Association Between Maternal Intimate Partner Violence and Incident Obesity in Preschool-Aged Children:

Results From the Fragile Families and Child Well-being Study

Renée Boynton-Jarrett, MD, ScD, **Jessica Fargnoli, MPH**, **Shakira Franco Suglia, MS, ScD**, **Barry Zuckerman, MD**, and **Rosalind J. Wright, MD, MPH**

Department of General Pediatrics, School of Medicine (Drs Boynton-Jarrett, Suglia, and Zuckerman) and Department of Epidemiology, School of Public Health (Ms Fargnoli and Dr Suglia), Boston University, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School (Dr Wright), and Department of Environmental Health, Harvard School of Public Health (Dr Wright), Boston, Massachusetts.

Abstract

Objective—To examine the impact of chronicity of maternal intimate partner violence (IPV) on obesity risk among preschool-aged children.

Design—Prospective cohort study.

Setting—Several large US cities.

Participants—A subsample of the Fragile Families and Child Well-being Study participants (n = 1595), who were children born between 1998 and 2000 and their parents interviewed at baseline and at 12, 36, and 60 months.

Main Exposure—Maternal report of restrictive, sexual, and physical abuse from an intimate partner. Chronic IPV was defined as any maternal IPV exposure during both pregnancy or infancy (0–12 months) and early childhood (36–60 months).

Main Outcome Measure—Repeated measures of child body mass index.

Results—Among the 1595 children, 16.5% were obese at age 5 years and 49.4% of the mothers reported some form of IPV. Compared with those who had no IPV exposure, children whose mothers reported chronic IPV had an elevated risk for obesity at age 5 years (adjusted odds ratio = 1.80; 95% confidence interval, 1.24–2.61). Stratified analyses indicated increased risk for obesity among girls with a maternal history of chronic IPV (adjusted odds ratio = 2.21; 95% confidence interval, 1.30–3.75) compared with boys (adjusted odds ratio = 1.66; 95% confidence interval,

0.94–2.93) and a larger effect of any maternal IPV on obesity among children living in less safe neighborhoods (adjusted odds ratio = 1.56; 95% confidence interval, 1.03–2.36).

Conclusions—Chronic maternal IPV is associated with increased risk of obesity among preschool-aged children. Preventing family violence and improving community safety may help reduce childhood obesity.

Childhood obesity is a growing public health epidemic.[1,2] Obese youth are more likely to become obese adults and disproportionately experience cardiovascular[3] and metabolic[4] comorbidities, reproductive dysfunction,[5,6] and emotional sequelae.[7] Despite recognition that the family environment has a significant influence on childhood obesity,[8] the role of adverse psychosocial exposures on obesity risk among preschool-aged children is poorly specified.

Of the 3 to 10 million children (aged 3–17 years) who witness intimate partner violence (IPV) annually,[9] a disproportionate number are aged 5 years and younger.[10] Exposure to IPV in childhood is associated with altered neuroendocrine system profiles,[11] impaired socioemotional development, cognitive functioning, attachment to caregivers, and emotional regulation, and poorer physical and mental health.[12–16]

Christoffel and Forsyth[17] postulated that severe early childhood obesity may be associated with patterns of family dysfunction. Compelling evidence from recent studies has established an association between childhood adversities, including household dysfunction and family violence, and adult obesity and excessive weight control.[18–20] To date, 4 prospective longitudinal studies[21–24] have demonstrated an association between physical abuse, neglect, and/or sexual abuse and obesity in late adolescence or young adulthood. Childhood abuse has been linked to disordered eating behaviors in adolescence and adulthood,[25–29] although studies have documented an inconsistent association.[30,31] Researchers hypothesize that behavioral changes, including inactivity and overeating, as a means of coping with the psychological impact of abuse may lead to disruption of metabolic systems and hormonal changes.[22]

An association between exposure to family violence or household dysfunction and childhood obesity may operate through several pathways. Family conflict may limit maternal emotional availability for caretaking needs and influence parental feeding style and patterns. Food may be used in excess as a tool for consoling or pacifying emotional needs of the child by the parent[31] or to self-soothe by the child.[32,33] Alternatively, family violence is distressing and may cause affective dysregulation, leading to decreased impulse control and excessive caloric intake.[34] More direct biological mechanisms are also plausible. Chronic stimulation of the hypothalamic- pituitary axis by environmental stressors, including family violence, may elevate cortisol levels, dysregulate neuroendocrine mediators of the reward pathway, and influence compulsive feeding practices[33] as well as visceral fat accumulation.[35]

Women who experience IPV may also be more likely to live in communities with higher levels of social disorganization,[36] disadvantage,[37] and violence.[38] Several studies have found evidence that familial violence exacerbates the adverse impact of child exposure to

community violence.[39] Neighborhood safety may serve as a barrier to the ability to exercise and play outdoors. Moreover, lack of safety may augment the social isolation of women who experience IPV through limiting contact with neighbors, impeding development of supportive relationships and thereby increasing childhood obesity risk by influencing caretaking ability.

While there is a growing appreciation of the impact of child maltreatment on cardiovascular disease risk over the life course,[40] there has been a paucity of research to date that has explored the impact of family violence on obesity risk in early life. To our knowledge, no study has explored the impact of maternal IPV experience and obesity risk among preschool-aged children. Based on prior empirical and theoretical work, this study investigated the following hypotheses: (1) maternal exposure to IPV will be associated with higher odds of obesity at age 5 years in their children; (2) maternal exposure to IPV will be associated with feeding practices and behaviors that elevate risk for childhood obesity; and (3) maternal perception of lower neighborhood safety will increase the effect of IPV on childhood obesity risk.

