Title

Exploring relationships among appetitive traits, negative affect, and binge eating in adults with overweight or obesity

Authors

Pasquale, Ellen K

Strong, David R

Manzano, Michael A

et al.

Publication Date

2024-04-01

DOI

10.1016/j.eatbeh.2024.101871

Peer reviewed

HHS Public Access

Author manuscript

Eat Behav. Author manuscript; available in PMC 2024 June 02.

Published in final edited form as:

Eat Behav. 2024 April ; 53: 101871. doi:10.1016/j.eatbeh.2024.101871.

Exploring Relationships Among Appetitive Traits, Negative Affect, and Binge Eating in Adults with Overweight or Obesity

Ellen K. Pasquale[1,2], **David R. Strong**[3], **Michael A. Manzano**[1,2], **Dawn M. Eichen**[4], **Carol B. Peterson**[5], **Kerri N. Boutelle**[2,3,4]

[1]San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, 6363 Alvarado Court, Suite 103, San Diego, CA 92120, USA

[2]Department of Psychiatry, University of California San Diego, 9500 Gilman Drive, La Jolla, CA, 92093 USA

[3]Herbert Wertheim School of Public Health and Human Longevity Science, University of California San Diego, 9500 Gilman Drive, La Jolla, CA, 92093 USA

[4]Department of Pediatrics, University of California San Diego, 9500 Gilman Drive, La Jolla, CA, 92093 USA

[5]Department of Psychiatry and Behavioral Sciences, University of Minnesota, F282/2A West 2450 Riverside Ave, Minneapolis, MN 55454, USA

Abstract

Binge eating (BE) is a significant public health concern due to its prevalence and impact on mental and physical health. While research has suggested both negative affect and appetitive traits are associated with BE, few studies have investigated these constructs concurrently. Structural equation modeling (SEM) evaluated relationships between negative affect, reward-related appetitive traits, and BE among 293 adults with overweight or obesity (OW/OB) seeking treatment for BE, overeating, and weight management (m age=46.6; m body mass index[BMI]=34.5; 81.2% female; 20.1% Latinx, 60.8% White non-Latinx). BE was related to negative affect (β=0.53; p<0.01) and appetitive traits (β=1.53; p<0.001). Negative affect and appetitive traits were related to one another (r=0.42; p<0.001), and the full model accounted for 77% of the variance in BE. In an exploratory follow-up analysis, multigroup SEM evaluated the above relationships in models stratified by sex. Exploratory findings demonstrated both negative affect and appetitive traits were related to BE across sex, particularly when examining BE cognitions and behaviors. However, relationships in men depended upon BE assessment tool. These findings highlight that both negative affect and appetitive traits are related to BE, and jointly

Correspondence concerning this article should be addressed to Ellen Pasquale, Department of Psychiatry, 9500 Gilman Drive, MC 0874, La Jolla, CA 92093 USA. ekpasquale@ucsd.edu.

Contributors

E.P., D.S., and K.B. conceptualized the manuscript. K.B. obtained funding for the trial and K.B., D.E., and D.S. directed and contributed to data collection. D.S. and M.M. aided in conducting analyses and interpreting results. E.P. wrote the first draft of the manuscript and prepared all tables and figures. D.E. and C.P contributed to writing and editing. All authors provided critical feedback and helped shape the research and final draft of the manuscript.

Conflict of Interest

All authors declare that they have no conflicts of interest.

Availability of Data and Materials: Data available upon reasonable request from the authors.

may represent significant risk and maintenance factors, particularly in adults with OW/OB. Our findings also highlight the importance of future investigation of sex differences in BE and the potential impact of assessment method.

Keywords

binge eating; structural equation modeling; appetitive traits; negative affect

1. Introduction

Binge eating (BE) is a serious public health concern due to its associations with several medical[1,2] and psychiatric comorbidities,[3] resulting in weight gain[4] and reduced quality of life.[5] BE is characterized by eating an objectively large amount of food while experiencing a loss of control (LOC) over one's eating. While BE is a feature of many eating disorders, it also occurs frequently in individuals without threshold eating disorders, particularly in adults with overweight or obesity (OW/OB).[6] Research has suggested that both BE and LOC eating are associated with significant distress and psychological comorbidities, regardless of whether size and frequency of episodes meets full criteria for an eating disorder.[7,8] Prevalence estimates have suggested that 28-37% of adults with OW/OB meet criteria for BED,[9,10] and subthreshold endorsement of BE features have been estimated significantly higher (57-100%).[11,12]

A large body of research has suggested that negative affect is a transdiagnostic antecedent of BE.[13] The affect regulation theory of BE purports that BE is used to reduce negative affect and distress.[14] Cross-sectionally, epidemiological studies and systematic reviews have demonstrated that higher levels of negative affect,[13] depression,[15] and anxiety[3] are associated with increased BE. Further, a systematic review supported the role of negative affect as a temporal antecedent to BE in bulimia nervosa and BED patients,[16] and this finding has also been supported among adults with OW/OB who engage in BE.[17] It is unclear in the literature if certain aspects of BE (i.e., frequency, cognitions, behaviors) have demonstrated greater associations with negative affect. However, it appears that a combination of both diagnostic requirements of a binge episode (i.e., LOC and objectively large amount of food) have been associated with greater levels of negative affect,[18,19] and worsened emotion dysregulation has been associated with increased BE severity.[20] Importantly, most of these studies have been conducted in women-only or majority-women samples, highlighting the need to evaluate these relationships in other gender identities, including cis-gender men.

While associations between negative affect and BE have been established, a growing body of evidence has suggested that reward-related appetitive traits may also impact BE. The Behavioral Susceptibility Theory (BST) suggests that appetitive traits are genetically determined and interact with the obesogenic environment to promote overeating and weight gain.[21,22] Two widely investigated appetitive traits are food responsiveness (FR) and satiety responsiveness (SR). FR includes urges to eat based on the sight, smell, or taste of palatable food while SR refers to stopping eating in relation to physical fullness sensations. Two

reviews which incorporated neuroimaging, neurocognitive, and behavioral tasks supported the relationship between FR and BE both cross-sectionally and prospectively.[23,24] Relatedly, reward-based eating is characterized by lack of control over eating, lack of satiation, and preoccupation with food.[25] Reward-based eating is not inherently pathological but reflects a strong eating drive in response to the reward of highly palatable food.[25] Reward-based eating includes aspects of FR, which may serve to maintain the high reward value of food and can override satiety signals.[25] Evidence from neuroimaging studies has suggested that increased reward-based eating is associated with BE.[26] While the role of SR in BE has been less frequently studied, results from standardized meal paradigms found that both children and adults with BE reported lower levels of SR as compared to individuals without BE.[27,28] Lastly, interventions that aimed to reduce FR through food cue exposure and improve SR through appetite awareness training have demonstrated reductions in BE and LOC eating episodes.[29,30]

While research has suggested that both negative affect and appetitive traits are associated with BE, few studies have investigated relationships among the 3 constructs concurrently. Evidence has suggested that negative affect and FR are related to one another both cross-sectionally[31] and prospectively,[32-34] and may impact one another as well as eating behavior. Recently, frameworks have been proposed integrating of aspects of negative affect, reward responsivity, and BE behavior among both youth and adults.[35,36] While these reviews have suggested that both negative affect and appetitive traits are related to one another, and may together exacerbate BE, more research is needed to investigate these relationships concurrently among adults with OW/OB.

