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Exploring Eating in the Absence of Hunger as a Mediator of the Association Between Exposure to Early Life Adversity and Adolescent Obesity Risk

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ABSTRACT

Adolescent obesity remains a prevalent and growing public health concern in the U.S., with 18.5% of children and youth classified as obese in 2015-2016. Eating in the absence of hunger (EAH) in response to negative affect may explain the association between exposure to early life adversity (ELA) during the preschool period and the development of obesity in adolescence. We examined the association between exposure to early life adversity—specifically cumulative sociodemographic risk at age 35 months—and adolescent obesity risk. Participants included 218 adolescents, drawn from a sample of rural households that experienced poverty. Data were collected through in-person home visits and web surveys; only individuals who completed in-person home visits were included in the analysis. Adolescents completed questionnaires and had their waist circumference measured during the home visit. Results showed that there was no direct association between exposure to ELA and adolescent waist circumference. Cumulative sociodemographic risk, however, significantly predicted EAH negative affect (p=0.05) and EAH negative affect significantly predicted waist circumference (p=0.003). Given no direct relation between adversity and obesity risk, we conclude that EAH negative affect did not mediate this association. These findings suggest that early life adversity may contribute to emotional eating behaviors in adolescence, which could have long term implications for obesity risk, but further research is needed on the mechanisms linking early life adversity and adolescent obesity.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

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Chapter 1

Introduction

Childhood Obesity in the United States

A significant public health issue that remains in the United States is childhood and adolescent obesity. About 1 in 5 children and adolescents in the U.S. have obesity, and this rate continues to rise (Centers for Disease Control and Prevention, 2024). In 2015-2016, 18.5% of children and youth classified as obese, with obesity prevalence being highest among adolescents (12-19 years old) at 20.6% (Hales et al., 2017). Adolescents, minority racial groups, and families who are experiencing poverty are disproportionately affected by obesity (Hales et al., 2017). For example, obesity rates were higher among non-Hispanic Black (22%) and Hispanic (25.8%) youth compared to non-Hispanic White (14.1%) and non-Hispanic Asian (11%) youth (Hales et al., 2017). In addition, there is a lower prevalence of obesity in youth from households with higher income compared to youth in households with lower income (Ogden et al., 2018). Given this persistent issue, it is important to gain a deeper understanding of the factors associated with adolescent obesity risk to help inform obesity prevention and intervention programs.

Sensitive Periods for the Study of Exposure to Adversity, Emotional Eating and Obesity

Early childhood is a sensitive period for the exposure of risk factors that lead to the development of adolescent obesity (Aagaard et al., 2024). During the preschool years (3-5 years), children’s brains undergo significant changes and growth, shaping their behavioral

patterns and neural development (Brown et al., 2012). During this period, the brain is more responsive to environmental changes/influences, making this period sensitive to the effects of early life adversity (ELA) (Davis et al., 2023). Furthermore, exposure to ELA during this period may interfere with typical neurodevelopmental growth, potentially causing long-term behavioral and emotional regulation changes later in life (Duffy et al., 2018). Exposure to ELA during these critical years has also been linked to difficulties in managing emotions, which may set the stage for health-risk behaviors such as the utilization of emotional eating as a coping mechanism (Duffy et al., 2018). While Davis et al. (2023) and Duffy et al. (2018) highlight how exposure to ELA in early childhood can lead to emotional health-risk behaviors, neither study is longitudinal nor fully explore the long-term consequences. These gaps underscore the need for longitudinal research to follow the long-term impact of ELA over time and the need to explore how it shapes the development of later health outcomes. This study aims to add to these findings by examining how exposure to ELA can lead to emotional eating behaviors in adolescence in a longitudinal study of youth from households who experienced adversity.

Aagaard et al. (2024) reviewed evidence from a National Institute of Health expert panel workshop on sensitive periods in early development that may influence obesity risk into adolescence. The participating researchers highlighted infancy and early childhood as critical periods in development when environmental, behavioral, and biological factors interact to affect risk for obesity. Factors such as diet quality and exposure to sociodemographic stressors (e.g., households experiencing poverty, single parent households, and affordability of healthy foods) during these formative years were highlighted as having lasting impacts on the development of obesity. By examining exposures such as stress, a deeper understanding of the complex biological, behavioral, and environmental interactions that contribute to an individual’s risk of

obesity during critical life stages can be gained. The workshop's findings advocate for an "exposome" approach to studying these sensitive periods, as this approach could reveal complex interactions between early-life exposures and adolescent obesity outcomes.

Adolescence is another sensitive stage of development where significant physical, social, and psychological changes occur, indicating that it is an essential stage to research for the development of obesity, problematic eating behaviors and body image concerns (Alberga et al., 2012; Shriver et al., 2020; Voelker et al., 2015). Perceived pressure, worries, mood fluctuations, body image issues, and weight concerns are all stressors that impact youth throughout adolescence (Voelker et al., 2015). Such stressors make adolescents more vulnerable to a multitude of health concerns (such as unhealthy emotional eating behaviors, anxiety, and depression), which may increase obesity risk (Shriver et al., 2020). With increasing vulnerability, the transition from childhood to adolescence may lead to a rise in emotional eating behaviors associated with obesity (van Strien, 2018). The present study aims to investigate the role of exposure to early life adversity and emotional eating behaviors in the development of obesity during adolescence, examining the potential ways that sociodemographic risk stressors may influence obesity risk during adolescence. Identifying risk factors in early childhood and adolescence is key to reducing the risk for youth and adulthood obesity and its consequences.

Exposure to Early Life Adversity and Risk for Obesity

Early life adversity (ELA) can be defined as exposure to a range of stressors during critical periods of development during the lifespan, such as during the preschool years, that requires the average child to adapt in significant ways (McLaughlin, 2016). Indicators of ELA

include sociodemographic factors (such as low socioeconomic status, parental education level, financial strain, etc.) (McLaughlin, 2016). Exposure to ELA is associated with heightened risk for health-risk behavioral issues (i.e., being physically inactive and eating high-fat, high-sugar foods) and maladaptive eating behaviors, which can predispose children to obesity (Duffy et al., 2018). Garasky et al. (2009) found that family stressors (i.e., financial strain and home environment factors) were significantly linked to higher risk of obesity and overweight during childhood among younger children (5-11 years). Among older children (ranging from 12-17 years) in the same study, those with obesity and overweight were more likely to live in households with household members that had increased mental and physical health issues and greater financial strain (Garasky et al., 2009). In a similar study, Lohman et al. (2009) found that food-insecure youth (ranging from 10-15 years) had a higher probability of being obese as maternal stressors (i.e., housing problems, family disfunction, disruption, conflict, financial strain, maternal physical and mental health) in the household increased. While there is strong evidence for these associations, much of the previous literature has been from cross-sectional studies which are limited in assessing causal inference. This thesis will fulfill this gap by using data from a longitudinal study to confirm the associations between exposure to ELA during the preschool period and obesity risk in adolescence.

