The Role of Self-esteem in Understanding the Link Between Subclinical Levels of Childhood Trauma and Risk Factors for Disease Later in Life

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ABSTRACT

The Role of Self-esteem in Understanding the Link Between Subclinical Levels of Childhood Trauma and Risk Factors for Disease Later in Life

A thesis presented to the Department of Psychology

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Current literature proposes a relationship between retroactive reports of childhood trauma and the cortisol response to acute psychosocial stress in delineating the pathways that may lead to future health problems. Less is known about sub-clinical levels of trauma and possible mediators of this relationship.

A sample of healthy adults (mean BMI =23.5 kg/m²), of both genders (female N=12, male N=15) with a mean age of 21.8 years (SD=4.6) provided self-ratings of childhood experiences using the Childhood Trauma Questionnaire (CTQ). Participants also completed the Rosenberg Self-esteem Scale to assess global self-esteem. All participants were exposed to the Trier Social Stress Test on two consecutive days to elicit an acute stress response. Saliva samples for assessment of cortisol were taken repeatedy before and after each stressor.
Mean CTQ total scores were in the low range (M=7.3, SD=2.0). Higher childhood trauma scores predicted higher cortisol responses ($\beta = .53$, $t(25) = 3.13$, $p = 0.004$), and lower self-esteem scores ($\beta = -0.55$, $t(25) = -3.26$, $p =0.003$), and lower self-esteem scores predicted higher cortisol responses ($\beta = -0.71$, $t(25) = -5.10$, $p <0.001$), upon second exposure to stress only. Self-esteem fully mediated the relationship between childhood trauma scores and the cortisol stress response to the second stress exposure, reducing it to non-significant ($\beta = 0.20$, $t(22) = 1.10$, $p = 0.29$), contrary to other personality measures tested, demonstrating its full mediation properties.

In summary, the results indicate that even sub-clinical levels of childhood trauma can have a negative effect on stress response systems; trauma need not be in the severe range to exert a powerful effect on these systems, particularly for repeated stress exposure. Although these effects may be rather benign in the short term, over time, they may lead to risk factors for several diseases. The mediating effects of self-esteem may help inform therapies that are geared towards helping those who have experienced subclinical levels of childhood trauma by including a self-esteem enhancing component.
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1 Introduction

Childhood trauma is a life-altering event that affects an estimated 794,000 children in the United States in a single year (U.S. Department of Health and Human Services, 2009). This type of trauma is life altering in that it has been shown to affect sufferers both psychologically in terms of developing anxiety and depressive disorders (Gibb, Chelminski, & Zimmerman, 2007; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Risbrough & Stein, 2006; Sachs-Ericsson et al., 2010; Watson et al., 2007) and physiologically in terms of developing risk factors for major physical illnesses (Carpenter et al., 2010; Gonzalez, Jenkins, Steiner, & Fleming, 2009; Heim et al., 2000; Heim et al., 2006; Lovallo, Farag, Sorocco, Cohoon, & Vincent, 2012; Luecken & Lemery, 2004; McEwen, 2003, 2008; Miller, Chen, & Parker, 2011; Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010; Repetti, Taylor, & Seeman, 2002; Taylor, Lerner, Sage, Lehman, & Seeman, 2004; van der Vegt, van der Ende, Huizink, Verhulst, & Tiemeier, 2010).

The physiological effects of trauma have been found to occur both at the time of the abuse and throughout the lifespan (Lupien, McEwen, Gunnar, & Heim, 2009; Rao, Hammen, Ortiz, Chen, & Poland, 2008; Tarullo & Gunnar, 2006). However, little is known about the impact of less traumatic, sub-clinical levels of childhood trauma and the stress response (Engert et al., 2010; Meaney et al., 1996; Miller et al., 2009; Pruessner, Lord, Meaney, & Lupien, 2004; Saridjan et al., 2010). The effects of childhood trauma are believed to be mediated through reciprocal relationships.
between stress response systems such as the hypothalamic-pituitary-adrenal (HPA) axis, sympathetic nervous system (SNS) and the immune system, as well as limbic structures and higher brain centers such as the frontal cortex, amygdala and hippocampus, ultimately resulting in dysregulation of these systems (Dickerson & Kemeny, 2004; McEwen, 2008; Rohleder, Chen, Wolf, & Miller, 2008; Stratakis & Chrousos, 1995).

Self-esteem is a personality construct that has been shown to be impaired in people who have had traumatic childhoods (Bolger, Patterson, & Kupersmidt, 1998; Cicchetti & Rogosch, 2009; Finzi-Dottan & Karu, 2006; Lopez & Heffer, 1998; Sachs-Ericsson et al., 2010; Schuck & Widom, 2001) and low self-esteem has been linked with maladaptive stress response patterns (Dickerson & Kemeny 2004; Lindahl, Theorell & Lindblad, 2005; Gruenwald, Kemeny, Aziz & Fahey, 2004; Pruessner, Lord, Meaney & Lupien, 2004; Zorrilla, DeRubeis & Redei, 1995). Because less severe childhood trauma may still impair self-esteem, low levels of childhood abuse may also be associated with maladaptive stress responses. Therefore, self-esteem may serve as an important mediator in the relationship between low levels of trauma that occurred in childhood and the adult cortisol stress response.

Very few studies have explored the later life consequences of low levels of childhood trauma. To the best of our knowledge, subclinical levels of trauma, self-esteem and the cortisol stress response have not been investigated together in one study. It is important to understand the impact that sub-clinical levels of childhood abuse may have on later life health outcomes so that proper preventative and intervention measures can be designed. Therefore, one of the goals of the present
study is to explore the relationship between low levels of childhood trauma and the adult cortisol stress response. The present study will also explore current levels of self-esteem as a possible pathway that may help explain the maladaptive stress response of people who have experienced low levels of childhood abuse.

It has been argued that humans are social animals that cannot survive in isolation, our social wellbeing is as important to our survival as our physical wellbeing (Baumeister & Leary, 1995; Bowlby, 1988; Gruenewald, Kemeny, Aziz, & Fahey, 2004; James, 1952). Therefore, perceived threats to our social well-being initiate a physiological response to help handle the threat. The stress response is the body’s way of dealing with physically and psychologically threatening situations and is therefore typically an adaptive, healthy chain of events (Sapolsky, Romero, & Munck, 2000). Threatening situations are appraised by the frontal cortex and thalamus, which sends signals to the hippocampus and amygdala so that the organism may respond mentally and emotionally to the threat (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Dickerson & Kemeny, 2004; Feldman, Conforti, & Weidenfeld, 1995). These signals also set in motion the HPA axis that secretes glucocorticoids (for example, cortisol) from the adrenal gland.

Timed just prior to the HPA axis activation, the SNS instigates the adrenal medulla to secrete epinephrine and norepinephrine into the bloodstream giving the body a fast dose of energy to fend off the threat. It has been hypothesized that norepinephrine also acts on pro-inflammatory pathways to induce inflammation, to help protect the organism from physical harm incurred during the threat (Bierhaus et al., 2003; Jessop, 1999).
Negative feedback loops, at all levels of the stress response system occur through the release of cortisol, again via the HPA axis, triggered by cytokines from the immune system once the stressful situation has abated, in order to shut down the HPA axis response and return the organism to a set point of homeostasis. Cortisol also regulates inflammation by binding to glucocorticoid receptors (GRs) located within immune cells, thereby acting as an anti-inflammatory agent (Lupien et al., 2009; Miller et al., 2011).

Certain levels of cortisol are needed for optimal functioning of several biological systems. For example, the stress response increases blood glucose levels in the body to produce energy to deal with stressful situations. However, consistently high levels of blood sugar have been associated with certain metabolic diseases such as diabetes. Similarly, catecholamines and other hormones rely on cortisol to help activate the cardiovascular system during stress. This activation produces vasoconstriction as well as increased heart rate needed to mobilize the organism during a threat. However, sporadic and chronic activation of the cardiovascular system during stress leaves it vulnerable to damage. This may eventually lead to health conditions such as high blood pressure and atherosclerosis, among other problems (Dickerson & Kemeny, 2004; McEwen, 2008). Digestion is also inhibited during the stress response so as to allocate as many resources as possible to deal with the stressor. Consequently, stress has been associated with gastrointestinal problems in humans (Bose, Unwin, Warakaulle, & Uthappa, 2009) and in animal studies (Dallman et al., 2006). Autoimmune diseases have also been associated with levels of
cortisol that are too low, resulting in under-suppression of the immune system (Segerstrom & Miller, 2004; Wolf, Nicholls, & Chen, 2008).

