
Parous	65 (15%)	97 (24%)	99 (24%)	128 (30%)	389 (23%)	
Missing	333 (74%)	224 (55%)	209 (50%)	184 (43%)	950 (56%)	
Birthweight-for-gestational age z-score, mean (SD)	0.04 (1)	0.02 (1)	-0.04 (1)	-0.13 (1)	-0.03 (1)	0.11
Missing, N (%)	8 (2%)	9 (2%)	6 (1%)	8 (2%)	31 (2%)	
Maternal pre-pregnancy BMI, mean (SD), kg/m ²	27.4 (8)	27.7 (7)	27.4 (7)	28.0 (8)	27.6 (7)	0.71
Missing, N (%)	74 (17%)	104 (26%)	111 (26%)	127 (30%)	416 (25%)	
Maternal age at delivery, years (SD)	28.5 (5)	29.2 (6)	28.8 (6)	29.1 (6)	28.9 (6)	0.32

	Perceived stress score (T-score)				Total (N=1,694)	P-value
	1st Quartile [22.4, 33.5] (n=447)	2nd Quartile (33.5, 42.3] (n=405)	3rd Quartile (42.3, 50.8] (n=419)	4th Quartile (50.8, 82] (n=423)		
Missing, N (%)	53 (12%)	21 (5%)	18 (4%)	14 (3%)	106 (6%)	

^aIncludes some college with no degree, associate's degrees, trade school, or higher levels of education (bachelor's, master's, or doctoral degrees).

^bAny type of private insurance for the biological mother, including employment-based, marketplace, TRICARE, VA, and HIS (Indian Health Services).

^cIncludes insurance coverage through Medicaid, Medical Assistance, CHIP, or any kind of state or government assistance plan based on income or disability of the biological mother or child.

BMI, body mass index; SD, standard deviation.

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Table 2.

Maternal perceived stress and child mean BMI percentile

	Crude (n=1694) β coefficient (95% CI) ^a	Fully Adjusted ^b (n=1694) β coefficient (95% CI)	Sex-Stratified - Male (n=897) β coefficient (95% CI)	Sex-Stratified - Female (n=797) β coefficient (95% CI)
Intercept	59.36 (55.52, 63.19)	36.46 (24.45, 48.48)	33.29 (16.57, 50.01)	43.62 (28.39, 58.85)
PSS 1st quartile	REF	REF	REF	REF
PSS 2nd quartile	-0.62 (-4.65, 3.41)	-1.27 (-5.14, 2.60)	-1.27 (-6.61, 4.08)	-2.29 (-7.88, 3.30)
PSS 3rd quartile	-1.82 (-5.85, 2.22)	-1.78 (-5.67, 2.12)	-1.62 (-6.91, 3.68)	-2.87 (-8.44, 2.71)
PSS 4th quartile	-1.27 (-5.40, 2.86)	-2.12 (-6.16, 1.91)	-0.78 (-6.4, 4.85)	-5.11 (-10.5, 0.27)
Maternal pre-pregnancy BMI		0.89 (0.68, 1.09)**	0.86 (0.58, 1.15)**	0.91 (0.61, 1.22)**
BW-for-GA z-score		3.91 (2.59, 5.24)**	3.79 (1.96, 5.62)**	4.12 (2.23, 6.01)**
Maternal race				
White		REF	REF	REF
Black		-1.86 (-6.02, 2.3)	0.04 (-0.38, 0.45)	-0.33 (-0.71, 0.05)
Other Race		-0.39 (-4.77, 4.00)	-1.47 (-7.35, 4.41)	-2.41 (-7.48, 2.66)
Maternal ethnicity				
Non-Hispanic		REF	REF	REF
Hispanic		8.85 (4.61, 13.10)**	-1.2 (-7.13, 4.73)	0.55 (-6.01, 7.12)
Maternal age at delivery		-0.11 (-0.40, 0.18)	5.84 (0.05, 11.64)*	12.82 (6.82, 18.82)**
Health insurance				
Private/Other		REF	REF	REF
Public/No		1.79 (-2.37, 5.96)	1.99 (-3.58, 7.56)	2.13 (-3.33, 7.59)

^aData are beta coefficients from linear mixed effects regression models reflecting change in child mean BMI percentiles.

^bModel adjusted for pre-pregnancy BMI (continuous), BW-for-GA z-score (continuous), maternal race, maternal ethnicity, maternal age at delivery, health insurance, with random intercept for ECHO cohort membership.

* p<0.05,

** p<0.01

PSS, Perceived Stress Scale; BMI, body mass index; BW, birthweight; GA, gestational age