Exposure to ELA, Emotional Eating Behaviors and Risk for Obesity

Exposure to ELA can be thought of as a stressor that can impact health-related behaviors, such as unhealthy eating patterns. Animal studies suggest that stress increases the craving for food, especially for indulgent, “comfort foods” (Patterson & Abizaid, 2013; Pecoraro et al.,

2004; Dallman, 2010). Emotional eating is defined as the inclination to eat, often excessively, in response to emotional states (Godet et al., 2022). These emotional states include stress, anxiety, or negative affect, rather than physical hunger (Stunkard & Messick, 1985). Emotional eating is seen as a maladaptive behavior, often involving the intake of high-calorie, palatable foods, and can contribute to the development of eating disorders, metabolic problems, and, ultimately, obesity (Godet et al., 2022; Zeeck et al., 2010; Koenders & van Strien, 2011). Exposure to chronic stress can be a potential driver of stress-induced or emotional eating, contributing to the effects that increase the risk of developing obesity (Pervanidou & Chrousos, 2011; Sinha & Jastreboff, 2013). Findings from a study by Kazmierski et al. (2022) show that for adolescent females with high exposure to ELA (prior to age 10 years), reports of negative affect were associated with greater consumption of calories and of solid fats, which may contribute to their obesity risk. Exposure to early stressors, such as sociodemographic risks, can lead to emotional eating behaviors, increasing vulnerability to obesity risk in adolescence (Kazmierski et al., 2022).

Emotional eating has been studied as a potential risk factor for obesity in adult populations (Dakanalis et al., 2023) but less is known about this association in adolescents. Limbers and Summers (2021) conducted a systematic review to examine the association between emotional eating and obesity risk in adolescents and concluded that there was no association. Alternatively, Nguyen-Michel et al. (2007) showed that that emotional eating was associated with a higher intake of highly palatable foods (i.e., sweet, energy-dense foods such as ice cream, chips, and soda) in predominantly Latino middle school students. Studying emotional eating behaviors during adolescence is critical for understanding the factors that increase the risk of obesity in youth. As there is a need for research on the association between

emotional eating and obesity risk in adolescents, this study aims to fill the gap in the literature by exploring this association specifically in adolescents, which may help inform targeted interventions to mitigate obesity risk that may persist into adulthood.

Eating in the absence of hunger (EAH) is a type of eating behavior that may be driven by external cues and emotional triggers and refers to the tendency to consume food even when physiological hunger cues are absent (Lansigan et al., 2015; Shomaker et al., 2013; Arnold et al., 2015). A systematic review of EAH behaviors in young children ages 12 years old or younger concluded that EAH is frequently prompted by external cues such as the availability of palatable foods, as well as by internal cues like negative emotions (Lansigan et al., 2015). While research on the link between stress and EAH in youth is limited, studies have shown positive associations between stress and EAH in youth populations (Francis et al., 2013, 2024; Miller et al., 2018). This thesis will build upon previous literature by using the same dataset used in Francis et al. (2023, 2024), but explores whether exposure to ELA influences EAH in adolescents, which in turn influences adolescent obesity risk. Francis et al. (2024) used an experimental design to investigate the potential ways that induced stress impacted eating in adolescents, but their study did not specifically address the role of ELA in stress-eating patterns; this study aims to fill this gap.

EAH that is driven by negative emotions has been measured in children and adolescents and is described as eating in response to feeling sad or depressed, feeling angry or frustrated, and feeling anxious or nervous, in the absence of hunger (Tanofsky-Kraff et al., 2008). Dakanalis et al. (2023) emphasize the ways that negative emotions can disrupt normal eating patterns, contributing to maladaptive behaviors that may increase the risk of obesity. Youth from households that experience poverty may be at increased risk; Cheon et al. (2024) found that low

subjective social status (low socioeconomic and social status) in youths (~13 years) was associated with greater BMI, fat mass, and EAH due to negative affect when facing weight-related teasing distress (an emotional social stressor). These findings highlight how emotional eating behaviors in response to negative affect, particularly in the context of low subjective social status, serve as contributors to obesity risk in adolescents. The current study expands on the study by Cheon et al. (2024) by using a more complex measure of ELA and examining its relationship with waist circumference compared to BMI. Waist circumference provides a more accurate estimate of obesity (Taylor et al., 2000). Understanding the relation between EAH in response to negative affect and obesity risk is especially relevant during sensitive periods such as adolescence.

The Biological Embedding and Biological Underpinnings of Obesity

Biological embedding refers to the process by which environmental stressors—such as sociodemographic risks—alter physiological systems, leaving lasting biological imprints that impact health (Hertzman, 1999). Biological embedding occurs when exposure to stressful adverse experiences leads to sustained physiological changes within biological systems, ultimately influencing health outcomes (Miller et al., 2011). These changes occur through mechanisms such as proinflammatory tendencies, altered gene expression, and posttranslational modification (Miller et al., 2011).

Exposure to stress can impact eating behavior and risk for obesity through a number of biobehavioral mechanisms. Michels (2019) provided a model that outlines biological and behavioral pathways through which stress may contribute to obesity, with a focus on impacts on

fat accumulation, appetite regulation and eating behavior. Stress may contribute directly to obesity through interactions between the stress hormone cortisol and lipid metabolism (Peckett et al., 2011). First, elevated cortisol levels can increase circulating non-esterified fatty acids (NEFA), which can then be used for the accumulation of fat in adipose tissue (Peckett et al., 2011); higher levels of cortisol contribute to abdominal fat deposition because of its high density of cortisol-binding glucocorticoid receptors (Björntorp, 2001). Second, cortisol may influence lipolysis, which is the degradation of adipose tissue to fatty acids. This means that cortisol may influence the storage and breakdown of fat (Peckett et al., 2011).

