Intimate Partner Violence and 5-Year Weight Change in Young Women: A Longitudinal Study

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Abstract

Background: Intimate partner violence (IPV) and obesity are national public health problems that are potentially associated. We examined the association between IPV exposure and 5-year weight gain in young women. We also examined whether depressive mood conferred additional increases in weight gain.

Materials and Methods: Analyses were conducted among women in Project EAT, a longitudinal cohort study of weight-related health, which has collected data at three 5-year survey waves: “EAT I” (mean age 15 years), “EAT II” (mean age 19 years), and “EAT III” (mean age 25 years). Height and body weight were self-reported at each survey wave. IPV victimization and depressive mood were assessed on the EAT II survey. The study comprised women with data on IPV and body mass index (BMI) ($N=619$). Linear regression analyses examined (1) 5-year (EAT II to EAT III) BMI change as a function of IPV exposure and (2) 5-year BMI change as a function of depressive mood at EAT II in women with and without IPV exposure.

Results: Almost 20% of the study sample reported IPV. Women exposed to both physical and sexual IPV at EAT II gained 1.1 kg/m^2 (95% CI -0.2 to 2.4) more, over 5 years, than women unexposed to IPV, although this did not reach statistical significance. Among those with IPV exposure, depressive mood at EAT II was associated with an additional increase in BMI of 1.8 kg/m^2 (95% CI 0.2 – 3.4) relative to no depressive mood.

Conclusion: Survivors of IPV with depressive mood may have accelerated weight gain. Trauma-informed obesity prevention strategies may be warranted in this group.

Keywords: intimate partner violence, dating violence, women, weight gain, obesity, depression

Introduction

RAPE AND/OR PHYSICAL ASSAULT by an intimate partner affect 25% of women in the United States and account for 76% of total violence against women.¹ In addition to causing psychological harm and acute physical injuries, emerging research suggests that physical and sexual intimate partner violence (IPV) may also contribute to obesity.^{2–4} Probable mechanisms for an IPV–obesity association include unhealthy strategies for coping with stress such as overconsumption of high-energy foods in an attempt to relieve negative emotions.^{5,6} Depressive mood, which is common among those exposed to IPV^{7,8} and has been linked to weight gain in some (although not all) studies,^{9–11} may also exacerbate unhealthy coping strategies and the weight-related impacts of IPV.

Although there is a large literature on the impact of violence from a parent during childhood on weight-related

outcomes,¹² studies on the association of violence from an intimate partner with weight status are more limited. We know of only a handful of studies that have examined weight as a function of IPV. Six have examined cross-sectional associations of IPV with weight status,^{3,13–16} with four finding a positive association.^{3,14–16} Two studies (one cross sectional, one longitudinal) have examined combined “interpersonal” violence exposure, without differentiating IPV from other types of violence exposure. Both found an association with body weight.^{17,18} To our knowledge, only one longitudinal study has examined the relationship between IPV and weight change. This study, which used data from the National Longitudinal Study of Adolescent Health, assessed physical and sexual IPV in adolescence and its impact on weight during the transition to adulthood; it found that women exposed to IPV had an overall 0.4 kg/m^2 greater 15-year body mass index (BMI) change than women unexposed to IPV.²

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Additional longitudinal studies are needed to confirm these findings.

The current literature also lacks studies examining how depression may operate in concert with IPV exposure to predict weight change. Depression has been found to be associated with problematic overeating^{19–21} and overweight.^{9,22,23} It is plausible that IPV-exposed women who suffer from depressive symptoms are at particularly high risk for weight gain, as depressive symptoms may impede women's capacity to cultivate healthy coping strategies. Depression may also be a marker for the most severe IPV experiences that place women at particularly high risk.

A better understanding of the association between IPV and weight status—including the role of depression—could inform interventions to prevent obesity-related disease risk in adulthood among the large number of women with IPV exposure. For example, existing interventions to improve mental health outcomes after IPV could potentially integrate obesity prevention strategies (e.g., strategies for addressing disordered overeating) to improve overall health outcomes in this high-risk population. Likewise, obesity prevention and intervention strategies might be more effective if appropriately tailored, for example, “trauma-informed,” for those with IPV histories by addressing the unique factors that contribute to weight gain in this population (e.g., depressive symptoms). We therefore aimed to estimate the association of physical and sexual IPV with 5-year weight change among women in Project EAT, a longitudinal cohort focused on weight-related health. In addition, we were interested in whether depressive mood conferred additional weight-related risks; we therefore examined the association between depressive symptoms and 5-year weight change in women with and without IPV exposure.

