

DISSERTATION

THE ROLE OF DEVELOPMENTAL TRAUMA IN DISORDERED EATING AND EXCESS
BODY WEIGHT IN ADOLESCENCE

Submitted by

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ABSTRACT

THE ROLE OF DEVELOPMENTAL TRAUMA IN DISORDERED EATING AND EXCESS BODY WEIGHT IN ADOLESCENCE

Psychological trauma and the human response to trauma have been characterized as one of the most important threats to public health. Developmental trauma, referring to traumatic events experienced during childhood and/or adolescence, is of particular concern given the potential biological, neurological, psychological, and relational impact of trauma on the developing child, with possible consequences lasting a lifetime. Youth may deal with developmental trauma by utilizing disordered eating behaviors like loss of control eating in efforts to cope with distress; loss of control eating can, in turn, cause excess weight gain. Adolescence is a particularly salient time for the initiation and maintenance of disordered eating, but it is unclear to what extent developmental trauma is associated with loss of control eating and excess weight in adolescence. Further, it is unknown what role developmental trauma has on affect and attachment, two developmental domains presumed to be affected by trauma, and how affect and attachment are associated with loss of control eating and excess weight gain in adolescence. This dissertation project utilized secondary data from a sample of adolescents at risk for excess weight gain, by having above-average body mass index (BMI ≥ 70 percentile for age and sex) or having a family history of overweight or obesity, to evaluate two aims. The first aim investigated associations between developmental trauma and disordered eating and developmental trauma and excess body weight, measured as BMI standard score, in adolescence. The second aim explored negative affect, measured as symptoms of depression and symptoms of

anxiety, and attachment, measured as emotional support, as mediators of the associations of developmental trauma with disordered eating and BMI standard score. Approximately 58% of the sample endorsed at least one traumatic event. Developmental trauma count was associated with greater depressive and anxiety symptoms and lower BMI standard score, but it was not significantly associated with global disordered eating or loss of control eating. An indirect effect of developmental trauma on global disordered eating via depressive symptoms was observed. Symptoms of anxiety and emotional support did not mediate the associations of developmental trauma with disordered eating or BMI standard score. Results highlight the unique role of depressive symptoms in the relationship of developmental trauma to disordered eating. If replicated, particularly with a longitudinal design, findings have the potential to inform prevention and intervention efforts for a particularly vulnerable population: youth who have experienced trauma and may be at risk for or are experiencing disordered eating and excess body weight.

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INTRODUCTION

Defining Trauma

The 2012-2013 Immediate Past President of the American Academy of Pediatrics, Dr. Robert W. Block, characterized trauma as the greatest unaddressed public threat facing our country today (Center for Youth Wellness, N.D.; CHILDREN NOW, N.D.). As many as two-thirds of children experience at least one traumatic event by the age of 18 years (Kilpatrick & Saunders, 1997). But what is this public health threat, what is trauma? Acknowledging the ongoing conversations about the complex and multi-dimensional nature and presentation of trauma and, by extension, the contradicting opinions presented within those larger conversations (for example, D'Andrea, Ford, Stolbach, Spinazzola, & van der Kolk, 2012; Friedman, Resick, Bryant, Strain, Horowitz, & Spiegel, 2011; Spitzer, First, & Wakefield, 2007), clinical trauma from a diagnostic perspective is the exposure to a traumatic event in which an individual experiences, witnesses, or is confronted with an event or events involving threatened or actual death or serious injury, or threat to the physical integrity of the individual or others. Following trauma, multiple symptoms can emerge and include re-experiencing the traumatic event(s), avoidance, and hyperarousal (American Psychiatric Association, 2000). Traumatic events have historically included experiences such as abuse (emotional, physical, and/or sexual), witnessing or experiencing serious interpersonal violence (including domestic violence), the unanticipated death of a loved one, war, ethnic cleansing and acts associated with terrorism, serious accidents, and natural disasters (Alisic, Zalta, Van Wesel, Larsen, Hafstad, Hassanpour, & Smid, 2014; Anda et al., 2006; Felitti et al., 1998; Scheeringa, Zeanah, & Cohen, 2011). It is critical to

understand the processes by which these events lead to sustained distress and ultimately adverse outcomes.

The term “developmental trauma” has been suggested to describe traumatic events that occur in childhood and adolescence that overwhelm an individual’s ability to integrate his or her experience and have the potential to negatively impact development in complex ways (Saakvitne, Gamble, Pearlman, Lev, 2000; van der Kolk, 2005). Proponents of this term posit that developmental trauma is more encompassing in that it not only reflects singular traumatic events, but also multiple or ongoing traumatic events, which present more frequently in clinical settings, thereby more accurately representing the diversity of trauma experienced during childhood and adolescence (Cloitre et al., 2009; Saakvitne et al., 2000; van der Kolk, 2005). Developmental trauma is used here to acknowledge the diversity and complexity of traumatic events experienced by children and adolescents during development. Despite the prevalence of developmental trauma and the growing recognition of its impact on physical and mental health, the role of developmental trauma in disordered eating and weight outcomes, such as excess body weight, in adolescence has not been clearly articulated. Adolescence is a developmental period in which disordered eating begins or becomes more severe and is a critical window for the trajectories of body weight. Both disordered eating and excess body weight put adolescents at risk for a host of complications and public health problems in adulthood (Mitchell & Crow, 2006; Reilly & Kelly, 2011).

Effects of Trauma

According to Dr. Bessel van der Kolk, Medical Director of the Trauma Center, Professor of Psychiatry at Boston University Medical School, and Co-Director of the National Center for Child Traumatic Stress Complex Trauma Network, “trauma, by definition, is unbearable and

intolerable...traumatic experiences...leave traces on our minds and emotions, on our capacity for joy and intimacy, and even our biology and immune systems” (van der Kolk, 2014, p. 1). Traumatic experiences or events are accompanied by stress. During childhood and adolescent development, our bodies are particularly sensitive to stress and its effects (Perry, Pollard, Blakley, Baker, & Vigilante, 1995). The Center on the Developing Child (2016) explains the precarious balance between enough stress, positive stress, in childhood to optimize development and too much stress, toxic stress, which is often the case in developmental trauma. Positive stress cultivates executive functioning including goal-directed behavior as well as self-regulation and resiliency, and establishes healthy and hearty immune responses. Toxic stress, a term which has been used interchangeably with traumatic stress, refers to the process by which the body, when threatened, responds with increased heart rate, blood pressure, and inflammatory reactivity, along with a cascade of stress hormones including cortisol and adrenaline (Center on the Developing Child, 2016; 2014). The continued activation of this stress response system results in wear and tear on the developing body and impacts brain structure, function, and plasticity such that the individual becomes hyper-reactive to the experience of stress (Center on the Developing Child, 2014). Prolonged exposure to toxic stress across development can manifest later in life as internalizing disorders including anxiety and depression; decreases in neurogenesis; learning and memory impairments; alterations in immune and hypothalamus-pituitary adrenal axis function – with decreases in immunity, increases in autoimmune diseases, and an eventual blunting or flattened response to acute stress sometimes observed; a doubled risk for obesity; and decreases in health-related quality of life (Anda et al., 2006; Corso, Edwards, Fang, & Mercy, 2008; Danese & Baldwin, 2017; Fuemmeler, Dedert, McClernon, & Beckham, 2009; Johnson, Riley, Granger, & Riis, 2013; Lupien, McEwen, Gunnar, & Heim, 2009; Olf & van Zuiden, 2017;

Shonkoff et al., 2012). The gravity of prolonged toxic stress during development and its effects on the individual throughout his or her later life are captured by Shonkoff and colleagues (2012): “Toxic stress can be viewed as the precipitant of a physiologic memory or biological signature that confers lifelong risk well beyond its time of origin” (p. e238).

