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Alcohol and Binge Eating as Mediators between Post-traumatic Stress Disorder Symptom Severity and Body Mass Index

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Abstract

Objective—Sexual minority women are at elevated risk for obesity, as well as of exposure to traumatic events. Rates of obesity are elevated in individuals with PTSD, but we know little about why this relationship exists. Behavioral mechanisms, such as eating patterns and alcohol use, are possible explanations that would be clinically useful to identify.

Methods—We longitudinally investigated binge eating and alcohol use as mediators of the relationship between PTSD symptom severity and body mass index (BMI) in a large sample of young adult sexual minority women (N = 425). We assessed PTSD symptom severity at baseline, binge eating and alcohol use 12-months later, and BMI 24-months after baseline.

Results—Using a multiple mediator model, we found that higher baseline PTSD symptom severity was significantly associated with higher BMI 2 years later, operating through binge eating behavior, but not through alcohol use. Exploratory moderator analyses found that this effect was higher for those with lower baseline BMI.

Conclusions—Results suggest that higher PTSD symptoms are longitudinally associated with increased BMI, and that binge eating behavior is one factor that explains this relationship.

Keywords

alcohol; binge eating; BMI; sexuality; women

Introduction

Sexual minority women, defined as women who self-identify as lesbian or bisexual or who engage in same-sex sexual behavior, tend to have higher body mass index (BMI) and

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associated elevated rates of obesity as compared to women in the general population (1). Sexual minority women also have increased risk for psychopathology thought to be due to increased stress related to minority status and the experience of discrimination (2). Posttraumatic stress disorder (PTSD) and alcohol use, in particular, both of which are highly prevalent among sexual minority women (3, 4), may confer specific risk for obesity (5, 6).

Sexual minority women are at elevated risk for both trauma exposure and PTSD (3). PTSD has been associated with obesity in both cross-sectional (7) and longitudinal studies (8) of women. In studies looking at trauma exposure as a predictor of disordered eating, PTSD emerges as a significant mediator of the relationship (9), suggesting that PTSD specifically, and not trauma exposure more generally, predicts unhealthy eating behavior (10). Moreover, the predictive relationship between PTSD and obesity remains even when demographics, depression, and substance use are controlled for (11), suggesting PTSD is a strongly relevant factor. Despite this, PTSD has not been examined as a specific risk factor for obesity among sexual minority women.

The relationship between PTSD and elevated BMI is likely explained by both physiological and health behavior pathways. Behavioral patterns are a clinically useful explanation for empirical study, as interventions targeted at these behaviors can help improve physical health for vulnerable individuals with PTSD. For example, PTSD is associated with bulimia nervosa and binge eating disorder (BED) in women (10). Binge eating behavior is a risk factor for elevated BMI (12), and theories suggest that disordered eating patterns may function as a maladaptive strategy to cope with the distress of having PTSD (13). Although, a recent study did not find that the presence of BED significantly mediated the relationship between PTSD and obesity (11), despite a significant relationship between PTSD and BED, this study was limited by looking only at BED status and by being cross-sectional, despite testing mediation processes. Thus, examining binge eating behavior more generally, as opposed to just diagnostic status, and looking prospectively at ability to predict BMI may be more informative in understanding eating behavior as a possible explanation of the relationship between PTSD and obesity. Notably, binge eating is elevated in sexual minority women compared to heterosexual women (14). However, the relationship between PTSD and binge eating behavior has not been directly examined in a sample of sexual minority women to date.

Sexual minority women also have higher rates of alcohol misuse as compared to heterosexual women (4). PTSD is thought to increase risk of alcohol misuse via self-medication (15), which may also contribute to increased rates of obesity. Laboratory-based studies suggest alcohol acutely disinhibits eating and that alcohol calories are added to total daily caloric intake rather than being compensated for through reduced food intake (16). However, among the general population research support for the relationship between alcohol use and obesity is mixed (6).

