

# UC Irvine

## UC Irvine Previously Published Works

### Title

Negative affect, childhood adversity, and adolescents' eating following stress.

### Permalink

<https://escholarship.org/uc/item/97j6b8r1>

### Authors

Kazmierski, Kelly FM

Borelli, Jessica L

Rao, Uma

### Publication Date

2022

### DOI

10.1016/j.appet.2021.105766

Peer reviewed

Negative Affect, Childhood Adversity, and Adolescent's Eating Following Stress

Kelly F. M. Kazmierski,<sup>1</sup> Jessica L. Borelli,<sup>2</sup> & Uma Rao<sup>1,2,3,4,5</sup>

1. University of California, Irvine, Department of Psychiatry and Human Behavior

2. University of California, Irvine, Department of Psychological Science

3. University of California, Irvine, Department of Pediatrics

4. University of California, Irvine, Center for the Neurobiology of Learning and Memory

5. Children's Hospital of Orange County

Correspondence concerning this article should be addressed to Uma Rao, Department of Psychiatry and Human Behavior, University of California, Irvine, [umar@hs.uci.edu](mailto:umar@hs.uci.edu).

### Abstract

Obesity commonly emerges by adolescence and is associated with serious health consequences. Emotional eating (consuming calories, fats, and sugars in response to negative affect) may promote obesity; however, evidence is mixed as to whether negative affect increases obesogenic eating. Early-life adversity may shape malleable neurobiological systems that govern inhibitory control, physiological regulation, coping strategies, and eating behavior, contributing to greater obesogenic eating in response to negative affect. Therefore, this study tested whether childhood adversity moderates the association between negative affect and food consumption in a diverse sample of female adolescents. After completing a childhood adversity assessment, 157 female adolescents (13-17 years; 28.7% African American, 39.5% Hispanic/Latina, 31.8% Non-Hispanic White) rated their negative affect in response to a standard social stress paradigm before consuming a buffet lunch, which was evaluated for calories, added sugars, and solid fats consumed. Results did not support that negative affect exerted a main effect on eating behavior. However, negative affect and childhood adversity interacted to predict calories and solid fats consumed, such that negative affect was associated with more obesogenic eating for those with high adversity exposure but not for those with low adversity exposure. Adversity and affect did not interact to predict added sugars consumed. Findings support that eating patterns in response to negative affect may differ by childhood adversity history. Reducing children's adversity exposure and bolstering emotion regulation techniques for adolescents who have been exposed to adversity may provide pathways to protect health and well-being by reducing maladaptive eating patterns.

*Keywords:* emotional eating, childhood adversity, adolescence, obesity, negative affect

## 1. Introduction

Obesity constitutes one of the greatest modifiable risks to health in the United States (U.S. Department of Health and Human Services, 2020). Through its association with a host of adverse health outcomes, such as diabetes, cancer, and heart disease, obesity is a leading contributor to disability and early death (National Institute of Health, 2013; Zhang, Rexrode, Van Dam, Li, & Hu, 2008). Moreover, obesity, once established, is resistant to treatment, as even obese individuals who are able to lose weight typically regain it (e.g., Tsai & Wadden, 2005). Therefore, understanding how and for whom obesogenic behaviors, such as emotional eating, arise, and doing so early in development, before the persistent and progressive course of obesity and its associated chronic health conditions have taken root, is critical in order to preserve health.

Emotional eating, the consumption of calories, sugars, and fats in response to negative affect, is associated with obesity and longitudinally predicts weight gain (Frayn & Knäuper, 2018; Ganley, 1989; Koenders & van Strien, 2011; van Strien, Herman & Verheijden, 2012). Consuming energy-rich, sweet, and fatty foods may regulate stress-induced negative affect by producing immediate hedonic pleasure, regulating opioidergic and dopaminergic pathways in the brain, enhancing the activity of the serotonergic system, and terminating activation of the hypothalamic-pituitary-adrenal (HPA) axis (Adam & Epel, 2007; Dallman et al., 2003; Gibson, 2006; Macht & Mueller, 2007). Although emotional eating provides short-term regulation, repeatedly consuming highly palatable foods in response to negative affect may lead to adaptation of these homeostatic systems, such that individuals eat increasingly greater quantities of energy-dense, sweet, and fatty foods in order to counteract negative affect, leading to positive energy balance and the development of obesity (Gibson, 2006).

Women are at higher risk for both emotional eating (Grunberg, & Straub, 1992;

O'Connor & Conner, 2011) and obesity (Hales, Carroll, Fryar, & Ogden, 2017). Moreover, adolescence constitutes a critical period for the development and course of obesity (Alberga, Sigal, Goldfield, Prud'homme, & Kenny, 2012), with emotional eating in adolescence linking earlier psychosocial risk factors to later adiposity (Shriver et al., 2019). Therefore, identifying factors that shape emotional eating among female adolescents may be particularly important for understanding developmental trajectories of obesity-related health.

Patterns of eating in response to stress-related negative affect are highly variable, with both appetite stimulation and suppression reported. In a review of experimental studies in low-risk adult populations, 43% of studies report increased eating in response to emotion, 39% report decreased eating in response to emotion, and 26% report no association between emotion and eating behavior (Macht, 2008); notably, however, this review pooled studies of eating in response to both positive and negative affect. Findings are also mixed in studies employing more ecologically valid daily diary methods (e.g., O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Stone & Brownell, 2004). Among adolescents, in a systematic review and meta-analysis, stress was frequently, but not consistently, associated with obesogenic eating behaviors (Hill, Moss, Sykes-Muskett, Conner, & O'Connor, 2018); however, associations between stress and eating may not be identical to associations between negative emotion and eating.

In response to the heterogeneity in affect-eating behavior associations, research has shifted towards the identification of moderators. To date, self-reported characteristic dietary behaviors, such as restrained and emotional eating, have received the most attention as possible moderators. Meta-analyses and systematic reviews have identified that restrained eaters (those who attempt to restrict food intake) and emotional eaters (those who report eating more when emotionally distressed generally) increase food intake in response to negative affect or stress

exposure (O'Connor & Conner, 2011; Cardi, Leppanen, & Treasure, 2015; Devonport, Nicholls, & Fullerton, 2019; Evers, Dingemans, Junghans, & Boevé, 2018). In contrast, relatively little research has focused on psychosocial factors that identify those at risk for eating in response to negative affect, though some evidence supports that use of emotion regulation strategies (Evers, Marijn Stok, & de Ridder, 2010), personality traits (O'Connor & O'Connor, 2004), and trait mindfulness (Pidgeon, Lacota, & Champion, 2013) may be candidate moderators among adults.

Childhood adversity may also shape whether negative affect induces obesogenic eating, by programming physiology and behavior during plastic developmental periods. Major adverse experiences in childhood, such as physical abuse, neglect, or the death of a caregiver, pose acute, uncontrollable sources of stress, which exceed children's coping resources. Exposure to these stressors might promote emotional eating via several pathways. First, childhood adversities evoke substantial, sustained, or repeated activation of the HPA axis; these forms of activation create wear-and-tear that recalibrates HPA functioning (Danese & McEwen, 2012) to both promote the consumption of highly palatable, energy-rich food and to increase the regulatory effect of eating on HPA activation (Dallman et al., 2003; Danese & Tan, 2014). Second, early adversities shape brain structure and function in ways that reduce inhibitory control (e.g., reduced volume and/or altered activation of the prefrontal cortex, Hart & Rubia, 2012), which may impair one's ability to resist impulses to engage in hedonic behaviors, such as eating, when under the cognitive load of negative affect (Hagger, Wood, Stiff, & Chatzisarantis, 2010; Herman & Polivy, 1984). Third, childhood adversity modifies dopaminergic reward circuits and reduces responsivity to rewarding activities, such as eating, which may lead to greater reward-seeking behavior (i.e., palatable food consumption) in order to reduce negative affect (Cottone et al., 2009; Duffy, McLaughlin, & Green, 2018). Finally, as children lack the ability to cope with

uncontrollable adverse experiences via direct, problem-focused strategies, they may acquire more emotion-focused or avoidant coping strategies, such as consuming rewarding foods, to cope with stress-induced negative affect (Leitenberg, Gibson, & Novy, 2004; Wadsworth, 2015).