Developmental trauma, which instigates a stress response in the developing body as previously described, has been theorized by Cook and associates (2005) to cause potential impairment in seven domains: (1) attachment – including difficulties with interpersonal awareness and interactions, social isolation, and perspective taking; (2) biology – including difficulties with sensorimotor development and somatization; (3) affect regulation – including difficulties with emotional regulation and communicating wants and needs; (4) dissociation – including alterations in states of consciousness, depersonalization, and derealization; (5) behavioral control – including difficulties with impulse control, disturbances in sleep and eating, and the maladaptive use of self-soothing behaviors; (6) cognition – including difficulties with executive functioning, planning and anticipation, language development, and even orientation to time and place; and (7) self-concept – including difficulties with developing a sense of self free from low self-esteem, shame, and/or guilt. The presumed impact of developmental trauma in childhood and adolescence on these seven domains constitutes the Developmental Trauma Model. **Figure 1** depicts this model and its primary assertion: that developmental trauma leads to a host of adverse physical and mental health outcomes through its complex impact on attachment, biology, affect regulation, dissociation, behavioral control, cognition, and self-concept. To understand better why these domains are assumed to be vulnerable to the effects of developmental trauma, it is essential to first understand the three principal ways that an

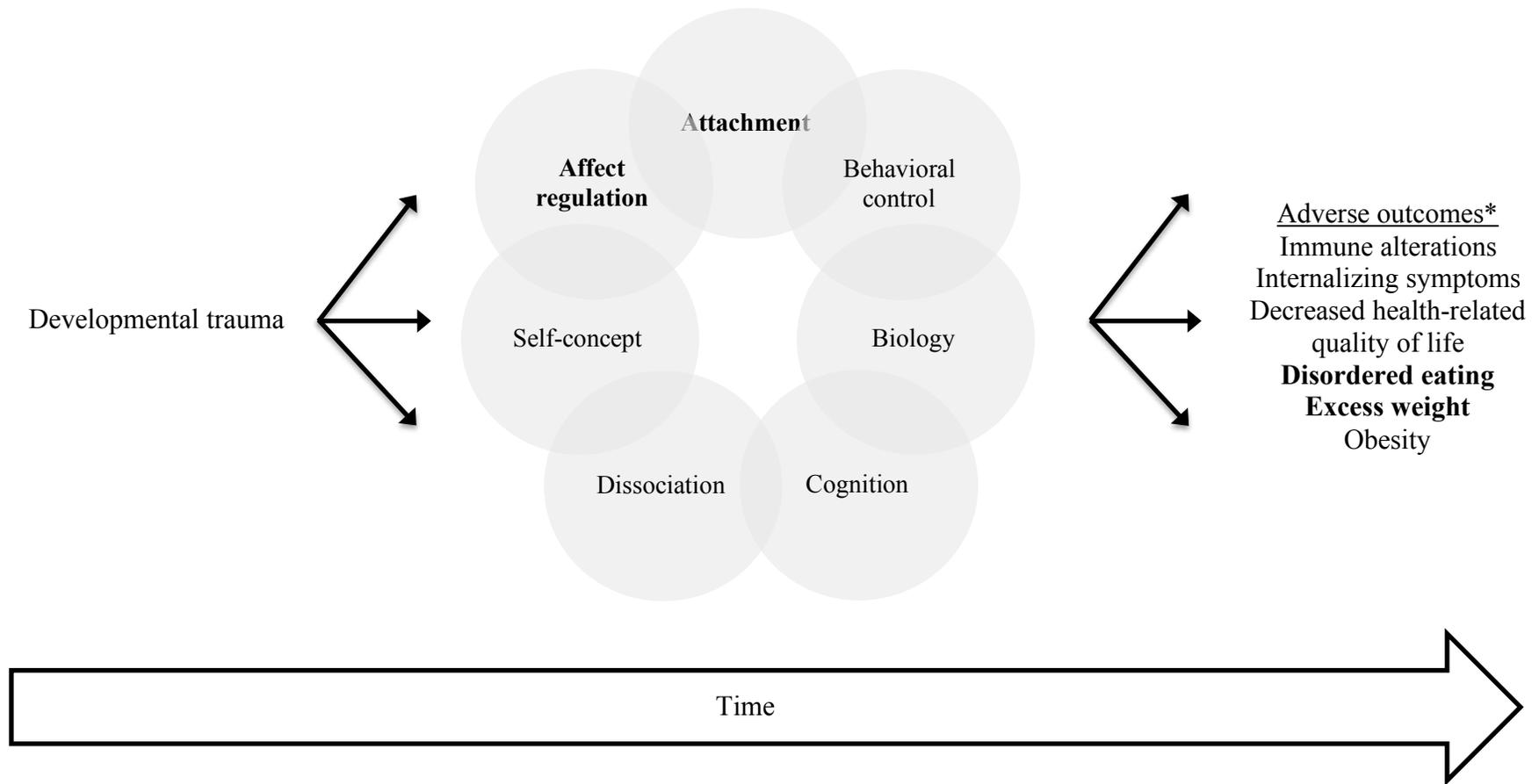


Figure 1
Theoretical Figure Guiding the Investigation

Notes: The asterisks attached to adverse outcomes is intended to acknowledge that many of these outcomes are inter-related.

individual can self-regulate during and after a traumatic event. Perry (2017) and van der Kolk (2014) provide an overview of regulation in trauma.

Trauma and Regulation

First, and perhaps the most primal, somatosensory regulation is used interchangeably with self-soothing and bottom-up regulation; these behaviors start in the womb, thus including sucking, and later manifesting as rhythmic motions that provide regulation for the body experiencing stress, including breathing, walking and running, rocking, doodling, humming and singing, and jumping and dancing (MacKinnon, 2012; Perry, 2017; 2009; 2008; van der Kolk, 2014, p. 3). Second, cortical modulation, or top-down regulation, develops over time and is specifically linked with the individual's cortical development; top-down regulation requires the individual to draw on more advanced cognitive processes like reframing and mindfulness (e.g., present-moment attention) techniques to process toxic stress (Perry, 2017; 2009; 2008; van der Kolk, 2014, p. 3). Ostensibly, these advanced processes are not typically accessible to children and some adolescents. Finally, dissociation, or in-out regulation, is the third way in which individuals can modulate their experiences during stress (Perry, 2017; 2009; 2008; van der Kolk, 2014, p. 3). Dissociation is considered a universal self-regulation mechanism in that all individuals, despite their development, can dissociate; in younger children, this process may include mind wandering, for example, and in adolescents and adults, this mechanism may include substances, medications, and/or disordered eating behaviors, like loss of control (LOC) eating (Heatherton & Baumeister, 1991), that are sought in an effort to numb oneself from fully experiencing the distress.

Disordered Eating and Overweight/Obesity

A review of cross-sectional, prospective, and retrospective data from both adolescent/young adult (for example, Fuemmeler et al., 2009; Hymowitz, Salwen, & Salis, 2017; Mamun, Lawlor, O'Callaghan, Bor, Williams, & Najman, 2007) and adult (for example, Anda et al., 2006; Armour, Müllerová, Fletcher, Lagdon, Burns, Robinson, & Robinson, 2016) samples suggests that developmental trauma increases the risk for overweight and obesity in adulthood. Overall, slightly more than two-thirds (69.0%) of adults have overweight or obesity (Ogden, Carroll, Kit, & Flegal, 2014). Further, preliminary retrospective evidence suggests that developmental trauma increases the risk for overweight and obesity even more immediately, in adolescence (Gooding, Milliren, Austin, Sheridan, & McLaughlin, 2015; Shin & Miller, 2012). During adolescence, nearly one-third of individuals (31.8%) have overweight or obesity, where overweight is defined as a body mass index (BMI) from the 85th and 94th percentiles for age and sex, and obesity is defined as a BMI at or above the 95th percentile (Ogden et al., 2014).

On a conceptual level, it would seem disordered eating behaviors, like LOC eating, may contribute to the increased overweight and obesity risk given: (i) the associations between LOC eating and excess weight gain (for example, Fairburn, Cooper, Doll, Norman, & O'Connor, 2000 and Tanofsky-Kraff, Yanovski, Wilfley, Marmarosh, Morgan, & Yanovski, 2004) and (ii) the potential use of LOC eating to self-regulate during and after traumatic events (Heatherton & Baumeister, 1991; Perry, 2017; 2009; 2008; van der Kolk, 2014, p. 3). LOC eating, referring specifically to the perception of overeating accompanied by a subjective feeling of lack of control over the type(s) or quantity of food consumed, is prevalent in adolescence with about one-quarter of non-treatment seeking youth endorsing some LOC eating and nearly 50% reporting LOC eating in adolescents seeking weight management (Tanofsky-Kraff et al., 2011;

Tanofsky-Kraff, Yanovski, & Yanovski, 2011). Cross-sectional, prospective, retrospective, and meta-analytic data indicate that disordered eating behaviors, primarily those distinguished by LOC, are associated with a greater likelihood of having experienced developmental trauma in a nationally representative sample of American adolescents (Fuemmeler et al., 2009), university students (Burns, Fischer, Jackson, & Harding, 2012; Hund & Espelage, 2006; Hymowitz et al., 2017; Kennedy, Ip, Samra, & Gorzalka, 2007), a nationally representative sample of English women (Armour et al., 2016), and 53 reviewed studies on the topic (Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). Further, a meta-analysis conducted on developmental trauma and long-term consequences found a three to five times increased risk of LOC eating phenotypes in individuals who experienced developmental trauma (Norman, Byambaa, De, Butchart, Scott, & Vos, 2012).

However, there is a significant limitation to this existing literature – retrospective designs; the work conducted to date is limited by a noteworthy time lag between traumatic events that may have been experienced in childhood and/or adolescence and the assessment of disordered eating and weight outcomes in early to mid-adulthood. In many studies, the time between developmental trauma and assessment of disordered eating and weight outcomes can be greater than two or three decades. This methodological limitation has led to concerns about recall and under-reporting because participants are tasked with reporting on events that may have occurred decades earlier (Fuemmeler et al., 2009; Gustafson & Sarwer, 2004). This limitation underscores the need for studies that assess disordered eating and weight outcomes earlier in the lifespan, in adolescence – much closer to the actual experience of developmental trauma events – to more precisely capture associations and articulate potential explanations for associations posited between developmental trauma and disordered eating and excess body weight in

adolescence. Indeed, a panel of experts in relevant fields have characterized the mechanisms linking developmental trauma to later disordered eating and weight outcomes as “not well understood” (Mason et al., 2016, p. 647).