Limited research has explored whether health-risk behaviors, including eating behavior and alcohol use, explain the relationship between PTSD and negative health outcomes, many of which are linked to elevated BMI (17), both generally, and within sexual minority women, specifically (18). This research has produced conflicting results, perhaps due to the diffuse

nature of trauma exposure sequelae (19) and the distal nature of most negative health outcomes (i.e., it can take decades for disease to emerge). As BMI is an established risk factor for distal negative health consequences that have been linked to PTSD (20), it may be more valuable to focus on BMI as the outcome of interest and to explore mechanisms that explain the relationship between PTSD and BMI. Identifying intermediate behavioral mechanisms, like binge eating and alcohol use that may help explain relationships between PTSD and BMI over time would inform indicated prevention approaches for individuals at risk for, or already experiencing, PTSD. Despite clear associations and a strong theoretical rationale for exploring binge eating and alcohol use behaviors as explanatory pathways between PTSD and BMI, prior studies have not simultaneously compared these behaviors to determine their relative contributions to BMI. Moreover, no studies have examined these relationships among individuals who may be at greater risk for PTSD, alcohol misuse, and obesity, such as sexual minority women. Thus, the purpose of this study is to test these relationships prospectively among young adult sexual minority women. It was hypothesized that frequency of binge eating and total weekly alcohol consumption assessed at 12-month follow-up would each independently mediate the relationship between baseline PTSD symptom severity and BMI at 24-month follow-up.

Method

Participants and Procedures

Paid online advertisements were placed on Facebook and Craigslist to recruit a national sample of self-identified sexual minority women ages 18–25 for a longitudinal study (N = 1,067) on women's health behaviors (21). As part of a planned missingness procedure, only 742 women were administered an assessment of binge eating behavior at 12-month follow-up, making them eligible for the present investigation. Of those who received the binge eating assessment, survey completion at 12-month follow-up was 72.4% (N = 537) and completion of 24-month follow-up was 63.3% (N = 470). Of these women, 429 (57.8%) provided complete data on model variables across the baseline, 12-month and 24-month surveys.

All assessments were completed online. Participants were paid $30 for completing each assessment and a $35 bonus for completing all assessments. All study procedures were approved by the University's Institutional Review Board.

Measures

Demographics—See Tables 1 and 2.

PTSD symptom severity—The PTSD Checklist-Specific version (PCL-S; 22) was used to assess the 17 DSM-IV symptoms that participants might have experienced in relation to a traumatic event. Each item is rated using a 5-point Likert-type scale where 1 = *Not at all* and 5 = *Extremely*, and items are summed to create a total score (range = 17–85). This measure had strong internal consistency (α=.95). The PCL-S has previously demonstrated strong convergent validity (r = .85–.93) with other measures of PTSD (22).

Alcohol use—Participants completed the Daily Drinking Questionnaire (DDQ; 23), which assesses average consumption in number of standard drinks on each day of typical week. Average total drinks per week was calculated as the sum of the total number of drinks reported. Prior studies have shown the DDQ to be highly correlated with other self-report measures of alcohol consumption (24).

Binge eating—To assess frequency of binge eating, participants were presented with the following question (25): "Sometimes people will go on an 'eating binge' where they eat an amount of food that most people, like their friends, would consider to be very large, in a short period of time. During the past year, how often did you go on an eating binge?" Response options were *never, less than once a month, 1–3 times a month, once per week,* and *more than once per week*. As the focus of this study was on the act of binge eating (i.e., consumption of an objectively large amount of food in a short period of time) versus the presence of binge-eating-related psychopathology (i.e., bulimia nervosa or BED), and a binge eating episode often occurs without feelings of loss of control (26), consideration of the frequency of binge eating was not predicated on the presence of loss of control in the present analyses. This item has been previously validated against a structured interview of disordered eating behaviors (26).

BMI—BMI was calculated based on self-reported height, weight and sex using the following formula: $BMI = 703 \left(\frac{weight\ in\ lbs.}{(height\ in\ inches)^2} \right)$. See Table 1 for descriptive statistics related to BMI.

Analyses

All analyses were completed in SPSS using the PROCESS (version 2.15) custom dialog for multiple mediation analyses (27, 28). PROCESS requires complete data for each participant and uses a bootstrapping resampling method (28). Prior to analyses, regression diagnostics were assessed and 4 participants were identified as influential outliers and excluded (final sample N = 425).

Results

Descriptive statistics, bivariate correlations and attrition analyses

Means, standard deviations, and correlations among model variables are presented in Table 2. Fisher *r* to *z* transformation identified a significant difference in 2 of 10 coefficients between completers and non-completers: (a) baseline PTSD symptom severity and binge eating at 12 months ($z = 2.75$, $p = .003$) and (b) baseline age and baseline total weekly drinking ($z = -2.56$, $p = .005$). No other correlations were statistically different between completers and non-completers at baseline. Therefore, the spread of the data in associations among model variables was largely similar across completers and non-completers.