Indeed, diverse forms of childhood adversity show robust associations with adult obesity (Danese & Tan, 2014; Hemmingsson, Johansson, & Reynisdottir, 2014), with some evidence supporting a dose-response relationship between adversity and obesity (e.g., Williamson, Thompson, Anda, Dietz, & Felitti, 2002). Whether these associations are due to adversity increasing the propensity to eat in response to negative affect is less clear, however. Evidence from clinical samples supports that childhood physical, sexual, and emotional abuse increase the odds of a binge eating disorder diagnosis in adolescence or adulthood (see Caslini et al., 2016 for meta-analysis). However, one study that directly measured calories consumed following an acute social-evaluative stressor found that energy consumption did not differ as a function of adverse childhood experiences (Wingenfield et al., 2017), though this study did not assess whether stress-related negative affect was differentially associated with food consumption based on adversity. Additional evidence that adversity might shape eating in response to emotion comes from predominantly cross-sectional mediation studies, which support that adverse childhood experiences, such as violence exposure or abuse, influence adult obesity through stress-related or binge eating (Greenfield & Marks, 2009; Rohde et al., 2008). However, these findings do not clarify whether childhood adversity merely increases negative emotionality, which contributes to obesity by providing more frequent opportunities to eat in response to negative affect (e.g., Midei, Matthews, & Bromberger, 2010), or whether adversity shapes regulatory systems, such that obesogenic eating is a more likely response to negative affect when it occurs. One cross-sectional mediation study supports that childhood trauma contributes to emotional eating through

emotion dysregulation (Michopoulos et al., 2015), such that individuals exposed to more trauma in childhood find their emotions more overwhelming, urgent, and difficult to cope with, therefore increasing their likelihood of coping by consuming palatable foods to regulate affect. However, existing research has relied primarily on self-reports of emotional eating, which correspond poorly to eating behavior in the lab or in the natural environment and are vulnerable to reporting biases (Bongers & Jansen, 2016). Observational data are needed to determine whether a history of adversity amplifies associations between negative affect and highly palatable food consumption.

Adolescence is a critical period for the development, course, and prevention of obesity, marked by changes in body composition, diet, and physical activity (Alberga et al., 2012). Adolescents face increasing exposure to stress (Ge, Natsuaki, & Conger, 2006), emotional reactivity and dysregulation (Ahmed, Bittencourt-Hewitt, & Sebastian, 2015), and engagement in health risk behaviors, such as emotional eating (Wardle et al., 1992). In adolescence, the effects of psychosocial factors on eating can be detected, allowing for the identification of risky health trajectories before obesity-related disease is established. For instance, in a longitudinal study of abused and non-abused girls, groups did not differ in obesity rates during childhood, and only trended towards showing group differences in adolescence; however, by early adulthood, abused women were more likely to be obese than were non-abused women (Noll, Zeller, Trickett, & Putnam, 2007), suggesting that childhood adversity may contribute to obesogenic processes during adolescence that progress to obesity by adulthood. Therefore, understanding how negative affect and childhood adversity drive adolescents' obesogenic behaviors may elucidate targets for prevention efforts that can be administered during this sensitive developmental window.

The present study builds on the literature by measuring the association between negative



affect and observed eating behavior and assessing whether this association differs based on exposure to childhood adversity. Constructs are measured in a diverse sample of female adolescents, in order to assess obesogenic processes in at-risk groups during an influential developmental period. Negative affect is assessed following exposure to a standardized stressor, in order to isolate the effect of negative affect from that of stress exposure, as individuals differ in the degree to which standardized stressors evoke negative affect (e.g., Aldao, McLaughlin, Hatzenbuehler, & Sheridan, 2014; Krkovic, Clamor, & Lincoln, 2018). Additionally, observational measures of food consumption are employed, given the limitations of self-reported emotional eating measures (Bongers & Jansen, 2016). Finally, models adjust for the effect of chronic stress on dietary behavior, to isolate the effect of early adversity, as childhood adversity is associated with experiencing a greater number of subsequent stressful life events (Hazel, Hammen, Brennan & Najman, 2008), and adolescent stress is also associated with obesogenic eating (Hsu & Raposa, 2021). Hypothesis 1 predicts that adolescents who report more negative affect following a stressor will consume more calories, solid fats, and added sugars. Hypothesis 2 predicts that the relationship between negative affect and food consumption will be moderated by childhood adversity, such that adolescents with higher levels of childhood adversity will exhibit greater obesogenic food consumption in association with higher negative affect, but adolescents with lower levels of childhood adversity will be buffered from this association.

## **2 Methods**

### **2.1 Participants**

Participants included 157 female adolescents (aged 13-17 years,  $M = 15.27$ ,  $SD = 1.47$ ), recruited to participate in a larger, ongoing study (Authors, in press) of adolescent stress and obesity-related health, who completed assessments of childhood adversity as well as negative

affect and food consumption following a standardized stressor. Participants were recruited from the community via fliers, online advertisements, and word of mouth. Participants were diverse in terms of race/ethnicity (28.7% African American, 39.5% Hispanic/Latina, 31.8% Non-Hispanic White), body-mass index (58.6% normal weight, 22.9% overweight, 18.5% obese), and annual family income (12.1% < \$25,000, 21.7% \$25-49,999, 24.8% \$50-99,999, 41.4% > \$100,000).

## **2.2 Procedures**

All procedures were approved by the Institutional Review Board (XXX IRB #2017-3441). Comprehensive information regarding procedures used in the larger study from which data are drawn are described elsewhere (Authors, in press); therefore, only procedures used to collect the present data are described. During an initial lab visit, adolescents provided assent and parents/guardians provided consent. Parents and adolescents both provided reports of demographics and completed standardized interviews assessing adolescents' exposure to adverse childhood experiences prior to age 10 years. Adolescents also completed a standardized interview assessing chronic stress over the preceding six months. Adolescent height and weight were measured to calculate body mass index (BMI).

Subsequently, adolescents returned to the lab to complete a stress-related food consumption paradigm. Adolescents arrived at the lab after fasting overnight (from 10 P.M. until their scheduled visit at 8-9 A.M.) and were provided a standard 210 calorie breakfast, consisting of a cereal bar and juice box. Following a 2.75 hour relaxation period spent watching movies with neutral content, adolescents were administered the Trier Social Stress Test (TSST), a 15-minute acute social-evaluative stressor (Buske-Kirschbaum et al., 1997). During the TSST, adolescents had five minutes to prepare and five minutes to present a story. Following the improvised story, adolescents completed a five-minute mental arithmetic task. Adolescents were

told that their performance was being evaluated, and both tasks were conducted in front of a panel of judges and a video camera. Adolescents completed a brief report of negative affect on six occasions surrounding the stress task (fifteen minutes before, immediately before, immediately after, ten minutes after, twenty minutes after, and thirty minutes after the task).

Thirty minutes after the stressor concluded, participants were offered a buffet lunch, composed of a wide variety of foods that adolescents commonly eat, which were diverse in nutritional and caloric content. Participants were instructed to eat whatever they would like, then left alone for 40 minutes. Following the meal, participants were debriefed regarding the stressor task.

## **2.3 Measures**

### **2.3.1 Negative Affect.**

Negative affect was measured using the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988), administered on six occasions surrounding the TSST (two preceding and four following the TSST). At each time point, participants rated the extent to which they felt each of eleven negative emotions (e.g., *upset*, *scared*) on a 5-point Likert Scale (1 = *Very slightly or not at all*, 5 = *Extremely*). Negative affect was calculated at each time-point as the mean of these eleven emotion ratings. The maximum negative affect score reached following the stressor (sampling occasions 3-6) was used in analyses.