Purpose of the Current Project

Accordingly, in this project, I aim to investigate associations of developmental trauma with disordered eating and with body weight in adolescence. Consistent with existing research conducted in early and mid-adulthood samples, and preliminary research in adolescent samples with regards to overweight/obesity, it is predicted that adolescents’ experiences of developmental trauma will relate to more disordered eating attitudes and behaviors, a greater odds of LOC eating in particular, and higher BMI. Secondly, I aim to examine, on an exploratory basis, plausible mediators of the associations of developmental trauma with disordered eating and body weight, wherein mediators examined will be two of the seven domains posited by the Developmental Trauma Model (Cook et al., 2005) to be impacted by traumatic events: affect regulation and attachment (**Figure 1**).

Theoretically-informed Mechanisms

The Developmental Trauma Model (Cook et al., 2005) specifies seven domains impacted by developmental trauma: attachment, biology, affect regulation, dissociation, behavioral control, cognition, and self-concept. While this model has been discussed in the scientific literature more than 1,000 times, to date, no test has been conducted to examine these domains as potential explanations for the observed association between developmental trauma and disordered eating and/or excess body weight, two of several diverse and inter-related adverse outcomes of developmental trauma. Referencing theories of disordered eating stands to inform

the selection of domains examined here as possible mechanisms: (i) affect regulation and (ii) attachment.

Affect regulation theory (Hawkins & Clement, 1984) proposes that increases in negative affect, including symptoms of depression and anxiety, lead to LOC eating in efforts to alleviate emotional distress; LOC eating in turn can lead to significant excess weight gain over time during childhood and adolescence (Tanofsky-Kraff, Yanovski, Schvey, Olsen, Gustafson, & Yanovski, 2009). Developmental trauma is associated with the development of depression and anxiety symptoms that may precipitate LOC eating behavior. In both youth and adults, a cumulative effect of developmental trauma on depression has been found such that as the number of traumatic events increase, so too does the severity of depressive symptoms (Felitti et al., 1998; Suliman, Mkabile, Fincham, Ahmed, Stein, & Seedat, 2009). While a significant cumulative effect of developmental trauma on anxiety has not been reported, those who experience any traumatic events in childhood and adolescence are more likely to endorse symptoms of anxiety as an adolescent and adult (Pine & Cohen, 2002; Suliman et al., 2009). A number of explanations have been offered for the associations observed between developmental trauma and symptoms of depression and anxiety including: the sensitizing effects of developmental trauma on the nervous system creating a unique vulnerability to internalizing symptoms later in life (Heim & Nemeroff, 2001), and insecure attachment such that developmental trauma can injure a child's attachment to his or her primary caregiver, which then is thought to result in increased affect dysregulation, decreased distress tolerance, and the inability to develop and maintain supportive relationships, all of which are thought to contribute to later risk for internalizing symptoms (Fowler, Allen, Oldham, & Frueh, 2013; Sroufe, Egeland, Carlson, & Collins, 2009). As such, both symptoms of depression and anxiety will be examined as mediators of the association of developmental

trauma with disordered eating and with excess body weight in adolescents. It is predicted that both symptoms of depression and anxiety will mediate the associations of developmental trauma with disordered eating and with excess body weight. The second component of that hypothesis is rooted in findings that depressive symptoms uniquely contribute to overweight/obesity onset prospectively in adolescent females (Blaine, 2008; Stice, Presnell, Shaw, & Rohde, 2005). Therefore, based on theoretical and empirical work, it is believed that developmental trauma will result in increased internalizing symptoms of depression and anxiety, which, in turn, will result in greater odds for LOC eating, increased disordered eating attitudes and behaviors, and excess body weight.

The interpersonal model (Ansell, Grilo, & White, 2012; Wilfley et al., 2002) of disordered eating expands on affect regulation theory in positing that interpersonal problems are a key precipitant of negative affect. Within this model, interpersonal problems are thought to lead to negative affect which, in turn, leads to LOC eating and excess weight gain. Individuals who experience developmental trauma differ from those who experience traumatic events as adults in that traumatic events during childhood and adolescence are associated consistently with interpersonal difficulties (Cloitre, Koenen, Cohen, & Han, 2002; Cloitre, Scarvalone, & Difede, 1997; D'Andrea et al., 2012). In childhood, attachment to a caregiver is critical for providing children the safety and security from which they can explore, learn, and socialize; secure attachment promotes affect and stress regulation, adaptability, resilience, and social relatedness (Bowlby, 2008; Cloitre et al., 2002; Rees, 2007). Developmental trauma can disrupt these processes resulting in dysregulation and interpersonal deficits. As children become adolescents, support, especially emotional support, emerges as a particularly important aspect of the safe haven component of the attachment behavioral system in relationships with parents, as well as

developing the ability to seek emotional support from peers (Allen et al., 2003; Chu, Saucier, & Hafner, 2010; Freeman & Brown, 2001). The same is true into adulthood during which time emotional support is considered “one of the most significant provisions of close relationships” (Burleson, 2003, p. 1). Emotional support is an expression of care and concern for another during times of distress from which we listen to, empathize with, legitimize, and actively explore the other person’s feelings along with encouraging, appreciating, reassuring, and validating (Burleson, 2003). Given: i) emotional support emerges as a key component of attachment in adolescence, ii) the salience of emotional support in relationships extends into adulthood, and iii) that developmental trauma can disrupt secure attachment which increases the likelihood of subsequent interpersonal deficits, emotional support will be examined as a mediator of developmental trauma and disordered eating and excess body weight in adolescents. It is predicted that emotional support will mediate associations between developmental trauma and disordered eating and between developmental trauma and excess body weight, because interpersonal difficulties in those with LOC eating and overweight/obesity have been documented (Arcelus, Haslam, Farrow, & Meyer, 2013; Duchesne, de Oliveira Falcone, de Freitas, D’Augustin, Marinho, & Appolinario, 2012; Tanofsky-Kraff et al., 2010). Thus, based on theoretical and empirical work, it is hypothesized that developmental trauma will decrease emotional support resulting in greater odds for LOC eating, increased disordered eating attitudes and behaviors, and excess body weight.

Importance of the Topic

Investigating associations between developmental trauma and disordered eating and weight outcomes and examining affect and attachment as plausible mechanisms between these associations is important for several reasons. First, this work directly addresses limitations of

past research by assessing disordered eating and weight outcomes much sooner in development than has been done historically, allowing for a more precise description of associations between these outcomes and traumatic events experienced in childhood and adolescence. While examining these associations in adolescence stands to address the temporal gap in events that limits past research, this approach also considers the vulnerability of adolescence as a developmental period in that many medical and psychiatric illnesses manifest and progress in adolescence including internalizing disorders, eating disorders, overweight and obesity, and other disease markers including insulin resistance as a precursor to type 2 diabetes (Cameron, Amin, De Beaufort, Codner, & Acerini, 2014; Giedd, Keshavan, & Paus, 2008; Steinberg, 2007; Tanofsky-Kraff et al., 2011). Second, it allows for a preliminary and exploratory test of the Developmental Trauma Model (Cook et al., 2005) in examining affect regulation and attachment as plausible mediators of associations of developmental trauma with disordered eating and with excess body weight. Finally, this study stands to inform intervention efforts (Mason et al., 2016) benefiting a particularly vulnerable population: those who experienced traumatic events in childhood and/or adolescence and those at risk for exacerbations of disordered eating and/or excess weight. Developmental trauma, disordered eating, and excess body weight are linked with a greater likelihood of many of the same diseases in adulthood including internalizing disorders like depression and anxiety, high blood pressure and cardiac disease, diabetes, arthritis, and autoimmune disorders (Goodwin & Stein, 2004; Mokdad et al., 2003; Versini, Jeandel, Rosenthal, & Shoenfeld, 2014). Accordingly, understanding the role of developmental trauma in disordered eating and body weight during adolescence may illuminate intervention targets, which in turn could stand to alleviate some of the burden that developmental trauma exerts on the individual and her or his overall health risk.

METHODS

Participants

The current dissertation project represents a secondary data analysis of an ongoing observational, longitudinal clinical trial to study eating behavior and excess weight gain in adolescence (ClinicalTrials.gov identifier: NCT03085160). Eighty-six adolescents ($M_{Age} = 14.25$, $SD = 1.68$ years), 42 males and 44 females, were included in analyses. On average, participants had overweight (Ogden et al., 2002) as represented by a mean BMI percentile of 92.50 ($SD = 7.65$). Adolescent participants mostly identified as non-Hispanic White (70.9%).

