CHILD SEXUAL ABUSE AND DISORDERED EATING: THE MEDIATING ROLE OF IMPULSIVE AND COMPULSIVE TENDENCIES

BY

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THESIS

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ABSTRACT

Empirical evidence suggests that the relationship between child sexual abuse (CSA) and disordered eating is complex and mediated by a number of factors. Indeed, it appears that child sexual abuse may lead to tendencies toward impulsivity and compulsivity, which may promote disordered eating behaviors. This mediated relationship was explored in regards to the severity of self-reported child sexual abuse and disordered eating behavior dimensions (i.e., binge eating, compensatory behaviors) in an undergraduate nonclinical sample of men and women. Structural equation modeling analyses revealed that impulsivity and compulsivity together accounted for the effect of child sexual abuse on binge eating (i.e., eating to excess) and compensatory behaviors (e.g., purging, laxative use) as assessed through the Minnesota Eating Behavior Survey. Further, in the context of this model, compulsivity was a stronger mediator than impulsivity of child abuse effects on binge eating, while both impulsivity and compulsivity were important in understanding compensatory behaviors. The present study provides much-needed empirical evidence to clarify theoretical accounts of predispositions to eating disorders. The findings suggest that CSA may predispose individuals to behavioral control problems in multiple ways, which may manifest differentially in symptoms of eating disorders.

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CHAPTER 1: INTRODUCTION

Survivors of child sexual abuse (CSA) are at an increased risk for a range of psychopathology (see Chen et al., 2010 and Putnam, 2003 for reviews), including disordered eating (Jarvis & Copeland, 1997), as well as predisposing personality traits like tendencies toward impulsivity and compulsivity (Caspi et al., 2008; Roy, 2005). Calls have been made to identify mediators of the relationship between CSA and disordered eating to identify potential mechanisms by which environmental adversity leads to psychopathology (Waller et al., 2001). There is evidence that impulsivity and compulsivity may mediate the relationship between CSA and disordered eating, although no empirical research has explored both variables in a single model. Another gap in the literature involves the fact that relationships between impulsivity and compulsivity and disordered eating are obscured by reliance on eating disorder diagnoses rather than distinct behavior dimensions such as binge eating and compensatory behaviors (Vitousek & Manke, 1994). Indeed, theory and empirical evidence suggest that impulsivity and compulsivity may be differentially related to disordered eating dimensions that are observed across eating disorder diagnoses (e.g., compensatory behaviors are similar to compulsivity in that they are typically used to compensate for negative affect and/or obsessive thoughts; binge eating is similar to impulsivity in that it often happens without premeditation (Altman & Shankman, 2009; Fahy & Eisler, 1993). To this end, the current study examines impulsivity and compulsivity as potential mediators of the relationship between CSA and distinct disordered eating behavior dimensions in a large nonclinical sample of young men and women.

1.1. Child Sexual Abuse and Disordered Eating

CSA appears to be a nonspecific risk factor in the etiology of at least some types of eating disorders (Fallon & Wonderlich, 1997; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). Meta-analyses have suggested that the prevalence of disordered eating in survivors of

CSA is high (see Chen et al., 2010 and Smolak & Murnen, 2002 for meta-analyses), and reviews of the literature indicate that self-reported CSA is a risk factor for both anorexia nervosa and bulimia nervosa (Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). In addition, the relationship of CSA to disordered eating may not be direct but instead indirect. That is, CSA may work in concert with a number of mediators to increase the risk for eating disorders.

1.2. Impulsivity and Compulsivity: Coping with Child Sexual Abuse

Impulsivity and compulsivity are two distinct but related tendencies toward behavioral dyscontrol that may be thought of as potential mediators of the CSA-disordered eating relationship. Both impulsivity and compulsivity refer to the presence of cognitive dyscontrol, which may manifest in uncontrollable and often disruptive or maladaptive behaviors. More specifically, impulsivity is characterized by the presence of deficient cognitive control, which may lead to disinhibition in behavior (e.g., eating on the spur of the moment; Skodol & Oldham, 1996, p. 3). In contrast, compulsivity is associated with a tendency to overcontrol and/or ruminate about behavior, which may lead to an initial resistance to engaging in particular behaviors, and ultimately behaviors that are harm avoidant and/or repetitive (e.g., exercising for many hours daily).

Correlational evidence has indicated that impulsivity and compulsivity both exist at higher prevalence and severity levels in survivors of self-reported CSA (Carter, Bewell, Blackmore, & Woodside, 2006; Caspi et al., 2008; Favaro et al., 2005; R. Lockwood, Lawson, & Waller, 2004). These findings suggest that CSA may impact behavioral control (Favaro et al., 2005; Shields, Cicchetti, & Ryan, 1994), either in the form of impulsivity or compulsivity, or both. Impulsivity and compulsivity may both be undertaken to cope with CSA, either in the pursuit of pleasure or to reduce intense negative affect (Cloninger, 1996; Cyders & Smith, 2008;

Hollander & Benzaquen, 1997; Sprague & Verona, 2010); indeed, they are thought to differ in terms of the typical motivations for their behavioral outcomes. Harmful consequences of behavior may be underestimated and risk-taking increased in impulsivity (Hollander, 1998; Hollander & Cohen, 1996, p. 268). In compulsivity, consequences of behavior are likely to be overestimated and risk-aversiveness increased, which in turn can lead to repetitive, uncontrollable behaviors thought to reduce risk.

While impulsivity and compulsivity appear to be distinct on a number of dimensions, they are not diametrically opposed. Rather, they exist on intersecting dimensions; for example, many eating disorders can be characterized by both compulsivity and impulsivity, either simultaneously or at different points in time (Hollander & Wong, 1995; Skodol & Oldham, 1996). For example, binge eating could be conducted without forethought or control, or after a great deal of rumination and planning (Raymond et al., 1999). While both impulsivity and compulsivity likely play a role in promoting disordered eating behaviors, there have been no systematic explorations of whether tendencies toward impulsivity or compulsivity are more important in promoting disordered eating behavior dimensions (e.g., binge eating, compensatory behaviors) after CSA.