There are very few studies that have examined whether relationships between negative affect, appetitive traits, and BE may differ by sex. Two studies reported a stronger relationship between negative affect and BE in women compared to men.[37,38] Although several studies have examined sex or gender differences in relationships between reward-related appetitive traits and BE, measured constructs have been inconsistent and results mixed.[39,40] Understanding factors that contribute to BE behavior in adults with OW/OB, examining these questions using validated measures and a rigorous statistical approach, and exploring these relationships by sex can inform BE prevention and treatment targets in OW/OB.

The present study was a secondary data analysis to elucidate the relationships among negative affect (depression and anxiety), appetitive traits (FR, SR and reward-based eating), and BE in a sample of treatment-seeking adults with OW/OB. We used 2 well-validated questionnaires to assess aspects of BE: BE frequency measured by the Eating Disorder Examination Questionnaire (EDE-Q) and BE cognitions and behaviors measured by the Binge Eating Scale (BES). We hypothesized that negative affect and appetitive traits would demonstrate significant independent positive relationships with BE and with one another. We also conducted an exploratory follow-up analysis that aimed to examine relationships among negative affect, appetitive traits, and BE in models stratified by sex.

2. Methods

Data for this secondary analysis were drawn from the Providing Adults Collaborative Interventions for Ideal Changes trial (NCT02516839), a randomized control trial targeting BE, overeating, and body weight for adults with OW/OB. Recruitment methods, measures, interventions, and outcomes have been detailed in full in previous publications.[41,42] Participant inclusion criteria included aged 18-65 years, BMI ≥ 25 and ≤ 45 kg/m^2, English language of at least the 5th grade reading level, and willingness to participate in study visits over 2 years. Exclusion criteria included serious current physical disease (e.g., diabetes), any medical condition that would make physical activity unsafe, current substance use disorder, current or planned pregnancy or lactation, and any medical or psychological problems that could make adherence to the study protocol difficult or dangerous (e.g., purging). Measures for the current study were collected during baseline assessments completed at the University of California San Diego (UC San Diego) prior to randomization. The study was approved by the Institutional Review Board at UC San Diego (151110) and written consent was obtained from all participants.

2.1 Measures

2.1.1. Demographics and Anthropometrics—Participants self-reported their age, sex assigned at birth (female or male), and race/ethnicity as part of baseline assessments. We use the term sex to refer to biological attributes associated with an individual's physical and physiological features. Height was measured in triplicate to the nearest 0.1 cm using a portable Schorr stadiometer (Schorr Inc., Olney, MD). Weight was measured in duplicate to the nearest 0.1 kg using a calibrated digital Tanita scale (model WB 110-A). The values obtained at the baseline assessment were averaged to calculate BMI (weight[kg]/height[m^2]).

2.1.2 The Patient Health Questionnaire—9 (PHQ-9)—The PHQ-9[43] is a 9-item questionnaire that assessed depression symptoms and severity. The PHQ-9 demonstrated acceptable internal consistency (α = 0.76, omega-hierarchical [ω_H] = 0.64).

2.1.3 The Generalized Anxiety Questionnaire—7 (GAD-7)—The GAD-7[44] is a 7-item questionnaire that assessed anxiety symptoms and severity. The GAD-7 demonstrated good internal consistency (α = 0.83, coefficient H [H] = 0.51).

2.1.4 The Reward-Based Eating Drive Scale (RED)—The RED[25] is a 9-item questionnaire that assessed lack of control over eating, lack of satiation, and preoccupation with food. The RED demonstrated strong internal consistency (α = 0.89, ω_H = 0.73).

2.1.5 Adult Eating Behavior Questionnaire (AEBQ)—The AEBQ is an adaptation of the Children's Eating Behavior Questionnaire (CEBQ)[45] that assessed appetitive traits. Language from the CEBQ was modified to create the adult version of the CEBQ's FR and SR subscales by the research team, maintaining the likeness of each item. The FR subscale demonstrated strong internal consistency (α = 0.85, H = 0.61), and the SR subscale demonstrated acceptable internal consistency (α = 0.70, H = 0.36).

2.1.6 Binge Eating Scale (BES)—

The BES[46] is a 16-item questionnaire that assessed BE symptoms and severity on a continuous scale with scores ranging from 0-46. In addition to BE behavior, the BES assessed upstream cognitions and attitudes related to BE. The BES demonstrated strong internal consistency (α = 0.87, ω_H = 0.74).

2.1.7 Eating Disorder Examination Questionnaire (EDE-Q)—

The EDE-Q[47] is a 28-item questionnaire adaptation of the Eating Disorder Examination interview that assessed eating disorder attitudes and behaviors. Item 15 from the EDE-Q assessed number of days the respondent has eaten an unusually large amount of food with a sense of LOC (i.e., the 2 diagnostic requirements of a binge episode) in the past month. Responses on this item range from 0-28 days.

2.2 Statistical Analysis

The proposed measurement model was comprised of 3 latent variables: negative affect, appetitive traits, and BE. Negative affect (indicated by PHQ-9 and GAD-7 total scores) and appetitive traits (indicated by AEBQ-FR and -SR subscale scores and RED total scores) were modeled as exogenous (independent) latent variables, and BE (indicated by BES total scores and EDE-Q BE frequency item) was modeled as an endogenous (dependent) latent variable. First, the measurement model was evaluated to ensure acceptable fit between observed and latent variables. Subsequently, SEM assessed relationships among negative affect, appetitive traits, and BE, adjusting for sex, age, ethnicity, race, and BMI. These covariates were chosen because demographic factors have been associated with differential BE,[48,49] and increased BE has been associated with weight gain.[4] As an exploratory follow-up analysis, multigroup SEM assessed the above relationships in separate models stratified by sex. In this multigroup model, SEM was performed concurrently in 2 separate models in which the latent BE construct was indicated by 1) BES total scores, which we define as BE cognitions and behaviors, and 2) EDE-Q BE frequency item which we define as BE frequency, singularly. BE measures were separated in the multi-group model by necessity to maintain positive variance of all outcome variables and establish plausible models.