Procedure and Assessment

Participants were recruited via a variety of methods such as targeted mailings to local families with adolescents, recruitment flyers in schools, informational sessions held at local schools, community listserv emails, and social media postings. To be included, participants needed to be between the ages of 12 to 17 years and in good general health, other than at risk for excess weight gain either by current above-average BMI in the participant ($\geq 70^{\text{th}}$ percentile for age and sex) or overweight or obesity ($BMI \geq 25$ kilograms/meters²) in both biological parents. Full-syndrome psychiatric disorders that in the investigators' opinion would impede study compliance, major medical conditions, the use of medication(s) affecting mood or body weight, and pregnancy in females were exclusionary.

After obtaining informed parental consent and participant assent, participants completed a screening visit that consisted of body measurements after an overnight fast and the completion of questionnaires, interviews, and laboratory assessments. Additionally, parents completed questionnaires and an interview pertaining to their child's development and health. All study

procedures were approved by the Institutional Review Board at Colorado State University and adolescent participants were compensated \$60 for their time and effort.

The following measures were utilized to assess the variables of interest for this project, including traumatic events as the independent variable, disordered eating and excess body weight as outcomes, and depressive symptoms, anxiety symptoms, and emotional support as potential mediators.

Traumatic events (developmental trauma). The *Schedule for Affective Disorders and Schizophrenia for School-Age Children* (K-SADS; Axelson, Birmaher, Zelazny, Kaufman, & Gill, 2009) is a semi-structured diagnostic interview that was administered to adolescents by masters and doctoral students under the supervision of a licensed clinical psychologist to assess current and lifetime episodes of psychopathology. A brief (~ 10 minutes) introductory section was conducted first to establish rapport and obtain information about the school, home, and social settings. Then, affective, psychotic, anxiety, behavioral, and substance abuse disorders were assessed. Pertaining to the assessment of developmental trauma in the current project, participants were asked if they had ever experienced the following, even if only one time: car and other accident(s), fire, natural disaster, witness or victim of violent crime, confronted with traumatic news, terrorism-related trauma, war zone, witness domestic violence, physical abuse, and sexual abuse. If a participant endorsed one or more traumatic experiences, he or she was asked to choose the one experience that was most distressing. Using that experience, interviewers then queried about avoidance/numbing, re-experiencing, and hyperarousal. The K-SADS has been used extensively to assess traumatic events experienced in childhood and adolescence (for example, Alisic, Zalta, Van Wesel, Larsen, Hafstad, Hassanpour, & Smid, 2014; Deblinger, Mannarino, Cohen, Runyon, & Steer, 2011; Kiser, Donohue, Hodgkinson,

Medoff, & Black, 2010). In the present study, developmental trauma was coded dichotomously such that the report of a traumatic event, a car accident in which the participant was injured and needed urgent medical care for example, was coded 1 where denying all traumatic events was coded 0. Then, traumatic events endorsed were summed to create a continuous traumatic events score, consistent with prior work and alternate measures of trauma (Gutermann, Schreiber, Matulis, Schwartzkopff, Deppe, & Steil, 2016; Schnurr, Vielhauer, Weathers, & Findler, 1999; Weathers, Blake, Schnurr, Kaloupek, Marx, & Keane, 2013).

As multiple individuals administered the K-SADS, it was necessary to calculate interrater reliability or a measure of agreement among assessors (McHugh, 2012). Interrater reliability here refers to agreement between two K-SADS administrators. For example, if a participant reported witnessing a robbery at a gas station and both the administrator who conducted the K-SADS coded this experience as a traumatic event and a second, independent administrator blind to initial ratings coded this experience as a traumatic event, both administrators are in agreement as to the experiences being coded as traumatic events. The study investigator (Pivarunas) reviewed 10% of all K-SADS; the cases reviewed were determined by a random number generator. As previously stated, the study investigator was blind to initial ratings and first listened to K-SADS audio recordings to determine her own ratings before learning of the initial ratings. Two statistics were calculated, Cohen's kappa, κ , and percent agreement, because kappa is sensitive to prevalence such that when prevalence is higher, kappa is generally observed to be weaker than when prevalence is lower (Byrt, Bishop, & Carlin, 1993; Sim & Wright, 2005). Kappa was .24 ($p < .001$) and percent agreement was 95%. As almost 58% of participants endorsed at least one traumatic event, it would seem the relatively high prevalence rate affected the κ statistic and that ultimately assessors demonstrated a level of agreement that suggests the data are reliable.

Disordered eating. To assess LOC eating in the past month, the Overeating Section of the *Eating Disorder Examination* (EDE) version 12.0D (Fairburn & Cooper, 1993) was administered to participants by a trained masters or doctoral student interviewer. Consistent with prior studies (Field et al., 2003; Sonnevile, Horton, Micali, Crosby, Swanson, Solmi, & Field, 2013; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Tanofsky-Kraff et al., 2006), LOC eating was treated as a categorical variable; endorsing one or more objective or subjective LOC episodes in the month prior to assessment was coded as presence and denying any episodes of LOC eating was coded as absence. The *Eating Disorder Examination Questionnaire* (EDE/EDE-Q; Fairburn & Beglin, 1994), a 36-item self-report measure derived from the EDE interview, was also administered. The EDE-Q includes a global disordered eating attitudes average score, as well as four specific subscales including dietary restraint, eating concern, shape concern, and weight concern. In the current study, we utilized the global score as a representation of disordered eating attitudes and behavior; higher global scores indicate greater disordered eating attitudes and behavior. The global score has previously demonstrated the highest internal consistency in a diverse adolescent sample compared to the subscales and two alternative variations of the questionnaire (Burke et al., 2017). In previous child and adolescent samples, the EDE and EDE-Q have both demonstrated strong inter-rater reliability and discriminant validity for eating episodes, attitudes and behaviors, respectively (Carter, Stewart, & Fairburn, 2001; Glasofer et al., 2007).

Body measurements. Height without shoes was obtained with a stadiometer, in triplicate and then averaged, and weight with a calibrated scale. Weight was measured in the morning following an overnight fast. Height and weight were used to compute body mass index (BMI;

kilograms/meters²). Standardized BMI scores for age and sex were determined according to the Centers for Disease Control and Prevention 2000 standards.

Depressive symptoms. The *Center for Epidemiological Studies Depression Scale* (CES-D; Radloff, 1977) is a 20-item questionnaire that assesses depressive symptoms in the past week on a four-point Likert scale (rarely or none of the time to most or all of the time). The CES-D was administered to measure symptoms of depression. Representative items include, “I felt depressed” and “I had crying spells.” Total score is calculated as the sum of all items, four items are reverse scored, and the total score ranges from 0-60 with higher scores indicating greater depressive symptoms. The scale has demonstrated adequate psychometric properties in adolescent samples (Radloff, 1991).

Anxiety symptoms. The *State-Trait Anxiety Inventory for Children* (STAI-C; Spielberger & Edwards, 1973) *Trait Version* is a 20-item questionnaire designed to measure trait anxiety (“I worry too much” and “I am secretly afraid;”) in youth. Trait anxiety refers to anxiety symptoms experienced over time rather than anxiety experienced in real-time as participants complete questionnaires, which is typically considered state anxiety (Endler & Kocovski, 2001; Spielberger, 1966). The total score is calculated as the sum of all items, with a possible range of 20-60 with higher scores indicating greater trait anxiety. The STAI-C has demonstrated internal and test-retest reliability along with construct and concurrent validity in adolescents (Myers & Winters, 2002).

Emotional support. Emotional support was assessed with a questionnaire developed and presented by the National Institutes of Health (NIH Toolbox; Gershon, Cella, Fox, Havlik, Hendrie, & Wagster, 2010). Seven questions such as, “I have someone to talk with when I have a bad day” and “I have someone who understands my problems” are responded to using a five-

point Likert scale (never to always). The total score is calculated as the sum of all items. Scores from 7-35 are possible with higher scores indicating greater emotional support. Raw summed scores are then converted to the NIH's recommended, uncorrected *t*-scores for group analyses. Existing psychometric data on the scale include strong internal reliability ($\alpha = .91$) in a sample of youth (Salsman et al., 2013).

Data Analytic Plan

Power. To ascertain the appropriate sample size necessary to detect meaningful effects, power calculations were conducted. Referencing studies conducted on developmental trauma and disordered eating during adolescence (Burns et al., 2012) and developmental trauma and overweight/obesity during late adolescence and young adulthood (Mamun et al., 2007), which found small effects (Cohen's $d = .36$ and $.33$ respectively), it was determined that a small effect (Cohen, 1988) could be found with power ($1 - \beta$ error probability) set at $.95$ and $\alpha = .05$ in a sample of 60 participants. This analysis was conducted with G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009) and accounted for three planned covariates: age, sex, and race.