### **2.3.2 Childhood Adversity.**

The Childhood Adversity Interview (CAI, Dienes et al., 2006; Henry & Hammen, 1988) assesses seven types of adversity commonly experienced in childhood: separation from or loss of caregivers; illness, injury, or loss; physical neglect; emotional abuse; physical abuse/assault; witnessing violence in the home; and sexual abuse/assault. Adolescents and parents were

interviewed separately about these experiences by a trained rater, who scored each domain of adversity on a 5-point scale, where 1 represents no adversity, 3 represents definite adversity, and 5 represents extreme adversity. Information from both parents and children were used to make consensus ratings describing the degree of adversity experienced in each domain; interviewers use information from both interviews to assign scores based on both severity and frequency of each type of adverse experience. When parent and youth reports differed, the reporter who described the abuse as more severe was prioritized in generating the consensus score, except in cases where the adversity occurred too early in development for the child to personally remember the experience, in which case the parent report was prioritized. Raters underwent extensive training and were required to achieve a minimum inter-rater reliability of  $r = .85$  on a training sample of interviews before administering and scoring the CAI with study participants. Only adverse experiences occurring before age 10 were included. Consensus scores were summed across the 7 categories of adversity, such that possible total scores range from 7 to 35.

### ***2.3.3 Food Consumption.***

Foods offered in the buffet before and after the food consumption period were measured using standard metrics. Calories, added sugar, and solid fat content of consumed foods were calculated using the Nutrition Data System for Research (NDSR, Feskanich, Sielaff, Chong, & Buzzard, 1989), a computerized program which employs the University of Minnesota Nutrition Coordinating Center (NCC) Food and Nutrient Database of nutritional information.

### ***2.3.4 Chronic Stress.***

Adolescents' experiences of chronic stress over the preceding six months were measured using the Chronic and Episodic Stress Interview (CESI, Hammen et al., 1987, Hammen, Adrian, & Hiroto, 1988). This interview assessment of chronic stress has demonstrated good reliability

and validity (e.g., Daley, Hammen, & Rao, 2000; Hammen, et al., 2009). Raters were trained to achieve a minimum inter-rater reliability of  $r = .85$  before administering interviews. Raters interviewed adolescents regarding eight common domains of chronic stress (close friendships, romantic relationships, social life, family relationships, work, finances, health, and academics). Stressful experiences in each domain were rated on a scale of one (representing no stressful experiences) to five (representing extreme stress), and the scores in all domains were averaged to compute the overall chronic stress score.

### **2.3.5 Demographics.**

Parents reported adolescents' age. BMI was calculated from adolescents' in-lab height and weight measurements. Height was measured to the nearest 0.1 centimeter on a free-standing stadiometer and weight was measured to the nearest 0.1 kilogram on a digital scale. BMI percentiles were calculated using age, height, and weight, according to the Centers for Disease Control and Prevention (CDC) growth charts (CDC, 2020). Socioeconomic status (SES) was calculated based on parent report of education and occupation of up to two parental figures who contribute to the adolescent's living expenses (Hollingshead, 1957).

## **2.4 Analytic Plan**

Hypotheses were tested using hierarchical, linear regression models in SPSS Version 26. Sensitivity power analyses of interaction models (Hypothesis 2) support that small effects ( $f^2 = 0.05$ ) can be detected with 80% power ( $\alpha = .05$ ) in a sample of 157. In order to ensure that the assumptions of regression were met, raw values were reviewed and values more than three standard deviations beyond the mean for each outcome variable (calories [ $N = 2$ ], solid fats [ $N = 1$ ], and added sugars [ $N = 2$ ] consumed) were reduced to  $M + 3 SD$  (Aguinis et al., 2013).

Demographic covariates (BMI, SES, age) and chronic stress were entered at step one. Negative affect was entered at step two. Childhood adversity was entered at step three. Variables that compose the interaction term (negative affect, childhood adversity) were mean-centered before entry in regressions. The interaction term (computed as the product of negative affect and childhood adversity) was entered in the final step. Interactions were probed at the region of significance using the Johnson-Neyman technique. Standardized conditional effect sizes were calculated at the region of significance using semi-partial correlations (see Bodner, 2017). Equations were plotted across all observed values of the independent variable (negative affect) and at the region of significance for the moderator (childhood adversity); when no observed values of the moderator were significant, high and low values were plotted as the highest or lowest value of childhood adversity observed.

### 3. Results

#### 3.1 Descriptive Statistics

Table 1 presents descriptive statistics and inter-item correlations for all study variables. Mean childhood adversity was relatively low in our non-clinical sample ( $M = 8.46$ , observed range = 7-16, possible range = 7-35). The most commonly endorsed forms of adversity were separation from or loss of a caregiver (30.6% of sample); illness, injury, or loss (25.5% of sample); physical abuse (24.8% of sample), and emotional abuse (19.7% of sample). Neither negative affect nor childhood adversity were associated with any food consumption variable, though adolescent's experiences of chronic stress were positively associated with solid fats consumed ( $r = .19, p = .02$ ). Adolescents who experienced more childhood adversity reported more chronic stress ( $r = .37, p < .001$ ) and experienced more negative affect ( $r = .20, p = .01$ ). Chronic stress in adolescence was associated with negative affect following the stressor ( $r = .23,$

$p = .004$ ). Adolescents with higher BMIs reported more childhood adversity ( $r = .16, p = .05$ ) and more chronic stress ( $r = .18, p = .02$ ); they consumed more calories ( $r = .19, p = .02$ ) and solid fats ( $r = .16, p = .04$ ).

### 3.2 Hypothesis 1

Hypothesis 1 was not supported, as negative affect was not associated with calories ( $b = 23.90, SE = 39.55, p = .54$ ), solid fats ( $b = 0.60, SE = 1.14, p = .60$ ), or added sugars ( $b = 1.40, SE = 1.62, p = .39$ ) consumed (see Table 2, Model 2).

### 3.3 Hypothesis 2

In partial support of Hypothesis 2, negative affect and childhood adversity interacted to predict calories and solid fats consumed, but not added sugars consumed (see Table 2, Model 4). Childhood adversity moderated the effect of negative affect on calories consumed ( $b = 37.33, SE = 16.36, p = .02$ ) such that adolescents with high adversity exposure ( $CAI \geq 11.31, M + 1.42 SD$ ) consumed more energy with greater negative affect ( $b = 107.04, SE = 54.17, p = .05$ , semi-partial  $r = 0.22$ ), whereas at the lowest level of childhood adversity observed (no adversity,  $CAI = 7, M - 1.46 SD$ ), negative affect was not associated with lower energy consumption ( $b = -53.78, SE = 51.77, p = .30$ , semi-partial  $r = -.11$ , see Figure 1A). This represents a small-to-moderate change in the magnitude of the effect of negative affect on energy consumption ( $\Delta r = 0.33$ , see Bodner, 2017). Similarly, childhood adversity moderated the effect of negative affect on solid fats consumed ( $b = 1.27, SE = 0.47, p = .008$ ), such that at high levels of childhood adversity ( $CAI \geq 11.21, M + 1.42 SD$ ), more negative affect was associated with more fat consumption ( $b = 2.76, SE = 1.40, p = .050$ , semi-partial  $r = .19$ ), whereas at the lowest level of childhood adversity observed (no adversity,  $CAI = 7, M - 1.46 SD$ ), negative affect was not associated with solid fat consumption ( $b = -2.06, SE = 1.48, p = .17$ , semi-partial  $r = -.14$ , see Figure 1B). This

represents a small-to-moderate change in the magnitude of the effect of negative affect on energy consumption ( $\Delta r = 0.23$ ). In contrast, the interaction of negative affect and childhood adversity did not predict added sugars consumed ( $b = 1.07$ ,  $SE = 0.68$ ,  $p = .12$ , see Figure 1C).