Furthermore, in more recent years, chronic inflammation has been implicated as the culprit to many of these disease processes. It has been proposed that atherosclerosis and ischemic strokes, and ultimately heart disease are the result of chronic inflammatory response to arterial injuries incurred doing repeated elicitation of the cardiovascular system (Libby and Therous, 2005; Ross, 1999). Inflammation may also play a key role in the development of certain elements of the metabolic syndrome such as hypertension and Type 2 diabetes (Bertoni et al., 2010; Pradhan, Manson, Rifai, Buring, & Ridker, 2001; Sesso et al., 2003). One study found a link between high levels of inflammatory molecules such as CRP and increases in these metabolic syndrome elements over time (Dandona, Aljada, Chaudhuri, Mohanty, & Garg, 2005). All in all, situations that repeatedly elicit the stress response, as well as differences in genetic and biological makeup, can eventually lead to health conditions that are problematic.

An abundance of studies have looked at childhood trauma and pathophysiology later in life. For example, one study found that women with a history of childhood physical or sexual abuse showed hyper-reactivity of the HPA axis to the widely used psychosocial stress paradigm, the Trier Social Stress Test (TSST), resulting in increased ACTH and cortisol levels (Heim et al., 2000). Heim et al., 2006 also found that women who had been abused and were depressed had higher cortisol and heart rate responses to stress induction than abused women without depression and non-abused women. Severe childhood trauma also significantly increases the risk
of developing Chronic Fatigue Syndrome (CFS) later in life (Heim et al., 2006). This study found that exposure to moderate to severe levels of childhood abuse was associated with a 3 to 8 fold increased risk for CFS depending on trauma subtypes. Severe childhood abuse is also associated with increased biomarkers for inflammation (Carpenter et al., 2010). Carpenter et al. found that after controlling for body mass index (BMI) and age, higher scores on the childhood trauma questionnaire were associated with higher delta and maximum Interleukin-6, a pro-inflammatory cytokine, measurements in response to the TSST. Greater stress induced increases in IL-6 were observed in the trauma group (women with moderate to severe childhood abuse) when controlling for depression, trait anxiety, quality of life, perceived stress and menstrual cycle, but not in the group of women without any childhood abuse (control group).

Other studies show that maltreatment is associated with blunted levels of cortisol. In a study of formerly maltreated adopted children, severe maltreatment was associated with lower basal cortisol levels compared to non-maltreated adoptees (van der Vegt et al., 2010). However, the adoptees with low cortisol levels were also diagnosed with anxiety disorders, which may have confounded the results. Another study by Lovallo et al., 2012 found a blunted cortisol response in participants with a history of childhood trauma (Lovallo et al., 2012). However, this study contained certain methodologies that are different from several of the studies already mentioned such as length and type of stressor, and calculation of cortisol reactivity. Also, the assessment of childhood physical trauma was coupled with trauma that is more likely to happen in adulthood such as being mugged. Although the results are conflicting,
collectively, these studies show an association between high scores on measures of childhood trauma, and overall dysregulation of the HPA axis and the immune system.

Less is known about low, subclinical levels of childhood trauma and the impact they may have on adult physiology. Animal studies have provided preliminary findings on the impact of low level, traumatic early experiences and the stress response (Meaney et al., 1996). Animals that are separated from their mother early on, or are exposed to physical trauma or endotoxins, exhibit enhanced HPA axis responsivity to stress. However, the findings in low, or subclinical, levels of trauma and the stress response in humans are conflicting. In one human study low maternal care was associated with blunted cortisol responses to repeated psychosocial stress (Engert et al., 2010). Other studies have found a link between low levels of trauma and high cortisol levels, although they focus on a single aspect of trauma such as social or socioeconomic adversity (Miller et al., 2009; Saridjan et al., 2010) or parental depression (Halligan, Herbert, Goodyer, & Murray, 2004). Overall, these findings indicate a need for further investigation into the relationship between subclinical levels of childhood trauma and the cortisol stress response.

Along with maladaptive physiological changes, unhealthy psychological functioning can result from an abusive childhood. Childhood abuse can have profoundly negative effects on personality characteristics. A wealth of literature suggests that there is a distinct relationship between childhood trauma and adult self-esteem. One study found that college students in Israel who experienced childhood emotional abuse tended to show decreased levels of self-esteem, as assessed by the Childhood Trauma Questionnaire and the Rosenberg Self-esteem Scale, compared to
those without childhood emotional abuse (Finzi-Dottan & Karu, 2006). Another study found that college students who had been physically abused showed lower self-concepts than those who had not been abused (Lopez & Heffer, 1998). Schuck and Widom, 2001 found that childhood abuse survivors reported significantly lower self-esteem than their matched counterparts in their study, although they did not find that self-esteem mediated the relationship between childhood abuse and alcohol abuse in women as hypothesized (Schuck & Widom, 2001).

Another study found that childhood trauma had more profound adverse effects on older adults with low self-esteem as opposed to those with high self-esteem. Again, though, contrary to prediction, in this study self-esteem did not mediate the relationship between childhood abuse and internalizing disorders in older adults (Sachs-Ericsson et al., 2010). In a longitudinal study, physically and sexually maltreated, but not emotionally maltreated, children scored significantly lower on self-esteem measures than matched controls (Bolger et al., 1998). Although the findings are somewhat heterogeneous, overall, childhood maltreatment has detrimental, long-lasting negative effects on self-esteem.

Some studies have found an association between cortisol responses and self-esteem. For example, one study found that a group of male subjects who had high cortisol responses to a repeated psychosocial stress paradigm (5 consecutive days of the TSST) scored lower on self-esteem measures than men who had low cortisol responses (Kirschbaum et al., 1995). In addition, when analyzing the social evaluative threat component of the TSST, it was found that cortisol increases were greater for people who reported low social self-esteem, but not for those who reported low
performance self-esteem (Gruenewald et al., 2004). In contrast to these results, one study found that people with low self-esteem showed a positive association with increasing age and a lower cortisol awakening response. This association was not found in people with high self-esteem (Pruessner et al., 2004).

One study in particular investigated the relationships between childhood trauma, personality factors and risk factors for heart disease. Van Reedt et al., 2012 found negative associations between personality factors such as openness to experience and agreeableness and overall metabolic risk (van Reedt Dortland, Giltay, van Veen, Zitman, & Penninx, 2012). Another study by Cicchetti and Rogosch, 2009 found that self-esteem, as well as other aspects of self-organization, can be used to successfully cope with the effects of traumatic childhoods (Cicchetti & Rogosch, 2009).

Situations that involve psychosocial stress are usually not a one-time occurrence, they tend to happen multiple times in a single day. This may be especially true for people who have low self-esteem, whose social skills may not be highly developed. It is possible that even situations that are relatively nonthreatening may be a source of psychosocial stress for people with low self-esteem, although this idea is based on speculation. It has been hypothesized that the stress response might show different patterns due to allostatic load and that these changes might affect disease processes (McEwen, 1998). The term allostatic load describes the damaging effects of allostasis, meaning the wear and tear on the body by repeated changes of homeostatic set points (McEwen & Stellar, 1993). Allostasis describes the process of achieving stability through changing the set points involved in homeostasis. In order to capture
the experience of multiple stressors that may contribute to the emergence of allostatic load, some studies perform at least two exposures to a laboratory stressor. There is also evidence that at least two exposures of psychosocial stress are needed to accurately assess the cortisol stress response that is not confounded with novelty (the first day of the laboratory stressor). Responses to repeated stress (at least 2 stress exposures) also correlate better with personality measures (Kirschbaum et al., 1995).