All study variables were analyzed using R version 4.1.0[50] using the “psych”[51] and “lavaan”[52] packages. Total or mean scores (if applicable) for observed variables were calculated and included in the present analysis if at least 50% of items were completed using mean item-imputation for missing data within a scale or subscale. Observations were excluded from analyses if any study variable required for SEM was missing. BES and RED scores were scaled by dividing by 10 to match scale of other included variables. Categorical race and ethnicity variables were dichotomized into White/non-White and Latinx/non-Latinx respectively, for incorporation into SEM.

Internal consistency was assessed using Cronbach’s alpha (α) and McDonald’s coefficient omega (ω). Omega-hierarchical (ω_H) was reported with $\omega_H \geq 0.65$ indicative of strong dimensionality.[53] For scales with less than 9 items, coefficient H from Mokken scale analysis (H) was reported as the second metric of dimensionality,[54] with $0.3 \leq H$ indicative of a weak scale and $H \geq 0.5$ indicative of a strong scale.[55] Overall model fit for both measurement and structural models was determined using 3 indices: the Comparative Fit

Index (CFI),[56] the root mean square error of approximation (RMSEA),[57] and standardized root mean residual (SRMR).[58] CFI values greater than .95 indicated good fit. RMSEA and SRMR values less than .08 indicated acceptable fit and values less than .05 indicated good fit. The likelihood ratio χ^2 was also reported.

3. Results

3.1 Demographics and Descriptive Statistics

In total, 293 participants had measurements for all included variables.[1] Participants had a mean age of 46.6 years, 81.2% (n = 238) of the sample was female, and 60.8% (n = 178) identified as non-Latinx White. Detailed demographic information for the sample by sex is presented in Table 1. Means and standard deviations by sex of scores on measures used to indicate latent variables are presented in Supplementary Table 1. The range of scores measuring BE cognitions and behaviors was somewhat wider for women (0 - 44) compared to men (0 - 31). Ranges of BE frequency were similar between women (0 - 28 days) and men (0 - 26 days). Approximately one quarter (26%, n = 76) of the sample met criteria for BED, which was somewhat higher in women (27.3%, n = 65) than men (20.0%, n = 11). Nonzero endorsement of BE frequency was similar between women (50.8%, n = 121) and men (47.3%, n = 26).

3.2 Measurement Model and Structural Equation Model

First, the measurement model containing the 3 hypothesized latent variables was evaluated, which demonstrated good fit to the data (CFI = 0.99; RMSEA = 0.06; SRMR = 0.04; $\chi^2(11) = 25.66$, $p = 0.007$).

Subsequently, the structural model was evaluated with pathways from negative affect and appetitive traits to BE (Figure 1). The model demonstrated acceptable fit to the data per all indices evaluated (CFI = 0.97; RMSEA = 0.05; SRMR = 0.05; $\chi^2(11) = 65.96$, $p = 0.008$). The direct effects from negative affect and appetitive traits to BE were both strong and statistically significant. The covariance between negative affect and appetitive traits was moderate ($r = 0.42$; $p < 0.001$). The overall R^2 for this model was 0.77.

3.3 Exploratory Multigroup Structural Equation Models by Sex

The exploratory multigroup structural model for women and men with pathways from negative affect and appetitive traits to BE cognitions and behaviors is presented in Figure 2 and for women and men with pathways from negative affect and appetitive traits to BE frequency is presented in Figure 3. Both exploratory multigroup models demonstrated good fit per all indices evaluated (CFI = 0.995; RMSEA = 0.02; SRMR = 0.05; equivalent for both models; BES $\chi^2(78) = 946.62$, $p < 0.001$; BE days $\chi^2(78) = 729.16$, $p < 0.001$). It was deemed inappropriate to test for sex differences between the pathways in models separated

[1] In total, 298 participants completed all questionnaires but 5 were excluded from analyses due to missing data. Of questionnaire data from 293 included participants, 0.3% of items (range for scales/subscales was 0.1 – 0.4%) were missing. 9.9% of cases required any imputation, and for most cases, this was only a single skipped item. The BES (16 items) was the only exception in which 3 items were imputed for 1 case.

by sex due to the small male sample size ($N = 55$).[59] Importantly, the results for men should be interpreted with caution, as there were only 55 men in the sample.

In the exploratory multigroup model for BE cognitions and behaviors, the relationship between BE cognitions and behaviors and negative affect was weak but statistically significant in both women and men. The relationship between BE cognitions and behaviors and appetitive traits was strong and statistically significant in both women and men. The relationships between BE cognitions and behaviors and planned covariates were nonsignificant across sex. Additionally, the covariance between negative affect and appetitive traits was moderate and statistically significant for both women and men. The overall R^2 for this model was 0.64 for women and 0.82 for men.

In the exploratory multigroup model for BE frequency, the relationship between BE frequency and negative affect was strong and statistically significant in women but was not significant in men. The relationship between BE frequency and appetitive traits was also strong and statistically significant in women but was not significant in men. The relationships between BE frequency and planned covariates were nonsignificant across sex. Additionally, the covariance between negative affect and appetitive traits was moderate and statistically significant for both women and men. The overall R^2 for this model was 0.28 for women and 0.12 for men.

4. Discussion

This study evaluated relationships between negative affect, appetitive traits, and BE among a sample of treatment-seeking adults with OW/OB. We found strong relationships between negative affect and BE and appetitive traits and BE, a moderate covariance between negative affect and appetitive traits, and that together these variables accounted for a large percentage of BE variance. These findings are consistent with existing literature, and provide evidence for the affect regulation model of BE and the role of appetitive traits in BE both singularly and jointly among treatment-seeking adults with OW/OB. These findings highlight the importance of assessing negative affect and appetitive traits concurrently due to their ability to account for significant variance in BE.

In all examined models, appetitive traits, compared to negative affect, demonstrated numerically stronger relationships with BE, suggesting that appetitive traits may be a more salient contributor to BE than negative affect. Further, negative affect and appetitive traits were significantly related to one another across sex, demonstrating a consistent, moderate covariance in all models. While examining the temporal relationship between constructs was beyond the scope of this study, our findings suggest that negative affect and appetitive traits were related to each other and to BE in both sexes. It is possible that together, negative affect and appetitive traits may present larger risk and/or more potent maintenance factors for BE than alone. Further, the hypothesis that the interaction of negative affectivity and elevated reward responsivity for food increases risk for the development of BED and adult obesity has been discussed in a previous review paper.[35] Thus, future research is needed to investigate if appetitive traits may potentially moderate the relationship between negative affect and BE.

