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ARTICLE

Obesity Risk for Female Victims of Childhood Sexual Abuse: A Prospective Study

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ABSTRACT

OBJECTIVE. Efforts are under way to articulate environmental, psychosocial, and biological conditions that may predispose the development and maintenance of obesity. There is increasing evidence that adverse childhood experiences such as childhood abuse may be implicated in the development of obesity. Given the dearth of prospective evidence for this link, the objective of this study was to track body mass across development (from childhood, through adolescence, and into young adulthood [ie, ages 6–27]) in a prospective, longitudinal study of abused and nonabused female subjects.

METHODS. Height and weight were obtained for 84 female subjects with substantiated childhood sexual abuse and 89 demographically similar comparison female subjects at 6 points during development. Obesity status was examined at various stages during development, and body-mass growth trajectories were contrasted across the 2 groups. It was hypothesized that, in comparison with their nonabused peers, abused female subjects would be more likely to (1) manifest obesity by early adulthood and (2) manifest high-risk growth trajectories throughout development.

RESULTS. Obesity rates were not different across groups in childhood or adolescence. By young adulthood (ages 20–27), abused female subjects were significantly more likely to be obese (42.25%) than were comparison female subjects (28.40%). Hierarchical linear modeling growth-trajectory analyses indicated that abused female subjects, on average, acquired body mass at a significantly steeper rate from childhood through young adulthood than did comparison female subjects after controlling for minority status and parity.

CONCLUSIONS. Psychosocial difficulties (eg, depression) and psychobiological conditions (eg, hypothalamic-pituitary-adrenal axis dysregulation) that have been shown to be related to both childhood abuse and obesity may help to explain these results. The identification of high-risk growth trajectories may improve health outcomes for victims. Systematic study of the mechanistic pathways and mediating processes that would help to explain the connection between childhood sexual abuse and later obesity is encouraged.

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Dr Noll analyzed the data.

Key Words
abuse, childhood obesity, obesity risk

Abbreviations
CPS— child protective services
SES—socioeconomic status
CDC—Centers for Disease Control and Prevention
zBMI—CDC population BMI *z* scores standardized for age and gender
HLM— hierarchical linear modeling
OR— odds ratio
df— degrees of freedom
CI— confidence interval
HPA— hypothalamic-pituitary-adrenal

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AFFECTING NEARLY 27% of adult Americans, the epidemic of obesity and its well-documented morbidity and mortality risks[1] constitute a major public health concern. Obesity has proved difficult to prevent and treat, in part because its cause is complex and not well understood. It is estimated that 40% to 70% of the variation in obesity is heritable,[2] leaving a substantial portion of obesity antecedents potentially characterized by relatively malleable mechanisms. Therefore, efforts are under way to articulate environmental, psychosocial, and even psychobiological conditions that may predispose one to the development and maintenance of obesity. Among these efforts are increasing mandates to elucidate mechanisms that operate early in development (eg, in childhood and/or adolescence) given that obesity in this period is highly predictive of obesity in adulthood.[3]

Research in the past 15 years has provided increasing evidence for a potential association between adverse childhood experiences and the subsequent development of obesity. In a 1974 population-based study of 756 pupils, teacher ratings of parental neglect were shown to be associated with obesity as assessed 10 years later in young adulthood.[4] In a recent study of extremely obese adult gastric bypass candidates, 69% retrospectively reported some form of childhood maltreatment.[5] Violations of a sexual nature are thought to be of particular salience. Sexual exploitation (eg, adulthood rape or childhood sexual molestation) was a significant correlate of adulthood obesity in a chart-review study of health maintenance organization subscribers,[6] with 60% of victims >50 lb overweight and 25% >100 lb overweight. Adults who reported more severe childhood abuse were significantly heavier than those who reported less severe forms of abuse.[7] Binge-eating disorder, often a comorbid condition that is seen in obese adults,[8] has also been associated with childhood sexual abuse.[9]

To date, there is no prospective evidence for a link between childhood sexual abuse and the subsequent development of obesity. The majority of the extant research is cross-sectional and correlational in nature, using retrospective reports of past abuse. Therefore, statements about a causal relationship between childhood abuse and obesity remain difficult to assert. This longitudinal, prospective study analyzed height and weight data over an 18-year period for 2 groups of female subjects: (1) those who were referred by child protective services (CPS) and had experienced substantiated familial sexual abuse and (2) a demographically similar group of nonabused female peers. The following hypotheses were tested: In comparison with their demographically similar nonabused peers, abused female subjects would be more likely to (1) manifest obesity by early adulthood and (2) manifest high-risk growth trajectories throughout development.