#### 4. Discussion

The current study tested the interactive effects of negative affect and childhood adversity on food consumption following a social stressor in a diverse sample of female adolescents. Negative affect did not exert a main effect on any food consumption measure. Rather, the effect of negative affect was conditional on exposure to childhood adversity, such that for adolescents high in adversity, more negative affect was associated with greater consumption of calories and solid fats but not added sugars. Adolescents with low levels of adversity were buffered from affect-eating associations.

Results are consistent with prior literature suggesting that negative affect has heterogeneous associations with food consumption (e.g., Macht, 2008); indeed, in our sample, the effect of global negative affect could be detected only when examined in the context of childhood adversity history. In line with theories positing that adverse childhood environments may sensitize youth to respond to negative affect with maladaptive coping behaviors generally (Wadsworth, 2015) and deleterious eating behaviors specifically (Danes & Tan, 2014), female adolescents with substantial adversity histories were vulnerable to consuming more calories and solid fats in the face of negative affect. Notably, effects were detected only at comparatively high levels of adversity, relative to the community sample from which participants were drawn ( $\geq M + 1.42$ - $1.47$   $SD$ ). Consistent with mechanistic frameworks that highlight the potential for repeated, severe, and/or uncontrollable adversities to alter children's physiological regulatory



patterns, inhibitory control, reward sensitivity, and coping strategies (e.g., Danes & Tan, 2014; Wadsworth, 2015), multiple and/or severe childhood adversity exposures may be required in order to produce the neural, endocrine, and behavioral adaptations that may promote obesogenic eating patterns in response to negative emotion. It is therefore notable that even small-to-moderate effects were detected in a community sample, in which markedly elevated childhood adversity scores were uncommon; observed associations may be even more pronounced among those sampled for substantial abuse or neglect. For severely maltreated adolescents, proneness to maladaptive eating patterns in response to negative affect may link childhood adversity to adult obesity (Greenfield & Marks, 2009).

For female adolescents who experienced low-to-moderate levels of adversity, negative affect was not associated with food consumption. This is consistent with findings from a sizable minority of studies, in which affect and eating behavior are not linked (Macht, 2008). However, observed null associations stand in contrast to models that posit that negative-affect-induced appetite suppression may constitute an adaptive response, as intense negative emotion may elicit physiological and behavioral responses that are adaptive to coping with stressors but antithetical to food consumption (Macht, 2008). For instance, the fight-or-flight response to fear prepares organisms to flee a threat, in part by directing resources away from digestion and toward the peripheral muscles needed for escape. Accordingly, appetite suppression is most commonly observed in response to stress in animal studies (Greeno & Wing, 1994). However, which pattern of eating in response to negative emotion (exhibiting reduced appetite versus showing no association between affect and eating behavior) is protective against obesity-related conditions remains to be tested.

Unexpectedly, interaction effects were observed for the consumption of calories and solid

fats, but not for the consumption of added sugars. The smaller, non-significant effects detected in models predicting added sugar consumption may be due, in part, to the differing ways in which female adolescents typically consume added sugars versus solid fats. American youth consume a great deal of added sugars and solid fats, with nearly 40% of their total daily energy consumption deriving from these categories (Reedy & Krebs-Smith, 2010). However, while solid fats are typically consumed as foods (with pizza and grain desserts constituting the top two sources of added fats for 14-18 year-olds), added sugars are most commonly consumed as beverages (with soda and fruit drinks constituting the top two sources of added sugars for this age group, Reedy & Krebs-Smith, 2010). At-risk adolescents may be more inclined to eat, rather than drink, in response to negative affect. For example, in a sample of middle school students, emotional eaters and non-emotional eaters did not differ in their intake of soda, though they did differ in their intake of sweet, energy dense foods (Nguyen-Michel, Unger, & Spruijt-Metz, 2007). Girls increasingly elect to drink artificially-sweetened diet sodas across adolescence (Striegel-Moore et al., 2006); the present study is unable to assess whether participants may have consumed sugar-free sweet beverages in lieu of sugar-sweetened beverages in response to negative affect.

Results from the present study provide preliminary evidence that childhood adversity and female adolescents' eating patterns in response to stress-induced negative affect may each provide worthwhile targets for the development of preventive interventions for obesity. For instance, girls who have a history of childhood adversity may particularly benefit from learning strategies to regulate negative affect (Evers et al., 2010). Already, mindfulness-based interventions have shown promise in reducing emotional eating and may be particularly well-suited to pediatric populations (e.g., Sato & Fahrenkamp, 2016). Additionally, incorporating trauma-informed approaches may enhance the impact of existing obesity interventions (Mason et

al., 2016), as even when weight loss efforts are successful, participants with histories of abuse are markedly more likely to regain lost weight (Felitti & Williams, 1988). Moreover, future biologically-focused interventions may be able to target neurobiological mechanisms (e.g., dopaminergic pathways that influence reward sensitivity) that may underpin associations between affect and obesogenic eating patterns for youth who have experienced early-life adversity (Gibson, 2012).

Notably, chronic stress was associated both with childhood adversity exposure and with solid fat consumption. Female adolescents who have been exposed to childhood adversity may enter risky trajectories, in which early adverse experiences set the stage for later stress exposure, negative affectivity, and obesogenic behavior (Midei et al., 2010; Hazel et al., 2008), with resultant adiposity further generating stress (Michels et al., 2015) and perpetuating risky cycles, whereby stress and adiposity jointly and multiplicatively generate inflammation, insulin resistance, and cardiometabolic disease (Black, 2006). Interventions that reduce adolescents' proximal stress exposure or enhance behavioral, interpersonal, and instrumental resources for coping with stressors may provide complimentary pathways to protecting obesity-related health, particularly for those individuals whose adversity exposure places them at greater risk.

The present study has several limitations. First, endorsement of any particular type of adversity was relatively low in this community sample, providing insufficient variability to test associations between the severity of any discrete form of adversity and eating behavior. Rather, in line with research suggesting that overall childhood adversity shows a dose-response relationship with obesity (e.g., Williamson et al., 2002), a combined, continuous measure of adversity was used, rather than examining specific forms of adversity separately. Therefore, we are unable to conclude whether any of the seven adversities assessed here uniquely contribute to

affect-induced eating. Additionally, although this study employed a rigorous standard interview to assess childhood adversity history, this measure relied on retrospective self-report of parents and children rather than assessing adversities as they occurred in childhood. Fourth, while the social stressor employed provides an ecologically valid experience of social-evaluative stress that evokes negative affect, findings are inherently limited by the laboratory nature of assessments; future studies should extend these findings to naturalistic settings, using daily diary or experience sampling methodologies. Additionally, the TSST may not evoke the forms of negative affect that are most strongly associated with binge eating in the natural environment (e.g., guilt, Berg et al., 2015). Fifth, findings from our all-female sample may not generalize to male adolescents. Sixth, causal conclusions cannot be drawn from these data. Finally, although adversity-induced alterations to neural and/or endocrine systems might account for observed moderation effects, this study is unable to test these mechanisms directly; rather, future large-scale, longitudinal studies that assess HPA activity and brain structure and function are required.

Despite these limitations, the present study is strengthened by its use of an observational measure of eating behavior, particularly given that, to date, the field has relied largely on insufficiently validated self-report instruments (Bongers & Jansen, 2016). Similarly, negative affect is measured directly, rather than assuming that a stress manipulation exerts similar degrees of negative affect across participants. Exposing participants to a stressor without assessing the degree of negative affect provoked may contribute to null findings reported elsewhere in the literature. By focusing on adolescence, the present study is well-situated to inform preventive efforts to mitigate the health impacts of obesity before they become entrenched. Effects of adversity on affect-eating behavior associations were observed even in a community sample of female adolescents, suggesting that modifying either children's adversity exposure or

adolescents' affective response to stress may have the potential to protect against obesogenic behaviors even in non-clinical samples. These results highlight the importance of early experiences in determining how one's immediate emotional responses influence health behaviors and point toward opportunities to protect children from adversity and promote adolescent emotion regulation in order to preserve health.