Materials and Methods

Sample

Our study used data from surveys administered by Project EAT, a longitudinal cohort study of adolescents initially recruited from Minneapolis-St. Paul public middle and high schools serving socioeconomically and racially/ethnically diverse communities. Of the 53 schools that were invited to participate, 31 agreed to take part. The surveys were administered at three time points, each 5 years apart, referred to as EAT I,^{24,25} EAT II,²⁶ and EAT III,^{27,28} respectively. Project EAT I was conducted in the 1998–1999 school year, with surveys administered in schools in health, physical education, and science classrooms. All students in participating classes were eligible to participate. For Project EAT II, 5 years later, the original participants were resurveyed by mailed questionnaire. EAT III resurveyed participants once again at 10 years from baseline; participants were mailed letters inviting them to complete online or paper versions of the Project EAT III survey. Survey development was guided by theory, pilot testing, and focus groups.^{29,30}

Participants aged 18+ years were asked about IPV on the EAT II survey. The sample for the present study included women in this age group ($N=940$), who responded to both the EAT II and EAT III questionnaire ($N=756$). We restricted analyses to women, because preliminary data analysis in men indicated that the numbers reporting IPV, particularly sexual IPV, were too limited ($N=20$) to obtain stable estimates. Participants who were pregnant or breastfeeding at EAT II or EAT III were excluded ($N=102$). In

addition, women who did not complete IPV information at EAT II ($N=21$) or body weight information at either EAT II or EAT III ($N=14$) were not included in the data analysis. This left 619 women for our analytic sample.

Women in our analytic sample were on average 20.3 years old and almost 30% nonwhite (8% black, 3% Hispanic, 13% Asian, and 4% other or mixed). Just over half had parents with less than a college education. Women who responded to all three Project EAT surveys (a requirement for inclusion in our sample) were more likely to be white and have parents with at least a college education than the original sample. Analyses were weighted to adjust for loss to follow-up, which corrected this distribution back to that of the original sample.

Measures

BMI change. BMI change was calculated as the difference between BMI at EAT III and BMI at EAT II in kilograms per meters squared (kg/m^2) based on self-reported weight and height. At EAT III, a substudy was conducted to validate the self-reported weight and height by measuring 125 participants and comparing their actual measurements and their self-reported measurements to establish the accuracy of their BMI; the validation study showed a high correlation between self-reported and measured BMI ($r=0.98$ for females).³¹ We chose to examine BMI change from EAT II to EAT III as our outcome, rather than BMI at EAT III alone, because we could be sure that change in BMI after EAT II (when IPV was measured) reflected the hypothesized temporal order—from IPV to BMI—rather than the reverse.

IPV exposure. IPV was assessed a single time at EAT II. Physical IPV was assessed with a question derived from the Conflict Tactics Scales³² asking participants if a dating partner had ever hit, shoved, held down, or used some other physical force against them either in the past year or before the past year. In a separate sexual IPV question, derived from the Sexual Experiences Survey,³³ participants were asked if they have ever been forced to touch their dating partner sexually or if their dating partner forced some type of sexual behavior on them either in the past year or before the past year. Because small numbers of participants reported past-year IPV, we combined past-year and prior IPV to create a dichotomous variable for physical IPV (ever/never) and sexual IPV (ever/never). In addition, we created a joint physical–sexual IPV variable to assess their independent and combined associations with BMI, categorizing women into four categories: no physical or sexual IPV, physical IPV alone, sexual IPV alone, and both physical and sexual IPV. Finally, we examined the association of any IPV with BMI change, where “any IPV” was defined with an indicator variable equal to one if a woman experienced either physical or sexual IPV.

Depressive mood. Because depression is common among women with IPV exposure,⁷ and has been linked, although inconsistently, to weight status,^{22,23} we were interested in whether depressive mood conferred additional risk of weight gain over and above IPV exposure. We therefore examined the interaction between depressive mood and IPV in the prediction of weight change, and also conducted depression–BMI change analyses stratified by IPV. Depressive mood was measured with the Kandel and Davies Scale³⁴ at EAT II and dichotomized at the recommended cutoff for clinically significant depressive symptoms.