Data preparation. Data were checked for missing values. Skew and kurtosis were examined for all variables to ensure each approximated a normal distribution. Outliers were adjusted to 1.5 times the interquartile range below the 25th percentile or above the 75th percentile – to the whiskers in Tukey's (1977) boxplot. Following these steps, descriptive statistics including mean and standard deviation were calculated. For traumatic events and LOC eating, frequency of each was examined. Correlations among variables were examined. Finally, independent samples *t*-tests were performed to describe differences between groups (traumatic events versus no traumatic events) with respect to LOC eating, global disordered eating attitudes, standardized BMI, depressive symptoms, anxiety symptoms, and emotional support.

Aim 1. Aim 1 was to investigate associations between developmental trauma and disordered eating and between developmental trauma and body weight. Three models were examined; in all models, developmental trauma was treated as a continuous variable with higher values representing more traumatic events. In model 1, a logistic regression model, traumatic events were regressed on LOC eating in the past 28 days. LOC eating was coded such that “0” represents no LOC eating in the past 28 days and “1” represents ≥ 1 LOC eating in the past 28 days. In model 2, a linear regression model, traumatic events were regressed on global disordered eating attitudes global score as captured by the EDE-Q; global disordered eating attitudes score was treated as a continuous variable. In model 3, a linear regression model, traumatic events were regressed on BMI *z*-score.

Aim 2. Aim 2 was to explore depressive symptoms, anxiety symptoms, and emotional support as mediators of the associations of developmental trauma with LOC eating, global disordered eating attitudes, and body weight. Three primary mediation models were tested. Mediation model 1 examined traumatic events on LOC eating in the past 28 days as mediated by: depressive symptoms, trait anxiety, and emotional support. Mediation model 2 examined traumatic events on global disordered eating attitudes as mediated by: depressive symptoms, trait anxiety, and emotional support. Mediation model 3 examined traumatic events on standardized BMI as mediated by: depressive symptoms, trait anxiety, and emotional support. Standard errors for indirect effects in these models were bootstrapped (10,000 samples).

Analyses were performed in SPSS following the procedures described by Hayes (2013). PROCESS, a syntax file which allows for the examination of mediation, was used to assess mediation models (Hayes, 2013). Historical approaches (for example, Baron & Kenny, 1986) to mediation posit an association between the independent and dependent variable as a precondition

before proceeding with an exploration of mediation – how the independent variable influences the dependent variable. More recently, several shortcomings have been elucidated in this approach (Hayes, 2013). Instead, the goal of mediation analysis in conditional process modeling is to establish the extent to which some independent variable, X, is associated with some proposed outcome, Y, through one or more mediator or intervening, M, variables (Hayes, 2012, p. 1). Conditional process modeling facilitates an examination of a direct effect, the independent variable's association with the dependent variable (X on Y) considering no change in a mediating variable, and an indirect effect, the independent variable's association with a mediating variable and the mediating variable's relationship with the dependent variable (X on Y through M) (Hayes, 2012). PROCESS employs conditional process modeling to parse apart the direct effect and, importantly, the indirect effect without presupposing an association between the independent and dependent variables (Hayes, 2013; 2012). As historical approaches to mediation are not based on the quantification of the indirect effect, mediation is considered qualitatively (none, partial, or complete) thereby making it more difficult to investigate nuanced questions about the size of effects for different mediators. Consequently, provided the goal of this study is to evaluate mediators, as they may be associated with traumatic events and disordered eating and excess body weight, conditional process modeling is a more appropriate approach to investigating mediation. PROCESS can accommodate simple and multiple linear regression as well as logistic regression analyses.

Covariates. Aim 1 investigated associations between traumatic events and disordered eating and between traumatic events and body weight via two linear regression models and one logistic regression model. For all models, hierarchical modeling was used such that three covariates, sex, race/ethnicity (Non-Hispanic versus Hispanic), and age, were first regressed on

each dependent variable. Then, traumatic events were regressed on each dependent variable to examine the unique effect of traumatic events on each dependent variable accounting for age, sex, and race. To clarify the process of accounting for covariates, aim 1's first model is utilized as an example to illustrate the analytical steps. In model 1, a logistic regression model, traumatic events were regressed on LOC eating status. In step one, age, sex, and race were regressed on LOC eating. In step two, traumatic events were regressed on LOC eating. This procedure is described by Cohen, Cohen, West, and Aiken (2003). The same covariates were accounted for in analyses pertaining to aim 2.

RESULTS

Data Cleaning and Preliminary Analyses

Data were examined for normality. BMI z -score and traumatic events were excessively kurtotic. As a result, BMI z -score and traumatic events were Winsorized by recoding extreme, but plausible outliers to the whiskers in Tukey's (1977) boxplot. One high outlier was affected by this adjustment for traumatic events. Two low outliers were affected by this adjustment for standardized BMI z -score; the corresponding BMI percentiles for these two cases were also trimmed accordingly. After these adjustments, all variables approximated a normal distribution.

Eighty-six adolescents ($M = 14.25y$, $SD = 1.68y$) participated in the study. Participants were almost evenly split between males ($n = 42$) and females ($n = 44$). A majority of participants (70.9%) were non-Hispanic White. The remainder were Hispanic (29.1%). Participants had a mean BMI z -score of 1.67 ($SD = 0.56$). More than half (57.4%) endorsed at least one traumatic event. Being confronted with traumatic news ($n = 33$; 38.4% of all participants in the sample) was the most frequently endorsed traumatic event followed by car ($n = 16$; 18.9%) and other ($n = 14$; 16.2%) accidents, witnessing a disaster such as a tornado or flood ($n = 8$; 9.5%), witnessing a violent crime ($n = 8$; 9.5%), and witnessing domestic violence ($n = 8$; 9.5%). In unadjusted, descriptive analyses comparing adolescents with and without any exposure to developmental trauma, participants who endorsed at least one traumatic event had a lower BMI z -score ($M = 1.51$, $SD = .62$ vs. $M = 1.81$, $SD = .43$, $t = -2.27$, $p < .05$) than adolescents who reported no traumatic events. Those who had at least one traumatic event also reported greater depressive symptoms ($M = 14.28$, $SD = 7.55$ vs. $M = 9.48$, $SD = 6.03$, $t = 2.86$, $p < .01$) and anxiety symptoms ($M = 35.45$, $SD = 9.51$ vs. $M = 31.40$, $SD = 6.13$, $t = 2.20$, $p < .05$) than adolescents

with no developmental trauma history. Those endorsing at least one traumatic event did not significantly differ from those without any developmental trauma history on global disordered eating or emotional support ($ps > .11$). The prevalence of LOC eating among those with a developmental trauma history (15.4%) compared to those without a developmental trauma history (6.2%) did not significantly differ ($\chi^2 = .53, p > .45$). Descriptive information for LOC eating, global disordered eating attitudes, BMI, depressive and anxiety symptoms, and emotional support are presented in **Table 1**. Correlations among key variables are presented in **Table 2**.

Analyses Evaluating Aim 1

Three hierarchical regression models were examined to investigate associations between developmental trauma (as a continuous variable) and LOC eating presence, developmental trauma and global, continuous disordered eating, and between developmental trauma and continuous BMI z-score. The order of entry into each regression model was as follows. First, the effects of age, sex, and race/ethnicity on LOC eating, disordered eating, and BMI z-score were examined. Then, continuous number of traumatic events was added to each model.

The overall logistic regression model for LOC eating, including all covariates and number of traumatic events, can be seen in **Table 3**. The overall model was not significant, $\chi^2 (4) = 8.78, p > .06$. Controlling for covariates, the addition of traumatic events to the model predicting LOC eating increased the variance accounted for in LOC eating status by a non-significant 3% ($p > .06$). Traumatic events did not significantly predict LOC eating status (Exp. $\beta = 1.35, p > .22$). With respect to demographic factors, adolescents' age did significantly ($p < .05$) predict LOC eating status; controlling for the effects of sex and race/ethnicity, for every one year increase in age, there was a 45% increase in the likelihood of reporting LOC eating. This effect

was slightly attenuated when accounting traumatic events were also included in the model ($p = .05$).

The overall multiple regression equation for global disordered eating attitudes, including all covariates and number of traumatic events, was significant, $F(4, 67) = 2.85, p < .05$ (**Table 4**). The covariates and developmental trauma explained a combined 15% of the variance in global disordered eating attitudes. Traumatic events, however, did not significantly contribute ($t = 0.49, p > .62$) to the prediction of global disordered eating attitudes. In terms of covariates, adolescents' age significantly contributed to the prediction of global disordered eating. Controlling for the effects of sex and race/ethnicity, for every one year increase in age, there was a .14 unit increase in global disordered eating score ($t = 2.09, p < .05$); this association remained significant after accounting for traumatic events ($t = 2.09, p < .05$).

