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**Maternal history of child abuse and obesity risk in offspring:
mediation by weight in pregnancy**

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ABSTRACT

Background. Women's experience of childhood adversity may contribute to their children's risk of obesity. Possible causal pathways include higher maternal weight and gestational weight gain, which have been associated with both maternal childhood adversity and obesity in offspring.

Methods. This study included 6718 mother-child pairs from the National Longitudinal Survey of Youth 1979 in the United States (1979-2012). We applied multiple log-binomial regression models to estimate associations between three markers of childhood adversity (physical abuse, household alcoholism, and household mental illness) and offspring obesity in childhood. We estimated natural direct effects to evaluate mediation by pre-pregnancy body mass index (BMI) and gestational weight gain.

Results. Among every 100 mothers who reported physical abuse in childhood, there were 3.7 (95% CI: -0.1, 7.5) excess cases of obesity in 2- to 5-year-olds compared with mothers who did not report physical abuse. Differences in pre-pregnancy BMI, but not gestational weight gain, accounted for 25.7% of these excess cases. There was no evidence of a similar relationship for household alcoholism or mental illness or for obesity in older children.

Conclusions. In this national, prospective cohort study, pre-pregnancy BMI partially explained an association between maternal physical abuse in childhood and obesity in preschool-age children. These findings underscore the importance of life-course exposures in the etiology of child obesity and the potential multi-generational consequences of child abuse. Research is needed to determine if screening for childhood abuse and treatment of its sequelae could strengthen efforts to prevent obesity in mothers and their children.

MeSH Manuscript Keywords: adult survivors of child adverse events; child abuse; child, preschool; obesity/etiology; pediatric obesity; physical abuse

INTRODUCTION

The prevalence of obesity in American children and adolescents has not improved over the past decade, affecting approximately one in six (1). Public health efforts to prevent child obesity have largely targeted exposures in childhood, but knowledge on how to prevent obesity in early childhood is limited (2). Evidence increasingly suggests that there are intergenerational causes of child obesity (3). Pre-pregnancy obesity and excessive gestational weight gain, in particular, have been consistently linked with obesity in children (4, 5). In numerous additional studies, researchers have also found adverse childhood experiences (ACE) to be associated with obesity in adulthood (6-9). Most recently, adversity in childhood has been associated with high pre-pregnancy body mass index (BMI) and excessive weight gain during pregnancy (10-12) as well as offspring obesity (13).

We sought to bridge the bodies of evidence connecting childhood adversity with maternal obesity and maternal obesity with child obesity (4, 5, 9). Findings suggest that childhood adversity may cause long-term emotional dysregulation and depression that can lead to unhealthy eating behaviors and obesity in adulthood (14-16). In turn, obesity and excessive weight gain in pregnant women may contribute to obesity in their children (4, 5). If maternal adversity in childhood does contribute to offspring obesity by causing high maternal weight and weight gain, prevention of abuse, neglect, or adversity in children and screening for ACE with treatment of its psychological consequences in women could potentially help efforts to prevent child obesity. We used 33 years of follow-up data from a diverse, national cohort study to investigate whether women's experience of childhood adversity is associated with their children's risk of obesity, and, if so, whether pre-pregnancy BMI and gestational weight gain partially mediate the association.

METHODS

Study population

The National Longitudinal Survey of Youth 1979 (NLSY79) is an ongoing, national, prospective cohort study designed to represent the U.S. population of youths aged 14-21 years old in 1979. The Bureau of Labor Statistics enrolled 12,686 youths in the study in 1979 and began enrolling the biological children of female participants into the NLSY Child and Young Adult study in 1986. Participants were visited in person and/or received questionnaires annually until 1994 and every two years thereafter. As shown in Figure 1, 11,512 mother-child pairs (4932 individual mothers) have participated in the studies as of 2012. A total of 3191 mothers with 7894 children participated in the 2012 survey wave (69% of the original sample), which is not an uncommon participation proportion for prospective cohort studies (17, 18). Custom sampling weights created by the Bureau of Labor Statistics are used to weight the sample to match the demographics of the original target population despite differential loss to follow-up. We included mother-child pairs in the final study sample if the child was delivered as a singleton during or after 1979 and data were available on ACE and child BMI (n = 6718). The University of California, Berkeley Committee for the Protection of Human Subjects approved the study.

Measures

Maternal Measures

In the 2012 wave of data collection, the survey administered three ACE questions, which were selected from the 11-item US Behavioral Risk Factor Surveillance System (BRFSS) questionnaire based on prioritization by a panel of child development specialists (19). We were unable to use the full set of BRFSS ACE questions because of questionnaire length limitations. Participants were asked to recall their experience of physical abuse, household alcohol abuse, and household mental illness before age 18 years with the following questions:

1. How often did a parent or adult in your home ever hit, beat, kick, or physically harm you in any way? Do not include spanking. Would you say never, once, or more than once?
2. Did you live with anyone who was a problem drinker or alcoholic?

3. Did you live with anyone who was depressed, mentally ill, or suicidal?

Ninety-nine percent of the respondents in 2012 answered the ACE questions. We dichotomized the history of physical abuse responses as ‘yes’ if abuse was reported as two or more times and ‘no’ otherwise to capture recurrent physical abuse.

The NLSY79 collected data on weight and height at each wave beginning in 1981. Pre-pregnancy weight and delivery weight were self-reported in the first survey postpartum starting in 1986. Specifically, within 2 years of delivery, women reported their weight “just before [they] became pregnant” and “just before [they] delivered.” We regression-calibrated height data using error data from the National Health and Nutrition Examination Survey. The reliability of recalled pre-pregnancy weight and the weight reported at the closest survey prior to that pregnancy was 0.9.(10) Pre-pregnancy body mass index (BMI) was calculated from pre-pregnancy weight and height (kg/m^2). Gestational weight gain was defined as the difference between delivery weight and pre-pregnancy weight. We converted gestational weight gain into weight gain-for-gestational age z-scores using pre-pregnancy BMI-specific z-score charts previously created by our team using serial prenatal weight measurements from women in Pennsylvania with healthy, term, singleton births (20, 21). Use of the z-scores accounts for the inherent link between duration of pregnancy and weight gain during pregnancy (22, 23). Pre-pregnancy BMI and gestational weight gain were tested as mediators of any associations between maternal ACE and child obesity (4, 10, 11, 13).