Baseline covariates. We included the following baseline (EAT I) sociodemographic variables in adjusted models because they are potential confounders of the IPV-BMI change relationship: age (continuous), race/ethnicity (white, non-white), and socioeconomic status (SES) based on parental educational attainment (parent education <college degree vs. ≥college degree). We also adjusted for baseline BMI, which may predict BMI trajectory.

Analysis

All analyses were conducted using the Statistical Analysis System (SAS Version 9.3; SAS Institute, Cary, NC). We first assessed the prevalence of IPV as reported by young women in EAT II and calculated the distribution of covariates by IPV exposure. Linear regression analyses were conducted to examine BMI change as a function of IPV. We first ran separate models examining the associations between physical and sexual IPV exposure and BMI change. We then ran a model regressing BMI change on the combined four-category physical and sexual violence variable, as well as a model regressing BMI change on an indicator for exposure to any IPV. For each of our exposures, we ran a crude model and a model adjusting for sociodemographic confounders plus BMI at study baseline. Models were weighted to adjust for loss to follow-up; weights were based on the inverse probability of response to each questionnaire cycle as a function of baseline age, race, and SES. Primary models were run as complete case analyses, that is, with adjusted models excluding observations with missing covariates ($N=44$).

We ran two supplemental models to assess the sensitivity of our findings to various modeling decisions. First, we re-ran our models using missing indicators for the race and SES covariates to assess whether our results would change when

the full sample was retained in adjusted models. Second, we excluded women with extreme values for the outcome (BMI change $\pm >3$ SD of the mean), to ensure our results were not driven by outliers.

To assess whether depressive mood was associated with BMI change in women with and without IPV, we first ran a model regressing 5-year BMI change on depressive mood, IPV, and their interaction term (plus sociodemographics and baseline BMI). We then regressed 5-year BMI change on depressive mood among all women, and separately in women with and without any IPV exposure (either physical or sexual), adjusting for sociodemographics and baseline BMI.

Results

Descriptive statistics

Of our analytic sample of 619, a total of 121 women (19.5%) reported a history of IPV. Of those women, 51 (8.2%) reported a history of sexual IPV and 100 (16.2%) reported a history of physical IPV. Of the women who reported IPV, 70 (57.9%) reported only physical IPV, 21 (17.3%) reported only sexual IPV, and 30 (24.8%) reported both types of IPV. The percent of women reporting IPV on the EAT II survey was similar between our analytic sample (*i.e.*, those who responded at all three time points) and those who responded only at EAT I and EAT II. Demographic and personal characteristics across each IPV category are shown in Table 1.

IPV and BMI change

On average, women in the sample gained 1.9 kg/m^2 in the 5 years between EAT II and EAT III. In crude estimates, women who were exposed to physical IPV gained 0.9 kg/m^2 (95% CI 0.2–1.6) more over 5 years than women who were

TABLE 1. COVARIATE AND BMI DISTRIBUTIONS BY INTIMATE PARTNER VIOLENCE EXPOSURE

Variable	Physical IPV		Sexual IPV		Joint physical and sexual IPV exposure			
	No (N = 519)	Yes (N = 100)	No (N = 568)	Yes (N = 51)	None (N = 498)	Physical abuse only (N = 70)	Sexual abuse only (N = 21)	Physical and sexual abuse (N = 30)
Continuous variables, mean (SD)								
Age at IPV assessment	20.3 (0.7)	20.4 (0.8)	20.3 (0.8)	20.4 (0.8)	20.3 (0.7)	20.3 (0.8)	20.3 (0.6)	20.5 (0.9)
BMI at study baseline (age 14–18 years)	22.0 (3.8)	23.0 (4.9)	22.2 (4.1)	22.0 (3.9)	22.1 (3.9)	23.2 (5.1)	21.0 (2.5)	22.7 (4.5)
BMI at IPV assessment (age 19–23 years)	23.5 (4.4)	23.9 (5.2)	23.6 (4.6)	23.3 (4.2)	23.6 (4.5)	23.8 (5.5)	22.4 (3.2)	24.0 (4.8)
BMI 5 years after IPV assessment (age 24–28 years)	25.2 (5.7)	26.5 (7.2)	25.4 (5.9)	26.1 (6.4)	25.3 (5.7)	26.0 (7.2)	23.6 (3.4)	27.8 (7.4)
Categorical variables, N (column%)								
Race								
White	371 (72.0)	69 (71.1)	401 (71.4)	39 (78.0)	356 (72.1)	45 (66.2)	15 (71.4)	24 (82.8)
Nonwhite	144 (28.0)	28 (28.9)	161 (28.7)	11 (22.0)	138 (27.9)	23 (33.8)	6 (28.6)	5 (17.2)
Parents < college education	259 (50.4)	64 (64.7)	296 (52.6)	27 (54.0)	251 (50.9)	45 (64.3)	8 (38.1)	19 (65.5)
Depressive mood at age 19–23 years	114 (22.1)	38 (38.8)	133 (23.7)	19 (37.3)	109 (22.1)	24 (35.3)	5 (23.8)	14 (46.7)

