Exploring the Relationship between Childhood Interpersonal Violence,

Cortisol Stress Response, and Stress Eating

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Histories of childhood interpersonal violence have been linked to overweight and obesity in adulthood. Despite the fact that victimization experiences are likely to co-occur within individuals, few studies have examined poly-victimization in the context of overweight or obesity. The current study aimed to: (1) determine whether poly-victimization was associated with body mass index and waist circumference in a sample of undergraduate women, and (2) explore plausible physiological and behavioral mechanisms accounting for the relationship, specifically cortisol and ad libitum eating in response to laboratory stress. Undergraduate women from the University of Pittsburgh responded to an online survey measuring history of six different childhood victimization experiences: physical abuse, sexual abuse, peer violence, intimate partner violence, community violence, and witnessing violence. Ninety-two healthy women were recruited into 2 study groups (n = 48 with no history of childhood victimization [controls]; and n = 44 endorsing a history of 2 or more different types of childhood victimization [poly-victims]). Study protocol occurred 3 hours after lunch and included standardized laboratory stressors (public speech and math task) followed by a 30 minute relaxation period that included ad libitum access to snack foods with varying caloric density. Salivary cortisol was assessed at three points across the laboratory session. Women reporting poly-victimization had higher body mass index after adjusting for childhood socioeconomic status, family history of obesity, and alcohol use. The relationship was weaker with the outcome of waist circumference. Poly-victims

did not eat more following stress, although poly-victims who perceived their performance as worse on the stress task consumed more calories, controlling for childhood socioeconomic status and hunger at study entry. Unexpectedly, poly-victims showed blunted cortisol response to the stress task compared to controls, after adjusting for childhood socioeconomic status and birth control use. Bootstrapping tests of mediation showed that neither cortisol response to stress nor stress eating were significant mediators in the relationship between childhood interpersonal violence and body mass index. In conclusion, results from the present study suggest that poly-victimization is a risk factor for body mass index, and cortisol and eating responses to stress do not appear to mediate the relationship.

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1.0   INTRODUCTION

Interpersonal violence is defined as behavior that threatens, attempts, or causes physical harm, and children are particularly vulnerable. Childhood interpersonal violence (CIV) has been shown to be associated with physical health outcomes, one of which is obesity. For the sake of the present literature review, obesity will be used as an umbrella term to refer to individuals in the overweight or obese categories. A review of the literature showed consistent positive relationships between physical abuse, sexual abuse, peer bullying, and obesity, with mixed findings for community violence (Midei & Matthews, 2011). Prospective and longitudinal studies starting in childhood provided the strongest empirical evidence. For instance, a study following sexually abused girls showed that they had a steeper trajectory of weight gain through adolescence and young adulthood compared to non-abused controls (Noll, Zeller, Trickett, & Putnam, 2007). Another study of adolescents found that peer bullying doubled the odds of becoming obese over four years (Sweeting, Wright, & Minnis, 2005). Physical abuse and sexual abuse were also linked to central adiposity in two recent studies (Midei, Matthews, & Bromberger, 2010; Thomas, Hypponen, & Power, 2008), suggesting that CIV may predict abdominal deposition of body fat. The qualitative review suggested that victims of various types of CIV have similar obesity outcomes.

Research in the field of CIV suggests that children who are exposed to one form of violence are three to six times more likely to be exposed to a second form of violence (Dong,

Anda, et al., 2004; Finkelhor, Ormrod, & Turner, 2007b), which is termed poly-victimization. An example would be that a child physically abused at home is more likely to be bullied at school. The Adverse Childhood Experiences study surveyed a large sample of HMO patients and found that exposures to abuse and household dysfunction were positively correlated; for a person reporting a single exposure, the probability of reporting a second exposure was on average 80%, and the probability of reporting two or greater additional adverse experiences was on average 56% (Felitti et al., 1998). The co-occurrence of different types of violence may partly explain the similar obesity outcomes across different types of violence. Only one study examined the relationship between poly-victimization and obesity. The authors found a dose-response relationship between the number of exposures to adverse experiences and severe obesity (Felitti et al., 1998).

Of primary importance is the consideration of *how* CIV may lead to later risk for obesity. Various literatures suggested that CIV may be linked to obesity through the following pathways: Hypothalamic-pituitary-adrenal (HPA) axis reactivity to stress and eating in response to stress (see Figure 1).
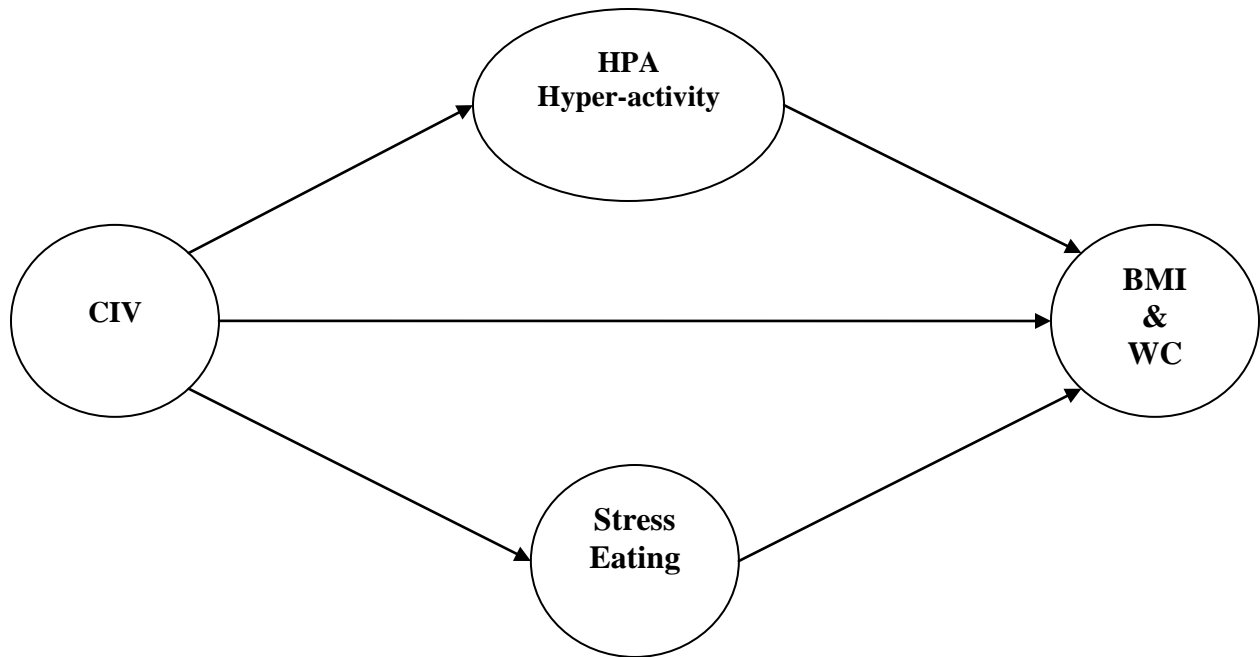
Figure 1. Individuals with high exposure to childhood interpersonal violence (CIV; poly-victimization) will have higher body mass index (BMI) and waist circumference (WC), compared to controls with no CIV exposure, and the relationship will be mediated by hypothathalamic-pituitary-adrenal axis hyperactivity and stress eating.

Theoretical and empirical support indicated that these mechanisms are plausible, although no studies tested for mediation. The primary purpose of the present study is to identify mechanisms that account for the relationship between CIV and obesity. Hypotheses examined associations between poly-victimization and obesity/central adiposity, as well as the mediating influence of HPA axis dysregulation and stress eating. Significant associations will reflect the importance of early-life psychosocial stressors on adult health, in addition to clarifying the physiological and behavioral pathways between CIV and obesity. Finally, the study findings may suggest a novel opportunity for possible intervention for victims of CIV.

2.0   LITERATURE REVIEW

2.1   CHILDHOOD INTERPERSONAL VIOLENCE AND BODY MASS INDEX / WAIST CIRCUMFERENCE

A recent systematic review reported a positive association between various types of CIV and obesity and central adiposity (Midei & Matthews, 2011). CIV from caregivers, such as physical abuse and sexual abuse, showed the strongest relationship with obesity. For example, a study following sexually abused girls showed that they had a steeper trajectory of weight gain through adolescence and young adulthood compared to non-abused controls (Noll et al., 2007). Another study tracked boys and girls with court-substantiated abuse and found that participants with a history of physical abuse had higher body mass index (BMI) in mid-life compared to non-abused controls (Bentley & Widom, 2009). Individuals who experienced interpersonal violence from caregivers were approximately 70% more likely to be obese in adulthood than non-abused individuals, based on an average of available odds ratios from the reviewed studies. A summary of the data suggest that among the studies deemed high-quality, six out of seven studies on physical abuse and five out of six studies on sexual abuse reported a positive association with BMI. Only one study examined witnessing violence and found a positive association with obesity. Two studies (out of two) explored the outcome of central adiposity and reported positive relationships with physical abuse, sexual abuse, and witnessing violence. Overall, CIV from

caregivers is a significant predictor of obesity in adulthood, with preliminary evidence for central adiposity.

CIV from peers has also been shown to be an important risk factor for obesity. There were two longitudinal studies that received high-quality ratings. A study of teenagers found that peer bullying doubled the odds of becoming obese over four years (Sweeting et al., 2005). The second study reported that obese females who were bullied by peers at age 12-13 had significant increases in BMI by age 16-17 (Adams & Bukowski, 2008). Five out of five cross-sectional, high-quality studies suggested that peer bullying was positively associated with obesity. The few studies that reported odds ratios suggested that individuals who experienced bullying were at least twice as likely to be obese compared to non-bullied individuals.

The evidence was inconsistent for CIV from the community; three out of the six cross-sectional, high-quality studies of neighborhood crime and lack of safety reported a positive association with obesity in childhood and adolescence. Two out of the three studies that found nonsignificant associations included boys and girls between the ages of three and five. This period of the life course may be too early to detect the onset of an obesity trajectory, as well as the cognitive, emotional and physiological burden of CIV. Furthermore, measures of CIV from the community did not identify whether individuals were targets of the neighborhood crime, which is a more valid assessment of CIV. The methodological concerns may be noteworthy limitations that preempt the exclusion of CIV from the community as a risk factor for obesity.

Some factors may be important moderators in the relationship between CIV and obesity. More severe forms of violence were associated with greater BMI, compared to less severe forms (Greenfield & Marks, 2009; Mamun et al., 2007). CIV was more consistently associated with obesity earlier in the life span (i.e., adolescence and young adulthood) than in later adulthood

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(Hamer, O'Donnell, Lahiri, & Steptoe, 2010; Mamun et al., 2007; Noll et al., 2007). This may be because 66% of Americans are overweight or obese in adulthood (Ogden et al., 2006) and CIV may account for less of the variance in obesity as adults move through the life course and gain more weight. In addition, significant relationships were more consistent in samples limited to females (e.g., Alvarez, Pavao, Baumrind, & Kimerling, 2007; Evenson, Scott, Cohen, & Voorhees, 2007; Jia, Li, Lesserman, Hu, & Drossman, 2004; Midei et al., 2010; Rohde et al., 2008) compared to samples with both males and females (Allison, Grilo, Masheb, & Stunkard, 2007; Chartier, Walker, & Naimark, 2009; Grilo et al., 2005; Romero et al., 2001). Moreover, some studies found significant relationships in only females but not males (Adams & Bukowski, 2008; Mamun et al., 2007).

The current study extends the literature by examining the relationship between poly-victimization and overweight while addressing some limitations in the previous literature. First, participants were recruited to compose two groups, one group reporting multiple exposures to CIV (poly-victims) and one group reporting no exposure to CIV (controls). Comparing a high-exposure group to a no-exposure group addresses the literature that suggests that poly-victimization is more often the rule rather than the exception (Finkelhor, 2008), and that physical abuse, sexual abuse, and peer bullying were all positively associated with obesity (Midei & Matthews, 2011). Second, the present study inquired about frequently studied CIV experiences (i.e., physical abuse, sexual abuse, and peer bullying), as well as under-examined CIV experiences (i.e., witnessing violence, intimate partner violence, and direct violence from the community). Third, participants were young women (mean age = 18.6) attending the University of Pittsburgh. This age period captures the obesity trajectory at a time when CIV appears to be more consistently related to obesity outcomes, as well as potentially minimizing the effect of

time on retrospective reporting of CIV. Moreover, women showed more consistent associations between CIV and obesity compared to men. Fourth, obesity was measured in the lab to address the limitation in previous studies of using self-reported weight and height, which has been shown to under-estimate measured BMI (Gillum & Sempos, 2005). Another limitation of previous research was that about a third of the studies reviewed by Midei and Matthews (2011) did not adjust for potential confounding variables when examining the association between CIV and obesity. Therefore, the current study controlled for relevant variables, such as childhood socioeconomic status and family history of obesity. Finally, few studies examined the relationship between CIV and central adiposity. Central adiposity is measured by waist circumference (WC) or waist-to-hip ratio (WHR) and predicts morbidity and mortality, independent of BMI (Prineas, Folsom, & Kaye, 1993). Thus, the current study tested associations between CIV and central adiposity. In summary, the present study's design not only addresses the methodological limitations in previous research, but also builds on the literature by exploring the importance of poly-victimization as a predictor of overweight/central adiposity.

2.2   MECHANISMS

Obesity is a heterogeneous condition with many contributing factors that lead to energy intake exceeding energy expenditure (Marcus & Wildes, 2009). Both energy intake and energy expenditure are important parts in the development of obesity; however, evidence targets excess energy intake as the primary force behind the obesity epidemic (Jeffery & Harnack, 2007). CIV may increase the risk for obesity by promoting increases in energy intake. In other words, individuals with CIV are likely to habitually eat an excess of calories, cumulatively leading to

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increases in BMI and WC. The intake of excess calories may occur in response to stressful situations, also known as stress eating. For the present study, stress eating is the first mechanism to be explored in the pathway between CIV and overweight/central adiposity.

The second mechanism to be explored is HPA axis dysregulation. Histories of childhood victimization have been linked with HPA axis activity, and HPA axis activity has been suggested as a contributor to obesity and central adiposity. It is also possible that stress eating may be a way of regulating HPA axis dysregulation. The following sections review the literature addressing stress eating and HPA axis dysregulation as possible mechanisms.

2.2.1 Stress Eating

In humans, previous literature has shown that stress affects eating in the following ways: approximately 40% of individuals eat less food and lose weight during stress, 20% have no changes in weight, and 40% of individuals eat more food and gain weight during stress (Block, He, Zaslavsky, Ding, & Ayanian, 2009; Oliver & Wardle, 1999). Given that people in developed countries are surrounded by an abundance of highly palatable and calorie dense food, it is likely that the increased food exposure contributes to increased eating. Indeed, animal models showed that rats exposed to stress and in an environment of highly-palatable food exhibit hyperphagia (e.g., Pijlman, Wolterink, & Van Ree, 2003).

Some individual differences predict who eats in response to stress. One individual difference that relates to stress eating is dietary restraint, or effortful control of eating. Restraint is conceptualized by some as a measure of chronic dieting (Greeno & Wing, 1994). Restrained eaters generally show increases in food consumption in response to laboratory stressors. For example, Mitchell & Epstein (1996) examined 16 women high on restraint and 16 women low on

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restraint and used the Stroop task to stress half of the women in each group. The authors found that restrained eaters in the stress condition ate more yogurt than the control group, and the non-restrained eaters in the stress condition ate less yogurt than the control group. Zellner and colleagues (2006) examined undergraduate woman in a stress condition and found that they consumed more high-calorie sweet food (M&Ms), and less low-fat healthy food (grapes), compared to women who were not stressed. Additionally, women who self-identified as stress eaters were largely restrained eaters and endorsed eating foods that they typically avoided (Zellner et al., 2006). In a real-life setting, 90 staff members of a department store were followed for six months to track work stress and diet (Wardle, Steptoe, Oliver, & Lipsey, 2000). High workload periods were associated with higher calorie, saturated fat, and sugar intake, measured by 24-hour dietary recall, but only for individuals reporting high restrained eating.

Some other factors appear to contribute to stress eating. Women may be more prone than men to stress eat (Grunberg & Straub, 1992; Zellner et al., 2006). Women also appear to be more susceptible to disinhibition, which is the inability to maintain control of self-imposed rules concerning food intake. Disinhibition has been linked with increased calorie consumption in response to stress (Oliver, Wardle, & Gibson, 2000; Weinstein, Shide, & Rolls, 1997).

The type of stressor may also be important as a predictor of stress eating. Stress eating laboratory paradigms have included cognitive tasks (Stroop), social-evaluative stressors (Trier Social Stress Test--TSST), and mood manipulations (unpleasant videos). Adam and Epel (2007) proposed that threatening stressors, but not challenging stressors, predict subsequent eating behavior. Threatening stressors often cause distress (feeling defeated or fearful) and are demanding situations in which one does not have the resources to cope. In contrast, a challenging stressor is a demanding but controllable situation in which one has adequate resources to cope.

Preliminary evidence suggests that a threatening task (TSST) predicted greater food consumption compared to challenging task (TSST with positive audience feedback), and perceptions of threat mediated this relationships (Cosley, McCoy, Ehle, Saslow, & Epel, 2007).

Finally, previous literature shows that stress alters the type of food that people eat with increasing preference for high-fat, high-sweet foods, such as chocolate candy and chocolate granola bars (Epel, Lapidus, McEwen, & Brownell, 2001; Grunberg & Straub, 1992; Zellner et al., 2006). Studies in the real world show similar patterns. When participants report periods of stress, there is an increase in consumption of "snack-type" foods (sweets, chocolates, cakes, cookies, savory snacks) and a decrease of consumption of "meal-type" foods (meat, fish, fruits, vegetables) (Kandiah, Yake, Jones, & Meyer, 2006; Oliver & Wardle, 1999). One study explored interactions associated with stress eating and food preference. Rutters and colleagues (2008) tested "eating in the absence of hunger" by providing snack foods available ad libitum after consumption of a standard lunch. The authors found that men and women who were stressed in the lab consumed more sweet snacks and total calories than the non-stressed group, even in the absence of hunger. Greater stress responses, which were measured by increases in anxiety scores, explained part of the relationship between stress and eating in the absence of hunger. Furthermore, this effect was stronger in participants with higher disinhibition scores. In summary, several factors predict an individual's likelihood of stress eating in response to threatening stressors in the laboratory and the real world: female gender, self-reported dietary restraint or disinhibition, and an environment of snack-type foods.

2.2.1.1 CIV and stress eating

CIV is undoubtedly perceived as a stressor each time that the violence occurs, and rumination about previous CIV experiences may also be experienced as stressful. Children and adolescents

may use food to manage their emotions. A habitual pattern of eating in response to CIV or rumination of CIV may generalize to other stressful experiences.

Previous research has suggested that when children and adolescents face stressors that are chronic, uncontrollable, and distressing, they often rely on emotion-focused coping rather than problem-solving coping (Ebata & Moos, 1994; Zeidner & Saklofske, 1996). Emotion-focused coping aims to reduce negative emotions associated with the stressor, rather than changing the stressor itself. Adolescents with emotion-focused coping have been shown to have unhealthy eating behaviors compared to children with problem-focused coping (Martyn-Nemeth, Penckofer, Gulanick, Velsor-Friedrich, & Bryant, 2009). Emotional eating and stress eating are often viewed as similar constructs. Studies in adults reported that participants who engaged in stress-eating did so for an increase in feelings of relaxation and comfort (Zellner et al., 2006) or to distract from feelings of distress or mask the source of the stress (Polivy & Herman, 1999).

Only one study has explored the relationship between CIV and stress eating. Greenfield and Marks (2009) measured childhood physical abuse (co-occurring with psychological abuse) and how men and women normally experienced a stressful event. Stress eating was assessed with the following items: "In response to stress, I eat more food than I usually do" and "In response to stress, I eat more of my favorite foods to make myself feel better." Results showed that frequent abusive experiences were significantly and positively associated with the use of food in response to stress. Stress eating is a plausible eating behavior that may in part explain the relationship between CIV and obesity.

2.2.1.2 Stress eating and body mass index/waist circumference

Only a few cross-sectional studies reported on the association between stress eating and obesity. BMI was highest among those who self-identified as stress eaters, and this relationship was

stronger in women (Laitinen, Ek, & Sovio, 2002). A study of adults showed that the use of food in response to stress was associated with a 2.14 odds ratio of being obese (Greenfield & Marks, 2009). Epel and colleagues (Epel et al., 2004) followed medical students over two exam periods through the course of a year. They reported that individuals who self-identified as eating more when stressed had significant increases on BMI compared to individuals who self-identified as eating less when stressed. Finally, Sims et al. (2008) reported that the relationship between perceived stress and emotional eating was stronger in overweight and obese black women compared to normal-weight black women. There may be a cyclical relationship between stress eating and obesity. Individuals who are stressed and eat in response to stress are likely to gain weight, and heavier individuals are more likely to stress eat, thus continuing the cycle.

The current study extends the literature in the following ways. First, an objective measurement of eating occurred in the laboratory, thus having high internal validity for eating that occurred immediately following stress. Second, the study included ad libitum access to snack-type foods that are high in fat and sugar in order to reflect palatable food choices that are commonly eaten in response to stress. Third, dietary restraint was measured and explored for moderating effects.

2.2.2 HPA Axis Dysregulation

A biological pathway that may influence the relationship between CIV and obesity is dysregulation of the HPA axis. The HPA axis is one of the fundamental neuroendocrine regulatory systems, which responds to perceived psychological and biological stressors through a cascade of physiological changes. The cascade begins with the hypothalamic release of corticotropin-releasing hormone (CRH), and then CRH stimulates the anterior pituitary to

produce and secrete adrenocorticotropic hormone (ACTH). Subsequently, ACTH stimulates the adrenal cortex, elevating secretion of glucocorticoids, the most important being cortisol. Cortisol has extensive effects on biological functions, such as making glucose available in the blood stream, releasing fatty acids for energy use, suppressing the immune system, and permitting catecholamine activity (Silverthorn, 2007). Cortisol has a strong diurnal rhythm, peaking after waking then decreasing over the course of a day. During brief, time-limited stressors, cortisol maintains homeostasis through negative-feedback loops to the hypothalamus and anterior pituitary, which occurs within minutes of cortisol release. However, chronic stress results in hyper-activity of the HPA axis, leading to hyper-secretion of cortisol and an eventual desensitization of the negative-feedback loop (McEwen, 2000).

The methodology used to measure HPA axis activity varies widely across studies and presents a challenge for clear conceptualization. HPA axis activity can be measured by assessing reactivity to laboratory psychological stressors or hormonal challenges, the morning awakening response, basal levels at various times across the day, total daily output, and diurnal rhythms. ACTH and cortisol are used as indices of HPA axis activity, although the magnitude of their correlation is unclear. Cortisol can be measured in urine, blood, saliva, hair, or cerebrospinal fluid. Each method of measuring HPA axis activity provides unique information about the functioning of the system in specific populations.

