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

Results From the Fragile Families and Child Well-being Study

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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.


C H I L D H O O D O B E S I T Y I S A growing public health epidemic.1,2 Obese youth are more likely to become obese adults and disproportionately experience cardiovascular3 and metabolic4 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 Forsyth17 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 studies21-24 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.32

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 or to self-soothe by the child. Alternatively, family violence is distressing and may cause affective dysregulation, leading to decreased impulse control and excessive caloric intake. 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 as well as visceral fat accumulation. 

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

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. 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.

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 preg-

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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. 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). 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 at the 36-month in-home assessment.
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’s $\alpha$ for this scale was .92.

### Statistical Analysis

Bivariate analyses of the relation between maternal IPV and covariates (Table 1) were performed using Pearson $\chi^2$ 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.$^{28}$ 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 the continuous CIDI-SF scale) as a mediator of the relation between both chronic maternal IPV and obesity, 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 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 compared with maternal chronic IPV (ad-
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. Maladaptive parenting behavior has been associated with eating disorders in adolescence. Family connectedness, perceived caring, and positive communication were found to be protective against disordered eating in girls. Second, witnessing family violence may be associated with emotional distress and emotion-focused coping using food to self-sooth and address negative emotions. Perceived stress influences feeding behaviors, including type, quantity, and pattern of eating. Glucocorticoids may drive intake of palatable, or “comfort,” foods that decrease feelings of stress and reinforce habitual behaviors at the expense of homeostatic regulators. Eating disturbances such as binge-eating disorder, which is correlated with obesity, and bulimia have been linked to emotional, physical, and sexual abuse. A recent meta-analysis of 53 studies concluded that a small, significant positive relation exists between child sexual abuse and disordered eating, including binge-eating disorder, which is 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. Other traumatic childhood experiences have been linked to altered serotonin and cortisol systems. Overlapping research has shown that bulimia is associated with decreased serotonin metabolites in cerebrospinal fluid, reduced platelet binding of serotonin reuptake inhibitors, reduced density of paroxetine-binding sites, and altered cortisol function. 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 but not all cross-sectional samples. 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.

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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 but not all cross-sectional samples.
Research exploring neighborhood safety and physical and sedentary 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 nearly 60% increase in risk of obesity at age 5 years. 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.

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