In summary, increased cortisol stress responses to a psychosocial stress challenge are associated with both maltreatment in childhood and low self-esteem. It has also been shown that people who grow up in abusive homes tend to have lower levels of self-esteem. Thus, it may be possible that these low levels of self-esteem are the driving force behind the exaggerated cortisol responses to acute psychosocial stress challenges. Finally, it is speculated that over time, increased cortisol stress responses are associated with negative health outcomes such as risk factors for heart disease and other health complications.

The overarching goal of the present study is to establish a relationship between low levels of childhood trauma and current cortisol responses to psychosocial stress, and to examine whether self-esteem helps explain this relationship by acting as a mediator. These relationships will be explored through the venue of repeated acute psychosocial stress to take into account the possible effects of multiple stressors. Although previous research has already established relationships between self-esteem, the stress response and trauma in childhood, this is the first study to investigate these three variables together and to test the role of self-esteem as a mediator. Furthermore, this is one of the first studies to investigate the relationship
between these variables and low levels of childhood trauma, as opposed to more severe levels of trauma. Specifically, we hypothesized that higher levels of trauma would predict higher cortisol responses to both the first and second exposure to psychosocial stress. Second we hypothesize that higher levels of trauma will predict lower levels of self-esteem. Third, we hypothesize that lower levels of self-esteem would predict higher cortisol responses to both the first and second exposure to stress. We also hypothesize that these relationships will be independent of current depression and anxiety levels, perceived stress and six personality measures. We further hypothesized that on the first and second exposure to stress, self-esteem would act as a full mediator in the relationship between childhood trauma and the cortisol stress response, reducing it to non-significant.
2 Methods

2.1 Participants

The sample consisted of young adults attending Brandeis University who were recruited via flyers posted on campus, campus newspapers and Internet posts. Potential participants were screened for medical and psychiatric illnesses, and excluded for the presence of either condition. Females were scheduled during their luteal phase to control for menstrual cycle hormonal variations, and excluded for the use of birth control. Participants (N=27; 15 male; 13 Caucasian) had a mean age of 21.8 years (SD = 4.6) and a mean Body Mass Index (BMI) of 23.5 kg/m2 (SD = 3.1). Participants received either monetary compensated or extra credit in an Introduction to Psychology course. Participants provided written informed consent and procedures were approved by the Institutional Review Board.

2.2 Procedure

Participants were invited to come to the lab at Brandeis University for three hours on two consecutive weekdays or the weekend between 1300 and 2000 hours to control for variations in circadian rhythms of stress systems. Participants were told during the screening process that they would be exposed to a standardized laboratory stress situation and would be asked to speak with other people about themselves and work on another problem solving task. After providing informed consent, a baseline saliva sample and height and weight measurements were taken. Subsequent saliva samples were taken after a rest period of 30 minutes one minute before the stress
induction (described below), directly after the stress induction, and then at 10, 30, 60, and 120 minutes post stress induction. Participants filled out self-report questionnaires assessing past and present physical and psychological well being (described below) during the rest period and after the stress induction. The same procedure was followed for day two of the experiment.

2.3 Acute stress induction

The Trier Social Stress Test (TSST) was employed to elicit a stress response. The TSST is a well-validated, standardized laboratory psychosocial stress situation that has been shown to reliably activate the HPA axis and SNS (Dickerson & Kemeny, 2004; Kirschbaum, Pirke, & Hellhammer, 1993). The stress test consists of a five-minute preparation period, a five-minute public speaking task in which the participant talks about their personal qualifications for a dream job, and a five-minute verbal mental arithmetic task in which the participant counts backwards from a large number in increments of 17 (on day one) or 13 (on day two). The participant presents into a microphone and a video camera in front of a neutral panel (one male, one female) wearing white lab coats. The participant is told that the panel is trained in evaluating verbal and non-verbal behavior and that their performance will be videotaped for later analysis.

2.4 Measures

2.4.1 Self-report measures

Evaluation of overall psychological well being was established by assessing depressive symptoms using the Center for Epidemiological Studies-Depression Scale
(CES-D) (Radloff, 1977) and participants’ existing stress levels were assessed using the Perceived Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983).

Childhood Trauma was measured using a shortened form of the Childhood Trauma Questionnaire, a highly used instrument that measures instances of childhood abuse and neglect (Bernstein et al., 1994). This modified version consists of 28 items that are scored on a five-point Likert scale with answers ranging from “never true” to “very often true”. The questionnaire is divided into five subscales with five items each: physical abuse, sexual abuse, emotional abuse, physical neglect and emotional neglect; three items assessed the validity of the responses.

Sample questions for the physical abuse subscale included, “I was punished with a belt, a board, a cord or some other hard object”; for the sexual abuse subscale, “Someone tried to touch me in a sexual way, or tried to make me touch them”; for the emotional abuse subscale, “People in my family called me things like ‘stupid’ ‘lazy’ and ‘ugly’”; for the physical neglect, “I didn’t have enough to eat”; and for the emotional neglect subscale all items were reversed scored such as, “I felt loved”. A higher total score overall and on each subscale represents higher instances of trauma or abuse. The total score was used in the analysis as opposed to each individual subscale due to a limited number of subjects in several categories of abuse. The scale had acceptable internal consistency (Cronbach’s $\alpha = 0.70$).

Self-esteem was measured using the Rosenberg Self-esteem Scale (RSE), a well-validated measure that quantifies global self-esteem (Rosenberg, 1965). The questionnaire consists of 10 items that are scored on a 4-point Likert scale with answers ranging from “strongly disagree” to “strongly agree”. Sample questions
include, “On the whole, I am satisfied with myself” and reverse coded items such as, “All in all, I am inclined to think that I am a failure”. A higher score on this questionnaire is indicative of higher self-esteem. The scale had high internal consistency (Cronbach’s $\alpha = 0.85$).

The personality factors: neuroticism, extraversion, openness to experience, conscientiousness, agency and agreeableness were assessed using a revised version of the Midlife Development Inventory (MIDI) Personality Scales (Prenda, K. & Lachman. M. (2001). The scale uses 31 adjectives scored on a 4-point Likert scale with answers ranging from “a lot” to “not at all”.

In addition to the CES-D measure, overall psychological distress was measured using the Hospital Anxiety and Depression Scale (HADS) (Zigmond, A.S. and Snaith, R.P., 1983). This instrument measures severity and frequency of anxiety disorders and depression in patients and in the general population.

2.4.2 Biological measures

For cortisol measurements, saliva was collected via Salivettes (Sarstedt, Newton, NC). Participants were instructed to swirl the cotton roll in their mouth around with their tongue for 2 minutes, and then spit it back into the tube. Samples were collected upon arrival (baseline S0), after a rest period of 30 minutes, 1 minute before the stress induction (S1) directly after the stress induction (S2), and then at 10 (S3), 30 (S4), 60 (S5) and 120 (S6) minutes post stress induction. Samples were stored at room temperature during the experiment and then at -30°C until analysis. Salivettes were centrifuged for 10 minutes at 2000g and 4°C, and salivary free cortisol concentrations were measured using a commercially available
chemiluminescence immunoassay (CLIA; IBL-International, Toronto, Canada). Intra- 
and inter-assay variability were below 10%. Body fat and weight was measured 
using a commercially available body fat scale (Seca; 720 Supra Plus Digital Scale; 
Hanover, MD, USA), which provides approximate percentages of body fat and water 
in participants using skin conductance.

2.5 Statistical analysis

Statistical analysis was performed using SPSS 19 software package (IBM, 
Chicago, IL, USA). Normal distribution and homogeneity of variance was determined 
using Kolmogorov-Smirnov and Levene’s test. Due to violation of normality, the 
childhood trauma variable was log transformed. Repeated measures ANOVA were 
conducted to test for changes in delta cortisol before and after the TSST, as well as 
the difference between the two TSST days. These analyses included seven cortisol 
time points as a within subjects factor, and TSST day (1 and 2) as a between subjects 
factor. This was followed by a paired samples t-test to see if the second stress 
exposure produced a significant decrease in cortisol response. Due to violation of the 
sphericity assumption, degrees of freedom were adjusted using the Greenhouse- 
Geisser technique.