Confounding maternal characteristics were selected using theory-based causal diagrams based on prior evidence (Supplemental Figure 1) (24). Confounding variables included early life characteristics (race/ethnicity, born in the U.S., born in the U.S. South, urban/non-urban childhood home at age 14, and maternal grandmother’s education level) (3, 13) and prenatal characteristics (age, education level, employment status, household income in year 2000 dollars

and equivalized for household size, race/ethnicity, marital status, parity, and smoked cigarettes during pregnancy) (4, 25).

Child Measures

Child BMI values were calculated using weight and height measurements that were either made in person by trained study interviewers (74% of weight and 82% of height measurements) or reported by the mother. We used the CDC growth chart SAS program to calculate age- and sex-specific BMI percentiles and identify biologically implausible values for exclusion (26). We assessed if a child was ever obese (BMI \geq 95th percentile) in early, middle, or late childhood (ages 2-5 years, 6-11 years, and 12-19 years, respectively), in accord with the age groups used by national child BMI surveillance methods (1).

Statistical Analysis

All statistical analyses accounted for the complex sampling design ('survey' package in R) (27) of the NLSY studies and family-level clustering. We first characterized the study sample and compared all variables between included, ineligible, and excluded mother-child pairs. The distributions of covariates were also compared across outcome groups to detect any patterns in the missingness of child BMI information. Multiple imputation with chained equations ('mice' package in R) (28) was used for missing covariate data (29). The percentage of missing observations for each covariate ranged from 0 to 15%, with a median of 3%.

To test our hypothesis, we first estimated the total effect of each maternal ACE exposure (physical abuse, household alcohol abuse, and household mental illness) on child obesity in each age group (2-5 y, 6-11 y, and 12-19 y) using log-binomial regression models, unadjusted and then adjusted for maternal early life characteristics. Because there was an overall association observed between maternal history of physical abuse and early childhood obesity in 2-5 year olds only, we limited further assessment of mediation by pre-pregnancy BMI and gestational weight gain to this age group. We followed the methods of Petersen et al. (30) to assess mediation, which we

describe step-by-step in Appendix A. In short, we applied the parametric g-formula to estimate the effect of childhood physical abuse in mothers on early childhood obesity in offspring if pre-pregnancy BMI and/or gestational weight gain were unrelated to childhood physical abuse. This estimate, the natural direct effect, assumes the maternal weight values to be those mothers would have attained in the absence of physical abuse. We adjusted these analyses for the covariates described previously. Statistical analyses were conducted in R version 3.1.1 (31).

Sensitivity Analyses

We pre-specified several sensitivity analyses to determine the robustness of the main results to methodological changes. (1) We added interaction terms between physical abuse and each mediator to models to detect effect modification. (2) We tested joint mediation by pre-pregnancy BMI and gestational weight gain. (3) We changed the outcome to childhood overweight or obesity (BMI \geq 85th percentile for age and sex). (4) We evaluated mediation by estimating controlled direct effects instead of natural direct effects. Natural direct effects provide estimates of the effect of maternal physical abuse on early childhood obesity if one were to intervene to prevent maternal physical abuse, and thereby change pre-pregnancy BMI or gestational weight gain. In contrast, controlled direct effects estimate the effect of maternal physical abuse on early childhood obesity if one were to intervene directly on pre-pregnancy BMI or gestational weight gain, setting them to pre-specified values.

RESULTS

The mothers of 17.3% of children in the study reported physical abuse in childhood, 11.0% reported living with someone in their childhood home who was mentally ill, and 22.2%

reported living with someone who was a problem drinker or alcoholic. Overall, mothers were mostly non-Hispanic white, born in the U.S., and had at least a high school education (Table 1). Compared to other mothers in the sample, a higher proportion of those who reported physical abuse as children were born to a mother who did not complete high school, did not complete high school themselves, had a low household income, and smoked during pregnancy. Consistent with previous findings in this sample (10), pre-pregnancy obesity and high gestational weight gain were more prevalent in women who experienced physical abuse, alcohol abuse in the household, and mental illness in the household, with the strongest association being between physical abuse and pre-pregnancy obesity. Compared to mother-child pairs in the final sample, those ineligible for the study (non-singleton birth, delivery before 1979, or not included in the 2012 survey) and those excluded for missing exposure or outcome data delivered in earlier years at younger ages and had lower prevalences of pre-pregnancy obesity and child obesity (Supplemental Table 1). Ineligible mother-child pairs reported a higher prevalence of physical abuse (21.3%) and alcohol abuse (25.7%), but a lower prevalence of household mental illness (7.8%) than those included in the final sample. There were no patterns detected in the missingness of child BMI information among the age groups.

Physical abuse in a woman's childhood was associated with a higher risk of her child being obese 2-5 years old (Figure 2, panels A and B). After adjusting for maternal early life characteristics, children 2-5 years old with mothers who reported childhood physical abuse were 21% more likely to be obese. Among every 100 mothers who were physically abused, there were 3.7 excess cases of early childhood obesity compared with mothers who were not physically abused. The predicted probability of early childhood obesity was 0.21 (95% CI: 0.18, 0.25) if women experienced physical abuse and 0.17 (95% CI: 0.16, 0.19) if women did not experience physical abuse.

After accounting for mediation by pre-pregnancy BMI, the association between maternal physical abuse and early childhood obesity was attenuated (Figure 2, panel C). Differences in pre-pregnancy BMI between physically abused and not physically abused mothers accounted for 25.7% of the excess cases of early childhood obesity that occurred among mothers who reported physical abuse. Accounting for mediation by gestational weight gain, without changing pre-pregnancy BMI, minimally attenuated the association between maternal physical abuse and early childhood obesity (Figure 2, panel D). Differences in gestational weight gain between mothers who reported physical abuse and those who did not were estimated to account for 5.7% of the excess cases of child obesity among mothers who reported physical abuse.