1.3. Impulsivity and Compulsivity as Mediators of Disordered Eating Behavior Dimensions

Given that the trauma of CSA is body-directed, it makes intuitive sense that impulsivity and compulsivity after CSA would result in under or over control of the body (i.e., disordered eating). Indeed, research evidence has confirmed relationships between impulsivity/compulsivity and disordered eating (e.g., Claes, Vandereycken, & Vertommen, 2002; Fernández-Aranda et al., 2008), although very few formal tests have been conducted of a model in which the former serve as mediators of the CSA-disordered eating relationship. The few studies that have statistically

tested mediation lend preliminary support to a mediated model involving impulsivity. In a study that compared female children receiving treatment for CSA with age-matched controls, Wonderlich and colleagues (Wonderlich et al., 2001) reported that behavioral impulsivity was one of several mediators in the relationship between self-reported CSA and compensatory behaviors. Favaro and colleagues found that self-reported child abuse (physical and sexual) predicted impulsivity, and, in turn, impulsivity was associated with higher levels of disordered eating in a sample of patients with eating disorders (Favaro et al., 2005). While a recent study of Korean patients with eating disorders found that obsessive-compulsive traits did not mediate the relationship between CSA and eating pathology, this study was limited in that it focused primarily on cognitive aspects of eating disorders (e.g., drive for thinness), rather than behavioral components (e.g., binge eating; Kong & Bernstein, 2009). Given that impulsivity and compulsivity overlap in terms of dyscontrol of behavior, but appear to be distinct in the manifestations of the dyscontrol, the first goal of the current study was to determine if both impulsivity and compulsivity mediate the CSA-disordered eating relationship. As well, in models that account for the shared variance between impulsive and compulsive tendencies, we hoped to explore the unique contributions of impulsivity and compulsivity to the mediation of CSA effects on disordered eating dimensions.

1.4. Distinct Disordered Eating Behavior Dimensions

While the relationship between CSA and disordered eating and the relationships between impulsivity/compulsivity and disordered eating have been extensively studied, the findings of these studies are often inconsistent (e.g., Fontenelle et al., 2002; Galanti, Gluck, & Geliebter, 2007; Raymond et al., 1999), potentially because of limitations in the operationalization of disordered eating. Specifically, findings of no relationship between CSA and eating disorders

may be, in part, due to a lack of examination of this relationship at the level of symptoms or behaviors present in disordered eating (Smolak & Murnen, 2002). While differences in impulsivity/compulsivity have not been consistently identified across diagnoses, several key eating disorder diagnoses are behaviorally similar (e.g., compensatory behaviors are present in both bulimia nervosa and anorexia nervosa), which may obscure distinct relationships with impulsivity versus compulsivity. Additionally, examining behavior dimensions rather than diagnoses permits further understanding of subclinical cases or individuals who do not fit into any specific diagnosis (e.g., not-otherwise-specified), which may be important given these cases may still be highly distressing (Bunnell, Shenker, Nussbaum, Jacobson, & Cooper, 1990). In that regard, college students are an appropriate population to examine, given the high rates of disordered eating reported in undergraduate samples (Zivin, Eisenberg, Gollust, & Golberstein, 2009) but that often do not rise to the attention of service providers. Therefore, the second goal of this study was to clarify the relationship of impulsivity/compulsivity and CSA to disordered eating by examining disordered eating at the level of individual dimensions of behavior.

At least two dimensions of disordered eating behavior (i.e., binge eating and compensatory behavior) are known to characterize eating disorders. Binge eating is characterized by the consumption of large amounts of food in a short period of time accompanied by a feeling of a lack of control, and compensatory behavior includes a number of methods to control weight such as vomiting, restricting, using diet pills and laxatives, and exercising (APA, 2000). While a large body of research has identified both impulsive (Favaro et al., 2005; Waxman, 2009) and compulsive (Cassin & von Ranson, 2005; Halmi et al., 2003; 2005) tendencies in eating disorder diagnoses that include both binge eating and compensatory behaviors (i.e., anorexia nervosa-

binge/purge type, bulimia nervosa-purging type, bulimia nervosa-nonpurging type), less research has examined disordered eating on a behavioral level.

The limited body of research on eating disorder dimensions indicates that binge eating involves tendencies toward impulsivity but provides less clear support for a relationship with compulsivity. Empirically, binge eating is often accompanied by a failure to consider the consequences of behavior before acting, which is also characteristic of impulsivity (Fahy & Eisler, 1993). Indeed, a meta-analysis identified that severe eating disorders characterized by binge eating are highly comorbid with borderline personality disorder (Sansone, Gaither, Songer, & Allen, 2005), which is typically characterized by impulsivity. Research that has examined impulsive tendencies through self-report has indicated that impulsivity is higher in binge eaters than non-binge eaters (Galanti et al., 2007), and that experimentally priming impulsivity can lead to overeating (Guerrieri, Nederkoorn, Schrooten, Martijn, & Jansen, 2009; Jansen et al., 2008). Impulsive behaviors also predict binge eating prospectively (Wonderlich, Connolly, & Stice, 2004).

However, theory and clinical anecdotal evidence provides important reasons to consider the role of compulsivity in binge eating. Binge eating and compulsions are typically characterized by intrusive, ruminative urges to engage in the behavior and a buildup of tension that is released through the act of binging (Raymond et al., 1999). Studies are mixed in terms of uncovering this relationship empirically, with some studies finding no relationship with compulsivity among obese individuals with eating disorders (Fontenelle et al., 2002; Galanti et al., 2007), while a second study found evidence of both self-reported impulsive and compulsive traits (measured using an inventory of obsessive-compulsive behaviors) in obese individuals who binge ate (Raymond et al., 1999). Further studies are needed in larger samples and in young

adults with higher base rates of different forms of disordered eating behaviors, such as college students.

While there is little empirical evidence on the relationship of compensatory behaviors, considered broadly, to impulsivity and compulsivity, compensatory behaviors have been characterized primarily by compulsivity. Compensatory behaviors share phenomenological similarities with compulsions: they are typically used to compensate for negative affect (e.g., distress following a binge episode) and/or obsessive thoughts (e.g., preoccupation with body shape; Altman & Shankman, 2009). Indeed, compensatory behaviors are associated with perfectionistic personality traits in women (Forbush, Heatherton, & Keel, 2007). This finding has been supported by a meta-analysis which identified a high comorbidity of obsessive-compulsive personality disorder and anorexia nervosa-restricting type, characterized by compensatory behaviors in the absence of binge eating (Sansone et al., 2005). It is less conceptually clear how compensatory behaviors relate to impulsivity, although tendencies to engage in behavior without thinking would promote at least some compensatory behaviors like purging. In fact, impulsive behaviors have been found to predict the presence of compensatory behaviors prospectively (Wonderlich, Connolly, & Stice, 2004). Given findings of comorbidity of compensatory behaviors with a range of impulse control disorders representing both impulsivity and compulsivity (Fernández-Aranda et al., 2008), it is possible that both types of behavior dysregulation are important for compensatory behaviors. However, a lack of research with direct comparisons of the relative importance of impulsivity and compulsivity in different disordered eating behavior dimensions limits our ability to speculate regarding specificity in these relationships.

