Testing Competing Mediation Models of the Effects of Weight Bias Internalization and Weight Suppression on Disordered Eating in Young Adults

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A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Psychology
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Abstract

The current study investigated a novel theoretical model of longitudinal relations between weight bias internalization (WBI), weight suppression, and disordered eating. Undergraduate students (N = 787) completed surveys at three time points. Path analysis was used to test competing models representing the temporal order of effects between WBI and weight suppression on disordered eating symptoms. Neither pathway was supported by the data, indicating that WBI and weight suppression were not related over time. However, results supported distinct effects of WBI versus weight suppression on the prediction of overall disordered eating, binge eating, and body dissatisfaction over six-month follow up. Taken together, these findings suggest that reducing WBI and identifying individuals who are weight suppressed may be important eating disorder prevention targets for undergraduate students across gender and weight status.

Keywords

disordered eating, weight bias internalization, weight suppression, binge eating, body dissatisfaction, longitudinal, path analysis
Summary for Lay Audience

Young adults are at high risk for the development of problematic eating behaviors which can have negative effects on physical and mental health. They also highly endorse negatively biased stereotypes and attitudes associated with being a higher weight, which are related to many negative effects including applying these negative attitudes to oneself, a process known as weight bias internalization. Weight bias internalization may motivate efforts to maintain a lower weight, which may lead to increasingly extreme eating and weight-control behaviors over time. The current study aimed to integrate distinct lines of research on the effects of weight bias internalization and weight loss maintenance, by examining whether these variables are related to each other and to problematic eating behaviors over time in young adults across weight status.

Undergraduate students completed three surveys over a six-month period, which included questionnaires regarding their current and previous weight in adulthood, internalization of weight-biased attitudes, and eating behaviors. Results indicated that, contrary to hypotheses, weight bias internalization and maintaining a lower weight were not related to each other. However, they each independently contributed to greater problematic eating behaviors over the follow-up period. In addition, weight bias internalization led to greater loss of control over eating and body dissatisfaction. Notably, weight loss maintenance also led to greater body dissatisfaction over time, suggesting that efforts to maintain a lower weight may have negative rather than positive effects on body-related attitudes. These findings have important implications for efforts to reduce weight bias internalization and prevent harmful eating behaviors in young adult populations of diverse weight ranges.
Acknowledgments

First and foremost, I am extremely grateful to my supervisor Dr. Lindsay Bodell for her unrelenting mentorship and belief in my ability throughout the duration of this project. Your expertise and integrity have had a profound influence on my development as a researcher. I also wish to thank the other members of my committee, Drs. Rachel Calogero, Samantha Joel, and Eva Pila, for their insightful advice and feedback. Thank you to Dr. Erin Kaufman for her guidance on earlier drafts of this thesis, which was influential in shaping my voice as a writer. Jason Chung, Justin Hopper, and Brianna Meddaoui also provided helpful comments and suggestions on drafts of this work. I am also grateful to my research assistant Jina Kim, whose help was instrumental in completing this project. Sincere thanks also to my lab mate Genevieve Bianchini for her kind help and support, and to my lab ‘twin’ Abbigail Kinnear, who has been a source of constant moral support, inspiration, and friendship throughout my graduate studies. As a first-generation student, I am indebted to my family for their unwavering support of my education. The completion of this thesis also would not have been possible without the encouragement and patient listening ear provided by my partner James Bushell. Finally, I would like to thank the individuals who took the time to participate in my study, without whom this research could not be completed.
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Introduction

Weight-based bias and discrimination against higher weight individuals is pervasive, and weight-biased attitudes are highly endorsed among youth and adults (Ambwani et al., 2014; Bucchianeri et al., 2016; O’Keeffe et al., 2020). Such attitudes include beliefs that weight is under personal control, and that those who fail to control their weight are lazy, impulsive, and lack self-discipline (Hunger et al., 2020; Pearl & Lebowitz, 2014; Rubino et al., 2020). There is strong evidence that experiencing weight bias (e.g. experiencing social rejection or overt discrimination based on one’s weight) is associated with a range of negative psychosocial and health outcomes, including disordered eating (Cheng et al., 2018; Emmer et al., 2020; Sutin et al., 2020; Vartanian & Porter, 2016; Wu & Berry, 2018). Moreover, the pervasiveness of weight bias may lead to weight bias internalization (WBI), in which individuals endorse and apply weight biases to themselves and devalue themselves based on their weight, which can further deteriorate well-being (Durso & Latner, 2008; Meadows & Calogero, 2018; Puhl et al., 2018). Like weight bias, WBI may have important implications for the development of disordered eating, such that holding such beliefs about oneself may lead to efforts to maintain a lower weight and subsequent disordered eating.

Disordered eating includes both cognitive (e.g., body dissatisfaction, shape/weight concerns) and behavioral (e.g., binge eating, skipping meals) symptoms that are not necessarily severe or frequent enough to meet criteria for a full eating disorder. Despite not reaching diagnostic thresholds, disordered eating has been associated with negative outcomes, including risk of developing an eating disorder and impaired quality of life (Mond et al., 2013; Pearson et al., 2017; Stice et al., 2017). The transition to university is a high-risk period for body dissatisfaction and disordered eating among young adults,
with one study finding that 40.2% of undergraduate men and women engaged in binge eating, and 30.2% reported engaging in compensatory behaviors such as self-induced vomiting, use of diet pills or laxatives, or excessive exercise (Girz et al., 2013; Lipson & Sonneville, 2017). Further, engaging in weight-control behaviors such as using diet pills and laxatives increases the risk of receiving a diagnosis of an eating disorder in young adulthood (Hazzard et al., 2021; Levinson et al., 2020).

Research indicates that WBI mediates associations between experiencing weight bias and disordered eating in higher-weight samples (Himmelstein et al., 2019; O’Brien et al., 2016). WBI has also been cross-sectionally associated with low self-esteem, poor quality of life, greater cardiometabolic risk, and symptoms of depression, anxiety, and disordered eating among higher-weight samples (Durso et al., 2012; Mensinger et al., 2016; Pearl & Puhl, 2018; Pearl et al., 2017; Schvey et al., 2013). However, extant investigations of WBI regularly restrict samples to individuals with higher weights (i.e., body mass indices [BMI] > 25 kg/m²). Although levels of WBI generally increase as BMI increases, WBI is highly prevalent across persons of varying weight status (Pearl & Puhl, 2018; Pearl et al., 2021; Puhl et al., 2018). Further, many studies rely on self-reported weight to determine weight category; however, WBI may have the effect of skewing respondents’ perceptions of their weight status. For example, Schvey & White (2015) found that respondents with underweight-to-normal BMIs (i.e., between 15-24.99 kg/m²) who endorsed high levels of WBI perceived themselves to be overweight. Most importantly, growing evidence indicates that WBI is associated with reduced quality of life, body dissatisfaction and eating pathology in lower weight samples (Burnette & Mazzeo, 2020; Lee et al., 2019; Pearl & Puhl, 2014; Purton et al., 2019; Romano et al.,
2021; Schvey & White, 2015). Taken together, these findings suggest that internalizing weight bias is problematic regardless of one’s weight status; thus, targeting WBI may reduce disordered eating risk broadly.

Although previous studies have established cross-sectional associations between WBI and disordered eating, more recent evidence also supports WBI as a longitudinal predictor of negative eating outcomes. In a 30-day ecological momentary assessment (EMA) study of higher weight participants, Carels et al. (2019) found that participants who reported daily high levels of WBI also reported greater overeating. The specific WBI domain of fear of experiencing weight bias has also been found to predict food addiction symptoms at follow up (Meadows & Higgs, 2020b). High WBI may also attenuate the effects of weight-neutral health promotion programs among higher-weight women (Mensing et al., 2016; Mensinger & Meadows, 2017). However, few studies have examined mechanisms that may underlie associations between WBI and eating pathology across weight status. In a cross-sectional study of two samples of over 1000 undergraduates, Romano et al. (2021) found support for a model of sequentially mediated effects between experiencing weight bias from others, internalizing weight bias, body dissatisfaction and disordered eating behaviors. The model was supported across weight status, with the exception that the association between body dissatisfaction and restricting behaviors was stronger among participants with underweight or normal category BMIs compared to higher weight participants. Additionally, in a sample of adolescents across weight status, Ahorsu et al. (2020) found that WBI indirectly predicted binge eating six months later via psychological distress and food addiction symptoms.
Beliefs in the controllability of one’s weight may also be an important mechanism linking WBI with eating behavior. Previous research has found that experimental exposure to weight-biased messaging increases motivation to lose weight as a means to avoid further bias, but also decreases perceived capacity to do so (Major et al., 2020). In higher-weight samples, high WBI has been negatively associated with perceived control over weight as well as intentions to engage in physical exercise (Fung et al., 2020; Reinka et al., 2021). By contrast, the combination of high WBI and weight-controllability beliefs in persons with lower BMIs is positively associated with both restrictive and binge eating (Reinka et al., 2021). WBI may mediate associations with weight-controllability beliefs and eating behavior, creating a “double-edged sword effect” in which WBI initially encourages restrictive eating to lose weight, but also increases binge eating via emotional distress (Reinka et al., 2021). Taken together, these findings suggest that WBI may motivate weight-loss behaviors that later contribute to disordered eating. However, the longitudinal pathways by which WBI may be related to efforts to lose weight and disordered eating remain unclear.

**Weight Suppression**

In order to lose weight, individuals with high WBI may attempt to reduce their food intake. Although caloric restriction can result in short-term weight loss, it also predicts future binge eating and weight gain (Stice et al., 2011). Further, significant weight loss (i.e., 10% or more of body weight) triggers biological processes that promote weight restoration, including reduced metabolic rate and increased appetite (Rosenbaum et al., 2010). Greater weight loss triggers greater activation of biological forces towards weight gain (Doucet et al., 2000; Stice et al., 2011). Psychological factors may also
contribute to weight regain: breaking a restrictive diet (e.g. by eating a ‘forbidden’ food) can lead dieters to temporarily abandon restriction and indulge in overeating, a response known as the abstinence violation effect (Carels et al., 2004; Linardon, 2018; Polivy & Herman, 2020; Stroebe et al., 2008). The combination of strong fears of weight gain and/or desires to lose weight with powerful psychological and physiological pressures to restore eating and regain weight are proposed to create a biobehavioral bind, in which individuals must engage in increasingly extreme eating behaviors to maintain a lower weight over time (Lowe, 1993; Lowe et al., 2018). The state of maintaining weight lost from a previous higher weight is referred to as weight suppression and is operationalized as the discrepancy between an individual’s highest adult weight (excluding weight gained due to pregnancy or medical conditions) and their current weight.

Weight suppression may be particularly pernicious during young adulthood, as this period is marked by both normative weight gain and high prevalence of disordered eating behaviors (Girz et al., 2013; Lipson & Sonneville, 2017; Smith-Jackson & Reel, 2012; Vadeboncoeur et al., 2015). Intentional weight suppression is common among undergraduate students and associated with greater body dissatisfaction, disordered eating and weight gain (Burnette et al., 2019; Chu et al., 2021; Lowe et al., 2019; Yoon et al., 2019). Further, the prevalence of unhealthy weight control behaviors such as self-induced vomiting and laxative or diet pill use appears to peak in late adolescence and young adulthood and is associated with depression and low self-esteem (Stephen et al., 2014). Importantly, associations between weight suppression and eating pathology appear to be similar across men and women, but may be stronger among persons with higher BMIs or a history of being a higher weight (Burnette et al., 2017, 2018). Weight suppression also
predicts the onset and maintenance of bulimia nervosa (BN) symptoms over 10- and 20-year follow up (Bodell et al., 2017; Keel & Heatherton, 2010). Thus, weight suppression appears to be a key risk factor for eating pathology (Gorrell et al., 2019). Although researchers have examined physiological and neuropsychological mechanisms of weight suppression (Bodell & Keel, 2015; Keel et al., 2019), few have investigated potential psychological mechanisms underlying the relation between weight suppression and disordered eating. One study found that increased drive for thinness mediated the association between weight suppression and BN symptoms (Bodell et al., 2017).