An underlying biological pathway to emotional eating caused by stress is through cortisol impacting appetite-related neuropeptides in the brain, particularly within the hypothalamus (Adam & Epel, 2007). Some functions of the nuclei in the hypothalamus are tied to stress response, reward sensitivity and appetite regulation (al'Absi, 2018). An increase in cortisol causes increased activity of neuropeptide Y (NPY; an appetite and reward inducer), and dysregulated levels of leptin and insulin, which are hormones responsible for suppressing hunger and reward (Adam & Epel, 2007). The continuous dysregulation of leptin causes the body to become resistant to its effects, which may result in increased appetite and increased food intake (Björntorp, 2001). Studies show that youth exposure to adversity were associated with emotional and dysregulated eating behavior (Michels et al., 2012; Thomas et al., 2020;; Huffhines et al., 2020; Miller et al., 2018). These studies found cross-sectional associations with youth eating behavior; the current study examines the longitudinal effects of ELA as a stressor from early childhood and its effect in adolescence. A longitudinal perspective is important, as chronic stress in childhood, in the form of exposure to ELA, may have lasting impacts on emotional eating behaviors and obesity risk in adolescence. Although the current study does not include biological

markers of appetite regulation, this study uses a behavioral measure of emotional eating (EAH in response to negative affect) that has been shown to result from dysregulated appetite control (Macht, 2008; Russel & Russel, 2021). Thus, EAH is a good proxy measure of this biological mechanism, thereby providing a rationale to examine if it mediates the effect of ELA on obesity risk. In sum, exposure to various forms of adversity during early life years can become biologically embedded in ways that affect fat accumulation, neural development, emotional regulation, appetite-regulation and eating behaviors that impact later health outcomes such as obesity.

Cumulative Sociodemographic Risk as a Measure of ELA

Sociodemographic risk factors (such as including low socioeconomic status, low parental education level, and low household income) may be indicators of ELA (Suglia et al., 2022). Lower socioeconomic status often serves as an indicator of adversity, as it reflects limited access to health aid resources, stable housing, and nutritious food options (Pampel et al., 2010). Williams et al. (2018) highlight that socioeconomic status was a major determinant of childhood obesity; children (6-11 years) from lower-income families were more likely to experience conditions linked to obesity such higher exposure to food insecurity (Kaur et al., 2015). Lee et al. (2013) examined social disadvantage (measured by low parent education) in a nationally representative sample of adolescents followed from grades 7-12 in 1995 into young adulthood and found that school level disadvantage (percent of students in households with low education parents) was linked to higher obesity risk in adolescence for both males and females. These

findings suggest the impact of sociodemographic risk factors, particularly socioeconomic factors, on obesity risk.

Poverty in early childhood is another sociodemographic risk factor that may lead to obesity risk (Wells et al., 2010). Poverty and related sociodemographic risks during early childhood can have lasting effects on brain development, specifically in the amygdala and hippocampus, the brain regions involved in memory, emotional regulation, and stress response (Luby et al., 2013). As a response to chronic exposure to ELA (e.g., poverty), the body goes through a "wear and tear" (referred to allostatic load) which leads to physiological changes (Evans & Fuller-Rowell, 2013). In a longitudinal study conducted by Evans and Fuller-Rowell (2013) of young adults, early childhood poverty was identified as a significant predictor of allostatic load. Findings from similar studies show an association between early childhood poverty and higher levels of allostatic load (measured as a composite index of chronic physiological stress that reflects the body's responses to prolonged environmental stressors) later in life (De France et al., 2022; Evans & De France, 2021). Children's higher levels of allostatic load have been linked to higher body mass index in youth (indicating obesity) in other studies (Cedillo et al., 2019; de la Rosa et al., 2023). Most of the studies on early childhood poverty focus on obesity outcomes later in life rather than in adolescence. This study aims to fill this gap by using a high-poverty sample, a more comprehensive measure of ELA with multiple indicators of adversity, and an objective measure of obesity risk in adolescence. Taken together, exposure to early life adversity may lead to emotional eating behaviors in adolescents, which in turn may increase the risk for adolescent obesity.

The Current Study

While existing research has explored links between exposure to ELA and emotional eating behaviors, exposure to ELA and obesity risk, and the potential ways emotional eating behaviors may impact obesity risk for adolescents, there is a lack of studies on the ways in which emotional eating behaviors, particularly EAH in response to negative affect, in adolescents may play a role in the association between ELA and obesity risk. Additionally, the role of emotional eating, or specifically eating without hunger in response to negative affect, has not been examined within one combined model. This study aims to fill this gap by exploring how early stress and poverty-related adversity in childhood interact with emotional eating behaviors in adolescence. This study will contribute to the understanding of (1) the potential ways that emotional eating behaviors develop in adolescent populations and (2) examining whether exposure to ELA influences eating in the absence of hunger behaviors which in turn impacts obesity risk among an adolescent population that live in rural areas, many of whom grew up in poverty. Specifically, this study hypothesizes that adolescents who experienced higher levels of exposure to ELA during the preschool years would show greater risk for obesity in adolescence. In addition, it was hypothesized that exposure to ELA during the preschool years (age 35 months) would be associated with an increased risk of developing obesity in adolescence. Lastly, it was hypothesized that associations between exposure to ELA and obesity in adolescents may be explained by increased emotional eating, measured as EAH in response to negative affect.

Chapter 2

Methods

Conceptual Model

The current study's focus is on understanding the potential mechanisms that early adversity shapes emotional eating behaviors (which are invoked in response to emotional states such as negative affect and stress) which in turn impacts later health outcomes such as obesity. Figure 1 shows a conceptual model of the hypothesized mechanism by which exposure to early life adversity (ELA) may be associated with adolescent obesity through emotional eating behaviors. Path 1 represents the hypothesis that children who experience high levels of exposure to ELA during the preschool years (age 35 months) may show greater risk for developing obesity in adolescence. Path 2 represents the hypothesis that exposure to ELA during the preschool years (age 35 months) may have more emotional eating behaviors in adolescence. Path 3 represents the hypothesis that emotional eating behaviors in adolescence is a mediator of the association between ELA and obesity risk in adolescence.