### Acknowledgements

Funding for this study was provided, in part, by the National Institutes of Health (NIH) grants R01MD010757, R01DA040966, R01MH108155, F32MD014050, and UL1TR001414.

The content is solely the responsibility of the authors and does not necessarily represent the NIH's official views. NIH did not play a role in the study design, collection, analysis or interpretation of the data, writing the manuscript, or the decision to submit the paper for publication. We would like to thank the families that participated in this research, as well as all members of the Biobehavioral Research on Adolescent Development Lab and the Children's Hospital of Orange County who assisted with the data collection, particularly Larissa Chau and Tomas Zurita, who helped coordinate the study. A preliminary version of these data were accepted for presentation at the Society for Research on Adolescence 18th Biennial Meeting. The data collection protocol described here is registered at ClinicalTrials.gov (NCT03369691).

Author Contributions: Dr. Kazmierski conceptualized the research question, conducted data analysis, interpreted findings, and drafted the manuscript. Dr. Borelli contributed to data interpretation and revised the manuscript. Dr. Rao designed the study, obtained financial support, oversaw all study procedures, supervised data analysis and interpretation of study findings, and reviewed and revised the manuscript. All authors have read and approved the final manuscript.

Declarations of Interest: none.

The corresponding author has full access to all data reported in this manuscript. Please contact Dr. Uma Rao with requests for data sharing approval.

## References

- Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior*, *91*(4), 449-458. <http://dx.doi.org/10.1016/j.physbeh.2007.04.011>
- Aguinis, H., Gottfredson, R. K., & Joo, H. (2013). Best-practice recommendations for defining, identifying, and handling outliers. *Organizational Research Methods*, *16*(2), 270-301. <https://doi.org/10.1177/1094428112470848>
- Ahmed, S. P., Bittencourt-Hewitt, A., & Sebastian, C. L. (2015). Neurocognitive bases of emotion regulation development in adolescence. *Developmental Cognitive Neuroscience*, *15*, 11-25. <https://doi.org/10.1016/j.dcn.2015.07.006>
- Alberga, A. S., Sigal, R. J., Goldfield, G., Prud'Homme, D., & Kenny, G. P. (2012). Overweight and obese teenagers: Why is adolescence a critical period? *Pediatric Obesity*, *7*(4), 261-273. <https://doi.org/10.1111/j.2047-6310.2011.00046.x>
- Aldao, A., McLaughlin, K. A., Hatzenbuehler, M. L., & Sheridan, M. A. (2014). The relationship between rumination and affective, cognitive, and physiological responses to stress in adolescents. *Journal of Experimental Psychopathology*, *5*(3), 272-288. <https://doi.org/10.5127/jep.039113>
- Authors (in press). Title omitted for blind review.
- Berg, K. C., Crosby, R. D., Cao, L., Crow, S. J., Engel, S. G., Wonderlich, S. A., & Peterson, C. B. (2015). Negative affect prior to and following overeating-only, loss of control eating-only, and binge eating episodes in obese adults. *International Journal of Eating Disorders*, *48*(6), 641-653. <https://doi.org/10.1002/eat.22401>
- Black, P. H. (2006). The inflammatory consequences of psychologic stress: relationship to

- insulin resistance, obesity, atherosclerosis and diabetes mellitus, type II. *Medical Hypotheses*, 67(4), 879-891. <https://doi.org/10.1016/j.mehy.2006.04.008>
- Bongers, P., & Jansen, A. (2016). Emotional eating is not what you think it is and emotional eating scales do not measure what you think they measure. *Frontiers in Psychology*, 7, 1932. <https://doi.org/10.3389/fpsyg.2016.01932>
- Bodner, T. E. (2017). Standardized effect sizes for moderated conditional fixed effects with continuous moderator variables. *Frontiers in Psychology*, 8, 562. <https://doi.org/10.3389/fpsyg.2017.00562>
- Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine*, 59(4), 419-426. <https://doi.org/10.1097/00006842-199707000-00012> <https://doi.org/10.1097/00006842-199707000-00012>
- Cardi, V., Leppanen, J., & Treasure, J. (2015). The effects of negative and positive mood induction on eating behaviour: A meta-analysis of laboratory studies in the healthy population and eating and weight disorders. *Neuroscience & Biobehavioral Reviews*, 57, 299-309. <https://doi.org/10.1016/j.neubiorev.2015.08.011>
- Caslini, M., Bartoli, F., Crocamo, C., Dakanalis, A., Clerici, M., & Carrà, G. (2016). Disentangling the association between child abuse and eating disorders: A systematic review and meta-analysis. *Psychosomatic Medicine*, 78(1), 79-90. <https://doi.org/10.1097/PSY.0000000000000233>
- Centers for Disease Control and Prevention. (2020). *BMI percentile calculator for child and teen. Metric Version*. <https://www.cdc.gov/healthyweight/bmi/calculator.html>.



Cottone, P., Sabino, V., Roberto, M., Bajo, M., Pockros, L., Frihauf, J. B., ... & Conti, B. (2009).

CRF system recruitment mediates dark side of compulsive eating. *Proceedings of the National Academy of Sciences*, *106*(47), 20016-20020.

<https://doi.org/10.1073/pnas.0908789106>

Daley, S. E., Hammen, C., & Rao, U. (2000). Predictors of first onset and recurrence of major depression in young women during the 5 years following high school graduation. *Journal of Abnormal Psychology*, *109*(3), 525. <https://doi.org/10.1037/0021-843X.109.3.525>

Dallman, M. F., Pecoraro, N., Akana, S. F., La Fleur, S. E., Gomez, F., Houshyar, H., ... & Manalo, S. (2003). Chronic stress and obesity: a new view of “comfort food”. *Proceedings of the National Academy of Sciences*, *100*(20), 11696-11701.

<https://doi.org/10.1073/pnas.1934666100>

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*(1), 29-39.

<https://doi.org/10.1016/j.physbeh.2011.08.019>

Danese A., & Tan M. (2014) Childhood maltreatment and obesity: Systematic review and meta-analysis. *Molecular Psychiatry*, *19*(5), 544–54. <https://doi.org/10.1038/mp.2013.54>

Devonport, T. J., Nicholls, W., & Fullerton, C. (2019). A systematic review of the association between emotions and eating behaviour in normal and overweight adult populations. *Journal of Health Psychology*, *24*(1), 3-24.

<https://doi.org/10.1177/1359105317697813>

Dienes, K. A., Hammen, C., Henry, R. M., Cohen, A. N., & Daley, S. E. (2006). The stress sensitization hypothesis: understanding the course of bipolar disorder. *Journal of Affective Disorders*, *95*(1-3), 43-49. <https://doi.org/10.1016/j.jad.2006.04.009>

- Duffy, K. A., McLaughlin, K. A., & Green, P. A. (2018). Early life adversity and health-risk behaviors: Proposed psychological and neural mechanisms. *Annals of the New York Academy of Sciences, 1428*(1), 151. <https://doi.org/10.1111/nyas.13928>
- Evers, C., Dingemans, A., Junghans, A. F., & Boevé, A. (2018). Feeling bad or feeling good, does emotion affect your consumption of food? A meta-analysis of the experimental evidence. *Neuroscience & Biobehavioral Reviews, 92*, 195-208. <https://doi.org/10.1016/j.neubiorev.2018.05.028>
- Evers, C., Marijn Stok, F., & de Ridder, D. T. (2010). Feeding your feelings: Emotion regulation strategies and emotional eating. *Personality and Social Psychology Bulletin, 36*(6), 792-804. <https://doi.org/10.1177/0146167210371383>
- Felitti, V., & Williams, S. (1998). Long term follow-up and analysis of more than 100 patients who each lost more than 100 pounds. *The Permanente Journal, 2*(3):17–21.
- Feskanich, D., Sielaff, B. H., Chong, K., & Buzzard, I. M. (1989). Computerized collection and analysis of dietary intake information. *Computer Methods and Programs in Biomedicine, 30*(1), 47-57. [https://doi.org/10.1016/0169-2607\(89\)90122-3](https://doi.org/10.1016/0169-2607(89)90122-3)
- Frayn, M., & Knäuper, B. (2018). Emotional eating and weight in adults: a review. *Current Psychology, 37*(4), 924-933. <https://doi.org/10.1007/s12144-017-9577-9>
- Ganley, R. M. (1989). Emotion and eating in obesity: A review of the literature. *International Journal of Eating Disorders, 8*(3), 343-361. [https://doi.org/10.1002/1098-108X\(198905\)8:3<343::AID-EAT2260080310>3.0.CO;2-C](https://doi.org/10.1002/1098-108X(198905)8:3<343::AID-EAT2260080310>3.0.CO;2-C)
- Ge, X., Natsuaki, M. N., & Conger, R. D. (2006). Trajectories of depressive symptoms and stressful life events among male and female adolescents in divorced and nondivorced families. *Development and psychopathology, 18*(1), 253-273.