Upon exploring these relationships by sex and separate BE constructs, we found that both negative affect and appetitive traits were related to BE across sex, particularly when examining BE cognitions and behaviors. However, this relationship was only observed in women when examining BE frequency. Numeric differences in R^2 values were observed across sex and BE models. Negative affect and appetitive traits accounted for a larger proportion of the variance in BE cognitions and behaviors in men while negative affect and appetitive traits accounted for a larger proportion of the variance in BE frequency in women. However, negative affect and appetitive traits did not account for as much variance in BE frequency in both sexes. This observed discrepancy may reflect the ability of the BE cognitions and behaviors construct to capture subthreshold BE, as compared to BE frequency, which measures the occurrence of a discrete behavior, and specifies both LOC and objectively large criteria. Due to our small sample of men we are not able to make statistical inferences about sex differences in BE relationships.

We were unable to form a plausible multigroup model separated by sex that maintained the latent BE construct, for which we offer a number of potential explanations. While there were more men in this study than in many other investigations of eating disorder symptoms in men, it was not large (18.8%, $n = 55$). It is possible that differential item functioning may have influenced the relationship between the BE frequency item and BES total scores in men, therefore rendering the model with the combined BE latent construct implausible. Multiple previous studies have found poorer psychometric functioning (i.e., lower criterion and predictive validity) of eating disorder measures among male compared to female respondents.[60,61] This hypothesis is also consistent with results of a previous study which found a dimensional measure of BE (the Eating Disorder Diagnostic Scale[62]) to yield greater specificity when examining gender differences in the severity of BE symptoms.[37] This phenomenon may have contributed to our exploratory findings which showed consistent relationships across sex between negative affect and appetitive traits with BE cognitions and behaviors when measured dimensionally, but a lack of relationship between negative affect and appetitive traits with BE frequency in men when measured categorically and for which endorsement of LOC was required. Literature suggests that men are just as likely as women to engage in overeating,[63] but women are more likely to endorse LOC eating.[64] Results from a qualitative study in men suggested that while overeating is consistent with the stereotypical, Western male gender role, LOC is not.[65] Thus, men may experience the same phenomenon as women but are more reluctant to endorse or label LOC. While we are unable to form conclusions due to the exploratory nature of this sex-based analysis, our findings highlight the need for more research on potential sex and gender bias in the measurement of eating disorder symptoms in men.[66]

The present study has several strengths. The study sample had some racial and ethnic diversity and the evaluation of appetitive traits, negative affect, and BE, was conducted concurrently, included multiple measures for each latent construct, and used advanced multivariable statistical modelling techniques with *a priori* model fit threshold cutoffs. However, several limitations are important in interpreting the study findings. This study was cross-sectional and included only treatment-seeking individuals who were predominantly women; thus, no causal inference can be made, results cannot be generalized to the population, and conclusions about BE in men should be interpreted with caution. Further, results cannot

be generalized to nonbinary, gender fluid, and transgender populations. Due to the small male sample, we were unable to evaluate for sex differences in SEM pathways, and the estimates we report may lack precision. While the BE frequency item from the EDE-Q assessed BE directly, it did not capture LOC eating which did not meet the size criteria for BE but may have been clinically significant, and it also required endorsement of the phenomenon of LOC, which some individuals may have been reluctant to endorse. Lastly, thorough psychometric testing has yet to be completed for the AEBQ FR and SR subscales adapted by the research team. Evaluating how BE symptoms and behavior may differ by sex and among gender identities, and examining potential sex and gender bias in existing eating disorder measurement tools and reporting of LOC will be useful to explore. Future research in this domain will benefit from choosing measurement tools that consider the dimensional nature of BE.

5. Conclusions

In sum, the present study demonstrated significant relationships between negative affect, appetitive traits, and BE, supporting the affect regulation model of BE and extension of the Behavioral Susceptibility Theory to BE, as well as their joint utility. Treatment approaches that address appetitive traits in addition to emotion regulation skills may demonstrate increased effectiveness in individuals with OW/OB who engage in BE. Research is needed to investigate the potential moderating role of appetitive traits on the relationship between negative affect and BE, to explore both sex and gender differences in BE experiences, and evaluate potential sex and gender bias in the current measurement of BE in men.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

The authors would like to thank the participants in the PACIFIC study and the staff at the Center for Healthy Eating and Activity Research (CHEAR), without whom this research would not be possible.

Role of Funding Sources

Funding for this study was provided by the National Institutes of Health (grants R01DK103554, UL1TR001442, and K23DK114480). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The National Institutes of Health had no role in the study design, collection, analysis or interpretation of the data, writing the manuscript, or the decision to submit the paper for publication.

Abbreviations:

BE	binge eating
OW/OB	overweight or obesity
BMI	body mass index
BES	Binge Eating Scale

EDE-Q	Eating Disorder Examination Questionnaire
CFI	comparative fit index
RMSEA	root mean square error of approximation
SRMR	standardized root mean residual
BED	binge eating disorder
FR	food responsiveness
SR	satiety responsiveness
UC San Diego	University of California San Diego
PHQ-9	Patient Health Questionnaire—9
GAD-7	Generalized Anxiety Disorder Questionnaire—7
RED	Reward-based Eating Drive Scale
AEBQ	Adult Eating Behavior Questionnaire
ω_H	omega-hierarchical
SEM	structural equation modeling
H	coefficient H from Mokken scale analysis

References

1. Mitchell JE. Medical comorbidity and medical complications associated with binge-eating disorder. Int J Eat Disord. 2016;49(3):319–323. doi:10.1002/eat.22452 [PubMed: 26311499]
2. Solmi F, Moreno AB, Lewis G, Angélica Nunes M, de Jesus Mendes da Fonseca M, Harter Griep R. Longitudinal association between binge eating and metabolic syndrome in adults: Findings from the ELSA-Brasil cohort. Acta Psychiatr Scand. 2021;144(5):464–474. doi:10.1111/acps.13356 [PubMed: 34333757]
3. Kessler RC, Berglund PA, Chiu WT, et al. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. Biol Psychiatry. 2013;73(9):904–914. doi:10.1016/j.biopsych.2012.11.020 [PubMed: 23290497]
4. Villarejo C, Fernández-Aranda F, Jiménez-Murcia S, et al. Lifetime obesity in patients with eating disorders: Increasing prevalence, clinical and personality correlates. Eur Eat Disord Rev. 2012;20(3):250–254. doi:10.1002/erv.2166 [PubMed: 22383308]
5. Ágh T, Kovács G, Pawaskar M, Supina D, Inotai A, Vokó Z. Epidemiology, health-related quality of life and economic burden of binge eating disorder: A systematic literature review. Eat Weight Disord - Stud Anorex Bulim Obes. 2015;20(1):1–12. doi:10.1007/s40519-014-0173-9
6. Mustelin L, Bulik CM, Kaprio J, Keski-Rahkonen A. Prevalence and correlates of binge eating disorder related features in the community. Appetite. 2017;109:165–171. doi:10.1016/j.appet.2016.11.032 [PubMed: 27899295]
7. Vannucci A, Theim KR, Kass AE, et al. What constitutes clinically significant binge eating? Association between binge features and clinical validators in college-age women. Int J Eat Disord. 2013;46(3):226–232. doi:10.1002/eat.22115 [PubMed: 23386591]