METHODS

Participants

Sexually abused female subjects ($N = 84$) were referred by CPS agencies in the Washington, DC, metropolitan area. Eligibility criteria included (1) age 6 to 16 years, (2) participation within 6 months of disclosure, (3) substantiated sexual abuse, including genital contact and/or penetration, (4) perpetration by a family member (eg, parent, grandparent, older sibling, uncle), and (5) participation of a nonabusing caregiver (usually the biological mother). CPS records indicated that the median age at abuse onset was 7.8 years, the median duration was 24 months, 70% experienced vaginal and/or anal penetration, and 60% of perpetrators were the primary father figure (biological fathers, stepfathers, or mothers' live-in boyfriends). These abuse characteristic were similar to comparable information reported in the 1988 National Incidence Study.[10]

Comparison female subjects ($N = 102$) were recruited via advertisements in newspapers and posters in welfare, child care, and community facilities in the same neighborhoods in which the abused participants lived. Comparison families contacted study personnel and were screened for eligibility, which included having no previous contact with CPS agencies and being demographically similar to a same-aged abused female. Comparison and abused female subjects were similar in terms of residing zip codes, racial/ethnic group, age (6–16 years), predisclosure socioeconomic status (SES), family constellation (1- or 2-parent families), and other nonsexual traumatic events. At some point after entry into the study, 13 comparison female subjects revealed some form of sexual abuse and were dropped from the study, resulting in a comparison sample of 89.

Fifty-four percent of the participants were white (abused: 48; comparison: 46), 43% were black (abused: 32; comparison: 41), 2% were Hispanic (abused: 3; comparison: 2), and 1% were Asian American (abused: 0; comparison: 1). The sample ranged from low to middle SES, with mean Hollingshead[11] scores of ~36 (defined as "blue collar," or working class). There were no statistical differences across groups regarding mean SES or percentage of minority (ie, white versus all minority categories).

Study Design

By design, the study was cross-sequential in nature: recruiting subjects represented a cross-section of development and followed this cross-section over time longitudinally (Table 1). This design permits analyses of both static, cross-sectional within-time effects and dynamic, repeated-measures within-person effects.[12] As illustrated in Table 1, the study began in 1987 (time 1), when participants were a mean age of 11 years. Five follow-up assessments were conducted (times 2–6). More than

TABLE 1 **Summary Statistics for the Sample and Numbers for Analyses**

Parameter	Total ($N = 173$)	Abused ($n = 84$)	Comparison ($n = 89$)
% minority[a]	46	39	51
SES, mean ± SD (range)[b]	36 ± 12 (11–44)	35 ± 14 (10–47)	37 ± 11 (12–43)
Age at assessment (dates), mean ± SD (range)			
Time 1 (1987–1989)	11 ± 3 (6–16)	11 ± 3 (6–16)	11 ± 3 (6–16)
Time 2 (1988–1991)	12 ± 3 (7–18)	12 ± 3 (7–17)	12 ± 3 (7–18)
Time 3 (1990–1992)	13 ± 3 (8–20)	13 ± 3 (8–18)	13 ± 3 (8–20)
Time 4 (1996–1998)	18 ± 4 (11–25	19 ± 4 (11–25)	18 ± 3 (11–23)
Time 5 (1999–2001)	20 ± 3 (13–26	21 ± 3 (13–26)	20 ± 3 (13–26)
Time 6 (2004–2006)	24 ± 3 (18–27)	25 ± 4 (18–27)	24 ± 3 (18–27)
No. per developmental period			
Childhood/early adolescence (age 6–14 y)[c]	146	70	76
Middle/late adolescence (age 15–19 y)[d]	139	66	73
Young-adulthood (age 20–27 y)[e]	156	75	81
No. obese at study entry	22	12	10
No. returning for follow-up 166 (times 4, 5, and/or 6; 96%)	166	82	84
No. used in growth curve analysis[f]	144	69	75
% with all 6 time points	48	42	52
% with only 5 time points	22	34	12
% with only 4 time points	11	13	9
% with only 3 time points	16	9	23
% with only 2 time points	3	2	4

[a] Minority was defined as black (90%), Hispanic (9%), or Asian American (1%).
[b] SES was defined by using Hollingshead ratings.[11]
[c] Three refused/were missing, and 24 were never assessed in childhood.
[d] Two refused/were missing, 28 were never assessed in adolescence, and 4 were invalid because of pregnancy.
[e] Four refused/were missing, 3 were never assessed in adulthood, and 10 were invalid because pregnancy.
[f] Retained sample minus number obese at study entry.

