January 2019

Unpacking Childhood Sexual Abuse And Eating Psychopathology Over Time: Impulsive Mechanisms And Deleterious Outcomes

Nicole M. Della Longa

UNPACKING CHILDHOOD SEXUAL ABUSE AND EATING PSYCHOPATHOLOGY OVER TIME: IMPULSIVE MECHANISMS AND DELETERIOUS OUTCOMES

by

Nicole Marie Della Longa
Bachelor of Science, Texas A&M University, 2013
Master of Arts, University of North Dakota, 2016

A Dissertation

Submitted to the Graduate Faculty

of the

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Grand Forks, North Dakota

September
2019

This dissertation, submitted by Nicole Marie Della Longa in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

John-Paul Legerski, Ph.D.

F. Richard Ferraro, Ph.D.

Joseph C. Miller, Ph.D.

Joelle Ruthig, Ph.D.

Justin Berg, Ph.D.

This dissertation is being submitted by the appointed advisory committee as having met all of the requirements of the School of Graduate Studies at the University of North Dakota and is hereby approved.

Grant McGimpsey
Dean of the School of Graduate Studies

08/28/2019

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Title	Unpacking Childhood Sexual Abuse and Eating Psychopathology Over Time: Impulsive Mechanisms and Deleterious Outcomes
Department	Psychology
Degree	Doctor of Philosophy

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Nicole M. Della Longa
08/28/2019

TABLE OF CONTENTS

LIST OF FIGURES

Figure Page

LIST OF TABLES

Table Page

ACKNOWLEDGMENTS

I wish to express my sincere appreciation to my advisor and committee chairperson, Dr. John-Paul Legerski. The guidance and encouragement he provided were paramount in the development and production of this project. I would also like to offer my gratitude to Dr. Justin Berg, Dr. F. Richard Ferraro, Dr. Alan King, Dr. Joseph C. Miller, and Dr. Joelle Ruthig, who served on my advisory committee during the course of this project. Their critiques and recommendations were instrumental in enhancing the quality of this dissertation. Furthermore, I am thankful for Dr. Ross Crosby, Dr. Stephen Wonderlich, and Li Cao for allowing me to utilize their original dataset to produce Study 1. I also appreciate their consultation on this study's statistical analyses. Finally, I wish to convey my profound gratitude to the donors of the Harold Randall Scholarship and to the Department of Psychology at the University of North Dakota, whose financial support compensated participants of Study 2.

To my parents, Barbara and Greg, for their unceasing love, faith, and encouragement.
Thank you.

ABSTRACT

The present investigation incorporated two independent studies to examine childhood sexual abuse (CSA) as a risk factor for eating psychopathology and to identify a potential conceptual model illustrating these constructs. Study 1 sought to investigate the associations between CSA, binge eating, and other variables over time. In a cross-sectional design, Study 2 aimed to examine these associations and additional risk-taking behaviors within a larger sample of young adults. Study 1 participants included adolescent girls, half of whom reported a history of CSA, and Study 2 participants were young adult men and women from a nationally representative community sample. In the first study, generalized estimating equations (GEE) analyzed the impact of CSA on binge eating over time. Mediation analyses in both studies were performed using bias-corrected bootstrap confidence intervals. In the adolescent sample, the GEE model indicated binge eating did not differ by abuse status, but impulsivity emerged as a significant mediator in the bootstrapping analyses, while substance use did not. In the young adult sample, impulsivity mediated the relationships between CSA and binge eating, purging/restriction, non-suicidal self-injury, and substance use in one combined model. Together, these results imply that impulsivity may be a pertinent mechanism in the CSA-eating disorder pathway and may also predispose CSA survivors to additional deleterious behavior.

CHAPTER I

INTRODUCTION

Disordered eating patterns and behaviors represent a form of psychopathology that is especially pernicious to the livelihood of individuals, beginning as early as infancy and potentially lasting throughout the lifespan (American Psychiatric Association, 2013). Anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED), and other specified feeding or eating disorder (OSFED) represent the primary eating disorders diagnoses defined by the American Psychiatric Association (2013). AN is depicted by enduring efforts to restrict one's caloric intake, which leads to low body weight. Individuals with AN may have a strong fear of weight gain, routinely engage in behaviors that prevent weight gain despite their low-weight condition, experience significant body weight or shape dissatisfaction, consider weight or shape as pertinent factors in self-evaluation, and/or lack awareness of the severity of their low-weight status. BN is characterized by recurring binge-eating episodes (i.e., eating an objectively large amount of food in a limited period of time while experiencing a loss of control) and compensatory behaviors (i.e., purging behaviors such as self-induced vomiting, misusing laxatives or diuretics, fasting, or excessive exercise), while experiencing an undue influence of weight and shape on self-evaluation. AN can also include episodes of binge-eating and purging behaviors, but the frequency of these behaviors does not meet criteria

for BN. Furthermore, BED describes the occurrence of binge-eating episodes and experiencing symptoms such as eating more rapidly than is typical, eating until uncomfortably full, eating when not hungry without compensatory behaviors, and experiencing negative affect after the binge-eating episode. Finally, OSFED explains sub-threshold levels of eating psychopathology including behaviors characterized by AN, BN, and BED that do not meet criteria for these diagnoses but are associated with impairment and/or psychological distress (American Psychiatric Association, 2013).

Due to the physical nature of these conditions, eating disorders can affect not only the psychological functioning of an individual but can also result in an assortment of medical complications (American Psychiatric Association, 2013). In fact, eating disorders represent mental illnesses with one of the highest mortality rates (Smink, van Hoeken, & Hoek, 2012), especially AN with a weighted mortality rate of 5.1 (Arcelus, Mitchell, Wales, & Nielsen, 2011). Less than satisfactory efficacy of the gold standard treatments for eating disorders (Juarascio, Manasse, Espel, Kerrigan, & Forman, 2015) may be contributing to the high mortality rates. Due to the severity of eating psychopathology and the poor outcomes associated with these disorders, researchers have strived to identify various etiological pathways leading to the development of eating disorder behaviors in hopes of informing prevention efforts, as well as treatments. A number of potential risk factors, including pubertal timing, negative self-evaluation, dieting, and weight concerns/negative body image have been identified. Researchers have also focused significant attention on adverse personal experiences, such as childhood maltreatment, when assessing relevant risk factors (Nicholls, 2007). However, given

some inconsistencies in the literature and the limited number of prospective investigations, more evidence is necessary to ascertain if and how childhood maltreatment exists as a risk factor for eating disorder development (Caslini et al., 2016).

Childhood Maltreatment

Childhood maltreatment is defined as violence (physical, sexual, and emotional) and neglect of children enacted by parents, caregivers, and other authority figures (World Health Organization, 2016) as well as exposure to domestic violence (Armiento, Hamza, Stewart, & Leschied, 2016). Estimates from state agencies indicate that childhood maltreatment is a prevalent concern in the United States. For instance, the Children's Bureau (2016) reported an estimate of 702,000 victims of child maltreatment in 2014. Not only does exposure to abuse or neglect place children at risk for immediate negative consequences (e.g., physical injury or even mortality), but it also predisposes individuals to having long-term negative outcomes. For example, individuals who have suffered from childhood abuse are at increased risk for neurological deficits (King, MacKay, & Sirnick, 2003), reproductive problems (Bohn & Holz, 1996), chronic pain conditions (Latthe, Mignini, Gray, Hills, & Khan, 2006), and other health issues (Chartier, Walker, & Naimark, 2007). Additionally, individuals who have experienced childhood maltreatment often experience negative outcomes in regard to their psychosocial functioning (Jonas et al., 2011). Specifically, childhood maltreatment has been linked to various mental health problems, including anxiety, depression, substance use (Scott, McLaughlin, Smith, & Ellis, 2012), emotional lability, behavioral disorders, and posttraumatic stress disorder (Kendall-Tackett, Williams, & Finkelhor, 1993; Spataro, Mullen, Burgess, Wells, &

Moss, 2004), poor self-esteem, and other indicators of inhibited psychological well-being (Herrenkohl, Klika, Herrenkohl, Russo, & Dee, 2012). Thus, childhood maltreatment appears to be a predictor of various pernicious outcomes, making it vital to continue to investigate the relationship among these variables.

Eating psychopathology represents another sequela that has been linked to childhood maltreatment in numerous studies. Multiple meta-analyses (Caslini et al., 2016; Rind, Tromovitch, & Bauserman, 1998; Smolak & Murnen, 2002) and narrative reviews (Brewerton, 2007; Kent & Walker, 2000) have provided evidence for the relationship among eating psychopathology and childhood maltreatment. These studies have identified associations of various forms of child abuse and neglect with sub-threshold and full-threshold levels of eating psychopathology. Given the physical and psychological negative outcomes associated both with eating disorders and childhood maltreatment, better understanding the relationship between these phenomena may be especially fruitful for informing assessment, treatment, and prevention efforts.

Childhood Sexual Abuse

Investigating childhood sexual abuse (CSA) above other forms of childhood maltreatment appears to be a special area of interest within the eating disorder literature. Researchers originally postulated CSA as a risk factor for eating disorders due to some of the similar symptoms reported by both sets of patients, including shame, body disparagement, and poor self-esteem (Thompson & Wonderlich, 2004). Thus, many studies have tested the relationship between CSA and eating disorder symptomatology in individuals of various ages.

Weight Dissatisfaction

Cross-sectional studies have suggested a link between CSA and cognitive symptoms of eating disorders. Weight-related attitudes, such as weight dissatisfaction, appear to be relevant when examining correlates of CSA. Weight dissatisfaction is characterized by a discrepancy between one's actual weight and her desired weight (Wirth, Blake, Hébert, Sui, & Blair, 2014). Empirical evidence suggests negative attitudes toward body and weight are risk factors for the development of eating psychopathology (Jacobi, 2005; McKnight, 2003; Stice, 2002). Individuals who express concerns about their weight are more likely to experience negative affect and engage in behaviors to modify their weight, including dietary restriction and bulimic symptomatology (Stice, 2002). In the case of CSA, an individual may have increased distress about her body in general, which could include weight and shape concerns (Thompson, Wonderlich, Crosby, & Mitchell, 2001) and perhaps a desire to repair or improve the body after experiencing the sexual trauma (Sansone & Sansone, 2007). Indeed, weight dissatisfaction has been linked to CSA in various samples. For instance, adolescent girls who have experienced CSA reported lower levels of weight satisfaction than girls without a history of CSA, despite their average-weight status (Moyer, DiPietro, Berkowitz, & Stunkard, 1997; Wonderlich et al., 2000; Wonderlich et al., 2001a). Furthermore, current weight dissatisfaction appears to be present in samples of adults who provide retrospective reports of CSA. Kenardy and Ball (1998) supply evidence that having previous unwanted sexual experiences was related to weight dissatisfaction in a group of young adult women. Similarly, a sample of undergraduates who reported a

history of CSA endorsed higher levels of weight concern (Villarroel, Penelo, Portell, & Raich, 2012). Thus, negative attitudes regarding weight, which are inherent in bulimic symptomatology and in many cases of anorexic symptomatology (American Psychiatric Association, 2013), may be an important factor to consider when investigating potential outcomes of sexual violence.