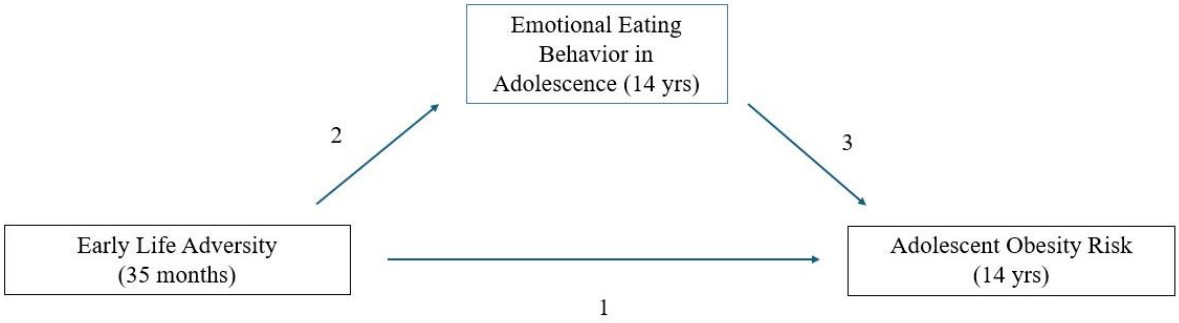

Figure 1: Conceptual Model of Associations Between Early Life Adversity and Adolescent Obesity Risk, through Emotional Eating Behavior

Participants

Data were drawn from the Family Life Project (FLP), a longitudinal, birth cohort study of predominantly poor youth and families residing in rural communities in the United States (Vernon-Feagans et al., 2012). Extensive details about study recruitment and data collection procedures have been published elsewhere (Vernon-Feagans et al., 2012). Briefly, pregnant, English-speaking mothers residing in six poor rural counties in eastern North Carolina and Central Pennsylvania were recruited between 2003 and 2004 at the time that they gave birth; low-income families and Black families were oversampled. A total of 1,292 families were recruited at birth, and nearly 1,000 were followed through age 12 years. A subsample of these families (39.4%) participated in an ancillary research study (the Family Health Study) that was designed to examine exposure to stress and its relation to obesity when youth were 14 years of age (samples sizes are described below). Eligibility criteria for participation in the FLP ancillary study included: (1) the absence of severe food allergies or chronic medical problems affecting food intake, and (2) the absence of dietary restrictions involving animal products; families were

not recruited based on weight status or concern about child weight. This study was approved by the Institutional Review Boards at the University of North Carolina and The Pennsylvania State University (protocol numbers 16-2343 and 2119, respectively); parents provided consent for adolescents' participation, and adolescents provided written assent.

Design and Procedures

The Family Health Study (FHS) utilized a planned missingness design (Graham et al., 2006) in which costly (and more valid) measures are collected alongside cost-effective (and less valid) measures of the same construct. Using this approach, web-based surveys were used to provide a subjective measure of EAH, and an objective assessment of EAH was administered during a home visit. FHS data collection took place from 2016 to 2020. Home visit families completed two 2.5-hour visits that were ~1 week apart, which varied based on youth exposure to a psychosocial stress task (i.e., one low-stress visit, one high-stress visit); we only report data collected during the low-stress home visit and through web surveys. Following the completion of each home visit, links to individual web surveys were sent to youth and primary caregivers (referred to as parents from here on). While FHS had extensive data collection, the current study focused specifically on the described measures and time points. A total of 303 individuals who completed in-person home visits were included in the initial sample. The sample was then reduced based on missing data on any of the main variables of interest, resulting in a final sample of 218 participants.

Upon arrival to the home, adolescents' (~14 years of age) waist circumference was measured by trained research assistants (RAs). Adolescents then completed a series of computer-

based questionnaires and tasks over the course of ~1.5 hours. Lastly, adolescents and their immediate family members were given a standard meal, followed by additional assessments and surveys. Timepoints of data collection for each measure are shown in Table 1.

Table 1: Timepoints of Measure Collection

Age of Child	**Reported By**	**Measure Collected**
35 months	Parent	Early childhood sociodemographic cumulative risk
12 years	Parent	Household sociodemographics
14 years	Adolescent	EAH negative affect
14 years	Adolescent	Waist circumference

Measures

Early Childhood Sociodemographic Cumulative Risk. Garmezy and colleagues (1984) and Rutter (1987) first proposed cumulative risk models. A validated, cumulative risk composite score was used as a measure of adolescents' exposure to adversity in early childhood (Burchinal et al., 2008). When children were 35 months, parents were asked to answer questions related to: maternal education, employment hours, job prestige, household density, household income-to-needs ratio (INR), constant spouse/partner living in the home, and neighborhood noise and safety. Household income was based on anyone who lived in the home ≥ 3 nights/week. Based on household income and size, INR was calculated using yearly poverty threshold values. At the end of each visit, trained research assistants rated each family's neighborhood in terms of noise,

safety, and community safety, and the ratings (1 to 4) were then averaged. Protective indicators (e.g., maternal education) were reverse coded, and each risk indicator was then standardized and averaged across all 4 time points to create the cumulative risk score. Cumulative sociodemographic risk was calculated as the mean of the standardized risk variables (mean = 0, SD = 1) within the sample (maternal education, parental unemployment, family income, single parent, neighborhood safety, number of children in the household, and number of negative life stressors).

EAH Questionnaire. The *Eating in the Absence of Hunger Questionnaire for Children and Adolescents (EAH-C)* was developed by Tanofsky-Kraff and colleagues (2008). The EAH-C is a 14-item measure designed to assess EAH in response to immediate factors (e.g., emotions, environmental cues) among youth ages 6 to 19 years, and consists of three subscales: Negative Affect, Fatigue/Boredom, and External Eating, and one total scale. Only the Negative Affect subscale was used for the purposes of this study. Adolescents are asked to "Imagine that you are eating a meal or snack at home, school, or at a restaurant. Imagine that you eat enough of your meal so that you are no longer hungry." Adolescents were then asked how often they **keep** eating for various reasons including "you are feeling sad or depressed." Adolescents are next asked, "Now imagine that you finished eating a meal or snack some time ago and you are not yet hungry." The adolescent was then asked how often they **start** eating for the same reasons. Response options ranged from 1 (never) to 5 (always). The original measure had a "I prefer not to answer" response option, which we omitted in the current study. All three subscales and the total scale had good internal consistency with Cronbach's alpha ranging from 0.82 to 0.94, which is consistent with past studies (Madowitz et al., 2013; Tanofsky-Kraff et al., 2008).