A delta score was calculated to capture the increase in cortisol relative to the 
time point immediately before the TSST (S1) by subtracting S1 from the peak 
measurement after the TSST. The delta calculation was used as the cortisol variable 
in both the correlation and the regression analyses reported below. To tests 
hypotheses, Pearson correlations were first performed to explore the bivariate 
relationship between the self-report variables and the delta cortisol variable. Second,
regression analyses were performed to test the prediction of delta cortisol from the childhood trauma responses, the prediction of self-esteem from the childhood trauma responses, the prediction of these two outcome variables when controlling for BMI and gender, and the mediation by self-esteem, as well as testing the mediating effects of several other variables including perceived stress (PSS), depression (CESD), personality (MIDI) and anxiety and depression (HADS). Tolerance and VIP statistics were checked to ensure non-violation of multicollinearity.

A bootstrapping analysis and Sobel test were performed to confirm mediation of self-esteem in the relationship between childhood trauma and cortisol stress reactivity. Subjects who had high baseline cortisol measurements (> 4) were excluded from the analysis to control for potentially confounding effects of anticipatory stress and baseline measurements. All data are reported as mean and standard deviation (mean ± standard error of the mean for figures) and the criterion for statistical significance was $p < 0.05$, two-tailed.
3 Results

3.1 Preliminary analysis

Average Perceived Stress Scale (PSS) scores were 17.78 (SD = 7.46), which is within the range of a healthy national sample of similar age (Cohen et al., 1983). Average Center for Epidemiologic Studies – Depression (CES-D) score in our sample was 13.26 (SD = 11.43), which is below the cut-off for clinical depression (Radloff, 1977). CES-D and PSS scores did not differ between genders (CES-D: $F_{1,21} = 0.62; p = 0.44$; PSS: $F_{1,21} = 0.05; p = 0.82$) and races (CES-D: $F_{3,21} = 0.37; p = 0.78$; PSS: $F_{3,21} = 0.59; p = 0.62$).

The Childhood Trauma Questionnaire scores (CTQ) and the Rosenberg Self-esteem scores (RSE) did not differ between gender in our sample (CTQ: $F_{1,21} = 0.005, p = 0.95$; RSE: $F_{1,21} = 0.43, p = 0.52$) or races (CTQ: $F_{3,21} = 1.44, p = 0.26$; RSE: $F_{3,21} = 0.67, p = 0.58$). Neither RSE nor CTQ scores were correlated with age, BMI or body fat (RSE: $p$’s $>$ 0.17; CTQ: $p$’s $>$ 0.37).

As expected, RSE and CTQ were both moderately to strongly correlated with PSS and CES-D (RSE and CES-D: $r = -0.64, p < 0.001$; RSE and PSS: $r = -0.57, p = 0.002$; CTQ and CESD: $r = 0.53, p = 0.005$; CTQ and PSS: $r = 0.50, p = 0.008$; CES-D and PSS: $r = 0.76, p < 0.001$).

None of the seven time points for cortisol for the first stress exposure (TSST1) were correlated with age, BMI or body fat (age: $p$’s $>$ 0.22; BMI: $p$’s $>$ 0.13; body fat: $p$’s $>$ 0.09), nor for the second stress exposure (TSST2) (age: $p$’s $>$ 0.21; BMI: $p$’s
> 0.11; body fat: p’s >0.07). Delta cortisol for the first stress exposure were not correlated with CESD (p = 0.54) or with PSS (p = 0.68) although they were for the second stress exposure (CES-D: r = 0.39, p = 0.04) and trending a significant correlation with PSS: r = 0.36, p = .06.

3.2 Effects of stress induction on delta cortisol

A repeated measures ANOVA revealed a significant main effect of time (TSST1: \(F_{1,37} = 14.16; p < 0.001\); TSST2: \(F_{2.35} = 9.03; p < 0.001\)) in delta cortisol immediately before (S1) and after the TSST (S2-S6) indicating, as expected, a cortisol response to the stress induction (see figure 1). A time by day interaction was also observed (\(F_{1.82, 46.73} = 4.41; p=0.02\)) indicating a difference between the stress response to TSST1 and TSST2. A paired samples t-test revealed a significant decrease in cortisol from TSST1 (M=6.14, SD=9.97) to TSST2 (M=0.80, SD= 6.38), \(t_{(26)} = 2.37, p = 0.03\) indicating habituation of the stress response (see also figure 1).

There was no significant effect of gender as a between subjects factor on overall cortisol for TSST1 (\(F_{1,25} = 0.006; p = 0.94\)), TSST2 (\(F_{1,25} = 1.31; p = 0.26\)) between TSST1 and TSST2 (gender by day interaction: \(F_{1,37, 34.24} = 0.80; p=0.38\)) nor with cortisol changes over time (gender by time interaction: TSST1 (\(F_{1,37, 34.24} = 0.70; p=0.45\)) TSST2 (\(F_{2.35, 58.75} = 0.31; p=0.77\)).

3.3 Self-report measures: sample characteristics

As shown in table 1, the means of each of the subscales for the Childhood Trauma Questionnaire were below the cutoff scores for clinically significant trauma (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997). Among the five subscales, emotional neglect had the highest mean (M = 10.19, SD = 3.67) followed by
emotional abuse (M = 8.59, SD = 3.72). Overall, the participants in the present study had very low levels of physical and sexual abuse, and physical neglect (physical abuse: M = 6.04, SD = 1.8; sexual abuse: M = 5.38, SD = 1.55; physical neglect: M = 6.11, SD = 2.12) considering the lowest score on each subscale is 5 and the highest score is 25.

The mean score on the Rosenberg Self-esteem Scale was just below the cutoff for low self-esteem (Isomaa, Vaananen, Frojd, Kaltiala-Heino, & Marttunen, 2012), (M = 23, SD = 6.38), indicating that a majority of the participants had borderline low self-esteem. The highest possible score on the Rosenberg Self-esteem scale is 40.

3.4 Childhood trauma and cortisol

We first examined the hypothesis that childhood trauma would predict a greater cortisol stress response to the first stress exposure (TSST1) and the second stress exposure (TSST2). A correlation analysis revealed a positive, non-significant relation between childhood trauma and delta cortisol for the first stress exposure (r = 0.05, p = 0.82) and a significant positive relation for the second stress exposure (r = 0.53, p = 0.004) (see figure 2). To further examine the relationship for the second exposure, we created a regression model with childhood trauma as the predictor variable and delta cortisol for the second stress exposure as the outcome variable. As expected, higher childhood trauma significantly predicted greater delta cortisol, β = 0.53, t(25) = 3.13, p = 0.004. Childhood trauma also explained a significant proportion of variance in delta cortisol, adjusted $R^2 = 0.25$, $F(1, 25) = 9.77$, $p = 0.004$.

To ensure that this relationship was not driven by BMI or gender, we entered these variables as covariates in a second step. When controlling for BMI and gender,
childhood trauma remained a significant predictor of delta cortisol for the second stress exposure, $\beta = 0.54$, $t(23) = 3.06$, $p = .0006$. BMI was not a significant predictor $\beta = -0.09$, $t(23) = -0.48$, $p = 0.64$ and gender was not a significant predictor $\beta = -0.12$, $t(23) = -0.69$, $p = 0.49$.

3.5 Childhood trauma and self-esteem

We next examined the hypothesis that higher childhood trauma would predict lower self-esteem. Correlation analysis revealed a negative relation between childhood trauma and self-esteem ($r = -0.55$, $p = 0.003$) (see figure 3). To further examine this relationship, we created a regression model with childhood trauma as the predictor variable and self-esteem as the outcome variable. As expected, higher childhood trauma significantly predicted lower self-esteem, $\beta = -0.55$, $t(25) = -3.26$, $p = 0.003$. Childhood trauma also explained a significant proportion of variance in self-esteem, adjusted $R^2 = 0.27$, $F(1, 25) = 10.62$, $p = 0.003$.

To ensure that this relationship was not driven by BMI or gender, we entered these variables as covariates in a second step. When controlling for BMI and gender, childhood trauma remained a significant predictor of self-esteem, $\beta = -0.57$, $t(23) = -3.34$, $p = 0.003$. BMI was not a significant predictor $\beta = 0.09$, $t(23) = 0.52$, $p = 0.61$ and gender was not a significant predictor $\beta = 0.22$, $t(23) = 1.26$, $p = 0.22$.