Eating Disorder Behaviors

Eating disorder symptomatology in the form of maladaptive behaviors is likewise frequently studied as a potential outcome of CSA in cross-sectional investigations. For instance, adolescent girls (from a general psychiatric inpatient unit) who reported a history of sexual abuse, when compared to girls without a history of CSA, had an increased likelihood of engaging in extreme weight-loss behavior (e.g., using self-induced vomiting, diet pills, laxatives, diuretics, or drinking only non-energy fluids; Isohookana, Marttunen, Hakko, Riipinen, & Riala, 2016). Thompson and colleagues (2001) revealed similar findings in an examination of three community samples (i.e., urban, rural, and statewide) of adolescent girls, such that sexual victimization was linked to an increased probability of weight-regulation tactics. Purging behaviors had an especially increased risk for adolescents in the urban sample who reported a history of sexual victimization. Further, in another sample of adolescents, Ackard and Neumark-Sztainer (2003) provided cross-sectional evidence that individuals who report previous experiences of CSA are more likely than peers who were not abused to engage in eating disorder behaviors, including fasting, binge eating, and various purging behaviors. In this same sample, multiple sexual victimizations were related to increased levels of eating

psychopathology and other poor psychological outcomes. Similar findings are present in numerous other cross-sectional tests of eating-disorder behaviors in adolescents who were survivors of CSA (Ackard, Neumark-Sztainer, Hannan, French, & Story, 2001; Chandy, Blum, & Resnick, 1996; Hopwood, Ansell, Fehon, & Grilo, 2011; Neumark-Sztainer et al., 2000; Wonderlich et al., 2001a).

In addition to adolescents, researchers have investigated levels of current eating psychopathology in adults who endorse previous experiences of sexual abuse when they were children. Retrospective reports of CSA are linked to bulimic symptomatology in female undergraduate students compared to students without a history of CSA (Dworkin, Javdani, Verona, & Campbell, 2014; Jenkins, Meyer, & Blissett, 2013; Rodriguez-Srednicki, 2001). Additionally, adult women from community samples who reported experiencing sexual abuse as a child also endorsed greater levels of eating disorder behaviors than women without a reported history of CSA (Wonderlich et al., 2001b; Wonderlich, Wilsnack, Wilsnack, & Harris, 1996). All together, these findings suggest that the experience of CSA is an important correlate, if not risk factor, for eating psychopathology.

Given the findings previously discussed, it is perhaps unsurprising that CSA is also salient in clinical populations. In fact, approximately 30% of individuals with eating disorders have reported being sexually abused in childhood, especially those individuals diagnosed with BN and BED (Behar, Arancibia, Sepúlveda, & Muga, 2016). This statistic is important to consider, especially given that in the national population, 20% of adult females and 5-10% of adult males report a history of childhood sexual assault or

abuse (The National Center for Victims of Crime, 2012). A recent meta-analysis of studies with adolescents and adults suggests strong associations between retrospective reports of CSA and multiple forms of eating disorders (e.g., AN, BN, BED, and eating disorder not otherwise specified) when compared to healthy control groups (Molendijk, Hoek, Brewerton, & Elzinga, 2017). Other meta-analyses and a review, including adolescents and adults, revealed a consistent link among CSA and patients with BN (Caslini et al., 2016; Wonderlich, Brewerton, Jocic, Dansky, & Abbot, 1997), BED (Caslini et al., 2016), and eating disorders in general (Chen et al., 2010). These findings provide additional evidence for childhood sexual victimization acting as a risk factor for eating disorder development.

Inconsistencies in the Literature

Despite the abundance of evidence proposing the link between CSA and eating disorder symptomatology, there are inconsistencies that exist in the literature. Some investigations of these factors suggested no relationship between CSA and eating disorder symptoms (e.g., Kent, Waller, & Dagnan, 1999; Kinzl, Traweger, Guenther, & Biebl, 1994; Korte, Horton, & Graybill, 1998) or between CSA and full-threshold eating disorders (e.g., Pribor & Dinwiddie, 1992; Rorty, Yager, & Rossotto, 1994). Additionally, some researchers initially revealed a relationship among sexual abuse and eating psychopathology; however, upon further investigation, they discovered that other variables (e.g., impulsive behaviors) better explained the relationship between these factors (e.g., Casper & Lyubomirsky, 1997). These discrepancies are consistent with findings from Smolak and Murnen's (2002) meta-analysis, which revealed a CSA-eating

disorder relationship that was characterized by significant heterogeneity. The authors interpreted this heterogeneity as a function of the methodological variation between previous studies and the likely diversity in the relationship between CSA and eating disorders. Therefore, the relationship between harmful childhood sexual experiences and eating psychopathology is likely not direct, and it may be influenced by other mediating and moderating factors. If these factors are not present, then CSA may not predict the development of eating disorders or may lead to the development of alternative psychopathology, such as depression, anxiety, and substance abuse (Maniglio, 2009). Consequently, some researchers have postulated that CSA is a non-specific risk factor for eating psychopathology (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). This position confers with the understanding that eating disorder etiology is multifactorial and is comprised of complex models, portraying how factors collaboratively develop disordered eating patterns (Stice, 2002). Thus, in the context of CSA, it is imperative to identify what specific factors make survivors of this abuse vulnerable to eating disorder symptomatology.

Conceptualizing CSA and Eating Psychopathology

Empirical Evidence

Many researchers have sought to explain the theoretical link between unwanted childhood sexual experiences and eating psychopathology. In this process, investigators have considered numerous variables as influential factors in this relationship. For instance, there is some evidence to suggest that negative affect/depressive symptoms account for the association between CSA and eating psychopathology in adolescents,

including dietary restraint (Hopwood et al., 2011), binge eating (Hopwood et al., 2011; Moyer et al., 1997), and other bulimic symptomatology (Sanci et al., 2008). Researchers have also proposed that cognitions often related to negative affect, including low self-esteem, serve as relevant factors in the relationship between CSA and eating disorder behaviors due to the link between poor self-concept and both forms of psychopathology (e.g., Ackard & Neumark-Sztainer, 2003; Harper, Richter, & Gorey, 2009; Kugu, Akyuz, Dogan, Ersan, & Izgic, 2006). However, other investigations have contradicted these findings (e.g., Wonderlich et al., 2001a) and have proposed that depression (e.g., Kent et al., 1999) and measures of self-esteem (e.g., Groleau et al., 2012) more accurately mediate the relationship between childhood emotional abuse and eating psychopathology. There is also evidence to suggest behaviors and beliefs more specific to eating disorders serve as mediators explaining the link between CSA and eating psychopathology. For example, dieting (Sanci et al., 2008) and weight satisfaction (Moyer et al., 1997) may represent important mechanisms relating CSA and BN symptoms in adolescents.

Alternatively, researchers have questioned the impact of temperamental factors, such as impulsivity, in the CSA-eating disorder relationship (Favaro et al., 2005; Thompson, Wonderlich, Crosby, Redlin, & Mitchell, 2002), and some investigators have provided evidence for the mediating role of impulsivity (Dworkin et al., 2014; Wonderlich et al., 2001a). It has also been proposed that adolescent substance use, which has been linked to CSA (e.g., Grilo, Sanislow, & Fehon, 1999; Maniglio, 2009) and impulsivity (e.g., Leon, Fulkerson, Perry, & Klump, 1999), impacts the relationship between CSA and eating psychopathology. Indeed, Wonderlich and colleagues (2001a)

demonstrated a relationship between unwanted childhood sexual experiences and purging and dietary restriction behaviors in a sample of adolescents. Therefore, there appears to be evidence for various factors accounting for the association among CSA and eating psychopathology. However, due to the lack of consistency and limited number of examinations, it is difficult to ascertain the nature of the relationship among these variables.

Proposed Developmental Models

Based on the evidence currently existing, researchers have proposed several risk models in attempt to conceptualize the link between CSA and eating disorders. Thompson and Wonderlich (2004) describe two models pertaining to impulsive personality traits. One model highlights the mediating roles of impulsivity and related psychopathology. This model postulates that exposure to CSA results in vulnerability to impulsive personality traits and dysregulated behaviors. Such vulnerability may be due to psychobiological alterations (Putnam & Trickett, 1997) and changes in neurotransmitter functioning (Post, Weiss, & Smith, 1994; Putnam & Tricket, 1997) that are linked to exposure to trauma. These individuals may then be more prone to experience impulsivity-related psychopathology and engage in behaviors consistent with borderline personality disorder (Waller, 1993), dissociation (Kent et al., 1999), self-harm, posttraumatic stress disorder, and substance use (Wonderlich et al., 1996; Wonderlich et al., 2001b). Eating disorder behaviors are then likely to originate after the development of the aforementioned impulsive behaviors, as an expression of this impulsivity-related psychopathology (Johnson & Connors, 1987; Nagata, Kawarada, Kiriike, & Iketani,

2001; Thompson et al., 2002). Binge eating, which often occurs without initial consideration of the consequences of this behavior, has been linked to impulsivity both in cross-sectional (e.g., Galanti, Gluck, & Geliebter, 2007) and prospective studies (e.g., Wonderlich, Connolly, & Stice, 2004). Impulsive behaviors have also predicted engagement in compensatory behaviors (Wonderlich et al., 2004). Further, there is evidence in a cross-sectional study of adolescents to suggest that impulsivity and substance use mediate the link between CSA and eating disorder behaviors (Wonderlich et al., 2001a).

However, findings from a longitudinal examination of adolescents suggest substance use does not independently predict eating psychopathology (Johnson, Cohen, Kotler, Kasen, & Brook, 2002). This inconsistency warrants the discussion of a similar but distinct CSA and eating disorder conceptual model. In this model, Thompson and Wonderlich (2004) postulate CSA predicts a general psychobiological dysregulation that can lead to a variety of psychopathology. Thus, a multitude of self-destructive behaviors develop in a co-morbid presentation (e.g., bulimic symptomatology, substance use, self-harm behaviors, and other psychopathology) without having a mediator role. Instead, these behaviors together exemplify impulsivity and impairment in self-regulation as a consequence of the sexual trauma. Evidence that CSA is related to "multi-impulsive" binge eating (i.e., binge eating and at least three impulsive and pernicious behaviors) supports this model (Wonderlich et al., 2001b). Findings suggesting that between one-fourth and one-third of individuals with BN display several modes of impulsivity are consistent with this model (Thompson & Wonderlich, 2004). While these

conceptualizations may provide an understanding of the risk CSA poses for the development of eating disorder symptomatology, limited empirical testing of these and other related models currently exists.

Methodological Concerns

While numerous investigations have depicted CSA as a risk factor for the development of eating disorder symptomatology, the majority of these studies have based their conclusions on cross-sectional examinations (e.g., Ackard & Neumark-Sztainer, 2003; Isohookana et al., 2016). These designs preclude the ability to determine temporal ordering of variables and thus cannot accurately depict risk factors (Caslini et al., 2016). In order to determine whether a variable represents a risk factor, it must prospectively predict another variable of psychopathology. Specifically, the risk factor must predict a disorder's onset in individuals who were originally without the disorder, or the risk factor must predict increase in symptomatology when original symptom levels are controlled (Stice, 2002). To date, there have been few prospective studies that have examined CSA and eating disorder symptomatology. These investigations provide evidence for the impact of unwanted childhood sexual encounters on eating psychopathology over time (Johnson, Cohen, Kasen, & Brook, 2002; Sanci et al., 2008; Smyth, Heron, Wonderlich, Crosby, & Thompson, 2008; Van der Kolk, Perry, & Herman, 1991; Vrabel, Hoffart, Rø, Martinsen, & Rosenvinge, 2010). However, all but two of these studies relied on adults' retrospective reports of CSA. Additionally, one of these investigations measured eating psychopathology over time beginning in adolescence, but it did not assess for CSA until participants were 24 years old.