METHODS

SAMPLE

The analysis presented in this article was conducted with public-use data from the Fragile Families and Child Well-being Study, a longitudinal birth cohort study of 4898 children born in the United States between 1998 and 2000. Investigators sampled families from 20 large US cities to obtain data on unwed parents and their children. The Fragile Families and Child Well-being Study design has been described in detail elsewhere.[41] Briefly, mothers were interviewed in the hospital shortly after giving birth (baseline) and were subsequently interviewed via telephone when their children were aged approximately 12, 36, and 60 months. A subsample of 2489 also participated in the in-home assessment of children at ages 36 and 60 months; of this group, 1612 had weight and height measures at both times. The present analysis included only children who had measures of weight and height from the 36- and 60-month in-home assessments and whose mothers answered the questions regarding IPV. A total of 1595 children were included in the final analysis. Sensitivity analyses revealed no differences between the full cohort at baseline and those who participated in the follow-up in-home assessments (the sample used in these analyses) by parental education or IPV; however, these groups did vary by race/ethnicity. The percentages of families lost to follow-up compared with those who participated in the in-home assessments were lower for black families (44.8% vs 53.3%, respectively) and higher for Asian families (29.3% vs 23.3%, respectively) and American Indian families (4.5% vs 2.8%, respectively). The analyses were approved by the institutional review board at Boston University School of Medicine, Boston, Massachusetts.

MEASURES

Maternal IPV—A measure of exposure to IPV was created using information on mother-father and mother–current partner relationships from the baseline and 12-, 36-, and 60-

month follow-up questionnaires. Questions regarding physical, sexual, and restrictive abuse were included.

Maternal exposure to physical abuse was measured at baseline and at 12, 36, and 60 months. Women were asked how often the baby's father "hits or slaps you when he is angry" during each assessment. On the 12-, 36-, and 60-month assessments, women were asked the same question in regard to a potential current partner who is not the baby's father. Women who responded with sometimes or often to at least 1 of these questions were considered to have been exposed to physical abuse. In addition, women were considered to have experienced physical abuse if they answered yes to the question "Were you ever cut/bruised or seriously hurt in a fight with the baby's father/current partner?" on the 12-, 36-, and 60-month surveys.

Maternal exposure to sexual abuse was measured on the 12-, 36-, and 60-month questionnaires. Women who reported that either the baby's father or their current partner "tries to make you have sex or do sexual things you don't want to" were categorized as having experienced sexual abuse.

Questions about restrictive and controlling behaviors of either the baby's father or the mother's current partner were asked on the 12-, 36-, and 60-month surveys. If a woman responded that either the baby's father or her current partner tried to isolate her from friends or family, prevent her from going to work or school, or withhold and control her money, she was categorized as having experienced restrictive abuse and controlling behavior.

Women were divided into 4 categories based on the timing and chronicity of any form of IPV exposure: no IPV; early IPV (reported IPV during baseline and/or the 12-month interview only); late IPV (IPV during the 36- and/or 60-month interview only); or chronic IPV (IPV both at baseline and/or the 12-month interview and at the 36- and/or 60-month interview). A woman was categorized as experiencing no IPV if she did not report physical, sexual, or restrictive abuse at any interval.

Child Obesity—Child weight and height were measured at the 36- and 60-month in-home assessments as previously described.[42] Body mass index (BMI) of the child was calculated by dividing the child's weight in kilograms by the squared value of the child's height in meters. Child BMI z scores and percentiles were calculated based on the 2000 Centers for Disease Control and Prevention growth charts.[43]

Demographic Variables and Covariates—Data on child sex and birth weight were collected on the baseline survey, as were maternal characteristics including age, race/ethnicity, education, immigration status, smoking during pregnancy, and relationship with the baby's father. The 36-month assessment collected data on child bottle use and hours of daily television viewing. Data on maternal mental health was also collected on the 36- and 60-month in-home assessments using the Composite International Diagnostic Interview Short Form (CIDI-SF) scale.[44,45] The CIDI-SF questions were scored consistent with the developer's guidelines, which follow the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition).[46] The CIDI-SF is based on a portion of the full CIDI and

estimates the probability of being a case; if the respondent's score is greater than 0.5, the respondent is classified as a probable case. Maternal BMI measurements were calculated based on height and weight measurements completed at the 36-month in-home assessment.

Neighborhood Safety—Maternal perception of neighborhood safety was assessed using the 8-item Neighborhood Environment for Children Rating Scale[47] at the 36-month in-home assessment. Mothers were asked to state how often the following activities happened in their neighborhood: drug dealers or users hanging around; drunk people hanging around; unemployed adults loitering; young adults loitering; occurrence of gang activity; seeing misbehaving groups of young children; seeing disorderly or misbehaving groups of teens; and seeing disorderly or misbehaving groups of adults. A scale representing neighborhood safety was created for each woman who responded to at least 6 of the 8 questions. For each question, women received 1 point for the response never, 2 points for the response rarely, 3 points for the response sometimes, and 4 points for the response frequently. Responses to the 8 questions were summed. Possible scores ranged from 6 to 32, with lower scores representing safer neighborhoods. Cronbach α for this scale was .92.