Adolescent Waist Circumference. Waist circumference was assessed using a validated protocol developed by Taylor et al. (2000). During the first home visit, adolescents' waist circumference was measured twice to the nearest 0.1 cm using a clinical-grade measuring tape (seca). The measurements aimed to estimate abdominal (visceral) fat and assess the adolescents' obesity-related disease risk. Trained research assistants took the measurements at the participant's side, focusing on measuring from the greatest protuberance of the abdomen. The average of the two measurements was used as the final waist circumference value. Any suspicious or discrepant measurements, such as differences greater than 0.2 cm or cases where participants refused the measurement, were flagged and set as missing.

Sociodemographic characteristics. At the 12-year home visit, parents reported on household sociodemographics, which included household income, maternal education, and state of residence. Household income was determined based on any earner who resided in the home. Individuals were considered residents of the household if they spent 3+ nights/week in the youth's home. Income-to-needs ratio (INR) was calculated using the 2007 poverty threshold values, based on household size and total household income (U.S. Census Bureau). For descriptive purposes, INR was categorized into seven groups based on categories outlined by Center on Urban Poverty and Community Development (n.d.) and Dalaker (2024): below 1.00 (below poverty), 1.00 (at poverty), 1.01–1.24 (near poverty), 1.25–1.49 (low income), 1.50–1.84 (moderate income), 1.85–1.99 (moderate to high income), and 2.00 or above (high income/financially secure).

Statistical Analyses

Descriptive statistics were calculated for all variables. Zero-order Spearman rank correlations were conducted to examine the bivariate associations between main variables (cumulative sociodemographic risk, adolescent EAH negative affect, and adolescent waist circumference) since the continuous measures were not normally distributed.

To examine the mediating role of adolescent EAH in response to negative affect in the associations between exposure to early life adversity (measured as cumulative sociodemographic risk at 35mo) and adolescent waist circumference, separate regression models were conducted for each mediation pathway as shown in Figure 2: Path A examined the association between cumulative sociodemographic risk and adolescent EAH in response to negative affect; Path B examined the association between adolescent EAH in response to negative affect and adolescent waist circumference; and Path C examined the direct effect of cumulative sociodemographic risk on adolescent waist circumference. All regression models were adjusted for household income, state of residence, adolescent age, and sex. Analyses were completed in R statistical software (v4.4.3; R Core Team 2021) and SAS software version 9.4 (SAS Institute, Inc., Copyright 2013, Cary, NC).

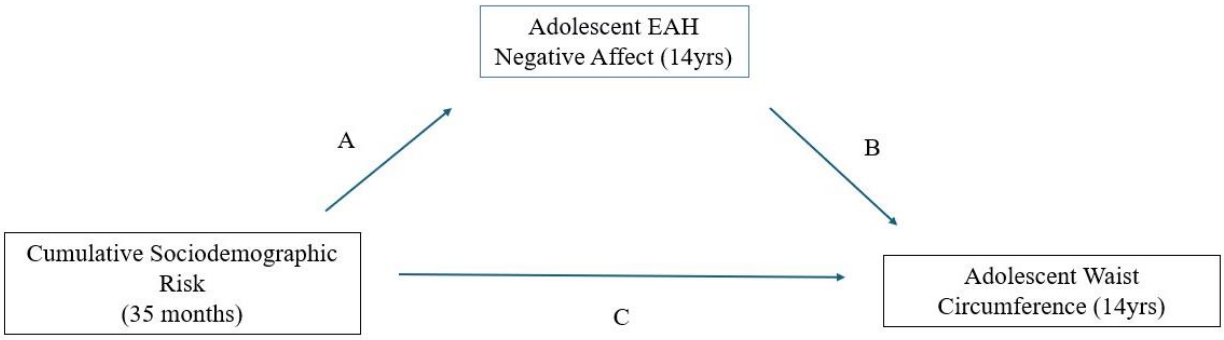

Figure 2: Analytical Model of Pathways Between Early Life Adversity and Adolescent Obesity Risk, through Emotional Eating Behavior.

Chapter 3

Results

Sociodemographic characteristics from the total sample at the 35-month visit are presented in Table 2. Adolescent age ranged from 12.5 years to 14.6 years (M = 12.9 years) and 45.16% were females. The majority of adolescents (76.61%) were non-Black with 30.73% of the total sample residing in North Carolina and 69.27% in Pennsylvania. Almost all (99.54%) of the primary caregivers were biological mothers. Based on education level at the 35-month visit, approximately 70% of the sample had a high school diploma, GED, or less. At the time of enrollment, 72% of households (n=157) were categorized as poor based on income, and at the 35-month visit, 32.11% of primary caregivers were single and 11.47% were separated or divorced. At the 35-month visit, 33.94% of primary caregivers were unemployed, a rate significantly higher than the 2004 national unemployment rate of 5.4% (National Archives and Records Administration, 2004).

Table 2 also presents descriptive statistics on the main variables. The cumulative sociodemographic risk had a mean score of -0.1 (SD = 0.64), with scores ranging from -2.05 to 2.71. Approximately 44% of the sample had cumulative sociodemographic risk scores above the sample mean. Adolescent-reported EAH negative affect had a mean score of 1.44 (SD = 0.70), with scores ranging from 1 to 4.17. This indicates a relatively low tendency to eat in response to negative affect. The proportion of adolescents with obesity (BMI $\geq 95^{th}$ percentile) in our sample (26.61%) was higher than the 2015–2016 national prevalence rate for U.S. adolescents aged 12 to 19 years (20.6%) (Hales et al., 2017).