While it is important to understand the origin and course of eating psychopathology in individuals of all ages, there are some limitations to drawing conclusions from samples of adults who report prior sexual victimization and current eating disorder behaviors. Due to the distal nature of the abuse, it is possible other factors may have impacted the eating disorder symptomatology reported at adulthood (Kenardy & Bell, 1998). Retrospective reports of CSA may also increase the chance of recall bias, thereby depicting an inaccurate account of childhood abuse in the sample (Sanci et al., 2008). Additionally, during adolescence and young adulthood, a number of developmental transitions tend to occur, along with alterations in the structure and function of the brain (Arnett, Žukauskienė, & Sugimura, 2014; McGorry, Goldstone, Parker, Rickwood, & Hickie, 2014). These adjustments make this developmental period a particularly vulnerable time, during which eating disorders are more likely to develop (American Psychiatric Association, 2013).

The Present Study

Research examining samples of individuals with a history of CSA have largely indicated a positive association between unwanted child sexual experiences and eating disorder symptomatology. These studies have examined these relationships primarily in cross-sectional designs and thereby have not been able to establish temporal precedence of the variables (Caslini et al., 2016). The current study will expand the work conducted by Wonderlich and colleagues (2001a), which compared a group of adolescent girls with a reported history of CSA to an aged-matched control group. Findings from this study revealed that reports of CSA were related to higher levels of weight dissatisfaction,

purging/restriction, impulsive behaviors, and substance use than controls. The study also revealed impulsivity mediated the relationship between abuse status and weight dissatisfaction. Additionally, impulsivity and substance use mediated the relationship between abuse status and purging/restriction.

Using the same dataset, Study 1 examined similar relationships identified by Wonderlich and colleagues (2001a) and included a 1- and 2-year follow-up. As a further expansion of Wonderlich and colleagues' findings, the present study introduced binge eating as a variable of interest. Thus, the primary aim of Study 1 was to investigate the impact of CSA on eating psychopathology over three time points (i.e., baseline, 1-year follow-up, and 2-year follow-up) in a sample of adolescent girls, half of whom reported a history of CSA. It was hypothesized that 1) CSA would predict binge eating over time, and this relationship would be mediated by weight dissatisfaction, impulsivity, and substance use, and 2) CSA would predict purging/restriction over time, and this relationship would be mediated by weight dissatisfaction, impulsivity, and substance use.

An additional follow-up study (Study 2) was conducted with young adults. The aim of Study 2, an investigation using cross-sectional data, was to compare associations between CSA and other factors measured in Study 1. Specifically, it was hypothesized that 1) impulsivity would mediate the relationship between CSA and binge eating and 2) impulsivity would mediate the association between CSA and purging/restriction. Further, Study 2 performed a preliminary examination of a risk model containing eating disorder behaviors and additional risk-taking behaviors, including non-suicidal self-injury and substance use. Given the scarcity of male participants in investigations of the relationship

between child maltreatment and eating psychopathology (Afifi et al., 2017; Caslini et al., 2015), Study 2 sought to narrow this gap by evaluating CSA outcomes in both women and men.

CHAPTER II

STUDY 1

Method

Participants

Participants were obtained from an archival dataset and consist of two groups of girls. One group (n = 20) includes girls who reported a history of sexual abuse. In the present study, sexual abuse is defined as 1) unwanted intrafamilial sexual activity or sexual activity with a family member that occurred with a family member who was at least 5 years older than the participant or 2) unwanted extrafamilial sexual activity or sexual activity with an individual at least 5 years older than the participant. The control group (n = 20) includes girls without a history of any type of childhood abuse as reported by their parents. The control group was matched to the group of abused girls based on their age and parents' level of education. Participants were recruited via school newsletters that were sent to parents. Participants' ages at the baseline measurement ranged from 10 to 16 (M = 13.30, SD = 1.69). The remaining participant characteristics are described in Table 1.

Table 1

Study 1 Participant Characteristics

		n	Percent
Race/Ethnicity	Asian	1	2.5%
	Black/African American	2	5.0%

Table 1 (continued).

	White	37	92.5%
Parental Education	Less than High School	1	2.5%
	High School	5	12.5%
	GED	1	2.5%
	VO-Tech	8	20.0%
	Partial College	12	30.0%
	College	12	30.0%
	Post College	1	2.5%
Parental Annual Income	$4,000-$9,999	3	7.5%
	$10,000-$15,999	8	20.0%
	$16,000-$25,999	12	30.0%
	$26,000-$35,999	5	12.5%
	$36,000+	12	30.0%

Note. VO-Tech abbreviates vocational-technical school.

Measures

Eating psychopathology. The McKnight Risk Factor Survey (MRFS-IV) is a 103-item self-report instrument assessing potential risk and protective factors for eating disorder development among preadolescent and adolescent girls. Two items were used to measure binge eating. Participants respond on a 5-point scale, ranging from 1 (“never”) to 5 (“always) with higher scores indicating greater psychopathology. The binge eating subscale’s internal consistency reliability was questionable for Time 1 ($\alpha = .61$), poor for Time 2 ($\alpha = .59$), and excellent for Time 3 ($\alpha = .91$). Psychometric properties of the MRFS-III, a previous version of the MRFS-IV, demonstrate acceptable levels of test-retest reliability ($r > .40$) and measures of validity (Shisslak et al., 1999).

The Kids Eating Disorders Survey (KEDS) is a 14-item self-report measure that assesses eating-disorder symptomatology in children and adolescents. Participants select "yes," "no," or "don't know" when responding to items. Responses were dichotomized, such that items with a positive response (i.e., "yes") received a score of 1 and items with all other responses (i.e., "no" or "don't know") received a score of 0. The KEDS is comprised of two subscales: weight dissatisfaction and purging/restriction (Childress, Jarrell, & Brewerton, 1993). The weight dissatisfaction subscale's internal consistency reliability was acceptable for Time 1 ($\alpha = .73$), Time 2 ($\alpha = .79$), and Time 3 ($\alpha = .77$). The purging/restriction subscale's internal consistency reliability was good for Time 1 ($\alpha = .84$), adequate for Time 2 ($\alpha = .72$), and unacceptable ($\alpha = .28$) for Time 3.

Impulsivity. The Impulsive Behavior Scale (IBS) is a self-report instrument consisting of 25 items. The IBS measures the frequency of various impulsive behaviors (e.g., sexual promiscuity, shoplifting, suicidal thoughts, etc.; Rossotto, Yager, & Rorty, 1998). Six items deemed inappropriate for children were excluded (e.g., reckless driving). Respondents indicate the number of times they have engaged in a particular impulsive behavior on a scale of 1 ("never") to 5 ("regularly") with higher scores suggesting greater levels of impulsivity. In the present study, the IBS's internal consistency reliability was excellent for Time 1 ($\alpha = .92$), good for Time 2 ($\alpha = .84$), and good ($\alpha = .87$) for Time 3. The IBS provides evidence for adequate indicators of validity (Anestis, Smith, Fink, & Joiner, 2009; Van Orden, Witte, Gordon, Bender, & Joiner, 2008).

Substance use. The Monitoring the Future Questionnaire (MTFQ) is a 167-item self-report measure asking respondents to rate "the way things are now and the way you think they ought to be in the future." Six items from this measure assess substance-use

patterns on a scale of 0 (“none”) to 5 (“all”) with higher values suggesting higher levels of substance use (Bachman, Johnston, & O’Malley, 1987). In the present study, the MTFQ’s internal consistency reliability was excellent for Time 1 (α = .91), Time 2 (α = .90), and Time 3 (α = .90). The MTFQ presents evidence for favorable indicators of validity (Johnston, O’Malley, Bachman, & Schulenberg, 2010).

Procedure

Following informed consent of parents, the researchers explained to the participants how to respond to the measures. Participants completed the demographics questionnaire, MRS-IV, KEDS, IBS, and MTFQ independently in a room without the research staff who were available for assistance if needed. Participants were contacted one and two years after the initial meeting to complete the same measures at each time point. Participants were awarded $75 for each year of participation.

Statistical Analyses

As previously noted, the purging/restriction subscale of the KEDS demonstrated unacceptable internal consistency reliability at Time 3 (α = .28). This Cronbach alpha value, which is strikingly lower than the Cronbach alpha values of the preceding years, suggests that this subscale is not consistently measuring purging/restriction behaviors at this timepoint. Further investigation of the KEDS at the item level prompted another cause for concern. The vast majority of the items from the KEDS inquire about respondents’ lifetime experience with disordered eating behaviors or cognitions (e.g., “Have you ever taken diet pills to lose weight?”; Childress et al., 1993). It appears that 32.5% of participants who endorsed a purging/restriction behavior at Time 1 or at Time 2 did not endorse the same behavior at subsequent timepoints. Given the wording of these

subscale items, which request reports of lifetime occurrence, this pattern of across-time discrepancy is difficult to fully explain. Items from the weight dissatisfaction subscale of the KEDS exhibited a similar pattern of discrepancy across timepoints to an even greater extent (i.e., 65% of participants). These observations, along with the unacceptable Cronbach alpha value of the Time 3 purging/restriction subscale, markedly call into question the reliability and veracity of the data collected from this scale. Thus, the KEDS measure was excluded from all further analyses; subsequently, the proposed hypotheses that included weight dissatisfaction and purging/restriction as variables of interest were not tested in Study 1.

Generalized estimating equations (GEE). The hypothesis that CSA predicts binge eating over time was tested using GEE. GEE models are utilized to examine longitudinal datasets (Zeger, Liang, & Albert, 1988) and are equipped to manage correlated repeated observations (Twisk, 2004). In the present study, the GEE model was performed using the gamma distribution with the log link function. The model was estimated utilizing a first-order autoregressive correlation structure. The significance threshold was set at .05. In this analysis, *abuse status* and *time* represented the independent variables and *binge eating* represented the dependent variable.

Mediation analyses. Two mediation analyses were conducted to examine the hypotheses. Mediation analyses were performed using a bootstrapping approach within Mplus Version 8 (Muthén & Muthén, 2017). This approach tests multiple paths among the variables of interest within a given mediation model. The *a* path represents the direct effect of the independent variable on the mediator. The *b* path serves as the direct effect of the mediator on the dependent variable. The *c'* path represents the direct effect of the

independent variable on the dependent variable when the mediation effect is held constant. The *c* path explains the total effect of the independent variable on the dependent variable. The *ab* path represents the indirect effect or the mediation effect. This path is the product of the effect of the independent variable on the mediator (*a*) and the effect of the mediator on the dependent variable (*b*). This bootstrapping method calculated 10,0000 samples and used bias-corrected bootstrap 95% confidence intervals. If the confidence interval of the indirect effect (*ab*) does not include zero, this outcome suggests a significant mediation effect (Preacher & Hayes, 2008). In these analyses, the independent variable was a dummy-coded variable: *CSA*. *Binge eating* was the dependent variable. *Impulsivity* and *substance use* were identified as mediator variables in separate models. Specifically, the first model examined the indirect effect of *CSA* on *binge eating* at Time 3 through Time 2 *impulsivity*. The second model tested the indirect effect of *CSA* on *binge eating* at Time 3 through Time 2 *substance use*. Furthermore, the indirect effects within each of these models were also tested cross-sectionally at Time 1, Time 2, and Time 3.