STATISTICAL ANALYSIS

Bivariate analyses of the relation between maternal IPV and covariates (Table 1) were performed using Pearson χ^2 tests to test differences in proportions. Multivariate logistic regression models were used to estimate odds ratios (ORs) while adjusting for factors associated with obesity risk using the SAS PROC LOGISTIC procedure (SAS Institute, Inc, Cary, North Carolina). Obesity was defined as BMI greater than the 95th percentile, and a dichotomous indicator was created. Multivariate logistic regression models were used to determine the association between IPV and childhood obesity. Known predictors of obesity that may also be associated with maternal IPV exposure were explored as confounders in univariate regression analyses and frequency distributions. A covariate was included in the multivariate analyses if theoretical or empirical evidence supported its role as a risk factor for obesity, if it was a significant predictor of obesity in univariate regression models, or if including it in the full multivariate model led to a 5% or greater change in the OR.[48] Model 1 includes maternal IPV exposure, race/ethnicity (black, white, Hispanic, other/unknown), child sex (male, female), maternal age (20–25, 26–28, 29–33, 34–50 years), maternal education (less than high school, high school graduation, beyond high school), maternal nativity (US born, yes or no), child age in months, relationship with father (yes or no), maternal smoking during pregnancy (yes or no), maternal depression (as measured by a CIDI-SF cutoff score 0.5), maternal BMI (normal/underweight, overweight, obese), low birth weight (<2500 g, 2500 g), whether the child takes a bottle to bed at age 3 years (yes or no), and average hours of child television viewing per day at age 3 years (<2 h/d, 2 h/d).

We formally tested for mediation of the relation between IPV and obesity by maternal depression using a bootstrap method.[49] Owing to theoretical and empirical data suggesting differing associations between family and neighborhood violence exposure and BMI by sex, additional stratified analyses were conducted and effect modification was tested using interaction terms. Analysis for model 1 was repeated stratified by sex and the neighborhood

safety scale (above and below the median score). A missing indicator was created for each covariate to preserve sample size.

Finally, we conducted sensitivity analysis to determine whether results were changed relative to different specification of the IPV categorizations.

RESULTS

The distributions of sociodemographic characteristics and covariates are shown based on obesity at age 5 years and IPV exposure categories in Table 1. Of the 1595 participants, 49.4% reported some form of IPV, including 8.9% early, 23.7% late, and 16.8% chronic IPV. Mothers were aged 20 to 50 years; 53.2% were black, 23.3% were Hispanic, and 20.5% were white; 10.6% were born outside the United States; 67.3% were obese or overweight; 34.0% had completed education beyond high school; 19.4% smoked during pregnancy; and 11.4% met depression criteria on the CIDI-SF. Among the children, 8.8% were born with low birth weight; 6.3% took a bottle to bed at age 3 years; and 34.4% watched less than 2 hours of television daily. Mothers with chronic IPV compared with mothers with no IPV were more likely to smoke during pregnancy (28.4% vs 12.9%, respectively; $P < .001$) and to be obese or overweight. Children whose mothers reported chronic IPV compared with those whose mothers reported no IPV were more likely to watch 2 or more hours of television daily (69.0% vs 60.6%, respectively; $P = .01$) and to take a bottle to bed (8.2% vs 5.6%, respectively; $P = .46$).

As demonstrated in Table 2, children whose mothers reported chronic IPV were 80% more likely to be obese at age 5 years than those with no maternal IPV in the model 1 analysis adjusted for all covariates (OR = 1.80; 95% confidence interval [CI], 1.24–2.61). This association was partially explained by maternal factors such as maternal BMI, smoking during pregnancy, and marital status and by child factors including television viewing, birth weight, and bottle feeding (data not shown but available on request). Children who were exposed to early or late maternal IPV only had a borderline, nonsignificant increase in risk. Excluding those who lived with their mother only part-time or whose mothers had not lived with a partner for 2 months (n = 327) did not substantively change our results.

We tested the role of maternal depression at 36 months (as measured by the continuous CIDI-SF scale) as a mediator of the relation between both chronic maternal IPV and maternal IPV prior to 36 months and obesity risk at age 60 months in separate models using the Preacher and Hayes bootstrapping method.[49] We found evidence for simple mediation of maternal IPV prior to 36 months and chronic maternal IPV by maternal depression.

In analyses stratified by sex, we found an increased risk for obesity among girls with maternal chronic IPV (adjusted OR = 2.21; 95% CI, 1.30–3.75) compared with boys (OR = 1.66; 95% CI, 0.94–2.93) (Table3). Analyses stratified by maternal perception of neighborhood safety revealed an increased obesity risk at age 60 months (adjusted OR = 1.56; 95% CI, 1.03–2.36) among those with any maternal IPV exposure and living in less safe neighborhoods. Children of mothers reporting any IPV and residing in a neighborhood they perceived to be safe did not have statistically significant increased odds for obesity at

age 5 years (adjusted OR = 1.38; 95% CI, 0.88–2.17) (Table 4). Interactions between IPV and neighborhood safety and between IPV and sex were not statistically significant.

COMMENT

In these prospective analyses, we found an association between chronic maternal IPV experience and risk for obesity in these preschool-aged children. This finding persisted even when controlling for obesity at age 3 years, several postulated intermediates (including child bottle-feeding and television viewing), and ostensible confounders such as maternal depression, maternal smoking during pregnancy, child birth weight, and other relevant covariates. In addition, our results suggest that girls with maternal exposure to chronic IPV were at greater risk for early childhood obesity than boys. Of note, there was also some suggestion that maternal perception of poor neighborhood safety may augment risk of obesity in early childhood among those exposed to maternal IPV. If substantiated, these findings may have implications for obesity prevention and reduction efforts. Therefore, interventions aimed at reducing obesity risk may be enhanced by incorporating strategies to address family violence.