96% of the sample was retained for follow-up assessments at times 4, 5, and/or 6 (abused: 82; comparison: 84). The study received approval from the university institutional review board and a federal certificate of confidentiality.

BMI and Obesity Assessments

Height and weight were obtained at each assessment by trained study personnel using a calibrated upright Health-O-Meter balance beam scale (model 400GZD; Continental Scale Corp, Bridgeview, IL). The exact apparatus and measurement procedures were used across all 6 assessments. At each assessment, participants were weighed and measured 1 time only in street clothing without shoes. These data were used to calculate BMI (kg/m^2). Obesity status was defined as per Centers for Disease Control and Prevention (CDC) guidelines: CDC population BMI *z* scores standardized for age and gender (zBMI) ≥95th percentile when participants were ≤19 years of age and BMI ≥30 once participants reached age 20. Obesity status was calculated for individuals who were assessed during the following distinct developmental periods: childhood to early adolescence (ages 6–14 years), middle to late adolescence (ages 15–19 years), and young adulthood (ages 20–27 years). Female subjects received a score of 1 when they were obese at any assessment point within the age range of each developmental period and a score of 0 when they were not. BMI calculations were considered invalid when participants were pregnant at the time of an assessment. No participant fluctuated from obese to nonobese within the age range of any developmental period.

Statistical Analyses

Because of the slight sample variation in minority status and potential racial variations in BMI,[13] minority status (1 = minority; 0 = white) was covaried in analyses. Because female subjects who have given birth were at greater risk for high BMI,[14] parity history (0 = nulliparous; 1 = primiparous; 2 = multiparous) was an additional covariate in subsequent analyses. All analyses were performed by using SAS 9.13 (SAS Institute, Cary, NC).

With covariates included, logistic regression was used via SAS/Logistic to test for significant group main effects in obesity status and to obtain corresponding likelihood ratios of being obese given group membership (abused = 1; comparison = 0). This analysis was repeated for each of the 3 developmental periods defined, and Bonferroni α corrections were imposed accordingly (ie, $\alpha/3$). Because of the cross-sequential study design, not every participant was assessed at every developmental stage. In addition, a handful of participants refused to be weighed or were pregnant at the time of assessments. Therefore, the sample size fluctuates slightly for each comparison (Table 1).

Hierarchical linear modeling (HLM)[15] via SAS/Mixed was used to estimate average growth trajectories across development on the basis of raw BMI scores arrayed from ages 6 to 27. The degree to which group membership (abused = 1; comparison = 0) could account for individual variation in parameter estimates (eg, intercept, slope) was then evaluated. An advantage of HLM is that maximum likelihood estimation methods can accommodate missing data, thereby allowing the analyst to make use of all available data so that any participant with multiple time points (or ages) can be included in the analysis of the entire trajectory. Using Bayes's estimation, individuals with more data are given more weight in the calculation, a procedure that is preferred to using listwise or pairwise deletion in analyses in which portions of the developmental curve are represented by differing individuals or any given portion of the curve is only sparingly represented.[16] Restricted maximum likelihood estimation with an estimated degrees of freedom procedure[17] was used to arrive at valid parameter estimates under the assumption of ignorable missing data. On the basis of population zBMI percentile scores, 22 participants (abused: 12; comparison: 10) were deemed to be obese at study entry (time 1). These 22 participants were removed from the HLM analysis to characterize growth trajectories for those who were not initially obese, thereby resulting in a total number for HLM analysis of 144. Although not necessarily contiguous, 70% of participants had at least 5 data points for the HLM growth analysis (see Table 1).