A number of other factors make it difficult to identify a clear relationship between CIV and HPA activity. Studies in children generally examine basal cortisol in contrast to studies in adults, which generally measure HPA reactivity. Additionally, normal HPA activity changes over the life course, such that basal cortisol levels increase from childhood through adolescence, and the timing of this increase has been linked to pubertal status (Netherton, Goodyer, Tamplin,

& Hertbert, 2004). Another challenge of interpreting HPA axis activity is that CIV victims often report comorbid depression or post-traumatic stress disorder (PTSD), and psychiatric disorders are independent predictors of HPA axis dysregulation. Finally, research has suggested that HPA axis hyper-activity and hypo-activity may not be mutually exclusive theories of impaired systems, but rather that hyper-activity may precede hypo-activity (Miller, Chen, & Zhou, 2007). In light of the numerous factors that may contribute to HPA activity measurement and interpretation, the following sections summarize trends in the literature between CIV and HPA activity, as well as obesity/central adiposity and HPA activity.

2.2.2.1 CIV and HPA axis dysregulation

CIV may be connected with HPA axis dysregulation because it is perceived as a chronic, threatening, and uncontrollable stressor. A meta-analysis reviewed studies measuring chronic stress and HPA activity (Miller et al., 2007), and a history of sexual abuse was coded with the features of being a physical threat, traumatic, and uncontrollable. Across all studies included in the meta-analysis, these three characteristics of stress generally showed HPA axis hyper-activity, measured by a flattened diurnal rhythm with lower morning values and higher afternoon/evening values. It should be noted that most of the sexual abuse studies included in the meta-analysis were of children, often with comorbid PTSD or depression. A similar pattern was recently reported in adults with chronic pain; a history of childhood maltreatment was positive associated with basal cortisol levels throughout the day (Nicolson, Davis, Kruszewski, & Zautra, 2010).

CIV may sensitize individuals to hyper-activity even in response to mild stressors. Several studies test HPA response to laboratory stressors such as the Trier Social Stress Test (TSST). Heim and colleagues (2000) found that women with physical or sexual abuse and major depressive disorder showed HPA axis hyper-activity, measured by ACTH and cortisol secretion,

compared to depressed women and controls. Another study reported that childhood abuse with current social anxiety disorder was linked to cortisol hyper-activity, compared to individuals with just social anxiety or controls (Elzinga, Spinhoven, Berretty, de Jong, & Roelofs, 2010). In both studies by Heim et al. and Elzinga et al., results showed that baseline values of cortisol were similar across groups, suggesting that basal levels of cortisol and ACTH were not driving group differences.

Several studies showed null or negative relationships between CIV and HPA axis activity in response to the TSST. For example, in a sample of healthy men and women, childhood traumatic experiences were associated with HPA hypo-activity (Elzinga et al., 2008). This effect was significant only for men, while there was a null relationship between adverse events and cortisol response for women. Carpenter et al. (2007) reported that adults with histories of abuse had significantly lower cortisol response to the TSST compared to non-abused participants, with the exception that sexual abuse was associated with greater peak cortisol response. A study of female adolescents (ages 12-16) found that abused and neglected participants showed cortisol hypo-activity compared to controls (MacMillan et al., 2009). Finally, one study measured exposure to violence in high school students and found that the number of exposures (i.e., poly-victimization) was not associated with cortisol response to verbal puzzles and verbal debate (Murali & Chen, 2005).

Psychiatric diagnosis may be an important moderator of HPA axis dysregulation. Men and women who reported a history of childhood abuse and current depression showed exaggerated HPA axis responses to stressors (e.g., Heim et al., 2000). Alternatively, childhood abuse with current PTSD was associated with suppression of HPA axis responses to stressors (Oquendo et al., 2003).

In summary, the literature suggests that CIV is associated with higher basal levels of cortisol. CIV was associated with HPA hyper-reactivity to laboratory stressors in several strong empirical studies, but the literature is inconsistent. Comorbid mental health status may moderate the relationship between CIV and HPA axis reactivity, and females may be particularly vulnerable to hyper-reactivity. Finally, there is a dearth of literature examining the relationship between poly-victimization and HPA axis activity.

2.2.2.2 HPA axis dysregulation and body mass index/waist circumference

HPA axis activity has been studied in the context of obesity and central adiposity. Cushing's syndrome provided the first evidence of a relationship; individuals with this metabolic disease have marked abdominal obesity and hypercortisolemia, and an andrenalectomy reverses obesity. Research examining the relationship with BMI has been inconsistent, in part because the measures of HPA activity varied widely (e.g., excretion, metabolic clearance, morning awakening response, basal levels). Higher BMI was associated with greater cortisol excretion and flatter diurnal rhythms in adults (Andrew, Phillips, & Walker, 1998; Daniel et al., 2006), and greater cortisol response to the TSST in children (Dockray, Susman, & Dorn, 2009). One study showed a nonsignificant relationship between BMI and cortisol (morning awakening response) in women (Therrien et al., 2007). Leading researchers in the field proposed that HPA axis dysregulation was stronger and more consistent in relation to abdominal adiposity (Björntorp & Rosmond, 2000).

The HPA axis has been targeted as the driver behind central adiposity, specifically visceral fat (Marniemi et al., 2002). Cortisol promotes differentiation of adipocyte precursors into adipocytes and stimulates lipogenesis in the presence of insulin (Gregoire, Genart, Hauser, & Remacle, 1991). Visceral fat is particularly sensitive to circulating cortisol because visceral

adipose tissue has greater blood flow and up to four times the number of glucocorticoid receptors compared to peripheral fat (Pedersen, Jonler, & Richelsen, 1994). Increases in HPA axis activity have been shown to increase deposition of body fat in cynomologus monkeys (Shively, Laber-Laird, & Anton, 1997), and adipose tissue can also release inflammatory markers to stimulate the HPA axis (Mastorakos, Chrousos, & Weber, 1993).

Empirical data have generally shown a positive relationship between cortisol secretion (postprandial or morning awakening) and central adiposity in men (Rosmond, Dallman, & Bjorntorp, 1998; Wallerius, Rosmond, Ljung, Holm, & Björntorp, 2003) and between cortisol (response to ACTH stimulation and urinary free cortisol) and central adiposity in women (Duclos et al., 2001; Marin et al., 1992; Pasquali et al., 1993). Additionally, women with high WHRs showed cortisol hyper-activity in response to the TSST (Epel et al., 2000). One study did not find an association between central adiposity and cortisol reactivity to a Stroop and speech task in women (Brydon et al., 2008). In sum, the empirical evidence suggests that central adiposity is related to HPA axis hyper-activity.

2.2.2.3 Interactions between HPA axis hyper-activity and stress eating

HPA axis reactivity has been related to increased food consumption, particularly when in an environment with highly palatable food. The animal literature has been reviewed previously (Adam & Epel, 2007); therefore, the following section highlights the relevant human studies. Tataranni and colleagues (1996) performed a double-blind, placebo-controlled study in twenty males to investigate how glucocorticoid administration affected ad libitum food intake. Men who were injected with glucocorticoids ate approximately 60% more calories than individuals injected with a placebo, with increases primarily in protein and carbohydrate intake. Another study stressed participants in a laboratory using the TSST; individuals showing high cortisol

response to the stressor ate more calorie-dense food post-stressor compared to individuals with low cortisol response (Epel et al., 2001). Another study had participants complete the TSST to classify them into high and low cortisol responders, then tracked the participants for 14 days (Newman, O'Connor, & Conner, 2007). The authors found that there was a positive association between daily hassles and snack intake, but only for individuals identified as high cortisol reactors.

Chronic glucocorticoid secretion promotes stress eating through a number of physiological pathways. As glucocorticoids increase, insulin secretion also increases, and the combination appears to drive the intake of fat and sugar calories, as opposed to calories from low-fat, bland food (Dallman, Pecoraro, & la Fleur, 2005). Glucocorticoids and insulin additionally stimulate the secretion of leptin from adipose cells, which signals satiety. However, chronically elevated GCs cause diminished leptin sensitivity, resulting in overeating despite high levels of leptin (Zakrzewska, Cusin, Sainsbury, Rohner-Jeanrenaud, & Jeanrenaud, 1997). Alternatively, stressors that elevate GCs may stimulate neuropeptide Y, which has the combined effect of lowering anxiety but increasing appetite (see review by Adam & Epel, 2007).

Stress eating may be a homeostatic response to HPA axis hyper-activity, such that individuals may be driven to consume calories to stabilize a dysregulated HPA axis. There is an absence of human research that has explored this theory, but some animal studies offer support for homeostatic eating. For example, stressed rats showed increases in HPA axis activity, and eating a high-fat diet subsequently dampened HPA axis activity, but only in the condition where rats could choose a high-fat and high-sugar diet (Foster et al., 2009; la Fleur, Houshyar, Roy, & Dallman, 2005). The authors proposed that the increased intake of a calorie-dense diet by the

stressed rats compares to the increased consumption of snack-type foods in humans when they are stressed, and that food choice/intake provides regulation of HPA axis activity.

The current study extended the literature in the following ways. This was the first study to test HPA axis activity, in the form of cortisol response to stress, as a mediator between CIV and overweight/central adiposity. Cortisol response to stress may be a particularly relevant marker of HPA axis activity because it has been associated with calorie consumption in previous literature. Moreover, this was the first study to test cortisol response to stress as a mediator between CIV and calorie consumption after stress. Cortisol response was measured using protocols recommended for maximum effects (Dickerson & Kemeny, 2004). For example, the stressor incorporated social-evaluative threat and uncontrollability, was short in duration, and was conducted in front of an audience. Specific study design improves measurement of cortisol response, such as laboratory sessions completed in the afternoon and cortisol samples 21-40 minutes from stressor onset. The present study aimed to clarify the role of HPA axis dysregulation in women with histories of CIV and in stress eating.

3.0   STATEMENT OF PURPOSE

To sum the previous literature, a considerable body of research showed a positive correlation between various types of CIV and obesity, with the relationship being more consistent in younger populations and for women. Few studies explored the mechanisms accounting for this relationship. Research has shown that stress eating may be a behavioral mechanism between CIV and obesity/central adiposity. Additionally, HPA axis dysregulation has been found in children and adults reporting CIV, generally reflecting a pattern of hyper-activity. Research also indicated that HPA axis dysregulation is a primary mechanism driving central adiposity, and waist circumference is highly correlated with BMI. Thus, HPA axis dysregulation may be a biological mechanism connecting CIV to obesity/central adiposity. Finally, stress eating may be a way of regulating HPA hyper-activity.

Several questions remain unanswered. First, is CIV (poly-victimization) associated with stress eating, and does stress eating mediate the relationship between CIV and BMI/WC? Second, is CIV associated with cortisol response to stress, and does cortisol response mediate the relationship between CIV and BMI/WC? Finally, does cortisol response mediate the relationship between CIV and stress eating?

The present study aimed to answer the stated questions using a quasi-experimental design. Participants were young women attending the University of Pittsburgh who responded to an online survey assessing CIV, demographics, emotions, and health behaviors. CIV experiences

included six different types of violence: physical abuse, sexual abuse, peer victimization, intimate partner victimization, witnessing violence, and community violence. Based on responses to the survey, participants for a laboratory study were recruited to fill two groups, one group with two to six exposures to CIV (poly-victims) and one group with zero exposures to CIV (controls). Participants in the lab study engaged in a social-evaluative stressor after which they had ad libitum access to snack foods to measure stress eating. Salivary cortisol was assessed at three points during the laboratory session in order to capture the cortisol response to stress.

There are several reasons why understanding the mechanisms between CIV and BMI/WC is important. Analyzing and clarifying the relationship between CIV and BMI increases our understanding of the pathogenesis of obesity. Additionally, knowledge regarding mechanisms may provide novel opportunities for obesity intervention. For example, school-based interventions that focus on increasing healthy coping strategies in response to CIV have shown positive mental health effects (Stein, Jaycox, Kataoka, Wong, et al., 2003). A similar intervention may also prevent children from becoming overweight or obese.

4.0   STUDY HYPOTHESES

4.1   PRIMARY HYPOTHESES

Hypothesis 1. Women reporting poly-victimization will have higher BMI and WC compared to controls.

Hypothesis 2: Women reporting poly-victimization will consume more calories after a stressor compared to controls.

Hypothesis 3: Stress eating will mediate the relationship between CIV and BMI and WC. See Figure 2.

Hypothesis 4:   Women reporting poly-victimization will have greater cortisol response in response to a laboratory stressor compared to controls.

Hypothesis 5: Cortisol response will mediate the relationship between CIV and BMI and WC. See Figure 3.

Hypothesis 6: Cortisol response will mediate the relationship between CIV and stress eating. See Figure 4.
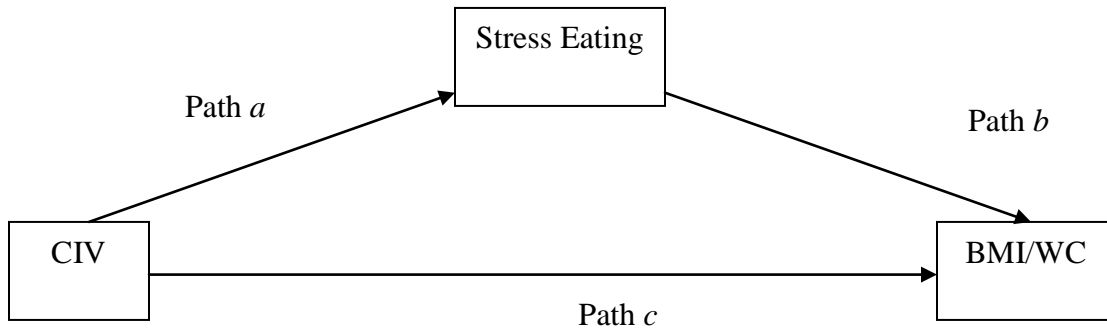
Figure 2. The hypothesized model suggested that stress eating would mediate the relationship between childhood interpersonal violence (CIV) and body mass index (BMI) / waist circumference (WC).
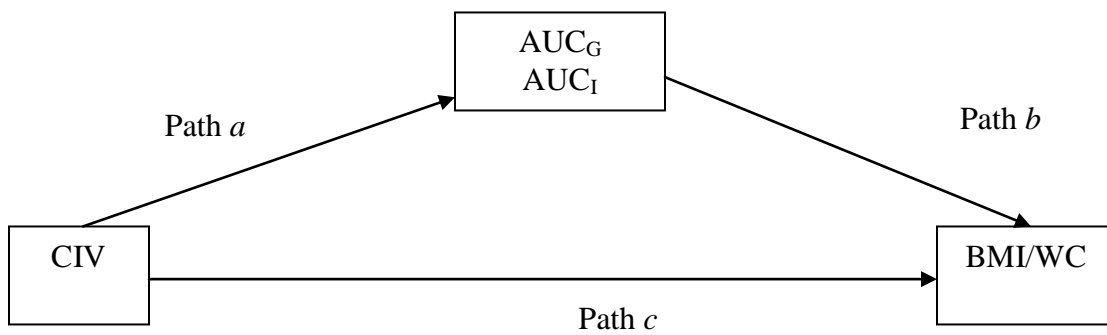
Figure 3. The hypothesized model suggested that cortisol reactivity would mediate the relationship between childhood interpersonal violence (CIV) and body mass index (BMI) / waist circumference (WC).
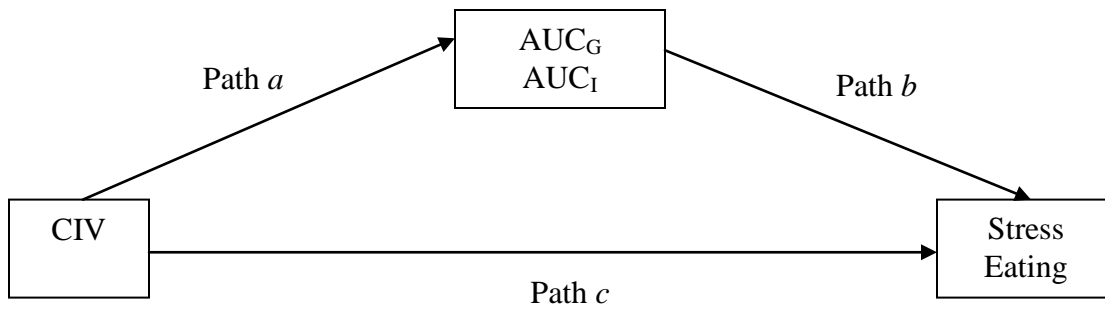
Figure 4. The hypothesized model suggested that cortisol reactivity would mediate the relationship between childhood interpersonal violence (CIV) and stress eating.

4.2   SECONDARY ANALYSES

Analyses will adjust for current violence exposure in order to test whether hypothesized relationships are independent of current violence. Additionally, due to the literature that suggests that restrained eating is a significant predictor of stress eating, restrained eating was tested as a moderator of the relationship between CIV and stress eating.

5.0   METHOD

5.1   PROCEDURE

Approval for the study protocol was obtained from the University of Pittsburgh Institutional Review Board. Participants were recruited from the psychology subject pool at the University of Pittsburgh from January 2011 to November 2011 and were invited to complete a survey on "Childhood Stress and Health." The recruitment survey was used to identify subjects who were eligible to participate in the laboratory component of this study. To complete the recruitment survey, subjects needed to be female and at least 18 years of age. Once the electronic survey was complete, all participants viewed a list of community resources, including Pitt Student Health, Pitt Counseling Center, and the Clinical Psychology Center. Participants received course credit as compensation for their involvement.

A total of 987 individuals opened the online survey, and of these, 876 participants provided complete data. See Figure 5 for a flow diagram that illustrates participation in the online survey. Of the 876 participants, 513 (58.6%) agreed to be contacted for a follow-up laboratory study.
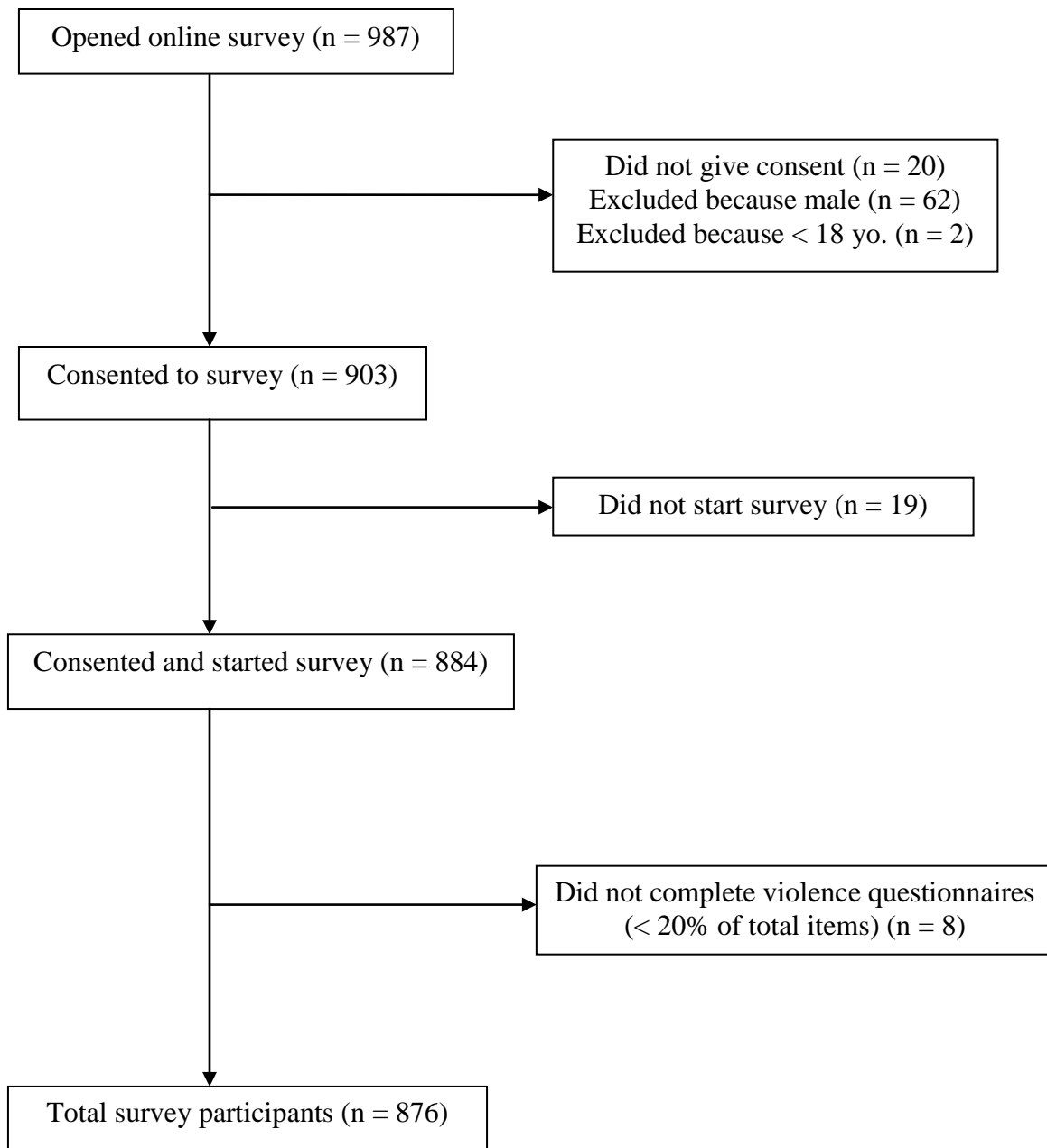
Figure 5. Participant flow diagram for the online survey.

Within the subset of 513 women, approximately 52% reported 0 exposures to violence and 21% reported 2 or more exposures to violence. Women were excluded from further participation if they: 1) used medications associated with weight gain/loss (e.g., Haldol, Lithium, Depakote, Glucocorticioids, Orlistat), 2) have been diagnosed or treated for post-traumatic stress disorder, 3) have Cushing's disease, high blood pressure, type II diabetes, or immune disorders, or 4) use recreational/illegal drugs more than two times per week. Once ineligible participants were excluded, names and contact information were copied to a word document without subsequent labeling of group membership. Thus, at the point of the recruitment phone calls for the laboratory session, all investigators were blinded to group membership. A total of 228 eligible participants were contacted and asked to be a part of a laboratory study, and 98 participants completed the laboratory study protocol.

Laboratory sessions began between 2:30 and 3:30 and lasted for 80 minutes. Participants were instructed to refrain from eating or drinking (excluding water) for three hours before the session. They were also asked to refrain from smoking, drinking alcohol, or exercising on the day of the laboratory session. The laboratory session followed a protocol (with minor modifications) suggested by the MacArthur Foundation for conducting the Trier Social Stress Test and measuring salivary cortisol response (Kirschbaum & Hellhammer, 2000; Kirschbaum, Pirke, & Hellhammer, 1993). Trained undergraduate research assistants contributed to various components of the protocol.

Upon arrival, participants provided written informed consent and began a 20-minute pre-task rest period. During this time they answered a questionnaire measuring health behaviors and current mood and relaxed with magazines. After the pre-task rest period, the first saliva sample was obtained.

At the beginning of the stressor, the experimenter informed the participant that she would have to deliver a speech (lasting five minutes) for a job application to an expert committee, which would be followed by a math subtraction task (lasting five minutes). The participant was then given five minutes to prepare for the stress task. The expert committee was trained to respond to the participant's performance in a non-accepting manner (e.g., not smiling, stony faces, and writing comments on a clipboard).