We used logistic regression to evaluate whether completion status (0 = complete case; 1 = missing measure) was predicted by baseline and 12-month measures of model variables. PTSD symptom severity significantly predicted completion status, Wald χ^2 ($df = 1$) = 19.602, $p < .001$. The odds of attrition were 1.02 times greater among those with a one unit higher baseline PCL score. PTSD symptom severity accounted for 3.54% of the variation in

attrition, and the model with PTSD symptom severity minimally improved classification of completion status (an increase from 58.5% to 59.4% correctly classified). Baseline drinking also significantly predicted completion status, Wald χ^2 (df = 1) = 4.548, p = .033. The odds of attrition were 1.01 times greater among those with a one unit higher baseline weekly drinking score. Weekly drinking accounted for less than 1% of the variation in attrition, and the model with total weekly drinking minimally improved classification of completion status (an increase from 58.1% to 58.4% correctly classified). No measures of model variables at 12-months predicted completion status.

Multiple mediation analysis

A multiple mediator model was selected for simultaneous estimation of two mechanisms (binge eating and drinking behavior, assessed at 12 months) expected to explain the relationship between baseline PTSD symptom severity and BMI 24 months later (PROCESS Model 4; 28, 29). Although other mechanisms may explain this relationship, we chose to focus on two externalizing behaviors that could be the target of preventive intervention in this population.

Controlling for age[1], PTSD symptom severity at baseline accounted for a significant proportion of the variance in binge eating at 12 months, R^2 = .074, F(2, 422) = 13.92, p < .001, with higher PTSD symptom severity predicting more frequent binge eating (B = .0181, SE = .0034), t = 5.2606, p <.001 (BCa 95% CI = .0114–.0249). Controlling for age, PTSD symptom severity at baseline did not account for a significant proportion of the variance in total weekly drinking at 12 months. Age, PTSD symptom severity, weekly drinking and binge eating were entered together in a multiple regression model that accounted for a small, but significant proportion of the variance in BMI at 24 months R^2 = .043, F(4, 420) = 3.67, p = .006, and we observed a significant, positive, direct effect of binge eating at 12-months (B = 1.2672, SE = .4821), t = 2.6268, p = .009 (BCa = .3196–2.2148) on 24-month BMI. The direct effect of baseline PTSD symptom severity on 24-month BMI was not significant when the effect of the mediator was removed.

Multiple mediation analyses to simultaneously compare the hypothesized longitudinal pathways between PTSD symptom severity and BMI operating indirectly through binge eating and drinking behavior, controlling for age, were conducted (see Table 3 for point estimates and standard errors from the model). This approach to examining theorized causal mechanisms is increasingly common (28, 29). Only the specific indirect effect of binge eating (B = .0230, SE = .0089, BCa = .0076–.0436) was significant, whereas weekly drinking was not. Figure 1 shows the significant effect of binge eating at 12 months on 24-month BMI.

[1]We ran an additional model to rule out the possibility that depression, as assessed by the Center for Epidemiologic Studies Depression Scale (CESD), which shares features with PTSD may have accounted for the findings. When baseline CESD scores were entered as a covariate in the model, the indirect effect of PTSD on BMI through binge eating remained significant. Thus, findings from the model with CESD scores are not reported here, but are available upon request from the first author.

Post hoc moderated mediation analyses

In addition to planned analyses, we ran post hoc analyses to determine whether the process by which PTSD is related to BMI over time was the same across all participants. We assessed whether baseline BMI moderated the effect of PTSD operating through 12-month binge eating on 24-month BMI (28; PROCESS Model 7). Baseline PTSD and BMI measures each significantly predicted 12-month binge eating, as did the product of baseline PTSD and BMI. Binge eating differed by baseline BMI across participant subgroups at the mean (M = 26.65) and plus (BMI = 34.66) or minus (BMI =18.64) one standard deviation (SD). The indirect effect of baseline PTSD on 24-month BMI through 12-month binge eating depended on the level of baseline BMI with significant positive effects observed for participants at −1 SD (B = .0219, SE = .0102, BCa = .0062–.0472) and at the mean (B = .0102, SE = .0067, BCa = .0003–.0267); index of moderated mediation (B = −.0015, SE = .0008; BCa = −.0035 – −.0003). There was not a significant effect for participants with a baseline BMI at +1 SD.