However, the potential role of WBI in efforts to suppress weight is under-examined. To date, the literatures on WBI and weight suppression are largely separate. Only one study has examined the potential association between WBI and weight suppression (Burnette & Mazzeo, 2020). The authors found that both variables predicted unique variance in dietary restraint among undergraduate men and women. However, although both WBI and weight suppression predicted binge eating behaviors in men, only WBI predicted binge eating in women. These findings suggest that the strength of associations between WBI and weight suppression may vary for specific eating behaviors (i.e., restrictive vs uncontrolled eating). However, the study did not explicitly examine whether or how WBI and weight suppression may influence one another. Importantly, the direction of effects between WBI and weight suppression could not be determined due to the use of a cross-sectional design. Longitudinal data are critical to establish whether WBI and weight suppression are related over time.
Modeling Relations Among WBI, Weight Suppression and Disordered Eating

Although findings from Burnette & Mazzeo (2020) suggest that WBI and weight suppression covary, cross-sectional data preclude any claims about whether these variables influence each other over time, or the potential temporal order of effects between them. In order to establish the direction of mediation effects, researchers must collect data on variables of interest at multiple time points using longitudinal designs (Hayes, 2018; MacKinnon et al., 2007; Selig & Preacher, 2009). Figures 1 and 2 depict alternative theoretical models of relations among WBI, weight suppression and eating pathology across three time points. Specifically, the relations between WBI and weight suppression in Models A and B reflect two competing models of temporal precedence, in which the effects of one construct on disordered eating is mediated via the other.

Figure 1

*Model A: Effect of Weight Bias Internalization (WBI) on Disordered Eating (DE) Mediated via Weight Suppression (WS)*
Consistent with Model A, Burnette & Mazzeo (2020) suggest individuals with high WBI may be motivated to suppress weight to reduce weight-related distress or avoid returning to a previous higher weight. Here, the effect of WBI at Time 1 on eating pathology at Time 3 is mediated by weight suppression at Time 2. This model is supported by evidence that the relation between WBI and restrictive eating behaviors is stronger at lower BMIs (Romano et al., 2021). Other studies find that participants with higher levels of WBI are more likely to report dieting in the previous year, and report strong motivations to lose weight to avoid future weight bias (Major et al., 2020; Puhl et al., 2018). Findings that body dissatisfaction mediates associations between WBI and disordered eating may suggest that body dissatisfaction resulting from WBI precipitates
engagement in disordered eating behaviors to change body shape or weight (Romano et al., 2021).

By contrast, Model B proposes that attempts to suppress weight at Time 1 result in eating pathology at Time 3 via increases in WBI at Time 2. For example, experiencing praise or reduced stigma following weight loss may drive greater fears of weight regain, particularly for individuals who previously had a higher weight status (Bodell et al., 2017; Burnette et al., 2019). In support of this interpretation, Schvey & White (2015) found that individuals who were currently dieting to lose weight reported higher levels of WBI than non-dieters even after controlling for BMI. Failed attempts to control weight may also exacerbate WBI, and even successful weight loss may not lead to decreases in WBI (Pearl et al., 2018; Reinka et al., 2021). Previous work indicates that WBI mediates associations between experience of weight bias and eating pathology — suggesting that disordered eating may occur in response to internalized self-devaluation associated with being the target of weight bias (Durso et al., 2012; O’Brien et al., 2016). Cycles of weight loss and weight gain driven by the biobehavioral bind may exacerbate self-devaluation associated with WBI, motivating increasingly extreme dieting attempts that lead to the development of an eating disorder (Lowe et al., 2018).

The Current Study

The current study seeks to establish the temporal order of effects of weight suppression and WBI on eating pathology by testing competing paths in a longitudinal mediation model. The study recruited undergraduate students, as this population traditionally falls within the median age of onset range for eating disorder (i.e., late adolescence/early adulthood), and well as exhibiting high reported prevalence of
disordered eating symptoms (Lipson & Sonneville, 2017; Solmi et al., 2021; Udo & Grilo, 2018). The current study allows examination of how WBI and weight suppression influence each other over time, accounting for baseline functioning. Further, the use of repeated measures will allow for investigation of stability within each construct over time. Longitudinal investigation contributes to greater understanding of potential pathways between WBI and weight suppression and their prospective effects on eating pathology.

To test models of temporal precedence, undergraduate students completed a series of online surveys including a baseline assessment (Time 1), and follow-up assessments after three (Time 2) and six months (Time 3). Length of follow-up period was chosen in part due to feasibility considerations; however, similar studies support the detection of changes in weight and eating disorder symptoms in college students over similar time periods (Cooley & Toray, 2001; Delinsky & Wilson, 2008; Girz et al., 2013; Sala & Levinson, 2016; Striegel-Moore et al., 1989). At each time point, participants completed measures of WBI and eating disorder symptoms, and their self-reported weight and height was used to calculate BMI and weight suppression indices.

Consistent with previous research, I predicted that higher levels of WBI and weight suppression at Time 1 would be associated with greater disordered eating symptoms at Time 1. Further, I predicted that WBI and weight suppression at Time 1 would predict increases in WBI, weight suppression, and disordered eating at Times 2 and 3. Given the dearth of research examining associations between WBI and weight suppression, no specific hypotheses were made regarding the relative strength of paths in
the longitudinal model (i.e., whether the relations in Model A would provide a stronger or weaker fit to the data compared to Model B).

Findings from the follow-up measures would contribute to a novel longitudinal model that integrates the disparate theoretical accounts of weight stigma and eating disorders in the extant literature. This longitudinal model also elucidates how WBI and weight suppression each contribute to increased risk of disordered eating over time. Results also would have important implications for developing comprehensive prevention programs that can interrupt the harmful effects of weight bias and reduce eating disorder risk.
Method

This study employed a longitudinal design involving surveys at three time points: baseline, three-month follow up, and six-month follow up.

Participants and Procedure

Participants ($N = 786$) were recruited via mass emails sent to all undergraduate students at Western University as well as posts on Western student-related Facebook groups. Participants were oversampled at baseline to account for attrition over follow-up. Undergraduates aged 17-25 and fluent in English were eligible to participate. Of participants invited to complete the follow-up surveys, 282 (35.9%) participants completed the three-month follow up, and 266 (33.8%) participants completed the six-month follow up. However, 46.8% completed baseline and at least one follow-up survey. Demographic characteristics of the sample are listed in Table 1. There were no significant differences in age, BMI, gender, or ethnicity among participants at each time point. There was a significant difference in participants’ year of study ($\chi^2 = 29.47, p = .043$), with post-hoc cellwise analysis indicating that a smaller proportion of first year students completed follow-up surveys compared to students in other years ($p = .001$).
### Table 1

**Demographic Characteristics of Sample Across Surveys**

<table>
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<tr>
<th>Variable</th>
<th>Baseline</th>
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<th>6 Month Follow-Up</th>
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</table>

*Significant at $p < .05$
Participants provided electronic consent and completed the baseline survey via Qualtrics (https://www.qualtrics.com). At the end of the survey, participants entered their email to be contacted for the follow up surveys, and their email was assigned to a randomly generated five-digit numerical ID. Numerical IDs were entered into a draw for one of four $25 Amazon gift cards for completing baseline questionnaires. Participants were invited to complete follow-up surveys via Qualtrics at three and six months following their baseline participation. Participants were invited to participate in the six-month follow up even if they had not completed the three-month follow-up. Numerical IDs were used to identify responses across follow-ups. Those who completed the three-month follow up were entered in a draw for one of four $50 gift cards; those who completed the six-month follow up entered a draw for one of two $100 gift cards. The baseline survey included all questionnaires and demographic items and took approximately 45-60 minutes to complete. The follow-up surveys did not include measures of demographics or dieting/weight history, and each follow-up survey took approximately 30-45 minutes to complete.

**Measures**

**Demographics**

At baseline, participants indicated their age in years, gender identity (female, male, nonbinary/genderqueer, or another identity not listed), racial/ethnic identity (“With which ethnic group(s) do you identify? (select all that apply)”), current height, and year of studies. At baseline and follow-up, participants reported their self-perception of their weight (“Given your age and height, you would say that you are currently: 1)
underweight 2) about the right weight or 3) overweight”). This item has been used in previous studies examining weight status perceptions (Edwards et al., 2010).

**Dieting and Weight Suppression**

Weight suppression and dieting were assessed using the Dieting and Weight History Questionnaire (DWHQ; Witt et al., 2013). The DWHQ includes 16 items assessing current weight, lowest previous weight and highest previous weight (e.g., “What is the most you have ever weighed since reaching your current height [do not count any weight gains due to medical conditions or medications]”). Respondents are also asked to indicate whether the difference between their highest and current weight was due to intentional weight loss. The questionnaire also includes items assessing stability of weight over the past 6 months, purpose of dieting (i.e., to lose weight or avoid gaining weight), length of diet and frequency of weight loss attempts. Although psychometric characteristics of this measure are not well established, the DWHQ was designed to standardize questions often used inconsistently in studies examining respondents’ history of dieting or weight loss. Both weight suppression and dieting are established risk factors predicting the onset of disordered eating (Bodell et al., 2017; Keel et al., 2019; Liechty & Lee, 2013; Pearson et al., 2017; Stice et al., 2017; Stice, et al., 2011). Previous studies have also found strong correlations between self-reported and measured weight (r = .99; Lin et al., 2012; Pursey et al., 2014), including our lab (Szczygłowski et al., 2021), supporting the reliability of self-report assessments of weight. All items of the DWHQ were included at baseline. At three- and six-month follow-up, participants were only asked items regarding their current weight and how their weight had changed over the last three months (i.e., “Which of these statements best
describes what has happened to your weight during the past 3 months? A) My weight has stayed about the same, B) I’ve been losing weight, C) I’ve been gaining weight or D) My weight has fluctuated a lot” [this was altered from the original item which asks about the past 6 months]).

**Calculating Weight Suppression.** Participant’s self-reported highest weight since adulthood, current weight at each time point, and height were used to calculate current and highest Body Mass Index (BMI). Consistent with recommendations from previous research, absolute and relative indices of weight suppression were then calculated (Burnette & Mazzeo, 2020; Piers et al., 2019; Schaumberg et al., 2016). Absolute weight suppression is represented as a difference score between highest and current BMI (highest BMI - current BMI). However, this score does not account for variation in highest previous weight across individuals, which may be problematic, given that the impact of weight loss may be greater depending on the BMI at which one begins to lose weight (e.g., losing weight when one is already at an underweight BMI vs overweight BMI; Lowe et al., 2018; Piers et al., 2019; Schaumberg et al., 2016). Relative weight suppression accounts for this variation by calculating the *percentage* decrease in weight from previous highest weight ((absolute weight suppression/highest BMI) *100). For both absolute and relative weight suppression scores, higher values indicate greater weight lost from highest previous weight. Negative values (i.e., indicating that current weight was higher than the highest previous weight provided) were recoded as zero.

**Weight Bias Internalization**

WBI was measured at all time points using an adapted version of the Weight Bias Internalization Scale (WBIS-M; Pearl & Puhl, 2014), altered to be inclusive of
respondents across the weight spectrum. The WBIS-M includes 11 items on a 7-point scale from 1= “Strongly disagree” to 7= “Strongly agree”. Example items include “I am less attractive than most other people because of my weight” and “I don’t feel that I deserve to have a really fulfilling social life, because of my weight”. Two items are reverse scored. A total score was computed using the average score across items, with higher scores reflecting greater WBI. Both the original and modified scales show good convergent, discriminant and predictive validity (Durso & Latner, 2008; Hilbert et al., 2014; M. S. Lee & Dedrick, 2016; Pearl & Puhl, 2014). The WBIS-M also shows high internal consistency in community and undergraduate samples (α = .89-.94; Burnette & Mazzeo, 2020; Pearl & Puhl, 2014). Internal consistency of the WBIS-M in the current study at baseline was excellent (α = .95).