Results

Preliminary Analyses

Table 2 indicates the means and standard deviations of the participants' scores on the variables of interest. Independent *t*-tests were performed comparing the variable means of participants with a history of CSA with those who denied a history of CSA. At Time 1, participants in the CSA group endorsed higher mean impulsivity scores than did participants in the control group. The mean scores of impulsivity at Time 2 and at Time 3 were not significantly different between the participants who had experienced CSA and

those without a history of CSA. At all three timepoints, there were no significant differences between the two groups' mean scores of binge eating and substance use.

Table 2

Study 1 Descriptive Statistics Using t-test for Group Mean Differences

	Control		Abused		
	M	*SD*	*M*	*SD*	*t*-test
Time 1					
Binge eating	1.70	0.64	1.75	0.62	0.25
Impulsivity	24.40	7.94	33.90	14.45	2.58*
Substance use	0.28	0.83	2.05	4.03	1.88
Time 2					
Binge eating	1.58	0.52	1.83	0.67	1.31
Impulsivity	24.45	6.41	29.45	8.99	2.03
Substance use	0.70	1.56	3.10	5.10	2.01
Time 3					
Binge eating	1.70	0.89	1.80	1.01	0.33
Impulsivity	27.45	8.48	29.65	10.63	0.72
Substance use	1.05	1.70	3.20	4.72	1.92

Note. The means and standard deviations are noted separately for the participants with a history of CSA (i.e., abused) and those without a history of CSA (i.e., control) at each timepoint. *$p < .05$.

Table 3 summarizes the bivariate Pearson correlations between the dummy-coded CSA variable as well as the mediating and dependent variables at each timepoint. CSA was significantly correlated with impulsivity at Time 1 and at Time 2 but was not significantly correlated with the remaining variables. The correlation between CSA and

substance use approached significance at Time 2 (p = .051). Binge eating was significantly correlated with impulsivity at overlapping timepoints. Binge eating at Time 2 was significantly associated with substance use at Time 3. Impulsivity and substance use were significantly correlated with each other across all timepoints. All correlations indicated positive relationships among the variables, and significant correlations demonstrated moderate to very strong relationships (rs = 0.31 to 0.85).

Table 3

Study 1 Bivariate Pearson Correlations

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. CSA (dummy-coded)	-									
Time 1										
2. Binge eating	0.04	-								
3. Impulsivity	0.39*	0.40*	-							
4. Substance use	0.30	0.05	0.73**	-						
Time 2										
5. Binge eating	0.21	0.34*	0.28	0.25	-					
6. Impulsivity	0.31*	0.25	0.61**	0.63**	0.40*	-				
7. Substance use	0.31	0.10	0.61**	0.81**	0.30	0.71**	-			
Time 3										
8. Binge eating	0.05	0.36*	0.18	0.12	0.57**	0.40*	0.10	-		
9. Impulsivity	0.12	0.29	0.64**	0.76**	0.30	0.80**	0.64**	0.36*	-	
10. Substance use	0.30	0.19	0.62**	0.71**	0.34*	0.64**	0.85**	0.05	0.65**	-

Note. Correlations are rounded to the nearest tenth. *p < .05. **p < .01.

GEE Model

The GEE model indicated that abuse status did not significantly predict binge eating, such that the frequency of binge eating did not significantly differ between participants with a history of CSA and participants without a history of CSA. Additionally, time did not significantly predict binge eating, indicating the frequency of binge eating that the participants endorsed did not change significantly across the three timepoints. There was no significant interaction between abuse status and time. Table 4 notes the parameter estimates from the GEE model.

Table 4

GEE Model Parameter Estimates

Parameter	β	*SE*	Lower CI	Upper CI	Wald Chi-Square	*df*	*p*
Abused Group	0.057	0.167	-0.271	0.385	0.117	1	.732
Control Group	-	-	-	-	-	-	-
Time 1	0.00	0.083	-0.163	0.163	0.000	1	1.000
Time 2	-0.076	0.082	-0.237	0.085	0.864	1	.353
Time 3	-	-	-	-	-	-	-
Abused Group x Time 1	-0.028	0.163	-0.347	0.291	0.030	1	.863
Abused Group x Time 2	0.090	0.136	-0.176	0.356	0.440	1	.507
Abused Group x Time 3	-	-	-	-	-	-	-
Control Group x Time 1	-	-	-	-	-	-	-

Table 4 (continued).

Control Group x Time 2	-	-	-	-	-	-	-
Control Group x Time 3	-	-	-	-	-	-	-

Note. SE = standard error. CI = confidence interval.

Mediation Analyses

In Model 1 (see Figure 1 and Table 5), results revealed a significant direct effect of CSA (Time 1) on impulsivity (Time 2; a_1), indicating a significant association between CSA and impulsivity. The direct effect of impulsivity (Time 2) on binge eating (Time 3; b_1) was not significant, which suggests no significant association between impulsivity and binge eating. The direct effect of CSA (Time 1) on binge eating (Time 3; c'_1) was not significant, suggesting CSA was not significantly related to binge eating when the indirect effect was held constant. The indirect effect of CSA (Time 1) on binge eating (Time 3; ab_1) was statistically different from zero, suggesting impulsivity mediates the relationship between these variables. The cross-sectional examinations of this model also revealed indirect effects of CSA on binge eating that were statistically different from zero at Time 1 (β = 0.173, *SE* = 0.095, 95% CI = [0.054 - 0.375], *p* = .069) and at Time 2 (β = 0.116, *SE* = 0.073, 95% CI = [0.022 - 0.272], *p* = .114), suggesting impulsivity mediated the relationship between CSA and binge eating at these two timepoints. There was no significant indirect effect of CSA on binge eating at Time 3 (β = 0.042, *SE* = 0.063, 95% CI = [-0.032 - 0.179], *p* = .506), indicating that impulsivity did not mediate the relationship between these variables at Time 3.

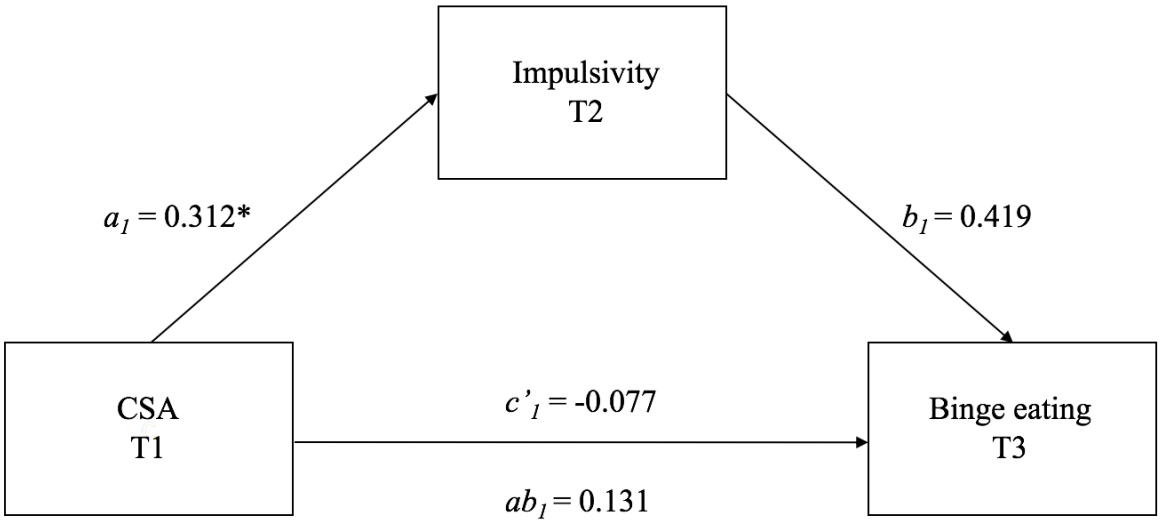

Figure 1. Model 1 standardized path coefficients. a_1 = direct effect of CSA on impulsivity. b_1 = direct effect of the impulsivity on binge eating. c'_1 = direct effect of CSA on binge eating. ab_1 = indirect effect of CSA on binge eating through impulsivity. ab_1 is statistically different from zero, as evidenced by its confidence interval. T1 = Time 1. T2 = Time 2. T3 = Time 3. *p < .05.

Table 5

Model 1 Mediation Analysis

Effect	Coefficient	*SE*	*p*	Lower CI	Upper CI
a_1	0.312	0.142	.028	0.052	0.523
b_1	0.419	0.220	.057	-0.018	0.708
c'_1	-0.077	0.165	.641	-0.334	0.210
ab_1	0.131	0.096	.172	0.016	0.351

Note. All coefficients are standardized. CI = confidence interval. a = direct effect of CSA on impulsivity. b_1 = direct effect of impulsivity on binge eating. c'_1 = direct effect of CSA on binge eating. ab_1 = indirect effect of CSA on binge eating through impulsivity.

In Model 2 (see Figure 2 and Table 6), results indicated a significant direct effect of CSA (Time 1) on substance use (Time 2; a_2), indicating a significant association

between CSA and substance use. The direct effect of substance use (Time 2) on binge eating (Time 3; b_2) was not significant, which suggests no significant association between substance use and binge eating. The direct effect of CSA (Time 1) on binge eating (Time 3; c'_2) was not significant, suggesting CSA was not significantly related to binge eating when the indirect effect was held constant. The indirect effect of CSA (Time 1) on binge eating (Time 3; ab_2) was not significant, revealing that substance abuse did not mediate the relationship between these variables. The cross-sectional examinations of this model also revealed no significant indirect effects of CSA on binge eating at Time 1 (β = 0.009, *SE* = 0.053, 95% CI = [-0.067 - 0.105], *p* = .866), at Time 2 (β = 0.081, *SE* = 0.074, 95% CI = [-0.017 - 0.222], *p* = .273), and at Time 3 (β = 0.012, *SE* = 0.061, 95% CI = [-0.070 - 0.133], *p* = .840). These cross-sectional models suggest substance use did not mediate the relationship between CSA and binge eating at Time 1, Time 2, and Time 3, respectively.

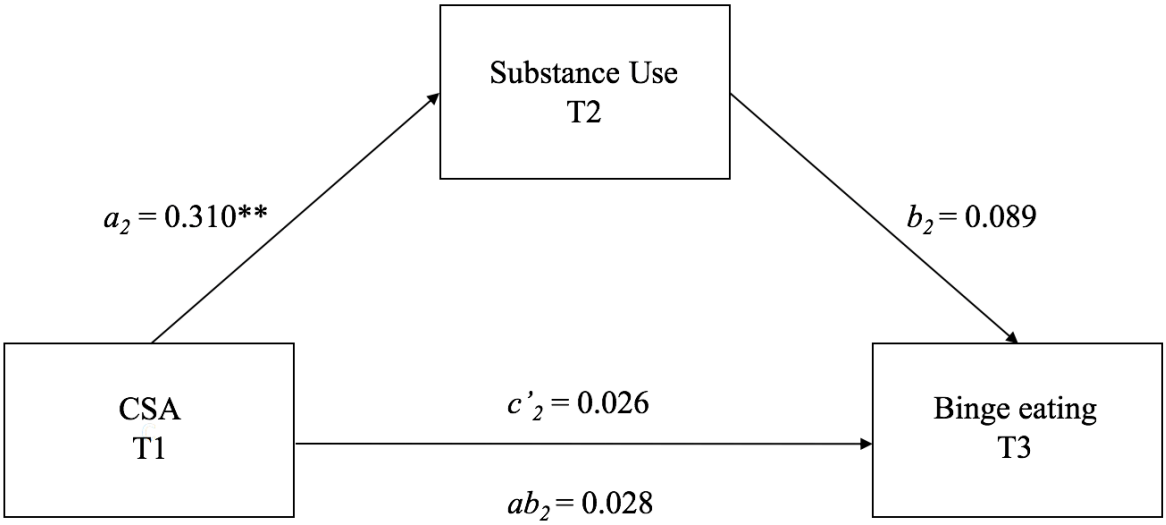

Figure 2. Model 2 standardized path coefficients. a_2 = direct effect of CSA on substance use. b_2 = direct effect of substance use on binge eating. c'_2 = direct effect of CSA on binge eating. ab_2 = indirect effect of CSA on binge eating through substance use. T1 = Time 1. T2 = Time 2. T3 = Time 3. **$p < .01$.