1.5. The Current Study

A conceptual challenge in the extant literature is the paucity of research that details *how* CSA may contribute to eating pathology. While impulsivity/compulsivity and disordered eating have been typically conceptualized as outcomes of CSA, there is evidence to suggest that a mediated relationship is warranted. Both CSA and disordered eating have been related to impulsivity and compulsivity, and research has suggested that the relationship between CSA and disordered eating is marked by complex pathways and likely accounted for by mediators rather than having a direct effect (Smolak & Murnen, 2002). However, very little research has tested whether tendencies toward impulsivity and/or compulsivity serve as mediators of the CSA-disordered eating relationship in a large sample of men and women. Further, since compulsivity has not been included in previous models, including compulsivity along with impulsivity in a multiple mediation model has the potential to account for previously unexplained variance and provide a more holistic conceptualization of the relationship of CSA to disordered eating. Therefore, the first goal of the current study was to empirically test a model in which impulsivity and compulsivity mediate the relationship of CSA to binge eating and compensatory behaviors. We hypothesize that a mediated model will be supported through formal methods of testing indirect effects (Preacher & Hayes, 2008).

The extant literature has identified relationships between key constructs, but the relative strengths of multivariate relationships have not been compared. Therefore, the second goal was to explore specificity in the mediated relationship between CSA and binge eating and compensatory behaviors. While it is difficult to predict the relative strength of mediated relationships, given the paucity of research directly comparing relationships explored in this model, preliminary hypotheses based on relevant theory and data include that: 1) the relationship

of CSA to binge eating through impulsivity will be stronger than the relationship of CSA to compensatory behaviors through compulsivity and 2) the relationship of CSA to compensatory behaviors through compulsivity will be stronger than the relationship of CSA to compensatory behaviors through impulsivity.

CHAPTER 2: METHODS

2.1. Participants

Seven hundred and eight undergraduate students completed a self-administered online questionnaire as part of their participation in an introductory psychology class. Six participants completed less than a third of the study measures and were excluded from further analyses, leaving a sample size of N = 702. About a third of participants who indicated their gender were male (n = 234), and 66% (n = 455) were female. Participants ranged in age from 18 to 30 years, and the average age was 19.3 years (*SD* = 1.43 years). The majority of participants who identified their race/ethnicity said that they were white (83.8%), while 16.2% identified as other races/ethnicities (6.3% Black/African American, 1.0% Latino/a/Hispanic, 0.6% American Indian/Native American, 5.5% Asian/Pacific Islander, 1.0% Middle Eastern, 1.6% multiracial). Participants signed an informed consent form that was approved by the university institutional review board.

2.2. Measures

2.2.1. Child sexual abuse severity. A fourteen-item measure assessing CSA severity asked participants to indicate whether they experienced a range of attempted or completed unwanted sexual experiences before age 14, and committed by a perpetrator at least five years older than the participant. Participants could answer either ―no" or ―yes" to each item. The CSA scale was adapted from several measures found to be reliable and valid (Ullman & Filipas, 2005; West, Williams, & Siegel, 2000). The reliability of the CSA scale in the current study was assessed at $\alpha = 0.86$ in the overall sample ($\alpha = .90$ for males, $\alpha = .86$ for females), indicating good reliability. From responses to this scale, an ordinal variable, *CSA severity*, was created to represent the most severe form of CSA experienced. Anchors were 0 = no abuse, 1 = exposure

only, 2 = attempted fondling, 3 = attempted penetration, 4 = completed fondling, and 5 = completed penetration. This is consistent with the method of ranking severity used by authors using a similar scale (Ullman & Filipas, 2005; Ullman, Filipas, Townsend, & Starzynski, 2005) and is based on guidelines established by Koss and colleagues (Koss, Gidycz, & Wisniewski, 1987).

2.2.2. Income. Income was assessed with a demographic item asking participants to indicate their family's annual household income. Anchors were 0 = Less than $30,000, 1 = $30,000-$65,000, 2 = $65,000-$100,000, 3 = $100,000-$200,000, 4 = Over $200,000, and 5 = Unknown. Family income was included as a proxy for socioeconomic status to address concerns voiced by some authors (e.g., Rind, Tromovitch, & Bauserman, 1998) that the apparent relationship between CSA and mental health outcomes can be accounted for by family environment variables.

2.2.3. Impulsive tendencies. Impulsive tendencies were assessed by summing responses from the 30-item self-report Barratt Impulsiveness Scale, version 11 (BIS-11; Patton, Stanford, & Barratt, 1995). This measure is assessed on a Likert scale ranging from 1 ("rarely/never") to 4 ("almost always/always"). Cronbach's α for the total score has been assessed at 0.83 (Stanford et al., 2009). There are three subscales in the BIS-11: motor impulsiveness (e.g., "I am self-controlled"), attentional impulsiveness (e.g., "I don't pay attention"), and non-planning impulsiveness (e.g., "I plan tasks carefully"). The total score was used for the purposes of this study to represent impulsivity unidimensionally. The overall reliability was assessed in the current study at $\alpha = 0.84$ ($\alpha = .85$ for females and $\alpha = .84$ for males), indicating good reliability.

2.2.4. Compulsive tendencies. In order to assess the presence of behavior patterns indicative of compulsive tendencies in participants, items indexing compulsivity were obtained

from the 18-item Obsessive-Compulsive Inventory (OCI-R; Foa et al., 2002). Items assessed how often each symptom has been distressing or bothersome to participants in the past month. These statements were assessed on a Likert scale ranging from 0 (~~"n~~ot at all") to 4 (~~"e~~xtremely"). Internal consistency of this scale has been assessed at between .81 and .93, and this scale has been used previously to measure compulsive traits in research on eating disorders (Foa et al., 2002; Wu, 2008).

The authors of the Obsessive-Compulsive Inventory- Revised suggest a six factor solution to this scale. Two of the subscales assess obsessional thoughts (i.e., obsessing, mental neutralizing), while the other four subscales assess compulsive tendencies. The latter include: *washing*, which assesses distress resulting from perceived contamination and/or a need to wash; *checking*, a subscale that measures how bothered participants are by a perceived need to check things; *ordering,* which measures the extent to which participants are bothered by a need for order, and *hoarding,* which contains questions assessing how distressed participants are by the accumulation of things that they have saved or failed to discard (Foa et al., 2002). Items from these four subscales were summed to create a variable representing compulsive tendencies. The internal consistency of this compulsivity score was assessed at $\alpha = 0.84$ ($\alpha = .82$ for females and $\alpha = .85$ for males) in the current sample, indicating good internal consistency.