The overall multiple regression equation for BMI z -score, including all covariates and number of traumatic events, was significant, $F(4, 59) = 3.74, p < .01$, and accounted for 20% of the total variance in BMI z -score (**Table 5**). Traumatic events contributed significantly ($t = -2.15, p < .05$) to the prediction of BMI z -score. Controlling for the effects of age, sex, and race/ethnicity, for every one-unit increase in traumatic events, there was a -.13 unit decrease in BMI z -score; traumatic events accounted for a significant 6% of added variance in BMI z -score ($p < .01$). In terms of covariates, sex contributed significantly to the prediction of BMI z -score such that controlling for the effects of race/ethnicity and age, females had a -.27 unit lower BMI z -score than males in this sample ($t = -2.11, p < .05$); sex remained a significant predictor of BMI z -score when also including traumatic events in the model ($t = -2.15, p < .05$). Further, race/ethnicity contributed significantly to the prediction of BMI z -score such that controlling for the effects of age and sex, Hispanic participants had a .28 unit higher BMI z -score than non-

Hispanic White participants ($t = 2.05, p < .05$); race/ethnicity remained a significant predictor of BMI z -score when traumatic events were included in the model ($t = 2.35, p < .05$).

Analyses Evaluating Aim 2

Nine mediation models were examined to explore depressive symptoms, anxiety symptoms, and emotional support as possible mediators of the associations of developmental trauma with the three outcomes of interest: LOC eating status, global disordered eating attitudes, and BMI z -score (**Figure 2**). Models were examined both without and with covariates and all of these results are summarized in **Tables 6-8**.

There were no significant direct or indirect effects for the association of developmental trauma with LOC eating status, with or without adjusting for covariates (**Table 6**). With respect to global disordered eating, traumatic events were positively associated with depressive symptoms (path $a = 1.83, p < .01$) and, in turn, depressive symptoms were positively associated with global disordered eating attitudes (path $b = 0.06, p < .01$; **Table 7**). Depressive symptoms served as a significant mediator of the association between traumatic events and global disordered eating attitudes, as evidenced through a significant indirect effect of traumatic events on global disordered eating attitudes through depressive symptoms (path $ab = 0.12, p < .05$). The indirect effect of traumatic events on global disordered eating attitudes through depressive symptoms was slightly attenuated after controlling for covariates ($p > .08$).

No significant indirect effects were found for BMI z -score, with or without adjusting for covariates (**Table 8**). However, even when accounting for covariates, it is noteworthy that traumatic events retained a direct, inverse association with BMI z -score in this cohort, even independent of depressive symptoms (path $c' = -0.13, p < .05$), anxiety symptoms (path $c' = -0.14, p < .05$), or emotional support (path $c' = -0.18, p < .05$).

Table 1
Descriptive Information about Study Participants

Variable	M (SD), Range	Frequency Statistics
Sex	---	Males, <i>n</i> = 42 (48.83%) Females, <i>n</i> = 44 (51.17%)
Age, years	14.25 (1.68), 12.01 - 17.53	---
BMI percentile (for age and sex)	92.50 (7.65), 62 - 99	---
BMI z-score (for age and sex)	1.67 (0.56), 0.22 - 2.64	---
Trauma count	---	0 events, <i>n</i> = 31 (42.60%) 1 event, <i>n</i> = 19 (26.00%) 2 events, <i>n</i> = 15 (20.50%) 3 events, <i>n</i> = 5 (6.80%) 4 events, <i>n</i> = 3 (4.10%)
Depressive symptoms	12.55 (7.29), 0 - 29	---
Anxiety symptoms	34.41 (8.48), 20 - 57	---
Emotional support total score	27.11 (6.41), 11 - 35	---
Emotional support <i>T</i> -score	46.88 (9.58), 26.90 - 64.20	---
≥1 LOC eating episode (past 28 days)	---	Absence, <i>n</i> = 56 (77.78%) Presence, <i>n</i> = 16 (22.22%)
Eating restraint	1.97 (1.05), 1.00 - 6.00	---
Eating concern	1.90 (0.95), 1.00 - 4.60	---
Shape concern	3.10 (1.40), 1.00 - 6.00	---
Weight concern	3.01 (1.39), 1.00 - 6.00	---
Global disordered eating attitudes	2.50 (0.99), 1.00 - 4.99	---

Notes: Trauma count (K-SADS) could range from 0-12. Depressive symptoms (CES-D) could range from 0-60. Anxiety symptoms (STAI-C) could range from 20-60. Emotional support (NIH Toolbox) could range from 7-35 for total score and 19.5-64.2 for uncorrected *T*-score. Eating restraint, eating concern, shape concern, weight concern, and global disordered eating attitudes (EDE-Q) could all range from 0-6. Higher scores indicate greater depressive and anxiety symptoms, emotional support, and disordered eating.

Table 2
Correlations among Key Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Age, years	---											
2. Trauma count	.09	---										
3. LOC eating, presence	.26*	.21	---									
4. Eating restraint	.15	.15	.27*	---								
5. Eating concern	.14	.27*	.37**	.38**	---							
6. Shape concern	.24*	.08	.32**	.32**	.71**	---						
7. Weight concern	.29*	.10	.35**	.38**	.70**	.89*	---					
8. Global disordered eating	.26*	.14	.39**	.60**	.83**	.92**	.93**	---				
9. BMI z-score	.20	-.19	.22	.28*	.21	.25*	.36**	.33**	---			
10. Depressive symptoms	.15	.31**	.23	.12	.41**	.44**	.34**	.40**	.21	---		
11. Anxiety symptoms	.11	.24*	.12	.16	.43**	.60**	.51**	.53**	.12	.74**	---	
12. Emotional support	.00	-.10	-.04	.02	-.07	-.03	-.02	-.03	-.17	-.35**	-.26*	---

Notes: LOC eating=loss of control eating, coded as 0 = absence, 1 = presence of at least one subjective or objective binge eating episode in the past 28 days.

* $p < .05$

** $p < .01$

Table 3

Estimates for Logistic Regression Predicting Loss of Control (LOC) Eating Presence

	Model 1 ($\Delta R^2 = .17$)		Model 2 ($\Delta R^2 = .20$)	
	<i>B (SE)</i>	Exp. β	<i>B (SE)</i>	Exp. β
Sex	-1.02 (.68)	.36	-.95 (.69)	.39
Race	.45 (.69)	1.57	.35 (.70)	1.41
Age, years	.37 (.18)*	1.45*	.36 (.19)	1.43
Trauma count			.30 (.25)	1.35

* $p < .05$

Notes: Sex was coded such that males were assigned 0 and females assigned 1. Race/ethnicity was also coded such that Non-Hispanic White or Caucasian was assigned 0 and Other 1.

Table 4

Unstandardized and Standardized Estimates for Multiple Linear Regression Predicting Global Disordered Eating

	Model 1 ($\Delta R^2 = .12^*$)		Model 2 ($\Delta R^2 = .15^*$)	
	<i>B (SE)</i>	β	<i>B (SE)</i>	β
Sex	.35 (.21)	.18	.42 (.23)	.21
Race	.33 (.23)	.15	.35 (.25)	.16
Age, years	.14 (.06)*	.24*	.14 (.07)*	.24*
Trauma count			.05 (.10)	.06

* $p < .05$

Notes: Sex was coded such that males were assigned 0 and females assigned 1. Race/ethnicity was also coded such that Non-Hispanic White or Caucasian was assigned 0 and Other 1.

Table 5

Unstandardized and Standardized Estimates for Multiple Linear Regression Predicting BMI z-score

	Model 1 ($\Delta R^2 = .14^*$)		Model 2 ($\Delta R^2 = .20^{**}$)	
	<i>B (SE)</i>	β	<i>B (SE)</i>	β
Sex	-.27 (.13)*	-.24*	-.28 (.13)*	-.25*
Race	.28 (.14)*	.24*	.34 (.14)*	.28*
Age, years	.06 (.04)	.19	.08 (.04)	.23
Trauma count			-.13 (.06)*	-.26*

* $p < .05$ ** $p < .01$

Notes: Sex was coded such that males were assigned 0 and females assigned 1. Race/ethnicity was coded such that Non-Hispanic White or Caucasian was assigned 0 and Other 1.