The association between maternal IPV and childhood obesity could operate through several pathways. First, if IPV influences maternal responsiveness to the socioemotional needs of the child, then feeding practices may be influenced.[17,33] Maladaptive parenting behavior has been associated with eating disorders in adolescence.[21] Family connectedness, perceived caring, and positive communication were found to be protective against disordered eating in girls.[25] Second, witnessing family violence may be associated with emotional distress and emotion-focused coping using food to self-soothe and address negative emotions.[20] Perceived stress influences feeding behaviors, including type, quantity, and pattern of eating.[50–53] Glucocorticoids may drive intake of palatable, or "comfort," foods that decrease feelings of stress and reinforce habitual behaviors at the expense of homeostatic regulators.[51] Eating disturbances such as binge-eating disorder, which is correlated with obesity, and bulimia have been linked to emotional,[54] physical,[26,55,56] and sexual[30] abuse. A recent meta-analysis of 53 studies concluded that a small, significant positive relation exists between child sexual abuse and disordered eating,[57] including binge eating,[21,30,31,58,59] body dissatisfaction, purging, and dietary restriction.[59,60] A small, yet relatively consistent body of research has documented a modest association between child sexual abuse and adult obesity in cross-sectional clinical,[61–64] community,[65] and national[66–69] samples. Third, chronic stress associated with family violence may alter hypothalamic-pituitary axis functioning, lead to dysregulation of neuroendocrine systems controlling appetite, and influence hormonal regulation of visceral fat distribution.[35] Other traumatic childhood experiences have been linked to altered serotonin and cortisol systems.[70] Overlapping research has shown that bulimia is associated with decreased serotonin metabolites in cerebrospinal fluid,[71–73] reduced platelet binding of serotonin reuptake inhibitors,[73] reduced density of paroxetine-binding sites, and altered cortisol function.[70] Thus, early-life disruption of neuroendocrine systems may elevate risk for disordered eating behaviors and suboptimal fat storage and distribution.

Our findings support previous research suggesting that neighborhood safety and community violence are associated with childhood obesity risk. Parental perception of neighborhood safety has been linked to increased risk for overweight in childhood in some[74] but not all cross-sectional surveys.[75] Research exploring neighborhood safety and physical[74,76–80] and sedentary[74,75] activities among youth has also revealed inconsistent findings, although many of these studies have been limited by measures of the neighborhood environment and use differing child or parental reports. Although prior research in this cohort revealed that maternal perception of neighborhood safety was not associated with obesity at age 3 years among offspring, we found that among those with any maternal IPV, those whose mothers perceived lower neighborhood safety had a nearly60% increase in risk of obesity atage5years. Hypothetically, community violence may influence norms regarding the use of violence for conflict resolution within and outside the home. Alternatively, neighborhood violence may augment the degree of social isolation faced by mothers exposed to IPV and impede caretaking ability. Finally, for children growing up with family conflict, exposure to community violence may have a cumulative impact on distress symptoms and thereby influence both physiological and behavioral coping strategies that elevate risk for early obesity.

While this study has a number of strengths, including the reasonably large sample, ethnic diversity, and ability to consider a number of important confounders, mediators, and moderators, there are also important limitations. First, a significantly reduced sample of our study population had available longitudinal data on BMI; therefore, if loss to follow-up is not nondifferential, the findings may be skewed. However, measured anthropometrics are more accurate than self-report and are thus more robust measures, justifying our focus on this reduced sample. Second, our study exposure is based on maternal self-report; therefore, we may be underestimating the prevalence of IPV owing to social desirability or recall bias. As the questions on IPV are from previously validated survey instruments, this is less likely. Moreover, reporting bias would most likely lead to underreporting and bias our results toward the null. Third, our assessment of IPV is limited to the questions included in the Fragile Families and Child Well-being Study, so we may have some imprecision in our measure of IPV. Finally, we lack detailed measures of several important predictors of obesity, including nutritional content of diet, dietary patterns, degree of breastfeeding, and physical activity; therefore, our ability to test plausible mechanisms is limited.

Our results suggest that exposure to chronic maternal IPV in early childhood is associated with an elevated risk for obesity as a preschooler, particularly among girls. Although not statistically significant, maternal perception of neighborhood safety may modify this association. Medical and public health practitioners must consider the impact of family violence on obesity risk when designing and implementing primary obesity prevention interventions. Interventions to prevent IPV, particularly those aimed at educating adolescents about healthy relationships prior to childbearing, may play a crucial role in prevention of early childhood obesity. Moreover, interventions aimed at improving neighborhood safety may have a benefit on reducing childhood obesity risk, even among those exposed to family violence.

Acknowledgments

REFERENCES

1. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA. 2002; 288(14):1723–1727. [PubMed: 12365955]

2. Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003–2006. JAMA. 2008; 299(20):2401–2405. [PubMed: 18505949]

3. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med. 1997; 337(13):869–873. [PubMed: 9302300]

4. Weigensberg MJ, Goran MI. Type 2 diabetes in children and adolescents. Lancet. 2009; 373(9677): 1743–1744. [PubMed: 19465216]

5. Linné Y. Effects of obesity on women's reproduction and complications during pregnancy. Obes Rev. 2004; 5(3):137–143. [PubMed: 15245382]

6. Loret de Mola JR. Obesity and its relationship to infertility in men and women. Obstet Gynecol Clin North Am. 2009; 36(2):333–346. ix. [PubMed: 19501317]

7. Dietz WH. Health consequences of obesity in youth. Pediatrics. 1998; 101(3, pt 2):518–525. [PubMed: 12224658]

8. Dunton GF, Kaplan J, Wolch J, Jerrett M, Reynolds KD. Physical environmental correlates of childhood obesity. Obes Rev. 2009; 10(4):393–402. [PubMed: 19389058]

9. Edelson JL. Children's witnessing of adult domestic violence. J Interpers Violence. 1999; 14(8): 839–870.

10. Fantuzzo J, Boruch R, Beriama A, Atkins M, Marcus S. Domestic violence and children. J Am Acad Child Adolesc Psychiatry. 1997; 36(1):116–122. [PubMed: 9000789]

11. Saltzman KM, Holden GW, Holahan CJ. The psychobiology of children exposed to marital violence. J Clin Child Adolesc Psychol. 2005; 34(1):129–139. [PubMed: 15677287]

12. Bair-Merritt MH, Blackstone M, Feudtner C. Physical health outcomes of childhood exposure to intimate partner violence. Pediatrics. 2006; 117(2):e278–e290. [PubMed: 16452335]