2.2.5. Disordered eating behavior dimensions. The Minnesota Eating Behavior Survey contains 30 items used to assess disordered body perceptions and eating behaviors (MEBS; von Ranson, Klump, Iacono, & McGue, 2005). This measure asked the participant to rate various statements relating to disordered eating. The possible answers, rated on a 0 to 3 Likert scale, were ~~"d~~efinitely false," ~~"p~~robably false," ~~"p~~robably true," and ~~"d~~efinitely true." In previous research, this survey has been found to have internal consistency ranging from 0.71 to 0.85

across its subscales and moderate to high concurrent validity (von Ranson, Klump, Iacono, & McGue, 2005). The MEBS has two primarily cognitive/affective subscales: *body dissatisfaction* (6 items, "My stomach is too big") and *weight preoccupation* (8 items, "My weight is very important to me"), and two primarily behavioral subscales: *binge eating* (7 items, "Sometimes I stuff myself with food") and *compensatory behaviors* (6 items focusing on purging and restriction behaviors, "Sometimes I make myself throw up (vomit) to control my weight") (Klump, McGue, & Iacono, 2000; Marmorstein, von Ranson, Iacono, & Succop, 2007). Since the focus of this study was on behavioral components of eating disorders, we considered only the latter two subscales in analyses. The current study identified adequate internal consistencies: α = 0.85 for *binge eating* (α = .86 in females and α = .79 in males) and α = 0.82 for *compensatory behaviors* (α = .80 in females and α =.88 in males).

2.3. Analyses

Skew and kurtosis were calculated on model variables to assess the assumption of normality. The variables representing severity of CSA and compensatory behaviors were found to be highly skewed and kurtotic, justifying the calculation of normalized variables using Blom's transformation (Blom, 1958).

Structural equation modeling with Mplus 6 (Muthén & Muthén, 1998-2009) was used to test conditional indirect effects. Cases were excluded listwise, leaving a sample size of N = 539 for these analyses. Given that the sample size in the current study was relatively large, model fit was assessed through fit indices beyond the chi-square statistic (which is commonly inflated to statistical significance in larger samples, potentially incorrectly suggesting model misfit; Kline, 1998), using the 2-index presentation strategy suggested by Hu and Bentler, the standardized root-mean-square residual (SRMR) supplemented by one of a number of additional fit indices

recommended by the authors (Hu & Bentler, 1998), specifically the comparative fit index (CFI). Hu and Bentler propose that values above .95 for the CFI and values below 0.08 for the SRMR indicate acceptable model fit. We also chose to use the more conservative *p* value cutoff of .01 to evaluate significance of parameter estimates given the large sample size. We compared two key models using these approaches. First, we evaluated a direct effects model, containing only direct effects from CSA to the mediators and eating behavior dimensions, with effects from mediators to outcomes constrained to zero (see Figure 1a). The hypothesized model, or indirect effects model, contained only the indirect paths from CSA to mediators to the eating behavior dimensions (see Figure 1b).

Rather than following the commonly-used causal step procedures to test mediation (Baron and Kenny, 1986), on the suggestion of MacKinnon and others (Mackinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008; Preacher, Rucker, & Hayes, 2007), we attempted to address the potentially problematic assumption that the sampling distribution of the indirect effects are normal by bootstrapping 5000 samples to create a bias-corrected 99% confidence interval around the indirect effects. Using maximum likelihood method (ML) estimation, we chose to examine 99% confidence intervals as a way to be more conservative given the large sample size and number of analyses conducted. When testing the presence of mediation, a finding that the confidence interval does not include zero would suggest the presence of a significant indirect effect. Such a finding would indicate with 99% confidence that zero is an unlikely a value for the indirect effect of CSA on the disordered eating behavior outcome. This approach has been found to increase power over other methods of testing indirect effects, while controlling Type I error (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Additionally, the bootstrapping approach has been suggested to be preferable over the causal

steps approach of Baron and Kenny (1986) when testing multiple mediation (Preacher & A. F. Hayes, 2008).

The bootstrapping approach was also used to test contrasting hypotheses regarding the mechanisms driving different disordered eating dimensions, using pairwise contrast syntax provided by Preacher and Hayes (2008). That is, we compared the magnitude of direct and indirect effects to each other (e.g., indirect effect through impulsivity vs. compulsivity). In this case, if zero was contained in a confidence interval, this indicated that the two effects were not statistically different in magnitude.

To address concerns voiced by some authors (e.g., Rind, Tromovitch, & Bauserman, 1998) that the apparent relationship between CSA and mental health outcomes can be accounted for by family environment variables, family income was used as a covariate on CSA. Second, given that a number of the focal variables tend to differ by gender (e.g., disordered eating), we tested whether model parameters in the hypothesized indirect effects model differed significantly by gender. By computing a chi-square difference test on the chi square values obtained by constraining all parameter estimates across gender and then allowing model parameters to vary by gender, we found no difference between gender-invariant ($\chi^2(14, N = 539) = 39.94$, $p = .001$; CFI = 0.95, SRMR = 0.05) and variant ($\chi^2(14, N = 539) = 39.08$, $p = .01$; CFI = 0.96, SRMR = 0.06), models ($\Delta\chi^2(7, N = 539) = 2.82$, *ns*). These findings indicated that the focal model did not vary by gender; thus, we collapsed across gender for future analyses.

CHAPTER 3: RESULTS

3.1. Correlations

Bivariate correlations between all focal variables (CSA severity, impulsivity, compulsivity, disordered eating behavior dimensions) are presented in Table 1. With the exception of CSA and impulsivity, all focal variables were significantly correlated with each other at $p < .01$. Generally, the correlations ranged widely, from small to moderate (rs = .08 to .64).

3.2. Descriptive Statistics

Descriptive statistics of focal variables for the total sample and within gender, along with tests of gender differences, can be found in Table 2.

3.2.1. Child sexual abuse. Overall, 15.8% of the sample reported experiencing some form of attempted or completed CSA. There were no gender differences in the prevalence of CSA (12.9% of males, 18.7% of females; $\chi^2(1, N = 650) = 3.49$, $p = .06$) or the severity of CSA ($t(507.20) = -1.94$, $p = .05$). Relative to other studies using a similar measure in a similar population, the frequencies we identified are comparable for men and the overall sample, although the females in our sample appeared to report less CSA than females in past research (e.g., 22.8% of the sample, 13.3% of males, 28.2% of females; Ullman & Filipas, 2005; Ullman, Filipas, Townsend, & Starzynski, 2005). The prevalence of the most severe form of CSA, completed penetration, was very similar to prevalence estimates using a similar measure in a similar population (Ullman & Filipas, 2005). However, among those experiencing CSA, we found a higher prevalence of attempted penetration than has been reported previously (26.1% of those reporting CSA in the current study vs. 4.3% in Ullman & Filipas, 2005).