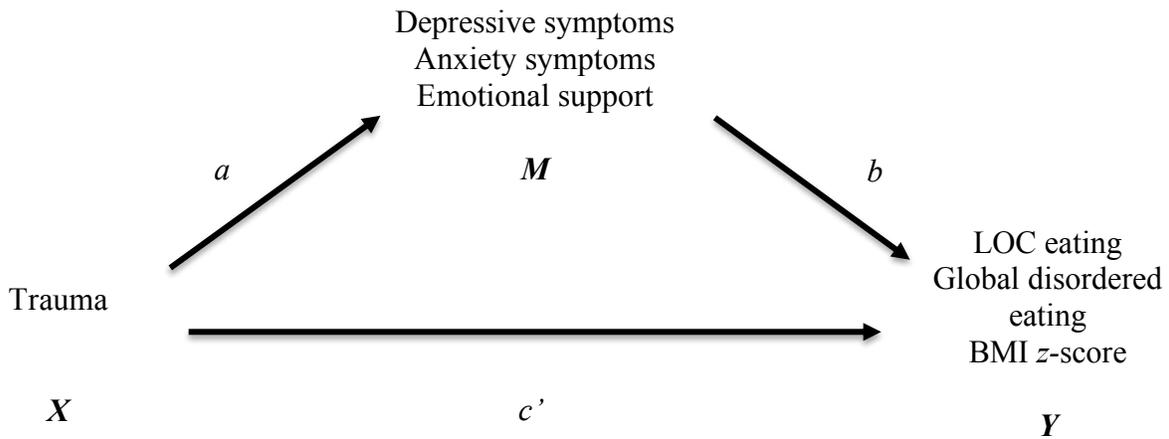


Figure 2
Mediation Figure Depicting the Tested Models Where Trauma was the Independent Variable, Depressive and Anxiety Symptoms and Emotional Support the Mediators, and Loss of Control (LOC) Eating, Global Disordered Eating Attitudes, and BMI z-score the Dependent Variables
 Notes: The direct effect (c') captures the effect of the independent variable (X) on the dependent variable (Y) that does not go through the mediating variable (M). The indirect effect (ab) of the independent variable (X) on the dependent variable (Y) that goes through the mediating variable (M) is calculated as a product of the a and b paths.

Table 6

Summary of Models Testing the Direct and Indirect Associations of Trauma Count with Loss of Control (LOC) Eating Presence Through Depressive Symptoms, Anxiety Symptoms, and Emotional Support

Without Covariates				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
			<i>Indirect Effect</i>	<i>Direct Effect</i>
Mediator	Coefficient (<i>SE</i>)			
Depressive symptoms	1.84 (.74)*	.08 (.04)	.15 (.11)	.25 (.26)
Anxiety symptoms	1.72 (.84)*	.04 (.04)	.06 (.07)	.32 (.24)
Emotional support	-.31 (1.10)	.00 (.03)	.00 (.07)	.43 (.25)
With Covariates (age, sex, and race)				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
			<i>Indirect Effect</i>	<i>Direct Effect</i>
Mediator	Coefficient (<i>SE</i>)			
Depressive symptoms	1.27 (.70)	.06 (.05)	.08 (.14)	.21 (.27)
Anxiety symptoms	1.13 (.82)	.01 (.04)	.02 (.08)	.28 (.25)
Emotional support	.08 (1.04)	.03 (.04)	.00 (.12)	.39 (.26)

* $p < .05$

Table 7

Summary of Models Testing the Direct and Indirect Associations of Trauma Count with Global Disordered Eating Attitudes Through Depressive Symptoms, Anxiety Symptoms, and Emotional Support

Without Covariates				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
			<i>Indirect Effect</i>	<i>Direct Effect</i>
Mediator	Coefficient (<i>SE</i>)			
Depressive symptoms	1.83 (.68)**	.06 (.02)**	.12 (.05)*	-.01 (.09)
Anxiety symptoms	1.64 (.80)*	.06 (.01)***	.10 (.05)	.01 (.09)
Emotional support	-.74 (1.02)	.00 (.01)	.00 (.02)	.10 (.10)
With Covariates (age, sex, and race)				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
			<i>Indirect Effect</i>	<i>Direct Effect</i>
Mediator	Coefficient (<i>SE</i>)			
Depressive symptoms	1.42 (.67)*	.05 (.02)**	.08 (.05)	-.03 (.09)
Anxiety symptoms	1.13 (.80)	.06 (.01)**	.06 (.04)	-.02 (.09)
Emotional support	-.51 (1.02)	.01 (.01)	.00 (.02)	.02 (.10)

* $p < .05$

** $p < .01$

*** $p < .001$

Table 8

Summary of Models Testing the Direct and Indirect Associations of Trauma Count with BMI z-score Through Depressive Symptoms, Anxiety Symptoms, and Emotional Support

Without Covariates				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
Mediator	Coefficient (<i>SE</i>)	Coefficient (<i>SE</i>)	<i>Indirect Effect</i> Coefficient (<i>SE</i>)	<i>Direct Effect</i> Coefficient (<i>SE</i>)
Depressive symptoms	1.27 (1.19)**	.02 (.01)*	.03 (.02)	-.11 (.06)
Anxiety symptoms	1.32 (.89)	.01 (.01)	.01 (.01)	-.11 (.06)
Emotional support	-1.13 (1.30)	-.01 (.01)	.02 (.03)	-.16 (.07)*
With Covariates (age, sex, and race)				
	<i>a</i>	<i>b</i>	<i>ab</i>	<i>c'</i>
Mediator	Coefficient (<i>SE</i>)	Coefficient (<i>SE</i>)	<i>Indirect Effect</i> Coefficient (<i>SE</i>)	<i>Direct Effect</i> Coefficient (<i>SE</i>)
Depressive symptoms	.52 (.72)	.02 (.01)	.01 (.02)	-.13 (.06)*
Anxiety symptoms	.68 (.86)	.01 (.01)	.01 (.01)	-.14 (.06)*
Emotional support	-.55 (1.25)	-.01 (.01)	.00 (.02)	-.18 (.07)*

* $p < .05$

** $p < .01$

DISCUSSION

In this study, I examined associations among developmental trauma – operationalized as number of traumatic events reported via semi-structured interviewing – disordered eating, and BMI standard score in a sample of mostly non-Hispanic White adolescents at risk for excess weight gain. Depressive and anxiety symptoms and emotional support were examined as mediators on an exploratory basis as a test of the Developmental Trauma Model (Cook et al., 2005). Consistent with published data (Anda et al., 2006; Kilpatrick & Saunders, 1997; Norman et al., 2012), nearly 58% of the sample reported at least one traumatic event.

Adolescents who experienced trauma reported significantly greater depressive and anxiety symptoms than their peers without a trauma background. An established body of work captures the association between developmental trauma and internalizing symptoms and disorders in both adolescence and adulthood (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Heim & Nemeroff, 2001; Hill, 2003; Suliman et al., 2009). It has been maintained that trauma sensitizes circuits in the central nervous system and primes salient psychological and social processes, thereby creating increased vulnerability to subsequent stressors and internalizing problems (Heim et al., 2008; Suliman et al., 2009). Further, there is evidence that developmental trauma is not only associated with depressive symptoms, but severity, course, and recurrence of depression (Hovens et al., 2010; Nanni, Uher, & Danese, 2012; Spinhoven et al., 2010). Taken together, finding that adolescents reporting trauma also report greater internalizing symptoms is consistent with prior findings.

Alternatively, adolescents with a trauma background were leaner than peers and there was a significant, negative association between developmental trauma and BMI standard score,

such that as the number of traumatic events reported increased, BMI was lower. Finding a negative association between developmental trauma and BMI standard score was in the opposite direction as was hypothesized, and this result is somewhat counterintuitive as developmental trauma has been associated with excess body weight and obesity in adulthood – an association that has been replicated in community (for example, Anda et al., 2006; Gunstad et al., 2006; Pederson & Wilson, 2009) and psychiatric (for example Aas et al., 2017 and Hepgul et al., 2012) samples. Positive cross-sectional and longitudinal associations between developmental trauma and BMI have also been found in adolescents in community (for example Gooding et al., 2015 and Shin & Miller, 2012) and psychiatric (for example Isohookana, Marttunen, Hakko, Riipinen, & Riala, 2016) samples. Thus, it is unexpected that a negative association between developmental trauma and BMI was observed in this sample. Two possible explanations warrant consideration.

First, this study did not consider compensatory or extreme unhealthy weight control behaviors such as self-induced vomiting, skipping meals, or using diet pills and/or laxatives as these behaviors are relatively infrequent in overweight youth (~3.5%) and with the sample size, unhealthy weight control behaviors could not be appropriately represented here as, consistent with prior estimates, only 3.5% of the current sample reported unhealthy weight control behaviors (Boutelle, Neumark-Sztainer, Story, & Resnick, 2002; Neumark-Sztainer, Wall, Eisenberg, Story, & Hannan, 2006). This limitation is critical to acknowledge as unhealthy weight control behaviors can affect BMI and there is evidence that youth who endorse developmental trauma may be more at risk for these behaviors than youth with no developmental trauma (Ackard & Neumark-Sztainer, 2002; Isohookana et al., 2016; Smyth, Heron, Wonderlich, Crosby, & Thompson, 2008). Unhealthy weight control behaviors may explain, in part, why

youth in this study with developmental trauma were leaner than peers and it is possible that unhealthy weight control behaviors may further be explanatory in understanding the association between traumatic events and BMI in adolescents. Second, this study is limited by a cross-sectional design, which does not allow for the evaluation of changes in the association between developmental trauma and BMI. Provided a positive association between developmental trauma and BMI has been found in both adolescent and adult samples, as previously described, it seems reasonable to expect the same finding would be reflected in this sample. However, there is a small body of research (for example, Schneiderman, Negriff, Peckins, Mennen, & Trickett, 2015) that suggests BMI trajectories in youth with developmental trauma may differ from trajectories for their peers such that youth with developmental trauma may reach their BMI apex in late adolescence, between 16 and 17 years of age, as opposed to earlier in adolescence as is the case in youth without developmental trauma. This may serve as preliminary indication for delayed weight gain trajectories in youth who experience developmental trauma. As such, subsequent investigations of developmental trauma in adolescence would be prudent to assess unhealthy weight control behaviors and examine associations between developmental trauma and BMI over time, ideally in early, mid, and late adolescence. Further, as the present study utilized a sample of adolescents who were at risk for excess weight gain and primarily were overweight, thus limiting generalizability, subsequent investigations should ideally utilize representative samples of adolescents with greater variability in the BMI spectrum, including underweight through obesity, to begin specifying effects, processes, and trajectories as a function of BMI.