In the sensitivity analyses, adding multiplicative interaction terms between physical abuse and the mediators to the models did not change the results (Supplemental Table 2). Additionally, joint mediation by pre-pregnancy BMI and gestational weight gain resulted in slightly smaller effect estimates as for mediation by pre-pregnancy BMI alone (Supplemental Table 2). Using child overweight or obesity (BMI percentile $\geq 85^{\text{th}}$ percentile) as the outcome instead of obesity (BMI percentile $\geq 95^{\text{th}}$ percentile) yielded attenuated effect estimates (Supplemental Table 2). Finally, controlled direct effects estimates were nearly identical to the natural direct effect estimates (Supplemental Table 3).

Physical abuse was not associated with obesity in 6- to 11 or 12- to 19-year-olds. Mental illness and alcohol abuse in a mother's childhood home were not associated with child obesity in any age group.

DISCUSSION

We found in a diverse, national sample that maternal history of physical abuse in childhood was related to obesity in the next generation of preschool-age children. Specifically, reporting one or more episodes of physical harm from a parent or adult in the mother's childhood

home was associated with 3.7 excess cases of early childhood obesity per 100 mother-child pairs—a 21% relative increase in risk. This relationship was partially mediated by maternal pre-pregnancy BMI, but not gestational weight gain. Although we found no evidence of a similar relationship for older children, or for two other ACE markers, our results shed light on a possible intergenerational pathway contributing to obesity in childhood.

Abuse in childhood may lead to obesity prior to pregnancy through a constellation of factors, including emotional eating used to cope with anxiety and depression (14-16), dysregulated cortisol response and chronic inflammation (15, 16), and obesogenic behaviors and environments related to socioeconomic disadvantages (6, 16). In turn, obesity in pregnancy may contribute to obesity in childhood through shared genetic predispositions, epigenetic effects on offspring metabolic regulation, and shared lifestyle factors (4, 5). This hypothesis is supported by recent findings in a large, national study of female nurses and their children, in which researchers reported a total association between a composite measure of physical, emotional, and sexual abuse in childhood and self-reported high BMI in adolescent offspring (13). This association was largely attenuated by adjusting for mother's concurrent BMI but unaffected by adjustment for the child's own exposure to abuse. Our study expands previous findings to include an overall association between physical abuse and early childhood obesity and a mediating role of maternal weight before conception in a diverse study population with mostly measured child weights and heights.

The results of this study may be informative for future interventions and research. Trials that aim to promote healthy weight gain during pregnancy have been conducted in part to hopefully reduce risk for child obesity (32, 33). Such interventions, however, have been met with limited success in preventing excessive gestational weight gain and large-for-gestational age births, and have not demonstrated an effect on child obesity (2, 32-34). For this reason, it has recently been suggested that interventions should target maternal weight before conception,

although no such randomized trials have been conducted (35). Our results underscore the need for preconception interventions for women who are overweight or obese and suggest that incorporation of childhood abuse screening and management could bolster effectiveness in reducing maternal and child obesity.

It is unknown why our findings are limited to physical abuse and early childhood obesity. However, our results are consistent with other findings of only physical abuse being associated with adult obesity and that other ACE exposures may confer different effects than physical abuse (6-8, 12). The prevalence of physical abuse in this study was similar to that among women in the BRFSS, but the prevalences of household mental illness and alcoholism were lower by 8.1 and 4.6 percentage points, suggesting that our study group may have underreported these exposures (36); therefore, we cannot confidently rule out their importance. We were also unable to assess other types of abuse and neglect, which could confound the association between physical abuse and early childhood obesity or be independent risk factors. Future studies that comprehensively assess abuse and neglect are needed. Additionally, an association of maternal physical abuse with offspring obesity limited to preschool-age children may be due to their younger age and closer proximity to pregnancy. Exposure to both positive and negative lifestyle factors at preschool ages may overshadow the effects of prenatal risk factors in school-age children and adolescents, although we did not study this possibility (37).

Several factors limit the results of this study. Mothers self-reported all their characteristics, which may have led to some misclassification. We regression-calibrated maternal height, but lacked calibration data for maternal weight. Although self-reported weight can be prone to error, a recent systematic review concluded that self-reported pre-pregnancy and delivery weight does not bias associations with perinatal outcomes in most studies (38). Adverse childhood experiences were recalled much later in life, although validation studies on

retrospective ACE measurement suggest our measures would likely result in false negatives and weaker associations than if ACE measures were collected earlier in life (39). We were also only able to study three ACE exposures because of questionnaire length limitations. We were therefore unable to use the full set of 11 ACE questions and could not assess the severity of ACE or other ACE exposures, which would better inform the relationships between ACE, maternal obesity, and child obesity. The validity of our approach could not be tested, but shortened ACE assessment has been shown to be appropriate (40). Additionally, the available data lacked information on several factors likely to confound the associations studied, including psychological measures, family history of obesity, paternal characteristics, pregnancy complications, and the child's exposure to ACE. The prevalences of pre-pregnancy obesity and preschool-age obesity were lower in NLSY mother-child pairs ineligible for this study, which could have inflated effect sizes in the included sample. The NLSY also only collected child weight and height measurements from 1986 to 2012, resulting in measurements not being recorded at certain ages if a child was born close to the beginning or the end of the study period.

A number of strengths counterbalance this study's limitations. The NLSY79 was designed to be nationally representative of youths and adolescents in 1979 and prospectively followed participants for 33 years, including 26 years of measuring the weights and heights of female participants' children. Additionally, trained study staff made 74% of weight and 82% of height measurements in person. These attributes strengthen both the internal and external validity of our results. Our mediation findings are strengthened by clear temporality, z-scores that standardized gestational weight gain for gestational duration (20), and mediation analysis methods that improve upon the conventional adjustment approach, which is often severely biased (30).