13. Kitzmann KM, Gaylord NK, Holt AR, Kenny ED. Child witnesses to domestic violence. J Consult Clin Psychol. 2003; 71(2):339–352. [PubMed: 12699028]

14. Kolbo JR. Risk and resilience among children exposed to family violence. Violence Vict. 1996; 11(2):113–128. [PubMed: 8933708]

15. Repetti RL, Taylor SE, Seeman TE. Risky families: family social environments and the mental and physical health of offspring. Psychol Bull. 2002; 128(2):330–366. [PubMed: 11931522]

16. Wolfe DA, Crooks CV, Lee V, McIntyre-Smith A, Jaffe PG. The effects of children's exposure to domestic violence. Clin Child Fam Psychol Rev. 2003; 6(3):171–187. [PubMed: 14620578]

17. Christoffel KK, Forsyth BW. Mirror image of environmental deprivation: severe childhood obesity of psychosocial origin. Child Abuse Negl. 1989; 13(2):249–256. [PubMed: 2743183]

18. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) Study. Am J Prev Med. 1998; 14(4):245–258. [PubMed: 9635069]

19. Williamson DF, Thompson TJ, Anda RF, Dietz WH, Felitti V. Body weight and obesity in adults and self-reported abuse in childhood. Int J Obes Relat Metab Disord. 2002; 26(8):1075–1082. [PubMed: 12119573]

20. Greenfield EA, Marks NF. Violence from parents in childhood and obesity in adulthood: using food in response to stress as a mediator of risk. Soc Sci Med. 2009; 68(5):791–798. [PubMed: 19185965]

21. Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. Am J Psychiatry. 2002; 159(3):394–400. [PubMed: 11870002]

22. Lissau I, Sorensen TI. Parental neglect during childhood and increased risk of obesity in young adulthood. Lancet. 1994; 343(8893):324–327. [PubMed: 7905145]

23. Noll JG, Zeller MH, Trickett PK, Putnam FW. Obesity risk for female victims of childhood sexual abuse. Pediatrics. 2007; 120(1):e61–e67. [PubMed: 17606550]

24. Thomas C, Hypponen E, Power C. Obesity and type 2 diabetes risk in midadult life: the role of childhood adversity. Pediatrics. 2008; 121(5):e1240–e1249. [PubMed: 18450866]

25. Fonseca H, Ireland M, Resnick MD. Familial correlates of extreme weight control behaviors among adolescents. Int J Eat Disord. 2002; 32(4):441–448. [PubMed: 12386908]

26. Welch SL, Fairburn CG. Childhood sexual and physical abuse as risk factors for the development of bulimia nervosa. Child Abuse Negl. 1996; 20(7):633–642. [PubMed: 8832119]

27. Wonderlich SA, Wilsnack RW, Wilsnack SC, Harris TR. Childhood sexual abuse and bulimic behavior in a nationally representative sample. Am J Public Health. 1996; 86(8):1082–1086. [PubMed: 8712265]

28. Ackard DM, Neumark-Sztainer D, Hannan PJ, French S, Story M. Binge and purge behavior among adolescents: associations with sexual and physical abuse in a nationally representative sample. Child Abuse Negl. 2001; 25(6):771–785. [PubMed: 11525525]

29. Everill JT, Waller G. Reported sexual abuse and eating psychopathology: a review of the evidence for a causal link. Int J Eat Disord. 1995; 18(1):1–11. [PubMed: 7670438]

30. Grilo CM, Masheb RM. Childhood psychological, physical, and sexual maltreatment in outpatients with binge eating disorder. Obes Res. 2001; 9(5):320–325. [PubMed: 11346674]

31. Moyer DM, DiPietro L, Berkowitz RI, Stunkard AJ. Childhood sexual abuse and precursors of binge eating in an adolescent female population. Int J Eat Disord. 1997; 21(1):23–30. [PubMed: 8986514]

32. Lehman AK, Rodin J. Styles of self-nurturance and disordered eating. J Consult Clin Psychol. 1989; 57(1):117–122. [PubMed: 2925962]

33. Francis LA, Hofer SM, Birch LL. Predictors of maternal child-feeding style: maternal and child characteristics. Appetite. 2001; 37(3):231–243. [PubMed: 11895324]

34. Wonderlich SA, Brewerton TD, Jocic Z, Dansky BS, Abbott DW. Relationship of childhood sexual abuse and eating disorders. J Am Acad Child Adolesc Psychiatry. 1997; 36(8):1107–1115. [PubMed: 9256590]

35. Adam TC, Epel ES. Stress, eating and the reward system. Physiol Behav. 2007; 91(4):449–458. [PubMed: 17543357]

36. Browning CR. The span of collective efficacy: extending social disorganization theory to partner violence. J Marriage Fam. 2002; 64(4):833–850.

37. Fox GL, Benson ML. Household and neighborhood contexts of intimate partner violence. Public Health Rep. 2006; 121(4):419–427. [PubMed: 16827443]

38. Osofsky JD, Wewers S, Hann DM, Fick AC. Chronic community violence: what is happening to our children? Psychiatry. 1993; 56(1):36–45. [PubMed: 8488211]

39. Richters JE, Martinez PE. Violent communities, family choices, and children's chances: an algorithm for improving the odds. Dev Psychopathol. 1993; 5:609–627.

40. Gilbert R, Widom CS, Browne K, Fergusson D, Webb E, Janson S. Burden and consequences of child maltreatment in high-income countries. Lancet. 2009; 373(9657):68–81. [PubMed: 19056114]

41. Reichman N, Teitler J, Garfinkel I, et al. Fragile Families: sample and design. Child Youth Serv Rev. 2001; 23(4–5):303–326.