**Disordered Eating**

Disordered eating was measured at all time points using the Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013). The EPSI is a self-report measure including 45 items rated on a 5-point scale from 0= “Never” to 4= “Very Often”. Responses across all items were summed to create a total score. Items were also scored according to eight subscales: body dissatisfaction, binge eating, purging, restricting, cognitive restraint, negative attitudes towards obesity, muscle building and excessive exercise. Higher total and subscale scores indicate greater disordered eating attitudes and behaviors. Reliability and validity of the EPSI is well-supported and gender norms have also been developed in young adult samples (Coniglio et al., 2018; Forbush et al., 2013, 2014). Cronbach’s alphas in the baseline sample were .90 for the total score and ranged from .73 (muscle building) to .90 (negative attitudes toward obesity) for the subscales.
**Attention Checks**

Consistent with recommendations by Abbey & Meloy (2017), multiple items were included in each survey to confirm that participants were attending to the survey. Each survey included a directed query item asking participants to select an arbitrary response (e.g., “Please select ‘somewhat disagree’”). At the end of each survey an “honesty check” was also included, in which participants were asked to rate how much attention and effort they had expended on the survey on a Likert-style scale from 1 (“a great deal”) to 5 (“none at all”). For each survey, participants who failed the directed query (i.e., by selecting the incorrect response) or the honesty check (i.e., a response of 5) were coded as missing.

**Analytic Strategy**

Data cleaning and preliminary analyses were conducted in SPSS Version 27.0 (IBM Corp., 2020), then transferred to R for path analyses (R Core Team, 2021). For aggregated scores, missing scale items were imputed with the participant mean based on completed items when ≤ 10% of items were missing (n = 23, or .03% of the total sample had item scores imputed). For participants with > 10% items missing, aggregate scores were recorded as missing. Scores were also recorded as missing for each survey when participants failed either of the attention checks. Participants were excluded from analyses if they were missing data on all key variables across time points (n = 124, or 15.6% of the total sample). Thus, a total of 663 participants were included in path analyses. Of this sample, 19.0% were missing at least one score at baseline. Of participants who completed the follow-up surveys, 11.7% were missing at least one score in the three-month follow up, and 14.7% in the six month follow up. Little’s MCAR tests
were significant for EPSI scores at baseline and WBIS-M scores at three-month follow up, indicating that missing data on these measures were not missing completely at random ($\chi^2(557) = 656.29, p = .002$ and $\chi^2(29) = 60.58, p = .001$). All other variables were non-significant.

All variables were examined to check whether assumptions were met for path analysis (i.e., linearity, normality of residuals). As expected, weight suppression scores were highly positively skewed and influenced by extreme outliers. Outliers greater than three times the interquartile range above the mean were corrected to the next highest value, which resulted in acceptable levels of skew (< 2) and kurtosis (< 5). However, examination of Q-Q plots of standardized residuals indicated that residuals were still severely non-normal. To account for non-normality and missing data, more robust estimation methods and test statistics were used in the path analyses and are described below.

Preliminary analyses using independent samples t-tests and one-way Analysis of Variance (ANOVA) were conducted to explore differences between participants who did vs did not complete follow-up surveys, as well as differences by gender (male, female, nonbinary/genderqueer) and ethnicity. Bivariate Pearson correlations among variables were also examined.

A cross-lagged panel model was tested using path analysis of a model including WBI, weight suppression, and EPSI total scores across time points (see Figure 3). Cross-lagged panel models assess interindividual change in variables included in the model and includes autoregressive paths that assess stability of a construct over time (Selig & Preacher, 2009). For example, the path coefficient between a variable at Time 1 and the
same variable at Time 2 represents the degree of stability in individual differences on the construct over time, with higher coefficients indicating greater stability. This model also allows for the estimation of multiple time-specific indirect effects as well as a total indirect effect representing the sum of all possible indirect effects. The model was recursive (i.e., all causal effects were unidirectional) and specified fewer parameters than observations ($df_M = 11$). Therefore, the model was overidentified, and model parameters could be estimated. Path analyses were conducted in R using the lavaan package (Rosseel, 2012). Given the high amount of missing data and non-normal distribution of weight suppression variables even after reining in outliers, full information robust maximum likelihood (MLR) estimation was used to generate path estimates. MLR estimates standard errors that are robust to potential non-normality in endogenous variables and estimate missing data efficiently (Kline, 2016). Parameter estimates of direct effects were determined to be significant at $p < .05$. Parameter estimates for indirect effects were evaluated using bias-corrected bootstrapping with 5000 resamples. Indirect effects were determined to be significant if the 95% confidence interval did not contain zero.
Figure 3

Cross-lagged Panel Model Analyzed using Path Analysis

Note: The product of paths (a1b1) + c1 represents the indirect effect hypothesized by Model A, whereas the product of paths (a2b2) + c2 represents the indirect effect hypothesized by Model B.

Based on Kline’s (2016) recommendations, model fit was evaluated using multiple indices. The Scaled $\chi^2$ test was reported but not interpreted, as this test becomes trivially significant with large samples (Hooper et al., 2007; Kline, 2016). The comparative fit index (CFI), Tucker-Lewis incremental fit index (TLI), root mean square error of approximation (RMSEA) and its 90% confidence interval, and standardized root mean square residual (SRMR) were also examined. Reasonable model fit was defined as CFI/TLI $\geq .90$ and RMSEA/SRMR $\leq .08$, while good fit was defined as CFI/TLI $\geq .95$ and RMSEA/SRMR $\leq .05$ (Hooper et al., 2007; Hu & Bentler, 1999). Robust estimates of model fit indices were examined to account for non-normality in the data.
Results

Preliminary Analyses

There were no significant differences on baseline WBI or weight suppression scores between participants who completed at least one follow-up survey compared to those who did not complete either of the follow-up surveys ($t(615) = .42, p = .673$, and $t(610) = 1.33, p = .185$ respectively\(^1\)). However, there was a significant difference in EPSI total score at baseline ($t(600) = 2.67, p = .008$), with participants who did not complete either follow-up survey reporting significantly greater overall disordered eating symptoms ($M = 109.07, SD = 24.11$) compared to those who completed at least one follow-up survey ($M = 104.01, SD = 21.79$), although the effect size was small ($d = .22$).

There were no significant ethnic/racial group differences in EPSI total or weight suppression scores at baseline ($F(8,562) = 1.78, p = .078$ and $F(8,45.17) = 1.85, p = .093$ respectively). There were significant differences in WBI at baseline ($F(8,44.81) = 2.60, p = .020$\(^2\)), with pairwise comparisons indicating that South Asian participants reported higher levels of WBI ($M = 4.09, SD = 1.55$) compared to East Asian participants ($M = 3.20, SD = 1.41, p = .009$). Given that differences were limited, primary analyses were collapsed across ethnic group.

\(^1\) Levene’s test was significant for weight suppression, $F = 6.15, p = .013$. The Welch test is reported to correct for heterogeneity of variance.

\(^2\) Levene’s test was significant for baseline WBI and weight suppression, $F(2, 578) = 2.01, p = .044$ and $F(2,573) = 3.06, p = .002$ respectively. The Welch test is reported to correct for heterogeneity of variance.
There were significant gender differences on baseline WBI ($F(2, 584) = 7.10, p = .001$), weight suppression ($F(2, 15.56) = 3.87, p = .043$) and EPSI scores ($F(2, 568) = 5.22, p = .006$). As expected, WBI at baseline was significantly higher among women ($M = 3.73, SD = 1.60$) compared to men ($M = 3.09, SD = 1.59, p = .001$), but neither were significantly different from nonbinary participants ($M = 3.26, SD = 1.39, p = .690$ and $p = .953$ respectively). Similarly, absolute weight suppression at baseline was significantly greater among female ($M = 1.59, SD = 1.33$) compared to male participants ($M = 1.23, SD = 1.22, p = .021$), with neither significantly different from nonbinary participants ($M = 2.30, SD = 2.29, p = .705$ and $p = .481$ respectively). Further, EPSI total scores were significantly higher for women ($M = 107.33, SD = 22.49$) compared to men ($M = 99.84, SD = 21.75, p = .007$) while nonbinary participants ($M = 97.50, SD = 27.47$) were not significantly different from women ($p = .436$) or men ($p = .956$). However, there were no gender differences in WBI at 3-month follow up ($F(2, 13.82) = 1.53, p = .251$) or 6-month follow up ($F(2, 234) = 0.61, p = .544$). There were also no significant gender differences on EPSI total scores at 3-month follow-up ($F(2, 255) = 2.14, p = .119$) or 6-month follow-up ($F(2, 232) = 0.26, p = .774$). Additionally, there were no gender differences in weight suppression at three months ($F(2, 12.21) = 0.89, p = .437$) or six months ($F(2, 7.56) = 0.26, p = .774$).

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3 Levene’s test was significant for baseline weight suppression, $F(2, 579) = 4.53, p = .011$. The Welch test is reported to correct for heterogeneity of variance.
4 Levene’s test was significant for three month WBI, $F(2, 254) = 5.07, p = .007$. The Welch test is reported to correct for heterogeneity of variance.
Given that there were no gender differences among endogenous variables, primary analyses were collapsed across gender.

Table 2 shows means, standard deviations and bivariate correlations among variables included in the path model. Absolute and relative indices of weight suppression showed very high positive correlations at baseline \((r(582) = .97, p < .001)\), 3-month \((r(268) = .99, p < .001)\) and 6-month follow up \((r(249) = .99, p < .001)\). As suggested by Schaumberg et al., 2016, and Piers et al., 2019, path models were analyzed separately using absolute versus relative weight suppression variables. Given the strong correlation among weight suppression calculations and the absence of differences in the models, only models using absolute weight suppression scores are reported. Consistent with hypotheses, weight suppression, WBI and overall disordered eating symptoms as measured by EPSI total scores were all significantly correlated at baseline. However, WBI showed a stronger correlation with disordered eating symptoms \((r(563) = .63)\) than did weight suppression \((r(545) = .26\) for absolute weight suppression scores).

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5 Levene’s tests were significant for three and six month weight suppression, \(F(2,265) = 6.64, p = .002\) and \(F(2,246) = 5.73, p = .004\) respectively. The Welch test is reported to correct for heterogeneity of variance.
Table 2

Means, Standard Deviations and Pearson Correlation Matrix for Continuous Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
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<tbody>
<tr>
<td>1. Baseline WBIS-M Mean Score</td>
<td>587</td>
<td>3.61</td>
<td>1.61</td>
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<tr>
<td>2. Baseline WS (Absolute Score)</td>
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<td>1.53</td>
<td>1.33</td>
<td>.27**</td>
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<tr>
<td>3. Baseline WS (Relative Score)</td>
<td>582</td>
<td>6.03</td>
<td>4.81</td>
<td>.17**</td>
<td>.97**</td>
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<tr>
<td>4. Baseline EPSI Total Score</td>
<td>571</td>
<td>105.86</td>
<td>22.59</td>
<td>.63**</td>
<td>.26**</td>
<td>.21**</td>
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<tr>
<td>5. Three Month WBIS-M Mean Score</td>
<td>257</td>
<td>3.74</td>
<td>3.76</td>
<td>.88**</td>
<td>.12</td>
<td>.04</td>
<td>.63**</td>
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<tr>
<td>6. Three Month WS (Absolute Score)</td>
<td>268</td>
<td>1.52</td>
<td>1.62</td>
<td>.21**</td>
<td>.76**</td>
<td>.72**</td>
<td>.14*</td>
<td>.11</td>
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<tr>
<td>7. Three Month WS (Relative Score)</td>
<td>268</td>
<td>6.01</td>
<td>5.82</td>
<td>.14*</td>
<td>.75**</td>
<td>.73**</td>
<td>.11</td>
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<td>.99**</td>
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<tr>
<td>8. Three Month EPSI Total Score</td>
<td>258</td>
<td>98.24</td>
<td>21.77</td>
<td>.64**</td>
<td>.15*</td>
<td>.12</td>
<td>.78**</td>
<td>.04</td>
<td>.14*</td>
<td>.11</td>
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<tr>
<td>9. Six Month WBIS-M Mean Score</td>
<td>237</td>
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<td>1.69</td>
<td>.86**</td>
<td>.14*</td>
<td>.07</td>
<td>.57**</td>
<td>.91**</td>
<td>.11</td>
<td>.05</td>
<td>.65**</td>
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<tr>
<td>10. Six Month WS (Absolute Score)</td>
<td>249</td>
<td>1.46</td>
<td>1.66</td>
<td>.19**</td>
<td>.75**</td>
<td>.71**</td>
<td>.16*</td>
<td>.12</td>
<td>.91**</td>
<td>.89**</td>
<td>.14</td>
<td>.09</td>
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<tr>
<td>Measure</td>
<td>M</td>
<td>WS</td>
<td>6.29</td>
<td>.13*</td>
<td>.73**</td>
<td>.71**</td>
<td>.14*</td>
<td>.08</td>
<td>.89**</td>
<td>.88**</td>
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<tr>
<td>11. Six Month WS</td>
<td>249</td>
<td>5.81</td>
<td>.03</td>
<td>.99**</td>
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<tr>
<td>12. Six Month EPSI</td>
<td>235</td>
<td>99.08</td>
<td>.09</td>
<td>.60**</td>
<td>.21**</td>
<td>.17*</td>
<td>.69**</td>
<td>.64**</td>
<td>.11</td>
<td>.11</td>
<td>.76**</td>
<td>.67**</td>
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</table>

WBIS-M = Modified Weight Bias Internalization Scale, WS = Weight Suppression, EPSI = Eating Pathology Symptoms Inventory. *p < .05, **p < .001
Path Analysis

Path analysis indicated that the overall model (Figure 3) fit the data well according to multiple indices of fit, $\chi^2(11) = 22.55, p = .020; \text{CFI} = .99, \text{TLI} = .97,$ RMSEA = .05, 90% CI [.02-.08], SRMR = .02. All indices except the scaled chi square test suggested good fit, so the model was retained.