Discussion

The current analyses provided partial support for study hypotheses. Specifically, when frequency of binge eating and weekly alcohol use were evaluated simultaneously as potential mediators of the relationship between PTSD symptom severity and BMI, binge eating alone emerged as an explanatory mechanism. This is an important finding as it provides us with crucial information on a behavior that if modified might significantly reduce the risk of the negative outcome of increased BMI for sexual minority women with PTSD, a population at risk for both. The current finding also lends support to theories that binge eating may be a strategy employed to cope with symptoms of PTSD. Moreover, it shows that this type of coping is prospectively associated with negative physical health outcomes (i.e., higher BMI), demonstrating the importance of targeting PTSD symptoms as well as associated maladaptive behaviors.

Binge eating is generally perceived as an undesirable, non-normative behavior, detracting from quality of life (30). Most women with bulimic-type eating behaviors do not seek help specifically for an eating disorder, but the majority do seek some type of general mental health services (31), which present opportunities for intervention. Primary care physicians are likely to be the first or only source of mental health care for most individuals (32); however, screening for binge eating is not yet routine and binge eating may not be recognized by practitioners as a contributing factor to obesity (33). Providers also may not assess sexual identity, sexual behavior, or trauma history. For sexual minority women this may create a barrier to obtaining appropriate care. Compared to heterosexual women, sexual minority women are less likely to seek out preventive care and screening and are more likely to receive suboptimal care (1, 34). At minimum, findings from this study suggest physicians would be advised to screen for binge eating in women who present with symptoms of PTSD, even if their sexual identity has not been disclosed.

Post hoc analyses revealed that the indirect effect of PTSD on BMI through binge eating was conditional. Average, or below average BMI participants reported significantly more frequent binge eating at 12 months and had higher BMIs at 24 months. This path was not

significant for the participants with a baseline BMI one SD above the mean. Thus, the importance of screening for binge eating among women presenting with symptoms of PTSD may be even higher for those with lower or average BMIs, which are probably even less likely to be asked about their eating habits than a woman with a higher BMI. As these analyses were exploratory, future research is warranted to replicate these findings.

Future research may also wish to explore the possible reciprocal relationship between PTSD and binge eating and its relation to BMI. While PTSD symptoms would seem like the most upstream intervention target based on the model tested in this study, and treating PTSD may affect symptoms shared by PTSD and disordered eating (35), it is also possible that interventions designed to change binge eating (e.g., meditation; 36) may positively impact PTSD symptoms. Better understanding the complex longitudinal relationships between PTSD symptoms, binge eating and BMI is necessary to determine the most effective components and course for intervention.

The absence of a relationship between PTSD symptoms and alcohol use is not wholly unexpected and has been found in other studies of young adults (37), and prior research exploring the relationship between alcohol use and BMI is mixed. In certain ways, the demonstration of a clear causal relationship from PTSD symptoms to BMI through binge eating has more clinically significant implications for intervention, as alcohol use is more likely to be considered normative (less likely to be perceived a problematic or as requiring intervention) by women in this age group (38).

This study had several strengths, including focusing on a proximal predictor of future negative health consequences (BMI), simultaneously examining modifiable behaviors (binge eating and alcohol use) that might explain the relationship between PTSD and BMI and could be targeted through preventive intervention, and evaluating these relationships in an underserved sexual minority population at higher risk for PTSD, binge eating, alcohol use and obesity. Additional strengths of this study were the use of a nationally-recruited sample, which increases generalizability, and use of a prospective design, with mediators assessed at a separate, intervening time point between baseline and final follow-up. While this is a significant improvement over other studies in this area that have used cross-sectional designs, it must be noted that binge eating was not assessed at baseline. Thus, the possibility remains that binge eating could have been static from baseline to 12-months, functioning instead as a baseline predictor. Future research in this area should address this possibility.

Additional limitations must also be considered. Specifically, as all assessment occurred online, data validity could be questioned. To address this concern, study staff obtained phone numbers and collateral contact information from participants to help verify identity and reduce missing data. Changes in demographic variables were also assessed longitudinally, to identify and remove participants with inconsistent responding, as this is one indicator of compromised data. Even among participants with consistent reporting, PTSD symptom severity, drinking, binge eating and BMI were all self-reported and may differ from values that would have been obtained via clinical examination or real-time assessment methods. However, this limitation is minimized through assurances of confidentiality and the use of psychometrically-validated measures, which increase validity of self-report (39), and

through prior research showing that self-report of BMI is generally accurate as an indicator of health risk when controlling for sociodemographic characteristics like age and sex (40) as was done in this study.