42. Kimbro RT, Brooks-Gunn J, McLanahan S. Racial and ethnic differentials in overweight and obesity among 3-year-old children. Am J Public Health. 2007; 97(2):298–305. [PubMed: 17194857]

43. National Center for Health Statistics; Centers for Disease Control and Prevention. [Accessed April 1, 2010] CDC growth charts: United States. 2000 May 30. http://www.cdc.gov/growthcharts/.

44. Kessler RC, Ustün TB. The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). Int J Methods Psychiatr Res. 2004; 13(2):93–121. [PubMed: 15297906]

45. Wittchen HU. Reliability and validity studies of the WHO–Composite International Diagnostic Interview (CIDI). J Psychiatr Res. 1994; 28(1):57–84. [PubMed: 8064641]

46. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed.. Washington, DC: American Psychiatric Association; 1994.

47. Coulton CJ, Korbin JE, Su M. Measuring neighborhood context for young children in an urban area. Am J Community Psychol. 1996; 24(1):5–32.

48. Greenland S. Modeling and variable selection in epidemiologic analysis. Am J Public Health. 1989; 79(3):340–349. [PubMed: 2916724]

49. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods. 2008; 40(3):879–891. [PubMed: 18697684]

50. Dallman MF. Stress-induced obesity and the emotional nervous system. Trends Endocrinol Metab. 2010; 21(3):159–165. [PubMed: 19926299]

51. Gibson EL. Emotional influences on food choice. Physiol Behav. 2006; 89(1):53–61. [PubMed: 16545403]

52. O'Connor DB, Jones F, Conner M, McMillan B, Ferguson E. Effects of daily hassles and eating style on eating behavior. Health Psychol. 2008; 27 suppl(1):S20–S31. [PubMed: 18248102]

53. Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. Nutrition. 2007; 23(11–12):887–894. [PubMed: 17869482]

54. Kent A, Waller G, Dagnan D. A greater role of emotional than physical or sexual abuse in predicting disordered eating attitudes. Int J Eat Disord. 1999; 25(2):159–167. [PubMed: 10065393]

55. Rorty M, Yager J, Rossotto E. Childhood sexual, physical, and psychological abuse in bulimia nervosa. Am J Psychiatry. 1994; 151(8):1122–1126. [PubMed: 8037244]

56. Kanter RA, Williams BE, Cummings C. Personal and parental alcohol abuse, and victimization in obese binge eaters and nonbingeing obese. Addict Behav. 1992; 17(5):439–445. [PubMed: 1442237]

57. Smolak L, Murnen SK. A meta-analytic examination of the relationship between child sexual abuse and eating disorders. Int J Eat Disord. 2002; 31(2):136–150. [PubMed: 11920975]

58. Striegel-Moore RH, Dohm FA, Pike KM, Wilfley DE, Fairburn CG. Abuse, bullying, and discrimination as risk factors for binge eating disorder. Am J Psychiatry. 2002; 159(11):1902–1907. [PubMed: 12411226]

59. Thompson KM, Wonderlich SA, Crosby RD, Mitchell JE. Sexual violence and weight control techniques among adolescent girls. Int J Eat Disord. 2001; 29(2):166–176. [PubMed: 11429979]

60. Wonderlich SA, Crosby RD, Mitchell JE, et al. Relationship of childhood sexual abuse and eating disturbance in children. J Am Acad Child Adolesc Psychiatry. 2000; 39(10):1277–1283. [PubMed: 11026182]

61. Felitti VJ. Long-term medical consequences of incest, rape, and molestation. South Med J. 1991; 84(3):328–331. [PubMed: 2000519]

62. Felitti VJ. Childhood sexual abuse, depression and family dysfunction in adult obese patients. South Med J. 1993; 86(7):732–736. [PubMed: 8322078]

63. Springs FE, Friedrich WN. Health risk behaviors and medical sequelae of childhood sexual abuse. Mayo Clin Proc. 1992; 67(6):527–532. [PubMed: 1434879]

64. Grilo CM, Masheb RM, Brody M, Toth C, Burke-Martindale CH, Rothschild BS. Childhood maltreatment in extremely obese male and female bariatric surgery candidates. Obes Res. 2005; 13(1):123–130. [PubMed: 15761171]

65. Aaron DJ, Hughes TL. Association of childhood sexual abuse with obesity in a community sample of lesbians. Obesity (Silver Spring). 2007; 15(4):1023–1028. [PubMed: 17426338]

66. Dansky BS, Brewerton TD, Kilpatrick DG, O'Neil PM. The National Women's Study: relationship of victimization and posttraumatic stress disorder to bulimia nervosa. Int J Eat Disord. 1997; 21(3):213–228. [PubMed: 9097195]

67. Gustafson TB, Sarwer DB. Childhood sexual abuse and obesity. Obes Rev. 2004; 5(3):129–135. [PubMed: 15245381]

68. Brewerton TD, O'Neil PM, Dansky BS, Kilpatrick DG. Links between morbid obesity, victimization, PTSD, major depression and bulimia in a national sample of women [abstract]. Obes Res. 1999; 7(suppl):56S.