Table 3 lists coefficients and significance for all paths and indirect effects, and Figure 4 overlays parameter estimates onto the model path diagram. Path coefficients between the same variable at later time points were all positive and significant, indicating stability of WBI, weight suppression and eating disorder symptoms over time. However, there were no significant paths between WBI and weight suppression at any time point. There was a significant direct effect of WBI at baseline on disordered eating symptoms three months later ($\beta = .19, p < .001$), but no effect of WBI at three months on disordered eating at six months ($p = .151$). Additionally, there was a significant direct effect of weight suppression at baseline on disordered eating symptoms six months later ($\beta = .18, p = .008$), but no effect on symptoms at three months ($p = .890$). Moreover, weight suppression at three months was not related to disordered eating at six months ($p = .220$).

The hypothesized relations among weight suppression, WBI, and disordered eating were largely not supported. There was no significant indirect effect of weight suppression at three months on the relation between WBI at baseline and disordered eating at six months ($\beta = -.00, 95\% \text{ CI } [-0.43, 0.18]; \text{Model A}$). Additionally, there was no significant indirect effect of WBI at three months on the relation between baseline weight suppression and disordered eating at six months ($\beta = -.01, 95\% \text{ CI } [-0.74, 0.12]; \text{Model B}$). Thus, neither mediation pathway explained the effects of WBI or weight
suppression on overall disordered eating symptoms. However, there was a significant indirect effect of baseline WBI on disordered eating symptoms at six months via disordered eating symptoms at three months, ($\beta = .13$, 95% CI [0.82, 2.98]), suggesting that WBI contributed to increasing disordered eating symptoms over time.

**Table 3**
*Unstandardized and Standardized Coefficients Testing Direct and Indirect Effects*

<table>
<thead>
<tr>
<th></th>
<th>$b$</th>
<th>$SE$</th>
<th>$\beta$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct Effects</strong></td>
<td></td>
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</tr>
<tr>
<td>Baseline WBI $\rightarrow$ 3 Month WBI**</td>
<td>0.87</td>
<td>0.03</td>
<td>.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI $\rightarrow$ 3 Month WS</td>
<td>0.04</td>
<td>0.04</td>
<td>.04</td>
<td>.358</td>
</tr>
<tr>
<td>Baseline WBI $\rightarrow$ 3 Month EPSI total**</td>
<td>2.56</td>
<td>0.66</td>
<td>.19</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI $\rightarrow$ 6 Month EPSI Total</td>
<td>0.34</td>
<td>1.45</td>
<td>.02</td>
<td>.816</td>
</tr>
<tr>
<td>Baseline WS $\rightarrow$ 3 Month WS**</td>
<td>0.96</td>
<td>0.07</td>
<td>.76</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WS $\rightarrow$ 3 Month WBI</td>
<td>-0.04</td>
<td>0.04</td>
<td>-.03</td>
<td>.267</td>
</tr>
<tr>
<td>Baseline WS $\rightarrow$ 3 Month EPSI total</td>
<td>0.09</td>
<td>0.63</td>
<td>.01</td>
<td>.890</td>
</tr>
<tr>
<td>Baseline WS $\rightarrow$ 6 Month EPSI Total*</td>
<td>3.08</td>
<td>1.16</td>
<td>.18</td>
<td>.008</td>
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<tr>
<td>3 Month WBI $\rightarrow$ 6 Month WBI**</td>
<td>0.96</td>
<td>0.04</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WBI $\rightarrow$ 6 Month WS</td>
<td>0.00</td>
<td>0.04</td>
<td>.00</td>
<td>.965</td>
</tr>
<tr>
<td>3 Month WBI $\rightarrow$ 6 Month EPSI Total</td>
<td>2.45</td>
<td>1.70</td>
<td>.17</td>
<td>.151</td>
</tr>
<tr>
<td>3 Month WS $\rightarrow$ 6 Month WS**</td>
<td>0.88</td>
<td>0.02</td>
<td>.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WS $\rightarrow$ 6 Month WBI</td>
<td>0.00</td>
<td>0.03</td>
<td>.00</td>
<td>.940</td>
</tr>
<tr>
<td>3 Month WS $\rightarrow$ 6 Month EPSI Total</td>
<td>-1.25</td>
<td>1.02</td>
<td>-.09</td>
<td>.220</td>
</tr>
<tr>
<td>Baseline EPSI total $\rightarrow$ 3 Month EPSI total**</td>
<td>0.65</td>
<td>0.05</td>
<td>.67</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month EPSI Total $\rightarrow$ 6 Month EPSI Total**</td>
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<td>0.07</td>
<td>.63</td>
<td>&lt; .001</td>
</tr>
<tr>
<td><strong>Indirect Effects</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Baseline WBI $\rightarrow$ 3 Month WS $\rightarrow$ 6 Month EPSI Total</td>
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<td></td>
<td>[-0.43, 0.18]</td>
<td></td>
</tr>
<tr>
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<td></td>
<td>[-1.02, 7.10]</td>
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<tr>
<td>Baseline WBI $\rightarrow$ 3 Month EPSI Total $\rightarrow$ 6 Month EPSI Total*</td>
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<td></td>
<td>[0.82, 2.98]</td>
<td></td>
</tr>
<tr>
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<td></td>
<td>[-1.76, 11.13]</td>
<td></td>
</tr>
<tr>
<td>Total Effect (Model A)</td>
<td>.16</td>
<td></td>
<td>[-6.03, 9.08]</td>
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</table>
Baseline WS -> 3 Month WBI -> 6 Month EPSI Total  
Baseline WS -> 3 Month WS -> 6 Month EPSI Total  
Baseline WS -> 3 Month EPSI Total -> 6 Month EPSI Total  
Total Indirect Effect (Model B)  
Total Effect (Model B)  

<table>
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<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>95% CI</th>
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<tr>
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<tr>
<td>Total Effect (Model B)</td>
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<td>[-1.54, 2.36]</td>
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</table>

WBI = weight bias internalization; WS = weight suppression; EPSI = Eating Pathology Symptoms Inventory. *Significant at \( p < .05 \) or 95% CI > 0. **Significant at \( p < .001 \).

Figure 4

Parameter Estimates for Path Analysis Examining Weight Bias Internalization (WBI), Weight Suppression (WS) and overall Disordered Eating (DE)

Post-Hoc Analyses

Given that WBI and weight suppression have been linked to both general and specific disordered eating symptoms, exploratory post-hoc analyses were conducted to examine whether the effects of WBI or weight suppression differed for specific disordered eating cognitions and behaviors. Following from previous research (Burnette & Mazzeo, 2020, Romano et al., 2021), path models were analyzed separately including
the Restricting, Binge Eating and Body Dissatisfaction subscale scores of the EPSI, rather than the total score.

**Restricting**

The Restricting model showed acceptable to good fit according to multiple indices, $\chi^2(11) = 30.31, p = .001; \ CFI = .99, \ TLI = .95, \ RMSEA = .06, \ 90\% \ CI [.04-.09], \ SRMR = .02$. Contrary to the overall disordered eating model, there were no direct effects of WBI or weight suppression on restricting behaviors at three or six months nor between WBI and weight suppression. There were also no significant indirect effects of either WBI or weight suppression on restricting behaviors, suggesting that neither variable directly nor indirectly explained restricting behaviors over time.

**Binge Eating**

Fit for the Binge Model was also acceptable, $\chi^2(11) = 32.82, p = .001; \ CFI = .98, \ TLI = .95, \ RMSEA = .06, \ 90\% \ CI [.04-.09], \ SRMR = .03$. There was a significant direct effect of WBI at baseline on binge eating three months later, ($\beta = .17, p = .003$), but no effect of WBI at baseline or three months on binge eating at six months ($\beta = .09, p = .569$ and $\beta = .17, p = .264$, respectively). There was also a significant effect of weight suppression at baseline on binge eating three months later ($\beta = .11, p = .040$), but no effects of weight suppression at baseline or three months on binge eating at six months ($\beta = .15, p = .133$ and $\beta = -.20, p = .075$, respectively). As with the overall disordered eating model, there were no indirect effects of WBI via weight suppression or weight suppression via WBI ($\beta = -.02, 95\% \ CI [-0.33, 0.03]$ and $\beta = -.00, 95\% \ CI [-0.21, 0.05]$, respectively). Thus, neither the Model A nor Model B mediation pathways explained the effects of WBI or weight suppression on binge eating. Consistent with the overall
disordered eating model, there was a significant indirect effect of WBI on binge eating at six months via binge eating at 3 months (β = .09, 95% CI [0.05, 0.86]), suggesting that WBI contributed to increasing binge eating symptoms over time. There were no indirect effects of weight suppression on binge eating symptoms.

**Body Dissatisfaction**

The Body Dissatisfaction model showed good to acceptable fit, $\chi^2(11) = 32.81$, $p = .001$; CFI = .99, TLI = .96, RMSEA = .07, 90% CI [.04-.09], SRMR = .02. Consistent with the model for overall disordered eating, WBI at baseline significantly predicted body dissatisfaction at three-month follow-up (β = .31, $p < .001$). Additionally, baseline weight suppression and WBI at three months predicted body dissatisfaction at six months (β = .12, $p = .030$ and β = .32, $p = .004$ respectively). As with the overall disordered eating model, neither the indirect effects of WBI via weight suppression on body dissatisfaction (Model A) nor the indirect effects of weight suppression via WBI on body dissatisfaction (Model B) were significant (β = -.00, 95% CI [-0.09, 0.04] and β = -.01, 95% CI [-0.28, 0.07], respectively). However, there was a significant indirect effect of baseline WBI on body dissatisfaction at six months via WBI at three months (β = .35, 95% CI [0.36, 2.29]) as well as an indirect effect of WBI on body dissatisfaction via body dissatisfaction at three months (β = .20, 95% CI [0.43, 1.28]). These indirect effects suggest that WBI at baseline contributed to both increasing WBI and body dissatisfaction over time. There were no indirect effects of weight suppression on body dissatisfaction.
Discussion

The current study investigated a novel theoretical model of longitudinal relations between WBI, weight suppression, and disordered eating among young adults. Competing mediational pathways between WBI, weight suppression, and disordered eating were tested to examine the potential temporal order of effects between WBI and weight suppression. Consistent with hypotheses and previous research, there were significant associations between WBI, weight suppression, and disordered eating at baseline. However, results of the path analysis only partially supported the hypothesized models. Specifically, baseline WBI and weight suppression indirectly and directly predicted overall disordered eating symptoms over six month-follow-up, respectively; however, there were no effects of WBI on weight suppression or vice versa. Moreover, post-hoc analyses examined whether the model explained specific disordered eating cognitions and behaviors. Neither weight suppression nor WBI were related to restricting behaviors across follow-up. Weight suppression directly predicted binge eating three months later, whereas WBI directly predicted binge eating three months later and indirectly predicted binge eating six months later via binge eating symptoms at three months. Finally, baseline WBI and weight suppression predicted body dissatisfaction three and six months later, respectively, and associations between baseline WBI and body dissatisfaction at six months were mediated by WBI and body dissatisfaction at three months. Across these analyses, there were no significant indirect effects that would be consistent with either hypothesized mediation pathway (Model A or Model B). These findings suggest that WBI and weight suppression may not be related over time in young adults.
Support for Model A

Contrary to relations hypothesized by Model A, there were no direct effects of WBI on weight suppression in any of the models tested, suggesting that higher levels of WBI at baseline did not predict greater weight suppression over follow up. These findings are consistent with Burnette & Mazzeo (2020), who found that weight suppression accounted for unique variance in disordered eating behaviors beyond the contribution of WBI, suggesting that these constructs have unique associations with disordered eating. One interpretation of the absence of an effect of WBI on weight suppression is that high levels of WBI may increase motivation to lose weight but not translate to actual weight loss (Major et al., 2020). Indeed, WBI was found to predict binge eating symptoms over follow-up, which may counteract attempts to lose weight. Other individual difference factors may also moderate potential relations between WBI and weight suppression. For example, BMI may moderate associations such that individuals with both high BMI and high WBI may also show higher weight suppression. Additionally, higher weight individuals who experienced weight-based victimization prior to losing weight may experience greater fears of regaining weight in the future, leading to greater suppression of weight.