Table 6

Model 2 Mediation Analysis

Effect	Coefficient	*SE*	*p*	Lower CI	Upper CI
a_2	0.310	0.111	.005	0.108	0.469
b_2	0.089	0.176	.612	-0.194	0.384
c'_2	0.026	0.175	.881	-0.268	0.309
ab_2	0.028	0.059	.639	-0.052	0.142

Note. All coefficients are standardized. CI = confidence interval. a_2 = direct effect of CSA on substance use. b_2 = direct effect of substance use on binge eating. c'_2 = direct effect of CSA on binge eating. ab_2 = indirect effect of CSA on binge eating through substance use.

CHAPTER III

STUDY 2

Method

Participants

Participants (N = 746) were recruited from Amazon's Mechanical Turk (MTurk). The MTurk assignment was restricted to MTurk workers with an 80% approval rating who resided in the United States and were between the ages of 18 and 29 years old. Only 432 participants were included in all data analyses, as the remaining participants did not complete the study or were categorized as careless responders. The latter endorsed incorrect answers to items that have overtly correct responses (e.g., "In the past 12 months, have you been to every country in the world?"), which suggested careless or dishonest responding (Meade & Craig, 2012). Participants were predominately women (55.6%), and their ages ranged from 18 to 29 (M = 25.81, SD = 2.60). The remaining participant characteristics are described in Table 7.

Table 7

Study 2 Participant Characteristics

		n	Percent
Race/Ethnicity	American Indian/Alaska Native	3	.7%
	Asian	23	5.3%
	Black/African American	46	10.6%
	Hispanic/Latino	32	7.4%
	Native Hawaiian/Other Pacific Islander	1	.2%

Table 7. (continued)

	White	316	73.1%
	Other	11	2.5%
Geographical Location	Midwest	77	17.8%
	Southwest	40	9.3%
	West	80	18.5%
	Northeast	94	21.8%
	Southeast	141	32.6%
Level of Education	High School Diploma/GED	45	10.4%
	Some College Credit	123	28.5%
	Associate Degree	158	36.6%
	Master's Degree	98	22.7%
	MD, JD, PhD, or Other Doctoral Degree	8	1.9%

Measures

CSA. The Comprehensive Child Maltreatment Scale (CCMS) for Adults is a self-report measure consisting of 22 items (Higgins & McCabe, 2001), assessing various modes of childhood abuse or neglect. With permission from the primary creator of the CCMS, the present study utilized an altered set of instructions that requested participants endorse their experiences of abuse prior to the age of 18 (rather than prior to the age of 13). Using items from the CCMS Sexual Abuse subscale, participants rated the extent to which they experienced CSA perpetrated by their mother, father, and another adult or older adolescent, respectively. Participants indicted the number of lifetime CSA episodes they have encountered on a scale from 0 ("Never") to 5 ("More than 20 times"). The CCMS Sexual Abuse subscale exhibited excellent internal consistency reliability (α =

.97). The CCMS for Adults has demonstrated concurrent criterion-related validity (Higgins & McCabe, 2001).

Eating psychopathology. The Eating Disorder Diagnostic Scale (EDDS) is a 22-item self-report measure that assesses symptoms of AN, BN, BED, and OSFED (Stice, Telch, & Ritzvi, 2000). Items on the EDDS measured frequency of binge eating and purging/restriction behaviors (i.e., self-induced vomiting, using laxatives or diuretics, and fasting). Ranging from 0 to 14, participants indicated the average frequency in which they engaged in each of these behaviors over the past 3 months. A purging/restriction variable was calculated by summing participants' responses to all of the items assessing frequency of purgative and fasting behaviors. Items comprising this variable exhibited good internal consistency reliability ($\alpha = .84$). The EDDS has demonstrated criterion-related and convergent validity (Stice et al., 2000).

Impulsivity. The Short Form of the UPPS-P Impulsive Behavior Scale (SUPPS-P; Cyders, Littlefield, Coffey, & Karyadi, 2014) is a 20-item self-report measure that assesses five facets of impulsivity, including positive urgency, negative urgency, lack of premeditation, lack of perseverance, and sensation seeking. Participants responded to items on a 4-point Likert scale. Reverse-scored items were recoded so that higher scores represented greater impulsivity. In the current study, responses to each item were averaged to formulate a composite of impulsivity (Liu et al., 2016). The SUPPS-P demonstrated good internal consistency reliability ($\alpha = .86$). Prior examination provides evidence for the validity of the SUPPS-P (Cyders et al., 2014).

Non-suicidal self-injury (NSSI). The Deliberate Self-Harm Inventory (DSHI; Gratz, 2001) consists of 16 yes/no items that assess lifetime incidence of specific

practices of NSSI, which result in tissue damage. An additional open-ended item asks respondents to describe any other lifetime practice self-harm that was not indicated in the previous items. A continuous variable of NSSI behaviors was created by totaling the number of self-harm behaviors endorsed (Fadoir, Lutz-Zois, & Goodnight, 2019), and items comprising this variable exhibited good internal consistency reliability (α = .84). The DSHI has demonstrated adequate measures of construct, convergent, and discriminant validity (Gratz, 2001).

Substance use. The Alcohol Use Disorders Identification Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) assesses alcohol use, symptoms of alcohol dependence and tolerance, and impairment related to alcohol consumption. This self-report measures consists of 10 items to which participants responded on a 5-point scale with higher scores suggesting more problematic alcohol use. The AUDIT demonstrated good internal consistency reliability (α = .90). Previous examination of the AUDIT provides evidence for its validity (e.g., Saunders et al., 1993).

The Drug Abuse Screening Test-10 (DAST-10; Skinner, 1982) is a self-report measure that assesses problematic substance use, excluding alcohol and tobacco use. Participants responded "yes" (scored 1) or "no" (scored 0) to 10 items inquiring about drug use in the past 12 months. Affirmative responses were summed to create a total score for each participant. The DAST-10 exhibited adequate internal consistency reliability (α = .79). The DAST-10 has demonstrated construct validity (e.g., McCann, Simpson, Ries, & Roy-Byrne, 2000; Skinner, 1982), convergent validity (e.g., Bedregal, Sobell, Sobell, & Simco, 2006), and predictive validity (e.g., McCann et al., 2000).

Procedure

Following informed consent, participants completed a battery of questionnaires, including the EDDS, CCMS, SUPPS-P, DSHI, AUDIT, and DAST-10. Participants accessed the survey online via MTurk and completed the measures using Qualtrics survey software (Qualtrics, 2015). Participants who completed the survey received $1.25 in compensation.

Statistical Analyses

A series of mediation analyses was used to examine Model 3, Model 4, and Model 5. Mediation analyses were conducted utilizing a bootstrapping approach within Mplus Version 8 (Muthén & Muthén, 2017). This bootstrapping method calculated 10,0000 samples and used bias-corrected bootstrap 95% confidence intervals (Preacher & Hayes, 2008). In Model 3, *CSA* represented the independent variable, *impulsivity* was the mediating variable, and *binge eating* represented the dependent variable. In Model 4, *CSA* represented the independent variable, *impulsivity* was the mediating variable, and *purging/restriction* served as the dependent variable. In Model 5, *CSA* represented the independent variable, and the mediating variable was *impulsivity*. *Binge eating*, *purging/restriction*, *NSSI*, *alcohol use*, and *drug use* served as the dependent variables. For all three models, bootstrapping analyses tested the indirect effects of CSA on all of the dependent variables through impulsivity.

Results

Preliminary Analyses

Approximately 22% of the sample (i.e., 96 participants) reported at least one experience of sexual abuse in childhood. Independent *t*-tests were performed comparing

the mediator and dependent variables' means (i.e., impulsivity, binge eating, purging/restriction, NSSI, alcohol use, and drug use) of the participants who endorsed at least one episode of CSA with those who denied a history of CSA. These descriptive statistics and the outcomes of the independent *t*-tests are reported in Table 8. For participants with a history of CSA, their mean scores on purging/restriction, impulsivity, NSSI, alcohol use, and drug use were significantly higher than those of participants' who denied episodes of CSA. While the mean score of binge eating was higher for participants who endorsed CSA than for those who did not, this difference was not statistically significant.

Table 8

Study 2 Descriptive Statistics Using t-test for Group Mean Differences

	No History of CSA		History of CSA		
	M	*SD*	*M*	*SD*	*t*-test
Binge eating	0.91	2.13	1.30	2.37	1.45
Purging/restriction	1.88	4.73	8.26	10.03	6.05***
Impulsivity	1.96	0.46	2.18	0.47	4.18***
NSSI	0.91	1.87	2.76	3.11	5.39***
Alcohol use	4.10	4.80	8.40	8.89	4.55***
Drug use	0.61	1.30	1.66	2.18	4.48***

Note. The means and standard deviations are noted separately for the participants with at least one episode of CSA and those without a history of CSA. ***$p < .001$.

Table 9 notes the bivariate Pearson correlations among all of the variables of interest. CSA was significantly correlated with all of the variables of interest, except binge eating. Binge eating was only significantly associated with impulsivity, NSSI,

alcohol use, and drug use. All remaining variables were significantly correlated with each other. Each of the correlations indicated positive relationships among the variables, and the size of the significant correlations ranged from low to moderate (*r*s = 0.10 to 0.56).

Table 9

Study 2 Bivariate Pearson Correlations

Variable	1.	2.	3.	4.	5.	6.	7.
1. CSA	-						
2. Binge eating	0.03	-					
3. Purging/restriction	0.56**	0.09	-				
4. Impulsivity	0.17**	0.17**	0.38**	-			
5. NSSI	0.19**	0.15**	0.18**	0.18**	-		
6. Alcohol use	0.45**	0.10*	0.44**	0.32**	0.26**	-	
7. Drug use	0.38**	0.10*	0.24**	0.28**	0.30**	0.43**	-

Note. Correlations are rounded to the nearest tenth. The CSA variable is derived from participants' scores on the CCMS Sexual Abuse subscale, indicating total number of CSA episodes. *$p < .05$. **$p < .01$.

Mediation analyses. In Model 3 (see Figure 3 and Table 10), results revealed a significant direct effect of CSA on impulsivity (a_3), indicating a significant association between CSA and impulsivity. The direct effect of impulsivity on binge eating (b_3) was significant, suggesting impulsivity was significantly associated with binge eating. The direct effect of CSA on binge eating (c'_3) was not significant, which suggests no significant association between CSA and binge eating when the indirect effect is held constant. The indirect effect of CSA on binge eating was significant (ab_3), indicating impulsivity mediated the relationship between these variables.