69. Chartier MJ, Walker JR, Naimark B. Health risk behaviors and mental health problems as mediators of the relationship between childhood abuse and adult health. Am J Public Health. 2009; 99(5):847–854. [PubMed: 18703446]

70. Steiger H, Gauvin L, Israel M, et al. Association of serotonin and cortisol indices with childhood abuse in bulimia nervosa. Arch Gen Psychiatry. 2001; 58(9):837–843. [PubMed: 11545666]

71. Jimerson DC, Lesem MD, Kaye WH, Brewerton TD. Low serotonin and dopamine metabolite concentrations in cerebrospinal fluid from bulimic patients with frequent binge episodes. Arch Gen Psychiatry. 1992; 49(2):132–138. [PubMed: 1372494]

72. Steiger H. Eating disorders and the serotonin connection: state, trait and developmental effects. J Psychiatry Neurosci. 2004; 29(1):20–29. [PubMed: 14719047]

73. Steiger H, Leonard S, Kin NY, Ladouceur C, Ramdoyal D, Young SN. Childhood abuse and platelet tritiated-paroxetine binding in bulimia nervosa: implications of borderline personality disorder. J Clin Psychiatry. 2000; 61(6):428–435. [PubMed: 10901341]

74. Lumeng JC, Appugliese D, Cabral HJ, Bradley RH, Zuckerman B. Neighborhood safety and overweight status in children. Arch Pediatr Adolesc Med. 2006; 160(1):25–31. [PubMed: 16389207]

75. Burdette HL, Whitaker RC. A national study of neighborhood safety, outdoor play, television viewing, and obesity in preschool children. Pediatrics. 2005; 116(3):657–662. [PubMed: 16140705]

76. Centers for Disease Control and Prevention. Neighborhood safety and the prevalence of physical inactivity: selected states, 1996. MMWR Morb Mortal Wkly Rep. 1999; 48(7):143–146. [PubMed: 10077460]

77. Molnar BE, Gortmaker SL, Bull FC, Buka SL. Unsafe to play? neighborhood disorder and lack of safety predict reduced physical activity among urban children and adolescents. Am J Health Promot. 2004; 18(5):378–386. [PubMed: 15163139]

78. Lee RE, Cubbin C. Neighborhood context and youth cardiovascular health behaviors. Am J Public Health. 2002; 92(3):428–436. [PubMed: 11867325]

79. Gordon-Larsen P, McMurray RG, Popkin BM. Determinants of adolescent physical activity and inactivity patterns. Pediatrics. 2000; 105(6):e83. [PubMed: 10835096]

80. Romero AJ, Robinson TN, Kraemer HC, et al. Are perceived neighborhood hazards a barrier to physical activity in children? Arch Pediatr Adolesc Med. 2001; 155(10):1143–1148. [PubMed: 11576010]

Table 1

Distribution of Sociodemographic Characteristics and Potential Risk Factors for Childhood Obesity by Severity of Maternal Intimate Partner Violence

Characteristic	Total (n=1595)	Children Obese at Age 5 y (n=263)	No IPV (n=807)	Early IPV Only[a] (n=142)	Late IPV Only[a] (n=378)	Chronic IPV[a] (n=268)	P Value[b]
			Maternal				
Age, quartile, y							
20–25	453 (28.4)	68 (25.9)	195 (24.2)	43 (30.3)	120 (31.6)	95 (35.5)	.004
26–28	346 (21.7)	67 (25.5)	169 (20.9)	39 (27.5)	78 (20.6)	60 (22.4)	
29–33	380 (23.8)	60 (22.8)	205 (25.4)	30 (21.1)	89 (23.3)	56 (20.9)	
34–50	416 (26.1)	68 (25.9)	238 (29.5)	30 (21.1)	91 (24.1)	57 (21.3)	
Maternal BMI							
Missing	106 (6.7)	23 (8.8)	44 (5.5)	16 (11.2)	29 (7.6)	17 (6.3)	.24
Normal	417 (26.1)	39 (14.8)	215 (26.6)	32 (22.5)	94 (25.9)	76 (28.4)	
Overweight	406 (25.5)	41 (15.6)	209 (25.9)	31 (21.8)	105 (27.8)	61 (22.8)	
Obese	666 (41.8)	160 (60.8)	339 (42.0)	63 (44.4)	150 (39.7)	114 (42.5)	
Race/ethnicity							
Black	849 (53.2)	134 (51.0)	434 (53.8)	79 (55.6)	211 (55.8)	125 (46.6)	.04
White	327 (20.5)	43 (16.4)	179 (22.2)	21 (14.8)	75 (19.8)	52 (19.4)	
Hispanic	371 (23.3)	84 (31.9)	171 (21.1)	27 (26.1)	79 (20.9)	84 (31.3)	
Other/unknown	48 (3.0)	2 (0.8)	23 (2.9)	5 (3.5)	13 (8.4)	7 (2.6)	
Education							
<High school	636 (39.9)	108 (41.1)	290 (35.9)	58 (40.9)	165 (43.7)	123 (45.9)	<.001
High school graduation	416 (26.1)	70 (26.6)	191 (23.7)	50 (35.2)	106 (28.0)	69 (25.8)	
>High school	543 (34.0)	85 (32.3)	326 (40.4)	34 (23.9)	107 (28.3)	76 (28.3)	
Immigration status							
Born in the United States	1426 (89.4)	233 (88.6)	722 (89.5)	128 (88.7)	339 (89.7)	239 (89.2)	.99
Born outside the United States	169 (10.6)	30 (11.4)	85 (10.5)	16 (11.3)	39 (10.3)	29 (10.8)	
Smoking during pregnancy							
No	1286 (80.6)	217 (82.5)	703 (87.1)	103 (72.5)	288 (76.2)	192 (71.6)	<.001
Yes	309 (19.4)	46 (17.5)	104 (12.9)	39 (27.5)	90 (23.8)	76 (28.4)	