The nature of the sample, while a strength, could also be considered a limitation. It is possible that the findings may only apply to young adult sexual minority women. This may be especially true regarding the absence of expected effects related to alcohol consumption. Although the majority of young adults consume alcohol at least on occasion, the fact that 30.8% of the sample was under the legal drinking age of 21 at 12-month follow-up may have contributed to the lower level of drinking reported. Thus, it is possible that alcohol consumption may emerge as important mediator of the relationship between PTSD symptoms and BMI in adult sexual minority women. The findings may also differ for older sexual minority women, who may have dealt with differing stressors and societal views on their sexual orientation than those affecting younger women, given rapid changes in cultural attitudes about sexual minorities within the United States. Findings also may not generalize to sexual minority men, who do not appear to have the same risk of obesity noted among sexual minority women, but who may have increased risk for other types of disordered eating. For heterosexual women, it would be important to replicate these findings, as they are also at high risk for trauma exposure and PTSD, but may differ in the experience of stressors thought to motivate binge eating among sexual minority women.

Additional limitations include potential for self-selection bias via attrition, which could limit internal validity. However, attrition does not always lead to imbalances. Participants who had complete data over 24 months had significantly lower PTSD symptom severity at baseline, which increases concerns about generalizability of our findings for participants at higher levels of PTSD symptom severity. At the same time, use of logistic regression identified only one significant predictor of completion status, and the small amount of explained variance (4%) indicates that the magnitude of the differences between completers and non-completers is likely small. However, as the PROCESS routine used to test study hypotheses does not support imputed data (27), it was not possible to analyze data from incomplete cases.

In summary, to our knowledge this study is the first to test two common yet challenging health behaviors, binge eating and alcohol use, as explanatory mechanisms of the relationship between PTSD and a physical health indicator, BMI. Our findings highlight binge eating behavior as a significant factor that explains this relationship in sexual minority women, a group at higher risk for PTSD and obesity, which opens up possibilities for prevention and intervention targets that could help combat the known problem of obesity among those with mental illness.

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What is already known about this subject?

- PTSD is associated with higher BMI, which individually and jointly confer greater risk for negative health outcomes, but we know little about why this relationship exists.
- Binge eating and alcohol use are two plausible causal mechanisms that explain the relationship between PTSD and higher BMI, and have been explored separately, often using cross-sectional data.
- Sexual minority women are a higher risk for PTSD, binge eating and alcohol use, and evidence higher rates of BMI in at least some studies.

What does your study add?

- To the best of the authors' knowledge, this study is the first to examine the potentially competing roles of binge eating and alcohol use as mediators of the relationship between PTSD and BMI simultaneously using a prospective (vs. cross-sectional) design.
- This study uses a large nationally-recruited sample of young adult sexual minority women, a population at high risk for obesity, alcohol misuse, and PTSD, to explore these relationships in a population that is routinely understudied and underserved.
- The findings from this study have direct implications for indicated prevention; specifically, targeting binge eating, which is not usually a part of routine health screening, may have greater impact on BMI than targeting alcohol use, which is more frequently assessed in health settings.

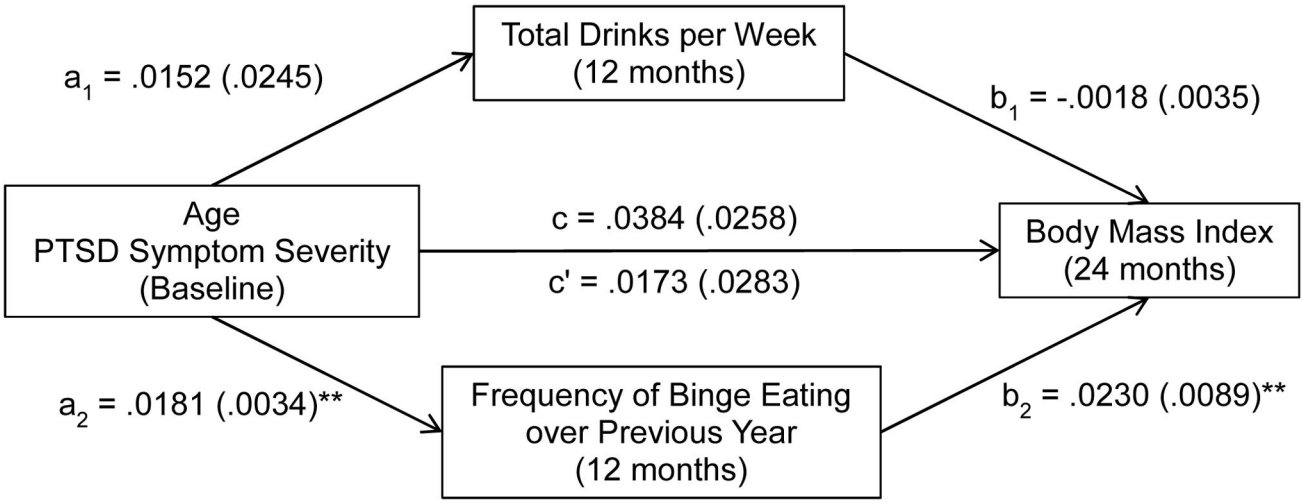

Figure 1.