3.2.2. Disordered eating behavior dimensions. Descriptive statistics for the MEBS can be found in Table 2. As expected, women had significantly higher scores than men on the total scale ($t(498.68) = -9.34$, $p < .001$), *binge eating* ($t(574.71) = -6.35$, $p < .001$), and *compensatory behaviors* ($t(543.53) = -5.27$, $p < .001$). While published norms are not available for mixed-gender or male-only university samples, norms for university women using a true/false response format (von Ranson, Cassin, Bramfield, & Fung, 2007) indicate that, when responses from the current study were recoded to match this format, the women in this sample scored higher overall ($M = 12.07$ (6.26) in the current study vs. $M = 9.85$ (6.35)), comparably for binge eating ($M = 12.07$ (6.26) in the current study vs. $M = 9.85$ (6.35)), and higher for compensatory behaviors ($M = 0.92$ (1.31) in the current study vs. $M = 0.52$ (0.88)).

3.3. Testing Mediation

To address the first goal of testing whether impulsivity and compulsivity mediate the effects of CSA on disordered eating behavior dimensions, we first compared a direct effects model above (Figure 1a: paths from the mediators to behavior dimensions constrained to zero; see Figure 2a for standardized path estimates) with an indirect effects model (Figure 1b: where these paths were free to vary; see Figure 2b for standardized path estimates). This test explores whether a mediated model is a better fit to the data than one in which CSA accounts for impulsivity/compulsivity and eating behavior dimensions. Fit indices of the direct effects model indicated poor fit ($\chi^2(9, N = 539) = 119.16$, $p < .001$; CFI = 0.74, SRMR = 0.11), suggesting that a model in which paths from impulsivity and compulsivity to disordered eating behavior dimensions are constrained to zero is not supported. In contrast, the indirect effects model appeared to be a good fit for the data ($\chi^2(7, N = 539) = 25.58$, $p < .001$; CFI = 0.96, SRMR = 0.05), and chi-square difference test confirmed that the two models were significantly different

($\Delta\chi^2(2)= 91.75$). This indicates that a model in which impulsivity and compulsivity mediate the relationship from CSA to disordered eating behaviors is a better fit to the data than a model in which impulsivity/compulsivity and disordered eating behaviors are outcomes of CSA[1].

Given that we established that the indirect effects model was an improvement over the direct effects model, we next wanted to test the presence of mediation formally by bootstrapping confidence intervals around the indirect effects (see Table 3 for specific and total indirect effects and confidence intervals). First, we considered total indirect effects (i.e., the total effect from CSA on each disordered eating behavior dimension through both impulsivity and compulsivity). The total indirect effects were significant for both binge eating ($\beta = .08$, $p < .001$, 99% CI [0.03, 0.14]) and compensatory behaviors ($\beta = .08$, $p < .001$, 99% CI [0.02, 0.14]), suggesting that the combination of impulsivity and compulsivity is important in explaining the effect of CSA on each disordered eating behavior dimension.

3.4. Examining Specificity for Disordered Eating Behavior Dimensions

To address the second goal of exploring specificity in the mediated relationship between CSA and binge eating and compensatory behaviors, we analyzed whether indirect paths to binge eating vs. compensatory behaviors differed in their regression weights (see Table 4 for detailed findings). When comparing the specific indirect effects of compulsivity on the two behavior dimensions, analyses revealed that the relationship of CSA to binge eating through compulsivity was stronger than the relationship of CSA to compensatory behaviors through compulsivity (difference in indirect effect, $Z = 3.68$, $p < .001$). In contrast, the indirect path of CSA to binge

[1] We tested the interaction of impulsivity and compulsivity in the context of a regression model predicting binge eating and compensatory behaviors, but only the main effects significantly predicted the disordered eating behaviors. Thus, we did not include an interaction term in structural models.

eating through impulsivity was not significantly different from the relationship of CSA to compensatory behaviors through impulsivity ($Z = 1.91$, $p = .056$). Thus, compulsivity accounted for the CSA relationships to binge eating more than to compensatory behaviors, although impulsivity effects were similar across both eating behavior dimensions.

Next, we explored the relative importance of impulsivity versus compulsivity for each outcome variable, in turn. Analyses revealed a differential impact of the mediators on binge eating, but a similar impact of each mediator on compensatory behaviors. That is, the path from CSA to binge eating through compulsivity was significantly stronger than the path through impulsivity ($Z = -2.79$, $p < .01$), but there was no difference in the specific indirect paths to compensatory behaviors ($Z = -2.22$, $p = .03$). Notably, there was no difference in the relationship of CSA to impulsivity or compulsivity ($Z = -1.29$, $p = .10$); thus, the greater mediating role of compulsivity relative to impulsivity for binge eating cannot be accounted for by their individual relationships to CSA alone. Thus, in our sample, compulsive vs. impulsive tendencies more strongly accounted for CSA relationships to binge eating but not compensatory behaviors.

We conducted post-hoc bootstrapped contrasts to examine whether specificity existed in the model after separating compensatory behaviors subscale of the MEBS into items representing purging (4 items) and items representing restricting (2 items) and using these behaviors as outcome variables, given their theoretically-distinct associations with impulsivity vs. compulsivity. We found no differences for relative importance of impulsivity and compulsivity in relation to purging and restricting. That is, there were no differences in the specific indirect paths through impulsivity ($Z = 1.25$, $p = .21$) or compulsivity ($Z = 0.49$, $p = .62$), or the indirect paths to purging ($Z = -1.87$, $p = .06$) or restricting ($Z = -2.12$, $p = .03$), Thus, our main results

cannot be accounted for by the inclusion of purging and restricting in the compensatory behaviors subscale.

CHAPTER 4: DISCUSSION

This study was the first to our knowledge to model the relationship between retrospectively reported CSA, tendencies toward impulsivity/compulsivity, and disordered eating behavior dimensions holistically in a multivariate context. Through this approach, we were able to compare the relative importance of impulsivity and compulsivity in accounting for the relationship between CSA and distinct disordered eating behaviors. In particular, the current study findings validate the extant literature that has identified that both impulsivity and compulsivity play an important role in the relationship of CSA to disordered eating behavior dimensions. However, previous studies have failed to incorporate both impulsivity and compulsivity to clarify their unique roles. The present findings suggest that identifying and targeting both impulsive and compulsive tendencies may be important in certain individuals.

Notably, this study expands the current understanding of disordered eating by identifying that compulsive tendencies appear to play a larger role than impulsivity in behaviors typically thought of as impulsive, such as binge eating. Since the current study focused on disordered eating behaviors rather than diagnoses in a nonclinical sample, the study findings may have clinical implications in a wide range of people struggling with disordered eating behaviors, particularly subclinical individuals, individuals with a primary diagnosis other than an eating disorder who display disordered eating behaviors, and patients who are diagnosed with eating disorder not otherwise specified.