3.6 Self-esteem and cortisol

We next examined the hypothesis that lower self-esteem would predict a higher cortisol stress response for the first stress exposure (TSST1) and the second stress exposure (TSST2). Correlation analysis revealed a negative, non-significant relation between self-esteem and delta cortisol for the first stress exposure ($r = -0.23$, $p = 0.12$).
and a significant negative relation for the second stress exposure ($r = -0.71$, $p < 0.001$) (see figure 4). To further examine the relationship for the second exposure, we created a regression model with self-esteem as the predictor variable and delta cortisol as the outcome variable. As expected, lower levels of self-esteem significantly predicted higher delta cortisol for the second exposure, $\beta = -0.71, t(25) = -5.10, p < 0.001$. Self-esteem also explained a significant proportion of variance in delta cortisol, adjusted $R^2 = 0.49, F(1, 25) = 25.97, p < 0.001$.

To ensure that this relationship was not driven by BMI or gender, we entered these variables as covariates in a second step. When controlling for BMI and gender, self-esteem remained a significant predictor of delta cortisol for the second exposure, $\beta = -0.72, t(23) = -4.85, p < 0.001$. BMI was not a significant predictor, $\beta = -0.30, t(23) = -0.21, p = 0.84$, and gender was not a significant predictor, $\beta = 0.05, t(23) = 0.34, p = 0.74$.

3.7 Mediation analysis

To test the hypothesis that the relationship between childhood trauma and cortisol stress response was mediated by self-esteem, we followed the criteria for mediation outlined by Baron and Kenny (Baron & Kenny, 1986). Since we did not find significant correlations between the first stress exposure (TSST1) and self-esteem or childhood trauma, we performed the mediation analysis for the second stress exposure (TSST2) only. BMI and gender were included as covariates for all steps of the mediation analysis.

We previously established that childhood trauma was a significant positive predictor of delta cortisol for the second stress exposure, which satisfied the first
criterion that the independent variable must significantly influence the dependent variable. Satisfying the second criterion, childhood trauma was also a significant negative predictor of self-esteem, the proposed mediator. Satisfying the third and fourth criteria, when childhood trauma and self-esteem were both included as predictors, the relationship between self-esteem and delta cortisol for the second stress exposure remained significant, $\beta = -0.60$, $t(22) = -3.37$, $p = 0.003$, and the relationship between childhood trauma and delta cortisol for the second stress exposure was reduced to non-significance, $\beta = 0.20$, $t(22) = 1.10$, $p = 0.29$ (see figure 5). When controlling for self-esteem, log transformed childhood trauma explained a significant proportion of variance in delta cortisol for the second stress exposure, adjusted $R^2 = 0.46$, $F (4, 22) = 6.42$, $p = 0.001$.

To further determine whether self-esteem mediated the relationship between childhood trauma and the cortisol stress response, bootstrapping analyses were conducted following the guidelines described by Preacher and Hayes (Preacher & Hayes, 2008). Delta cortisol for the second stress exposure was entered as the dependent variable, childhood trauma as the predictor variable, and self-esteem as the proposed mediator in the SPSS macro created by Preacher and Hayes for bootstrap analyses with a single mediator and Sobel test (Preacher & Hayes, 2004). The results indicated that the indirect effect of childhood trauma on delta cortisol for the second stress exposure through self-esteem was significant, with a 95% bootstrap confidence interval of 1.57 to 35.88, $z = 2.38$; $p = 0.02$. Thus, self-esteem fully mediated the relationship between childhood trauma and delta cortisol for the second stress exposure.
3.8 Alternative mediators

We tested several other possible mediators to see if self-esteem was indeed a unique mediator in the relationship between childhood trauma (CTQ) and delta cortisol. Again, this was only done for delta cortisol for the second stress exposure since childhood trauma and self-esteem were not significantly correlated with delta cortisol on the first stress exposure. Due to the high correlations between childhood trauma, depression, perceived stress and self-esteem, we examined whether depression as measured by the CES-D scale or perceived stress as measured by the PSS might mediate this relationship. We also tested anxiety and depression symptoms as assessed by the Hospital Anxiety and Depression Scale (HADS) to see if mediation effects of self-esteem are unique to self-esteem and not confounded with other personality constructs. Finally, we tested each of the revised Midlife Development Inventory (MIDI) personality subscales for mediation since these personality constructs are also related to self-esteem (Trzesniewski, Donnellan, & Robins, 2003).

Our primary goal in this process was to see if any of these measures had an impact on the relationship between childhood trauma and delta cortisol for the second stress exposure by reducing this relationship. To test for mediation, we first examined bivariate correlations between the variables, and then entered these variables into a regression model.

We already established earlier that CTQ and delta cortisol on the second stress exposure are positively correlated ($r = 0.53$, $p = 0.004$), and created a regression with CTQ as the predictor variable and delta cortisol as the outcome variable. As expected, higher CTQ significantly predicted higher delta cortisol, $\beta = .53$, $t(25) = 3.13$, $p =
0.004 (satisfying the first criterion for mediation). As previously mentioned in the preliminary analysis, that CTQ and depression as measured by the CES-D are positively correlated \( r = 0.53, p = 0.005 \). To further examine this relationship, we created a regression model with CTQ as the predictor variable and CES-D as the outcome variable. As expected, higher CTQ significantly predicted a higher CES-D, \( \beta = 0.53, t(25) = 3.12, p = 0.005 \) (satisfying the second criterion). We also established in the preliminary analysis that CES-D and delta cortisol are positively correlated \( r = 0.39, p = 0.04 \). To further examine this relationship, we created a regression model with CES-D as the predictor variable and delta cortisol as the outcome variable. As expected, higher CES-D significantly predicted higher delta cortisol responses, \( \beta = 0.39, t(25) = 2.13, p = 0.04 \) (satisfying the third criterion).

To see whether CES-D reduced the relationship between CTQ and delta cortisol, we repeated the regression predicting the cortisol response from CTQ, adding CES-D as a predictor. Adding CES-D as a predictor did reduce the prediction of CTQ \( (\beta = 0.45, t(24) = 2.22, p = 0.04) \) although CES-D itself ceased to remain a significant predictor \( (\beta = 0.16, t(24) = 0.77, p = 0.45) \).

To test mediation of perceived stress as measured by the PSS, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure \( \beta = 0.53, t(25) = 3.13, p = 0.004 \). As previously mentioned in the preliminary analysis, CTQ and PSS are positively correlated \( r = 0.50, p = 0.008 \). To further examine this relationship, we created a regression model with CTQ as the predictor variable and PSS as the outcome variable. As expected, higher CTQ significantly predicted a higher PSS, \( \beta = \)
0.50, \( t(25) = 2.90, p = 0.008 \) (satisfying the second criterion). We also established in the preliminary analysis that PSS and delta cortisol responses are trending a significant correlation \( r = 0.36, p = 0.06 \). To further examine this relationship, we created a regression model with PSS as the predictor variable and delta cortisol response as the outcome variable. As expected, higher PSS marginally significantly predicted higher delta cortisol responses, \( \beta = 0.36, t(25) = 1.95, p = 0.06 \) (satisfying the third criterion).

To see whether PSS reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding PSS as a predictor. Adding PSS as a predictor did reduce the prediction of CTQ \( (\beta = 0.47, t(24) = 2.34, p = 0.03) \) although PSS itself ceased to remain a significant predictor \( (\beta = 0.13, t(24) = 0.66, p = 0.52) \).

To test mediation of anxiety as measured by the HADS, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure \( \beta = 0.53, t(25) = 3.13, p = 0.004 \). Next, a bivariate correlation revealed a positive relationship between CTQ and anxiety \( (r = 0.47, p = 0.02) \). To further examine this relationship, we created a regression model with CTQ as the predictor variable and anxiety as the outcome variable. As expected, higher CTQ significantly predicted higher anxiety, \( \beta = 0.47, t(25) = 2.62, p = 0.02 \) (satisfying the second criterion). A bivariate correlation also revealed a trending significant relationship between anxiety and the delta cortisol response \( (r = 0.33, p = 0.09) \) To further examine this relationship, we created a regression model with anxiety as the predictor variable and delta cortisol response as
the outcome variable. As expected, higher anxiety marginally predicted higher delta cortisol responses, ($\beta = 0.33, t(25) = 1.74, p = .09$) (satisfying the third criterion).