Following completion of the stressor, the participant completed questionnaires evaluating the task and labeling current mood. The participant then began a 30-minute post-stress recovery period during which she had ad libitum access to snack food while watching a video (Planet Earth). The participant was invited to eat with the statement, "These snacks are leftover from a meeting that just ended. Feel free to help yourself while you are watching the video." Approximately ten minutes within the recovery period (~30 minutes after initiation of the stressor), the second saliva sample was obtained. At the end of the recovery period (~50 minutes after initiation of the stressor), the participant provided the third and final saliva sample. See Figure 6 for a timeline of the laboratory protocol. At the conclusion of the video, participants were weighed, measured, and debriefed.
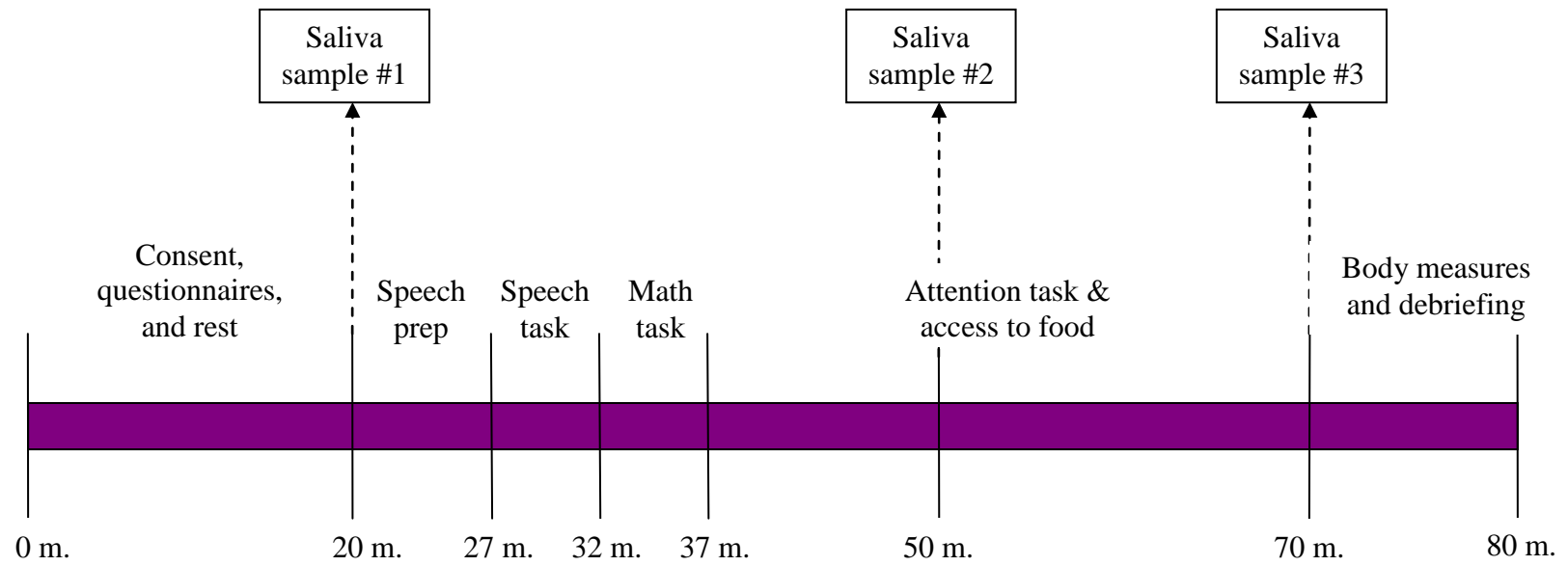
Figure 6. Timeline of laboratory session.

5.2   PARTICIPANTS

Ninety-eight participants completed the laboratory study; 49 were identified as poly-victims (2-5 types of violence exposures) and 49 were controls (0 types of violence exposures). Out of the 98 subjects, 6 had to be excluded because of errors in subject recruitment and statistical software. Of the remaining 92 subjects, 44 were identified as poly-victims and 48 as controls. Five more subjects were excluded from cortisol analyses; three participants were taking steroid medication for immediate infections, one participant had un-measurable cortisol data based on assay reports, and one participant reported being moderately sick on the day of the lab session. Thus, cortisol analyses included a total of 87 participants. See Figure 7 for a flow diagram of participant sample size.
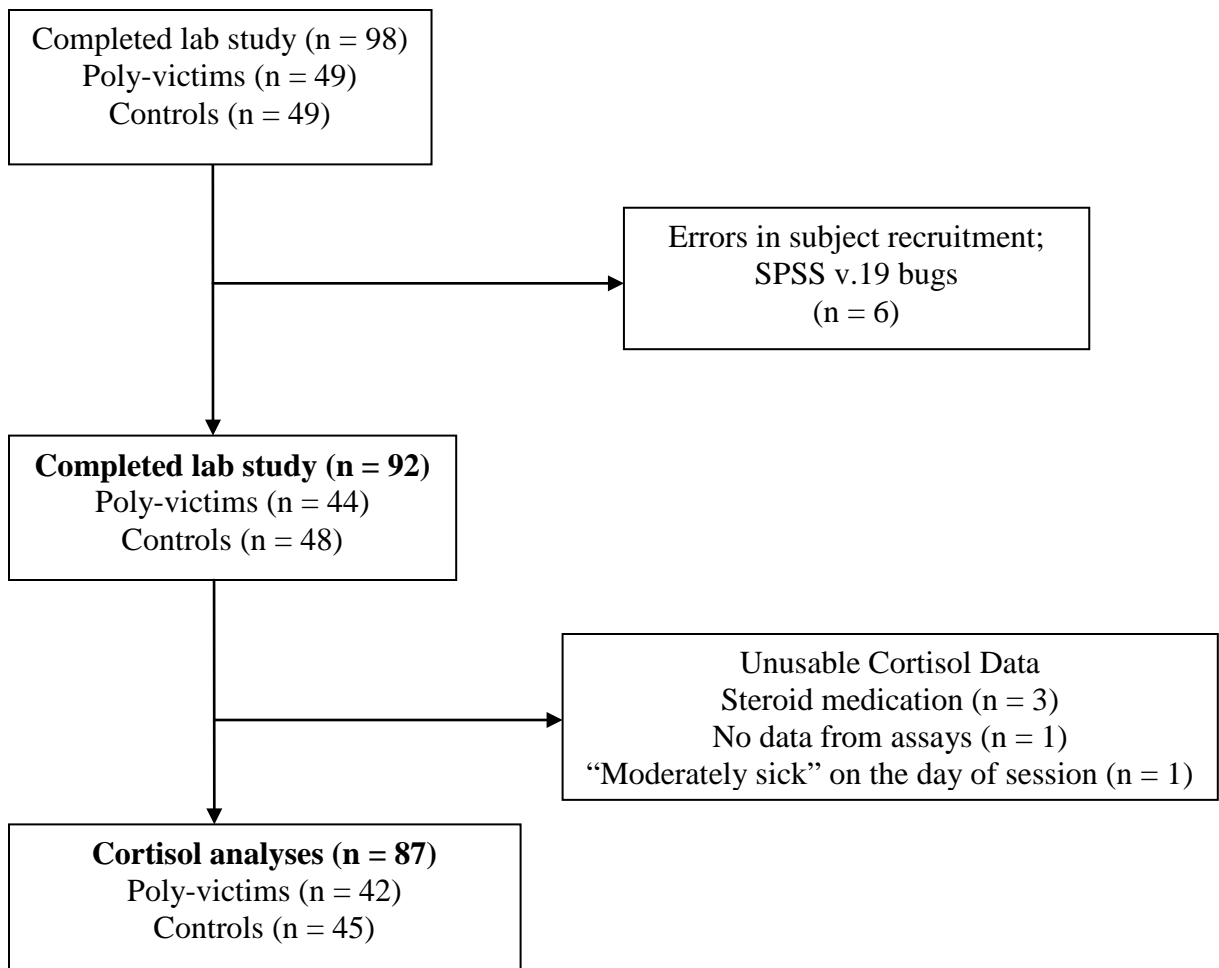
Figure 7. Participant flow diagram for the laboratory study.

5.3   RECRUITMENT SURVEY

The recruitment survey was used to identify poly-victims and controls. At the end of each violence questionnaire, an item asked whether participants were *currently* experiencing the specific type of victimization.

5.3.1   Demographics and Background Information

A short questionnaire addressed demographic characteristics and health factors, including participants' age, race/ethnicity, current medications, health conditions, and recreational/illegal drug use. Alcohol use was addressed by the question: "How many servings of alcohol do you drink per week, on average? A serving is equal to 12 oz of beer, 4 oz of wine, or 1.25 oz of hard liquor." Physical activity was assessed by the following item: "Structured physical activity is activity that lasts at least 10 minutes in a row without breaks, with intensity equal to or greater than a brisk walk. On average, how many minutes (or hours) per week do you engage in structured physical activity?" Parental obesity was identified by a positive to the following question: "Is your mother or father obese?" This item has been suggested as a reliable marker of family history of obesity (Paradis, Pérusse, Godin, & Vohl, 2008).

5.3.2 Violence Questionnaires

5.3.2.1 Physical abuse from family members or caregivers

Participants answered five items from the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998) to ascertain childhood physical abuse from family members or caregivers before the age of 18. An example item is, "People in my family hit me so hard that it left me with bruises or marks" and responses were on a five-point scale (1 = never true, 2 = rarely true, 3 = sometimes true, 4 = often true, 5 = very often true). Items were summed to yield scores ranging from 5 to 25, and participants with a score ≥ 8 were classified as positive for physical abuse. This clinical cut-point for physical abuse has been validated and has sensitivity and specificity at 0.85 or higher relative to clinical interview (Bernstein & Fink, 1998; Bernstein et al., 2003; Walker et al., 1999). The CTQ has strong test-retest reliability and convergent validity with clinical interview and therapist ratings (Bernstein et al., 1994; Bernstein et al., 2003). Among all of the women who completed the recruitment survey, Cronbach's alpha for the physical abuse scale was .67.

5.3.2.2 Sexual abuse from family members or caregivers

Participants answered five additional questions from the CTQ to ascertain childhood sexual abuse from family members or caregivers. Some items in the scale are phrased in objective and behavioral terms (e.g., "When I was growing up, someone touched me in a sexual way or made me touch them"), whereas other items require subjective evaluation (e.g., "When I was growing up, I believe I was sexually abused"). Items are rated on a 5 point scale as above and were summed to yield scores ranging from 5 to 25. Participants with a score ≥ 8 were classified as positive for sexual abuse. This clinical cut-point for sexual abuse has been validated and has

sensitivity and specificity at 0.85 or higher relative to clinical interview (Bernstein & Fink, 1998; Bernstein et al., 2003; Walker et al., 1999). Among all of the women who completed the recruitment survey, Cronbach's alpha for the sexual abuse scale was .93.

5.3.2.3 Peer victimization

Three items assessed victimization from peers and were based on a questionnaire developed by Romito and Grazzi (2007). Participants were asked whether they had ever experienced the following from peers, classmates, or friends (but not romantic partners or family) before the age of 18: 1) Being insulted, denigrated, threatened, or repetitively controlled; 2) Being slapped or kicked repetitively, being beaten up, hit with an object, burned or other serious physical aggressions; or 3) Being fondled without consent, being forced to touch another person, or being raped or attempted rape. Possible answers were "no = 1," "once or twice = 2," or "more often = 3." Participants who respond with "more often" to at least one item of violence by peers were scored as positive for peer victimization. Previous use of this measure showed that females reporting peer victimization were significantly more likely to have depressive symptoms, panic attacks, and eating problems compared to females not endorsing peer victimization (Romito & Grassi, 2007). Among all of the women who completed the recruitment survey, Cronbach's alpha for the peer victimization scale was .41.

5.3.2.4 Intimate partner victimization

The same measure used for peer victimization will also be used to assess intimate partner violence, except that participants were asked whether they had ever experienced psychological, physical, or sexual violence from romantic partners. Possible answers are "no = 1," "once or twice = 2," or "more often = 3." Participants who respond with "more often" to at least one item

of violence by peers were scored as positive for intimate partner victimization. In a previous report by Romito and Grassi (2007), females reporting intimate partner victimization had significantly higher odds of having depressive symptoms, panic attacks, eating problems, and suicide attempts compared to females not endorsing intimate partner victimization. Among all of the women who completed the recruitment survey, Cronbach's alpha for the intimate partner victimization scale was .57.

5.3.2.5 Community violence

The Community Experiences Questionnaire (CEQ; Schwartz & Proctor, 2000) was a self-report questionnaire instructing participants to report violence incidents that occurred prior to age 18. Eleven items measured exposure to community violence by direct victimization (e.g., "How many times has somebody threatened to hurt you physically?" and "How many times has someone stolen something from you using violence?"). Participants were asked to report the frequency of which they had experienced each item on a 4-point rating scale (1 = never, 2 = once, 3 = a few times, 4 = lots of times). Follow-up questions were added to three items of physical violence to verify that the exposure had not been previously reported. For example, "Was this experience reported in response to an earlier question in this survey?" Possible answers are "Yes—the event I'm thinking of has been addressed by another questionnaire" or "No—the event I am thinking of has not been reported previously." For the purpose of the present study, we dichotomized summed scores in order to match severity and frequency indices with other victimization scales. Participants were classified as positive for community violence if they reported (at a minimum) any item of direct victimization occurring "a few times" or three separate items occurring "once." Among all of the women who completed the recruitment survey, Cronbach's alpha for the community violence scale was .62.

5.3.2.6 Witnessing victimization

The CEQ included a second scale that had 14 items measuring witnessing violence. Sample items were "How many times have you seen somebody get robbed or have something stolen from them by force--like a person beating somebody up and then taking their money?" and "How many times have you seen somebody else get chased by gangs, kids, or adults?" Participants were asked to report the frequency of which they had experienced each item on a 4-point rating scale (1 = never, 2 = once, 3 = a few times, 4 = lots of times). Due to high reported rates of witnessing violence, the threshold for this scale was increased to identify individuals who were victimized by witnessing violence. Participants were classified as positive for witnessing violence if they reported (at a minimum) three items that occurred with a frequency of "a few times" or "lots of times." Among all of the women who completed the recruitment survey, Cronbach's alpha for the witnessing violence scale was .80.

5.3.3   Emotion Questionnaires

5.3.3.1 PTSD symptoms

Symptoms of current PTSD were measured by the Impact of Events-Revised scale (Weiss & Marmar, 1997). Even though participants were excluded on previous diagnosis of or treatment for PTSD, sub-clinical symptoms of PTSD were measureable. The 22-item questionnaire measured domains of intrusion, avoidance, and hyper-arousal. Instructions were as follows: "Below is a list of difficulties people sometimes have after experiencing a dangerous threat to their own well-being, or witnessing a threat to someone else's well-being. Please read each item, and then indicate how distressing each difficulty has been for you during the past month. If you have never experienced or witnessed a threat to well-being, circle '0' for 'not at all' in response

to all items." Respondents were asked to rate each item on a five-point scale (0 = not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit, and 4 = extremely). Sample items were: "I found myself acting or feeling as though I was back at that time" and "I feel watchful or on guard." The Impact of Events scale has shown good internal consistency and validity for detecting relative differences in the responses to the traumatic events of varying severity (Weiss & Marmar, 1997). For the purposes of the current study, the instructions asked for reflection on the past month, rather than the past week (as in the original scale), in order to assess chronic PTSD-like symptoms.

5.3.3.2 Depressive symptoms

Depressive symptoms were assessed by the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977). The CES-D is a 20-item scale measuring depressive symptoms on a four-point rating scale indicating frequency of experiencing each symptom in the past week (0 = rarely or none of the time, 1 = some or a little of the time, 2 = occasionally or a moderate amount of time, and 3 = most or all of the time). The CES-D has well-established reliability (Cronbach's alpha = 0.85) and has been shown to correlate well with other depressive symptom questionnaires and with interview assessments of severity of depression (Fechner-Bates, Coyne, & Schwenk, 1994).

5.3.3.3 Trait anger

Participants completed the Trait Anger scale of the State-Trait Personality Inventory (Spielberger, Jacobs, Russell, & Crane, 1983). The Spielberger Trait Anger scale contained 10 statements concerning the frequency with which the emotion of anger is experienced. Example items reflecting Trait Anger were, "I am quick-tempered" and "I feel infuriated when I do a job

and get a poor evaluation." Each statement was rated on a four-point scale (1 = almost never, 2 = sometimes, 3 = often, 4 = almost always); high scores reflected high levels of anger. Previous work showed that the Trait Anger scale has good internal consistency (0.81-0.92) and validity with other measures of anger and hostility (Spielberger et al., 1983).

5.3.3.4 Trait hostility

Participants completed a subset of the Cook and Medley Hostility Scale (Cook & Medley, 1954), consisting of 26 items that loaded on three factors related to CHD incidence: Cynicism, Hostile Affect, and Aggressive Responding (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). The scale also provided a summary score for Total Hostility. Sample items were, "It is safer to trust nobody" and "It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important." Participants indicated whether statements were false or true and higher scores indicated greater hostility. Scores on the items from the Total Hostility Scale were relatively stable across four years in adolescents, r (85) = .56 (Woodall & Matthews, 1993).

5.3.3.5 Trait positive and negative affect

The trait version of the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) was included in the survey of questionnaires. Participants were asked to "Indicate to what extent that you generally feel this way, that is, how you feel on average?" The PANAS questionnaire consisted of 27 items, 10 items each measuring global positive and negative dispositional affect, as well as 7 specific emotional states: angry, sad, happy, cheerful, sleepy, calm and tired. Each adjective was rated on a 5-point scale (1 = very slightly or not at all, 2 = a little, 3 = moderately, 4 = quite a bit, and 5 = extremely). The PANAS assesses positive

and negative activation and has been shown to have strong internal reliability (Watson et al., 1988). Crawford and Henry (2004) examined the measurement properties of the PANAS in a large non-clinical sample and Cronbach's alpha for positive affect and negative affect scales were .89 and .85 respectively, and the scales were relatively independent of each other.

5.3.3.6 Perceived Stress

The Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983) measured appraisal of situations in the participant's life over the past month. The 10-item self-report questionnaire ascertained the perception that one's life is unpredictable, uncontrollable, and overloaded. Items were rated on a 5-point scale: 0 = never, 1 = almost never, 2 = sometimes, 3 = fairly often, 4 = very often. A sample items is: "In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?" The Perceived Stress Scale has adequate reliability and has been correlated with life-event scores, depression, and anxiety (Cohen et al., 1983).

5.3.3.7 Emotion regulation

The Emotion Regulation Questionnaire (Gross & John, 2003) was used to measure trait-like differences in two emotion regulation strategies, emotional suppression and cognitive reappraisal. Examples of suppression items are "I control my emotions by not expressing them" and "I keep my emotions to myself." Examples of cognitive reappraisal items are "When I'm faced with a stressful situation, I make myself think about it in a way that helps me stay calm" and "When I want to feel less negative emotion (such as sadness or anger), I change what I'm thinking about." Participants rated each item on a seven point scale (1 = strongly disagree, 2 = somewhat disagree, 3 = disagree, 4 = neither, 5 = agree, 6 = somewhat agree, and 7 = strongly

agree). Previous research on the validity of the Emotion Regulation Questionnaire (Gross & John, 2003) suggested that emotion suppression was associated with experiencing less positive emotion, greater negative emotion, worse interpersonal functioning, and a low sense of well-being. On the other hand, cognitive reappraisal was linked to experiencing greater positive emotion, less negative emotion, better social relationships, and a stronger sense of well-being. The Emotion Regulation Questionnaire has been shown to have strong convergent and discriminant validity (Gross & John, 2003).

5.3.4   Eating Behavior Questionnaires

5.3.4.1 Binge eating

The Binge Eating Scale (BES; Gormally, Black, Daston, & Rardin, 1982) was originally developed to identify binge eaters within an obese population. Severity of binge eating was assessed by behavioral indicators (eating large amounts of food) and feelings or cognitions during a binge episode (loss of control, guilt, and fear of being unable to stop eating). The 16 items do not specify a time frame and are differentially weighted yielding a continuous measure of binge eating pathology ranging from 0 to 46. Scores of ≥ 27 have conventionally served as a cut-point for identifying the presence of severe binge eating and ≤ 17 as a cut-point for mild or no binge eating (Greeno, Marcus, & Wing, 1995). The BES has good test-retest reliability ($r = .87$, $p < .001$) (Timmerman, 1999), and scores have been shown to be positively associated with fasting blood sugar, diastolic blood pressure, and BMI (Meneghini, Spadola, & Florez, 2006).

5.3.4.2 Dietary restraint, emotional eating, and uncontrolled eating

The Three Factor Eating Questionnaire (TFEQ, Stunkard & Messick, 1985) was a self-report questionnaire measuring dietary restraint as well as other eating behaviors. Originally 51 items, Karlsson and colleagues (2000) developed a revised version of the TFEQ containing 18 items. The scale measured three domains of eating behaviors: cognitive restraint (6 items; "I do not eat some foods because they make me fat"), emotional eating (3 items; "When I feel anxious, I find myself eating"), and uncontrolled eating (9 items; "Sometimes when I start eating, I just can't seem to stop"). Participants rated each item on a 4-point Likert scale (1 = definitely false, 2 = mostly false, 3 = mostly true, and 4 = definitely true). Responses to items within each domain were summed. Higher scores indicated a greater tendency to exhibit that domain's behavior. Although the measure was originally validated in obese subjects, later research suggested that the TFEQ was valid in non-obese populations of varying age ranges (de Lauzon et al., 2004; Hyland, Irvine, Thacker, Dann, & Dennis, 1989). The TFEQ has been shown to correlate highly with another measure of eating behavior, the Dutch Eating Behavior Questionnaire (Hyland et al., 1989). Each of the scales has good internal consistency, convergent validity, and discriminant validity (de Lauzon et al., 2004).

5.4   LABORATORY MEASURES

5.4.1   Health Behavior Questionnaire

The following health behaviors occurring on the day of and the day previous to the laboratory session were measured due to their associations with cortisol stress response (Stewart & Seeman,

2000): exercise, eating, tobacco use, alcohol use, medication use, sleep duration the previous night, menstrual cycle (days since the beginning of last menstruation), and perceived health status.

5.4.2   Childhood Socioeconomic Status (SES)

Childhood SES was measured using the MacArthur Scale of Subjective Social Status (Adler et al., 1994). A "social ladder" was presented on a worksheet and the participant was asked to place an "X" on the rung on which they felt that their family stood on the ladder compared to other Americans. One ladder reflecting traditional SES factors (money, education, job) was used to represent childhood and family SES, with a scale ranging from 1 to 9. The distribution of responses was leptokurtic; 86% of the sample reported being between a 5 and 7 on the ladder. Therefore, we divided the responses into three equally distributed categories based on responses. With regard to our sample, the lower SES category represented 2 through 5 on the ladder (n = 30), the middle SES category represented a 6 on the ladder (n = 33), and the higher SES category represented a 7 through 9 on the ladder (n = 29).

5.4.3   State Affect

The state version of the PANAS (Watson et al., 1988) was administered to participants during the pre-task rest period, post-task recovery period, and post-video period. Participants were asked to indicate "How do you feel right now, that is, at the present moment?" The PANAS questionnaire consisted of 27 items, 10 items each measuring global positive and negative dispositional affect, as well as 7 specific emotional states: angry, sad, happy, cheerful, sleepy,

calm and tired. Each adjective was rated on a 5-point scale (1 = very slightly or not at all, 2 = a little, 3 = moderately, 4 = quite a bit, and 5 = extremely). Responses within the positive affect category and negative affect category were summed.