RESULTS

Figure 1 includes obesity status by group comparisons in childhood/early adolescence, middle/late adolescence, and young adulthood. During childhood/early adolescence 25.42% of abused and 21.88% of comparison female subjects were obese (odds ratio [OR]: 1.25; degrees of freedom [*df*] = 1141; 95% confidence interval [CI: −0.05 to 3.00; $P = .52$). Later in adolescence, 27.87% of abused and 15.49% of comparison female subjects were obese (OR: 2.03; $df = 1134$; 95% CI: 0.54–4.60; $P = .09$). By young adulthood, 42.25% of abused and 28.40% of comparison female subjects were obese (OR: 2.85; $df = 1151$; 95% CI: 1.06–4.64; $P = .009$). Results thus indicate that obesity rates were not significantly different across groups in childhood or adolescence. However, abused female subjects were 2.85 times more likely to be obese by young adulthood.

Figure 2 depicts the variation in raw BMI trajectories for all participants as well as the mean at each age from 6 to 27. These unconditional HLM results revealed a significant overall omnibus χ^2 ($\chi^2_{5,146} = 508.79$, $P < .0001$) with the linear slope coefficient (1.06) significantly different from 0 ($t_{1,244} = 10.00$, $P < .0001$) and the quadratic coefficient (−0.02) significantly different from 0 ($t_{1,245} = -4.62$, $P < .001$). These results suggest

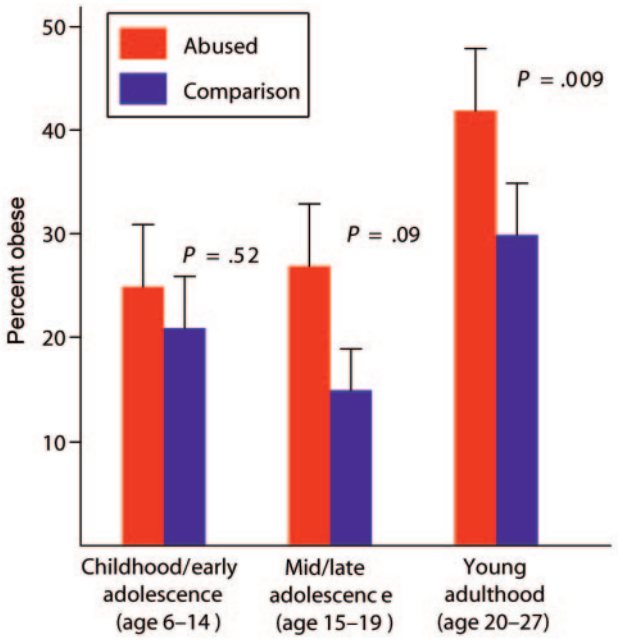

FIGURE 1
Percentage obesity for sexually abused and comparison female subjects according to developmental stage. *P* values correspond to logistic regression log odds parameter estimates of the likelihood of being obese given membership in the abused group. Minority status and parity covaried.

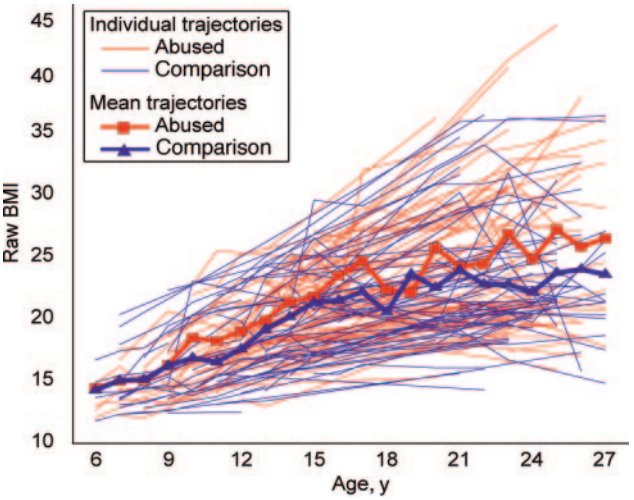

FIGURE 2
Raw BMI trajectories for abused ($n = 69$) and comparison ($n = 75$) female subjects across development. Individual and group mean trajectories are shown.

that the sample, on average, showed a linear positive trend of BMI accumulation across development from childhood to young adulthood but that this trend leveled off in the early 20s.