4.1. The Dual Importance of Impulsive and Compulsive Tendencies in Disordered Eating

A major contribution of the current study is the finding that both impulsive and compulsive tendencies are important in explaining the effect of CSA on disordered eating behavior dimensions. We established that this effect is best explained through a model in which

impulsive and compulsive tendencies mediate the relationship between CSA and disordered eating behavior dimensions, rather than a direct-effects model. When we examined the mediated model, the total indirect effects were significant for both binge eating and compensatory behaviors.

These findings indicate that the effect of CSA on disordered eating behavior dimensions appears to be ~~–e~~arried" by general dysregulation in behavioral control and provide support for the argument that the same behavior may be impulsive or compulsive across individuals and time. Indeed, this study found that compensatory behaviors, as well as purging and restriction independently, appear to be equally related to impulsive and compulsive tendencies. This study thus replicates previous literature that has identified a role for both impulsive and compulsive tendencies in disordered eating (Fernandez-Aranda et al., 2008; Raymond et al., 1999), while expanding on this literature by examining this relationship in a holistic model. While other authors have suggested that the coexistence of behavioral impulsivity and self-reported restraint in anorexia nervosa may suggest of a lack of self-awareness in the participant's reports, as these constructs appear to be contradictory (Butler & Montgomery, 2005), the current study provides new evidence that indeed tendencies toward both impulsivity and compulsivity may play a role in the same eating behaviors.

Evidence of the coexistence of impulsive and compulsive tendencies in disordered eating has a number of clinical implications. Namely, it suggests that coupling clinical approaches found to be effective in treating impulsivity (e.g., Dialectical Behavior Therapy; Robins & Chapman, 2004) with those effective in treating compulsivity (e.g., exposure and response prevention; Butler, Chapman, Forman, & Beck, 2006) may be most effective in preventing and treating disordered eating. The exposure-based cognitive therapy for depression described by

Kumar and colleagues (Kumar, Feldman, & A. Hayes, 2008) may be one such promising treatment, as it combines mindfulness training with cognitive behavioral therapy, although its effectiveness in treating disordered eating behaviors has not been examined.

4.2. The Unique Importance of Compulsive Tendencies

While the current study identified both impulsive and compulsive tendencies as important, our findings highlighted the unique (and largely unexamined) role of compulsive tendencies in explaining variance in disordered eating behavior dimensions. When considering the indirect effects through each mediator individually, we identified that compulsivity appears to play a larger role in binge eating than impulsivity, while neither impulsivity nor compulsivity was more important for compensatory behaviors. These findings are surprising, given that we expected binge eating to relate to impulsive tendencies and compensatory behaviors to relate to more strongly to compulsive tendencies. However, it makes conceptual sense that binge eating would be related more strongly to compulsive tendencies—indeed, colloquially binge eating is often referred to as "compulsive eating." Like compulsions, binges may be characterized by intrusive, ruminative urges and a buildup of tension preceding a binge episode (Raymond et al., 1999). Empirical evidence has shown that the use of exposure and response prevention of binge episodes is associated with a faster recovery over the use of exposure and response prevention of purge episodes or cognitive behavioral therapy alone (Carter, McIntosh, Joyce, Sullivan, & Bulik, 2003).

There are several reasons that this finding may have emerged. First, compulsivity may play a heightened role in pathological eating among high-functioning participants, including the college students who participated in this study. That is, compulsivity is strongly linked to traits such as perfectionism, and such traits may make students more competitive college applicants

and potentially facilitate behaviors (e.g., studying) that may make them less likely to drop out (and thus, participate in our study). However, compulsive and perfectionistic traits can simultaneously facilitate maladaptive behaviors (e.g., binge eating) that do not necessarily interfere with their studies. In contrast, impulsive tendencies might be a barrier to reaching or engaging in higher education. Later research should address this possibility by testing this model in community samples or in those with lower educational attainment.

Second, compulsive tendencies may play a unique role in survivors of sexual abuse. The unpredictability of control over one's body involved in experiencing CSA may result in behavior regulation that is primarily overcontrolled in order to avoid future abuse (as in compulsivity), and less commonly, undercontrolled (as in impulsivity), resulting in several manifestations of disordered eating. Given that the trauma of CSA is specifically bodily-directed, it is not surprising that this symptomatology takes the form of over-control of body weight and eating habits. Third, the relationship between compulsivity and binge eating may itself be indirect. That is, compulsive tendencies may indirectly promote binge eating through the promotion of body dissatisfaction, and subsequent dietary constraint may increase risk for binge eating (Stice, Davis, Miller, & Marti, 2008). Longitudinal research is needed to verify the temporal sequence of events, cognitions and behaviors that leads to disordered eating. Finally, these findings may be artifacts of differences in the measures used to assess impulsive and compulsive tendencies, with the former being more strongly tied to obsessive-compulsive disorder. Future studies should use more similar measures to assess these constructs.

Although causal assumptions cannot be made, the current study findings suggest that clinical approaches targeting binge eating can incorporate treatment of compulsive tendencies more generally. Indeed, treatment outcome studies that manipulate to what extent compulsive

tendencies are addressed may provide compelling evidence for the causal impact of compulsivity on eating disorders. Techniques found to be effective in treating compulsivity, such as exposure and response prevention, may be particularly useful.

4.3. Limitations

There are several limitations present in the current study. By nature of their enrollment at a university and engagement with class requirements (i.e., participating in human subjects research), this sample is biased towards high-functioning individuals. Certain demographic characteristics, including young age, white race, and high socieconomic status, were overrepresented in the current sample as a result of the population sampled. However, it has been suggested that young adulthood is an ideal age at which to study problems with impulsivity and compulsivity, as these problems may decrease with age (Abramowitz & Berenbaum, 2007). Additionally, nonclinical samples may be useful to study, since treatment-seekers are not representative of the general population who struggle with behavioral dysregulation and eating disorders (Galbaud Du Fort, Newman, & Bland, 1993). Since the current study focused on disordered eating behaviors rather than diagnoses, the study findings may have clinical implications in a wide range of people struggling with disordered eating behaviors, particularly subclinical individuals, individuals with a primary diagnosis other than an eating disorder who display disordered eating behaviors, and patients who are diagnosed with eating disorder-not otherwise specified (EDNOS).