5.4.4   Manipulation Check

As used in previous studies of the Trier Social Stress Test (Gruenewald, Kemeny, Aziz, & Fahey, 2004), a questionnaire with 10 items measured perception of the stress task. Example items were "Overall, on the speech task I thought I performed: (1 = extremely poor to 7 = extremely well)" and "Do you think the panel of evaluators liked you? (1 = not at all to 7 = very much)." All responses were on a 7-point likert scale.

A principal components analysis was conducted on the 10 items with oblique rotation (direct oblimin). An initial analysis was run to obtain eigenvalues for each component in the data. The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis, KMO = .65. Bartlett's test of sphericity x^2 (45) = 410.62, p < .01, indicated that correlations between items were sufficiently large for a principal component analysis.

Three components had eigenvalues over Kaiser's criterion of 1 and included items with communalities above .70. The three components explained 66.35% of the variance. Two items had loadings less than .40 ("Overall, on the math task I thought I performed…" and "I thought the task was threatening."); therefore, we excluded these items from the identified components. Component 1 represented perceived task performance with three items (e.g., "How do you think the panel of evaluators would rate your performance overall?") with an eigenvalue of 3.48. Component 2 represented perceived task difficulty with two items (e.g., "Overall, I thought the speech and math tasks were challenging.") with an eigenvalue of 1.69. Component 3 represented

perceived interpersonal evaluation with three items (e.g., "Do you think the panel of evaluators accepted you?") with an eigenvalue of 1.47. Each item loaded heavily on one component; factor loadings were between .79 and .95 for each item on its component. Therefore, factor scores were created by summing the responses to the items within each component. Table 1 shows the correlations between factors.

Table 1. Correlations between factors created from participants' impressions of the Trier Social Stress Test

	Perceived Task Performance [a]	Perceived Task Difficulty [b]	Perceived Interpersonal Evaluation [c]
Perceived Task Performance	-	-.08	.39**
Perceived Task Difficulty		-	-.07

** $p < .001$

5.4.5   Body mass index

Weight was measured without shoes and in light indoor clothing using a balance beam scale. Height was measured without shoes using a fixed stadiometer. BMI was calculated as weight in (lbs/height in inches^2)*703.

5.4.6 Waist circumference

WC was measured in centimeters at the level of the natural waist, defined as the narrowest part of the torso as seen from the anterior aspect. In cases where a waist narrowing was difficult to identify, the measurement was taken at the smallest horizontal circumference in the area between the ribs and the iliac crest.

5.5 STRESS EATING

Based on laboratory eating paradigms suggested by Oliver et al. (2000), snacks were high fat and low fat options within categories of bland, salty, and sweet foods. Bland low fat and high fat foods were raw carrots and raw almonds. Salty low fat and high fat foods were pretzels and potato chips. Sweet low fat and high fat foods were LifeSavers Gummies® and Peanut M&Ms®. Each food was weighed in its bowl prior to the experimental procedure. After the participant finished the protocol, the bowls with food were weighed again to identify grams of food consumed. Manufacturers' information was used to convert gram weight consumption of each food into calorie intake. Calories eaten were summed across food types, and a total calorie intake variable was created. The variable of total calories consumed was highly correlated with total sugar calories consumed ($r = .87$, $p < .001$) and total fat calories consumed ($r = .91$, $p < .001$).

5.6   CORTISOL STRESS RESPONSE

Salivary free cortisol strongly reflects the amount of free cortisol in plasma (Kudielkaa & Kirschbaum, 2005). Three samples were obtained: 1) just prior to the stressor, 2) ~30 minutes after initiation of the stressor, and 3) ~50 minutes after initiation of the stressor. Saliva samples were collected with salivettes, plastic vials with cotton dental rolls inside, and frozen until laboratory analysis. Samples were shipped to Dr. Clemens Kirschbaum's laboratory at the University of Dresden. Salivary cortisol concentrations were analyzed using a commercially available chemiluminescence assay (CLIA, IBL-International; Hamburg, Germany). The intra- and inter-assay coefficients of variance were both below 8%. The lower limit of sensitivity was 0.4 nmol/L and the upper limit of sensitivity was over 110 nmol/L. Of the 87 subjects with valid cortisol data, 2 subjects were missing their time 1 cortisol sample, due to un-measurable cortisol based on assay reports. The missing data were imputed by using the mean of the other two samples for each subject.

6.0   ANALYTIC PLAN

CIV was measured as a summary score ranging from zero to six depending on endorsement of physical abuse, sexual abuse, peer victimization, intimate partner violence, community violence, and witnessing violence. For the dichotomous CIV variable, control participants were given a score of 0 and poly-victims were given a score of 1.

The variables of BMI and WC were positively skewed with two participants having scores ≥ 3 standard deviations from the mean. Therefore, regression diagnostics were conducted to examine each case's influence on the regression parameters and included: Cook's distance, leverage, Mahalanobis distances, DFBeta, and DFFit (for review see textbook by Field, 2009). Regression diagnostics were run for analyses associated with each hypothesis. Outliers on BMI and WC were not unduly influencing the results of linear regression analyses; therefore, results presented here include all participants run in the lab session (n = 92 or n = 87 for cortisol).

6.1   APPROACHES TO PRIMARY HYPOTHESES

6.1.1   Hypothesis 1

Women reporting poly-victimization will have higher BMI and WC compared to controls. BMI and WC were highly correlated in the full sample ($r = .94$, $p < .001$); therefore, BMI and WC

were examined as dependent variables in separate models. Hypothesis 1 was tested using linear regression, with CIV (categorical: controls and poly-victims) as the independent variable and BMI or WC as the dependent variable.

6.1.2 Hypothesis 2

Women reporting poly-victimization will consume more calories after a stressor compared to controls. Hypothesis 2 was tested using linear regression, with CIV as the independent variable and calories consumed during the free-access snack period as the dependent variable.

6.1.3 Hypothesis 3

Stress eating will mediate the relationship between CIV and BMI and WC. Hypothesis 3 was tested using the bootstrap method (Efron & Tibshirani, 1993; Shrout & Bolger, 2002). The bootstrap method is preferable for small sample sizes and for data that violate normality assumptions. From the bootstrap sampling distribution, the significance test of the mediated effect and confidence interval estimate was obtained. To compute path *a*, the independent variable was CIV and the dependent variable was calories consumed during stress eating. To compute path *b*, the independent variable was calories consumed and the dependent variable was BMI or WC, adjusting for CIV. If path *a* or path *b* was not significant, then the significance of the indirect path was not computed.

6.1.4   Hypothesis 4

Women reporting poly-victimization will have greater cortisol response in response to a laboratory stressor compared to controls. Hypothesis 4 was analyzed using mixed design factorial ANOVA (first factor was CIV, second repeated factor was cortisol trials). In the case of significant interaction effects, between-subjects comparisons were examined at single time points using Sidak corrections for multiple comparisons.

Mauchly's test indicated that the assumption of sphericity had been violated for the main effect of trials, χ^2 (2) = 18.76, $p < .001$. Therefore, the degrees of freedom were corrected using the Huynh-Feldt estimates of sphericity (ε = .86 for the main effect of trials). The Huynh-Feldt estimates were used for analyses with and without covariates.

Cortisol values at each time point were not normally distributed. Results from the mixed design factorial ANOVA showed that the raw cortisol data at time 2 and time 3 violated the assumption of homogeneity of variance using the Levene's test, which may compromise the accuracy of the F-test for the between-subjects variable. Therefore, log transformed cortisol values were used for mixed design factorial ANOVA models. Models with transformed cortisol showed homogeneity of variance.

Supplemental analyses included linear regression tests of cortisol "reactivity" and "recovery." Cortisol reactivity was calculated as cortisol at trial 2 minus cortisol at trial 1, controlling for cortisol at trial 1. Cortisol recovery was calculated as cortisol at trial 3 minus cortisol at trial 1, controlling for cortisol at trial 1 and 2.

Two other variables were created to capture repeated measurements of cortisol. Area under the curve with respect to ground (AUC_G) is the total area under the curve of all measurements and reflects total cortisol output. AUC_G takes into account both the overall

intensity of the response (distance of the measures from the ground) and sensitivity of response (difference between the measurements). Area under the curve with respect to increase (AUC_I) is calculated in reference to the baseline measurement and reflects changes over time, thus emphasizing the sensitivity of the response. AUC_G and AUC_I were calculated using the logged cortisol values in order to reduce skew. There was a moderate positive correlation between AUC_G and AUC_I ($r = .38$, $p < .001$). Table 2 provides a correlation matrix of the various cortisol measurements.

Table 2. Correlations between cortisol measurements. Values above the diagonal are correlations with logged values; values below the diagonal are correlations with raw values (n = 87).

	Trial 1	Trial 2	Trial 3	Mean Output	AUC_G	AUC_I	Δ Trial 1 - 2	Δ Trial 1 - 3
Trial 1	-	.41	.49	.71	.61	-.44	-.44	-.39
Trial 2	.49	-	.78	.90	.90	.62	.64	.45
Trial 3	.55	.79	-	.89	.81	.46	.36	.62
Mean output	.75	.91	.91	-	.93	.32	.29	.31
AUC_G	.67	.95	.87	.98	-	.38	.37	.31
AUC_I	-.25*	.71	.52	.44	.54	-	.98	.87
Δ Trial 1 - 2	-.21*	.75	.46	.45	.56	.99	-	.77
Δ Trial 1 - 3	-.38	.39	.56	.27	.30	.82	.72	-

Note: All correlations are significant $p < .01$ unless otherwise noted. AUC_G = area under the curve with respect to ground; AUC_I = area under the curve with respect to increase

* $p < .05$

6.1.5   Hypothesis 5

Cortisol response will mediate the relationship between CIV and BMI and WC. Mediation in hypothesis 5 was tested using the bootstrap method. To compute path *a*, the independent variable was CIV and the dependent variable was cortisol response (AUC_G or AUC_I). To compute path *b*, the independent variable was cortisol response and the dependent variable was BMI or WC, adjusting for CIV.

6.1.6   Hypothesis 6

Cortisol response will mediate the relationship between CIV and stress eating. Mediation in hypothesis 6 was tested using the bootstrap method. To compute path *a*, the independent variable was CIV and the dependent variable was cortisol response (AUC_G or AUC_I). To compute path *b*, the independent variable was cortisol response and the dependent variable was calories consumed after the stressor, adjusting for CIV.

6.2   EFFECT SIZE ESTIMATION

There was minimal literature reporting an effect size for the relationship between poly-victimization, cortisol reactivity, stress eating, and BMI/WC. Hence, calculations were used to predict an effect size estimate based on number of subjects. To calculate an effect size for univariate general linear models, the following information was needed: u = number of independent variables; v = *df* for the error variance = N – (u +1); significance level = .05; and

power = .80. Power analyses were run using G*Power (Faul, Erdfelder, Buchner, & Lang, 2009). For hypotheses 1 and 2 there was one independent variable (CIV). The resultant effect size for 92 participants would be $f^2 = 0.09$. For mediation in hypothesis 3, there were two independent variables (for path *b*: CIV and calories consumed), and the resultant effect size for 92 participants would be $f^2 = 0.11$. Hypotheses 4 through 6 have similar numbers of independent variables, but effect sizes were calculated for 87 participants. The resulting effect size estimates were $f^2 = 0.09$ and $f^2 = 0.12$. Overall, it appears that the proposed study will be able to detect a medium effect size, which can also be expressed as $r^2 = 0.12$. Figure 8 portrays the relationship of sample size, power, and effect size over a wide range of participants.
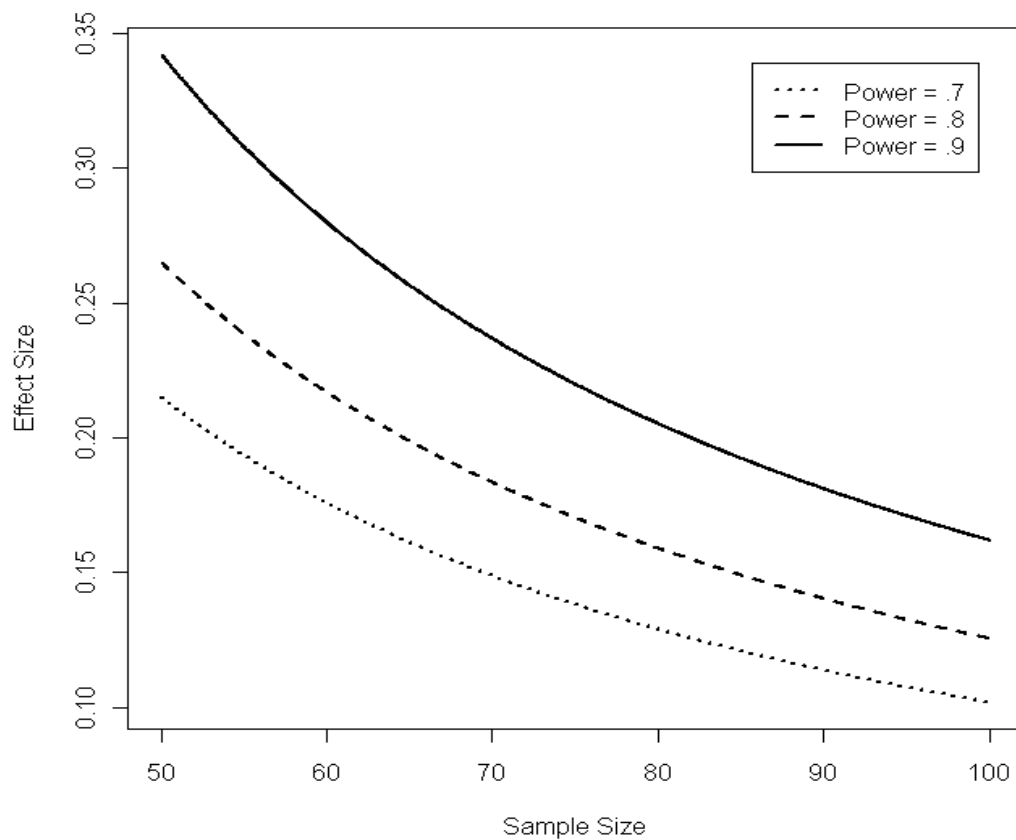
Figure 8. Graph representing the interrelationships between effect size, sample size, and power.

Although no studies examine the magnitude of relationships for poly-victimization, there is literature that examines variables similar to those in the current study. Epel and colleagues (2001) looked at the relationship between cortisol reactivity and eating post-task on days with stress and a day without stress. They showed that high cortisol reactors ate more calories than low cortisol reactors, but only on the stress day, Cohen's d = 2.56 (r^2 = .79). Heim and colleagues (2000) looked at the relationship between childhood abuse and cortisol reactivity. The relationship between abuse (w/ and w/o depression) and peak cortisol was Cohen's d = 0.82 (r^2 = .38). Elzinga et al. (2010) completed a study that looked at childhood abuse (w/ social anxiety) and cortisol reactivity, and the estimated effect size was f^2 = 0.42, (r^2 = .30). Epel et al. (2000) examined the relationship between cortisol reactivity and WHR and found an effect size of Cohen's d = 0.71 (r^2 = .33). Finally, Laitinen et al. (2002) looked at individuals at age 31 who endorsed stress eating and showed that stress eating predicted obesity, controlling for BMI at age 14, OR = 3.24, p < .001. In sum, previous literature suggests that relationships similar to those in my dissertation find medium to large effect sizes.

6.3   COVARIATES

Undergraduate women were examined in the current study, which means that there was a restricted age range. In the full sample of 92 women, age was not significantly correlated with CIV, BMI, WC, total calories consumed, or cortisol at any time point (*p*s > .13). Therefore, age was not included as a covariate in subsequent analyses.

Birth control pill (BCP) use was not significantly correlated with CIV, BMI, WC, or total calories consumed (*p*s > .23), but BCP use was significantly and positively correlated with

cortisol at time 2 ($r = .20$, $p = .06$) and time 3 ($r = .23$, $p = .03$). Thus, BCP use was included as a covariate in analyses examining cortisol response.

Poly-victims tended to have lower childhood SES than controls (χ^2 (1) = 2.64, p ≤ .10). In order to control for the CIV group trend, all hypotheses adjusted for childhood SES.

Several analyses were conducted in order to understand the composition and influence of race in the study sample. Cell sizes were too small to use a chi-square for specific racial categories within the control and poly-victim groups. However, chi-square analyses comparing the counts of whites and non-whites in the control and poly-victim groups suggest nonsignificant differences (χ^2 (1) = 1.58, $p = .21$). There was no significant difference between whites and nonwhites on BMI (t (90) = .77, $p = .45$) or total calories consumed (t (90) = -.19, $p = .85$). There was no significant difference between whites and nonwhites on AUC_G (t (86) = .98, $p = .33$) or AUC_I (t (86) = .29, $p = .78$). Due to the relative homogeneity of the sample with regard to race, as well as the lack of relationship between race and BMI, calories consumed, or cortisol, analyses did not adjust for race.

The design of the study allowed for collection of data that may confound or suppress significant relationships. Table 3 shows correlations between possible variables of interest and BMI, total calories consumed, and AUC_G.

Table 3. Pearson correlations between potential covariates and BMI, stress eating, and cortisol response

	BMI	Total Calories	AUC$_G$
	(n = 92)	(n = 92)	(n = 87)
Lifestyle Variables			
Childhood SES (0 = Low SES, 1 = Middle SES, 2 = High SES)	-.16	-.03	-.08
Family history of obesity (0 = no, 1 = yes)	.20**	.07	.05
BCP use (0 = no, 1 = yes)	-.01	.01	.09
Alcohol use (servings per week)	.19*	-.04	.12
Physical activity (hours per week)	-.08	.07	-.04
Laboratory Study Variables			
Start time (0 = 2:30-2:59, 1 = 3:00-3:30)	.003	-.15	-.08
Amount of time since last ate	-.07	.15	-.06
Hunger at study entry (1 = not at all hungry to 5 = very hungry)	-.09	.31***	.03
Health over past day (1 = healthy, 2 = a little sick)	.07	.12	.14
Health over past week (1 = health, 2 = a little sick)	.11	-.08	.07
Sleep duration on previous night	-.01	.11	-.01
Days since last menstrual cycle	-.13	-.12	-.08

* $p \leq .10$, ** $p \leq .05$, *** $p \leq .01$

Note: BMI = body mass index; AUC$_G$ = area under the curve with respect to ground; SES = socioeconomic status; BCP = birth control pill

In analyses with BMI, additional covariates include family history of obesity and alcohol use. In analyses with stress eating, additional covariates include hunger at study entry. In analyses with cortisol response, no additional covariates were identified. Table 4 provides a summary of the covariate decisions.

Table 4. Summary of covariate decisions

Analyses with BMI/WC (Hypothesis 1, Hypothesis 3, Hypothesis 5)

    1. Univariate

    2. Adjust for childhood SES, family history of obesity, and alcohol use

Analyses with Stress Eating (Hypothesis 2, Hypothesis 3, Hypothesis 6)

    1. Univariate

    2. Adjust for childhood SES and hunger at study entry

Analyses with Cortisol Response to Stress (Hypothesis 4, Hypothesis 5, Hypothesis 6)

    1. Univariate

    2. Adjust for childhood SES and BCP use

Note: BMI = body mass index; WC = waist circumference; SES = socioeconomic status

There were several subjects taking medications including selective serotonin reuptake inhibitors (e.g., Prozac, Celexa; n = 6), asthma medications (e.g., Albuterol, Singulair; n = 6), allergy medications (e.g., Zyrtec, Claritin; n = 6), and cold medications (e.g., Dayquil, Sudafed; n = 4). Chi-square analyses showed that the number of subjects taking each group of medications was not significantly different between controls and poly-victims ($ps > .34$). In order to examine

whether medication use was confounding hypothesized relationships, analyses were re-run excluding individuals taking the medications within each group (i.e., SSRIs, asthma meds, allergy meds, and cold meds). Findings remained unchanged, so the results are reported using the full sample.

7.0   RESULTS

7.1   PARTICIPANT CHARACTERISTICS

Table 5 shows participant characteristics for the full sample. The sample was 80% white and approximately 18 years of age. About a third of the sample reported having lower childhood SES (2 – 5 on the SES ladder) and about a quarter of the sample had at least one obese parent. Approximately half the participants were taking BCPs. On average, participants consumed less than three servings of alcohol per week and exercised more than 4 hours per week. Finally, the mean BMI and WC were in the normal/healthy range. Seventeen percent of the sample was overweight and 11% of the sample was obese.

Table 5. Sample characteristics for the full sample (N = 92)

	M (SD)	Range	% (n)
Age	18.6 (1.5)	18 - 29	
Race			80 (74) White
			9 (8) Asian
			4 (4) Black
			6 (5) Mixed
			1 (1) Hispanic
Lower Childhood SES			32.6 (30)
SES family ladder	5.9 (1.2)	2 - 9	
Alcohol, svgs/wk	2.7 (4.0)	0 - 20	
Physical Activity, hrs/wk	4.2 (3.1)	0.0 – 15.0	
WC, centimeters	73.7 (9.6)	61.0 – 116.50	
BMI, lbs/in^2	23.9 (4.8)	16.1 – 43.5	
Normal weight			71.7 (66)
Overweight			17.4 (16)
Obese			10.9 (10)
At least one obese parent			23.9 (22)
BCP use			45.7 (42)

Table 6 compares poly-victims and controls on study variables. There were no differences on age, BCP use, alcohol use, physical activity per week, or family history of obesity.

Table 6. Sample characteristics by group

	Controls (n = 48)	Poly-victims (n = 44)	p value from t-test or chi-square
Age, M (SD)	18.4 (.64)	18.9 (2.1)	.13
Race, % (n)	85% (41) White	75% (33) White	.21[a]
	6% (3) Asian	11% (5) Asian	
	2% (1) Black	7% (3) Black	
	6% (3) Mixed	5% (2) Mixed	
		1% (1) Hispanic	
% (n) Lower Childhood SES (2 – 5 on the SES ladder)	25.0% (12)	40.9% (18)	.10
% (n) taking BCPs	39.6% (19)	52.3% (23)	.22
Alcohol svgs/wk, M (SD)	2.4 (3.6)	3.0 (4.5)	.49
Physical Activity hrs/wk, M (SD)	4.7 (3.3)	3.7 (2.7)	.14
% (n) with Obese parents	18.8% (9)	29.5% (13)	.23

[a] Chi-square analysis examined group differences in whites and non-whites.

Table 7 compares poly-victims and controls on psychosocial variables that were measured in the recruitment survey. Poly-victims reported faring worse on most psychosocial variables, including PTSD symptoms, depressive symptoms, trait hostility, negative affect, perceived stress, and binge eating symptoms. Poly-victims also tended to have greater trait anger and lower positive affect. Controls reported having significantly more emotional eating.