Figure 3 shows the conditional (by group) HLM results. There was not a significant group × intercept effect ($t_{1,108} = 0.14$, $P = .89$) or a group × quadratic time effect ($t_{1,108} = 0.0014$, $P = .79$). These results indicate that abused and comparison female subjects did not differ with respect to BMI at intercept (age 6) or with respect

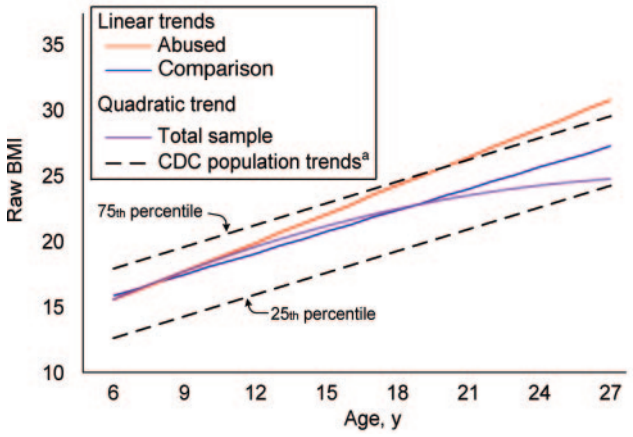

FIGURE 3
Average BMI growth trajectories across development for sexually abused ($n = 69$) and comparison ($n = 75$) female subjects. The average linear slope was significantly steeper for abused female subjects (red) than for comparison female subjects (blue). The average quadratic function was not significantly different across groups (full sample curve is depicted in purple). Minority status and parity covaried. Female subjects who were obese at study entry were excluded. [a] Population growth trends were obtained from the 2000 CDC growth charts (www.cdc.gov/growthcharts). Parallel trajectories representing the 75th and 25th population percentiles for female subjects are depicted (black). The average linear slope for abused female subjects was significantly steeper than the population slope, whereas the linear slope for comparison female subjects was similar to the population slope.

to the quadratic trend in BMI accumulation across development (ie, the leveling off of BMI in young adulthood at approximately age 20). Results did, however, reveal a group × linear time interaction estimate (0.18) that was significantly different from 0 ($t_{1,131} = 3.03$, $P = .01$) with minority status and parity covaried. This result indicates that abused female subjects, on average, acquired body mass at a significantly steeper rate during development (from age 6 to age 27) than did comparison female subjects. Thus, abused female subjects, as a group, were heavier earlier in development and remained heavier throughout development than did comparison female subjects when controlling for minority status and parity. Figure 3 also depicts population statistics representing the 25th and 75th percentile trajectories as provided by the CDC (extrapolated to age 27). The average linear trend for comparison female subjects mirrors that of the population ($t_{1,244} = 0.93$, $P = .34$) and falls well within this boundary. However, the linear trend for abused female subjects is steeper than the CDC population trend across development ($t_{1,244} = 2.99$, $P = .01$) and exceeds the 75th percentile by young adulthood.

DISCUSSION

These results provide some of the first prospective evidence that childhood sexual abuse may place female individuals at inordinately high risk for developing and maintaining obesity. Differential obesity rates were not evident at the start of this longitudinal study, when participants were approximately the mean age of 11. However, those with histories of substantiated familial sexual abuse were more than twice as likely to be obese by young adulthood (mean age 24) as compared with their nonabused peers after controlling demographic variables and parity histories. The observed young-adulthood obesity rate in the group of sexually abused women (just over 42%) is somewhat lower than reports from other studies (eg, 69%)[6]; however, the prospective study design, stringent inclusion criteria of substantiated childhood sexual abuse, and comparison with a demographically similar nonabused peer group constitute methodologic improvements over past studies. We also observed differential rates of body-mass accrual across development, suggesting an increased likelihood that abused female individuals may manifest identifiable high-risk BMI growth trajectories from childhood through adolescence and into young adulthood.

Caveats

It should be made clear that we are not purporting a causal link between sexual abuse and obesity per se but are suggesting a plausible link between the various consequences that are associated with severe childhood adversity and the subsequent development of obesity. Given the documented vast individual differences in responses to childhood adversity[18] and the potential for resilience[19] in many victims, we are also not asserting the development of obesity as an inevitability for abuse victims. We simply wish to underscore the need for systematic study of the mechanistic and mediating processes that would help to explain the connection between childhood abuse and later obesity. Although some of our demographic matching and statistical control represent methodologic improvement over past studies, we were unable to control for several conditions that may place children at high risk for obesity development. For example, familial histories of psychiatric or substance use disorders and numerous alternative adverse environmental factors such as comorbid neglect, poor nutrition and dietary habits, family dysfunction, and greater social isolation may contribute to the development of obesity in this population independent of the experience of childhood abuse. The extent to which sexual abuse is, in itself, a particularly salient risk factor remains theoretical, because the definitive study to compare alternative forms of childhood adversity has not yet been adequately executed. Moreover, given that our sample was recruited from CPS agencies and a nonabusing caregiver was required to participate, abused participants may represent a select set of the abused population (ie, those who had reported, substantiated abuse and whose caregiver was available/supportive).