BMI, body mass index; IPV, intimate partner violence.

TABLE 2. DIFFERENCE IN BMI CHANGE FROM EAT II (AGE 19–23 YEARS) TO EAT III (AGE 24–31 YEARS) AS A FUNCTION OF LIFETIME IPV EXPOSURE AS OF EAT II

IPV exposure	N	Model			
		Crude		Adjusted for sociodemographics ^a and baseline BMI	
		BMI difference	95% CI	BMI difference	95% CI
Physical IPV					
No	519	0.0	REF	0.0	REF
Yes	100	0.9	0.2 to 1.6	0.7	−0.1 to 1.4
Sexual IPV					
No	568	0.0	REF	0.0	REF
Yes	51	0.7	−0.3 to 1.7	0.6	−0.5 to 1.6
Joint physical and sexual IPV					
None	498	0.0	REF	0.0	REF
Physical IPV	70	0.5	−0.3 to 1.4	0.5	−0.4 to 1.3
Sexual IPV	21	−0.6	−2.2 to 1.0	−0.1	−1.8 to 1.6
Both	30	1.7	0.4 to 2.9	1.1	−0.2 to 2.4
Any IPV					
No	498	0.0	REF	0.0	REF
Yes	121	0.6	−0.0 to 1.3	0.4	−0.3 to 1.1

^aAge (continuous in years), race (white vs. nonwhite), and parental education (<college vs. ≥college).

unexposed to physical IPV (Table 2). When adjusted for sociodemographics and baseline BMI, the association was slightly attenuated to 0.7 kg/m² (95% CI −0.1 to 1.4). For sexual IPV, BMI increased 0.7 kg/m² (95% CI −0.3 to 1.7) more among exposed versus unexposed women; adjustment attenuated this to 0.6 (95% CI −0.5 to 1.6).

When physical and sexual IPV were examined jointly, women who were exposed to both physical and sexual IPV had a crude 1.7 kg/m² (95% CI 0.4–2.9) greater change in BMI relative to women without any IPV exposure. After adjustment, this association was attenuated to a suggestive, but nonstatistically significant, 1.1 kg/m² (95% CI −0.2 to 2.4) difference in weight change in women with physical plus sexual IPV. Supplemental analyses yielded largely similar results, although using missing indicators rather than excluding observations with missing covariates in complete case analyses yielded a slightly larger, and statistically sig-

nificant, effect estimate for the joint impact of physical plus sexual IPV (Supplementary Table S1; Supplementary Data are available online at www.liebertpub.com/jwh).

Depressive mood and BMI change

Analyses of the association of depression with 5-year weight change among women with and without IPV exposure revealed a substantial increase in weight change associated with depression among women with IPV (Table 3). When adjusted for age, race, SES, and baseline BMI, the estimated 5-year BMI increase associated with depression among women with IPV was 1.8 kg/m² (95% CI 0.2–3.4); in contrast, there was no difference in BMI change associated with depression among women without IPV. The interaction between IPV and depressive mood was statistically significant ($p = 0.004$).

Discussion

In this study, we found that the combination of physical and sexual IPV is associated with a suggestive increase in 5-year BMI change among young adult women. Even after adjustment for age, race, baseline SES, and baseline BMI, the association of joint exposure to both physical and sexual IPV with BMI change remained substantial (>1 kg/m²), although no longer statistically significant, possibly due to limited power. Differences in BMI of this magnitude are associated with increases in type 2 diabetes risks of 10%–20% in women (with greater risks in some subpopulations such as Hispanics and Asians).³⁵ If these accelerated weight trajectories continue for women with histories of IPV, then they are likely to be at even greater risk for weight-related disease as they enter middle and older age.