Table 7. Sample characteristics on psychosocial variables for the full sample and by group, means and standard deviations [a]

Psychosocial Variables	Full Sample (n = 92)	Controls (n = 48)	Poly-victims (n = 44)	T-test ($df = 90$) and p value
PTSD symptoms	0.30 (0.52)	0.09 (.29)	0.53 (.62)	-4.4, < .001
Depressive symptoms	13.3 (10.0)	9.1 (6.1)	17.9 (11.4)	-4.7, < .001
Trait Anger	17.6 (5.0)	16.7 (4.1)	18.5 (5.8)	-1.7, = .09
Hostility	37.2 (4.7)	36.3 (4.5)	38.2 (4.9)	-2.0, < .05
PANAS Negative Affect	30.1 (8.4)	28.2 (7.1)	32.2 (9.2)	-2.3, < .05
PANAS Positive Affect	45.7 (8.5)	47.1 (7.7)	44.2 (9.1)	1.7, < .10
Perceived Stress Scale	26.7 (6.6)	24.9 (6.5)	28.8 (6.2)	-2.9, < .01
ERQ suppression	14.4 (5.8)	14.4 (5.8)	14.4 (5.9)	.003, = .99
ERQ reappraisal	30.4 (5.7)	31.3 (5.1)	29.5 (6.3)	1.5, = .14
Binge eating score	10.7 (8.5)	8.6 (7.0)	12.9 (9.5)	-2.5, < .05
TFEQ restraint	15.7 (2.0)	15.8 (2.0)	15.5 (2.1)	0.6, = .53
TFEQ emotional eating	8.7 (2.4)	9.1 (2.0)	8.2 (2.6)	2.0, = .05
TFEQ uncontrolled eating	24.3 (3.6)	24.7 (2.9)	23.9 (4.2)	1.1, = .27

[a] All psychosocial variables are presented as sum scores except for PTSD symptoms, which is an item mean score

The distribution of violence exposure for the 44 poly-victims is presented in Table 8. Community violence and witnessing violence were the most frequent types of experiences, while sexual abuse was the most infrequent experience. Table 9 shows correlations between violence subtypes. Based on meeting threshold scores, peer violence was correlated with most other subtypes of violence exposure, and intimate partner violence was correlated with the least number of other subtypes of violence exposure. See Table 10 for a qualitative list of how violence exposures grouped together in the present sample.

Table 8. Frequency of types of victimization within the 44 poly-victims

Number of Exposures	% (n)
2 exposures	63.6 (28)
3 exposures	29.5 (13)
4 exposures	4.5 (2)
5 exposures	2.3 (1)
Type of Exposure	
Physical abuse	36.4 (16)
Sexual abuse	9.1 (4)
Peer victimization	18.2 (8)
Intimate partner victimization	20.5 (9)
Community Violence	79.5 (35)
Witnessing Violence	81.8 (36)

Table 9. Correlations between violence exposures; The numbers above the diagonal are Spearman's correlations between measures using threshold scores. The numbers below the diagonal are Pearson's correlations between measures using continuous scores.

	Physical Abuse	Sexual Abuse	Peer Violence	Intimate Partner violence	Community Violence	Witnessing Violence
Physical Abuse	-	.18	.27**	-.15	.29**	.46**
Sexual Abuse	.05	-	.31**	-.07	.05	.27**
Peer Violence	.13	.17	-	.29**	.24*	-.01
Intimate Partner Violence	-.08	-.03	.66**	-	.19	.04
Community Violence	.34**	.05	.33**	.13	-	.66**
Witnessing Violence	.37**	.23*	.27**	.08	.58**	-

* p ≤ .05, ** p ≤ .01

Table 10. Patterns of poly-victimization (n = 44)

	Number of Participants
2 Victimization Experiences	
Community + Witness	16
Physical + Witness	4
Partner + Community	2
Partner + Witness	2
Physical + Community	1
Sexual + Witness	1
Peer + Community	1
Peer + Physical	1
3 Victimization Experiences	
Physical + Community + Witness	7
Partner + Community + Witness	2
Peer + Partner + Community	2
Sexual + Community + Witness	1
Physical + Peer + Community	1
4 Victimization Experiences	
Physical + Sexual + Peer + Witness	1
Physical + Peer + Community + Witness	1
5 Victimization Experiences	
Physical + Sexual + Peer + Community + Witness	1

7.2   IMPRESSIONS OF THE TRIER SOCIAL STRESS TEST

There were no group differences in impressions of the TSST. Controls and poly-victims reported similar ratings on perceptions of task performance (t (90) = 0.18, p = .86), perceptions of task difficulty (t (90) = 1.04, p = .30), and perceptions of interpersonal evaluation (t (90) = 0.30, p = .76). Additionally, controls and poly-victims reported similar decreases in PANAS positive mood (t (90) = -1.06, p = .29) and increases in PANAS negative mood (t (90) = -0.23, p = .82) across the stress task. See Table 11 for group means.

Table 11. Group differences on impressions of the Trier Social Stress Test

	Controls	Poly-victims	T-test (df = 90) and
	M (SD)	M (SD)	p value
Perception of Task Performance [a]	8.79 (3.58)	8.66 (3.42)	0.18, p = .86
Perception of Task Difficulty [b]	10.73 (1.90)	10.27 (2.33)	1.04, p = .30
Perception of Interpersonal Evaluation [c]	9.13 (3.27)	8.89 (4.29)	0.30, p = .76
Change score in Positive Affect	-7.85 (6.30)	-6.52 (5.63)	-1.06, p = .29
Change score in Negative Affect	3.83 (5.34)	4.08 (4.96)	-0.23, p = .82

[a] Possible range is 3 to 21; high scores indicate better performance.
[b] Possible range is 2 to 14; high scores indicate more difficult task.
[c] Possible range is 3 to 21; high scores indicate more positive evaluation.

7.3   HYPOTHESIS 1

Women reporting poly-victimization will have higher BMI and WC compared to controls. See Table 12 for descriptive data on BMI and WC in controls and poly-victims.

Table 12. Mean, (standard deviation), and range for main outcome variables for the full sample and by group

	Full Sample (n = 92)	Controls (n = 48)	Poly-victims (n = 44)
Body Mass Index, lbs/in^2	23.92 (4.76), 16.10 – 43.50	22.68 (3.74), 16.10 – 31.15	25.28 (5.39), 17.67 – 43.50
Waist Circumference, cm	73.68 (9.60), 61.00 – 116.50	71.46 (7.60), 61.00 – 92.00	76.10 (10.97), 61.00 – 116.50
Total Calories Consumed	388.78 (272.98), 0 – 1197.04	393.06 (275.47), 0 – 1085.01	384.11 (273.35), 0 – 1197.04

Univariate linear regressions showed that CIV was associated with higher BMI (B = 2.61, $p <$ .01) and higher WC (B = 4.65, p = .02). The relationship between CIV and BMI remained significant after controlling for childhood SES, family history of obesity, and alcohol use (B = 2.03, p = .04). The relationship between CIV and WC became a trend after adjusting for childhood SES, family history of obesity, and alcohol use (B = 3.63, p = .07). See Table 13 for a

summary of the linear regression model with covariates. In sum, poly-victims had higher BMI than controls and tended to have higher WC than controls.

Table 13. Unstandardized coefficients from linear regression models examining associations between CIV and BMI or WC, adjusting for childhood SES, family history of obesity, and alcohol use.

	BMI			WC		
	B	*SE*	*p*	B	*SE*	*p*
Childhood SES	-0.76	0.60	.21	-1.00	1.22	.41
Obese parent	2.06	1.11	.07	5.10	2.27	.03
Alcohol use	0.24	0.12	.05	0.32	0.24	.19
CIV	2.03	.96	.04	3.63	1.97	.07

Note: CIV = childhood interpersonal violence (0 = controls, 1 = poly-victims); BMI = body mass index; WC = waist circumference; SES = socioeconomic status

7.4   HYPOTHESIS 2

Women reporting poly-victimization will consume more calories after a stressor compared to controls. See Table 12 for descriptive data on calorie consumption for controls and poly-victims. Univariate linear regressions showed that CIV was not associated with total calories consumed (B = -8.95, p = .88). Further analyses adjusted for childhood SES and hunger at study entry and showed similar nonsignificant results between CIV and total calories consumed (B = -20.42, p =

.72). See Table 14 for a summary of the linear regression model with covariates. Poly-victims did not consume more calories after a stressor compared to controls.

Table 14. Unstandardized coefficients from linear regression models examining associations between CIV and total calories, adjusting for childhood SES and hunger at study entry.

	Total calories		
	B	*SE*	*p*
Childhood SES	-1.72	34.99	.96
Hunger at study entry	81.64	26.36	< .01
CIV	-20.42	55.93	.72

Note: CIV = childhood interpersonal violence (0 = controls, 1 = poly-victims);

SES = socioeconomic status

7.5   HYPOTHESIS 3

Stress eating will mediate the relationship between CIV and BMI and WC. See Figure 2 for a model representing the hypothesized mediation pathway. Path *a* was not significant (hypothesis 2); therefore, bootstrapping methods to test mediation were unnecessary. Stress eating did not mediate the relationship between CIV and BMI/WC. Table 15 shows correlations between BMI, WC, and calories consumed.

Table 15. Correlations between anthropometric variables and calorie consumption

	BMI	WC	Total Calories Consumed
BMI	-	.94*	.11
WC		-	.07

* $p < .001$; Note: BMI = body mass index; WC = waist circumference

7.6   HYPOTHESIS 4

7.6.1   Mixed Design Factorial ANOVA (no covariates)

Women reporting poly-victimization will have greater cortisol response in response to a laboratory stressor compared to controls. Table 16 presents the raw and logged cortisol values for each trial for the full sample and by CIV group.

Table 16. Mean (standard deviation) and range of cortisol values at each trial for the full sample and by CIV group

	Full sample (n = 87)	Controls (n = 45)	Poly-victims (n = 42)
Raw Cortisol (nmol/L), Trial 1	9.1 (4.6), 2.6 – 34.1	9.7 (5.1), 2.6 – 34.1	8.5 (4.0), 3.1 – 18.8
Raw Cortisol (nmol/L), Trial 2	10.6 (6.8), 2.4 – 37.7	12.2 (7.9), 2.4 – 37.7	8.9 (4.8), 3.6 – 24.8
Raw Cortisol (nmol/L), Trial 3	8.5 (5.2), 1.4 – 33.1	9.9 (6.0), 2.3 – 33.1	6.9 (3.5), 1.4 – 19.2
Average cortisol level (nmol/L)	9.4 (4.8), 2.8 – 32.5	10.6 (5.5), 2.8 – 32.5	8.1 (3.6), 2.8 – 19.4
Logged Cortisol (nmol/L), Trial 1	0.91 (0.21), 0.42 – 1.53	0.94 (0.21), 0.42 – 1.53	0.88 (0.21), 0.49 – 1.27
Logged Cortisol (nmol/L), Trial 1	0.95 (0.24), 0.37 – 1.58	1.01 (0.26), 0.37 – 1.58	0.90 (0.21), 0.55 – 1.39
Logged Cortisol (nmol/L), Trial 1	0.86 (0.24), 0.14, 1.52	0.93 (0.24), 0.36 – 1.52	0.78 (0.23), 0.14 – 1.28
Average logged cortisol level (nmol/L)	0.93 (0.20), 0.44 – 1.51	0.98 (0.20), 0.44 – 1.51	0.87 (0.18), 0.45 – 1.29

Note: CIV = childhood interpersonal violence

Table 17 presents the main effects and interaction effects from the mixed design factorial ANOVA with logged cortisol values.

Table 17. Mixed Design Factorial ANOVA (without covariates), CIV predicting logged cortisol levels

	F test [a]	p value
Cortisol Trials	8.38	.001
CIV	6.58	.01
Cortisol Trials x CIV	2.00	.15

[a] Huynh-Feldt degrees of freedom for within-subject and interaction F-tests are (1.72, 145.84); degrees of freedom for between subject F-tests are (1, 85)

Note: CIV = childhood interpersonal violence

There was a significant within-subjects main effect of cortisol trials, suggesting that cortisol levels differed between trials. Pairwise comparisons from the ANOVA show that cortisol at trial 2 was significantly higher than cortisol at trial 3. Results also showed a significant between-subjects main effect for CIV. Poly-victims had lower average cortisol levels compared to controls. The interaction between cortisol trials and CIV group was nonsignificant.

Supplementary analysis tested whether CIV was associated with cortisol reactivity and recovery. See Table 18 for means and standard deviations of logged cortisol change scores by CIV group. Linear regressions showed that poly-victims tended to have lower cortisol reactivity compared to controls (B = -.09, p = .07), calculated as the change between trial 1 and trial 2 cortisol, controlling for cortisol level at trial 1. Poly-victims also tended to have greater cortisol

recovery, calculated as the change between trial 1 and trial 3 cortisol, controlling for cortisol at

trial 1 and trial 2 (B = -.06, p = .08).

Table 18. Means and standard deviations for change scores on logged cortisol levels

	Controls	Poly-victims
Cortisol Reactivity		
Δ Trial 1 and Trial 2 Cortisol	.070 (.275)	.011 (.213)
Cortisol Recovery		
Δ Trial 1 and Trial 3 Cortisol	-.008 (.245)	-.100 (.211)

7.6.2 Mixed Design Factorial ANCOVA (with covariates)

Table 19 shows the mixed design factorial ANCOVA adjusting for childhood SES and BCP use.

Table 19. Mixed Design Factorial ANOVA (adjusting for childhood SES and BCP use); CIV predicting logged cortisol levels

	F test [a]	*p* value
Cortisol trials	7.62	.001
Childhood SES	1.72	.19
BCP use	1.05	.31
CIV	8.43	.005
Cortisol x CIV	2.59	.09

[a] Huynh-Feldt degrees of freedom for within-subject and interaction F-tests are (1.74, 144.56); degrees of freedom for between subject F-tests are (1, 83)

Note: CIV = childhood interpersonal violence; SES = socioeconomic status; BCP = birth control pill

Results showed a significant within-subjects main effect for cortisol trials. Similar to the univariate analysis, cortisol at trial 2 was significantly greater than cortisol at trial 3. There was a significant between-subjects main effect for CIV; poly-victims had lower mean cortisol levels compared to controls. The mixed design factorial ANCOVA also showed a trend for the interaction between trials and CIV. See Figure 9 for a plot of cortisol by CIV group.
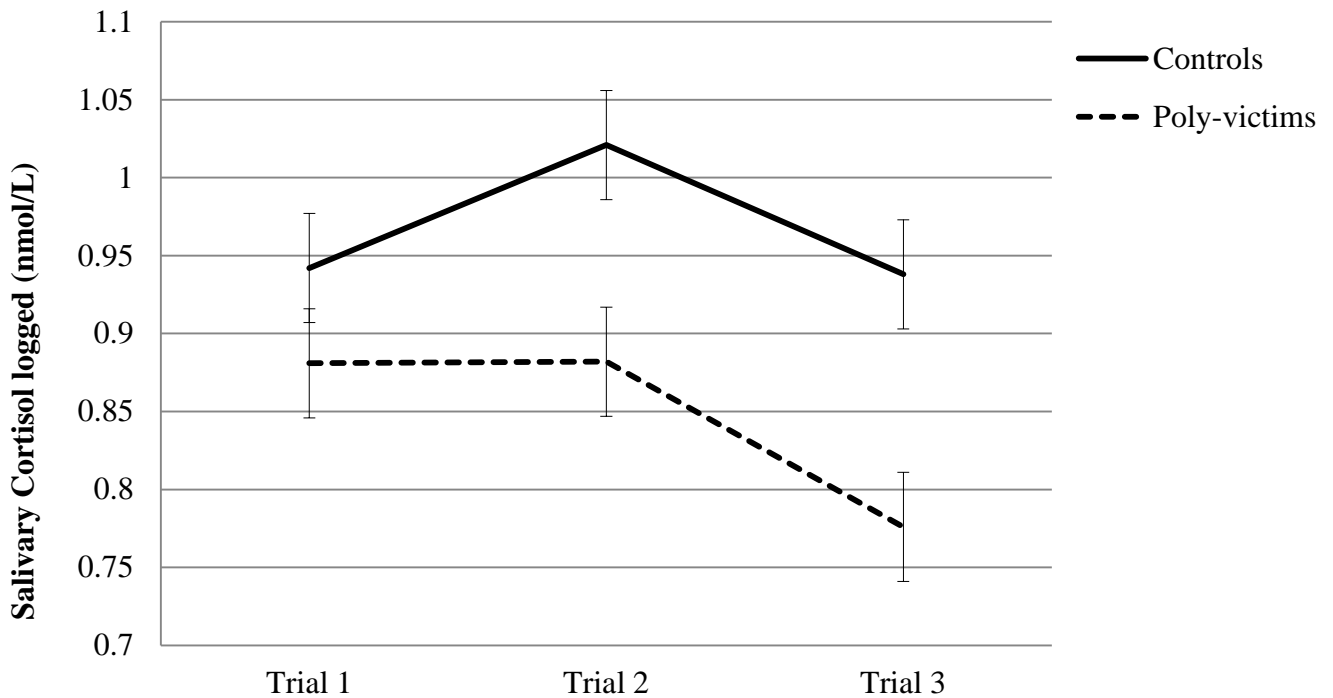
Figure 9. Mean and standard error of the mean of cortisol concentrations before and after psychosocial stress induction in women with a history of poly-victimization (n = 42) and controls (n = 45). Values are from models controlling for childhood socioeconomic status and birth control use.

Supplementary analysis tested whether CIV was associated with cortisol reactivity and recovery, adjusting for childhood SES and BCP use. Linear regressions showed that poly-victims had significantly blunted cortisol reactivity compared to controls (B = -.11, p = .02), calculated as the change between trial 1 and trial 2 cortisol, controlling for cortisol level at trial 1. Poly-victims did not differ from controls on cortisol recovery, calculated as the change between trial 1 and trial 3 cortisol, controlling for cortisol at trial 1 and trial 2 (B = -.05, p = .11). See Table 20 for a summary of the linear regression models.

Table 20. Unstandardized coefficients from linear regression models examining associations between CIV and cortisol reactivity and recovery.

	Cortisol Reactivity [a]				Cortisol Recovery [b]		
	B	*SE*	*p*		B	*SE*	*p*
Childhood SES	-0.05	0.03	.11		0.04	0.02	.09
BCP use	0.07	0.05	.17		0.07	0.03	.04
CIV	-0.11	0.05	.02		-0.05	0.03	.11

[a] Change score between trial 1 and trial 2 cortisol, controlling for trial 1 cortisol

[b] Change score between trial 1 and trial 3 Cortisol, controlling for trial 1 and trial 2 cortisol

Note: CIV = childhood interpersonal violence (0 = controls, 1 = poly-victims); SES = socioeconomic status; BCP = birth control pill

7.6.3 Linear Regressions with AUC_G and AUC_I

Table 21 presents group means for AUC_G and AUC_I.

Table 21. Mean (standard deviation) and range of AUCG and AUCI (calculated with logged cortisol values)

	Full sample (n = 87)	Controls (n = 45)	Poly-victims (n = 42)
AUC_G	52.5 (11.4), 22.8 – 84.9	55.2 (11.4), 22.8 – 84.9	49.5 (10.7), 26.3 – 80.5
AUC_I	0.6 (9.3), -19.8 – 35.9	1.9 (10.1), -14.6 – 35.9	-0.8 (8.1), -19.8 – 15.9

Note: AUC_G = area under the curve with respect to ground; AUC_I = area under the curve with respect to increase

Univariate linear regressions showed that poly-victims had significantly lower AUC_G (B = -5.78, p = .02), and this relationship persisted after adjusting for childhood SES and BCP use (B = -6.70, p < .01). Univariate linear regressions found no significant difference in AUC_I by CIV group (B = -2.66, p = .18). Models that adjusted for childhood SES and BCP use showed that poly-victims tended to have lower AUC_I (B = -3.40, p = .09). See Table 22 for a summary of the linear regression model with covariates. In sum, poly-victims had lower total cortisol output compared to controls and tended to have less sensitive cortisol responses.

Table 22. Unstandardized coefficients from linear regression models examining associations between CIV and AUCG or AUCI, adjusting for childhood SES and BCP use.

	AUC_G			AUC_I		
	B	*SE*	*p*	B	*SE*	*p*
Childhood SES	-1.81	1.54	.24	-0.91	1.27	.48
BCP use	2.69	2.41	.27	360	1.99	.07
CIV	-6.70	2.43	.007	-3.40	2.01	.09

Note: CIV = childhood interpersonal violence (0 = controls, 1 = poly-victims); AUC_G = area under the curve with respect to ground; AUC_I = area under the curve with respect to increase; SES = socioeconomic status; BCP = birth control pill

7.7 HYPOTHESIS 5

Cortisol response will mediate the relationship between CIV and BMI and WC. See Figure 3 for a model representing the hypothesized mediation pathway. AUC_G did not significantly predict BMI (B = 0.01, p = .83) or WC (B = 0.02, p = .81). Similar nonsignificant results were found for AUC_I (ps > .40). Path b was not significant; therefore, bootstrapping methods to test mediation were unnecessary. Cortisol response did not mediate the relationship between CIV and BMI or WC.

7.8 HYPOTHESIS 6

Cortisol response will mediate the relationship between CIV and stress eating. See Figure 4 for a model representing the hypothesized mediation pathway. AUC_G did not predict total calories consumed (B = -1.44, p = .58), nor did AUC_I predict total calories (B = -3.11, p = .34). Path c and Path b were not significant; therefore, bootstrapping methods to test mediation were unnecessary. Cortisol response did not mediate the relationship between CIV and stress eating.

7.9 SECONDARY ANALYSES

We intended to examine whether current victimization was a significant predictor of the outcome variables of interest; thus suggesting a need to control for current victimization. The frequency of current victimization exposure was very low. One separate participant reported current exposure

to violence in each of the following categories: physical or sexual abuse, peer bullying, community violence, and witnessing violence. Three of the four participants with current violence exposure were not statistically different from the group means on BMI, total calorie intake, and cortisol AUC_G or AUC_I (z-score < 1.96). The one participant with current peer victimization had a BMI z-score of 2.07. The low frequency of current violence exposure preempted its use as an independent variable.

We explored whether dietary restraint moderated the relationship between CIV and stress eating. An interaction term between dietary restraint (subscale of the Three Factor Eating Questionnaire) and CIV was created. In models controlling for the main effects of dietary restraint and CIV, the interaction term did not significantly predict total calories consumed (B = -4.50, p = .88). The relationship between CIV and total calorie intake did not vary by level of dietary restraint.