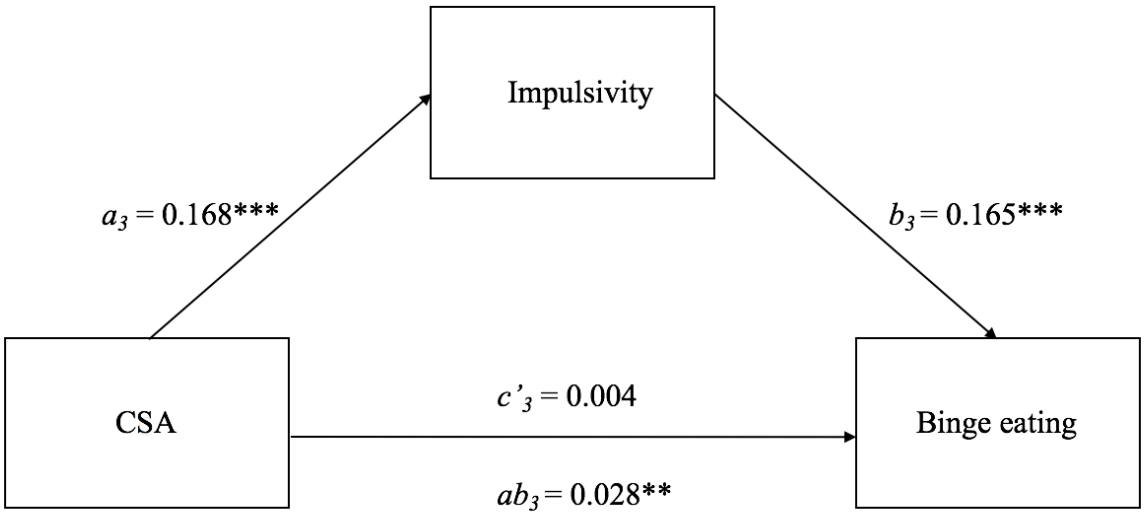

Figure 3. Model 3 standardized path coefficients. a_3 = direct effect of CSA on impulsivity. b_3 = direct effect of the impulsivity on binge eating. c'_3 = direct effect of CSA on binge eating. a_3 = indirect effect of CSA on binge eating through impulsivity. $^{**}p < .01$. $^{***}p < .001$.

Table 10

Model 3 Mediation Analysis

Effect	Coefficient	*SE*	*p*	Lower CI	Upper CI
a_3	0.168	0.040	<.001	0.101	0.231
b_3	0.165	0.045	<.001	0.090	0.238
c'_3	0.004	0.047	.933	-0.065	0.090
ab_3	0.028	0.010	.006	0.014	0.048

Note. All coefficients are standardized. CI = confidence interval. a_3 = direct effect of CSA on impulsivity. b_3 = direct effect of the impulsivity on binge eating. c'_3 = direct effect of CSA on binge eating. ab_3 = indirect effect of CSA on binge eating through impulsivity.

In Model 4 (see Figure 4 and Table 11), results indicated a significant direct effect of CSA on impulsivity (a_3), suggesting CSA and impulsivity were significantly related to each other. There was a significant effect of impulsivity on purging/restriction (b_4), indicating a significant association between impulsivity and purging/restriction. The direct effect of CSA on purging/restriction (c'_4) was significant, which suggests a significant relationship between CSA and purging/restriction when the indirect effect is held constant. The indirect effect of CSA on purging/restriction (ab_4) was significant, revealing that impulsivity mediated the relationship between these variables.

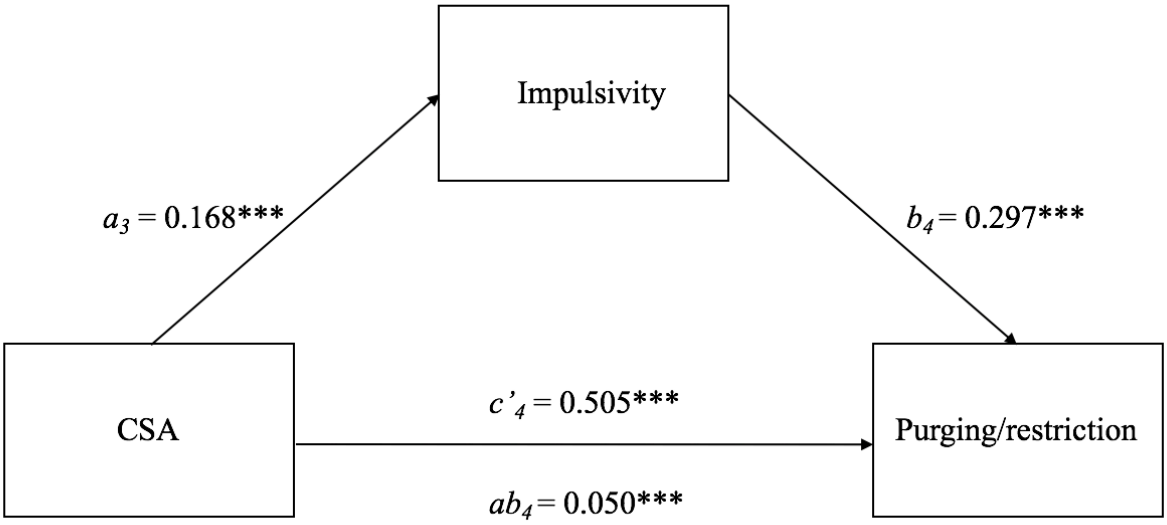

Figure 4. Model 4 standardized path coefficients. a_3 = direct effect of CSA on impulsivity. b_4 = direct effect of the impulsivity on purging/restriction. c'_4 = direct effect of CSA on purging/restriction. ab_4 = indirect effect of CSA on purging/restriction through impulsivity. ***$p < .001$.

Table 11

Model 4 Mediation Analysis

Effect	Coefficient	*SE*	*p*	Lower CI	Upper CI

Table 11 (continued).

a_3	0.168	0.040	<.001	0.101	0.231
b_4	0.297	0.040	<.001	0.230	0.362
c'_4	0.505	0.075	<.001	0.381	0.625
ab_4	0.050	0.012	<.001	0.031	0.072

Note. All coefficients are standardized. CI = confidence interval. a_3 = direct effect of CSA on impulsivity. b_4 = direct effect of the impulsivity on purging/restriction. c'_4 = direct effect of CSA on purging/restriction. ab_4 = indirect effect of CSA on purging/restriction through impulsivity.

In Model 5 (see Figure 5 and Table 12), results revealed a significant direct effect of CSA on impulsivity (a_3), indicating a significant association between CSA and impulsivity. The direct effects of impulsivity on binge eating (b_3), impulsivity on purging/restriction (b_4), impulsivity on NSSI (b_5), impulsivity on alcohol use (b_6), and impulsivity on drug use (b_7) were significant. These direct effects indicate significant relationships between impulsivity and binge eating, purging/restriction, NSSI, alcohol use, and drug use, respectively. The direct effects of CSA on purging/restriction (c'_4), CSA on NSSI (c'_5), CSA on alcohol use (c'_6), and CSA on drug use (c'_7) were significant. When indirect effects are held constant, the aforementioned direct effects suggest significant associations between CSA and purging/restriction, NSSI, alcohol use, and drug use, respectively. The direct effect of CSA on binge eating (c'_3) was not significant, which indicates no significant relationship between CSA and binge eating when the indirect effect is held constant. There were significant indirect effects of CSA on all the dependent variables (ab_3-ab_7), indicating impulsivity mediated the relationships between CSA and binge eating, purging/restriction, NSSI, alcohol use, and drug use.

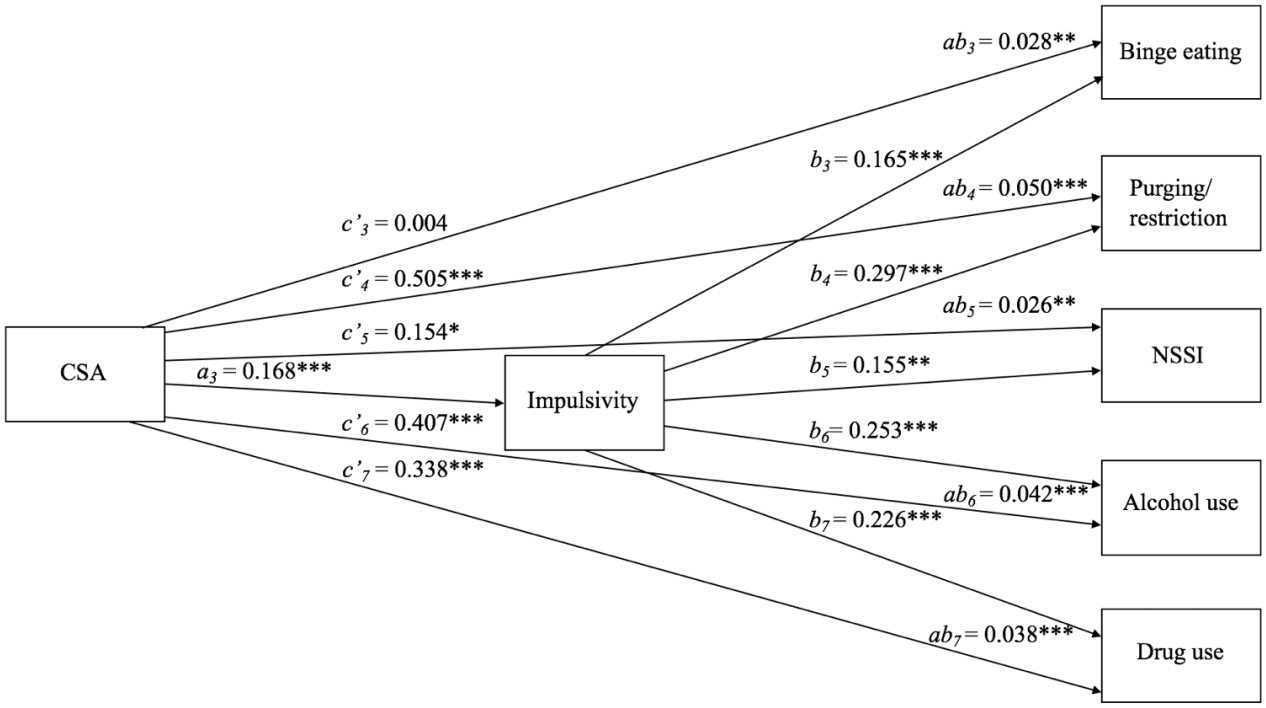

Figure 5. Model 5 standardized path coefficients. a_3 = direct effect of CSA on impulsivity. b_3, b_4, b_5, b_6, b_7 = direct effects of the impulsivity on the dependent variables. c'_3, c'_4, c'_5, c'_6, c'_7 = direct effects of CSA on the dependent variables. ab_3, ab_4, ab_5, ab_6, ab_7 = indirect effects of CSA on the dependent variables through impulsivity. *p < .05. **p < .01. ***p < .001.