Although the mediation pathway represented by Model A was not supported, results did support an indirect effect of WBI on overall disordered eating symptoms such that disordered eating at three months mediated associations between baseline WBI and disordered eating at six months. Additionally, WBI contributed indirectly to body dissatisfaction at six-month follow-up via greater WBI and body dissatisfaction at three months. Thus, WBI appears to have cumulatively negative effects on body satisfaction
and overall disordered eating symptoms over time. These findings are consistent with previous research across the weight spectrum (Meadows & Calogero, 2018; Romano et al., 2021) and have important implications for eating disorder risk in young adult populations, as body dissatisfaction is a known predictor of eating disorder onset (Buchianeri & Neumark-Sztainer, 2014; Stice et al., 2017; Stice et al., 2011).

Further, WBI at baseline directly predicted binge eating three months later, and indirectly predicted binge eating six months later via symptoms at three months. These findings are consistent with previous research demonstrating cross-sectional and longitudinal effects of WBI on binge eating symptoms (Ahorsu et al., 2020; Burnette & Mazzeo, 2020; Lee et al., 2019; Romano et al., 2021; Schvey & White, 2015). WBI may lead to greater symptoms of psychological distress and food addiction, both of which may underly increased binge eating (Ahorsu et al., 2020). WBI has also been associated with greater use of eating to cope with stress (Pearl et al., 2021). Further, there has been increasing research attention on weight bias as a form of minority stress (Sikorski et al., 2015). According to this model, weight bias contributes to chronic stress through both distal stressors of weight-based victimization and proximal stressors including WBI. These stressors activate psychological risk factors, such as impaired emotion regulation and coping, which contribute to negative mental health outcomes. Thus, WBI may activate other psychological risk factors that have been associated with binge eating, such as emotion dysregulation or impulsivity (Lavender et al., 2015; Smith et al., 2018). However, further research is needed to establish whether this model explains relations between WBI and binge eating symptoms.
The finding that WBI had no direct or indirect effects on restrictive eating behavior was inconsistent with previous research, as WBI has been concurrently associated with restrained eating in previous studies (Burnette & Mazzeo, 2020; Zuba & Warschburger, 2017). The absence of longitudinal effects may suggest that WBI is associated with concurrent severity of restricting behavior, but not the development or maintenance of restriction over time. WBI may also have differential effects on cognitive efforts to restrain food intake compared to actual behavioral restriction (Major et al., 2020). Indeed, cognitive restraint is a distinct construct from behavioral restriction, as cognitive efforts to restrict calories, avoid eating or avoid certain types of food may not translate to objective reductions in food intake (Stice et al., 2007). Consistent with the idea that WBI may be more related to cognitive restraint versus food restriction, supplemental post-hoc analyses indicated that WBI at baseline predicted cognitive restraint symptoms three and six months later (see Appendix B). Thus, WBI may contribute to greater efforts to restrain eating, but not translate to behavioral restriction of food intake.

**Support for Model B**

As with the findings for Model A, there were no effects of weight suppression on WBI over follow up, indicating that weight suppression did not contribute to increasing WBI over time. Additionally, there was no indirect effect of weight suppression on disordered eating outcomes via WBI, thus Model B was not supported. A potential explanation for this finding is that maintaining a lower weight attenuates WBI by supporting beliefs that one can successfully control their weight and/or avoid experiencing weight bias. Consistent with this idea, previous research has found that
individuals with lower BMIs endorse lower levels of WBI. Moreover, Bodell et al. (2017) identified drive for thinness at 10-year follow up as a mediator between weight suppression and bulimia symptoms over 20-year follow up. Thus, preoccupation with thinness, rather than WBI, may better explain the effects of weight suppression on disordered eating.

The finding that weight suppression at baseline predicted greater body dissatisfaction six months later was a novel contribution of this study. Few studies have examined associations between weight suppression and body dissatisfaction, with inconsistent results using cross-sectional designs (Burnette et al., 2019; Van Son et al., 2013). Concurrent associations may reflect weight loss motivations to change body shape/weight and reduce dissatisfaction. However, longitudinal findings from the current study suggest that maintaining weight loss may have the opposite effect than intended in terms of improving one’s body satisfaction over time. In the context of the biobehavioral bind, the increasing effort required to maintain a suppressed weight may contribute to greater dissatisfaction.

The effect of weight suppression on binge eating at three months may be consistent with the dietary restraint model of binge eating, where initial efforts to restrict food intake in order to lose weight eventually lead to binge episodes (Fairburn et al., 2003). However, the absence of effects between weight suppression and binge eating six months later is inconsistent with previous work where higher weight suppression has been cross-sectionally and prospectively linked with binge eating and loss of control over eating (Bodell et al., 2017; Burnette et al., 2019). Notably, other studies have found that weight suppression is not associated with binge eating or purging symptoms in
community and clinical samples (Bodell et al., 2016; Call et al., 2021; Lavender et al., 2015; Stice et al., 2011; Van Son et al., 2013; Zunker et al., 2011). The inconsistency of findings suggests that study design (i.e., cross-sectional versus longitudinal) and length of follow up may be particularly important to understanding the long-term effects of weight suppression on binge eating. Additionally, other variables may moderate these effects, such as impulsivity, depression symptoms, or eating-related self-efficacy (Goldschmidt et al., 2012; Linardon, 2018).

The absence of effects of weight suppression on restrictive behaviors also was inconsistent with previous research, as weight suppression has been associated with greater dietary restraint in undergraduate men and women (Burnette et al., 2017; 2018; 2019). However, these studies all employed cross-sectional designs, which may lead to biased estimates of weight suppression as a predictor of restricting in non-clinical samples. Additionally, dietary restraint was measured using the Eating Disorders Examination Questionnaire (Fairburn & Beglin, 1994), which largely reflects cognitive efforts to restrict calories or avoid eating whether or not they are successful, obscuring effects unique to behavioral restriction. It is also possible that existing evidence is influenced by publication bias: in an unpublished thesis, Jones (2016) similarly found that weight suppression did not predict dietary restriction over four weeks in a sample of undergraduate women. Alternatively, the undergraduate sample included in the current study may not have included a large enough range of severity in either weight suppression or restricting symptoms. Mean weight suppression in the current sample was relatively low (i.e., absolute scores indicating < 2 BMI unit change), whereas significant weight loss is likely more prevalent in clinical samples. Further, only 15.2% of the
sample at baseline \((n = 101)\) reported that they were currently dieting to lose weight. Indeed, weight suppression has been linked to greater severity of restricting symptoms in multi-diagnostic eating disorder patient samples (Bodell et al. 2016; Lavender et al., 2015). However, in community samples of adolescents and young adults, restriction may more strongly predict weight \emph{gain} rather than weight loss, reflecting the long-term ineffectiveness of calorie deficit as a weight loss strategy (Chu et al., 2021; Yoon et al., 2019). Additionally, participants in the current study who were already weight suppressed at baseline may have been more vulnerable to weight gain over follow-up (Lowe et al., 2019).

Length of follow up in the current study may also have contributed to inconsistent predictive effects of weight suppression on disordered eating. For example, weight suppression at baseline predicted overall disordered eating and body dissatisfaction six months later, but not at three months. Thus, the three-month period between assessments may not be long enough for effects of weight suppression on disordered eating symptoms to appear. Additionally, it may take longer than six months for the contribution of weight suppression to restriction and binge eating symptoms to manifest, leading to non-significant findings in this study. However, the findings do suggest that it is possible to observe effects on body dissatisfaction and overall eating pathology within a six-month time frame. Follow-up periods over multiple years may be necessary to clearly elucidate the effects of long-term efforts to maintain a suppressed weight.

**Implications and Future Directions**

Consistent with previous research, both weight suppression and WBI were found to prospectively predict general and specific disordered eating symptoms. Distinct effects
of WBI versus weight suppression were found on the prediction of overall disordered eating, binge eating, and body dissatisfaction over six-month follow up. Taken together, these findings provide support for WBI and weight suppression being important risk factors for disordered eating in young adults across weight status. These findings have important implications for prevention and intervention strategies targeted at undergraduate students. Prevention programs should address weight-biased attitudes held by students which may inform internalization and subsequent self-devaluation. Interventions challenging beliefs about weight and personal responsibility as well as promoting empathy towards higher weight persons have been examined to reduce weight biased attitudes held against others, but their effects on WBI in undergraduate populations has not been examined (Daníelsdóttir et al., 2010; Lee et al., 2014). Specific programs could also be developed to reduce WBI regardless of one’s weight status, as many existing programs focus on higher weight individuals and have been disseminated in combination with weight-loss interventions (Meadows & Calogero, 2018; Pearl et al., 2018, 2020). WBI may be an important prevention target in undergraduate populations, as it predicted greater body dissatisfaction, binge eating and overall disordered eating symptoms in this study. Additionally, screening for weight suppression and high levels of WBI may be easily implemented to identify those most at risk for disordered eating.

Future research should examine additional mechanisms or moderators that contribute to the effects of WBI and weight suppression on disorder eating, such as negative affect, emotion dysregulation, and weight history prior to adulthood. Within-individual variability in weight change over time as well as subjective perceptions of weight may also have important implications for weight-control behaviors that may
become extreme over time. Although the cross-lagged panel model analyzed in this study did assess interindividual change in variable, it was not able to capture within-person influences. Additionally, the longitudinal effects of WBI and weight suppression should be further explored in populations at particularly high risk for disordered eating behaviors, such as higher-weight individuals exposed to weight-based victimization, individuals currently engaged in dieting for weight loss, and clinical eating disorder samples.

Future research should more closely examine similarities and differences among genders in longitudinal effects of WBI and weight suppression on eating behavior. In the current study, women showed greater levels of WBI, weight suppression and disordered eating compared to men at baseline, which is consistent with previous literature (Puhl et al., 2018; Stephen et al., 2014). However, gender differences disappeared over follow up, and other studies have found limited or no gender differences (Burnette et al., 2017; Lipson & Sonneville, 2017). It should be noted that very few men, and even fewer nonbinary individuals, completed the follow-up measures; thus, any interpretation of these findings should be made with caution. Men may feel stronger pressures to gain muscle rather than lose weight or may alternate between periods of restrictive and binge eating in order to achieve masculine body ideals of high muscle mass and low body fat (Murray et al., 2017; Nagata et al., 2018). WBI may also be greater among specific groups of men, such as sexual minorities (Austen et al., 2020). Importantly, transgender and nonbinary individuals may be at heightened risk for weight-based victimization and disordered eating, and experience unique body image and eating concerns associated with gender dysphoria and transition (Coelho et al., 2019; Diemer et al., 2018; Himmelstein et
al., 2017; Obarzanek & Munyan, 2021). Thus, larger samples of gender diverse young adults are needed for more nuanced investigation of gendered effects in future studies.