<https://doi.org/10.1017/S0954579406060147>

Gibson, E. L. (2006). Emotional influences on food choice: Sensory, physiological and psychological pathways. *Physiology & Behavior*, *89*(1), 53-61.

<https://doi.org/10.1016/j.physbeh.2006.01.024>

Gibson, E. L. (2012). The psychobiology of comfort eating: Implications for neuropharmacological interventions. *Behavioural Pharmacology*, *23*(5 and 6), 442-460.

<https://doi.org/10.1097/FBP.0b013e328357bd4e>

Greenfield, E. A., & Marks, N. F. (2009). Violence from parents in childhood and obesity in adulthood: Using food in response to stress as a mediator of risk. *Social Science & Medicine*, *68*(5), 791-798. <https://doi.org/10.1016/j.socscimed.2008.12.004>

Greeno, G. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin*, *115*, 444–464. <https://doi.org/10.1037/0033-2909.115.3.444>

Grunberg, N. E., & Straub, R. O. (1992). The role of gender and taste class in the effects of stress on eating. *Health Psychology*, *11*, 97-100. <https://doi.org/10.1037/0278-6133.11.2.97>

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. (2010). Ego depletion and the strength model of self-control: a meta-analysis. *Psychological Bulletin*, *136*(4), 495.

<https://doi.org/10.1037/a0019486>

Hales, C. M., Carroll, M. D., Fryar, C. D., & Ogden, C. L. (2017). Prevalence of obesity among adults and youth: United States, 2015–2016. *NCHS Data Brief*, *288*, 1-8.

Hammen, C., Adrian, C., Gordon, D., Burge, D., Jaenicke, C., & Hiroto, D. (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology*, *96*, 190-198. <https://doi.org/10.1037/0021-843X.96.3.190>

Hammen, C., Adrian, C., & Hiroto, D. (1988). A longitudinal test of the attributional

- vulnerability model in children at risk for depression. *British Journal of Clinical Psychology*, 27(1), 37-46. <https://doi.org/10.1111/j.2044-8260.1988.tb00751.x>
- Hammen, C., Kim, E. Y., Eberhart, N. K., & Brennan, P. A. (2009). Chronic and acute stress and the prediction of major depression in women. *Depression and Anxiety*, 26(8), 718-723. <https://doi.org/10.1002/da.20571>
- Hart, H., & Rubia, K. (2012). Neuroimaging of child abuse: A critical review. *Frontiers in Human Neuroscience*, 6, 52. <https://doi.org/10.3389/fnhum.2012.00052>
- Hazel, N. A., Hammen, C., Brennan, P. A., & Najman, J. (2008). Early childhood adversity and adolescent depression: The mediating role of continued stress. *Psychological Medicine*, 38(4), 581-589. <https://doi.org/10.1017/S0033291708002857>
- Hemmingsson, E., Johansson, K., & Reynisdottir, S. (2014). Effects of childhood abuse on adult obesity: A systematic review and meta-analysis. *Obesity Reviews*, 15(11), 882-893. <https://doi.org/10.1111/obr.12216>
- Henry, R., & Hammen, C. (1998). *The Childhood Adversity Interview*. Los Angeles, CA: University of California at Los Angeles.
- Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. *Research Publications-Association for Research in Nervous and Mental Disease*, 62, 141.
- Hill, D. C., Moss, R. H., Sykes-Muskett, B., Conner, M., & O'Connor, D. B. (2018). Stress and eating behaviors in children and adolescents: Systematic review and meta-analysis. *Appetite*, 123, 14-22. <https://doi.org/10.1016/j.appet.2017.11.109>
- Hollingshead, AB. *Two factor index of social position*. Memo. New Haven, Connecticut: Yale University. 1957.

- Hsu, T., & Raposa, E. B. (2021). Effects of stress on eating behaviours in adolescents: a daily diary investigation. *Psychology & Health, 36*(2), 236-251.  
<https://doi.org/10.1080/08870446.2020.1766041>
- Koenders, P. G., & van Strien, T. (2011). Emotional eating, rather than lifestyle behavior, drives weight gain in a prospective study in 1562 employees. *Journal of Occupational and Environmental Medicine, 53*(11), 1287-1293.  
<https://doi.org/10.1097/JOM.0b013e31823078a2>
- Krkovic, K., Clamor, A., & Lincoln, T. M. (2018). Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery. *Psychoneuroendocrinology, 94*, 112-120. <https://doi.org/10.1016/j.psyneuen.2018.04.028>
- Leitenberg, H., Gibson, L. E., & Novy, P. L. (2004). Individual differences among undergraduate women in methods of coping with stressful events: The impact of cumulative childhood stressors and abuse. *Child Abuse & Neglect, 28*(2), 181-192.  
<https://doi.org/10.1016/j.chiabu.2003.08.005>
- Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite, 50*(1), 1-11.  
<https://doi.org/10.1016/j.appet.2007.07.002>
- Macht, M., & Mueller, J. (2007). Immediate effects of chocolate on experimentally induced mood states. *Appetite, 49*(3), 667-674. <https://doi.org/10.1016/j.appet.2007.05.004>
- Mason, S. M., Austin, S. B., Bakalar, J. L., Boynton-Jarrett, R., Field, A. E., Gooding, H. C., ... & Sogg, S. (2016). Child maltreatment's heavy toll: The need for trauma-informed obesity prevention. *American Journal of Preventive Medicine, 50*(5), 646. <https://doi.org/10.1016/j.amepre.2015.11.004>
- Michels, N., Sioen, I., Boone, L., Clays, E., Vanaelst, B., Huybrechts, I., & De Henauw, S.

- (2015). Cross-lagged associations between children's stress and adiposity: The children's body composition and stress study. *Psychosomatic Medicine*, 77(1), 50-58.  
<https://doi.org/10.1097/PSY.0000000000000122>
- Michopoulos, V., Powers, A., Moore, C., Villarreal, S., Ressler, K. J., & Bradley, B. (2015). The mediating role of emotion dysregulation and depression on the relationship between childhood trauma exposure and emotional eating. *Appetite*, 91, 129-136.  
<https://doi.org/10.1016/j.appet.2015.03.036>
- Midei, A. J., Matthews, K. A., & Bromberger, J. T. (2010). Childhood abuse is associated with adiposity in mid-life women: Possible pathways through trait anger and reproductive hormones. *Psychosomatic Medicine*, 72(2), 215.  
<https://doi.org/10.1097/PSY.0b013e3181cb5c24>
- National Institute of Health, National Heart, Lung, and Blood Institute. (2013). Managing overweight and obesity in adults: Systematic evidence review from the obesity expert panel. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/obesity-evidence-review.pdf>.
- Nguyen-Michel, S. T., Unger, J. B., & Spruijt-Metz, D. (2007). Dietary correlates of emotional eating in adolescence. *Appetite*, 49(2), 494-499.  
<https://doi.org/10.1016/j.appet.2007.03.005>
- Noll, J. G., Zeller, M. H., Trickett, P. K., & Putnam, F. W. (2007). Obesity risk for female victims of childhood sexual abuse: A prospective study. *Pediatrics*, 120(1), e61-e67.  
<https://doi.org/10.1542/peds.2006-3058>
- O'Connor, D.B. & Conner, M. (2011). Effects of stress on eating behavior. Chapter in *Handbook of Stress Science: Psychology, Biology and Health*. Richard J. Contrada and Andrew