Table 12

Model 5 Mediation Analysis

Pathway	Coefficient	*SE*	*p*	Lower CI	Upper CI
a_3	0.168	0.040	<.001	0.101	0.231
b_3	0.165	0.045	<.001	0.090	0.238
b_4	0.297	0.040	<.001	0.230	0.362
b_5	0.155	0.045	.001	0.082	0.228
b_6	0.253	0.040	<.001	0.185	0.317
b_7	0.226	0.039	<.001	0.164	0.293

Table 12 (continued).

c'_3	0.004	0.047	.933	-0.065	0.090
c'_4	0.505	0.075	<.001	0.381	0.625
c'_5	0.154	0.074	0.036	0.051	0.298
c'_6	0.407	0.071	<.001	0.283	0.517
c'_7	0.338	0.088	<.001	0.187	0.473
ab_3	0.028	0.010	.006	0.014	0.048
ab_4	0.050	0.012	<.001	0.031	0.072
ab_5	0.026	0.010	.009	0.012	0.046
ab_6	0.042	0.011	<.001	0.025	0.062
ab_7	0.038	0.010	<.001	0.022	0.057

Note. All coefficients are standardized. CI = confidence interval. a_3 = direct effect of CSA on impulsivity. b_3, b_4, b_5, b_6, b_7 = direct effects of the impulsivity on the dependent variables. c'_3, c'_4, c'_5, c'_6, c'_7 = direct effects of CSA on the dependent variables. ab_3, ab_4, ab_5, ab_6, ab_7 = indirect effects of CSA on the dependent variables through impulsivity.

CHAPTER IV

DISCUSSION

The primary aim of these investigations was to further elucidate the relationship between CSA and eating psychopathology. A myriad of literature provides evidence for a positive correlation between sexual abuse in childhood and disordered eating behaviors (e.g., Smolak & Murnen, 2002). However, much of the evidence base supporting this link has neglected to examine these variables prospectively in child and adolescent samples, thereby curbing conclusions that identify CSA as a possible risk factor for eating disorder symptomatology (Sanci et al., 2008; Stice, 2002). Furthermore, while some researchers have indeed asserted that CSA remains a non-specific risk factor for eating psychopathology (e.g., Jacobi et al., 2004), consensus regarding mechanisms that explain the association between these variables is lacking. Study 1 intended to address some of the methodological limitations in the existent literature. This study examined whether CSA predicts binge eating over two years in a sample of girls, while also testing the mediating influence of impulsivity and substance use. Unfortunately, due to poor psychometric properties of the KEDS, Study 1 was unable to investigate weight dissatisfaction and purging/restriction as outcomes of CSA. Utilizing a young adult sample of women and men, Study 2 endeavored to replicate findings from the proceeding literature and from Study 1 that suggest impulsivity mediates the relationship between CSA and eating psychopathology. As an additional aim, this second study explored a

conceptual model of CSA, impulsivity, eating psychopathology, and other deleterious behaviors.

Study 1

Binge Eating (GEE Model)

As indicated by the GEE model, while the frequencies of reported binge eating were greater in participants with a history of abuse than participants in the control group, these reports did not differ at a statistically significant level. This outcome diverges from this author's hypothesis and suggests that accounts of binge eating are not dependent on abuse status. Additionally, binge eating did not change over time nor did the interaction between abuse status and time affect participants' report of binge eating. Thus, when compared to girls without a reported history of sexual abuse, girls who have experienced CSA engage in similar frequencies of binge eating, and this relationship remains stagnant across two years.

These findings conflict with that of Sanci and colleagues (2008), who, at a 1-year follow-up, discovered a 2.5 increased risk of bulimic symptomatology in adolescents with a reported episode of CSA. However, this investigation has notable distinctions from Study 1, which may explain the contrasting results. For example, while Study 1 solely analyzed binge eating over time, Sanci and colleagues (2008) measured subthreshold BN, which characterizes additional eating psychopathology (e.g., purging behaviors and overconcern with weight and shape). Thus, in younger individuals, it is possible that sexual abuse in childhood does not predict binge eating in itself and that binge eating only represents a potential outcome of CSA when compounded with weight and shape dissatisfaction and/or purging symptoms. Additionally, while Sanci and colleagues

(2008) used a prospective design to measure eating disorder symptoms, participants did not endorse their histories of CSA until the age of 24, which introduces the possibility of recall bias and inaccurate accounts of childhood abuse. Study 1 circumvented this concern by recruiting youth participants and their parents or guardians, who could corroborate the incidence of CSA.

Comparing these findings to other preceding investigations presents as a challenge due to the limited prospective examinations of the influence of sexual trauma on eating psychopathology in adolescents. Longitudinal examinations of CSA and disordered eating in adult samples have often neglected to examine binge eating in isolation (e.g., Smyth et al., 2008; Vrabel et al., 2010) or have investigated full-threshold eating disorders (e.g., Dinwiddie et al., 2000). However, some evidence from a prospective design does suggest that adult women endorse similar levels of binge eating regardless of their histories of child sexual violence (Vogeltanz-Holm et al., 2000). Additionally, this non-significant finding contrasts with outcomes from cross-sectional studies of school children, such that children with a history of CSA endorsed elevated levels of binge eating compared to their peers with no reported sexual abuse (Ackard & Neumark-Sztainer, 2003; Chandy et al., 1996). Of note, these studies included both male and female participants and possessed substantially larger sample sizes than Study 1, which may have enhanced their representative quality. Therefore, while the nonsignificant outcomes of the GEE model provide unique insights into the CSA-binge-eating relationship, replication is essential in order to draw stronger conclusions regarding the longitudinal impact of CSA on adolescent binge eating.

Binge Eating and Impulsivity (Model 1)

As predicted, impulsivity mediated the relationship between CSA and binge eating in Model 1. The direct effect of CSA on impulsivity aligns with existing cross-sectional evidence of the impact of sexual abuse in childhood on the development of impulsive personality traits (Liu, 2019). Although the direct effect of impulsivity at Time 2 on binge eating at Time 3 was not significant, the strength of this effect and the associated confidence interval suggests this non-significant outcome may be the result of inadequate power to reject the null hypothesis. If the null hypothesis is truly false and the proceeding interpretation is accurate, Model 1 suggests that girls with a history of sexual abuse have an increased chance of developing impulsivity, which may augment their risk of binge eating 2 years later. Evidence of the predictive nature of impulsivity on adolescent binge eating exists in a community adolescent sample (Wonderlich et al., 2004). However, the inconsistencies regarding this relationship in the literature bear mentioning. Such inconsistent findings are likely the result of limited longitudinal designs and diversity in the measurement of impulsivity (Wonderlich et al., 2004). Regardless of the direct effect of impulsivity on binge eating, the model observed an indirect effect of CSA on binge eating, indicating that impulsivity is important in understanding how history of CSA may predispose adolescents to engage in binge eating. Thus, Model 1 adds to the scarcity of longitudinal designs employed to examine this relationship and represents the first prospective examination of impulsivity as a mediator of CSA and adolescent binge eating.

The cross-sectional mediation models demonstrated similar findings, such that impulsivity mediated the relationship between CSA and binge eating at Time 1 and at

Time 2, respectively. However, at Time 3, impulsivity did not emerge as a significant mediator among these variables. It may be that as time progresses, the combined effects of sexual abuse and impulsivity have less influence on adolescents' engagement in binge eating. Other pertinent factors, such as dietary restraint and negative affect (Mason, Smith, Lavender, & Lewis, 2018), may become more predictive of this eating disorder behavior. Investigating impulsivity, binge eating, and other suspected mediators at subsequent timepoints (i.e., beyond 2 years) is warranted to further trace and discern the pathway from CSA to binge eating.

Binge Eating and Substance Use (Model 2)

In contrast to the author's hypothesis, the results of Study 1 revealed substance use did not mediate the association between CSA and binge eating. Substance use also did not emerge as a significant mediator in the cross-sectional examinations of CSA and binge eating at all three timepoints. Further, while there was a direct effect of CSA on substance use at Time 2, substance use at Time 2 did not directly affect binge eating at Time 3. The latter finding resembles the work of Wonderlich and colleagues (2004), which demonstrated that substance use did not predict the subsequent onset of binge eating in a sample of adolescents. At the same time, this outcome conflicts with other prospective evidence, in which past 12-month substance use (i.e., predominantly marijuana use) predicted binge eating in adults at a 5-year follow-up (Vogeltanz-Holm et al., 2000). However, both of the aforementioned studies did not evaluate these variables in the context of CSA, which may have altered the substance use-binge-eating association. Thus, for CSA survivors, it may be that substance use does not necessarily predict binge eating but instead represents an alternative sequela of sexual abuse.

Alternatively, the temporal order of substance use and eating disorder behaviors in CSA survivors may depend on the specific eating disorder symptoms endorsed (e.g., binge eating only or dietary restriction and binge eating; Thompson et al., 2002).

Similar to eating disorder behaviors, researchers have postulated that individuals might utilize substances as a method of coping with distress related to their trauma histories (Briere & Scott, 2007; Ullman, Relyea, Peter-Hagene, & Vasquez, 2013; Polusny & Follette, 1995). In the case of adolescents, there is longitudinal evidence to support that CSA predicts substance use (e.g., Shin, Edwards, & Heeren, 2009). The non-significant indirect effect of substance use in the present study approximates work by Casper and Lyubomirsky (1997), which uncovered other variables (e.g., impulsivity) but not substance use that accounted for a sexual abuse-bulimic symptomatology link. Altogether, this evidence encourages the investigation of alternative mechanisms explaining binge eating as a possible outcome of CSA.

Study 2

Binge Eating and Impulsivity (Model 3)

Consistent with this author's hypothesis, impulsivity mediated the relationship between CSA and binge eating in the young adult sample. The direct effect of impulsivity on binge eating in this sample accords with proceeding cross-sectional research of the impulsivity-binge eating link in adults (e.g., Galanti et al., 2007). Model 3 resembles the indirect effect of sexual trauma in childhood on binge eating that Dworkin and colleagues (2014) identified in a sample of undergraduate women. This outcome also corroborates the longitudinal CSA-impulsivity-binge-eating mediation encountered in the adolescent sample of Study 1 (Model 1), suggesting that impulsivity is a pertinent mechanism in

understanding how binge eating develops from CSA both in adolescents and in young adulthood. Similar to Model 1, there was no significant direct effect of CSA on binge eating in Model 3. Thus, while individuals with a history of CSA, may have an increased likelihood of engaging in binge eating, this relationship may only be present if these individuals also possess impulsive temperaments. Indeed, psychobiological changes and alterations in neurotransmitter functioning (Putnam & Tricket, 1997) may increase CSA survivors' susceptibility to impulsive traits. Limited cognitive control and behavioral disinhibition characterize impulsivity; thus, individuals with these traits are at increased risk of participating in risk-taking behaviors (Skodol & Oldham, 1996). Binge eating may therefore serve as a strategy of reducing negative affect (Polivy & Herman, 1993) that emerges from their trauma histories.

Purging and Restriction Behaviors (Model 4)

As hypothesized, CSA was positively related to purging/restriction through impulsivity. This result corroborated the findings of Dworkin and colleagues (2014) who, with a sample of undergraduate women, demonstrated that impulsivity mediates the association between CSA and purging and restriction behaviors. Further, Wonderlich et al. (2001a) identified a similar mediation relationship in a sample of adolescents. This mediation relationship suggests that young adult CSA survivors may be at risk for engaging in dietary restriction and purging behaviors partially as a function of their impulsive tendencies. Such tendencies may especially be the case with purging pathology, as evidence suggests that purging symptoms, in comparison to binge eating, are significantly associated with impulsive behaviors (Favaro et al., 2005). Similar to the direct effect of impulsivity on purging and restriction behaviors in Model 4, prospective

investigation has also demonstrated that impulsive behaviors predict subsequent compensatory behaviors (Wonderlich et al., 2004). Given that sexual abuse inherently targets the body, survivors of CSA may observe their bodies with shame and disgust (Andrews, 1997). Already vulnerable to risk-taking behaviors, CSA survivors who experience bodily contempt may choose to utilize dietary restriction and purging methods, as the intended function of these techniques is to alter one's body (Dworkin et al., 2014). To this end, purging/restriction behaviors may represent one impulsive, coping technique toward which individuals with a history of CSA gravitate.