Table 2: Descriptive Statistics of the Main Variables and Sociodemographics (n=218)

Main Variables		Mean Score	SD	Range
EXPOSURE TO EARLY LIFE ADVERSITY				
Cumulative sociodemographic risk (35mo)		-0.10	0.64	-2.06 - 2.71
ADOLESCENT EATING BEHAVIOR AND OBESITY RISK				
Adolescent EAH Negative Affect		1.44	0.70	1 - 4.17
Adolescent Waist Circumference		76.96	15.06	52 - 123.75
Average Adolescent Age (years)		12.91	0.35	12.53 – 14.59
SOCIODEMOGRAPHICS				
Household Income-to-Needs Ratio		2.04	1.48	0 - 10.39
		Estimate		
		n	%	
Household Economic Level				
High		61	27.98	
Low		157	72.02	
State of Residence	North Carolina	67	30.73	
	Pennsylvania	151	69.27	
Parent Sex	Female	216	99.54	
Parent Education Level (35mo)	Less than high school	21	9.63	
	High school/GED	64	29.36	
	Some post HS/GED training/college	65	29.82	
	Associate's Degree	22	10.09	
	Bachelor's Degree and Professional (i.e., Master's, J.D., Ph.D.) Degree	46	21.2	

Parent Relation to Child at 35mo	Mother	216	99.53
	Grandparent	1	0.47
Parent Employment Status	Employed	144	66.06
	Not Employed	74	33.94
Parent Marital Status	Single	70	32.11
	Married	123	56.42
	Separated	9	4.13
	Divorced	16	7.34
Adolescent Sex	Female	98	45.16
	Male	119	54.84
Adolescent Race	Not Black	167	76.60
	Black	51	23.40

Bivariate Associations Among Variables

Table 3 shows the Spearman correlation coefficients for associations between the main variables. Cumulative sociodemographic risk was positively and significantly associated with adolescent-reported EAH negative affect (r = 0.13, p=0.050). Cumulative sociodemographic risk was positively but not significantly correlated with adolescent waist circumference (r = 0.05, p=0.424). Adolescent-reported EAH in response to negative affect was significantly and positively correlated with adolescent waist circumference (r = 0.20, p=0.003).

Table 3: Correlation Matrix

	1	2
1. Cumulative Sociodemographic Risk (35mo)		
2. Adolescent EAH Negative Affect	0.13*	
3. Adolescent Waist Circumference (mean)	0.05	0.2**

*Note. n = 218 ***p<0.001, **p<0.01, *p<0.05*

Exploring the Role of EAH in the Link Between Early Life Adversity and Waist Circumference

Multiple regression analyses were conducted to examine all pathways adjusting for adolescent sex, household income at the 14-year visit, adolescent race (Black/non-Black), and age. As shown in Figure 3 and Table 4, the direct pathway (Path C) from cumulative sociodemographic risk to adolescent waist circumference revealed no significant effect (β = 0.65, p=0.758). Next, the effect of cumulative sociodemographic risk on adolescent-reported EAH in response to negative affect (Path A) also showed no significant association (β = 0.08, p=0.402). Finally, the pathway from adolescent-reported EAH in response to negative affect to adolescent waist circumference (Path B) demonstrated a positive significant effect (β = 4.63, p=0.002).

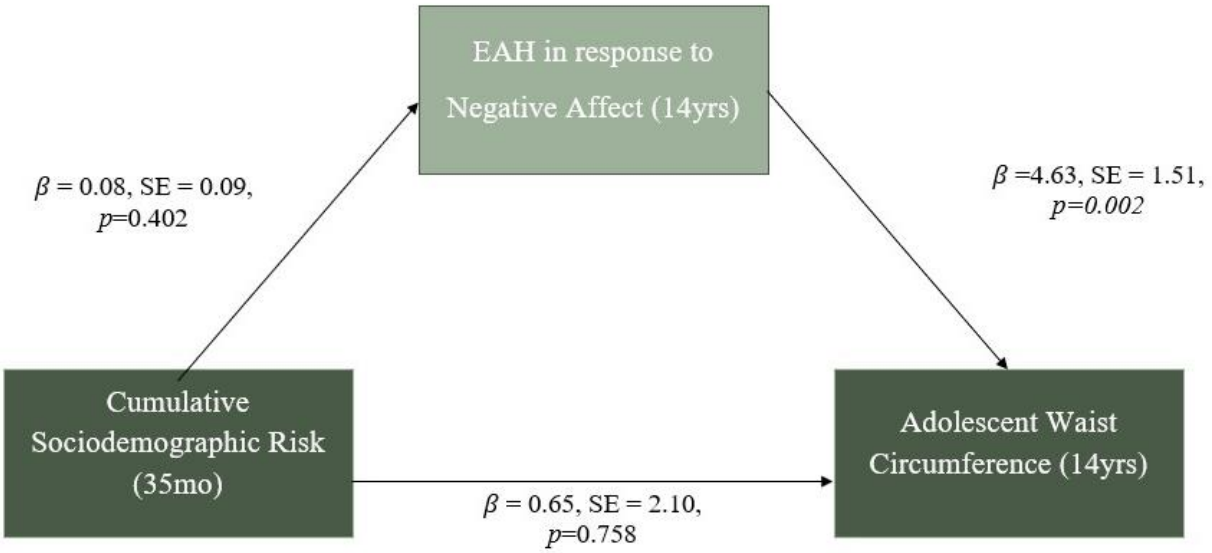

Figure 3: Model Examining the Mediating Effects of Eating in the Absence of Hunger in response to Negative Affect on the Relation Between Cumulative Sociodemographic Risk (35mo) and Adolescent Waist Circumference.

Table 4: Examining the Mediating Effects of Eating in the Absence of Hunger in response to Negative Affect on the Relation Between Cumulative Sociodemographic Risk (35mo) and Adolescent Waist Circumference.

	β	SE	*p*
Cumulative Risk Predicting EAH (Path A)			
Cumulative Risk (35mo)	0.08	0.09	0.402
EAH Predicting Waist Circumference (Path B)			
EAH Negative Affect	4.63	1.51	0.002
Cumulative Risk Predicting Waist Circumference (Path C)			
Cumulative Risk (35mo)	0.65	2.10	0.758

Note. Adjusted for household income, state of residence, adolescent age, and child sex.

Chapter 4

Discussion

The present study sought to address the hypothesis that adolescents' dysregulated eating would mediate the association between exposure to early childhood adversity and adolescent obesity risk (measured by waist circumference). We examined the associations between exposure to cumulative sociodemographic risk in early childhood and waist circumference, in adolescence, with EAH in response to negative affect as a mediator. Our primary hypothesis was not supported—while results adolescent reported EAH in response to negative affect was significantly associated with waist circumference, cumulative sociodemographic risk did not directly affect EAH in response to negative affect nor predict waist circumference. This significant pathway of EAH in response to negative affect remained a significant predictor of adolescents' waist circumference even after controlling for all covariates.