Additionally, exploring associations among developmental trauma and excess weight and weight-related behaviors across adolescence may elucidate why others have found positive associations between developmental trauma and global disordered eating and LOC eating (for

example, Fuemmeler et al., 2009; Isohookana et al., 2016; Mazzeo & Espelage, 2002), but this study did not. Fuemmeler and colleagues (2009) dichotomized trauma, finding an association between developmental trauma and disordered eating in a sample of young adults; Isohookana and colleagues (2016) found an association between developmental trauma, as measured with the same semi-structured interview used in the present study, and disordered eating in a clinical sample of adolescents; and Mazzeo and Espelage (2002) utilized a self-report, continuous measure of developmental trauma, finding an association between developmental trauma and LOC eating in a sample of young adults. Thus, differences in the operationalization of developmental trauma and study samples may also explain why previous studies have reported associations between developmental trauma and disordered eating, LOC eating included. It is also possible the onset of disordered eating attitudes and behaviors comes later in adolescence (Neumark-Sztainer et al., 2006), provided that significant associations have been found in older adolescent samples (sample mean ages from 15.5 years to 21.75 years whereas the average age of the current cohort was 14.25 years). Additionally, it is also possible that the effect of developmental trauma on disordered eating is partially mediated by depressive symptoms.

A significant indirect effect of developmental trauma on disordered eating through depressive symptoms was found in the current cohort when not accounting for covariates; this indirect effect was attenuated when accounting for covariates. This indirect effect, developmental trauma influencing disordered eating through depressive symptoms, mirrors what has been found twice before in samples of older adolescents (Mazzeo & Espelage, 2002) and fits existing theory, Affect Regulation Theory (Hawkins & Clement, 1984), which stipulates disordered eating is an attempt to alleviate emotional distress (Dakanalis et al., 2014; Stice, Shaw, & Nemeroff, 1998). This study may have been slightly underpowered to detect this

indirect effect, and other indirect effects, when considering covariates and, as such, future studies would be well suited by larger samples. Nevertheless, this is the first study to find an indirect effect of developmental trauma on disordered eating through depressive symptoms in an adolescent sample. This finding suggests the poorly understood processes instigated by developmental trauma, and ultimately leading to adverse longer-term outcomes, are underway well before adulthood, the developmental period most studied in research conducted to date on developmental trauma. Further, this study contributes something unique in that the non-clinical sample utilized was at risk for excess weight gain and primarily comprised of overweight youth. A meta-analysis found that depressive symptoms, especially among adolescent females, significantly increase the risk for later obesity (Blaine, 2008). Current findings are not only consistent with meta-analytic findings, but suggest, perhaps, that over time developmental trauma may affect depressive symptoms in adolescence which, in turn, ultimately could further exacerbate excess weight gain.

Further investigation of developmental trauma's effect on disordered eating in adolescence through depressive symptoms is important for two, primary reasons. First, understanding the role of depressive symptoms could help in identifying at-risk youth: youth with a history of developmental trauma and elevated depressive symptoms, but who are not yet demonstrating disordered eating behaviors. If these youths can be identified, targeted prevention may be appropriate to support healthy weight and weight-related behaviors. Second, if depressive symptoms are indeed a precursor to disordered eating in adolescents with developmental trauma, existing interventions that address depressive symptoms could, potentially be customized and tested for the reduction of disordered eating and/or excess body weight in youth with developmental trauma. Highly informed intervention is critical for

individuals struggling with disordered eating or excess weight and comorbid trauma histories as evidence suggests developmental trauma, unless properly addressed in treatment, predicts early treatment dropout, less weight lost in clinical weight reduction programs, and continued impairment (Adams et al., 2007; Brewerton, 2007; Fassino, Pierò, Tomba, & Abbate-Daga, 2009; King, Clark, & Pera, 1996). Provided the prevalence of developmental trauma, its public health toll, and the physical and mental health risk associated with trauma, empirically-supported interventions for youth with developmental trauma are necessary (Anda et al., 2006; Corso et al., 2008; Danese & Baldwin, 2017; Center for Youth Wellness, N.D.; CHILDREN NOW, N.D.; Kilpatrick & Saunders, 1997; Fuemmeler et al., 2009; Johnson et al., 2013; Lupien et al., 2009; Olf & van Zuiden, 2017; Shonkoff et al., 2012). While non-significant findings in this cohort suggest the provision of additional emotional support may not be a critical component of these preventions and interventions, this is worth confirming in the future as the present sample is limited in size, ethnic heterogeneity, and perhaps diversity of developmental trauma reported.

Moreover, the present study is limited by several other factors including an incomplete assessment of each participant's development including hypothetically salient individual factors such as temperament and personality (Strelau & Zawadzki, 2005; Thomas et al., 2014; Yoon, Jun, An, Kang, & Jun, 2009). Further, salient biological factors were not assessed. To elaborate, it is evident developmental trauma has a complex impact on physiological indicators of health and stress including cortisol, inflammatory markers like C-reactive protein, and DNA methylation. The association between trauma and cortisol is not entirely understood because a great deal of evidence documents a blunted, hypocortisol awakening response and response to stressors in those with trauma backgrounds (Carpenter et al., 2007; Monaco et al., 2016; Suzuki, Poon, Papadopoulos, Kumari, & Cleare, 2014), while other evidence indicates the opposite

pattern of hypercortisolism (Heim & Nemeroff, 2001). In contrast, data appear to converge in capturing a positive association between developmental trauma and increased inflammation (Coelho, Viola, Walss-Bass, Brietzke, & Grassi-Oliveira, 2014; Danese, Moffitt, Pariante, Ambler, Poulton, & Caspi, 2008; Hepgul et al., 2012; Pace et al., 2006) and alterations in DNA methylation (Houtepen et al., 2016; McGowan et al., 2009). It is undeniable that trauma has a long-term impact on the body and such was not measured or considered in any meaningful way in the present study.

Moreover, the operationalization of developmental trauma in the present study is not without limitations. While many studies (for example, Alisic et al., 2014; Anda et al., 2006; Gutermann et al., 2016; Smyth et al., 2007) have treated trauma as a continuous count variable, as was done here, other studies have examined particular kinds of trauma, for example, physical abuse, sexual abuse, and neglect (for example, Burns et al., 2012; Hund & Espelage, 2006; Mamun et al., 2007; Smyth et al., 2007), whereas others have assessed trauma by type – interpersonal versus impersonal (Fowler et al., 2013; Smyth et al., 2007). Each alternate method of operationalizing developmental trauma is not without its flaws (Brock & Davis, 2008; Gutermann et al., 2016). A continuous count disregards severity and personal relevance/proximity. Specific type disregards the cumulative effect of different traumatic events and the effect of traumatic events not included, and broad type disregards the cumulative effect of trauma and severity to some extent. In the current study, operationalizing developmental trauma as a continuous count variable was the most practical provided the study's sample size and the low count of interpersonal trauma such as physical and sexual abuse. To extend on aforementioned recommendations, a 1) longitudinal, well-powered investigation that 2) includes a more extensive assessment of temperament, personality, unhealthy extreme weight control

behaviors, and biological factors and 3) implements an analytic plan that allows for developmental trauma to be operationalized as a continuous variable, specific type variable, and broad type variable would be well-positioned to address the limitations herein and provide more precise findings on associations among developmental trauma and disordered eating and excess weight in adolescence; these contributions would all ultimately inform prevention and intervention efforts insofar as elucidating when and with whom prevention is most appropriate and when, developmentally and with regards to demonstration of risk, intervention is necessary.

In conclusion, developmental trauma is of tremendous concern for the fields of psychology, psychiatry, public health, social work, child welfare, medicine, and even government. Developmental trauma is pervasive and insidious in the lifelong physical and mental health risks it confers (Anda, Butchart, Felitti, & Brown, 2010; Dube, Felitti, Dong, Giles, & Anda, 2003; Heim & Nemeroff, 2001; Shonkoff et al., 2012). Findings from the present study suggest the developmental trauma may be uniquely related to lower body mass index in early adolescents and indirectly related to disordered eating through depressive symptoms in the same developmental period. Longitudinal research is needed to disentangle the effects of developmental trauma on body mass trajectories independent of normal body maturation during adolescence and clarify the impact of developmental trauma on depressive symptoms and disordered eating.

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