8. Sonneville KR, Horton NJ, Micali N, et al. Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: Does loss of control matter? JAMA Pediatr. 2013;167(2):149–155. doi:10.1001/2013.jamapediatrics.12 [PubMed: 23229786]
9. Hay P, Girosi F, Mond J. Prevalence and sociodemographic correlates of DSM-5 eating disorders in the Australian population. J Eat Disord. 2015;3:19. doi:10.1186/s40337-015-0056-0 [PubMed: 25914826]
10. Spitzer RL, Yanovski S, Wadden T, et al. Binge eating disorder: its further validation in a multisite study. Int J Eat Disord. 1993;13(2):137–153. [PubMed: 8477283]
11. Greeno CG, Wing RR, Shiffman S. Binge antecedents in obese women with and without binge eating disorder. J Consult Clin Psychol. 2000;68(1):95–102. doi:10.1037/0022-006X.68.1.95 [PubMed: 10710844]
12. Mason TB, Do B, Chu D, Belcher BR, Dunton GF, Lopez NV. Associations among affect, diet, and activity and binge-eating severity using ecological momentary assessment in a non-clinical sample of middle-aged fathers. Eat Weight Disord - Stud Anorex Bulim Obes. 2022;27(2):543–551. doi:10.1007/s40519-021-01191-8
13. Leehr EJ, Krohmer K, Schag K, Dresler T, Zipfel S, Giel KE. Emotion regulation model in binge eating disorder and obesity - a systematic review. Neurosci Biobehav Rev. 2015;49:125–134. doi:10.1016/j.neubiorev.2014.12.008 [PubMed: 25530255]
14. Lacey JH. Pathogenesis. In: Current Approaches: Bulimia Nervosa. Duphar; 1986:17–26.
15. Araujo DMR, Santos GF da S, Nardi AE. Binge eating disorder and depression: A systematic review. World J Biol Psychiatry. 2010;11(2-2):199–207. doi:10.3109/15622970802563171 [PubMed: 20218783]
16. Haedt-Matt AA, Keel PK. Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. Psychol Bull. 2011;137(4):660–681. doi:10.1037/a0023660 [PubMed: 21574678]
17. Goldschmidt AB, Crosby RD, Cao L, et al. Ecological momentary assessment of eating episodes in obese adults. Psychosom Med. 2014;76(9):747–752. doi:10.1097/PSY.0000000000000108 [PubMed: 25373891]
18. Racine SE, Horvath SA. Emotion dysregulation across the spectrum of pathological eating: Comparisons among women with binge eating, overeating, and loss of control eating. Eat Disord. 2018;26(1):13–25. doi:10.1080/10640266.2018.1418381 [PubMed: 29384463]
19. Berg KC, Crosby RD, Cao L, et al. Negative affect prior to and following overeating-only, loss of control eating-only, and binge eating episodes in obese adults. Int J Eat Disord. 2015;48(6):641–653. doi:10.1002/eat.22401 [PubMed: 25808854]
20. Mikhail ME, Fowler N, Burt SA, et al. A daily diary study of emotion regulation as a moderator of negative affect-binge eating associations. Int J Eat Disord. 2022;55(10):1305–1315. doi:10.1002/eat.23768 [PubMed: 35779074]
21. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. Am J Clin Nutr. 2008;88(1):22–29. doi:10.1093/ajcn/88.1.22 [PubMed: 18614720]
22. Llewellyn C, Wardle J. Behavioral susceptibility to obesity: Gene–environment interplay in the development of weight. Physiol Behav. 2015;152:494–501. doi:10.1016/j.physbeh.2015.07.006 [PubMed: 26166156]
23. Kober H, Boswell RG. Potential psychological & neural mechanisms in binge eating disorder: Implications for treatment. Clin Psychol Rev. 2018;60:32–44. doi:10.1016/j.cpr.2017.12.004 [PubMed: 29329692]
24. Stojek M, Shank LM, Vannucci A, et al. A systematic review of attentional biases in disorders involving binge eating. Appetite. 2018;123:367–389. doi:10.1016/j.appet.2018.01.019 [PubMed: 29366932]
25. Epel ES, Tomiyama AJ, Mason AE, et al. The Reward-Based Eating Drive Scale: A self-report index of reward-based eating. PLOS ONE. 2014;9(6):e101350. doi:10.1371/journal.pone.0101350 [PubMed: 24979216]
26. DeGuzman M, Frank GKW. Neuroimaging to study brain reward processing and reward-based learning in binge eating pathology. In: Frank GKW, Berner LA, eds. Binge

Eating: A Transdiagnostic Psychopathology. Springer International Publishing; 2020:121–135. doi:10.1007/978-3-030-43562-2_9