The absence of a direct association between early life adversity and adolescents' obesity risk is contrary to findings in the literature. Gardner et al. (2019) found that exposure to adverse experiences in childhood was associated with increased adolescent obesity risk. De Visser et al. (2024) found that adolescents' exposure to adverse experiences in childhood before the age of 9 years was associated with increased BMI at 9 and 13 years. While our hypothesis was not supported, our study was one of the first to examine how adolescent EAH in response to negative affect may have mediated the association between exposure to early life adversity during the preschool years and adolescent obesity risk.

Despite no evidence of a mediating effect of EAH, there were some interesting findings at the bivariate level. For instance, cumulative sociodemographic risk was significantly correlated to EAH negative affect, but this association was no longer significant after running adjusted regression analyses. However, prior research suggests that sociodemographic risk is associated with emotional eating behaviors (Proffitt Levya et al., 2020; West et al., 2021). In children aged 5-12, experiencing stress increased emotional eating, consumption of sweet foods, and external eating (Michels et al., 2015). The absence of a significant association between sociodemographic risk and EAH negative affect may suggest that additional sociodemographic factors (e.g., financial strain, parenting style, food insecurity) may have more prominent influences on emotional eating. It is also possible that the association between adversity and EAH negative affect varied by sex, thus masking our findings, but this was not a focus of the current study. Future studies should explore sex differences in the pathways we examined in the current study, as well as additional sociodemographic risk factors (e.g., parenting styles and food insecurity), to gain deeper insights on the influences on emotional eating behaviors.

Our findings contribute to the growing body of literature examining the role of early life adversity in shaping adolescent eating behaviors. Consistent with previous research, exposure to adversity in early childhood was not directly linked to adolescent emotional eating (Kazmierski et al., 2022), although others have found a positive, significant association between exposure to adversity and emotional eating in children (Thomas et al., 2020; Miller et al., 2018). The absence of a direct association between early life adversity and waist circumference may suggest the association between adversity and obesity is complex and may be influenced by additional mediators (Schroeder et al., 2021). Future studies should examine additional factors of adversity to better understand their roles in the development of emotional eating behaviors.

Surprisingly, there was no direct association between exposure to early adversity and waist circumference in this study, indicating that our findings did not support the hypothesis that EAH negative affect would mediate the association between early life adversity and waist circumference. This is inconsistent with prior literature suggesting that emotional eating is a key mechanism linking early life adversity to obesity risk (Huffhines et al., 2020; Cheon et al., 2024). One possible explanation for our finding is that other forms of emotional/dysregulated eating, such as binge eating or dietary restraint, may play a more significant role in this relation. Additionally, physiological factors such as HPA axis dysregulation may contribute to obesity risk beyond the effects of emotional eating alone (Adam & Epel, 2007; Bose et al., 2009; Rutters et al., 2012). Future studies should examine additional biological pathways, such as neurobiological and behavioral mechanisms, linking early life adversity to adolescent obesity.

Strengths and Limitations

This study had several strengths. One of the primary strengths is the use of a longitudinal dataset, which allowed for the examination of early childhood exposure to adversity and its potential long-term effects on adolescent eating behaviors and waist circumference. By assessing ELA during the preschool years and following outcomes into adolescence, this study provides insight into the potential critical periods of development of obesity by linking early-life risk factors to later health behaviors. Additionally, including a diverse sample across two states enhances the generalizability of findings to a broader population, while statistically controlling for household income, sex, and state of residence ensures that the results are more reliable and accurate.

However, the study also has limitations that should be considered when interpreting the results. First, while the longitudinal design is a strength, ELA was only assessed at a specific time point in early childhood (35 months), which may not fully capture the cumulative impact of adversity across development and into adolescence. Longitudinal studies tracking changes in eating behaviors and adiposity over time could provide a clearer understanding of how early life adversity influences obesity risk later in life. Additionally, although EAH was measured as a possible mediator between ELA and obesity; other factors such as stress-related physiological changes or physical activity were not accounted for, limiting a more comprehensive understanding of the pathways leading to adolescent obesity. For instance, Michels (2019) outlined several biological pathways that link stress to eating behaviors and obesity, including the microbiota-gut-brain axis, appetite-related molecules, and hormones (such as NPY, GLP, ghrelin, and leptin), highlighting the complex interplay of biological and behavioral factors in obesity development. The present study did not include such biological mechanisms, which are crucial to understanding how stress impacts eating behavior and obesity risk. In addition to these factors, key metabolic hormones such as insulin, ghrelin, and leptin, which play crucial roles in regulating appetite and energy balance (Michels, 2019), were also not examined in this study.

Additionally, the reliance on self-reported measures of emotional eating may have introduced reporting bias, particularly among adolescents. While the self-report EAH questionnaire (Tanofsky-Kraff et al., 2008) provided valuable insight into adolescents' perceptions of their eating behaviors, objective measures of eating in response to negative affect (e.g., laboratory-based eating tasks) may yield different findings. Finally, the study's sample size and its focus on a primarily low-income population may have limited the ability to find significant associations between early life adversity and adolescent obesity risk. It is possible that

determining associations with early life adversity was difficult due to a sample with higher-than-average rates of adversity exposure.

Despite these limitations, this study contributes valuable knowledge to the field by highlighting the role of emotional eating in adolescent obesity and demonstrating that early exposure to adversity may influence eating behaviors differently depending on sex. Future research should explore additional pathways, consider additional measures of ELA, use diverse methodological approaches, and examine interventions that target emotional eating as a potential mechanism for mitigating obesity risk in adolescents exposed to early life adversity.