Impulsivity and Deleterious Behaviors (Model 5)

In the proposed exploratory model, impulsivity mediated the relationship between CSA and a variety of risky behaviors, including binge eating, purging/restriction, NSSI, alcohol use, and drug use. This model explains that encountering sexual abuse in childhood coincides with an enhanced susceptibility to developing impulsive personality traits. As a result, one's disposition is marked by sensation-seeking, limited premeditation and perseverance, and rash actions in the face of negative or positive affect (Cyders et al., 2014). With such a temperament, an individual is more prone to partake in multiple self-destructive behaviors that may serve as methods of escaping or coping with distress (Heatherton & Baumeister, 1991; Sprague & Verona, 2010). Additionally, while the direct effect of CSA on binge eating was not significantly different from zero, this finding was not the case for the remaining risky behaviors, all of which demonstrated significant pathways from CSA when the indirect effect was held constant. Thus, it appears that unlike binge eating, engaging in purging/restriction, NSSI, or substance use may be relevant, possible outcomes of CSA regardless of one's impulsive traits. Finally,

Model 5 suggests that, similar to Model 2, substance use and other risk behaviors may not necessarily predate eating disorder symptoms but rather might occur separately or comorbidly. This conceptualization supports conclusions from researchers who view CSA as a predictor of a multitude of negative psychological outcomes, not specific to eating disorders (e.g., Jacobi et al., 2004). Of course, due to the cross-sectional nature of this study, replication of this model in a longitudinal design is necessary to substantiate this interpretation.

Model 5 resembles a conceptualization proposed by Thompson and Wonderlich (2004). Thompson and Wonderlich postulated psychobiological dysregulation results from child sexual trauma and leads to the development of a myriad of psychopathology. In this model, eating psychopathology and other deleterious behaviors represent an outcome of multi-impulsivity (i.e., binge eating and various impulsive behaviors). However, unlike the model Thompson and Wonderlich described, findings of Study 2 emphasize impulsivity as an independent construct that explicates the relationship between child sexual violence and various harmful behaviors. Due to developmental limitations of adolescents based on self-awareness, cognition, or language, some researchers rely on inventories that assess behavioral expressions of impulsivity, which may require less introspection than personality trait measures. Subsequently, behavioral measurements of impulsivity may lend themselves to more accurate assessment in a younger age group (Shiner, 1998; Wonderlich et al., 2004). At the same time, representing impulsivity strictly in the form of behaviors may oversimplify or potentially mischaracterize this construct, as contemporary research is increasingly identifying impulsivity as multidimensional (e.g., Cyders, & Coskunpinar, 2011). Additionally, a

meta-analysis of the child maltreatment and impulsivity literature discovered that in studies utilizing general trait measurements of impulsivity, the magnitude of the CSA-impulsivity association did not differ depending on the age of the participants (Liu, 2019). Thus, modeling impulsivity as a temperament that increases an individual's likelihood of engaging in harmful behaviors may better delineate the risk pathway from CSA and may also create space for investigating other relevant personality traits as possible mediators.

Indeed, this model of CSA, impulsivity, and deleterious behaviors draws support from prior investigations of these variables of interest. Meta-analytic data corroborate that sexual abuse in childhood predisposes individuals to an impulsive temperament (Liu, 2019) and corresponds with engagement in NSSI (Liu, Scopelliti, Pittman, & Zamora, 2018) and substance use (Hillberg, Hamilton-Giachritsis, & Dixon, 2011). In addition to disordered eating, an abundance of literature exists, linking impulsivity to NSSI (Casillas, & Clark, 2002; Herpertz, Sass, & Favazza, 1997; Ogle & Clements, 2008), alcohol use (Lejuez et al., 2010; Moeller & Dougherty, 2002), and drug use (de Wit, 2009; Moeller & Dougherty, 2002). While some researchers have uncovered impulsivity's role in the connection between CSA and subsequent harmful behaviors (e.g., Oshri, Sutton, Clay-Warner, & Miller, 2015), much of these investigations have assessed overall childhood maltreatment or have employed divergent definitions and measures of impulsivity (Arens, Gaher, & Simons, 2012; Arens, Gaher, Simons, & Dvorak, 2014; Bailey & McCloskey, 2005; Wardell, Strang, & Hendershot, 2016). To the author's knowledge, this study represents the first to evaluate these variables in a cohesive mediation model, which elicits evidence for all five behaviors as outcomes of CSA through impulsivity.

General Conclusions

Despite, their divergent methodological and sample characteristics, Study 1 and Study 2 had a similar objective of elucidating the relationships between CSA, impulsivity, and binge eating. Upon scrutinizing these variables in mediation models, some noteworthy findings emerged. Study 1 and Study 2 provided cross-sectional evidence for the indirect effect of CSA on binge eating through impulsivity in adolescent girls and young adults. These findings further substantiate the hypothesis that impulsivity represents a mechanism through which CSA predicts binge eating. Study 1 further corroborates this claim by bestowing evidence for the mediating role of impulsivity in a longitudinal design. Moreover, both Study 1 and Study 2 failed to demonstrate a significant the direct effect of CSA on binge eating. Together, these findings underscore the importance of accounting for impulsivity when identifying binge eating as an outcome of CSA; otherwise, this relationship may be inconsequential.

However, as previously noted, Study 1 did not observe impulsivity as a significant mediator of CSA and binge eating in the cross-sectional model at Time 3. If indeed this finding implies that the combined effect of CSA and impulsivity on binge eating decreases over time, the CSA-impulsivity-binge-eating relationship in the adult sample of Study 2 is perplexing. This distinction may relate to the manner in which each study measured impulsivity. Study 1 elected to measure impulsive behaviors (e.g., shoplifting, sexual promiscuity, and suicidal thoughts), whereas Study 2 measured trait impulsivity. Perhaps the prevalence of engaging in impulsive behaviors increases with age, regardless if one is a survivor of CSA. Thus, as time progresses, other variables may develop or become more pertinent in increasing one's likelihood of engaging in binge eating.

Conversely, once impulsivity as a personality trait develops, it is possible this temperament may exist as a more stagnant mechanism in the CSA-binge eating relationship. Future investigations that incorporate multiple measures of impulsivity in various age groups may prove fruitful in better understanding this factor as it relates to sexual abuse and binge eating.

Limitations

The current studies are not without limitations, which are critical to consider when interpreting these findings. First, the MRFS-IV binge eating subscale demonstrated ambiguous internal consistency reliability at Times 1 and 2, which is important to recognize in the context of the nonsignificant findings of the GEE model. Additionally, unacceptable internal consistency reliability and across-time discrepancies from the KEDS data precluded Study 1 from testing the effects of CSA on weight dissatisfaction and purging/restriction. In future investigations of eating psychopathology in adolescent samples, researchers may consider taking supplemental steps to minimize the incidence of unreliable and invalid reporting (e.g., selecting measures with excellent reliability estimates, incorporating validity check items, etc.; Fan et al., 2006). Another limitation to acknowledge is that participants from Study 1 within the abused group were receiving treatment throughout the course of the study, which may have impacted their endorsement of psychopathology. Future studies may benefit from including measures that more closely monitor access and responses to ongoing therapy interventions. Furthermore, due to the small sample size in Study 1, the mediation analyses likely did not contain enough power to control for initial levels of binge eating, which may constrain interpretations of abuse status's effect through the examined mediators (Stice,

2002). However, the items of the binge eating subscale of the MRFS-IV request that participants endorse their frequency of binge eating "in the past year" (Shisslak et al., 1999). Therefore, while the mediation analyses did not statistically control for binge eating at Time 1, the subscale's instructions and the annual measurements may have protected against the possibility of statistical overlap at Time 3.

In Study 2, the cross-sectional design precludes the ability to determine temporal ordering of the variables. Thus, the results cannot accurately define CSA as a risk factor of eating psychopathology and other deleterious behaviors in young adults (Caslini et al., 2016). Furthermore, there are some constraints to drawing conclusions from the sample of adults from Study 2 who reported prior sexual victimization. Due to the distal nature of the abuse, it is possible other factors may have impacted the eating disorder behaviors and other risk behaviors reported at adulthood (Kenardy & Bell, 1998). Retrospective reports of CSA may also increase the chance of recall bias, thereby potentially depicting an inaccurate account of childhood maltreatment in the sample (Sanci et al., 2008). However, this study attempted to minimize the potential adverse effects of retrospective reporting of CSA by recruiting young adult participants. Finally, Study 2 collected data online in an uncontrolled environment via self-report measures. Such methodology predisposes the data to response bias and enables the potential for misinterpretation of scale items.

Future Directions

The outcomes of the current study present the opportunity for several future investigations. Most importantly, given the inconsistencies in the child maltreatment and eating disorder literature, reproducing these analyses is crucial 1) to further substantiate

the observed outcomes and 2) to reduce methodological heterogeneity in the literature. These replication studies may also benefit from including both trait and behavioral measures of impulsivity in order to more precisely discern this construct in the context of child sexual trauma and eating psychopathology. Furthermore, addressing the research questions of Study 1 in a larger, more gender-diverse sample may improve representation of all the intended outcome variables (i.e., binge eating, dietary restriction, and purging behaviors) and may remedy the restrictions placed on this study's analyses. The exploratory conceptual model produced in Study 2 also merits reproduction. Upon replication, examination of this model in a longitudinal design and in an adolescent sample, is paramount to draw causal conclusions about the development of eating disorder behaviors and other self-destructive actions in CSA survivors through impulsivity. Other conceptualizations of CSA and eating disorder symptomatology, such as those Thompson and Wonderlich (2004) postulated, likewise warrant further investigation.

CHAPTER V

CONCLUSION

The present investigation sought to further illuminate the impact of sexual abuse in childhood on eating disorder symptomatology and to uncover the mechanisms through which CSA predicts eating disorder behaviors and related psychological consequences. By evaluating a group of adolescent CSA survivors and an age-matched control group in a longitudinal design, Study 1 addressed methodological limitations that have plagued the literature. Additionally, this investigation provided evidence against binge eating as an outcome of CSA in adolescents unless these individuals also endorse impulsivity. In contrast, substance use did not emerge as a significant mediator through which CSA predicts binge eating. Using a nationally representative sample of young adult men and women, Study 2 cultivated additional evidence to support the positive relationship between CSA and purging/restriction behaviors. Moreover, this investigation originated support for a conceptual model that illustrates impulsivity as a possible pathway linking CSA and eating psychopathology, NSSI, and substance use.

The outcomes from these studies generate meaningful implications for eating disorder prevention efforts. Programs aimed at preventing eating psychopathology may consider tailoring their approaches for individuals with a history of sexual abuse in childhood. For example, CSA survivors participating in these programs may benefit from learning specific strategies to manage impulsive tendencies and employ adaptive coping

methods in response to distress. Acquiring such techniques as an adolescent or a young adult may inhibit dysregulatory processes and impede the development of eating disorder behaviors. Programs designed to prevent self-harm behaviors and substance use disorders may also consider incorporating similar approaches. Ultimately, these studies highlight the importance of understanding the nuances of the relationship between CSA and eating psychopathology across age groups, in order to best inform prospective research and clinical efforts.

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