Similarly, there are likely nuanced influences of racial or ethnic background that were not captured by this study. Although the current study identified few ethnic differences, South Asian participants reported higher levels of WBI compared to East Asian participants. Most studies examining WBI have examined majority White and Western samples, and few have investigated potential differences by ethnic or national background (Pearl et al., 2021). Cultural attitudes towards weight and dieting behaviors likely influence exposure to and endorsement of weight-stigmatizing messages as well as other risk factors for disordered eating (Hart et al., 2016; Scott & Rosen, 2015; Yanover & Thompson, 2010). Weight-based victimization may also compound or intersect with exposure to other forms of discrimination, including racial discrimination (Ciciurkaite & Perry, 2018). However, studies describing and identifying mechanisms of ethnic differences in WBI are lacking. Similarly, while previous studies of ethnic differences in disordered eating symptoms have found inconsistent results, there is increasing evidence that disordered eating risk may be compounded among individuals with multiple marginalized social identities (Austin et al., 2013; Bucchianeri et al., 2016; Burke et al., 2020; Rodgers et al., 2017). Future research should examine whether unique patterns of effects may explain relations among WBI, weight suppression, and disordered eating in diverse samples.

**Strengths and Limitations**

The current study has several important strengths, particularly through its longitudinal design. Cross-sectional mediation analyses have been heavily criticized, as
mediation implies a process that unfolds over time, which cannot be captured by observation at only one time point (O’Laughlin et al., 2018). Simulation studies have also found that cross-sectional analyses can produce biased results (Maxwell & Cole, 2007). The current study addresses this issue by capturing change in variables across three waves of measurement, allowing for a fully longitudinal design in which all direct effects include data at more than one time point. This study also benefits from the large sample included in path analyses. Additionally, this study examined a novel theoretical model which integrated the previously separate WBI and weight suppression literatures and tested competing models of relations between these two constructs. Examination of both general disordered eating as well as specific behavioral subscales also elucidated the relative contribution of WBI and weight suppression to distinct behaviors in addition to overall psychopathology.

There are also important limitations that should be considered when interpreting the results of this study. Path analyses using SEM assume that all observed variables are measured with perfect reliability, an assumption that is likely to be problematic especially when relying on single indicators (Kline, 2016). Measurement error within any of the analyzed variables could lead to biased parameter estimates, and error in mediating variables can lead to underestimation of indirect effects and overestimation of direct effects (Cole & Maxwell, 2003). Although the WBIS-M showed high internal consistency in the current sample, factor analysis of the original item set has indicated that the single factor design of the WBIS-M may not be appropriate, and may not adequately capture self-devaluation associated with WBI (Austen et al., 2020; Meadows & Higgs, 2020a). Self-reported weight and height were used to calculate weight
suppression scores, as self-reports of these variables have been shown to be highly correlated with objective measures (Pursey et al., 2014). However, weight suppression as operationalized in this study does not account for weight trajectories prior to adulthood. Attrition over the follow-up period may also have biased estimates for variables at follow-up. Participants who did not complete either of the follow-up surveys reported significantly higher disordered eating symptoms at baseline, suggesting that symptoms at three and six months may have been underestimated. It should be noted, however, that the effect size for this difference was small, and represented only a five-point difference in EPSI total scores, which may not reflect clinically significant differences in symptoms.

A further assumption inherent to the use of cross-lagged panel model longitudinal designs is that the time lag between waves of measurement is sufficient to capture the theorized change process among variables (Selig & Preacher, 2009). The use of a three-month lag between assessments in the current study may not have been long enough to capture causal effects. It could also be the case that, in a sample of undergraduates, relationships among these variables may have been established long before participants were recruited. Indeed, WBI has been prospectively related to emotional problems and restrained eating in girls and boys aged 7-11 years (Zuba & Warschburger, 2017). Future research should examine longitudinal relations among these variables over extended time periods. Given that eating disorders commonly onset during adolescence, it may be especially important to track associations between weight trajectories, attitudes about weight, and eating behaviors before and during this developmental stage.

Additionally, although the use of a longitudinal design allowed for the examination of temporal precedence, establishing temporal precedence between two
variables is not sufficient to determine causation. This study did not employ random assignment or experimental manipulation of any of the variables analyzed, both of which inform stronger claims compared to those relying on observational data (Bullock et al., 2010). Further, relations among WBI, weight suppression, and disordered eating may be influenced by other variables that were not included or accounted for in these analyses, such as negative affect or distress (Ahorsu et al., 2020).

Finally, it is important to note that data collection for this study took place during periods of lockdown caused by the COVID-19 pandemic. Recruitment of participants began shortly after the first wave began in Spring 2020, with collection of follow-up data continuing throughout Fall 2020 and Winter 2021. The pandemic has had important impacts on various forms of psychopathology including disordered eating, although the long-term impact remains to be seen (Dozois, 2020; Jenkins et al., 2021; Phillipou et al., 2020). Participants in this study may have experienced important changes in their daily routine, access to social supports and ability to use adaptive coping strategies, which may have influenced their responses to questionnaires (Rodgers et al., 2020). Pandemic-related stressors may have contributed to disordered eating symptoms such as binge eating and restriction, particularly among young adults already at risk for eating pathology (Ramalho et al., 2021; Simone et al., 2021). Public health measures may have also had effects on food consumption and physical activity which potentially led to weight changes (Gallo et al., 2020; Romero-Blanco et al., 2020), although self-reports of weight and activity changes may also be more strongly influenced by shape/weight concerns or weight misperception than objective weight changes (Keel et al., 2020).
Thus, more research is needed to establish whether effects observed in the current study will generalize to samples post-COVID.
Conclusion

Internalization of weight-biased attitudes may motivate young adults to engage in unhealthy weight-control behaviors to suppress their weight, which may become increasingly extreme over time and lead to disordered eating. Although previous literature has linked WBI and weight suppression to disordered eating, these lines of research are largely separate, and potential relations between these two variables are underexamined. The aim of the current study was to integrate research on these constructs by examining competing theories of how WBI and weight suppression influence each other, as well as disordered eating, over time. Longitudinal path analysis models supported WBI and weight suppression as distinct predictors of disordered eating, binge eating, and body dissatisfaction over a six-month time frame. However, neither direction of effects between WBI and weight suppression was supported by the models, suggesting that these variables are not related over time in young adults. Identifying those as highest risk for disordered eating based on their levels of weight suppression and WBI will be important to target for prevention and intervention strategies to reduce eating disorder risk in undergraduate populations.


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Appendix A: Supplemental Tables

Table A1
Direct and Indirect Effects Tested in Restricting Model

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<th>$\beta$</th>
<th>$p$</th>
</tr>
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<td>Baseline WBI -&gt; 3 Month WBI**</td>
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<td>0.03</td>
<td>.90</td>
<td>&lt; .001</td>
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<td>Baseline WBI -&gt; 3 Month WS</td>
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<td>0.04</td>
<td>.04</td>
<td>.378</td>
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<td>Baseline WBI -&gt; 3 Month Restricting</td>
<td>0.17</td>
<td>0.14</td>
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<td>.208</td>
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<tr>
<td>Baseline WBI -&gt; 6 Month Restricting</td>
<td>-0.05</td>
<td>0.39</td>
<td>-.02</td>
<td>.901</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS**</td>
<td>0.96</td>
<td>0.07</td>
<td>.76</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI</td>
<td>-0.04</td>
<td>0.04</td>
<td>-.03</td>
<td>.244</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Restricting</td>
<td>-0.26</td>
<td>0.15</td>
<td>-.07</td>
<td>.080</td>
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<tr>
<td>Baseline WS -&gt; 6 Month Restricting</td>
<td>0.50</td>
<td>0.33</td>
<td>.12</td>
<td>.136</td>
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<tr>
<td>3 Month WBI -&gt; 6 Month WBI**</td>
<td>0.96</td>
<td>0.03</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WS</td>
<td>0.00</td>
<td>0.04</td>
<td>.00</td>
<td>.942</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month Restricting</td>
<td>-0.01</td>
<td>0.42</td>
<td>-.00</td>
<td>.980</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WS**</td>
<td>0.88</td>
<td>0.02</td>
<td>.897</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WBI</td>
<td>-0.00</td>
<td>0.03</td>
<td>0.00</td>
<td>.940</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month Restricting</td>
<td>-0.04</td>
<td>0.27</td>
<td>-.01</td>
<td>.152</td>
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<tr>
<td>Baseline Restricting -&gt; 3 Month Restricting**</td>
<td>0.77</td>
<td>0.04</td>
<td>.78</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month Restricting -&gt; 6 Month Restricting**</td>
<td>0.74</td>
<td>0.05</td>
<td>.75</td>
<td>&lt; .001</td>
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</table>

<table>
<thead>
<tr>
<th>Indirect Effects</th>
<th>$\beta$</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WS -&gt; 6 Month Restricting</td>
<td>.00</td>
<td>[-0.05, 0.13]</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI -&gt; 6 Month Restricting</td>
<td>.01</td>
<td>[-0.96, 0.93]</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Restricting -&gt; 6 Month Restricting</td>
<td>.03</td>
<td>[-0.18, 0.41]</td>
</tr>
<tr>
<td>Total Indirect Effect (Model A)</td>
<td>.04</td>
<td>[-0.88, 1.06]</td>
</tr>
<tr>
<td>Total Effect (Model A)</td>
<td>.03</td>
<td>[-0.34, 0.59]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI -&gt; 6 Month Restricting</td>
<td>.00</td>
<td>[-0.08, 0.06]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS -&gt; 6 Month Restricting</td>
<td>.05</td>
<td>[-0.65, 1.00]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Restricting -&gt; 6 Month Restricting</td>
<td>-.04</td>
<td>[-0.49, 0.11]</td>
</tr>
<tr>
<td>Total Indirect Effect (Model B)</td>
<td>.00</td>
<td>[-0.81, 0.89]</td>
</tr>
</tbody>
</table>

Total Effect (Model B)  .00  [-0.63, 0.66]

WBI = weight bias internalization; WS = weight suppression. **Significant at p < .001

Table A2
Direct and Indirect Effects Tested in Binge Eating Model

<table>
<thead>
<tr>
<th>Direct Effects</th>
<th>b</th>
<th>SE</th>
<th>β</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI**</td>
<td>0.87</td>
<td>0.03</td>
<td>.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WS</td>
<td>0.04</td>
<td>0.04</td>
<td>.04</td>
<td>.321</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Binge Eating*</td>
<td>0.69</td>
<td>0.24</td>
<td>.17</td>
<td>.003</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 6 Month Binge Eating</td>
<td>0.34</td>
<td>0.60</td>
<td>.08</td>
<td>.569</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS**</td>
<td>0.96</td>
<td>0.07</td>
<td>.76</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI</td>
<td>-0.04</td>
<td>0.04</td>
<td>-.03</td>
<td>.249</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Binge Eating*</td>
<td>0.53</td>
<td>0.26</td>
<td>.11</td>
<td>.040</td>
</tr>
<tr>
<td>Baseline WS -&gt; 6 Month Binge Eating</td>
<td>0.76</td>
<td>0.51</td>
<td>.08</td>
<td>.133</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WBI**</td>
<td>0.96</td>
<td>0.03</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WS</td>
<td>0.00</td>
<td>0.04</td>
<td>.00</td>
<td>.940</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month Binge Eating</td>
<td>0.73</td>
<td>0.65</td>
<td>.17</td>
<td>.264</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WS**</td>
<td>0.88</td>
<td>0.02</td>
<td>.897</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WBI</td>
<td>0.00</td>
<td>0.03</td>
<td>.00</td>
<td>.918</td>
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<tr>
<td>3 Month WS -&gt; 6 Month Binge Eating</td>
<td>-0.81</td>
<td>0.46</td>
<td>.20</td>
<td>.075</td>
</tr>
<tr>
<td>Baseline Binge Eating -&gt; 3 Month Binge Eating**</td>
<td>0.54</td>
<td>0.05</td>
<td>.57</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month Binge Eating -&gt; 6 Month Binge Eating**</td>
<td>0.53</td>
<td>0.08</td>
<td>.50</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indirect Effects</th>
<th>β</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WS -&gt; 6 Month Binge Eating</td>
<td>-.02</td>
<td>[-0.33, 0.03]</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI -&gt; 6 Month Binge Eating</td>
<td>.08</td>
<td>[-1.21, 1.90]</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Binge Eating -&gt; 6 Month Binge Eating*</td>
<td>.09</td>
<td>[0.05, 0.86]</td>
</tr>
<tr>
<td>Total Indirect Effect (Model A)</td>
<td>.11</td>
<td>[-2.07, 3.03]</td>
</tr>
<tr>
<td>Total Effect (Model A)*</td>
<td>.30</td>
<td>[0.56, 1.94]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI -&gt; 6 Month Binge Eating</td>
<td>-.00</td>
<td>[-0.21, 0.05]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS -&gt; 6 Month Binge Eating</td>
<td>-.17</td>
<td>[-2.55, 0.56]</td>
</tr>
</tbody>
</table>
Baseline WS -> 3 Month Binge Eating -> 6 Month Binge Eating  .05  [-0.10, 0.65]
Total Indirect Effect (Model B)  -0.27  [-3.63, 0.81]
Total Effect (Model B)  -0.20  [-1.99, 0.22]

WBI = weight bias internalization; WS = weight suppression. *Significant at $p < .05$ or 95% CI > 0. **Significant at $p < .001$.