CONCLUSION

In conclusion, maternal physical abuse in childhood was associated with a modest increase in children's risk of obesity at pre-school ages. Pre-pregnancy BMI explained approximately a quarter of this association. In contrast, weight gain during pregnancy explained a very small portion of the association. No associations were found with later childhood obesity or with maternal experience of mental illness or alcohol abuse in the childhood household. This study's results expand our limited knowledge of intergenerational contributors to obesity in young children and the importance of efforts to prevent childhood abuse. Additionally, they suggest that interventions to promote healthy weight prior to pregnancy and prevent childhood obesity should consider evaluating the impact of screening and management of maternal adverse childhood experiences, particularly physical abuse.

REFERENCES

1. Ogden CL, Carroll MD, Lawman HG, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2012-2014. *JAMA* 2016;315:2292-2299.
2. Waters E, de Silva-Sanigorski A, Burford BJ, et al. Interventions for preventing obesity in children. *Cochrane Database of Systematic Reviews* 2011(12).
3. Power C, Kuh D, Morton S. From developmental origins of adult disease to life course research on adult disease and aging: insights from birth cohort studies. *Annu Rev Public Health* 2013;34:7-28.
4. Woo Baidal JA, Locks LM, Cheng ER, Blake-Lamb TL, Perkins ME, Taveras EM. Risk factors for childhood obesity in the first 1,000 days: a systematic review. *Am J Prev Med* 2016;50:761-79.
5. Alfaradhi MZ, Kusinski LC, Fernandez-Twinn DS, et al. Maternal obesity in pregnancy developmentally programs adipose tissue inflammation in young, lean male mice offspring. *Endocrinology* 2016;157:4246-4256.
6. Thomas C, Hypponen E, Power C. Obesity and type 2 diabetes risk in midadult life: the role of childhood adversity. *Pediatrics* 2008;121:e1240-1249.
7. Bentley T, Widom CS. A 30-year follow-up of the effects of child abuse and neglect on obesity in adulthood. *Obesity* 2009;17:1900-1905.
8. Rehkopf DH, Headen I, Hubbard A, et al. Adverse childhood experiences and later life adult obesity and smoking in the United States. *Ann Epidemiol* 2016;26:488-492.e5.
9. Hemmingsson E, Johansson K, Reynisdottir S. Effects of childhood abuse on adult obesity: a systematic review and meta-analysis. *Obes Rev* 2014;15:882-93.
10. Ranchod YK, Headen IE, Petito LC, Deardorff JK, Rehkopf DH, Abrams BF. Maternal childhood adversity, prepregnancy obesity, and gestational weight gain. *Am J Prev Med* 2016;50:463-9.
11. Diesel JC, Bodnar LM, Day NL, Larkby CA. Childhood maltreatment and the risk of pre-pregnancy obesity and excessive gestational weight gain. *Matern Child Nutr* 2016;12:558-568.
12. Nagl M, Steinig J, Klinitzke G, Stepan H, Kersting A. Childhood maltreatment and pre-pregnancy obesity: a comparison of obese, overweight, and normal weight pregnant women. *Arch Womens Ment Health* 2016;19:355-65.
13. Roberts AL, Galea S, Austin SB, Corliss HL, Williams MA, Koenen KC. Women's experience of abuse in childhood and their children's smoking and overweight. *Am J Prev Med* 2014;46:249-58.

14. Vámosi M, Heitmann BL, Kyvik KO. The relation between an adverse psychological and social environment in childhood and the development of adult obesity: a systematic literature review. *Obes Rev* 2010;11:177-184.
15. Michopoulos V, Powers A, Moore C, Villarreal S, Ressler KJ, Bradley B. The mediating role of emotion dysregulation and depression on the relationship between childhood trauma exposure and emotional eating. *Appetite* 2015;91:129-136.
16. Ehlert U. Enduring psychobiological effects of childhood adversity. *Psychoneuroendocrinology* 2013;38:1850-1857.
17. Elovainio M, Ferrie JE, Singh-Manoux A, et al. Socioeconomic differences in cardiometabolic factors: social causation or health-related selection? Evidence from the Whitehall II Cohort Study, 1991-2004. *Am J Epidemiol* 2011;174:779-789.
18. Gordon-Larsen P, Harris KM, Ward DS, Popkin BM, National Longitudinal Study of Adolescent Health. Acculturation and overweight-related behaviors among Hispanic immigrants to the US: the National Longitudinal Study of Adolescent Health. *Soc Sci Med* 2003;57:2023-2034.
19. Centers for Disease Control and Prevention (CDC). Behavioral Risk Factor Surveillance System Questionnaire. 2010; www.cdc.gov/brfss/questionnaires/pdf-ques/2010brfss.pdf. Accessed Jan 11, 2017.
20. Hutcheon JA, Platt RW, Abrams B, Himes KP, Simhan HN, Bodnar LM. A weight-gain-for-gestational-age z score chart for the assessment of maternal weight gain in pregnancy. *Am J Clin Nutr* 2013;97:1062-1067.
21. Hutcheon JA, Platt RW, Abrams B, Himes KP, Simhan HN, Bodnar LM. Pregnancy weight gain charts for obese and overweight women. *Obesity* 2015;23:532-535.
22. Hutcheon JA, Bodnar LM, Joseph KS, Abrams B, Simhan HN, Platt RW. The bias in current measures of gestational weight gain. *Paediatr Perinat Epidemiol* 2012;26:109-116.
23. Bodnar LM, Hutcheon JA, Parisi SM, Pugh SJ, Abrams B. Comparison of gestational weight gain z-scores and traditional weight gain measures in relation to perinatal outcomes. *Paediatr Perinat Epidemiol* 2015;29:11-21.
24. Hernán MA, Hernández-Díaz S, Werler MM, Mitchell AA. Causal knowledge as a prerequisite for confounding evaluation: an application to birth defects epidemiology. *Am J Epidemiol* 2002;155:176-184.
25. Sharp GC, Lawlor DA, Richmond RC, Fraser A, Simpkin A, Suderman M, et al. Maternal pre-pregnancy BMI and gestational weight gain, offspring DNA methylation and later offspring adiposity: findings from the Avon Longitudinal Study of Parents and Children. *Int J Epidemiol* 2015;44:1288-1304.