The measurement of the focal variables in this study had a number of limitations that temper interpretability and generalization of the study results. First, the operationalization of severity of sexual abuse by the degree of violation involved in the act does not account for other indicators of severity (e.g., duration, relationship to perpetrator). Indeed, there is some

suggestion that the chronicity of trauma has neurodevelopmental implications that may include impulsivity (Perry, 1997). However, operationalizing CSA according to degree of violation has been used in similar studies (e.g., Callahan, Price, & Hilsenroth, 2003; Senn, Carey, Vanable, Coury-Doniger, & Urban, 2007), and similar severity indicators have been associated with negative psychosocial outcomes (Senn et al., 2007). Second, the measurement of CSA in this study involved self-report and retrospective recall of CSA. Concerns have been raised regarding the validity of such data, particularly as it relates to CSA (Briere, 1992). While some studies have attempted to account for this using longitudinal designs with samples restricted to corroborated cases, this excludes the large proportion of victims who never reported their abuse. Self-report may be the best available option, particularly in light of findings that, while individuals who have been abused sometimes do not report their abuse in self-report measures, those who have not been sexually abused do not falsely report being abused, and false negatives appear to not affect estimates of post-abuse psychopathology (Fergusson, Horwood, & Woodward, 2000). Finally, the cross-sectional and correlational design limits our ability to draw causal conclusions. Although there is evidence that CSA typically occurs before the onset of eating disorders (Deep, Lilenfeld, Plotnicov, Pollice, & W H Kaye, 1999), we echo other calls (e.g., Wonderlich et al., 2001) for longitudinal research examining mediators between CSA and mental health outcomes, so that causal inferences may be drawn.

4.4. Conclusions

The results of the current study further explicate the relationship of CSA to impulsivity/compulsivity and disordered eating behavior dimensions. In particular, they highlight the importance of considering both impulsivity and compulsivity, and provide new information about the unique role of compulsivity in the relationship between CSA and binge eating.

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TABLES

Table 1

Bivariate Pearson Correlations between Focal Variables

Variable	1	2	3	4	5
1. CSA (normalized)	--				
2. Impulsivity	.08	--			
3. Compulsivity	.23*	.18*	--		
4. Binge Eating	.17*	.22*	.38*	--	
5. Compensatory Behaviors (normalized)	.15*	.27*	.33*	.64*	--

*$p < .01$

Table 2

Descriptive Statistics for Total Sample and Within Each Gender

	Total Sample (*N* = 708)	**Men (*n* = 234)**	**Women (*n* = 455)**	**Statistical Difference Tests**
	M(SD)	***M(SD)***	***M(SD)***	
CSA Scale	0.53(1.33)	0.39(1.15)	0.59(1.38)	$t(507.20) = -1.94$, $p = .05$
	Frequency (%)	***Frequency (%)***	***Frequency (%)***	
No CSA	543 (77.4%)	189 (80.8%)	352 (77.4%)	
Exposure	29 (4.1%)	8 (3.4%)	21 (4.6%)	
Attempted Fondling	12 (1.7%)	4 (1.7%)	8 (1.8%)	
Attempted Penetration	6 (0.9%)	2 (0.9%)	4 (0.9%)	
Completed Fondling	43 (6.1%)	8 (3.4%)	35 (7.7%)	
Completed Penetration	21 (3.0%)	6 (2.6%)	13 (2.9%)	
Any CSA	111 (15.8%)	28 (12.0%)	81 (17.8%)	$\chi^2(1, N = 650) = 3.49$, $p = .06$
	M(SD)	***M(SD)***	***M(SD)***	
BIS Total Score	62.97(9.94)	64.81(9.68)	62.04(10.01)	$t(619) = 3.29$, $p = .001$
Attentional Impulsiveness	16.62(3.65)	16.91(3.70)	16.49(3.64)	$t(669) = 1.43$, *ns*
Motor Impulsiveness	21.79(3.76)	22.57(3.64)	21.40(3.77)	$t(654) = 3.80$, $p < .001$
Non-planning Impulsiveness	24.59(4.81)	25.29(4.60)	24.18(4.89)	$t(661) = 2.83$, $p < .01$
Compulsivity	24.58(8.85)	25.11(9.33)	24.32(8.55)	$t(658) = 2.08$, *ns*
Washing	4.88(2.64)	5.22(2.88)	4.69(2.48)	$t(407.66) = 2.35$, $p = .02$
Hoarding	7.03(3.23)	7.01(3.15)	7.05(3.27)	$t(679) = -0.15$, *ns*
Ordering	6.76(3.20)	6.64(3.00)	6.82(3.30)	$t(503.93) = -0.71$, *ns*
Checking	5.99(2.70)	6.39(2.91)	5.78(2.55)	$t(416.05) = 2.71$, $p < .01$
MEBS Total Score	61.77 (16.09)	54.25 (13.12)	65.58 (16.10)	$t(498.68) = -9.34$, $p < .001$
Binge Eating	13.59 (4.83)	12.10 (3.85)	14.35 (5.11)	$t(574.71) = -6.35$, $p < .001$
Compensatory Behavior	8.47 (3.50)	7.54 (2.90)	8.90 (3.64)	$t(543.53) = -5.27$, $p < .001$

Note: BIS = Barratt Impulsiveness Scale; MEBS = Minnesota Eating Behavior Survey

Table 3
Specific and Total Indirect Effects

	Standardized Estimate	SE	*Z*	Bias corrected 99% CI	
				Lower	Upper
Binge Eating					
Impulsivity	0.02	0.01	2.00	0.00	0.04
Compulsivity	0.07	0.02	3.65*	0.02	0.11
Total	0.08	0.02	3.92*	0.03	0.14
Compensatory Behaviors					
Impulsivity	0.02	0.01	2.17	0.00	0.04
Compulsivity	0.06	0.02	3.33*	0.01	0.11
Total	0.08	0.02	3.64*	0.02	0.14

* $p < .01$

Table 4

Hypothesis Testing of Differences Between Paths

H_0	Unstandardized Estimate	SE	*Z*	Bias corrected 99% CI Lower	Upper
CSA → IMP = CSA → COMP	-1.29	0.78	-1.65	-3.25	0.83
IMP → BE = COMP → BE	-0.10	0.04	-2.89*	-0.20	-0.02
IMP → CB = COMP → CB	0.01	0.01	1.68	-0.01	0.03
IMP → BE = IMP → CB	0.06	0.02	3.33*	0.01	0.11
COMP → BE = COMP → CB	0.15	0.02	7.02*	-0.21	-0.10
CSA → IMP → BE = CSA → IMP → CB	0.09	0.05	1.91	0.00	0.26
CSA → COMP → BE = CSA → COMP → CB	0.42	0.11	3.68*	0.16	0.79
CSA → IMP → BE = CSA → COMP → BE	-0.38	0.14	-2.79*	-0.81	-0.06
CSA → IMP → CB = CSA → COMP → CB	-0.05	0.02	-2.22	0.01	0.12