Table A3

Direct and Indirect Effects Tested in Body Dissatisfaction Model

<table>
<thead>
<tr>
<th>Direct Effects</th>
<th>$b$</th>
<th>$SE$</th>
<th>$\beta$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI**</td>
<td>0.87</td>
<td>0.03</td>
<td>.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WS</td>
<td>0.04</td>
<td>0.04</td>
<td>.04</td>
<td>.347</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Body Dissatisfaction**</td>
<td>1.19</td>
<td>0.22</td>
<td>.31</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 6 Month Body Dissatisfaction</td>
<td>-0.13</td>
<td>0.40</td>
<td>-0.03</td>
<td>.742</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS**</td>
<td>0.96</td>
<td>0.07</td>
<td>.76</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI</td>
<td>-0.04</td>
<td>0.03</td>
<td>-0.03</td>
<td>.283</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Body Dissatisfaction</td>
<td>0.01</td>
<td>0.19</td>
<td>0.00</td>
<td>.969</td>
</tr>
<tr>
<td>Baseline WS -&gt; 6 Month Body Dissatisfaction*</td>
<td>0.58</td>
<td>0.27</td>
<td>.12</td>
<td>.030</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WBI**</td>
<td>0.96</td>
<td>0.03</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WS</td>
<td>0.00</td>
<td>0.04</td>
<td>0.00</td>
<td>.953</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month Body Dissatisfaction*</td>
<td>1.33</td>
<td>0.46</td>
<td>.32</td>
<td>.004</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WS**</td>
<td>0.88</td>
<td>0.02</td>
<td>.897</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WBI</td>
<td>0.00</td>
<td>0.03</td>
<td>0.00</td>
<td>.954</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month Body Dissatisfaction</td>
<td>-0.29</td>
<td>0.22</td>
<td>-0.07</td>
<td>.186</td>
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<tr>
<td>Baseline Body Dissatisfaction -&gt; 3 Month Body Dissatisfaction**</td>
<td>0.55</td>
<td>0.06</td>
<td>.58</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month Body Dissatisfaction -&gt; 6 Month Body Dissatisfaction**</td>
<td>0.63</td>
<td>0.07</td>
<td>.59</td>
<td>&lt; .001</td>
</tr>
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Indirect Effects

<table>
<thead>
<tr>
<th>$\beta$</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WS -&gt; 6 Month Body Dissatisfaction</td>
<td>-.00</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI -&gt; 6 Month Body Dissatisfaction*</td>
<td>.35</td>
</tr>
</tbody>
</table>
Baseline WBI -> 3 Month Body Dissatisfaction -> 6 Month Body Dissatisfaction* 0.20 [0.43, 1.28]
Total Indirect Effect (Model A)* 0.55 [1.16, 3.09]
Total Effect (Model A)* 0.48 [1.25, 2.49]
Baseline WS -> 3 Month WBI -> 6 Month Body Dissatisfaction -0.01 [-0.28, 0.07]
Baseline WS -> 3 Month WS -> 6 Month Body Dissatisfaction -0.03 [-0.79, 0.44]
Baseline WS -> 3 Month Body Dissatisfaction -> 6 Month Body Dissatisfaction -0.02 [-0.39, 0.22]
Total Indirect Effect (Model B) -0.06 [-1.01, 0.49]
Total Effect (Model B) 0.02 [-0.63, 0.75]

WBI = weight bias internalization; WS = weight suppression. *Significant at \( p < .05 \) or 95% CI > 0. **Significant at \( p < .001 \).
Appendix B: Supplemental Analyses of Cognitive Restraint Subscale

Supplemental analyses were conducted to examine whether there would be differential effects of WBI and weight suppression on cognitive efforts to restrict food intake (e.g. avoiding certain types of food). A separate path analysis model was tested with the Cognitive Restraint subscale of the EPSI. Fit for the Cognitive Restraint Model was good according to multiple indices of fit, $\chi^2(11) = 23.81$, $p = .014$; CFI = .99, TLI = .97, RMSEA = .05, 90% CI [.02-.08], SRMR = .02. There was a significant direct effect of WBI at baseline on cognitive restraint six months later, ($\beta = .22$, $p = .036$), but no effect of WBI at baseline on cognitive restraint at three months ($\beta = .10$, $p = .382$). There was also no effect of WBI at three months on cognitive restraint at six months ($\beta = -.10$, $p = .339$). As with the overall disordered eating model, there were no indirect effects of WBI via weight suppression or weight suppression via WBI ($\beta = -.01$, 95% CI [-0.09, 0.03] and $\beta = -.00$, 95% CI [-0.01, 0.07], respectively). Thus, neither the Model A or Model B mediation pathways explained the effects of WBI or weight suppression on cognitive restraint. Consistent with the overall disordered eating model, there was a significant indirect effect of WBI on cognitive restraint at six months via cognitive restraint at three months ($\beta = .15$, 95% CI [0.09, 0.48]), suggesting that WBI contributed to increasing cognitive restraint symptoms over time. There were no direct or indirect effects of weight suppression on cognitive restraint symptoms.

Table B1

<table>
<thead>
<tr>
<th>Direct Effects</th>
<th>$b$</th>
<th>SE</th>
<th>$\beta$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI**</td>
<td>0.87</td>
<td>0.03</td>
<td>.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month WS</td>
<td>0.04</td>
<td>0.04</td>
<td>.04</td>
<td>.328</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Cognitive Restraint</td>
<td>0.21</td>
<td>0.10</td>
<td>.10</td>
<td>.382</td>
</tr>
<tr>
<td>------------------------------------------</td>
<td>------</td>
<td>------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 6 Month Cognitive Restraint*</td>
<td>0.43</td>
<td>0.20</td>
<td>.22</td>
<td>.036</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS**</td>
<td>0.96</td>
<td>0.07</td>
<td>.76</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI</td>
<td>-0.04</td>
<td>0.04</td>
<td>-0.03</td>
<td>.242</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Cognitive Restraint</td>
<td>0.10</td>
<td>0.11</td>
<td>.04</td>
<td>.382</td>
</tr>
<tr>
<td>Baseline WS -&gt; 6 Month Cognitive Restraint</td>
<td>0.39</td>
<td>0.25</td>
<td>.16</td>
<td>.121</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WBI**</td>
<td>0.96</td>
<td>0.03</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month WS</td>
<td>0.00</td>
<td>0.04</td>
<td>.00</td>
<td>.953</td>
</tr>
<tr>
<td>3 Month WBI -&gt; 6 Month Cognitive Restraint</td>
<td>-0.20</td>
<td>0.21</td>
<td>-.10</td>
<td>.339</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WS**</td>
<td>0.88</td>
<td>0.02</td>
<td>.897</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month WS -&gt; 6 Month WBI</td>
<td>0.00</td>
<td>0.03</td>
<td>.00</td>
<td>.929</td>
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<td>3 Month WS -&gt; 6 Month Cognitive Restraint</td>
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<td>0.21</td>
<td>-.06</td>
<td>.613</td>
</tr>
<tr>
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<td>0.66</td>
<td>0.05</td>
<td>.66</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>3 Month Cognitive Restraint -&gt; 6 Month Cognitive Restraint**</td>
<td>0.66</td>
<td>0.06</td>
<td>.68</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

**Indirect Effects**

<table>
<thead>
<tr>
<th>Baseline WBI -&gt; 3 Month WS -&gt; 6 Month Cognitive Restraint</th>
<th>β</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline WBI -&gt; 3 Month WBI -&gt; 6 Month Cognitive Restraint</td>
<td>-.11</td>
<td>[-0.70, 0.23]</td>
</tr>
<tr>
<td>Baseline WBI -&gt; 3 Month Cognitive Restraint -&gt; 6 Month Cognitive Restraint</td>
<td>.15</td>
<td>[0.09, 0.48]</td>
</tr>
<tr>
<td>Total Indirect Effect (Model A)</td>
<td>.03</td>
<td>[-0.48, 0.54]</td>
</tr>
<tr>
<td>Total Effect (Model A)*</td>
<td>.24</td>
<td>[0.16, 0.68]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WBI -&gt; 6 Month Cognitive Restraint</td>
<td>.00</td>
<td>[-0.01, 0.07]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month WS -&gt; 6 Month Cognitive Restraint</td>
<td>-.05</td>
<td>[-0.61, 0.36]</td>
</tr>
<tr>
<td>Baseline WS -&gt; 3 Month Cognitive Restraint -&gt; 6 Month Cognitive Restraint</td>
<td>.01</td>
<td>[-0.16, 0.19]</td>
</tr>
<tr>
<td>Total Indirect Effect (Model B)</td>
<td>-.04</td>
<td>[-0.65, 0.44]</td>
</tr>
<tr>
<td>Total Effect (Model B)</td>
<td>.09</td>
<td>[-0.13, 0.52]</td>
</tr>
</tbody>
</table>

WBI = weight bias internalization; WS = weight suppression. *Significant at p < .05 or 95% CI > 0. **Significant at p < .001.
Appendix C: Letter of Information and Consent

**Project Title:** Weight Stigma Internalization and Quality of Life among Undergraduate Students

**Document Title:** Letter of Information and Consent.

**Principal Investigator:** Dr. Lindsay Bodell, lbodell@uwo.ca, (519) 661-2111 x 80486

**Graduate Student Researcher:** Samantha Withnell, withnell@uwo.ca, (519) 661-2111 x 87316

**Additional Research Staff:** Jina Kim, Abbigail Kinnear

**Invitation to Participate**

You are being invited to participate in a study regarding body image, eating behaviors, and quality of life.

The purpose of this letter is to provide you with information required for you to make an informed decision regarding participation in this research.

**Purpose of Study**

The aim of this study is to examine the effect of internalizing weight stigma on body image, eating behaviors and quality of life over time.

**Inclusion Criteria**

Undergraduate students who are 17-25 years old and fluent in English are eligible to participate in this study.

**Procedures**

If you agree to participate, you will be asked to complete three surveys. The first survey includes 8 questionnaires following this letter which will take approximately 30-60 minutes to complete. The nature of the questions under investigation will include topics such as weight, eating and exercise behaviors, attitudes towards your body, and quality of life.
At the end of this survey you will be asked to provide your email so that you can be contacted to participate in follow-up surveys after three months and six months. The follow-up surveys will each take approximately 30-45 minutes to complete.

**Risks and Harms**
There are no known risks or harms of completing this study. However, some questions may be related to a sensitive topic, and participants may refuse to answer any questions. If after completing this study you have concerns about your safety or mental health, please ask the experimenter for information on where you can obtain mental health services or use any of the resources included at the end of the survey.

**Benefits**
There are no direct benefits from completing this study. Information gathered from this study may supplement the psychological community in the knowledge of the relations between university life and disordered eating.