- Baum (Eds). New York: Springer Publishing Company. pp 275-286.
- O'Connor, D. B., Jones, F., Conner, M., McMillan, B., & Ferguson, E. (2008). Effects of daily hassles and eating style on eating behavior. *Health Psychology, 27*(1S), S20.  
<https://doi.org/10.1037/0278-6133.27.1.S20>
- O'Connor, D. B., & O'Connor, R. C. (2004). Perceived changes in food intake in response to stress: The role of conscientiousness. *Stress & Health, 20*, 279–291.  
<https://doi.org/10.1002/smi.1028>
- Pidgeon, A., Lacota, K., & Champion, J. (2013). The moderating effects of mindfulness on psychological distress and emotional eating behaviour. *Australian Psychologist, 48*(4), 262-269. <https://doi.org/10.1111/j.1742-9544.2012.00091.x>
- Reedy, J., & Krebs-Smith, S. M. (2010). Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. *Journal of the American Dietetic Association, 110*(10), 1477-1484. <https://doi.org/10.1016/j.jada.2010.07.010>
- Rohde, P., Ichikawa, L., Simon, G. E., Ludman, E. J., Linde, J. A., Jeffery, R. W., & Operskalski, B. H. (2008). Associations of child sexual and physical abuse with obesity and depression in middle-aged women. *Child Abuse & Neglect, 32*(9), 878-887.  
<https://doi.org/10.1016/j.chiabu.2007.11.004>
- Sato, A. F., & Fahrenkamp, A. J. (2016). From bench to bedside: Understanding stress-obesity research within the context of translation to improve pediatric behavioral weight management. *Pediatric Clinics, 63*(3), 401-423. <https://doi.org/10.1016/j.pcl.2016.02.003>
- Shriver, L. H., Dollar, J. M., Lawless, M., Calkins, S. D., Keane, S. P., Shanahan, L., & Wideman, L. (2019). Longitudinal associations between emotion regulation and adiposity in late adolescence: Indirect effects through eating behaviors. *Nutrients, 11*(3), 517.

<https://doi.org/10.3390/nu11030517>

Stone, A. A., & Brownell, K. D. (1994). The stress-eating paradox: Multiple daily measurements in adult males and females. *Psychology and Health*, 9(6), 425-436.

<https://doi.org/10.1080/08870449408407469>

Striegel-Moore, R. H., Thompson, D., Affenito, S. G., Franko, D. L., Obarzanek, E., Barton, B.

A., ... & Crawford, P. B. (2006). Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *The Journal of Pediatrics*, 148(2), 183-187. <https://doi.org/10.1016/j.jpeds.2005.11.025>

Tsai, A. G., & Wadden, T. A. (2005). Systematic review: An evaluation of major commercial weight loss programs in the United States. *Annals of Internal Medicine*, 142(1), 56-66.

<https://doi.org/10.7326/0003-4819-142-1-200501040-00012>

U.S. Department of Health and Human Services, Office of Disease Prevention and Health

Promotion (2020). 2020 Leading Health Indicators: Nutrition, physical activity, and obesity. <http://www.healthypeople.gov/2020/leading-health-indicators/2020-lhi-topics/Nutrition-Physical-Activity-and-Obesity>.

van Strien, T., Herman, C. P., & Verheijden, M. W. (2012). Eating style, overeating and weight gain. A prospective 2-year follow-up study in a representative Dutch sample. *Appetite*,

59(3), 782-789. <https://doi.org/10.1016/j.appet.2012.08.009>

Wadsworth, M. E. (2015). Development of maladaptive coping: A functional adaptation to chronic, uncontrollable stress. *Child Development Perspectives*, 9(2), 96-100.

<https://doi.org/10.1111/cdep.12112>

Wardle, J., Marsland, L., Sheikh, Y., Quinn, M., Fedoroff, I., & Ogden, J. (1992). Eating style and eating behaviour in adolescents. *Appetite*, 18(3), 167-183.



[https://doi.org/10.1016/0195-6663\(92\)90195-C](https://doi.org/10.1016/0195-6663(92)90195-C)

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54(6), 1063. <https://doi.org/10.1037/0022-3514.54.6.1063>

Williamson, D. F., Thompson, T. J., Anda, R. F., Dietz, W. H., & Felitti, V. (2002). Body weight and obesity in adults and self-reported abuse in childhood. *International Journal of Obesity*, 26(8), 1075-1082. <https://doi.org/10.1038/sj.ijo.0802038>

Wingenfield, K., Kuehl, L. K., Boeker, A., Schultebrasucks, K., Ritter, K., Hellmann-Regen, J., ... & Spitzer, C. (2017). Stress reactivity and its effects on subsequent food intake in depressed and healthy women with and without adverse childhood experiences. *Psychoneuroendocrinology*, 80, 122-130. <https://doi.org/10.1016/j.psyneuen.2017.03.014>

Zhang, C., Rexrode, K. M., Van Dam, R. M., Li, T. Y., & Hu, F. B. (2008). Abdominal obesity and the risk of all-cause, cardiovascular, and cancer mortality. *Circulation*, 117(13), 1658-1667. <https://doi.org/10.1161/CIRCULATIONAHA.107.739714>

Table 1.

*Descriptive statistics and inter-item correlations for all study variables.*

Variable	M (SD)	Range	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Calories Consumed <sup>1</sup>	995.54 (388.18)	0.82- 2212.99	-	-	-	-	-	-	-	-	-
2. Added Sugars Consumed <sup>1</sup>	26.09 (16.06)	0.00- 77.06	.66***	-	-	-	-	-	-	-	-
3. Solid Fats Consumed <sup>1</sup>	22.29 (11.29)	0.00- 56.24	.85***	.54***	-	-	-	-	-	-	-
4. Negative Affect	1.99 (0.80)	1.00- 4.55	.06	.08	.06	-	-	-	-	-	-
5. Childhood Adversity	8.46 (1.94)	7-16	.08	.07	.11	.20*	-	-	-	-	-
6. Chronic Stress	2.08 (0.42)	1.30- 3.15	.12	.11	.19*	.23**	.37***	-	-	-	-
7. Age	15.27 (1.47)	13-17	-.09	-.16*	-.11	.13	.09	.19*	-	-	-
8. SES	43.40 (11.62)	13-61	<-.01	.11	-.07	.04	-.13 <sup>†</sup>	-.19*	-.13	-	-
9. BMI	73.89 (22.58)	11-99	.19*	.13	.16*	<.01	.16*	.18*	-.01	-.11	-

1. Outlying values (greater than 3 standard deviations beyond the mean) were reduced to  $M + 3 SD$ . Two outlying values were reduced for calories consumed, two outlying values were reduced for added sugars consumed, and one outlying value was observed for solid fats consumed.

*Note.* BMI = body mass index percentile, SES = socioeconomic status.

<sup>†</sup>  $p < .10$ , \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$

Table 2.