27. Dalton M, Blundell J, Finlayson G. Effect of BMI and binge eating on food reward and energy intake: Further evidence for a binge eating subtype of obesity. Obes Facts. 2013;6(4):348–359. doi:10.1159/000354599 [PubMed: 23970144]
28. Mirch MC, McDuffie JR, Yanovski SZ, et al. Effects of binge eating on satiation, satiety, and energy intake of overweight children. Am J Clin Nutr. 2006;84(4):732–738. [PubMed: 17023698]
29. Boutelle KN, Knatz S, Carlson J, Bergmann K, Peterson CB. An open trial targeting food cue reactivity and satiety sensitivity in overweight and obese binge eaters. Cogn Behav Pract. 2017;24(3):363–373. doi:10.1016/j.cbpra.2016.08.003 [PubMed: 29269997]
30. Boutelle KN, Pasquale EK, Strong DR, Eichen DM, Peterson CB. Reduction in eating disorder symptoms among adults in different weight loss interventions. Eat Behav. 2023;51:101787. doi:10.1016/j.eatbeh.2023.101787 [PubMed: 37639734]
31. Coakley KE, Le H, Silva SR, Wilks A. Anxiety is associated with appetitive traits in university students during the COVID-19 pandemic. Nutr J. 2021;20(1):45. doi:10.1186/s12937-021-00701-9 [PubMed: 33985515]
32. Wagner DD, Boswell RG, Kelley WM, Heatherton TF. Inducing negative affect increases the reward value of appetizing foods in dieters. J Cogn Neurosci. 2012;24(7):1625–1633. doi:10.1162/jocn_a_00238 [PubMed: 22524295]
33. Hepworth R, Mogg K, Brignell C, Bradley BP. Negative mood increases selective attention to food cues and subjective appetite. Appetite. 2010;54(1):134–142. doi:10.1016/j.appet.2009.09.019 [PubMed: 19815043]
34. Loxton NJ, Dawe S, Cahill A. Does negative mood drive the urge to eat? The contribution of negative mood, exposure to food cues and eating style. Appetite. 2011;56(2):368–374. doi:10.1016/j.appet.2011.01.011 [PubMed: 21238524]
35. Tanofsky-Kraff M, Schvey NA, Grilo CM. A developmental framework of binge-eating disorder based on pediatric loss of control eating. Am Psychol. 2020;75:189–203. doi:10.1037/amp0000592 [PubMed: 32052994]
36. Schaefer LM, Forester G, Dvorak RD, Steinglass J, Wonderlich SA. Integrating aspects of affect, reward, and cognition to develop more comprehensive models of binge-eating pathology. Int J Eat Disord. 2023;56(8):1502–1510. doi:10.1002/eat.23971 [PubMed: 37084184]
37. Rosenbaum DL, White KS. The relation of anxiety, depression, and stress to binge eating behavior. J Health Psychol. 2015;20(6):887–898. doi:10.1177/1359105315580212 [PubMed: 26032804]
38. Sultson H, Kreegipuu K, Akkermann K. Exploring the role of momentary positive and negative affect in overeating and binge eating: Evidence for different associations among men and women. Appetite. 2022;168:105758. doi:10.1016/j.appet.2021.105758 [PubMed: 34655665]
39. Levallius J, Monell E, Birgegård A, Clinton D, Forsén Mantilla E. Binge eating and addictive-like behaviours in males and females. Psychol Rep. 2022;125(1):148–166. doi:10.1177/0033294120971750 [PubMed: 33174818]
40. Eneva KT, Murray S, O'Garro-Moore J, et al. Reward and punishment sensitivity and disordered eating behaviors in men and women. J Eat Disord. 2017;5(1):6. doi:10.1186/s40337-017-0138-2 [PubMed: 28228946]
41. Boutelle KN, Eichen DM, Peterson CB, Strong DR, Rock CL, Marcus BH. Design of the PACIFIC study: A randomized controlled trial evaluating a novel treatment for adults with overweight and obesity. Contemp Clin Trials. 2019;84:105824. doi:10.1016/j.cct.2019.105824 [PubMed: 31400516]
42. Boutelle KN, Eichen DM, Peterson CB, et al. Effect of a novel intervention targeting appetitive traits on body mass index among adults with overweight or obesity: A randomized clinical trial. JAMA Netw Open. 2022;5(5):e2212354. doi:10.1001/jamanetworkopen.2022.12354 [PubMed: 35583870]
43. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9. J Gen Intern Med. 2001;16(9):606–613. doi:10.1046/j.1525-1497.2001.016009606.x [PubMed: 11556941]

44. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: The GAD-7. Arch Intern Med. 2006;166(10):1092–1097. doi:10.1001/archinte.166.10.1092 [PubMed: 16717171]
45. Wardle J, Guthrie CA, Sanderson S, Rapoport L. Development of the Children's Eating Behaviour Questionnaire. J Child Psychol Psychiatry. 2001;42(7):963–970. doi:10.1017/S0021963001007727 [PubMed: 11693591]
46. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. Addict Behav. 1982;7(1):47–55. doi:10.1016/0306-4603(82)90024-7 [PubMed: 7080884]
47. Fairburn CG, Beglin Sarah J. Christopher G. Fairburn, Zafra Cooper and Marianne E. O'Connor APPENDIX B Eating Disorder Examination Questionnaire. In: Cognitive Behavior Therapy and Eating Disorders. Guilford Publications; 2008.
48. Kelly-Weeder S, Jennings KM, Wolfe BE. Gender differences in binge eating and behavioral correlates among college students. Eat Weight Disord - Stud Anorex Bulim Obes. 2012;17(3):e200–e202. doi:10.1007/BF03325348
49. Reslan S, Saules KK. Assessing the prevalence of and factors associated with overweight, obesity, and binge eating as a function of ethnicity. Eat Weight Disord - Stud Anorex Bulim Obes. 2013;18(2):209–219. doi:10.1007/s40519-013-0022-2
50. R Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. 2021. https://www.R-project.org/
51. Revelle W. psych: Procedures for psychological, psychometric, and personality research. Northwestern University, Evanston, Illinois. 2021;R package version 2.1.9, https://CRAN.R-project.org/package=psych
52. Rosseel Y lavaan: An R package for structural equation modeling. J Stat Softw. 2012;48(2), 1–36. doi:10.18637/jss.v048.i02
53. Nájera Catalán HE. Reliability, population classification and weighting in multidimensional poverty measurement: A monte carlo study. Soc Indic Res. 2019;142(3):887–910. doi:10.1007/s11205-018-1950-z
54. Mokken RJ. A Theory and Procedure of Scale Analysis: With Applications in Political Research. De Gruyter Mouton; 1971. doi:10.1515/9783110813203
55. Stochl J, Jones PB, Croudace TJ. Mokken scale analysis of mental health and well-being questionnaire item responses: a non-parametric IRT method in empirical research for applied health researchers. BMC Med Res Methodol. 2012;12:74. doi:10.1186/1471-2288-12-74 [PubMed: 22686586]
56. Bentler PM. Comparative fit indexes in structural models. Psychol Bull. 1990;107:238–246. doi:10.1037/0033-2909.107.2.238 [PubMed: 2320703]
57. Steiger JH. Structural model evaluation and modification: An interval estimation approach. Multivar Behav Res. 1990;25(2):173–180. doi:10.1207/s15327906mbr2502_4
58. Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Struct Equ Model Multidiscip J. 1999;6(1):1–55. doi:10.1080/10705519909540118
59. Wolf EJ, Harrington KM, Clark SL, Miller MW. Sample size requirements for structural equation models: An evaluation of power, bias, and solution propriety. Educ Psychol Meas. 2013;76(6):913–934. doi:10.1177/0013164413495237 [PubMed: 25705052]
60. Richson BN, Johnson SN, Swanson TJ, Christensen KA, Forbush KT, Wildes JE. Predicting probable eating disorder case-status in men using the Clinical Impairment Assessment: Evidence for a gender-specific threshold. Eat Behav. 2021;42:101541. doi:10.1016/j.eatbeh.2021.101541 [PubMed: 34332312]
61. Forbush KT, Richson BN, Swanson TJ, et al. Screening for eating disorders across genders in college students: Initial validation of the brief assessment of stress and eating. Int J Eat Disord. 2022;55(11):1553–1564. doi:10.1002/eat.23815 [PubMed: 36135594]
62. Stice E, Telch CF, Rizvi SL. Development and validation of the Eating Disorder Diagnostic Scale: a brief self-report measure of anorexia, bulimia, and binge-eating disorder. Psychol Assess. 2000;12(2):123–131. doi:10.1037//1040-3590.12.2.123 [PubMed: 10887758]