To see whether anxiety reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding HADS anxiety as a predictor. Adding HADS anxiety as a predictor did reduce the prediction of CTQ ($\beta = 0.48, t(24) = 2.48, p = 0.02$) although anxiety itself ceased to remain a significant predictor ($\beta = 0.11, t(24) = 0.54, p = 0.59$).

To test mediation of depression as measured by the HADS, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = .53, t(25) = 3.13, p = 0.004$. Next, a bivariate correlation revealed a positive relationship between CTQ and depression ($r = 0.49, p = 0.009$). To further examine this relationship, we created a regression model with CTQ as the predictor variable and depression as the outcome variable. As expected, higher CTQ significantly predicted higher depression, $\beta = 0.49, t(25) = 2.83, p = 0.009$ (satisfying the second criterion). A bivariate correlation also revealed a positive relationship between depression and the delta cortisol response ($r = 0.50, p = .008$) To further examine this relationship, we created a regression model with depression as the predictor variable and delta cortisol response as the outcome variable. As expected, higher depression predicted higher delta cortisol responses, ($\beta = 0.50, t(25) = 2.88, p = 0.008$) (satisfying the third criterion).

To see whether depression reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding depression as a predictor. Adding depression as a predictor did reduce the
prediction of CTQ to marginally non-significant ($\beta = 0.38$, $t(24) = 1.99$, $p = 0.06$) although depression itself ceased to remain a significant predictor ($\beta = 0.31$, $t(24) = 1.67$, $p = 0.11$).

To test mediation of neuroticism as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = .53$, $t(25) = 3.13$, $p = 0.004$. Next, a bivariate correlation revealed a positive relationship between CTQ and neuroticism ($r = 0.41$, $p = 0.03$). To further examine this relationship, we created a regression model with CTQ as the predictor variable and neuroticism as the outcome variable. As expected, higher CTQ significantly predicted higher neuroticism, $\beta = 0.41$, $t(25) = 2.24$, $p = 0.03$ (satisfying the second criterion). A bivariate correlation also revealed a positive relationship between neuroticism and the delta cortisol response ($r = 0.46$, $p = .02$) To further examine this relationship, we created a regression model with neuroticism as the predictor variable and delta cortisol response as the outcome variable. As expected, higher neuroticism predicted higher delta cortisol responses, ($\beta = 0.46$, $t(25) = 2.60$, $p = 0.02$) (satisfying the third criterion).

To see whether neuroticism reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding neuroticism as a predictor. Adding neuroticism as a predictor did reduce the prediction of CTQ ($\beta = 0.41$, $t(24) = 2.28$, $p = 0.32$) although neuroticism itself ceased to remain a significant predictor ($\beta = 0.29$, $t(24) = 1.63$, $p = 0.12$).
To test mediation of extroversion as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = .53, t(25) = 3.13, p = 0.004$. Next, a bivariate correlation revealed a negative relationship between CTQ and extroversion ($r = -0.43, p = 0.03$). To further examine this relationship, we created a regression model with CTQ as the predictor variable and extroversion as the outcome variable. As expected, higher CTQ significantly predicted lower extroversion, $\beta = -0.43, t(25) = -2.37, p = 0.03$ (satisfying the second criterion). A bivariate correlation also revealed a negative relationship between extroversion and the delta cortisol response ($r = -0.49, p = 0.02$) To further examine this relationship, we created a regression model with extroversion as the predictor variable and delta cortisol response as the outcome variable. As expected, higher extroversion predicted lower delta cortisol responses, ($\beta = -0.49, t(25) = -2.80, p = 0.01$) (satisfying the third criterion).

To see whether extroversion reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding extroversion as a predictor. Adding extroversion as a predictor did reduce the prediction of CTQ ($\beta = 0.39, t(24) = 2.18, p = 0.04$) and extroversion itself remained a marginally significant predictor ($\beta = -0.32, t(24) = -1.77, p = 0.09$).

To test mediation of openness to experience as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = .53, t(25) = 3.13, p = 0.004$. Next, a bivariate correlation revealed a negative relationship between
CTQ and openness to experience \( (r = -0.49, p = 0.01) \). To further examine this relationship, we created a regression model with CTQ as the predictor variable and openness to experience as the outcome variable. As expected, higher CTQ significantly predicted lower openness to experience, \( \beta = -0.49, t(25) = -2.79, p = 0.01 \) (satisfying the second criterion). A bivariate correlation also revealed a negative relationship between openness to experience and the delta cortisol response \( (r = -0.47, p = .02) \) To further examine this relationship, we created a regression model with openness to experience as the predictor variable and delta cortisol response as the outcome variable. As expected, higher openness to experience predicted lower delta cortisol responses, \( (\beta = -0.47, t(25) = -2.67, p = .01) \) (satisfying the third criterion).

To see whether openness to experience reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding openness to experience as a predictor. Adding openness to experience as a predictor did reduce the prediction of CTQ \( (\beta = 0.39, t(24) = 2.08, p = 0.05) \) although openness to experience itself was no longer a significant predictor \( (\beta = -0.28, t(24) = -1.47, p = 0.15) \).

To test mediation of conscientiousness as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure \( \beta = .53, t(25) = 3.13, p = 0.004 \). Next, a bivariate correlation revealed a negative relationship between CTQ and conscientiousness \( (r = -0.41, p = 0.04) \). To further examine this relationship, we created a regression model with CTQ as the predictor variable and conscientiousness
as the outcome variable. As expected, higher CTQ significantly predicted lower conscientiousness, $\beta = -0.41$, $t(25) = -2.23$, $p = 0.04$ (satisfying the second criterion). A bivariate correlation revealed a marginally significant relationship between conscientiousness and the delta cortisol response ($r = -0.32$, $p = 0.10$). To further examine this relationship, we created a regression model with conscientiousness as the predictor variable and delta cortisol response as the outcome variable. As expected, higher conscientiousness predicted lower delta cortisol responses, ($\beta = -0.32$, $t(25) = -1.70$, $p = 0.10$) (satisfying the third criterion).

To see whether conscientiousness reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding conscientiousness as a predictor. Adding conscientiousness as a predictor did reduce the prediction of CTQ ($\beta = 0.48$, $t(24) = 2.55$, $p = 0.002$) although conscientiousness itself was no longer a significant predictor ($\beta = -0.13$, $t(24) = -0.68$, $p = 0.50$).

To test mediation of agency as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = 0.53$, $t(25) = 3.13$, $p = 0.004$. Next, a bivariate correlation revealed a non-significant relationship between CTQ and agency ($r = -0.20$, $p = 0.33$). Since this relationship was not significant, no further mediation analysis was performed.

To test mediation of agreeableness as measured by the MIDI, again, the first criterion for mediation has been established, higher CTQ significantly predicted higher delta cortisol responses to the second stress exposure $\beta = 0.53$, $t(25) = 3.13$, $p = 0.004$. Next, a bivariate correlation revealed a non-significant relationship between CTQ and agreeableness ($r = -0.20$, $p = 0.33$). Since this relationship was not significant, no further mediation analysis was performed.
Next, a bivariate correlation revealed a negative relationship between CTQ and agreeableness ($r = -0.38, p = 0.05$). To further examine this relationship, we created a regression model with CTQ as the predictor variable and agreeableness as the outcome variable. As expected, higher CTQ significantly predicted lower agreeableness, $\beta = -0.38, t(24) = -2.04, p = 0.05$ (satisfying the second criterion). A bivariate correlation also revealed a negative relationship between agreeableness and the delta cortisol response ($r = -0.45, p = 0.02$). To further examine this relationship, we created a regression model with agreeableness as the predictor variable and delta cortisol response as the outcome variable. As expected, higher agreeableness predicted lower delta cortisol responses, ($\beta = -0.45, t(24) = -2.46, p = 0.02$) (satisfying the third criterion).