*Hierarchical regression analysis examining childhood adversity as a moderator of the effect of negative affect on ad libitum food consumption (calories, solid fats, and added sugars) among female adolescents following an acute stressor.*

	Calories				Solid Fats				Added Sugars			
	b (SE)	F	F $\Delta$	Adj R <sup>2</sup>	b (SE)	F	F $\Delta$	Adj R <sup>2</sup>	b (SE)	F	F $\Delta$	Adj R <sup>2</sup>
Model 1		2.22†	2.22†	.03		2.93*	2.93*	.05		2.96*	2.96*	.05
Age	-28.50 (21.34)				-1.11† (0.62)				-1.87* (0.87)			
SES	0.81 (2.70)				-.03 (0.08)				0.17 (0.11)			
BMI	2.87† (1.39)				.06 (0.04)				0.08 (0.06)			
Chronic Stress	107.89 (75.98)				4.93* (2.19)				5.61† (3.11)			
Model 2		1.84	0.37	0.3		2.39*	0.28	.04		2.41*	0.74	.05
Age	-29.73 (21.78)				-1.15† (0.62)				-1.94* (0.88)			
SES	0.65 (2.72)				-.04 (0.08)				0.16 (0.11)			
BMI	2.90* (1.39)				.06 (0.04)				0.08 (0.06)			
Chronic Stress	97.27 (78.14)				4.67* (2.24)				4.99 (3.20)			
Negative Affect	23.90 (21.54)				0.60 (1.14)				1.40 (1.62)			
Model 3		1.54	0.06	.02		2.01†	0.16	.04		2.10†	0.06	.04
Age	-29.75 (21.55)				-1.15† (0.62)				-1.94* (0.88)			
SES	0.70 (2.74)				-.04 (0.08)				0.17 (0.11)			
BMI	2.87* (1.40)				.06 (0.04)				0.08 (0.06)			
Chronic Stress	91.13 (82.27)				4.38† (2.37)				4.74 (3.37)			
Negative Affect	22.52 (40.07)				0.54 (1.16)				1.34 (1.64)			
Childhood Adversity	4.28 (17.40)				0.20 (0.50)				0.18 (0.71)			
Model 4		2.10*	5.21*	.05		2.84**	7.34**	.08		2.17*	2.51	.05
Age	-23.34 (21.43)				-0.93 (0.61)				-1.74* (0.89)			
SES	0.95 (2.70)				-0.03 (0.08)				0.17 (0.11)			
BMI	2.54† (1.39)				0.05 (0.04)				0.07 (0.06)			
Chronic Stress	88.92 (81.15)				4.31† (2.32)				4.68 (3.35)			
Negative Affect	0.79 (40.65)				-0.20 (1.16)				0.71 (1.68)			
Childhood Adversity	-8.16 (18.01)				-0.23 (0.52)				-0.18 (0.74)			
NA x Ch Adversity	37.33* (16.36)				1.27** (0.47)				1.07 (0.68)			

†p < .10, \*p < .05, \*\*p < .01

BMI = body mass index; SES = socioeconomic status; NA = Negative Affect; Ch Adversity = Childhood Adversity

Figure 1A.

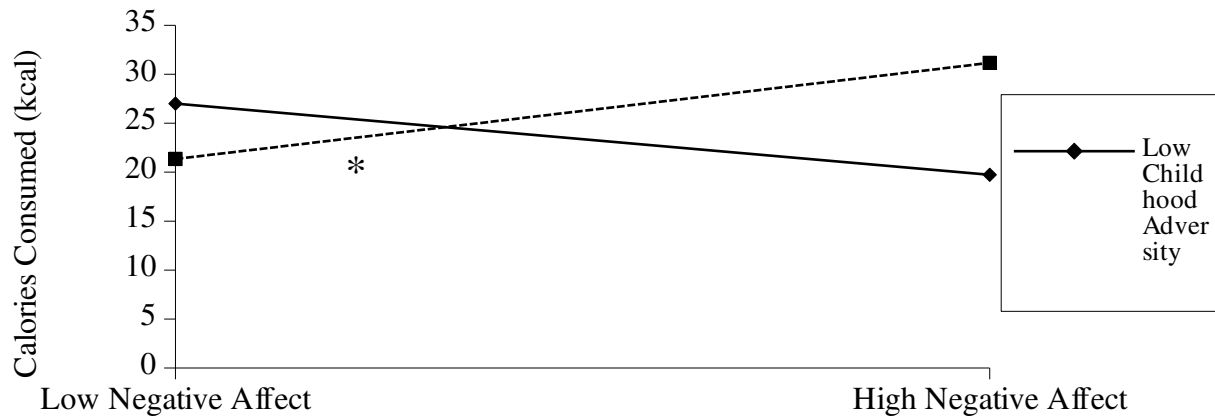


Figure 1B.

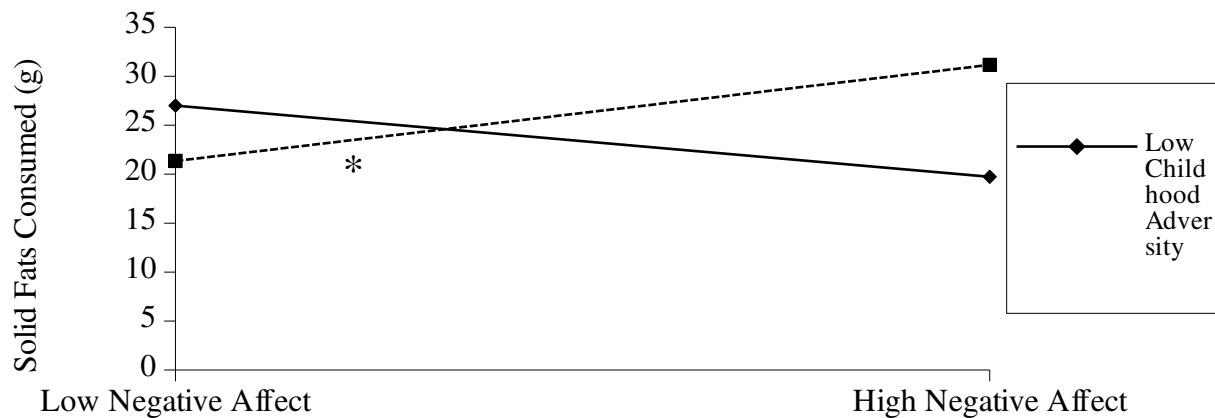
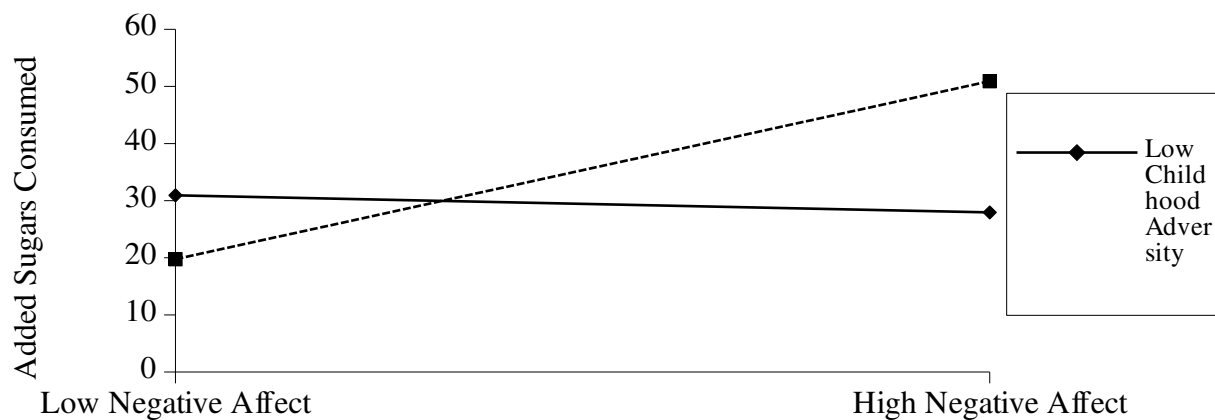


Figure 1C.



*Figure 1.* Interaction of negative affect and childhood adversity on calories (Figure 1A), solid fats (Figure 1B), and added sugars (Figure 1C) consumed following a stressor. Asterisks indicate significant slopes. Interaction is displayed across all observed values of negative affect; high childhood adversity is plotted at the region of significance when interactions were significant (Figure 1A and 1B) and at the highest observed value when interactions were non-significant (Figure 1C); low childhood adversity is plotted at the lowest levels observed (no adversity).