63. Striegel-Moore RH, Rosselli F, Perrin N, et al. Gender difference in the prevalence of eating disorder symptoms. Int J Eat Disord. 2009;42(5):471–474. doi:10.1002/eat.20625 [PubMed: 19107833]

64. Reslan S, Saules KK. College students' definitions of an eating "binge" differ as a function of gender and binge eating disorder status. Eat Behav. 2011;12(3):225–227. doi:10.1016/j.eatbeh.2011.03.001 [PubMed: 21741023]

65. Carey JB, Saules KK, Carr MM. A qualitative analysis of men's experiences of binge eating. Appetite. 2017;116:184–195. doi:10.1016/j.appet.2017.04.030 [PubMed: 28465183]

66. Brown TA, Keel PK. Eating Disorders in Boys and Men. Annu Rev Clin Psychol. 2023;19(1):177–205. doi:10.1146/annurev-clinpsy-080921-074125 [PubMed: 36737595]

Highlights

- Negative affect and appetitive traits both strongly related to binge eating (BE)
- Negative affect and appetitive traits accounted for significant variance in BE
- Negative affect and appetitive traits related moderately to one another

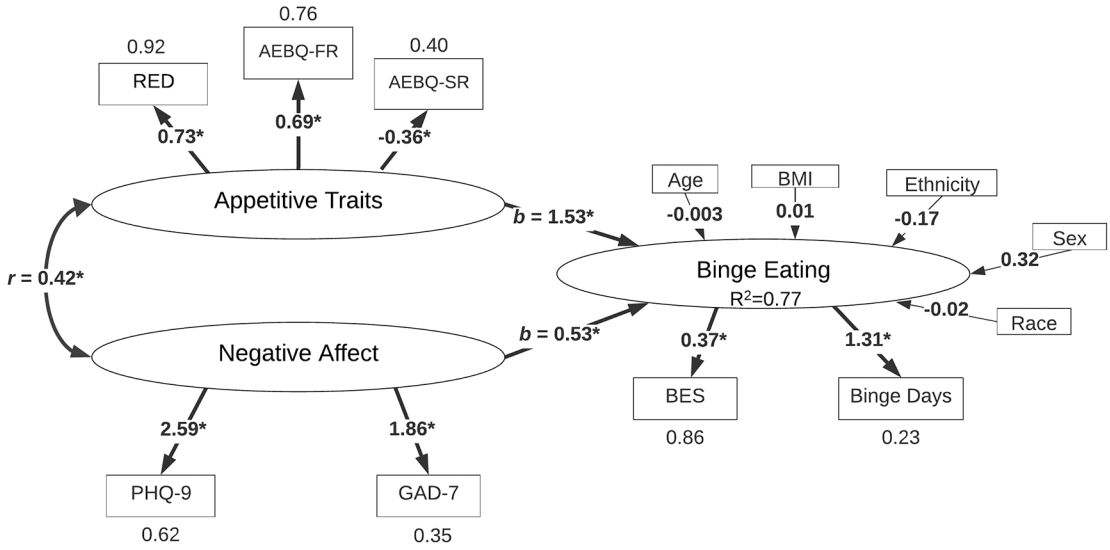

Figure 1. Path Diagram for the Combined Structural Model

Abbreviations: RED = Reward-Based Eating Drive Scale; AEBQ-FR = Adult Eating Behavior Questionnaire Food Responsiveness Scale; AEBQ-SR = Adult Eating Behavior Questionnaire Satiety Responsiveness Scale; PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalized Anxiety Questionnaire-7; BE = binge eating; BMI = body mass index

* Denotes statistically significant loading or correlation ($p \leq 0.01$)