Clinical and Public Health Implications

Addressing early life adversity and emotional eating in youth is important for reducing obesity risk. Clinicians working with adolescents, particularly those from socioeconomically disadvantaged backgrounds, should consider incorporating stress management strategies into treatment plans. Programs that teach positive coping behaviors in response to negative emotions, such as mindfulness, emotional regulation, and stress-reduction techniques, may help mitigate the development of emotional eating (Kudlek et al., 2024; Katterman et al., 2014). For instance, Kudlek et al. (2024) aimed to reduce emotional eating through an Acceptance and Commitment Therapy (ACT)-based management intervention. This intervention promoted self-awareness and taught alternative coping strategies such as preparation (i.e., changing food shopping habits and food environment in the home to keep tempting foods away), substitution (i.e., exchanging their usual comfort foods for emotional eating with healthier food options), and acceptance (included allowing themselves to fully experience the emotion or emotional eating trigger without reacting

to it, letting it pass naturally) (Kudlek et al., 2024). Participants learned to disconnect emotions from eating behaviors, which helped them manage emotional eating more effectively over time (Kudlek et al., 2024). While many found the intervention to be beneficial, some struggled to participate because of an external locus of control or complicated emotional experiences, highlighting the need for more personalized and longitudinal/ongoing support (Kudlek et al., 2024). Clinicians should also take into consideration the potential impact of early life adversity on eating behaviors and obesity risk, ensuring that they assess for emotional eating and other dysregulated eating behaviors when they assess adolescent health.

Public health interventions should focus on reducing the impact of early life adversity and promoting healthier coping strategies for emotional stress, especially in rural and low-socioeconomic status communities. One example of an intervention is the Strengthening Families Program (SFP), an evidence-based initiative that improves family relationships and teaches essential coping skills (such as bonding and creating warm relationships with family and friends, setting firm and clear boundaries, and monitoring emotional well-being) to both parents and their youth (Strengthening Families Program, 2025). This program aims to decrease child maltreatment, youth substance abuse, and youth depression and aggression, while also increasing family bonding and parental involvement by teaching parents parenting skills and youth life skills (Strengthening Families Program, 2025). By enhancing parents' abilities and providing youth with tools to manage stress and make positive decisions, SFP tries to help reduce the negative effects of early life adversity on youth behavioral development (Strengthening Families Program, 2025). Another effective intervention is the Family Check-Up (FCU). The FCU is an intervention designed to improve parent-child interactions and reduce problematic behaviors in children and adolescents (Family Check-Up, 2025). By enhancing positive parenting practices

and emotional regulation, it helps children manage emotions and prevent risky behaviors (Family Check-Up, 2025). FCU is especially beneficial for families facing adversities like low socioeconomic status, promoting healthier family dynamics (Family Check-Up, 2025).

Programs that target emotional eating, such as school-based mental health initiatives and community support groups, could be effective in reducing the risk of obesity in adolescents (Wilksch et al., 2015). One effective program was the Media Smart program, a school-based initiative conducted by Wilksch et al. (2015), which demonstrated significant benefits in reducing eating disorder and obesity risk factors among adolescents. A randomized controlled trial with adolescents (~ 13 years) showed that girls who participated in Media Smart had half the rate of onset of clinically significant concerns about shape and weight compared to the control group (Wilksch et al., 2015). Media Smart participants reported more physical activity and less screen time, with sustained improvements in eating concerns at the 12-month follow-up (Wilksch et al., 2015). Additionally, addressing structural barriers that limit access to resources for families facing adversity, such as affordable mental health services and healthy food options, is essential. A successful intervention for affordable mental healthcare services is the National Alliance on Mental Illness (NAMI) Keystone Pennsylvania program. NAMI Keystone PA improves the well-being of children, adolescents, adults, and families that are impacted by mental illness by providing recovery-oriented support, advocacy, and mental health education (National Alliance on Mental Illness, 2025). An example of a structural-level healthy food accessibility program is the Supplemental Nutrition Assistance Program (SNAP). SNAP is a federal program that provides low-income families with financial help to purchase the food that they need, allowing them to have more accessibility to healthier food options without the financial burden (Food and Nutrition Service U.S. Department of Agriculture, 2025). Tailoring

interventions to the specific needs of these populations will improve their ability to engage in healthier behaviors and reduce the long-term effects of early life adversity on obesity risk.

Conclusions

This study highlights the complex relations between early life adversity, emotional eating, and obesity risk in adolescence. While cumulative sociodemographic risk was significantly associated with adolescent-reported EAH negative affect, no mediation effect of EAH negative affect on waist circumference was observed. However, the lack of associations with waist circumference may suggest that the effects of early life adversity experienced during adolescence may be a more powerful predictor of obesity risk, which was not measured in this study. These findings contribute to the growing literature on childhood adversity and adolescent obesity risk, emphasizing the need for further research to identify mechanisms underlying the association between early life adversity and obesity risk. Future research should further explore the physiological and neurobiological pathways that impact the relation between ELA and obesity, including the role of allostatic load and other biomarkers of chronic stress. Identifying these pathways is essential for developing targeted interventions that address both the biological and behavioral aspects of obesity prevention in adolescents exposed to early life adversity. Furthermore, longitudinal studies are needed to more thoroughly map the pathways through which exposure to early life adversity shapes later health outcomes, providing deeper insights into how interventions could target these underlying biological processes.

Appendix

TC EAH-C Questionnaire

Imagine that you are eating a meal or snack at home, school, or at a restaurant. Imagine that you eat enough of your meal so that you are no longer hungry.

*In this situation, how often do you **keep** eating because…*

	Never	**Rarely**		**Some-times**	**Often**	**Always**
1. You are feeling sad or depressed?	1	2		3	4	5
2. You are feeling angry or frustrated?	1	2		3	4	5
3. You are feeling anxious or nervous?	1	2		3	4	5
4. You are feeling tired?	1	2		3	4	5
5. You are feeling bored?	1	2		3	4	5
6. The food looks, tastes, or smells so good?	1	2		3	4	5
7. Others are still eating?	1	2		3	4	5

Now imagine that you finished eating a meal or snack some time ago and you are not yet hungry.

*In this situation, how often do you **start** eating because…*

	Never	**Rarely**	**Some-times**	**Often**	**Always**
8. You are feeling sad or depressed?	1	2	3	4	5
9. You are feeling angry or frustrated?	1	2	3	4	5
10. You are feeling anxious or nervous?	1	2	3	4	5
11. You are feeling tired?	1	2	3	4	5
12. You are feeling bored?	1	2	3	4	5
13. The food looks, tastes, or smells so good?	1	2	3	4	5
14. Others are still eating?	1	2	3	4	5

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