7.10   EXPLORATORY ANALYSES

7.10.1 Moderation in CIV and Stress Eating

The results of hypothesis 2 showed that CIV did not predict stress eating. Exploratory analyses examined whether perceptions of the TSST moderated the relationship between CIV and total calories consumed. The manipulation check regarding the TSST provided three factor scores representing the participant's perception of task performance, perception of task difficulty, and perception of interpersonal evaluation. There was a significant interaction between CIV and perception of task performance on total calories consumed (B = -36.43, p = .03). For poly-

victims, a one point decrease in perception of (worse) task performance was associated with an increased consumption of 29 calories (B = -28.82, p = .02). For controls, there was no relationship between perceived task performance and calorie intake for the control group (B = 7.61, p = .50). See Figure 10 for a graph of the interaction.
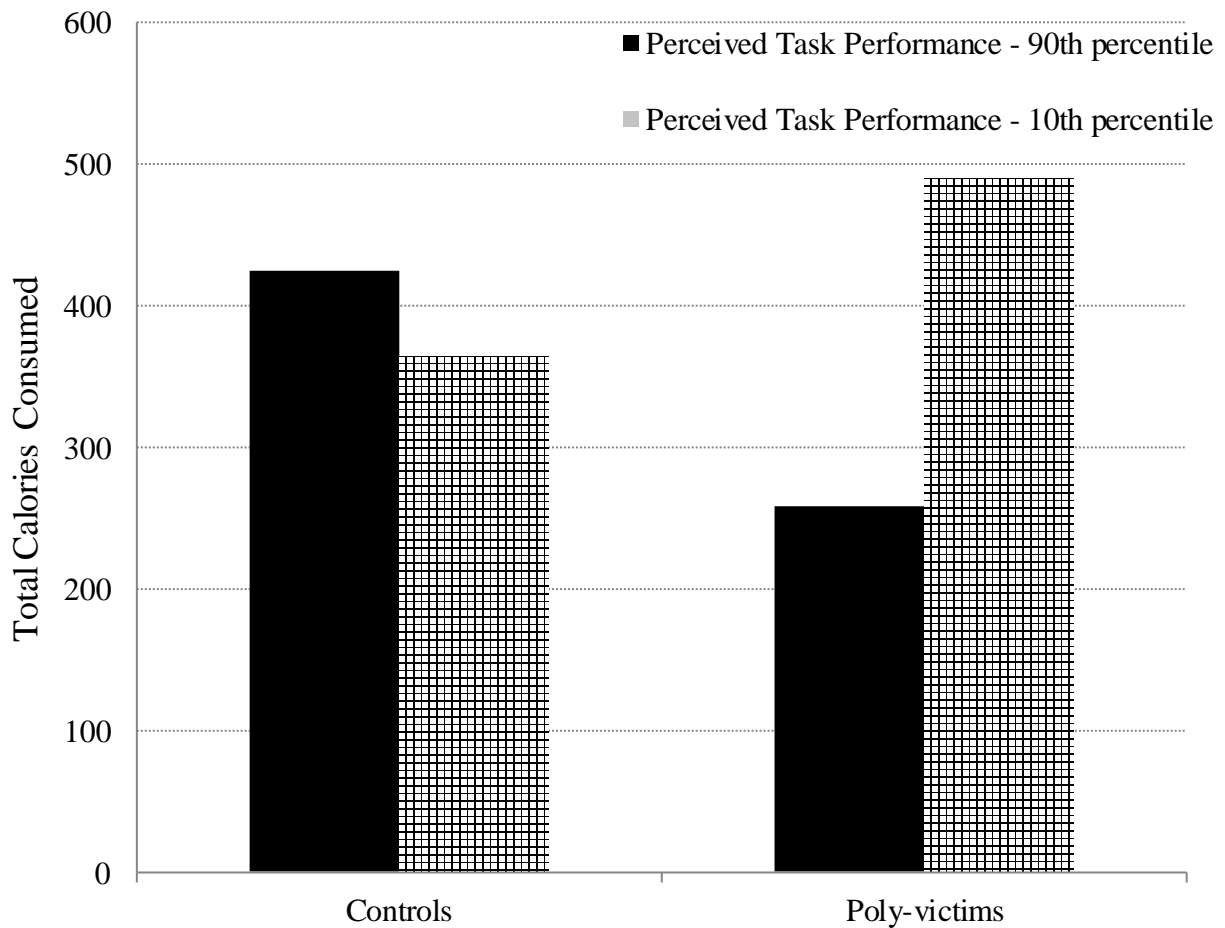
Figure 10. There was a significant interaction between CIV and perceived stress task performance on total calorie consumption after the stressor. Poly-victims ate more calories when they perceived worse task performance.

Perception of task performance continued to moderate the relationship between CIV and total calories consumed after adjusting for childhood SES and hunger at study entry (B = -31.14, p = .05). Interactions between CIV and the two other factors (perceived task difficulty and perceived interpersonal evaluation) were nonsignificant (ps > .66).

7.10.2 Mediation between CIV and BMI/WC

Stress eating, defined as calorie consumption after the stressor, did not mediate the relationship between CIV and BMI/WC. However, the present study assessed disordered eating with additional questionnaires in the recruitment online survey. Poly-victims reported greater binge eating symptoms, measured by the Binge Eating Scale, than controls (Table 7); therefore, we examined whether binge eating symptoms mediated the relationship between CIV and BMI/WC.

Bootstrapping was used to test for the significance of the indirect effect, or the product of the coefficients of path *a* and path *b*. The indirect path of CIV to BMI through binge eating symptoms was nonsignificant (indirect effect = 0.41, SE = 0.40, 95% bias corrected CI = -0.05 – 1.63). The indirect path of CIV to WC through binge eating was also nonsignificant (indirect effect = 1.00, SE = 0.88, 95% bias corrected CI = -0.01 – 3.54). Mediation was likely to be nonsignificant because of path *b*; binge eating was positively associated with BMI/WC but not once CIV was included in the model. The indirect effect remained nonsignificant when controlling for childhood SES, family history of obesity, and alcohol use.

The previous section of exploratory analyses reported an interaction effect between CIV and perceived task performance on calorie intake after the stressor. As an extension of

hypothesis 3, bootstrapping was used to explore moderated mediation (perceived task performance functioned as a moderator in path *a*). Figure 11 portrays the model.
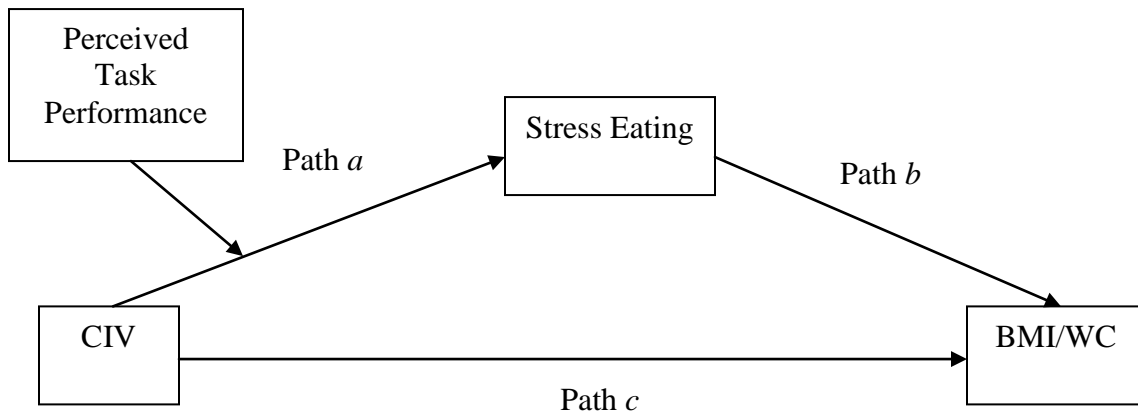
Figure 11. A model of moderated mediation--the strength of the indirect effect depends on the level of another variable. Perceived task performance functions as a moderator in path a.

The indirect path of CIV to BMI through total calorie intake was nonsignificant at various levels of perceived task performance, such as when perceived task performance was at the 10th percentile (indirect effect = 0.25, SE = 0.32, 95% bias corrected CI = -0.13 – 1.28) or at the 90th percentile (indirect effect = -0.33, SE = 0.36, 95% bias corrected CI = -1.47 – 0.14).

7.10.3 Mediation between CIV and Cortisol Response

We examined whether PTSD symptoms, measured by the Impact of Events Scale, mediated the relationship between CIV and blunted cortisol response, measured via AUC_G. Bootstrapping was used to test for the significance of the indirect effect. The indirect path of CIV to cortisol response through PTSD symptoms was nonsignificant (indirect effect = -1.30, SE = 1.21, 95% bias corrected CI = -4.06 – 0.64). Results remained nonsignificant when controlling for childhood SES and BCP use (indirect effect = -1.50, SE = 1.26, 95% bias corrected CI = -4.41 – 0.64).

8.0   DISCUSSION

The present study examined retrospective reports of childhood interpersonal violence (CIV) in a sample of undergraduate women. One aim of the study was to test for an association between CIV, in the form of poly-victimization, and overweight/central adiposity. A second aim of the study was to investigate the relationship between CIV and stress eating, as well as CIV and cortisol response to stress. Finally, if significant associations were found, then stress eating and cortisol reactivity were tested as mediators between CIV and BMI/WC. The present study had the unique capability to test behavioral and physiological pathways linking poly-victimization to overweight/central adiposity.

8.1   POLY-VICTIMIZATION

Examining the construct of poly-victimization is a novel approach within the field of CIV and health outcomes. The CIV literature suggests that poly-victimization is the norm rather than the exception (Dong, Anda, et al., 2004; Finkelhor et al., 2007b). Frequency rates from the recruitment survey showed that about 45% of all individuals reporting any CIV were poly-victims. Although this rate is lower than that reported by Dong et al. (87% of victims were poly-victims) and Finklehor et al. (69% of victims were poly-victims), it should be noted that the present study used stricter thresholds to identify participants with CIV. A study using stricter

thresholds, such as cut-off scores in the CTQ, found that 38% of victims were poly-victims in a sample of 967 men and women (Scher, Forde, McQuaid, & Stein, 2004).

In the present study, women reporting poly-victimization endorsed worse mental health. Poly-victims had greater depression, PTSD symptoms, hostility, negative affect, perceived stress, and binge eating symptoms compared to women reporting no CIV. This is consistent with recent literature suggesting that adolescent poly-victims had significantly more anxiety, depression, anger, life stress, suicide risk, and risky behaviors compared to adolescents with a single violence exposure or no violence exposure (Finkelhor, Ormrod, & Turner, 2007a; Nurius, Russell, Herting, Hooven, & Thompson, 2009). It appears that poly-victims in the present sample may not be qualitatively different from poly-victims in other samples, at least with regard to mental health outcomes.

8.2   CHILDHOOD INTERPERSONAL VIOLENCE AND OVERWEIGHT / CENTRAL ADIPOSITY

As predicted, results showed that women reporting poly-victimization had greater BMI than women reporting no victimization. On average, poly-victims had BMIs that were 2.6 points higher, which is equivalent to approximately 15 pounds. Poly-victims were generally in the overweight range while controls were in the normal-weight range. The relationship between CIV and BMI remained even when controlling for potential confounding variables.

The finding that CIV is positively associated with BMI is consistent with previous literature linking physical abuse, sexual abuse, or peer bullying to BMI (Adams & Bukowski, 2008; Bentley & Widom, 2009; Midei & Matthews, 2009; Noll et al., 2007; Singh, Kogan, &

van Dyck, 2008; Sweeting et al., 2005). Our definition of CIV also includes understudied types of violence such as community violence, witnessing violence, and intimate partner violence. Many studies do not control for other variables that may confound the relationship between CIV and BMI. When our analyses adjusted for childhood SES, family history of obesity, and alcohol use, CIV continued to predict BMI.

Two other studies provided results with which to compare our data. Lovallo et al. (2012) reported a trend for significantly different BMI means in their study of lifetime adversity in young men and women. Poly-victims had a mean BMI of 25.0 and the no victimization group had a mean BMI of 23.0; these results compare closely with the means from the present study. Gustad et al. (2006) reported that the total number of early life stressors accounted for 4% of the variance in BMI in their sample of men, but the relationship was nonsignificant in women. In comparison, the present study did find a significant and positive relationship between CIV and BMI in late adolescent women. The measures used to assess CIV differed between studies and may partially account for the varying outcomes. In the studies by Lovallo et al. and Gunstad et al., only one item was used to measure each type of violence exposure, whereas the present study used measures ranging from 3 to 14 items per CIV subtype, increasing validity and reliability of violence measurement. In addition, we utilized cut-off scores that generally reflected at least moderate frequency and moderate severity of victimization experiences. It may be that as violence intensity/frequency increases, the relationship with BMI becomes stronger.

Similar results were found for the outcome of WC; women reporting poly-victimization had 4.62 centimeters (1.8 inches) higher WC than women reporting no CIV. WC means for both groups were in the healthy range (i.e., WC < 88 centimeters). When covariates were included in

the model, the relationship between CIV and WC became a trend, suggesting that the relationship was not as reliable as with BMI.

There is a pattern in the literature of a slightly weaker relationship between CIV and WC, compared to the outcome of BMI (Midei et al., 2010; Thomas et al., 2008). It is possible that behavioral factors (e.g, alcohol use) or genetic factors play a larger mediating or moderating role for central adiposity than for overall overweight. Alternatively, CIV may impart greater risk for a certain component of central adiposity, specifically visceral adipose tissue. Visceral adiposity has been identified as the particularly toxic component of WC (which includes both subcutaneous and visceral adipose tissue in its measurement) and has stronger associations with metabolic syndrome components than subcutaneous adipose tissue (Fox et al., 2007; Preis et al., 2010). Future studies should consider utilizing computed tomography scans to quantify visceral adiposity. CIV may show stronger relationships with visceral adipose tissue rather than the measure of WC.

8.3   CIV AND STRESS EATING

This was the first study to examine CIV group differences in stress eating using a laboratory paradigm. Poly-victims did not consume more calories after the stressor compared to controls However, poly-victims who perceived that they did poorly on the tasks did eat more calories compared to those victims who perceived that they did better. There was no relationship between perceived task performance and calorie consumption in women without CIV. Perceived task performance was a statistically derived factor that was composed of three items measuring the

participant's self-evaluation of effectiveness during the stressor, with an emphasis on the speech task.

Perceived task difficulty and perceived interpersonal evaluation were factors that did not moderate the relationship between CIV and stress eating. Perceived task difficulty included items asking whether the tasks were difficult or challenging and may reflect the inherent properties of the stressor and stressor validity. In general, participants perceived the task to be relatively difficult and challenging, although their ratings of task difficulty were uncorrelated to perceived task performance and perceived interpersonal evaluation. Hence, appraisal of the stressor itself appears to be set apart from self-evaluations of effectiveness or likeability. Perceived interpersonal evaluation included items addressing whether the panel liked and accepted the participant, and also whether the participant liked the panel. Perceived task performance and perceived interpersonal evaluation were moderately correlated; thus, when participants rated their performance as better, they also felt more accepted by the panel. It is possible that perceived interpersonal evaluation is secondary to perceptions of task performance. Self-evaluations of task performance may be the key component of stressor appraisal.

Early work determined that the appraisal of stress was an important component for the stress response. The same environmental demand can elicit wide variability in individuals' reactions and emotions, suggesting that an individual's subjective perception and interpretation of the stressor is critical in understanding responses to stress (Lazarus & Folkman, 1984). Part of the appraisal process is whether an individual views the situation as threatening and believes that she/he has the necessary resources to cope with the situation. In the present study, poly-victims who faced a stressor and rated their performance as poor, which may suggest that they perceived themselves as being unable to cope with the stressor, subsequently ate more calories.

Alternatively, poly-victims who faced a stressor and perceived their performance as being strong engaged in less stress eating. It should be noted that the results discussed here were exploratory and not a primary hypothesis. In order to further explore a potential moderating effect of stressor and coping appraisal, future studies of CIV and stress responses should include adequate measures to test the relationship reported in this study.

8.4   STRESS EATING AS A MEDIATOR

Stress eating did not mediate the relationship between CIV and BMI or WC. Moderated mediation (Figure 10) was also explored, which asked whether stress eating accounted for the relationship between CIV and BMI, when the indirect pathway varied by level of perceived task performance. The indirect effect was nonsignificant, most likely due to calorie consumption being unrelated to BMI. Interestingly, other studies using laboratory eating paradigms also reported a nonsignificant correlation between BMI and calories intake (Epel et al., 2001) or did not report the correlation (Mitchell & Epstein, 1996; Oliver et al., 2000; Rutters et al., 2008; Zellner et al., 2006). Our findings contrast with the results of Greenfield and Marks (2009), who found that use of food in response to stress, measured by questionnaire, accounted for the relationship between physical and psychological abuse and BMI.

One concern of the present study is the external validity of the stress eating paradigm. The mean calorie intake during ad libitum access to food was 389 calories, suggesting that on average, participants were not inhibited from eating. Self-reported hunger at study entry was also correlated with total calories consumed. However, total calories consumed was not significantly correlated with BMI or questionnaires assessing dysfunctional eating behaviors, such as binge

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eating symptoms, dietary restraint, emotional eating, or uncontrolled eating (ps > .31). Eating after stress in the laboratory paradigm may not generalize to stress eating in real world environments. There are limited data on the ecological validity of laboratory eating paradigms. For example, no studies use the same participants to compare stress eating behaviors measured in the laboratory and measured in a naturalistic setting. Future studies would benefit from qualitative and quantitative measures of participant's interpretation of the stress eating paradigm and comparisons to everyday experiences.

Exploratory analyses tested whether another measure of maladaptive eating behavior, specifically binge eating symptoms, accounted for the relationship between CIV and BMI/WC. Poly-victims reported more binge eating symptoms, and binge eating symptoms were positively associated with BMI and WC. However, results showed that the variance in BMI/WC explained by binge eating was largely accounted for by a history of CIV. This was the second study to test whether dysfunctional eating behaviors explained the relationship between CIV and BMI. Rhodes and collages (Rohde et al., 2008) reported that clinically significantly binge eating partially mediated the relationship between physical or sexual abuse and BMI, although there was no test of significance for mediation (i.e., bootstrapping or Sobel test). In sum, eating behaviors measured objectively in the laboratory or subjectively measured in a questionnaire did not explain the relationship between CIV and overweight/central adiposity.

8.5   CIV AND CORTISOL RESPONSE

It was hypothesized that CIV would be related to exaggerated cortisol response to stress. The results did not support the hypothesis; in fact, the findings showed a reverse association. Women

reporting poly-victimization had lower overall cortisol response to stress compared to women with no CIV. These findings were supplemented by analyses predicting AUC_G, which reflects the intensity of the cortisol response and is highly correlated to mean cortisol output. Poly-victims had significantly lower AUC_G than controls after adjusting for childhood SES and BCP use. The effect of lower overall cortisol response does not seem to be driven by lower baseline levels, as cortisol at trial 1 did not significantly differ between controls and poly-victims. Furthermore, the finding does not seem to be driven by diminished psychological responses to the stressor, as there were no CIV group differences on impressions of the TSST or on changes in negative or positive affect across the stress task. Additional analyses examined whether CIV was associated with cortisol reactivity or recovery. After adjusting for childhood SES and BCP use, poly-victims had lower cortisol reactivity than controls but similar patterns of cortisol recovery.

One can compare the current results of the control women to "normative" responses to the TSST. In developing the TSST, Kirschbaum and colleagues (1992) ran several studies to understand the normative range of cortisol response. Over three studies with a total of 71 female participants, females showed a mean cortisol increase of 4.1 to 6.2 nmol/L to the TSST, which equaled to a 1.5 to 2.5-fold increase. Additionally, females generally peaked at 9.8 to 10.7 nmol/L. The controls in the present study showed a response reaching closer to normal with 2.5 nmol/L, and there were wide variability in the data. Based on the suggested normative cortisol response to stress, it appears that poly-victims in the present sample show a blunted cortisol response.

Several recent studies show a similar pattern of blunted cortisol reactivity in individuals with a history of CIV. Elzinga et al. (2008) reported that young adult poly-victims had blunted

cortisol reactivity to the TSST compared to individuals reporting zero or one adverse event. The finding appeared to be driven by men, and no relationship was found in women. A second recent study by Lovallo and colleagues (2012) found a dose-dependent cortisol response to the TSST, such that cortisol response was progressively lower with increasing lifetime adverse events (most occurring before the age of 18). Another study of adults reported that the total CTQ score, which included measures of emotional and physical neglect, predicted lower overall cortisol response to the TSST (Carpenter et al., 2007). Finally, MacMillan et al., (2009) completed a study of female adolescents at about 14 years of age. Although there was no direct test of poly-victimization and cortisol response, a majority of participants in the childhood maltreatment group had more than one exposure to CIV (including physical and emotional neglect). The authors reported that maltreated children had flattened cortisol reactivity and recovery slopes after the TSST compared to controls.

There may be several reasons why the present study found that poly-victims showed hypo-activity rather than the hypothesized hyper-activity. The relationship between CIV and cortisol hypo-activity may be more common in healthy subjects who were not recruited for studies of depression (Elzinga et al., 2010; Klaassens et al., 2009). Indeed, the present study did not recruit participants based on co-occurring diagnoses and included individuals reporting subclinical or minimal depressive symptoms. Clinically significant PTSD diagnosis has generally been associated with cortisol hypo-activity (Miller et al., 2007; Yehuda, 2006). The present study was able to test whether PTSD symptoms mediated the relationship between CIV and blunted cortisol output. However, bootstrapping mediation analysis showed that the indirect effect was nonsignificant, suggesting that subclinical PTSD did not explain the relationship between CIV and cortisol hypo-activity found in the current study. Duration or timing of CIV

may be another factor related to differential HPA axis responses to stress. For example, the number of early life traumatic events occurring before puberty showed an inverse relationship with peak plasma cortisol response to the TSST, but the number of traumas after puberty showed no significant relationship with cortisol (Heim et al., 2002). The women in the present study may have experienced victimization earlier in the lifespan.

Previous literature has suggested ways in which CIV leads to blunted cortisol response to stress. Poly-victimization, possibly indicating a history of chronic victimization, may cause persistently elevated HPA axis activity which then develops into dampened cortisol responses to stress (De Bellis, 2002; Tarullo & Gunnar, 2006; Trickett, Noll, Susman, Shenk, & Putnam, 2010a). The dampened response to stress may be a result of down-regulation of the HPA axis. It is unclear at what point in the lifespan HPA axis dampening may occur, although this question has been the focus of several recent studies. For example, one research group followed a sample of sexually abused and non-abused girls from age 6 through 32. They reported that the abused participants showed basal hyper-cortisol in childhood and then hypo-cortisol in adulthood, in addition to blunted cortisol response to stress in late adolescence (Shenk, Noll, Putnam, & Trickett, 2010; Trickett, Noll, Susman, Shenk, & Putnam, 2010b). The present sample may provide additional evidence that an attenuated cortisol response is measurable by late adolescence.

Another somewhat related theory suggests that early life adversity initially causes HPA hyper-activation, which then interacts with gene expression to reduce hippocampal volume, thereby causing HPA axis hypo-activation (Charney & Manji, 2004; Gilbertson et al., 2002). Recent research supports this theory. Teichera and colleagues (2012) found that histories of maltreatment were associated with reduced volume in certain areas of the hippocampus,

specifically areas that are the most sensitive to glucocorticoids. These same areas in the hippocampus are associated with outputs that regulate HPA responses following psychological stressors (Herman, Dolgas, & Carlson, 1998). Interestingly, the authors tested whether major depressive disorder or PTSD mediated the relationship between childhood maltreatment and hippocampal volume. Mediation was not supported, and the authors speculated that reduced hippocampal volume was a precursor for PTSD rather than a consequence of the disorder. Reduction of hippocampal volume may account for hypo-secretion of cortisol and confer vulnerability for PTSD and/or depression over time.

8.6   CORTISOL RESPONSE AS A MEDIATOR

8.6.1   CIV → Cortisol Response → BMI/WC

Cortisol response to stress did not mediate the positive association between CIV and BMI or WC. Although many previous studies speculate that HPA axis dysregulation is the mechanism linking CIV to overweight/central adiposity, this was the first study to empirically test the hypothesis. Cortisol response to stress was unrelated to BMI or WC. A recent study also failed to show an association between cortisol reactivity and BMI (Hamer et al., 2010). However, the results are inconsistent with two previous reports that cortisol reactivity is positively related to overweight/central adiposity in children and women (Dockray et al., 2009; Epel et al., 2000). Overall, results from studies appear to be conflicting, suggesting that HPA axis activity may be a weak physiological pathway leading to overweight and central adiposity.