To see whether agreeableness reduced the relationship between CTQ and cortisol, we repeated the regression predicting the cortisol response from CTQ, adding agreeableness as a predictor. Adding agreeableness as a predictor did reduce the prediction of CTQ ($\beta = 0.47, t(23) = 2.66, p = 0.02$) although agreeableness itself was no longer a significant predictor ($\beta = -0.27, t(23) = -1.52, p = 0.14$).
4 Discussion

These results support the hypothesis that childhood trauma scores would predict a greater increase in the cortisol stress response to the second stress exposure. However, the data fails to support the other half of this hypothesis that this relationship would also emerge for the first exposure to stress. More specifically, in our sample of healthy young college students, higher childhood trauma scores predicted higher cortisol stress responses to the second, but not the first, exposure to the Trier Social Stress Test (TSST). These results indicate that even people who have grown up in relatively low levels of trauma will exhibit a higher cortisol stress response when exposed to repeated psychosocial stress.

The second hypothesis that childhood trauma scores would predict self-esteem scores was supported by the data. In our sample, higher childhood trauma scores predicted lower self-esteem scores. These results support the idea that people who have grown up in abusive environments tend to have lower self-esteem in their young adult years due to their traumatic experiences in childhood, replicating prior research.

The present study findings support the third hypothesis that self-esteem would predict cortisol responses to the second stress exposure. Again, however, the data failed to support the other half of the hypothesis that this relationship would occur for the first stress exposure. More specifically in our sample of healthy, young college students, low self-esteem scores predicted higher cortisol stress responses upon repeated, but not a single, exposure to the TSST. These results suggest that people
who have low self-esteem may find repeated psychosocial stress more threatening and thus respond with higher spikes in cortisol.

Finally, these results support our hypothesis that self-esteem would mediate the relationship between childhood trauma scores and the cortisol stress response. Again, though, these results only emerged upon repeated exposure to stress (TSST2), not the first exposure to stress. When we controlled for the variation in self-esteem, the relationship between childhood trauma and the cortisol response to the second stress exposure became non-significant. This result indicates that the driving force behind higher cortisol responses to repeated, but not single, instances of psychosocial stress in people who have had abusive childhoods is their current level of self-esteem.

Taken together, these results reveal that although childhood trauma in itself can have lasting effects on how the body handles stress, self-esteem, learned through these childhood experiences, can act as a powerful pathway that prolongs its destructive impact. These results hold true regardless of characteristics such as gender and body mass index. Cortisol responses to the first and second stress exposure were also not associated with traits such as age, BMI and body fat.

We further tested the specificity of self-esteem as a mediator in the relationship between childhood trauma and the cortisol response to the second stress exposure. We examined current levels of stress, depression, anxiety and personality measures. All of these measures (except agency) reduced the relationship between childhood trauma and the cortisol response to the second stress exposure, each exhibiting a minor role in this relationship. However, none of these measures were candidates for mediation as they did not satisfy all of the mediation criterion outlined
by Baron and Kenney (Baron & Kenny, 1986). These results suggest both a strong relationship between childhood trauma and the cortisol response to repeated stress exposure, and a full mediation effect of self-esteem.

Our findings that a relationship with cortisol and personality measures (self-esteem) were found on the second but not the first exposure to stress are in line with the results of a study conducted by Kirschbaum et al., 1995 that found that repeated stress exposure seemed to correlate better with personality measures than a single stress exposure (Kirschbaum et al., 1995). Other studies also did not find associations between subclinical levels of depression (Schommer, 2003) and slightly marginally significant associations with vital exhaustion (Kudielka et al., 2006) and cortisol responses to repeated psychosocial stress.

Also, overall, our findings that higher childhood trauma scores were associated with higher cortisol stress response to psychosocial stress is consistent with some of the literature (Heim et al., 2000), but as discussed earlier, not with other studies (Lovallo et al., 2012). Although the fact that childhood trauma only exhibited a relationship with the second and not initial exposure to stress in the present study is inconsistent with the study done by Engert et al, 2010, who found this relationship on both days of the stress test. As mentioned earlier, though, Engert et al. assessed varying levels of maternal care; the present study assessed childhood trauma (Engert et al., 2010). Although it may be possible that childhood trauma could incorporate low maternal care, the reverse is unlikely. Comparisons between studies using different cortisol measurements such as basal levels (van der Vegt et al., 2010) and the present study that assessed cortisol reactivity to a psychosocial stressor, should be
made with caution. Although van der Vegt et al. found lower basal cortisol in
formerly maltreated adoptees compared to non-maltreated adoptees, it cannot be
assumed that these subjects would respond to psychosocial stress also with a blunted
cortisol response.

Similarly, our finding that higher childhood trauma scores are related to lower
levels of self-esteem is consistent with the majority of the literature (Finzi-Dottan &
Karu, 2006; Lopez & Heffer, 1998; Schuck & Widom, 2001), although the present
study is distinct from other studies in a number of ways. For example, the majority of
participants in the present study had very low levels of physical and sexual abuse, and
physical neglect. Caution should be used when making comparisons between studies
such as Schuck and Widom (2001) and Lopez and Heffer (1998) whose subjects
experienced moderate to high levels of physical and sexual abuse. Results for the
present study are consistent with results from Finzi-Dottan (2006) in that those who
had experienced emotional abuse had lower self-esteem than those without this type
of abuse. However, it should be noted that several of these studies used a non-
maltreated control group whereas the present study did not have this condition.

The association between low self-esteem and high cortisol responses to a
stressor found in the present study is also in line with previous studies (Gruenewald et
al., 2004; Kirschbaum et al., 1995; Pruessner et al., 2004). The present findings that
self-esteem is a full mediator of the relationship between childhood trauma and the
cortisol response to the second stress exposure is largely in line with the study by
Cicchetti and Rogosch (2009) that found a moderating effect of self-esteem (Cicchetti
& Rogosch, 2009). This finding was enhanced by our quest to find alternative
mediators. Although all of the personality factors (with the exception of agency) had an impact on the relationship between childhood trauma and the cortisol stress response, self-esteem was able to fully mediate this relationship.

It was an unexpected finding (and contrary to our hypothesis) that the relationship between self-esteem and the cortisol response, as well as childhood trauma and the cortisol response, to the first stress exposure did not reach significance. A handful of studies have also not found a relationship between personality measures and cortisol responses (Kirschbaum, Bartussek, Strasburger, 1992; Kirschbaum, 1995; Schommer, 2003). One possibility for why these relationships ceased to exist for the first stress exposure could be that the variability in the stress response was exceedingly high due to the confounding effect of novelty and unpredictability (Kirschbaum, 1995). The second stress exposure does not contain the novelty and uncertainty inherent in the first stress exposure, and may therefore be able to produce a more pure response to the psychosocial stress situation.

There are several possible mechanisms by which childhood trauma may affect physiological responses to acute psychosocial stress later in life. The present study found current level of self-esteem to be one of those mechanisms. One body of literature suggests that individuals who grew up in abusive homes may have learned a “negative cognitive style” (Abramson, Metalsky, & Alloy, 1989) that sets the stage for developing low self-esteem. Persons with a “negative cognitive style” may attribute the occurrence of negative circumstances (maltreatment) to some factor of their personality or character (feelings of worthlessness) (Alloy et al., 2004; Sachs-Ericsson et al., 2010; Seligman, Abramson, Semmel & von Baeyer, 1979).
For example, as a way of coping with the reality of being abused by caretakers, victims may have blamed themselves for the abuse and think that they deserved the abuse because they think they are worthless, which in turn leads to judgments about the self that are critical and demeaning, and which hold little value for the self (low self-esteem).