26. Centers for Disease Control and Prevention (CDC). A SAS Program for the 2000 CDC Growth Charts (ages 0 to <20 years). <http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm>. Accessed Jan 11, 2017.
27. Lumley T. Analysis of complex survey samples. *J Stat Softw* 2004;9:1-19.23.
28. van Buuren S, Groothuis-Oudshoorn K. mice: Multivariate imputation by chained equations in R. *J Stat Softw* 2011;45(3).
29. Sterne JAC, Carlin JB, Spratt M, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *BMJ* 2009;338:b2393.
30. Petersen ML, Sinisi SE, van der Laan MJ. Estimation of direct causal effects. *Epidemiology* 2006;17:276-84.
31. R Core Team. R: A Language and Environment for Statistical Computing. Vienna, Austria: R Foundation for Statistical Computing; 2016. <http://www.r-project.org/>.
32. Poston L, Bell R, Croker H, et al. Effect of a behavioural intervention in obese pregnant women (the UPBEAT study): a multicentre, randomised controlled trial. *Lancet Diabetes Endocrinol* 2015;3:767-777.
33. Vesco KK, Karanja N, King JC, et al. Efficacy of a group-based dietary intervention for limiting gestational weight gain among obese women: a randomized trial. *Obesity* 2014;22:1989-1996.
34. Bleich SN, Segal J, Wu Y, Wilson R, Wang Y. Systematic review of community-based childhood obesity prevention studies. *Pediatrics* 2013;132:e201-e210.
35. Opray N, Grivell RM, Deussen AR, Dodd JM. Directed preconception health programs and interventions for improving pregnancy outcomes for women who are overweight or obese. *Cochrane Database Syst Rev* 2015;7(CD010932).
36. Centers for Disease Control and Prevention (CDC). Behavioral Risk Factor Surveillance System (BRFSS): Prevalences of ACEs. http://www.cdc.gov/violenceprevention/acestudy/ace_brfss.html. Accessed Jan 11, 2017.
37. Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation. Editors: Glickman D, Parker L, Sim LJ, Cook HD, Miller EA. Washington, DC: The National Academies Press; 2012.
38. Headen I, Cohen AK, Mujahid M, Abrams B. The accuracy of self-reported pregnancy-related weight: a systematic review. *Obes Rev* 2017;(in press).
39. Hardt J, Rutter M. Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *J Child Psychol Psychiatry* 2004;45:260-273.
40. Wade R, Jr., Becker BD, Bevans KB, Ford DC, Forrest CB. Development and evaluation of a short adverse childhood experiences measure. *Am J Prev Med* 2017;52:163-172.

Table 1. Maternal and child characteristics in total sample and by maternal ACE^a

	Total	Physical	Alcohol	Mental
		abuse	abuse in	illness in
			household	household
	(n = 6718)	(n = 1181)	(n = 1414)	(n = 636)
	%	%	%	%
Child obesity status				
Age 2-5 years ^b	17.2	20.3	16.5	18.7
Age 6-11 years	25.3	24.5	21.6	21.2
Age 12-19 years	22.5	23.0	20.5	22.6
Early life characteristics of				
mother				
Maternal grandmother completed	62.9	59.0	61.3	69.8
high school				
Race/ethnicity				
Non-Hispanic white or other	74.4	76.3	79.6	85.4
Non-Hispanic black	17.1	13.3	13.1	10.0
Hispanic	7.6	9.2	6.8	4.6
Asian	1.0	1.2	0.5	0.0
Born in the U.S.	95.3	91.9	95.3	93.7
Born in southern U.S.	31.7	25.0	26.4	28.5
Urban childhood home	78.3	79.3	76.7	78.2
Prenatal characteristics^c				
Pre-pregnancy BMI (kg/m ²)				
Underweight (< 18.5)	7.3	8.6	8.0	8.6
Normal weight (18.5 to < 25)	64.3	60.4	63.0	64.2
Overweight (25 to < 30)	17.7	15.6	16.2	14.3
Obese (≥ 30)	10.7	15.4	12.0	12.9
Gestational weight gain ^d				
Inadequate	29.2	25.7	25.5	29.2
Adequate	29.4	26.0	29.3	26.6
Excessive	41.5	48.3	45.2	44.2
Age				
< 20 years	7.0	8.9	7.7	5.7
20-30 years	55.9	57.3	56.7	56.7
≥ 30 years	37.0	33.8	35.6	37.6
Education				
Less than high school	15.7	24.5	18.3	13.0
completion				
High school graduate	63.3	62.5	65.1	65.2
College graduate	20.9	13.0	16.6	21.8

Employment				
Unemployed	34.2	40.3	36.2	36.5
Part-time	28.3	27.7	29.3	28.7
Full-time	37.5	32.0	34.5	34.8
Equivalized household income				
Lowest quartile	24.9	31.7	25.5	22.4
Second quartile	25.1	28.6	29.9	30.7
Third quartile	25.1	20.0	22.7	27.0
Highest quartile	25.8	19.6	21.9	19.8
Married	74.9	68.4	74.7	74.5
Smoked cigarettes in pregnancy	25.9	33.6	30.0	26.9
Primiparous	43.0	41.8	43.6	44.9
Child birthyear				
<1980	7.7	7.6	8.6	5.9
1980-1990	54.1	57.0	53.9	56.3
>1990	38.2	35.3	37.5	37.8

ACE, adverse childhood experience; BMI, body mass index

^a Percentages are weighted for the survey sampling design. Sample sizes are not weighted and refer to number of mother-child pairs.

^b Child obesity percentages includes children who had an obese BMI at least once during the specified age range.

^c Pre-pregnancy BMI and gestational weight gain were assessed as mediators. All other maternal characteristics in pregnancy were treated as confounding variables.

^d Based on Institute of Medicine 2009 recommendations.

Figure 1. Selection flowchart of mother-child pairs for the study. Participants not included in the 2012 survey wave either were intentionally dropped from the original cohort due to funding constraints, lost to follow-up, or died.