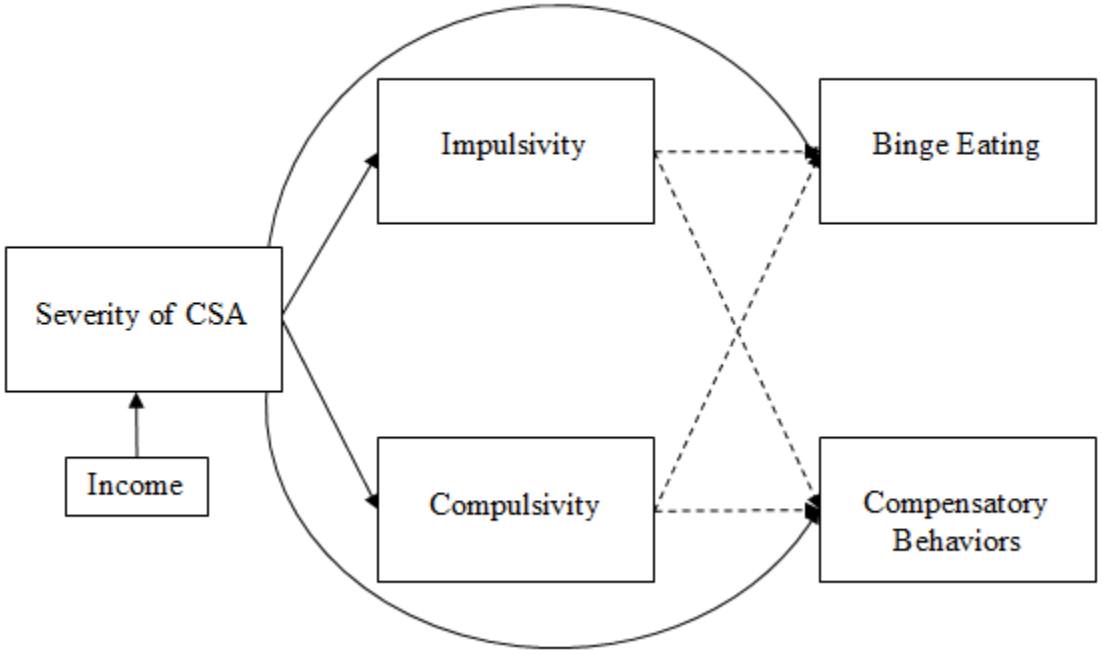
IMP: Impulsivity, COMP: Compulsivity, BE: Binge Eating, CB: Compensatory Behaviors

*$p < .01$

FIGURES

Figure 1

1.1. Direct Effects Model



1.2. Indirect Effects Model

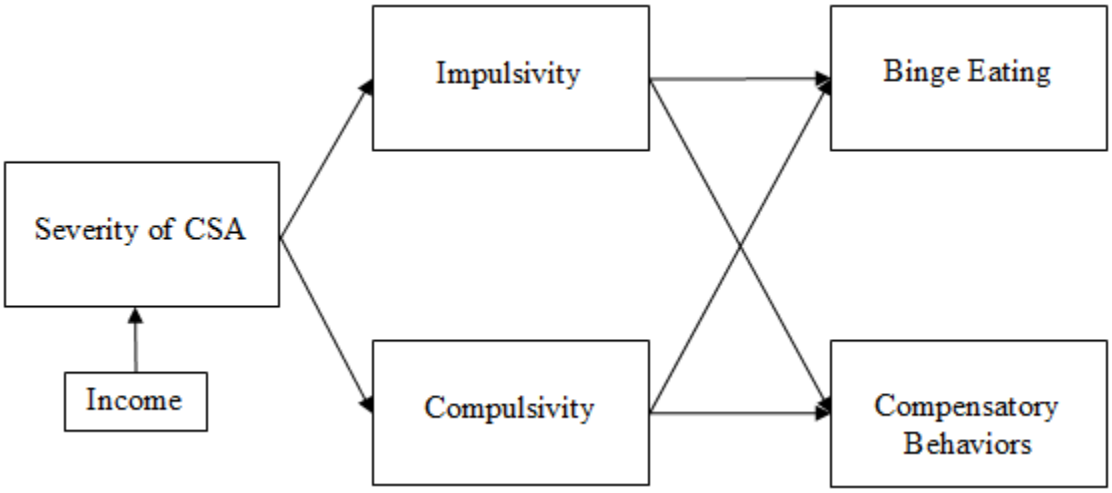
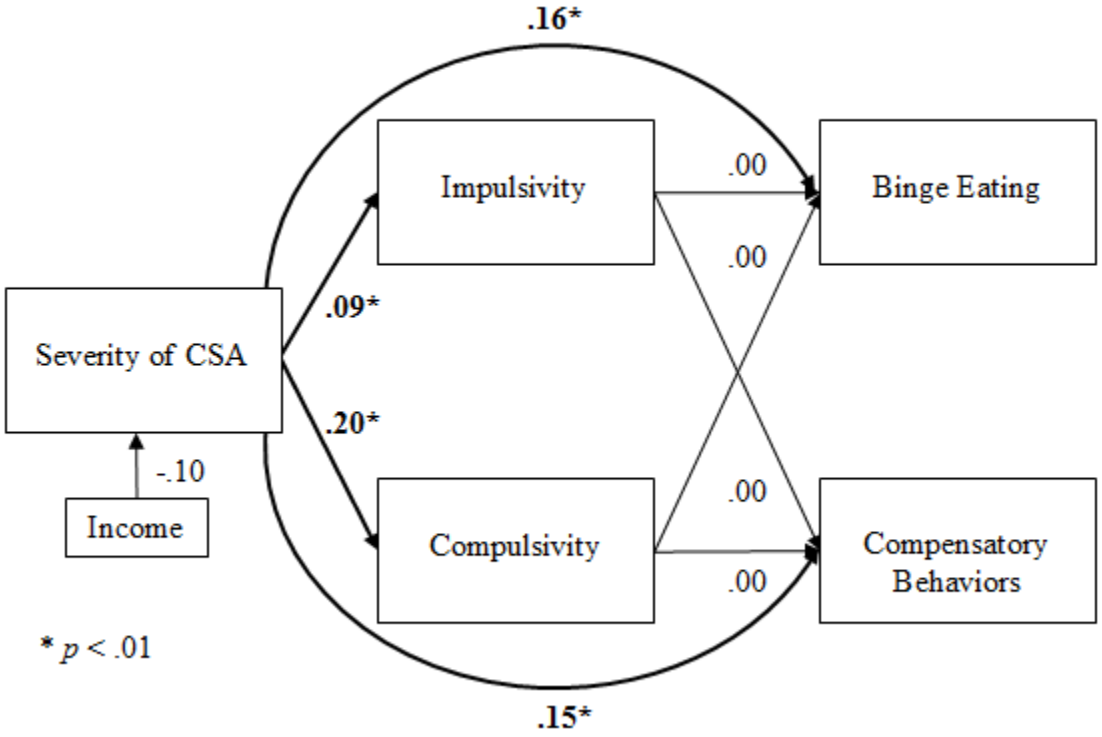


Figure 2

2.1. Results: Direct Effects Model



2.2. Results: Indirect Effects Model