It is possible that the developmental pattern of HPA axis dysregulation after early life adversity (high output in childhood, low output by late adolescence) determines a critical window of risk between cortisol and BMI/WC. For instance, cortisol hyper-activity in childhood and early adolescence may be a key mechanism driving increases in adiposity during that specific period. In fact, the previously mentioned study by Dockray et al. assessed children 8-13 years old and found a positive association between cortisol reactivity to the TSST and BMI. Future research should consider how HPA axis dysregulation co-varies with increasing overweight/central adiposity over the life course.

8.6.2   CIV → Cortisol Response → Stress Eating

Cortisol response did not mediate the relationship between CIV and stress eating because cortisol response was not correlated to total calorie intake. The present study's results differ from the one previous study that examined the relationship between HPA axis activity and calorie consumption after a laboratory stressor. Epel and colleagues (2001) showed that cortisol high-responders, defined as participants with AUC_G above the median split, ate more calories after the TSST. The authors noted that there was actually a weak correlation between cortisol AUC_G measured continuously and calorie consumption. A second study using ecological momentary assessment showed that daily hassles were positively associated with snack intake over 14 days, but only for cortisol high-responders, defined as those with any cortisol increase from baseline in response to the TSST (Newman et al., 2007). It is worth nothing that the "high-reactors" in the Epel et al. and Newman et al. studies appear to actually have normal responses to the TSST (a 1.5 to 2.5-fold increase), based on previously established normative response patterns (Kirschbaum et al., 1992). The results could be interpreted differently; participants with blunted

cortisol reactivity ate less food after laboratory stress or daily hassles. In sum, the inconsistency of results between the previous two studies and the present study suggests that more research is needed to further understand the association between HPA axis dysregulation and calorie consumption.

8.7   IMPLICATIONS

8.7.1   CIV and Overweight/Central Adiposity

There are several implications related to the findings of the present study. Women with histories poly-victimization have higher BMI. Overweight and obesity is the second leading cause of preventable death and associated with morbidity and mortality, in addition to costing the United States $147 billion dollars in medical care (Finkelstein, Trogdon, Cohen, & Dietz, 2009; National Heart Lung and Blood Institute, 1998). The present study, among others (see review by Midei & Matthews, 2011), suggests that childhood violence is a significant risk factor for overweight, and that this relationship is evident in late adolescence. CIV is a threat to the physical well being of exposed individuals.

Several avenues exist for prevention and intervention. Primary intervention strategies are aimed at the general population and at preventing violence before it occurs. General strategies include public service announcements on positive parenting practices or student education such as anti-bullying programs. Secondary prevention activities also aim for prevention and are targeted at populations that are at high-risk for violence, such as low-income neighborhoods or substance-abusing parents. Activities may include parent education programs located in high

schools for teen parents or family resource centers in neighborhoods with high levels of poverty. Results from the present study suggest that community and witnessing violence were "common denominators" for poly-victims; secondary prevention that is effectively targeted at communities with high rates of violence may prevent additional exposures to violence. Finally, tertiary prevention focuses on families and children for whom victimization has already occurred, such as mental health services for children and parent mentor programs. Tertiary prevention seeks to reduce the negative consequences of CIV. For example, empirically supported treatments are available for children and adolescents who experienced victimization, including Trauma-focused Cognitive Behavioral Therapy and School-based Group Cognitive Behavioral Treatment. These treatments aim to reduce psychological distress and increase healthy coping strategies; it is possible that these targeted intervention programs for victimized children could also prevent unhealthy weight gain. No published studies have examined this possibility; however, Kessler and colleagues (2008) reported that early intervention with abused/neglected children in private foster care reduced prevalence of cardiometabolic disorders in adulthood, such as type II diabetes, hypertension, and heart disease. In summary, it is recommended that public policy and public health campaigns increase efforts within primary, secondary, and tertiary prevention of CIV.

This is the first study to examine the relationship between poly-victimization and WC. Even though results showed a weaker relationship with WC compared to BMI, it is worth discussing the potential implications uniquely associated with central adiposity. Central adiposity may be an important pathway by which early life adversity is associated cardiovascular disease. CIV has been linked to ischemic heart disease, hypertension, and diabetes in adults (Dong, Giles, et al., 2004; Rich-Edwards et al., 2010; Riley, Wright, Jun, Hibert, & Rich-Edwards, 2010). It is

possible that central adiposity may be an early marker of cardiovascular risk, such that we found a trend for the relationship between CIV and WC in late adolescent women.

8.7.2 CIV and Stress Eating

Poly-victims with negative self-evaluations after stress showed increased stress eating, while poly-victims with positive self-evaluations after stress showed decreased stress eating. One implication of these findings is that women with histories of CIV may be more vulnerable to negative self-talk, subsequently leading to negative health behaviors. It is possible that poly-victims who believe themselves to be less competent at coping with stress engage in unhealthy coping strategies (such as stress eating), thus reinforcing thoughts of inadequacy. Women with CIV may benefit from interventions aimed at improving self-efficacy or countering negative self-talk. Indeed, some common components among psychosocial interventions for victimized children are skills related to emotion regulation and cognitive misappraisals (Silverman et al., 2008). Interventions that aim to decrease negative self-evaluations and increase positive self-evaluations may improve dysfunctional eating behaviors.

8.7.3 CIV and Cortisol Stress Response

Although opposite of expectations, CIV was associated with cortisol hypo-activity, which may have several negative health implications. A recent review by Miller and colleagues (2011) suggests that early life adversity affects later health through proinflammatory tendencies, such as exaggerated cytokine response and decreased sensitivity to inhibitory signals. Cortisol is one such inhibitory signal. Cortisol binds to glucocorticoid receptors on macrophages and other

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immune cells and essentially interrupts the "turning-on" of proinflammatory genes. Thus, HPA axis hypo-activity creates an environment of immune activation and inflammation (Raison & Miller, 2003; Sapolsky, Romero, & Munck, 2000). Miller et al. also suggested that childhood stress promotes excessive threat vigilance, mistrust of others, poor social relationships, impaired self-regulation, and unhealthy lifestyle choices. HPA axis dysregulation may even be a pathway to these negative behavioral, emotional, and social outcomes. Recent evidence showed that HPA axis hypo-activity mediated the relationship between CIV and social and behavioral problems in 12-year-old children (Ouellet-Morin et al., 2011). In sum, HPA axis hypo-activity may contribute to negative physical health outcomes by decreasing inhibition to proinflammatory tendencies and by increasing social and behavioral problems.

8.7.4   Mechanisms linking CIV to BMI/WC

Findings from the present study aimed to clarify the mechanisms contributing to overweight. However, none of the proposed mediators were supported by the data including stress eating, binge eating, or cortisol reactivity. The association between poly-victimization and BMI/WC is unlikely due to stress eating or HPA axis dysregulation, at least as measured in this study at one time point. Tests of mediation were largely nonsignificant because stress eating and HPA activity were not related to BMI/WC.

With regard to the lack of association between stress eating and BMI/WC, there are two possible implications based on the results of this study. First, a laboratory-based assessment of stress eating (measured in one sitting with restricted food options) may be unable to capture dysfunctional eating behaviors following stress, thus having low ecological validity. Stress eating should be measured differently in order to clarify its role, or lack thereof, in the pathway

to weight gain. For example, poly-victims may choose higher calorie "comfort foods" during periods of stress eating, when they are able to choose from a broad array of food. Alternatively, the number of stress eating episodes in a given time period may predict BMI/WC rather than calories consumed at each sitting. A second implication of the present study's lack of association may be that stress eating is independent from BMI/WC, at least in a sample of undergraduate women with and without histories of CIV. Nguyen-Rodriguez and colleagues (2008) conducted a study of 500 minority adolescents and showed that perceived stress was associated with emotional eating, but there was no relationship between emotional eating and BMI. The authors reported that emotional eating was a concern for normal weight individuals, as well as overweight and obese adolescents. Poly-victims may engage in other eating behaviors that link histories of victimization with BMI/WC, such as eating in the absence of hunger or eating beyond fullness. Future studies should examine other dysfunctional eating behaviors as possible mediators.

There are several implications with regard to the lack of association between HPA activity and BMI/WC. It is possible that cortisol stress reactivity is not a primary physiological mechanism behind increased overweight/central adiposity in late adolescent women. There may be other aspects of HPA axis activity that is associated with BMI/WC, such as diurnal slope over the course of the day or cortisol uptake within visceral adipose tissue. HPA axis dysregulation may also be a consequence of overweight/central adiposity that develops over time. Another implication is that other physiological systems may be better targets as possible mediators between CIV and BMI/WC, for instance, inflammatory processes or sympathetic nervous system activity. Future theoretical and empirical research should consider alternative mechanisms linking CIV to overweight/central adiposity.

8.8   LIMITATIONS

The current study has several limitations, one being the measurement of CIV. There are several challenges to studying CIV. First, one has to determine the types of victimization to be included. Victimization categories can be distinguished by perpetrator, such as violence from caregivers, peers, intimate partners, and the community. Alternatively, victimization categories can be distinguished by type of violence without regard to perpetrator, such as physical assault, sexual assault, and witnessing violence. The childhood victimization literature tends to blend these approaches, suggesting that there is not an agreed-upon definition of poly-victimization. The present study took a similar approach of blending methods; perpetrator was the main distinguishing factor (e.g., violence from caregivers, peers, intimate partners, and the community) but physical and sexual abuse (both from caregivers) were considered as separate categories.

The second challenge to studying CIV is to find measures that adequately assess each type of victimization. A combination of measures was used in the present study, some with strong psychometric properties (e.g., CTQ) and some without validity and reliability data (e.g., peer violence). Psychometric data were obtained through the online survey for the peer violence and intimate partner violence scales and generally showed low reliability. This is not unexpected as each questionnaire had three items measuring very different violence exposures from the same source. Unfortunately, there is a lack of sound questionnaires used to assess poly-victimization or even certain types of victimization occurring in childhood and adolescence, such as dating violence. It is clear that additional assessment methods are needed within the field of childhood victimization.

A third challenge to studying CIV is having continuity and similarity across measures. The present study aimed to identify poly-victims by creating similar cut-offs across questionnaires that reflected at least moderate violence with moderate frequency. These criteria were met in part by established thresholds of the original scale (e.g., CTQ, Bernstein et al., 2003), or by determining similar thresholds on the un-validated questionnaires (a response of "more frequently" for peer or intimate partner violence). Thresholds for the community violence and witnessing violence scales were based on a theoretical approach to match other violence subtypes, but also with a quantitative approach because of the high frequency of reported exposure. For example, the threshold for the witnessing violence scale was increased from one item to three items that were endorsed "a few times" or "lots of times." In the full survey sample from which study participants were recruited (n = 876), 24% reported victimization from the community and 33% reported witnessing violence. These rates are similar to rates reported in other studies on the prevalence of community violence (Stein, Jaycox, Kataoka, Rhodes, & Vestal, 2003) and witnessing violence (Zinzow et al., 2009). Interestingly, in individuals who reported two or more exposures to violence (n = 189), the prevalence of community and witnessing violence increased to 81% and 85% respectively, which is similar to the prevalence in the laboratory study's sample of poly-victims (Table 8). The high prevalence of community and witnessing violence in the lab sample may not be due to lenient cut-off scores for each scale, but rather that risky and dangerous environments co-vary with children's vulnerability to victimization.

Retrospective assessments of CIV may be another limitation of the present study. Retrospective reports may be affected by distorted memories or mood at time of recall (Briere, 1992). However, recent views are that concerns about reliability and validity are often

overemphasized (Miller et al., 2011). Previous research suggests that when early life adverse events (specifically maltreatment) are reported retrospectively, the events are likely to have occurred (Brewin, Andrews, & Gotlib, 1993; Hardt & Rutter, 2004). Details of timing and duration may be more vulnerable to inaccurate memory recall. Furthermore, the main concern about validity is that even with well-documented reports of abuse/neglect, about a third of victims do not report its occurrence when asked about the abuse/neglect in adulthood (Hardt & Rutter, 2004). CIV has more potential to be underreported than over-reported. The present study measured various forms of victimization other than maltreatment, and it is possible that validity and reliability of retrospective reports may differ across violence subtypes. However, no research has examined this concern.

The study was cross-sectional and unable to make claims regarding causality of the observed relationships. The data are consistent with longitudinal studies that link CIV to steeper trajectories of weight gain by young adulthood (Noll et al., 2007). However, it is also possible that heavier children are more likely to be victimized, whether because it makes them targets for peer bullying (Adams & Bukowski, 2008), or because higher rates of obesity are found in neighborhoods with increased likelihood of community violence (Singh et al., 2008). The present study aimed to identify mediators linking CIV to overweight/central adiposity as a way to improve understanding of the factors that increase risk for overweight/central adiposity. However, cross-sectional study design makes it impossible to test true mechanisms in the relationship. Thus, the current study was essentially designed to identify factors that co-varied within individuals, at one point in time, as a way of initiating a deeper exploration into the behavioral and physiological pathways linking CIV to physical health.

As only undergraduate women participated in this study, the results may not generalize to men or to participants with varying age, SES, and ethnicity. Although less consistent and generally weaker, several studies that included men have shown that CIV is linked to BMI, cortisol response to stress, and eating behaviors (Greenfield & Marks, 2009; Lovallo et al., 2012). The women in the present study were able to attend college, which may reflect a level of resilience or economic advantage that could be protective in the relationship between CIV and health outcomes. However, it is worth nothing that the women reporting poly-victimization in the present sample reporter greater depressive symptoms, PTSD symptoms, hostility, negative affect, and perceived stress compared to controls, which calls the poly-victims' resilience into question. Significant relationships were found between CIV, BMI, cortisol response, and stress eating in the present study; one might expect stronger relationships in samples of low SES participants.

8.9   FUTURE DIRECTIONS

Continuing to work on the mechanisms connecting CIV to BMI and WC will improve understanding of how CIV affects morbidity and mortality. Chronic and stress-induced inflammation has been linked to CIV and BMI and warrants further study as a plausible mechanism (Bensona et al., 2009; Miller et al., 2011). Interview-based assessments may improve CIV construct validity and reliability and allow for questions on timing, duration, perpetrator, and severity of victimization experiences. Furthermore, a future study may benefit from using continuous scores of violence exposure to increase power and potentially clarify the relationship

between CIV and health outcomes. For example, the relationship between CIV and BMI may be linear or it may be curvilinear.

The present study found that a laboratory-based assessment of stress eating did not mediate the relationship between CIV and BMI/WC. A future study could explore eating behaviors in the field by using ecological momentary assessment with the intent to improve ecological validity. For example, Newman and colleagues (2007) examined daily hassles and snack intake by having participants complete questionnaires and food diaries at the end of each day for 14 days. Ecological momentary assessment would provide additional data; participants could be alerted every hour to record their daily experience and food intake. Such a study may be able to capture patterns, possibly stress-related, that influence eating behaviors in women reporting poly-victimization.

Another direction for future research is the timing of CIV and weight gain. There has been evidence to suggest that overweight and obese children are vulnerable to violence from peers (Adams & Bukowski, 2008). A longitudinal assessment of children and adolescents would help to identify the direction of the relationship. A longitudinal assessment may also clarify the theory of changing HPA dysregulation during childhood and adolescent development. Future research would benefit from adding various health-related measures to the longitudinal assessments, such as body mass index, visceral adiposity, immune factors, and insulin sensitivity. Health behavior measurements, such as diet, physical activity, substance use, and social ties, would further clarify the relationship between CIV and negative health outcomes. Understanding the trajectory of mental and physical health after CIV would enable interventions to be tailored to development stage and critical periods of health risk.

8.10   SUMMARY

Previous literature has suggested that certain types of childhood interpersonal violence (CIV), such as physical and sexual abuse, were associated with overweight status. However, few studies explored poly-victimization, or the experience of multiple forms of CIV, even though victimization experiences are likely to co-occur within individuals. Additionally, there was a dearth of literature examining mechanisms to explain relationships with BMI. The current study aimed to determine whether poly-victimization was associated with BMI and WC in a sample of undergraduate women. The current study sought to test the relationship between poly-victimization and cortisol response to stress, as well as eating in response to stress. It was hypothesized that cortisol response and stress eating may be mediators accounting for a positive relationship between poly-victimization and overweight/central adiposity.

Women reporting poly-victimization had higher BMI after adjusting for childhood SES, family history of obesity, and alcohol use. The relationship was weaker with the outcome of WC. Poly-victims did not eat more following stress, although poly-victims who perceived their performance as worse on the stress task consumed more calories. Unexpectedly, poly-victims showed blunted cortisol response to the stress task. Formal tests of mediation showed that neither cortisol response to stress nor stress eating were significant mediators in the relationship between CIV and BMI.

Overall, the results are consistent with earlier reports that early life victimization is related to increased BMI and WC (Midei & Matthews, 2011) and attenuated cortisol responses to stress (Lovallo et al., 2012). This was the first study to use a laboratory paradigm to measure stress eating in poly-victims. Future research should continue to explore behavioral, emotional,

and physiological pathways linking childhood interpersonal violence to later health outcomes and the moderators of these relationships.

BIBLIOGRAPHY

Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior, 91*, 449-458.

Adams, R. E., & Bukowski, W. M. (2008). Peer victimization as a predictor of depression and body mass index in obese and non-obese adolescents. *Journal of Child Psychology and Psychiatry, 49*, 858-866.

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: The challenge of the gradient. *American Psychologist, 49*, 15–24.

Allison, K. C., Grilo, C. M., Masheb, R. M., & Stunkard, A. J. (2007). High self-reported rates of neglect and emotional abuse, by persons with binge eating disorder and night eating syndrome. [doi: DOI: 10.1016/j.brat.2007.05.007]. *Behaviour Research and Therapy, 45*(12), 2874-2883.

Alvarez, J., Pavao, J., Baumrind, N., & Kimerling, R. (2007). The relationships between child abuse and adult obesity among California women. *American Journal of Preventive Medicine, 33*, 28-33.

Andrew, R., Phillips, D. I. W., & Walker, B. R. (1998). Obesity and gender influence cortisol secretion and metabolism in man. *Journal of Clinical Endocrinology and Metabolism, 83*, 1806 -1809.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook-Medley Hostility Scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46-57.

Bensona, S., Arckb, P. C., Tanc, S., Mannc, K., Hahnd, S., Janssene, O. E., . . . Elsenbruch, S. (2009). Effects of obesity on neuroendocrine, cardiovascular, and immune cell responses to acute psychosocial stress in premenopausal women. *Psychoneuroendocrinology, 34*, 181–189.

Bentley, T., & Widom, C. S. (2009). A 30-year follow-up of the effects of child abuse and neglect on obesity in adulthood. *Obesity, 17*, 1900-1905.

Bernstein, D. P., & Fink, L. (1998). *Childhood Trauma Questionnaire*. San Antonio, TX: Psychological Corp.

Bernstein, D. P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., . . . Ruggiero, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry, 151*, 1132-1136.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., . . . Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect, 27*, 169-190.

Björntorp, P., & Rosmond, R. (2000). Obesity and cortisol. *Nutrition, 16*, 924–936.

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Block, J. P., He, Y., Zaslavsky, A. M., Ding, L., & Ayanian, J. Z. (2009). Psychosocial stress and change in weight among US adults. *American Journal of Epidemiology, 170*, 181-192.

Brewin, C. R., Andrews, B., & Gotlib, I. H. (1993). Psychopathology and early experience: a reappraisal of retrospective reports. *Psychological Bulletin, 113*, 82-98.

Briere, J. (1992). Methodological issues in the study of sexual abuse effects. *Journal of Consulting and Clinical Psychology, 60*, 196-203.

Brydon, L., Wright, C. E., O'Donnell, K., Zachary, I., Wardle, J., & Steptoe, A. (2008). Stress-induced cytokine responses and central adiposity in young women. *International Journal of Obesity, 32*, 443–450.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., . . . Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62*, 1080–1087.

Charney, D. S., & Manji, H. K. (2004). Life stress, genes, and depression: multiple pathways lead to increased risk and new opportunities for intervention. *Science's STKE, 225*. doi: DOI: 10.1126/stke.2252004re5

Chartier, M. J., Walker, J. R., & Naimark, B. (2009). Health risk behaviors and mental health problems as mediators of the relationship between childhood abuse and adult health. *American Journal of Public Health, 99*, 847–854.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385-396.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic virtue scales for the MMPI. *Journal of Applied Psychology, 38*, 414-418.

Cosley, B., McCoy, S., Ehle, M., Saslow, L., & Epel, E. (2007). Does stress make you fat? 'Good' stress, 'bad' stress and comfort food eating. Memphis, TN: Society for Personality and Social Psychology.

Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology, 43*, 245–265.

Dallman, M. F., Pecoraro, N. C., & la Fleur, S. E. (2005). Chronic stress and comfort foods: Self-medication and abdominal obesity. [doi: DOI: 10.1016/j.bbi.2004.11.004]. *Brain, Behavior, and Immunity, 19*(4), 275-280.

Daniel, M., Moore, S., Decker, S., Belton, L., DeVellis, B., Doolen, A., & Campbell, M. K. (2006). Associations among education, cortisol rhythm, and BMI in blue-collar women. *Obesity, 14*, 327–335.

De Bellis, M. D. (2002). Developmental traumatology: a contributory mechanism for alcohol and substance use disorders. *Psychoneuroendocrinology, 27*, 155–170.

de Lauzon, B., Romon, M., Deschamps, V., Lafay, L., Borys, J.-M., Karlsson, J., . . . Charles, M. A. (2004). The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. *The Journal of Nutrition, 134*, 2372-2380.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355-391.

Dockray, S., Susman, E. J., & Dorn, L. D. (2009). Depression, cortisol reactivity, and obesity in childhood and adolescence. *Journal of Adolescent Health, 45*, 344-350.

Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., . . . Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect, 28*, 771–784.

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P., & Anda, R. F. (2004). Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. *Circulation, 110*, 1761-1766.

Duclos, M., Gatta, B., Corcuff, J. B., Rashedi, M., Pehourcq, F., & Roger, P. (2001). Fat distribution in obese women is associated with subtle alterations of the hypothalamic-pituitary-adrenal axis activity and sensitivity to glucocorticoids. *Clinical Endocrinology, 55*, 447–454.

Ebata, A. T., & Moos, R. H. (1994). Personal, situational, and contextual correlates of coping in adolescence. *Journal of Research on Adolescence, 4*, 99-125.

Efron, B., & Tibshirani, R. (1993). *An introduction to the bootstrap*. New York, NY: Chapman & Hall/CRC.

Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events: A study among healthy young subjects. *Psychoneuroendocrinology, 33*, 227–237.

Elzinga, B. M., Spinhoven, P., Berretty, E., de Jong, P., & Roelofs, K. (2010). The role of childhood abuse in HPA-axis reactivity in Social Anxiety Disorder: A pilot study. *Biological Psychiatry, 83*, 1-6.

Epel, E. S., Jimenez, S., Brownell, K., Stroud, L., Stoney, C., & Niaura, R. (2004). Are stress eaters at risk for the metabolic syndrome? *Annals of the New York Academy of Sciences, 1032*(Biobehavioral Stress Response: Protective and Damaging Effects), 208-210.

Epel, E. S., Lapidus, R., McEwen, B., & Brownell, K. (2001). Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology, 26*, 37-49.