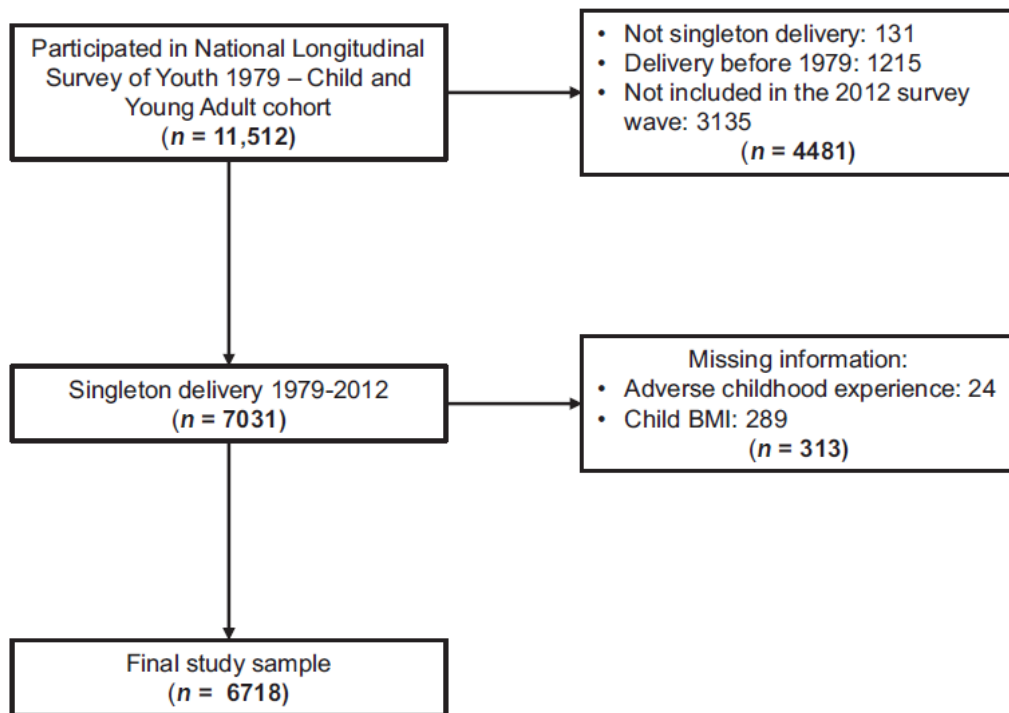
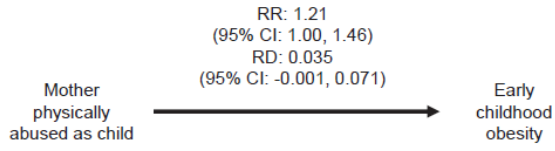
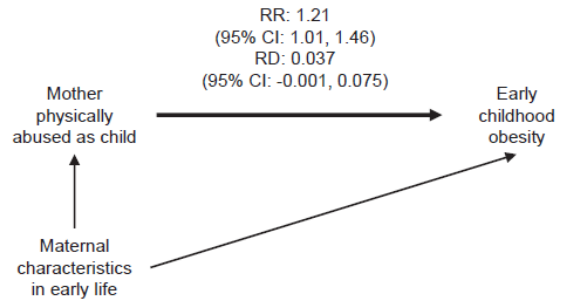


Figure 2. Diagrams of estimates of unadjusted (A) and adjusted (B) total effects of maternal history of physical abuse in childhood on risk of obesity in children ages 2-5 years, and natural direct effect estimates for mediation by pre-pregnancy BMI (C) and gestational weight gain (D). Risk ratios (RR) and risk differences (RD) with 95% confidence intervals (CI) shown correspond to each emboldened pathway.