We were also unable to control for parental weight status, a known potent predictor of child and adolescent obesity that arguably is the result of a gene–environment interaction.[20] However, our intentional omission

of initially obese individuals from the growth analysis should bolster confidence that we have presented BMI trajectories for individuals whose genetic potential for obesity may have been relatively low or unexpressed at study entry. Participant height and weight were measured only once during each assessment visit, the reliability of which could have been improved had we averaged 2 (or even 3) measurements that were obtained at each visit.

Plausible Explanations and Future Research

Caveats aside, these results suggest that the consequences of abuse may amplify or exacerbate the psychosocial risks that are associated with developing or maintaining obesity. For example, depression, body image disturbances, poor peer relations, low self-esteem, and the development of binge-eating disorders are psychosocial conditions that have been studied as both sequelae of abuse[9,18,21–23] and correlates of childhood and adolescent obesity.[24] It has also been suggested that obesity might function as a defense against sexual advances, thereby serving an adaptive purpose for some victims.[9] Research designs that include these various comorbid psychosocial conditions will be essential in any attempt to elucidate the independent contribution of childhood abuse to the development of obesity.

There may also be psychobiological conditions that develop as a result of severe childhood trauma and that may directly contribute to the development of obesity. For example, a dysregulated hypothalamic-pituitary-adrenal (HPA) axis that manifests in the hypersecretion of the stress hormone cortisol has been shown to be a response to severe childhood deprivation.[25] Preclinical studies in rodents and primates found that early deprivation in primates is associated with permanent alterations in hormonal responses to stressors.[26] Inordinately high catecholamine and cortisol activity has been documented in samples of abused children and adolescents.[27–29] As shown in both human and animal models, HPA axis hormones play an important role in the deposition and metabolism of fat.[30–33] Adrenocortical deficiency during weight loss and the high rates of obesity in Cushing syndrome underscore the role of glucocorticoids in obesity.[34] Cortisol promotes differentiation of adipocyte precursors into adipocytes and stimulates lipogenesis in the presence of insulin.[35,36] Several studies have reported a direct association between high cortisol levels and relatively large waist-to-hip ratios in women.[37] Abnormal HPA regulation and heightened cortisol have been implicated in the development of metabolic disorders, abdominal obesity, and type 2 diabetes.[38–40] Therefore, additional examination of conditions that result in HPA disruption (eg, childhood abuse) may advance our understanding of antecedents to obesity.

CONCLUSIONS

Although this study was not designed to give causal conclusions regarding the links between sexual abuse and obesity, the results provide a basis for the additional testing of this hypothesis. Unresolved, persistent, or untreated conditions that are commonly associated with childhood abuse (eg, depression, posttraumatic stress disorder, eating disorders) may be implicated in the development of obesity. Coping mechanisms that serve to eradicate these symptoms as well as reduce the emotional and physiologic stress associated with recovery should be encouraged. Psychological treatment of childhood abuse typically does not extend into adolescence or young adulthood, when issues that are reminiscent of the abuse (eg, the onset of dating, initiation into sexual activity) become developmentally salient. Therefore, these results suggest that childhood abuse treatment extending beyond the acute phases of recovery or that is revisited throughout development may improve health outcomes for abuse survivors.

Pediatricians who are aware of incidences of family violence may better serve patients by closely tracking BMI accumulation across development and suggesting specific treatments that might curtail obesity. With adequate child advocacy support, standard pediatric medical history intake may need to include inquiries into traumatic histories to promote optimal care. Results also suggest that female individuals with traumatic pasts may have particularly high-risk growth trajectories in late childhood and adolescence and that obesity prevention efforts targeting these points in development may be warranted. Such prevention and intervention programs that integrate both psychosocial and biological correlates of obesity may prove more efficacious than those focused on a single process.

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