Epel, E. S., McEwen, B., Seeman, T., Matthews, K. A., Castellazzo, G., Brownell, K., . . . Ickovics, J. R. (2000). Stress and body shape: Stress-induced cortisol secretion is consistently greater among women with central fat. *Psychosomatic Medicine, 62*, 623-632.

Evenson, K. R., Scott, M. M., Cohen, D. A., & Voorhees, C. C. (2007). Girls' perception of neighborhood factors on physical activity, sedentary behavior, and BMI. *Obesity, 15*, 430-445.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods, 41*, 1149-1160.

Fechner-Bates, S., Coyne, J. C., & Schwenk, T. L. (1994). The relationship of self-reported distress to depressive disorders and other psychopathology. *Journal of Consulting and Clinical Psychology, 62*, 550-559.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., . . . Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine, 14*, 245–258.

Field, A. (2009). *Discovering Statistics Using SPSS* (Third Edition ed.). Los Angeles, CA: SAGE.

Finkelhor, D. (2008). *Childhood victimization: Violence, crime, and abuse in the lives of young people*. New York, NY: Oxford University Press.

Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007a). Poly-victimization: a neglected component in child victimization. *Child Abuse & Neglect, 31*, 7–26.

Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007b). Re-victimization patterns in a national longitudinal sample of children and youth. *Child Abuse & Neglect, 31*, 479-502.

Finkelstein, E. A., Trogdon, J. G., Cohen, J. W., & Dietz, W. (2009). Annual medical spending attributable to obesity: Payer- and service-specific estimates. *Health Affairs, 28*, w822-w831.

Foster, M. T., Warne, J. P., Ginsberg, A. B., Horneman, H. F., Pecoraro, N. C., Akana, S. F., & Dallman, M. F. (2009). Palatable foods, stress, and energy stores sculpt corticotropin-releasing factor, adrenocorticotropin, and corticosterone concentrations after restraint. *Endocrinology, 150*(5), 2325-2333.

Fox, C. S., Massaro, J. M., Hoffmann, U., Pou, K. M., Maurovich-Horvat, P., Liu, C. Y., . . . O'Donnell, C. J. (2007). Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation, 116*, 39-48.

Gilbertson, M. W., Shenton, M. E., Ciszewski, A., Kasai, K., Lasko, N. B., Orr, S. P., & Pitman, R. K. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience, 5*, 1242–1247.

Gillum, R. F., & Sempos, C. T. (2005). Ethnic variation in validity of classification of overweight and obesity using self-reported weight and height in American women and men: the Third National Health and Nutrition Examination Survey. *Nutrition Journal, 4*, 27-35.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*, 47-55.

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.

Greeno, C. G., Marcus, M. D., & Wing, R. R. (1995). Diagnosis of binge eating disorder: discrepancies between a questionnaire and clinical interview. *International Journal of Eating Disorders, 17*, 153–160.

Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin, 115*, 444-464.

Gregoire, F., Genart, C., Hauser, N., & Remacle, C. (1991). Glucocorticoids induce a drastic inhibition of proliferation and stimulate differentiation of adult rat fat cell precursors. *Experimental Cell Research, 196*, 270-278.

Grilo, C. M., Masheb, R. M., Brody, M., Toth, C., Burke-Martindale, C. H., & Rothschild, B. S. (2005). Childhood maltreatment in extremely obese male and female bariatric surgery candidates. *Obesity Research, 13*, 123-130.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348-362.

Gruenewald, T. L., Kemeny, M. E., Aziz, N., & Fahey, J. L. (2004). Acute threat to the social self: Shame, social self-esteem, and cortisol activity. *Psychosomatic Medicine, 66*, 915-924.

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.

Gunstad, J., Paul, R. H., Spitznagel, M. B., Cohen, R. A., Williams, L. M., Kohn, M., & Gordon, E. (2006). Exposure to early life trauma is associated with adult obesity. *Psychiatry Research, 142*, 31– 37.

Hamer, M., O'Donnell, K., Lahiri, A., & Steptoe, A. (2010). Salivary cortisol responses to mental stress are associated with coronary artery calcification in healthy men and women. *European Heart Journal, 31*, 424–429.

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: reviewof the evidence. *Journal of Child Psychology and Psychiatry, 45*, 260–273.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., . . . Nemeroff, C. B. (2000). Pituitary adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284*, 592-597.

Heim, C., Newport, D. J., Wagner, D., Wilcox, M. M., Miller, A. H., & Nemeroff, C. B. (2002). The role of early adverse experience and adulthood stress in the prediction of neuroendocrine stress reactivity in women: a multiple regression analysis. *Depression and Anxiety*, 117-125.

Herman, J. P., Dolgas, C. M., & Carlson, S. L. (1998). Ventral subiculum regulates hypothalamopituitary-adrenocortical and behavioural responses to cognitive stressors. *Neuroscience, 86*, 449–459.

Hyland, M. E., Irvine, S. H., Thacker, C., Dann, P. L., & Dennis, I. (1989). Psychometric analysis of the Stunkard-Messick Eating Questionnaire (SMEQ) and comparison with the Dutch Eating Behavior Questionnaire (DEBQ). *Current Psychology: Research and Reviews, 8*, 228–233.

Jeffery, R. W., & Harnack, L. J. (2007). Evidence implicating eating as a primary driver for the obesity epidemic. *Diabetes, 56*, 2673-2676.

Jia, H., Li, J. Z., Lesserman, J., Hu, Y., & Drossman, D. A. (2004). Relationship of abuse history and other risk factors with obesity among female gastrointestinal patients. *Digestive Diseases and Sciences, 49*, 872-877.

Kandiah, J., Yake, M., Jones, J., & Meyer, M. (2006). Stress influences appetite and comfort food preferences in college women. [doi: DOI: 10.1016/j.nutres.2005.11.010]. *Nutrition Research, 26*(3), 118-123.

Karlsson, J., Persson, L. O., Sjostrom, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International JOurnal of Obesity and Related Metabolic Disorders, 24*, 1715–1725.

Kessler, R. C., Pecora, P. J., Williams, J., Hiripi, E., O'Brien, K., English, D., . . . Sampson, N. A. (2008). Effects of enhanced foster care on the long-term physical and mental health of foster care alumni. *Archives of General Psychiatry, 65*, 625-633.

Kirschbaum, C., & Hellhammer, D. (2000). Salivary cortisol and challenge tests Retrieved June 8, 2010, from http://www.macses.ucsf.edu/research/allostatic/challenge.php#trier

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81.

Kirschbaum, C., Wust, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine, 54*, 648-657.

Klaassens, E. R., van Noorden, M. S., Giltay, E. J., van Pelt, J., van Veen, T., & Zitman, F. G. (2009). Effects of childhood trauma on HPA-axis reactivity in women free of lifetime psychopathology. *Progress in Neuro-pspychopharmacology and Biological Psychiatry, 33*, 889-894.

Kudielkaa, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology, 69*, 113-132.

la Fleur, S. E., Houshyar, H., Roy, M., & Dallman, M. F. (2005). Choice of lard, but not total lard calories, damps adrenocorticotropin responses to restraint. *Endocrinology, 146*(5), 2193-2199.

Laitinen, J., Ek, E., & Sovio, U. (2002). Stress-related eating and drinking behavior and body mass index and predictors of this behavior. [doi: DOI: 10.1006/pmed.2001.0948]. *Preventive Medicine, 34*(1), 29-39.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer Publishing Company.

Lovallo, W. R., Farag, N. H., Sorocco, K. H., Cohoon, A. J., & Vincent, A. S. (2012). Lifetime adversity leads to blunted stress axis reactivity: studies from the Oklahoma Family Health Patterns Project. *Biological Psychiatry, 71*, 344–349.

MacMillan, H. L., Georgiades, K., Duku, E. K., Shea, A., Steiner, M., Niec, A., . . . Schmidt, L. A. (2009). Cortisol response to stress in female youths exposed to childhood maltreatment: Results of the Youth Mood Project. *Biological Psychiatry, 66*, 62-68.

Mamun, A. A., Lawlor, D. A., O'Callaghan, M. J., Bor, W., Williams, G. M., & Najman, J. M. (2007). Does childhood sexual abuse predict young adult's BMI? A birth cohort study. *Obesity, 15*, 2103-2110.

Marcus, M. D., & Wildes, J. E. (2009). Obesity: Is it a mental disorder? *International Journal of Eating Disorders, 42*, 739-753.

Marin, P., Darin, N., Amemiya, T., Andersson, B., Jern, S., & Bjorntorp, P. (1992). Cortisol secretion in relation to body fat distribution in obese premenopausal women. *Metabolism, 41*, 882-886.

Marniemi, J., Kronholm, E., Aunola, S., Toikka, T., Mattlar, C.-E., Koskenvuo, M., & Ronnemaa, T. (2002). Visceral fat and psychosocial stress in identical twins discordant for obesity. *Journal of Internal Medicine, 251*, 35-43.

Martyn-Nemeth, P., Penckofer, S., Gulanick, M., Velsor-Friedrich, B., & Bryant, F. B. (2009). The relationships among self-esteem, stress, coping, eating behavior, and depressive mood in adolescents. *Research in Nursing & Health, 32*(1), 96-109.

Mastorakos, G., Chrousos, G. P., & Weber, J. S. (1993). Recombinant interleukin-6 activates the hypothalamic–pituitary–adrenal axis in humans. *The Journal of Clinical Endocrinology and Metabolism, 77*, 1690–1694.

Meneghini, L. F., Spadola, J., & Florez, H. (2006). Prevalence and associations of binge eating disorder in a multiethnic population with type 2 diabetes. *Diabetes Care, 29*, 2760-2760.

Midei, A. J., & Matthews, K. A. (2009). Social relationships and negative emotional traits are associated with central adiposity and arterial stiffness in healthy adolescents. *Health Psychology, 28*, 347-353.

Midei, A. J., & Matthews, K. A. (2011). Interpersonal violence in childhood as a risk factor for obesity: A systematic review of the literature and proposed pathways. *Obesity Reviews, 12*, e159–e172. doi: doi: 10.1111/j.1467-789X.2010.00823.x

Midei, A. J., Matthews, K. A., & Bromberger, J. T. (2010). Childhood abuse and neglect are associated with adiposity in mid-life women: A possible role for trait anger and reproductive hormones. *Psychosomatic Medicine, 72*, 215-223.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*, 959-097. doi: doi: 10.1037/a0024768

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133*, 25-45.

Mitchell, S. L., & Epstein, L. H. (1996). Changes in taste and satiety in dietary-restrained women following stress. *Physiology & Behavior, 60*, 495-499.

Murali, R., & Chen, E. (2005). Exposure to violence and cardiovascular and neuroendocrine measures in adolescents. *Annals of Behavioral Medicine, 30*, 155-163.

National Heart Lung and Blood Institute. (1998). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults–the evidence report. *Obesity Research, 6*, 51S–209S.

Netherton, C. M., Goodyer, I. M., Tamplin, A., & Hertbert, J. (2004). Salivary cortisol and dehydroepiandrosterone in relation to puberty and gender. *Psychoneuoendocrinology, 29*, 125–140.

Newman, E., O'Connor, D. B., & Conner, M. (2007). Daily hassles and eating behaviour: The role of cortisol reactivity status. *Psychoneuroendocrinology, 32*, 125–132.

Nguyen-Rodriguez, S. T., Chou, C.-P., Unger, J. B., & Spruijt-Metz, D. (2008). BMI as a moderator of perceived stress and emotional eating in adolescents. *Eating Behaviors, 9*, 238–246.

Nicolson, N. A., Davis, M. C., Kruszewski, D., & Zautra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine, 72*, 471-480.

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*, e61-67.

Nurius, P. S., Russell, P. L., Herting, J. R., Hooven, C., & Thompson, E. A. (2009). Risk and protective profiles among never exposed, single form, and multiple form violence exposed youth. *Journal of Child and Adolescent Trauma, 2*, 106–123.

Ogden, C. L., Carroll, M. D., Curtin, L. R., McDowell, M. A., Tabak, C. J., & Flegal, K. M. (2006). Prevalence of overweight and obesity in the United States, 1999-2004. *Journal of American Medical Association, 295*(13), 1549-1555.

Oliver, G., & Wardle, J. (1999). Perceived effects of stress on food choice. *Physiology & Behavior, 66*, 511-515.

Oliver, G., Wardle, J., & Gibson, E. L. (2000). Stress and food choice: a laboratory study. *Psychosomatic Medicine, 62*, 853-865.

Oquendo, M. A., Echavarria, G., Galfalvy, H. C., Grunebaum, M. F., Burke, A., Barrera, A., . . . Mann, J. (2003). Lower cortisol levels in depressed patients with comorbid post-traumatic stress disorder. *Neuropsychopharmacology, 28*, 591-598.

Ouellet-Morin, I., Odgers, C. L., Danese, A., Bowes, L., Shakoor, S., Papadopoulos, A. S., . . . Arseneault, L. (2011). Blunted cortisol responses to stress signal social and behavioral problems among maltreated/bullied 12-year-old children. *Biological Psychiatry, 70*, 1016–1023.

Paradis, A.-M., Pérusse, L., Godin, G., & Vohl, M.-C. (2008). Validity of a self-reported measure of familial history of obesity. *Nutrition Journal, 7*, 27.

Pasquali, R., Cantobelli, S., Casimirri, F., Capelli, M., Bortoluzzi, L., Flamia, R., . . . Barbara, L. (1993). The hypothalamic-pituitary-adrenal axis in obese women with different patterns of body fat distribution. *Journal of Clinical Endocrinology & Metabolism, 77*, 341-346.

Pedersen, S., Jonler, M., & Richelsen, B. (1994). Characterization of regional and gender differences in glucocorticoid receptors and lipoprotein lipase activity in human adipose tissue. *Journal of Clinical Endocrinology and Metabolism, 78*, 1354-1359.

Pijlman, F. T., Wolterink, G., & Van Ree, J. M. (2003). Physical and emotional stress have differential effects on preference for saccharine and open field behaviour in rats. *Behavioral Brain Research, 139*, 131–138.

Polivy, J., & Herman, C. P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders, 26*(2), 153-164.

Preis, S. R., Massaro, J. M., Robins, S. J., Hoffmann, U., Vasan, R. S., Irlbeck, T., . . . Fox, C. S. (2010). Abdominal subcutaneous and visceral adipose tissue and insulin resistance in the Framingham Heart Study. *Obesity, 18*(11), 2191-2198.

Prineas, R. I., Folsom, A. R., & Kaye, S. A. (1993). Central adiposity and increased risk of coronary artery disease mortality in older women. *Annals of Epidemiology, 3*, 35-41.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.

Raison, C. L., & Miller, A. H. (2003). When not enough is too much: The role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *American Journal of Psychiatry, 160*, 1554–1565. doi: doi:10.1176/appi.ajp.160.9.1554

Rich-Edwards, J. W., Spiegelman, D., Lividoti Hibert, E. N., Jun, H. J., Todd, T. J., Kawachi, I., & Wright, R. J. (2010). Abuse in childhood and adolescence as a predictor of Type 2 diabetes in adult women. *American Journal of Preventive Medicine, 39*, 529 –536. doi: doi:10.1016/j.amepre.2010.09.007

Riley, E. H., Wright, R. J., Jun, H. J., Hibert, E. N., & Rich-Edwards, J. W. (2010). Hypertension in adult survivors of child abuse: Observations from the Nurses' Health Study II. *Journal of Epidemiology and Community Health Psychology, 64*, 413–418. doi: doi:10.1136/jech.2009.095109

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*, 878-887.

Romero, A. J., Robinson, T. N., Kraemer, H. C., Erickson, S. J., Haydel, F., Mendoza, F., & Killen, J. D. (2001). Are perceived neighborhood hazards a barrier to physical activity in children? *Archives of Pediatric and Adolescent Medicine, 155*, 1143-1148.

Romito, P., & Grassi, M. (2007). Does violence affect one gender more than the other? The mental health impact of violence among male and female university students. *Social Science & Medicine, 65*, 1222-1234.

Rosmond, R., Dallman, M. F., & Bjorntorp, P. (1998). Stress-related cortisol secretion in men: relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. *Journal of Clinical Endorinology and Metabolism, 83*, 1853–1859.

Rutters, F., Nieuwenhuizen, A. G., Lemmens, S. G. T., Born, J. M., & Westerterp-Plantenga, M. S. (2008). Acute stress-related changes in eating in the absence of hunger. *Obesity, 17*(1), 72-77.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews, 21*, 55–89. doi: doi:10.1210/er.21.1.55

Scher, C. D., Forde, D. R., McQuaid, J. R., & Stein, M. B. (2004). Prevalence and demographic correlates of childhood maltreatment in an adult community sample. *Child Abuse & Neglect, 28*, 167–180.

Schwartz, D., & Proctor, L. J. (2000). Community violence exposure and children's social adjustment in the school peer group: The mediating roles of emotion regulation and social cognition. *Journal of Consulting and Clinical Psychology, 68*, 670-683.

Shenk, C. E., Noll, J. G., Putnam, F. W., & Trickett, P. K. (2010). A prospective examination of the role of childhood sexual abuse and physiological asymmetry in the development of psychopathology. *Child Abuse & Neglect, 34*, 752–761.

Shively, C. A., Laber-Laird, K., & Anton, R. F. (1997). Behavior and physiology of social stress and depression in female cynomolgus monkeys. *Biological Psychiatry, 41*, 871–882.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods, 7*, 422–445.

Silverman, W. K., Ortiz, C. D., Viswesvaran, C., Burns, B. J., Kolko, D. J., Putnam, F. W., & Amaya-Jackson, L. (2008). Evidence-based psychosocial treatments for children and adolescents exposed to traumatic events. *Journal of Clinical Child & Adolescent Psychology, 37*, 156–183.

Silverthorn, D. U. (2007). *Human Physiology: An Integrated Approach*. San Francisco, CA: Pearson Education.

Sims, R., Gordon, S., Garcia, W., Clark, E., Monye, D., Callender, C., & Campbell, A. (2008). Perceived stress and eating behaviors in a community-based sample of African Americans. [doi: DOI: 10.1016/j.eatbeh.2007.06.006]. *Eating Behaviors, 9*(2), 137-142.

Singh, G. L., Kogan, M. D., & van Dyck, P. C. (2008). A multilevel analysis of state and regional disparities in childhood and adolescent obesity in the United States. *Journal of Community Health, 33*, 90-102.

Spielberger, C. D., Jacobs, G., Russell, S., & Crane, R. S. (1983). Assessment of anger: The State-Trait Anger Scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol. 2, pp. 159-187). Hillsdale, NJ: Lawrence Erlbaum.

Stein, B. D., Jaycox, L. H., Kataoka, S., Rhodes, H. J., & Vestal, K. D. (2003). Prevalence of child and adolescent exposure to community violence. *Clinical Child and Family Psychology Review, 6*, 247-264.

Stein, B. D., Jaycox, L. H., Kataoka, S. H., Wong, M., Tu, W., Elliot, M. N., & Fink, A. (2003). A mental-health intervention for schoolchildren exposed to violence. *JAMA, 290*, 603-611.

Stewart, J., & Seeman, T. (2000). Salivary Cortisol Measurement Retrieved April 24, 2012, from http://www.macses.ucsf.edu/research/allostatic/salivarycort.php

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29*, 71-83.

Sweeting, H., Wright, C., & Minnis, H. (2005). Psychosocial correlates of adolescent obesity, 'slimming down' and 'becoming obese'. *Journal of Adolescent Health, 37*, 409.e409-409.e417.

Tarullo, A. R., & Gunnar, M. R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior, 50*, 632–639.

Tataranni, P. A., Larson, D. E., Snitker, S., Young, J. B., Flatt, J. P., & Ravussin, E. (1996). Effects of glucocorticoids on energy metabolism and food intake in humans. *American Journal of Physiology-Endocrinology and Metabolism, 271*, E317-E325.

Teichera, M. H., Anderson, C. M., & Polcari, A. (2012). Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proceedings of the National Academy of Science, 109*, E563–E572.

Therrien, F., Drapeau, V., Lalonde, J., Lupien, S. J., Beaulieu, S., Tremblay, A., & Richard, D. (2007). Awakening cortisol response in lean, obese, and reduced obese individuals: effect of gender and fat distribution. *Obesity, 15*, 377–385.

Thomas, C., Hypponen, E., & Power, C. (2008). Obesity and type 2 diabetes risk in midadult life: The role of childhood adversity. *Pediatrics, 121*, e1240-1249.

Timmerman, G. M. (1999). Binge Eating Scale: Further assessment of validity and reliability. *Journal of Applied Biobehavioral Research, 4*, 1-12.

Trickett, P. K., Noll, J. G., Susman, E. J., Shenk, C. E., & Putnam, F. W. (2010a). Attenuation of cortisol across development for victims of sexual abuse. *Developmental Psychopathology, 22*, 165–175.

Trickett, P. K., Noll, J. G., Susman, E. J., Shenk, C. E., & Putnam, F. W. (2010b). Attenuation of cortisol across development for victims of sexual abuse. *Development and Psychopathology, 22*, 165–175.

Walker, E. A., Gelfan, A., Katon, W. J., Koss, M. P., Von Korff, M., Bernstein, D., & Russo, J. (1999). Adult health status of women with histories of childhood abuse and neglect. *American Journal of Medicine, 107*, 332-339.

Wallerius, S., Rosmond, R., Ljung, T., Holm, G., & Björntorp, P. (2003). Rise in morning saliva cortisol is associated with abdominal obesity in men: a preliminary report. *Journal of Endocrinology Investigations, 26*, 616–619.

Wardle, J., Steptoe, A., Oliver, G., & Lipsey, Z. (2000). Stress, dietary restraint and food intake. *Journal of Psychosomatic Research, 48*, 195-202.

Watson, D., Clark, L., & Tellegen, A. (1988). Development and validation of brief measure of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.

Weinstein, S. E., Shide, D. J., & Rolls, B. J. (1997). Changes in food intake in response to stress in men and women: Psychological factors. *Appetite, 28*, 7-18.

Weiss, D., & Marmar, C. (1997). The Impact of Event Scale - Revised. In J. Wilson & T. Keane (Eds.), *Assessing psychological trauma and PTSD*. New York, NY: Guildford.

Woodall, K. L., & Matthews, K. A. (1993). Changes in and stability of hostile characteristics: Results from a 4-Year Longitudinal Study of Children. *Journal of Personality and Social Psychology, 64*, 491-499.

Yehuda, R. (2006). Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Annals of the New York Academy of Sciences, 1071*, 137–166.

Zakrzewska, K. E., Cusin, I., Sainsbury, A., Rohner-Jeanrenaud, F., & Jeanrenaud, B. (1997). Glucocorticoids as counterregulatory hormones of leptin: Toward an understanding of leptin resistance. *Diabetes, 46*, 717–719.

Zeidner, M., & Saklofske, D. (Eds.). (1996). *Adaptive and maladaptive coping*. New York City, New York: Wiley.

Zellner, D. A., Loaiza, S., Gonzalez, Z., Pita, J., Morales, J., Pecora, D., & Wolf, A. (2006). Food selection changes under stress. *Physiology & Behavior, 87*(4), 789-793.

Zinzow, H. M., Ruggiero, K. J., Resnick, H., Hanson, R., Smith, D., Saunders, B., & Kilpatrick, D. (2009). Prevalence and mental health correlates of witnessed parental and community violence in a national sample of adolescents. Journal of Child Psychology and Psychiatry, 50, 441-450.