**Confidentiality**
Contact information will be collected from participants in order to participate in follow-up surveys. This information will be kept separate from participant data, and a unique code will be assigned to participants’ responses. De-identified data will be retained for seven years as per regulatory guidelines. Representatives of The University of Western Ontario Non-Medical Research Ethics Board may require access to your study-related records to monitor the conduct of the research.

Your survey responses will be collected through a secure online survey platform called Qualtrics. Qualtrics uses encryption technology and restricted access authorizations to protect all data collected. In addition, Western’s Qualtrics server is in Ireland, where privacy standards are maintained under the European Union safe harbour framework. The data will then be exported from Qualtrics and securely stored on Western University's server.

In reports of this study, only aggregated group data will be presented.

**Compensation**
You will be entered into a prize draw for one of four $25 Amazon gift cards using the email you provide at the end of the survey. You will be entered into additional draws for one of 4 $50 gift cards for the three-month survey, and one of 2 $100 gift cards for the six-month survey. The prize draws will take place after the six-month follow up period.

**Rights of Participants**
Participation in this study is voluntary. You may refuse to participate, refuse to answer any questions at any point in this study, and decide to withdraw from this study at any time without penalty. You do not waive any legal rights by consenting to participate.

**Contact Information**
Please contact Dr. Lindsay Bodell or Samantha Withnell with any questions you may have.

If you have any questions about your rights as a research participant or the conduct of this study, you may contact The Office of Human Research Ethics.

**Consent**
This study has been explained to me and any questions I had have been answered. I know that I may leave the study at any time. You indicate your voluntary agreement to participate by responding to the questions.
Appendix D: Baseline and Three-Month Debriefing Form

Project Title: Weight Stigma Internalization and Quality of Life among Undergraduate Students

Principal Investigator: Dr. Lindsay Bodell, The University of Western Ontario
e-mail: lbodell@uwo.ca; phone: 519-661-2111 x 80486.

Graduate Student Researcher: Samantha Withnell, The University of Western Ontario. E-mail: swithnell@uwo.ca; phone: 519-661-2111 x 87316

Thank you for your participation in this study! The data you provided will help us better understand the impacts of weight stigma on young adults, and other factors that contribute to negative body image and disordered eating. In the future, this information could inform efforts to reduce weight stigma and prevent eating disorders. We realize that thinking about and answering some of the questions may have been unpleasant, but we hope that you found the experience of participating in this study worthwhile, because you are in a sense helping others.

This study includes follow up surveys after three and six months. Your email entered below will be kept separately from the data you provided and used to contact you for the follow up surveys. Your email will also be used as your entry for the gift card draw.

If you would like to learn more about this study or have any further questions or concerns, please contact the Psychobiology of Eating and Related Disorders (PEAR) lab at Western University at 519-661-2111. If after completing this study you have concerns about your mental health, please contact the experimenter for information on where you can obtain mental health services or use any of the resources listed below:
London Area Resource List

Canadian Mental Health Association – Middlesex
24/7 Mental Health and Addictions Crisis Centre

Western University Psychological Services (for students)
Western Student Services Building

Reach Out 24/7 Crisis Services

The Adult Eating Disorders Service
London Health Sciences Center

Hope’s Garden
Eating Disorders Support
Appendix E: Six-Month Debriefing Form

**Project Title:** Weight Stigma Internalization and Quality of Life among Undergraduate Students

Principal Investigator: Dr. Lindsay Bodell, The University of Western Ontario

E-mail: [redacted]

Graduate Student Researcher: Samantha Withnell, The University of Western Ontario. E-mail: [redacted]

Thank you for your participation in this study! The data you provided will help us better understand the impacts of weight stigma on young adults, and other factors that contribute to negative body image and disordered eating. In the future, this information could inform efforts to reduce weight stigma and prevent eating disorders. We realize that thinking about and answering some of the questions may have been unpleasant, but we hope that you found the experience of participating in this study worthwhile, because you are in a sense helping others.

Previous research has shown that efforts to maintain significant weight loss can lead to weight gain and disordered eating behaviors. However, no study has examined whether internalizing negative stereotypes about being overweight explains these outcomes. We think that internalization of weight stigma will be related to changes in body image, quality of life, and eating behaviors over time. If you would like to learn more about this study or have any further questions or concerns, please contact the Psychobiology of Eating and Related Disorders (PEAR) lab at Western University at [redacted]. If after completing this study you have concerns about your mental health, please contact the experimenter for information on where you can obtain mental health services or use any of the resources listed below:
London Area Resource List

Canadian Mental Health Association – Middlesex
24/7 Mental Health and Addictions Crisis Centre

Western University Psychological Services (for students)
Western Student Services Building
Room 4113

Reach Out 24/7 Crisis Services

The Adult Eating Disorders Service
London Health Sciences Center

Hope’s Garden
Eating Disorders Support
Curriculum Vitae
Samantha Jade Withnell, B.A. (Hons.)
Department of Psychology, University of Western Ontario

Education

<table>
<thead>
<tr>
<th>Year</th>
<th>Degree</th>
<th>Institution</th>
<th>Advisor(s)</th>
</tr>
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<tbody>
<tr>
<td>2019 – 2021</td>
<td>M.Sc. Candidate, Psychology (Clinical Science and Psychopathology), University of Western Ontario</td>
<td>Lindsay Bodell, Ph.D.</td>
<td></td>
</tr>
<tr>
<td>(expected)</td>
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<tr>
<td>2011 - 2017</td>
<td>B.A. (Honors), Psychology, University of Calgary</td>
<td>First Class, Women’s Studies Minor</td>
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<tr>
<td></td>
<td></td>
<td>Thesis: Priming attentional biases in women with high and low body satisfaction</td>
<td>Kristin M. von Ranson, Ph.D., FAED &amp; Christopher Sears, Ph.D.</td>
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Peer-Reviewed Publications  
Orcid ID 0000-0002-3770-6379


Manuscripts in Progress


Conference Presentations


2. Withnell, S., & Bodell, L. (March 2021). *Attempts to maintain weight loss mediate the relationship between internalization of weight stigma and disordered eating in women*, oral presentation at the Western Research Forum (institutional graduate research conference), Virtual.


Invited Talks (*indicates shared co-authorship*)


2. Withnell, S. (2019). *Fitness and Thinness on Social Media*, workshop presented for the Women’s Resource Centre at the University of Calgary, Calgary, AB.

Grants, Awards, & Recognitions *all amounts in Canadian Dollars*

<table>
<thead>
<tr>
<th>Year</th>
<th>Grant/Recognition</th>
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<tbody>
<tr>
<td>2021-2022</td>
<td>Ontario Graduate Scholarship, <em>University of Western Ontario</em> ($15,000)</td>
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<tr>
<td>2021</td>
<td>Student/Early Career Researcher Travel Fellowship, <em>Academy for Eating Disorders (AED)</em></td>
</tr>
<tr>
<td></td>
<td><em>Included complementary registration to 2021 International Conference on Eating Disorders including Clinical/Research Training Days and Chapter Events</em></td>
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<tr>
<td>2020-2021</td>
<td>Western Graduate Research Award, <em>University of Western Ontario</em> ($500)</td>
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<tr>
<td></td>
<td><strong>Project:</strong> Does internalization of weight bias explain the negative effects of suppressing weight in undergraduate students?</td>
</tr>
<tr>
<td>2017</td>
<td>Best Paper Presentation Award, <em>Department of Psychology Annual Student Conference, University of Calgary</em></td>
</tr>
</tbody>
</table>
Research Experience

2019 - Present  **Psychobiology of Eating and Related Disorders (PEAR) Lab**, Dr. Lindsay Bodell, University of Western Ontario

    **Role:** Lab Co-Coordinator and Graduate Research Assistant
    **Responsibilities:** Recruit, train and manage research assistants; update lab website; design and conduct studies; write and submit manuscripts for publication

2016 - 2018  **Cognition and Emotion Lab**, Dr. Christopher Sears, University of Calgary

    **Role:** Research Assistant
    **Responsibilities:** Designed and conducted study examining the effect of body satisfaction on visual attention using an eye-tracking paradigm; prepared and submitted manuscript for publication

2015 - 2018  **Eating Behaviors Lab**, Dr. Kristin M. von Ranson, University of Calgary

    **Role:** Research Assistant
    **Responsibilities:** Updated lab website; responded to lab inquiries; assisted PI and students with data checking, preliminary analyses and reference management; contributed to design, data collection and analysis for study examining “thinspiration” and “fitspiration” on social networks; contributed to preparation and submission of manuscript for publication

Teaching Experience

Fall 2019 -  **Teaching Assistant**

    **Courses:**
    Abnormal Psychology (Psychology 2310)
    Introduction to Clinical Psychology (Psychology 3301)
    Psychology of Gender (Psychology 2074)
    Introduction to Social Psychology (Psychology 2720)

    **Lectures:**
    Intellectual and Cognitive Assessments (Psychology 3301, 1.5 hour)
    Applying to Graduate Programs in Clinical Psychology (Psychology 3301, 1 hour)

    **Responsibilities:** Moderate discussion and chat in online classroom; deliver weekly interactive tutorials; grade written assignments, participation and examinations; hold one-on-one appointments; proctor student examinations
Clinical Experience

January 2020 - **Coach**, iAIM EDU Mobile Intervention Study, Virtual (Supervisor: Ellen Fitzsimmons-Craft, PhD)

Provides ongoing coaching to college students enrolled in an online guided self-help program for anxiety, depression and eating disorders; maintains user engagement and personalizes program content; answers questions and messages from users; attends weekly supervision with a licensed psychologist

Winter 2020 **Assessment Practicum Student**, Child and Youth Development Clinic, University of Western Ontario (Supervisor: Colin King, PhD)

Administered neuropsychological and psychoeducational assessments (Wechsler Intelligence Scale for Children Fifth Edition, Wechsler Individual Achievement Test Third Edition); scored and interpreted assessments; prepared integrated assessment report; received supervision on diagnostic assessment with children, case conceptualization, and developing school system recommendations

Leadership and Service

May 2021 - Member, *Western Psychology Equity, Diversity, and Inclusion Graduate Committee – Clinical Sub-Committee*

March 2021 - Member, *Social Media Committee, Coalition for the Advancement and Application of Psychological Science*

October 2020 - Member, *Marketing and Outreach Committee, Advocacy Through Action – Western University*

September 2020 - Student Representative, *Graduate Affairs Committee, Western University Department of Psychology*

May 2020 - Treasurer, *UWO Psychology Graduate Student Association*

Summer 2020 - Volunteer, *Middlesex-London Health Unit COVID-19 Support Line*

**Ad-Hoc Reviewer** *Western Undergraduate Psychology Journal*

**Professional Memberships**

*Academy for Eating Disorders*

*Ontario Psychological Association (Student Affiliate)*

*Society for a Science of Clinical Psychology*
### Specialized Training

<table>
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<tr>
<th>Month</th>
<th>Workshop Title</th>
<th>Duration</th>
<th>Training Leaders</th>
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<tbody>
<tr>
<td>May 2021</td>
<td>Introduction to Structural Equation Modeling</td>
<td>Three-day</td>
<td>Drs. Patrick Curran and Daniel Bauer</td>
</tr>
<tr>
<td>April 2021</td>
<td>Brief Therapy and Single Sessions</td>
<td>Four-hour</td>
<td>Drs. Naomi Wiesenthal and Jared French</td>
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<tr>
<td>December 2020</td>
<td>Suicide Assessment</td>
<td>Four-hour</td>
<td>Dr. Marnin Heisel</td>
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<tr>
<td>August 2020</td>
<td>Visualization Fundamentals for Scientists</td>
<td>Four-hour</td>
<td>Kelly Bullock</td>
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<tr>
<td>January 2020</td>
<td>Exposure in Eating Disorders Treatment</td>
<td>One-day</td>
<td>Dr. Glenn Waller</td>
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<tr>
<td>January 2020</td>
<td>CBT-T: Evidence-Based Brief CBT for Adults with Non-Underweight Eating Disorders</td>
<td>One-day</td>
<td>Dr. Glenn Waller</td>
</tr>
<tr>
<td>December 2019</td>
<td>Emotion-Focused Therapy</td>
<td>Four-hour</td>
<td>Dr. Carey Ann DeOliveira</td>
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