Among women with IPV exposure, depressive mood was associated with a large additional increase in 5-year BMI change. It is not clear from our data whether depressive mood is simply a marker of more severe IPV, or plays a more mechanistic role (*e.g.*, is a pathway by which IPV influences

TABLE 3. DIFFERENCE IN BMI CHANGE FROM EAT II (AGE 19–23 YEARS) TO EAT III (AGE 24–31 YEARS) AS A FUNCTION OF DEPRESSIVE MOOD AT EAT II IN ALL WOMEN, AND STRATIFIED BY IPV EXPOSURE

IPV stratum and depression category	N	BMI difference ^a	95% CI
All women			
No depressive mood	461	0.0	REF
Depressive mood	152	0.5	−0.1 to 1.1
Women with no IPV			
No depressive mood	385	0.0	REF
Depressive mood	109	0.0	−0.7 to 0.7
Women with IPV			
No depressive mood	76	0.00	REF
Depressive mood	43	1.8	0.2 to 3.4

^aAdjusted for age (continuous in years), race (white vs. nonwhite), and parental education (<college vs. ≥college).

weight gain because depressive symptoms undermine healthy coping strategies). In either case, these findings suggest that women with IPV histories who experience depressive mood would benefit substantially from targeted obesity prevention, although future work is needed to identify the types of prevention strategies that are most likely to be effective in this group. If depressive mood is indeed on the pathway between IPV and weight gain, then intervening on depressive symptoms themselves may be an important obesity prevention tool in women with IPV histories. The reasons for the observed lack of association between depressive mood and BMI change in women without IPV exposure are unclear, but may reflect milder symptoms in this group, or different phenotypic expressions of depressive symptoms.

Our results are consistent with prior literature, which has found that IPV exposure is associated with weight-related behaviors³⁶ and obesity-related chronic diseases such as hypertension, diabetes, stroke, and coronary artery disease.^{13,37,38} Our results are also consistent with a cross-sectional study, in which a substantial association was found between IPV exposure and obesity ($\geq 30 \text{ kg/m}^2$) in women; however, this study was among Egyptian women, and it is unknown if the results are generalizable to women in the United States. A single prior longitudinal study also found small accelerations in 15-year BMI change associated with a history of IPV in adolescence.² Several other published studies have focused on the association of IPV exposure on psychological outcomes of abuse, for example, depression, anxiety, and somatization.^{7,8,39,40} To our knowledge, this study is only the second longitudinal examination of weight change as a function of IPV.

Our study was limited by the use of single-item questions rather than full validated scales to measure physical and sexual IPV. Use of full IPV scales may have picked up additional IPV experiences that participants did not report on our single-item measures; it is unclear whether such experiences would be related to BMI in the same way as IPV captured by our single-item measures. Nonetheless, the prevalence of IPV found in our sample was similar in our study to estimates in national samples,^{2,41} providing some reassurance that our measures are performing similarly to more extensive scales. Self-reported BMI may be prone to error; while validation studies in the overall sample suggest accurate self-report, women with depression may be less accurate in their reporting and influence the results of our stratified analysis. Restriction of our analytic sample to those who responded to all three Project EAT questionnaires resulted in a select group; in particular, comparisons of our analytic sample to responders to the original Project EAT survey suggest that our analytic sample was more likely to be white and have higher parental education than the original Project EAT sample. However, after weighting to correct for loss to follow-up, the analytic sample was similar to the original respondents on these characteristics. Restricting our analysis to women, due to the small number of IPV reports among men in this cohort, is an additional limitation, and future studies should examine these associations in larger samples of men.

Despite its limitations, our study has several notable strengths, including a community-based sample, prospectively measured IPV, and BMI self-reports that perform well against measured BMI in the overall sample. Furthermore, longitudinal data allowed for identification of the temporal

order from IPV to weight change; our ability to adjust for baseline BMI, which may predict later BMI trajectory, provides additional support for this temporal order. Although small sample sizes in our IPV-exposed group resulted in wide confidence intervals around our IPV estimates, our results suggest a clinically important association that warrants further investigation.

Conclusions

The present study suggests that women who experience physical and sexual IPV in conjunction with depressive mood are significantly more vulnerable to accelerated weight gain than women who do not have this constellation of experiences. Greater understanding of this association may inform development of obesity prevention strategies tailored for this high-risk group; such strategies might include trauma-informed approaches that address potential contributors to IPV-related weight gain such as depressive mood and unhealthy coping strategies.

Author Disclosure Statement

No competing financial interests exist.

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