Characteristic	Total (n=1595)	Children Obese at Age 5 y (n=263)	No IPV (n=807)	Early IPV Only[a] (n=142)	Late IPV Only[a] (n=378)	Chronic IPV[a] (n=268)	P Value[b]
No. (%)							
Mother meets depression criteria by CIDI-SF scale							
No	1413 (88.6)	236 (89.7)	750 (92.9)	130 (91.6)	319 (84.4)	214 (79.9)	<.001
Yes	182 (11.4)	27 (10.3)	57 (7.1)	12 (8.4)	59 (15.6)	54 (20.1)	
Child							
Sex							
Girl	779 (48.8)	135 (48.7)	420 (52.0)	71 (50.0)	198 (52.4)	127 (47.4)	.56
Boy	816 (51.2)	128 (51.3)	387 (48.0)	71 (50.0)	180 (47.6)	141 (52.6)	
Low birth weight							
Missing	41 (2.6)	3 (1.1)	19 (2.4)	5 (3.5)	11 (2.9)	6 (2.2)	.48
Yes	140 (8.8)	16 (6.1)	59 (7.3)	14 (9.9)	39 (10.3)	28 (10.5)	
No	1414 (88.7)	244 (92.8)	729 (90.3)	123 (86.6)	328 (86.8)	234 (87.3)	
Takes bottle to bed at age 3 y							
No	1495 (93.7)	239 (90.9)	762 (94.4)	132 (93.0)	355 (93.9)	246 (91.8)	.47
Yes	100 (6.3)	24 (9.1)	45 (5.6)	10 (7.0)	23 (6.1)	22 (8.2)	
Television viewing							
Missing	43 (2.7)	4 (1.5)	15 (1.9)	3 (2.1)	13 (3.4)	12 (4.5)	.01
<2 h/d	549 (34.4)	79 (30.0)	303 (37.5)	46 (32.4)	129 (34.1)	71 (26.5)	
2 h/d	1003 (62.9)	180 (68.5)	489 (60.6)	93 (65.5)	236 (62.4)	185 (69.0)	

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CIDI-SF, Composite International Diagnostic Interview Short Form; IPV, intimate partner violence.

[a] Early IPV indicates exposure to IPV at baseline and/or the 12-month interview only; late IPV, exposure to IPV during the 36- and/or 60-month interview only; and chronic IPV, exposure to IPV both at baseline and/or the 12-month interview and at the 36- and/or 60-month interview.

[b] Differences between multigroup comparisons of IPV (using χ^2 test).

Table 2

Multivariate Models of the Association Between Timing of Maternal Intimate Partner Violence and Obesity in Preschool-Aged Children[a]

	No. (%)		
IPV	Total	Obese	Model 1, OR (95% CI)
No IPV	807 (50.6)	115 (14.3)	1 [Reference]
Early IPV only	142 (8.9)	24 (16.9)	1.12 (0.67–1.85)
Late IPV only	378 (23.7)	62 (16.4)	1.25 (0.88–1.77)
Chronic IPV	268 (16.8)	62 (23.1)	1.80 (1.24–2.61)

Abbreviations: CI, confidence interval; IPV, intimate partner violence; OR, odds ratio.

[a]Model controls for race/ethnicity (black, white, Hispanic, other/unknown), child sex (male, female), maternal age (20–25, 26–28, 29–33, 34–50 years), maternal education (less than high school, high school graduation, beyond high school), maternal nativity (US born, yes or no), child age in months, relationship with father (yes or no), maternal body mass index (normal/underweight, overweight, obese), maternal smoking during pregnancy (yes or no), whether the child takes a bottle to bed at age 3 years (yes or no), average number of hours of child television viewing per day at age 3 years (<2 h/d, 2 h/d), low birth weight (<2500 g, 2500 g), and maternal depression (as measured by a Composite International Diagnostic Interview Short Form cutoff score of 0.5).

Table 3

Multivariate Models of the Association Between Timing of Maternal Intimate Partner Violence and Obesity in Preschool-Aged Children Stratified by Sex[a]

	OR (95% CI)	
IPV	**Boys**	**Girls**
No IPV	1 [Reference]	1 [Reference]
Early IPV only	1.10 (0.53–2.27)	0.99 (0.46–2.13)
Late IPV only	1.09 (0.65–1.82)	1.31 (0.78–2.19)
Chronic IPV	1.66 (0.94–2.93)	2.21 (1.30–3.75)

Abbreviations: CI, confidence interval; IPV, intimate partner violence; OR, odds ratio.

[a]Model controls for race/ethnicity (black, white, Hispanic, other/unknown), child sex (male, female), maternal age (20–25, 26–28, 29–33, 34–50 years), maternal education (less than high school, high school graduation, beyond high school), maternal nativity (US born, yes or no), child age in months, relationship with father (yes or no), maternal body mass index (normal/underweight, overweight, obese), maternal smoking during pregnancy (yes or no), whether the child takes a bottle to bed at age 3 years (yes or no), average number of hours of child television viewing per day at age 3 years (<2 h/d, 2 h/d), low birth weight (<2500 g, 2500 g), and maternal depression (as measured by a Composite International Diagnostic Interview Short Form cutoff score of 0.5).

Table 4

Multivariate Models of the Association Between Maternal Intimate Partner Violence and Obesity in Preschool-Aged Children Stratified by Neighborhood Collective Efficacy and Neighborhood Safety[a]

Any IPV	Neighborhood Safety, OR (95% CI)	
	Safe	**Unsafe**
Never IPV	1 [Reference]	1 [Reference]
Ever IPV	1.38 (0.88–2.17)	1.56 (1.03–2.36)

Abbreviations: CI, confidence interval; IPV, intimate partner violence; OR, odds ratio.

[a]Model controls for race/ethnicity (black, white, Hispanic, other/unknown), child sex (male, female), maternal age (20–25, 26–28, 29–33, 34–50 years), maternal education (less than high school, high school graduation, beyond high school), maternal nativity (US born, yes or no), child age in months, relationship with father (yes or no), maternal body mass index (normal/underweight, overweight, obese), maternal smoking during pregnancy (yes or no), whether the child takes a bottle to bed at age 3 years (yes or no), average number of hours of child television viewing per day at age 3 years (<2 h/d, 2 h/d), low birth weight (<2500 g, 2500 g), and maternal depression (as measured by a Composite International Diagnostic Interview Short Form cutoff score of 0.5).