Longitudinal Multiple Mediation Model of the Indirect Effects of Drinking and Binge Eating on the Relationship between PTSD and BMI, Controlling for Age

Note: ** $p < .01$. N = 425. Baseline age included as a covariate. Independent variable = PTSD symptom severity at baseline. Dependent variable = BMI at 24 months. Mediating variables = Total drinks per week reported at 12 months and frequency of binge eating over the previous year reported at 12 months. Unstandardized beta coefficient for each path (Standard Error of the Estimate); a paths are direct effects, b paths are indirect effects, c′ is the direct effect and the c path is the total effect.

Table 1

Sample Demographics

Variable	% / value
Racial Identity	
White or Caucasian	72.9
Black or African American	8.0
Asian or Asian American	2.1
American Indian or Alaskan Native	0.2
Multiracial	13.9
Ethnic Identity	
Hispanic/Latinx	9.3
Sexual Orientation	
Bisexual	48.0
Lesbian	37.2
Queer	8.7
Pansexual	2.3
Questioning	2.1
Two-spirit	0.2
Asexual	0.2
Baseline BMI Mean (SD)	26.65 (8.01)
BMI <18	5.2
BMI 18 to <25	52.0
BMI 25 to <30	17.6
BMI 30 to <35	8.0
BMI 35+	17.2
24-Month BMI Mean (SD)	27.21 (8.45)
BMI <18	5.9
BMI 18 to <25	49.6
BMI 25 to <30	16.7
BMI 30 to <35	8.7
BMI 35+	19.1

Note. N = 425. Racial identity and sexual orientation categories do not sum to 100%; the remainder reported “other” on these items.

Table 2

Means, Standard Deviations and Pearson Correlations among Mediation Model Variables

	All (N = 1067)	Non-Completers (N = 313)	Completers (N = 425)	OR [95% CI]	1	2	3	4	5
Baseline Variable									
1. PTSD Symptom Severity	37.90 (17.16)	41.28 (17.69)	35.57 (16.53)	1.020 ** [1.011–1.028]	-	.166 **	.047	-.001	.272 **
2. Total Weekly Drinking	8.33 (11.48)	9.45 (12.33)	7.57 (10.96)	1.014 ** [1.001–1.027]	.135 *	-	-.074	.222 **	.038
3. Body Mass Index	26.89 (8.02)	26.83 (7.67)	21.39 (2.07)	1.003 [0.984–1.022]	.058	.012	-	.092	.172 **
4. Age	21.37 (2.09)	21.28 (2.10)	20.87 (2.08)	.972 [0.906–1.042]	.016	.033	.202 **	-	.009
12 month Variable									
5. Binge Eating	0.74 (1.08)	0.66 (0.95)	0.76 (1.10)	.918 [0.730–1.155]	−.058	−.012	.253 *	0.175	-

Notes.

** $p < .01$.

* $p < .05$.

Mean (Standard Deviation). Binge eating was only assessed at 12-month follow-up. OR = Exponentiated beta. 95% CI = Upper and lower bounds of 95% confidence interval. Correlation matrix shows *completers* above the diagonal and *non-completers* below the diagonal.

Table 3

Mediation Model of the Effect of PTSD Symptom Severity on Body Mass Index through Drinking and Binge Eating, Controlling for Age

	Coefficients		BCa 95% CI	
	Point Estimate	***SE***	**Lower**	**Upper**
1. Baseline Age	.2401	.2143	−.1813	.6614
2. Baseline PTSD Symptom Severity (path c')	.0173	.0283	−.0384	.0730
3. Weekly Drinking (path b_1)	−.0018	.0035	−.0113	.0029
4. Binge Eating (path b_2)	.0230	.0089	.0076	.0436*

Note. N = 425. 5000 bootstrap samples to generate bias-corrected and accelerated bootstrap 95% confidence intervals (BCa 95% CI).

*CIs not containing zero are significant at the 0.05 level.