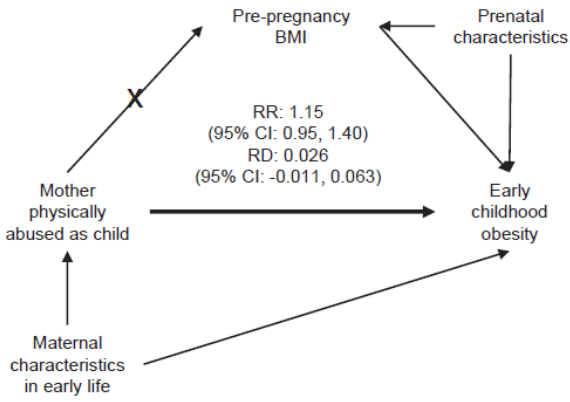
A



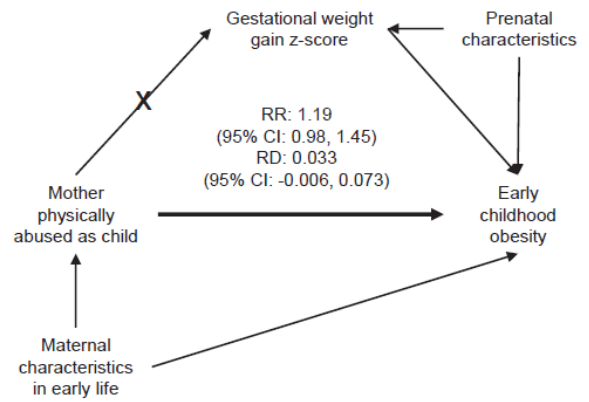
B

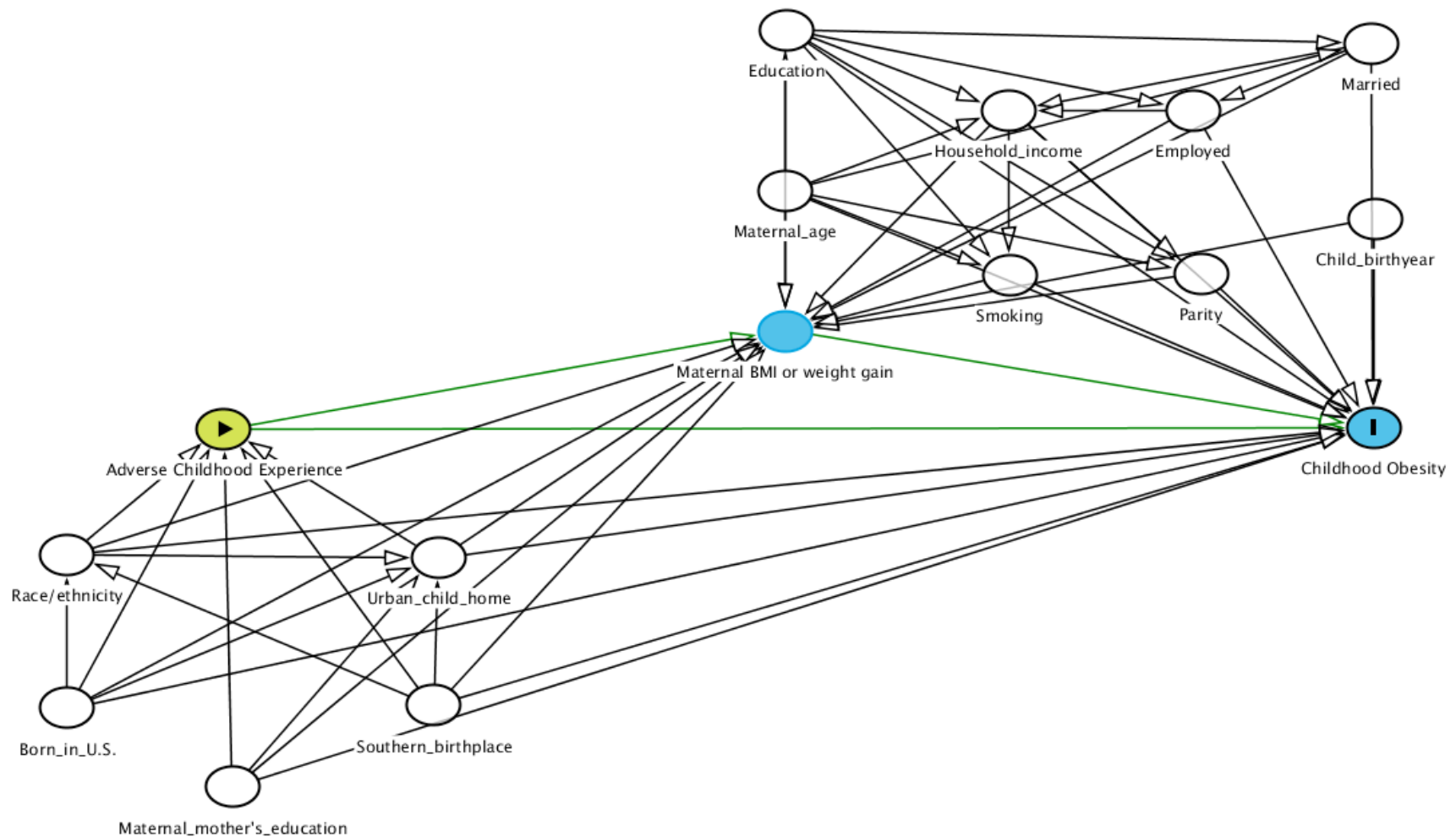


C



D





Supplemental Figure 1. Directed acyclic graph to determine confounding variables. The covariates on the lower, left-hand side are maternal characteristics in early life and were used in modeling the associations between maternal adverse childhood experiences and childhood obesity. The covariates on the upper, right-hand side are prenatal characteristics and were used in modeling the associations between maternal pre-pregnancy BMI and childhood obesity. The same set of covariates were used in testing both pre-pregnancy BMI and gestational weight gain z-score as mediators. Green lines indicate direct pathways and black lines indicate confounding pathways.

Appendix A: Detailed methods to estimate natural direct effects and controlled direct effects

Below, we outline the steps of our analysis using, as an example, pre-pregnancy BMI as the mediator. We repeated these steps for gestational weight gain z-score as the mediator, gestational weight gain adjusted for pre-pregnancy BMI, and with childhood overweight/obesity as the outcome.

1. We applied log-binomial regression to estimate the total effect of physical abuse on early childhood obesity.
2. We updated the log-binomial regression model to adjust for pre-pregnancy BMI and all confounders.
3. We replicated the data into a second dataset and set all physical abuse observations to 'no' (0) in one dataset (all unexposed) and to 'yes' (1) in the second dataset (all exposed).
4. In each dataset, we predicted all confounding maternal characteristics at pregnancy using regression models with physical abuse, pre-pregnancy BMI, and all confounding maternal characteristics in early life as predictors.
5. We applied linear regression to estimate the association between physical abuse and pre-pregnancy BMI, adjusting for all covariates. In the unexposed dataset, we predicted pre-pregnancy BMI using the regression model. We set pre-pregnancy BMI in both the unexposed and exposed datasets to this expected value if unexposed to physical abuse ("the natural value").
6. In each dataset, we predicted the expected probabilities of early childhood obesity using the total effect log-binomial regression model from Step 2.
7. We calculated the risk ratio for the natural direct effect (NDE) by dividing the mean of the expected probabilities of obesity in the exposed dataset by the mean of the expected probabilities of obesity in the unexposed dataset. The risk difference for the NDE was calculated as the difference of these two quantities.
8. We bootstrapped the analyses 1000 times for statistical inference.
9. The steps above were repeated with the addition of an interaction term between physical abuse and pre-pregnancy BMI.
10. To estimate the controlled direct effect (CDE), we repeated the same steps; however, in Step 4 we set pre-pregnancy BMI for all women in each dataset to 25 kg/m² and we did not predict the natural value of pre-pregnancy BMI (Step 6).
11. We repeated the CDE estimation for values of 22 and 20 kg/m² for a range of estimates.
12. For gestational weight gain z-score mediation analysis, we set z-score to 0 (the standard mean) for CDE estimation. When using the Institute of Medicine gestational weight gain recommendation categories as the mediator in sensitivity analyses, we also adjusted for gestational duration.