It is possible that people who have a negative cognitive style are also making negative appraisals to ambiguous stress situations (such as the Trier Social Stress Test). It has been hypothesized that children who have grown up in stressful and unpredictable environments may view the world as a threatening place, and are more inclined to appraise ambiguous situations as threats rather than as challenges (Chen & Matthews, 2003). Heightened threat appraisal (furnished by the amygdala mentioned earlier) may then lead to both a greater physiological reactivity to psychosocial stress situations such as the TSST, as well as more frequent elicitation of this response. These activities may be rather benign in the short run, but over time, may culminate in the development of risk factors for numerous health conditions. For example, as mentioned earlier, frequent activation of the cardiovascular system can lead to hypertension and atherosclerosis (McEwen, 2008) due to unregulated inflammatory response to arterial wall injuries (Libby & Therous, 2005; Ross, 1999). Activation of the stress response also inhibits digestion; frequent activation of this system may lead to gastrointestinal problems (Bose et al., 2009).

As the results of the present study suggest, low levels of childhood abuse are important to consider because even when the level of trauma is not severe, individuals may still respond to stress with maladaptive physiological patterns. This may be
because the negative experiences are powerful enough to produce an underlying physiological pathology that has not yet surfaced at the clinical level – meaning the experiences were not extreme enough to create documented physical or mental illness, but they are harmful enough to have some kind of negative effect on the body. This negative affect on bodily systems is speculated to start in early childhood and continue through adolescence and adulthood (Lupien et al., 2009). As noted earlier, it is not clear whether maltreatment in childhood results in a hypo or a hyper secretion of cortisol, both in basal concentrations and in response to acute stress. Since the present study found elevated concentrations of cortisol in response to acute stress, speculation on potential mechanisms will favor this direction.

A recent article posits the Biological Embedding of Childhood Adversity model to explain how trauma experienced in childhood impacts physiological processes during that time and in ensuing years, and may eventually lead to detrimental health effects later in life (Miller et al., 2011). An important assertion of the model is that early life stress gets “embedded” in the cells through epigenetic processes thereby preprogramming the HPA axis and inflammatory processes early in life. Ideally, a negative feedback system allows cytokines such as IL-1β to trigger the HPA axis to secrete cortisol, which then binds to glucocorticoid receptors located within macrophages and other immune cells to slow down the inflammatory process.

However, sensitivity, or lack thereof, to this inhibitory effect is programmed into the immune cells genetic material through early exposure to stress. The article cites a study by Miller and Chen (2010) that found a positive association between participants who reported growing up in a harsh family environment and
desensitization of the anti-inflammatory property of cortisol during a follow-up period. So even though the participants in the present study had heightened cortisol responses to stress, this does not mean that their increased level of cortisol would necessarily be able to appropriately inhibit inflammation that is associated with disease related processes. As mentioned earlier, chronic inflammation may play a key role in the development of Type 2 diabetes (Bertoni, 2010; Pradhan et al., 2001; Sesso, 2003; Wang, 2007; Dandon, 2005). Under suppression of the immune system has also been associated with autoimmune diseases (Sergerstrom, 2004; Wolf, 2008).

The “embedded model” also speculates that early life stress promotes hyper vigilance for threat, mistrust of others and poorer social relationships (Miller et al., 2011). This is thought to be accomplished through early life sculpting of corticolimbic structures such as the amygdala that processes threat appraisals. The amygdala sends signals to hypothalamic complexes that regulate outflow from the SNS and HPA axis, as well as other neuroendocrine routes. Consequently, the amygdala has a powerful influence over the hormonal environment of inflammatory processes. Hyper vigilance, mistrust of others and poor social interactions learned from early adversity also fits in well with the negative cognitive style mentioned earlier. It suggests another avenue for developing low self-esteem in addition to the negative cognitions of blaming oneself for the abuse. If people who have grown up in an abusive environment develop a negative cognitive style, they may be more likely to also be mistrustful and hyper vigilant of others, which may lead to less positive social interactions and low self-esteem. Future studies investigating connections
between self-esteem, negative cognitive style and physiological responses to stress are needed to more accurately understand these processes.

Two basic assumptions need to be made about the present study in order to draw realistic implications from the findings. One is that participant’s responses to the TSST will mimic real life instances of acute, repeated psychosocial stress for them. Another assumption is that increased cortisol responses to psychosocial stress are indeed maladaptive and can lead to risk factors for health problems. If these assumptions are correct, then the study’s finding that self-esteem can help explain hyper secretion of cortisol in response to psychosocial challenges has important implications for therapeutic interventions and preventative measures. Programs for at risk children and adolescents might benefit from incorporating a focus on increasing self-esteem. Therapeutic techniques that are specifically designed to address self-esteem issues can also help adults who have been raised in abusive environments.

The study’s other major finding that instances of sub-clinical levels of trauma can have maladaptive later life stress response patterns speaks more to the general population, since it is probable that many more people suffer sub-clinical levels of childhood abuse rather than severe abuse. Trauma does not have to be severe in order for it to have detrimental physical and psychological impacts. However, with the right therapeutic approach, including a focus on self-esteem, it may be possible to reverse or prevent some of the ensuing health problems.

The results of the present study should be interpreted keeping some limitations in mind. One limitation is that our assessment of childhood trauma was based on self-report and retrospective in nature. However, the scale has very good
internal consistency and validity, and has been widely used in both clinical and
general populations. To more accurately assess dysfunctional family environments
that may be similar to sub-clinical levels of trauma, a questionnaire such as the Risky
Families Questionnaire would be preferable over the Childhood Trauma
Questionnaire used in the present study. Another limitation is that we were not able to
split up the abuse categories, due to small sample sizes in physical neglect and abuse
and sexual abuse, which would have made comparisons between the present study
and others more meaningful. Similarly, the amount of subjects reporting an absence
of abuse was too small to comprise a control group, limiting our ability to compare
non-abused and abused participants as was done in other studies.

Future studies may want to include either a larger sample size with increased
variability in childhood trauma sub categories or a questionnaire that specifically
assesses dysfunctional family environments. Also, although we tested several valid
alternative mediators and found self-esteem to produce full mediation, it should be
noted that there are other possible mediators or factors influencing the relationship
between childhood trauma and the cortisol stress response that we did not test.

Finally, the sample used in this study was unusually healthy; interpretation of the
results to the general population should be made bearing this in mind.

In conclusion, the present study sought to investigate the relationship between
childhood trauma and the cortisol stress response later in life. We showed that people
who have had even low levels of trauma respond to psychosocial challenges with
increased cortisol, which may have consequences for future health problems. These
results show that even lower levels of trauma should be taken seriously because of
their potential effects on health. We were also able to show that current self-esteem levels are related to childhood trauma and help explain the relationship between trauma and cortisol responses. This finding may help inform therapies that aim to help people who have had abusive childhoods by including a focus on increasing self-esteem levels.
5 Tables and Figures

Table 1

Means, standard deviations and range of Childhood Trauma Questionnaire (CTQ) scores by subscale.

<table>
<thead>
<tr>
<th>CTQ Subscale</th>
<th>Min.</th>
<th>Max.</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotional abuse</td>
<td>5</td>
<td>19</td>
<td>8.59</td>
<td>3.72</td>
</tr>
<tr>
<td>Physical abuse</td>
<td>5</td>
<td>13</td>
<td>6.04</td>
<td>1.79</td>
</tr>
<tr>
<td>Sexual abuse</td>
<td>5</td>
<td>13</td>
<td>5.37</td>
<td>1.55</td>
</tr>
<tr>
<td>Emotional neglect</td>
<td>5</td>
<td>19</td>
<td>10.19</td>
<td>3.67</td>
</tr>
<tr>
<td>Physical neglect</td>
<td>5</td>
<td>15</td>
<td>6.11</td>
<td>2.12</td>
</tr>
</tbody>
</table>
Figure 1. Mean cortisol concentrations for day one (TSST1) and day two (TSST2) of the stress exposure. Shaded area indicates when the TSST was administered.

Figure 2. Scatterplot of Childhood Trauma scores and delta cortisol for the second stress exposure (TSST2).

Figure 3. Scatterplot of Rosenberg Self-esteem scores and Childhood Trauma scores.

Figure 4. Scatterplot of Rosenberg Self-esteem scores and delta cortisol for the second stress exposure (TSST2).
Figure 5. Mediation of the relationship between childhood trauma (CTQ) and the cortisol stress response (delta cortisol) by self-esteem. $p \leq 0.01, p \leq 0.001$ (after controlling for self-esteem).
6 References