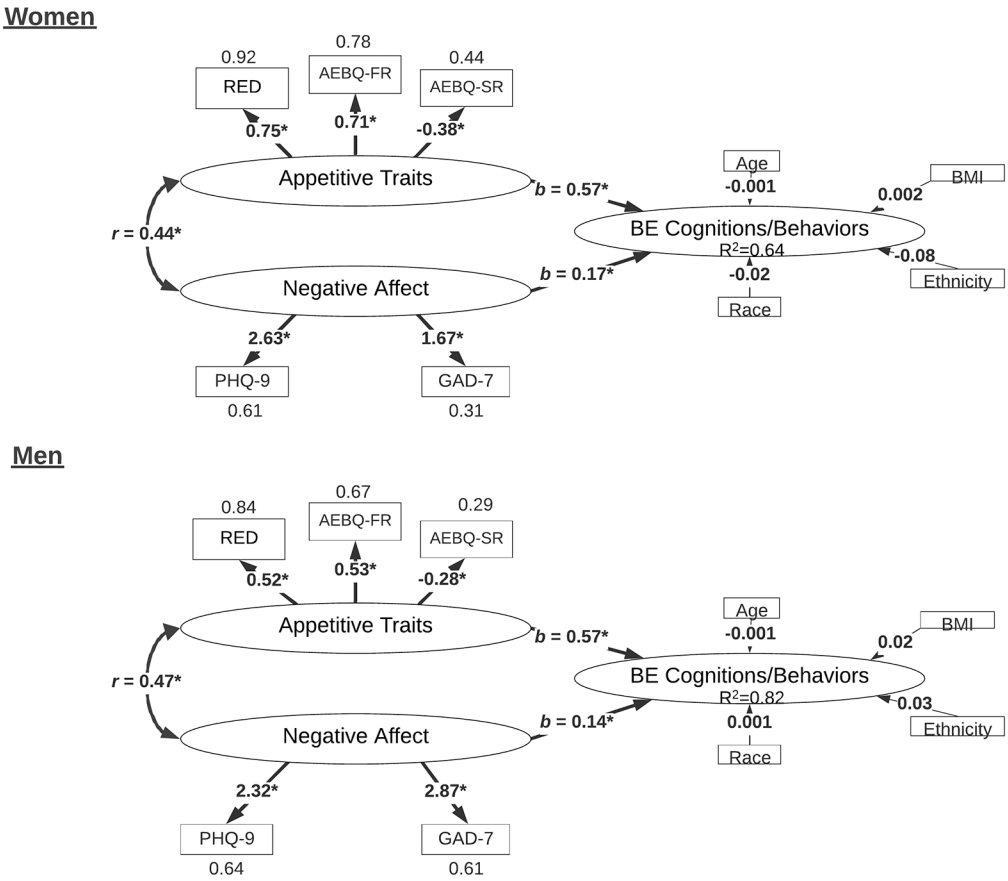

Figure 2. Binge Eating Cognitions and Behaviors Path Diagram for Women and Men

Abbreviations: RED = Reward-Based Eating Drive Scale; AEBQ-FR = Adult Eating Behavior Questionnaire Food Responsiveness Scale; AEBQ-SR = Adult Eating Behavior Questionnaire Satiety Responsiveness Scale; PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalized Anxiety Questionnaire-7; BE = binge eating; BMI = body mass index

*Denotes statistically significant loading or correlation ($p \leq 0.01$)

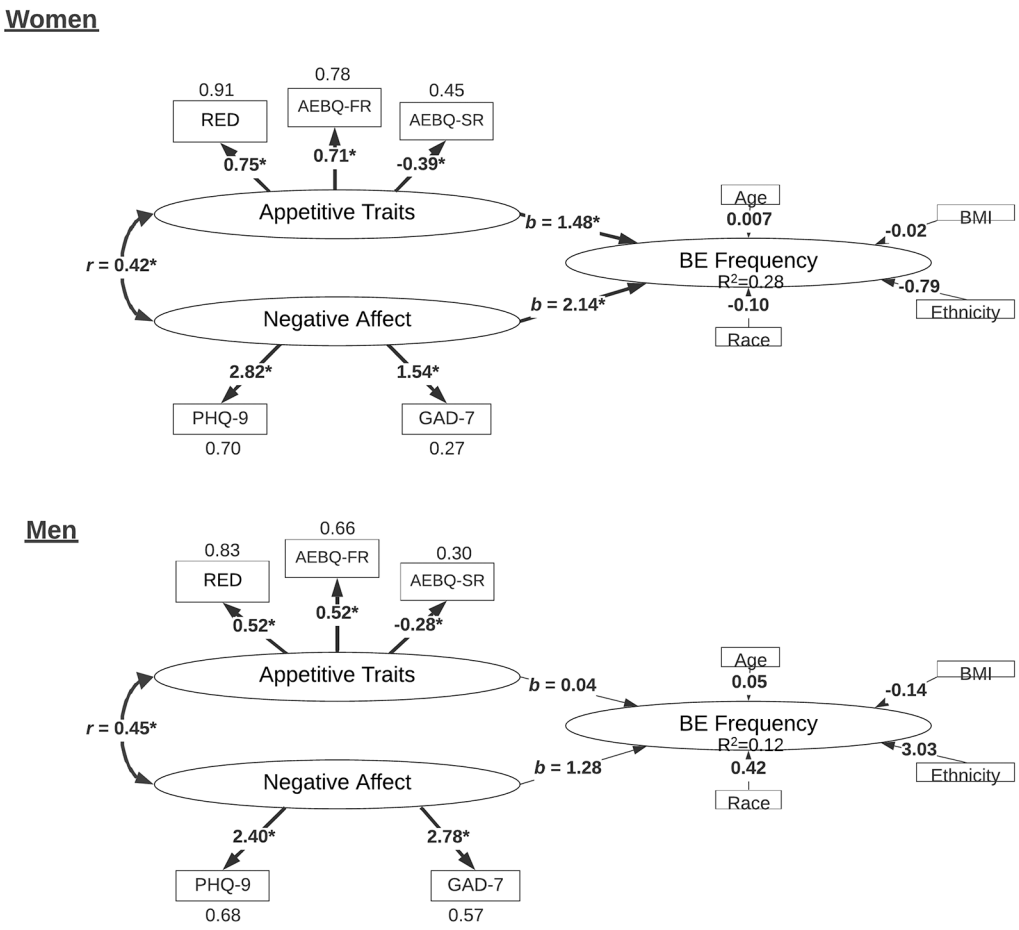

Figure 3. Binge Eating Frequency Path Diagram for Women and Men

Abbreviations: RED = Reward-Based Eating Drive Scale; AEBQ-FR = Adult Eating Behavior Questionnaire Food Responsiveness Scale; AEBQ-SR = Adult Eating Behavior Questionnaire Satiety Responsiveness Scale; PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalized Anxiety Questionnaire-7; BE = binge eating; BMI = body mass index

*Denotes statistically significant loading or correlation ($p \leq 0.01$)

Table 1.

Sample Characteristics

Demographics, *N* (%) unless stated otherwise*	**Full Sample (*N*=293)**	**Women (*N*=238)**	**Men (*N*=55)**
Age (years), Mean (SD)	46.6 (11.9)	46.8 (11.9)	45.9 (12.3)
Race/Ethnicity			
Latinx	59 (20.1%)	48 (20.2%)	11 (20.0%)
Non-Latinx, White	178 (60.8%)	146 (61.3%)	32 (58.2%)
Black	18 (6.1%)	15 (6.3%)	3 (5.5%)
Asian/Pacific Islander	22 (7.5%)	15 (6.3%)	7 (12.7%)
American Indian	3 (1.0%)	3 (1.3%)	0 (0.0%)
Multiracial**	13 (4.4%)	12 (5.0%)	1 (1.8%)
Unreported	17 (5.8%)	12 (5.0%)	5 (9.1%)
BMI (kg/m^2), Mean (SD)	34.5 (5.2)	34.5 (5.3)	34.9 (4.9)
Household Income			
<$50 000/year	56 (19.1%)	47 (19.7%)	9 (16.4%)
$50 000-$99 999/year	94 (32.1%)	83 (34.9%)	11 (20.0%)
>$100 000/year	118 (40.3%)	87 (36.6%)	31 (56.4%)
Prefer not to answer answer/Unreported	25 (8.5%)	21 (8.8%)	4 (7.3%)

Abbreviations: BMI = body mass index

*No significant sex differences observed between demographic categories (all *p*'s > 0.05).

**Note: race/ethnicity percentages add up to > 100% due to selection of multiple categories by some respondents.