Supplemental Table 1. Characteristics of mother-child pairs included in the final sample, ineligible for the study, and eligible but excluded from the final sample^a

	Final sample	Ineligible sample	Excluded sample
	(n = 6718)	(n = 4477)	(n = 317)
	%	%	%
Adverse childhood experiences			
Physical abuse of child	17.3	21.3	0
Alcohol abuse in household	22.2	25.7	11.1
Mental illness in household	11.0	7.8	2.8
Child obesity status			
Age 2-5 years ^b	17.2	14.3	14.4
Age 6-11 years	25.3	16.0	17.0
Age 12-19 years	22.5	20.8	19.0
Early life characteristics of mother			
Maternal grandmother completed high school	62.9	60.3	65.7
Race/ethnicity			
Non-Hispanic white or other	74.4	75.0	81.3
Non-Hispanic black	17.1	17.2	11.9
Hispanic	7.6	7.2	6.3
Asian	1.0	0.6	0.5
Born in the U.S.	95.3	95.0	94.6
Born in southern U.S.	31.7	34.7	31.2
Urban childhood home	78.3	78.6	79.1
Prenatal characteristics^c			
Pre-pregnancy BMI (kg/m ²)			
Underweight (< 18.5)	7.3	11.3	9.7
Normal weight (18.5 to < 25)	64.3	71.1	71.0
Overweight (25 to < 30)	17.7	13.0	13.7
Obese (≥ 30)	10.7	4.6	5.5
Gestational weight gain ^d			
Inadequate	29.2	31.7	29.8
Adequate	29.4	29.1	30.8
Excessive	41.5	39.3	39.3
Age			
< 20 years	7.0	26.9	12.9
20-30 years	55.9	47.3	55.9
≥ 30 years	37.0	25.7	31.2
Education			
Less than high school completion	15.7	16.7	16.8
High school graduate	63.3	60.6	61.4
College graduate	20.9	22.7	21.8
Employment			
Unemployed	34.2	39.4	39.5
Part-time	28.3	25.3	25.6

Full-time	37.5	35.3	34.9
Equivalized household income			
Lowest quartile	24.9	25.0	24.9
Second quartile	25.1	23.4	23.9
Third quartile	25.1	24.4	24.5
Highest quartile	25.8	27.3	26.7
Married	74.9	77.7	77.9
Smoked cigarettes in pregnancy	25.9	32.8	31.6
Primiparous	43.0	43.8	45.8
Child birthyear			
<1980	7.7	31.4	14.8
1980-1990	54.1	42.4	53.5
>1990	38.2	26.2	31.8

BMI, body mass index

^a Percentages are weighted for the survey sampling design. Sample sizes are not weighted and refer to number of mother-child pairs.

^b Child obesity percentages includes children who had an obese BMI at least once during the specified age range.

^c Pre-pregnancy BMI and gestational weight gain were assessed as mediators. All other maternal characteristics in pregnancy were treated as confounding variables.

^d Based on Institute of Medicine 2009 recommendations.

Supplemental Table 2. Results of total effect and additional natural direct effect estimates for the association between history of physical abuse in childhood and children’s overweight/obesity status age 2-5 years.

	Obese	Overweight/Obese
Unadjusted total effect		
RR (95% CI)	1.21 (1.00, 1.46)	1.10 (0.97, 1.24)
RD (95% CI)	0.035 (-0.001, 0.071)	0.030 (-0.010, 0.072)
Adjusted total effect		
RR (95% CI)	1.21 (1.01, 1.46)	1.10 (0.97, 1.24)
RD (95% CI)	0.037 (-0.001, 0.075)	0.032 (-0.010, 0.074)
NDE: pre-pregnancy BMI		
RR (95% CI)	1.15 (0.95, 1.40)	1.07 (0.94, 1.21)
RD (95% CI)	0.026 (-0.011, 0.063)	0.022 (-0.019, 0.063)
NDE: gestational weight gain		
RR (95% CI)	1.19 (0.98, 1.45)	1.09 (0.96, 1.24)
RD (95% CI)	0.033 (-0.006, 0.073)	0.028 (-0.015, 0.070)
NDE: pre-pregnancy BMI and physical abuse interaction		
RR (95% CI)	1.18 (0.97, 1.44)	1.09 (0.96, 1.23)
RD (95% CI)	0.031 (-0.008, 0.070)	0.027 (-0.015, 0.069)
NDE: gestational weight gain and physical abuse interaction		
RR (95% CI)	1.19 (0.98, 1.46)	1.09 (0.95, 1.24)
RD (95% CI)	0.034 (-0.006, 0.074)	0.028 (-0.017, 0.073)
NDE: joint mediation by pre-pregnancy BMI and gestational weight gain		
RR (95% CI)	1.14 (0.94, 1.38)	1.06 (0.93, 1.20)
RD (95% CI)	0.024 (-0.013, 0.060)	0.019 (-0.022, 0.060)

RR, risk ratio; RD, risk difference; CI, confidence interval; NDE, natural direct effect; BMI, body mass index

Total effects adjusted for maternal grandmother’s education, maternal race/ethnicity, born in U.S., born in southern U.S., urban childhood home. Natural direct effects additionally adjusted for age, education, employment status, equivalized household income, marital status, parity, smoking, and child birth year.

Supplemental Table 3. Results of controlled direct effect (CDE) estimates for the association between history of physical abuse in childhood and children’s overweight/obesity status age 2-5 years.

	Obese	Overweight/Obese
CDE: pre-pregnancy BMI set to normal weight		
RR (95% CI)	1.17 (0.96, 1.43)	1.08 (0.95, 1.22)
RD (95% CI)	0.027 (-0.008, 0.062)	0.023 (-0.017, 0.062)
CDE: gestational weight gain set to within IOM recommendation		
RR (95% CI)	1.19 (0.98, 1.45)	1.09 (0.96, 1.23)
RD (95% CI)	0.033 (-0.006, 0.072)	0.028 (-0.015, 0.070)

RR, risk ratio; RD, risk difference; CI, confidence interval; CDE, controlled direct effect; BMI, body mass index; IOM, Institute of Medicine

Adjusted for maternal grandmother’s education, maternal race/ethnicity, born in U.S., born in southern U.S., urban childhood home, maternal age at pregnancy, education, employment status, equivalized household income, marital status, parity, smoking, and child birth year. CDE for IOM recommendations additionally adjusted for gestational duration.

