EFFECTS OF ACUTE AND CHRONIC STRESS ON ATTENTION AND PSYCHOBIOLOGICAL STRESS REACTIVITY IN WOMEN

By

Charissa Andreotti

Dissertation

Submitted to the Faculty of the

Graduate School of Vanderbilt University

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

in

Psychology

August, 2013

Nashville, Tennessee

Approved:

Professor Bruce E. Compas

Professor David Cole

Professor Michael DeBaun

Professor Bunmi Olatunji

Professor Sohee Park

ACKNOWLEDGMENTS

This research was generously supported by a National Science Foundation Graduate Research Fellowship and a gift from Patricia and Rodes Hart.

I would especially like to thank Dr. Bruce Compas, my advisor and mentor. I am unendingly grateful for his dedication to helping me develop as a scholar and scientist over the past four years. I feel extremely honored to be a member of the Stress and Coping Lab and to have his guidance and support as I continue my education and training. I am extremely privileged to have had the opportunity to work with him.

This work would not have been possible without the Stress and Attention Project Team, whose endless hard work made the realization of this project possible. Paige Garrard, Sneha Venkatraman, and Kelly Watson were instrumental to the collection of data by serving as excellent Noisy Neighbors, saliva collectors, and neurocognitive testers. I would also like to thank the entire Stress and Coping Lab for their support and friendship.

I am extremely grateful to Dr. Linda Luecken for providing her guidance on the development and execution of this project and Dr. Sohee Park for serving as a member of my committee for the past four years and providing constant support, encouragement, and new ideas.

To the members of my many milestone committees, Professors David Cole, Michael DeBaun, Brad Folley, Steve Hollon, Louis Muglia, Bunmi Olatunji, and Sohee Park, thank you for your generous donation of time in reading my work and enthusiastically serving at my meetings.

Finally, I am grateful to the support of my family and friends for their patience, love, compassion, and inspiration.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

A large body of research has provided evidence for a causal relationship between stress and disease. Within this vast literature, at least two independent lines of research have been important in understanding the effects of stress on both mental and physical health. First, biases in attention to threatening stimuli are thought to be based in the evolutionary recognition of natural environmental threats (Ohman, Flykt, & Esteves, 2001). And second, biological and physiological processes of stress reactivity have been tied to the "fight-or-flight" response involved in aiding humans' ability to overcome or avoid environmental stressors. These include the activation of both the hypothalamic-pituitary-adrenal (HPA) axis, leading to release of neuroendocrine hormones such as cortisol, and the sympathetic-adrenal-medullary (SAM) axis, leading to heart rate and other autonomic changes.

Past research has found attentional biases and stress reactivity to be altered in individuals suffering from various forms of psychopathology, including depression and anxiety, as well as those exposed to chronic life stress. For example, anxious individuals as well as children subjected to abuse exhibit altered attentional processing of socially threatening stimuli during tasks of attentional bias (e.g., Bar-Heim et al., 2007; Mogg et al., 2004; Pine et al., 2005). In addition, individuals with diagnosed affective disorders as well as those exposed to low socioeconomic status, a significant chronic life stressor, have altered biological responses, including heart rate, galvanic skin response, immunologic, and salivary cortisol responses, when presented with a laboratory-based stressor (e.g., Kirschbaum et al., 1993; Pace et al., 2006).

The use of coping and emotion-regulation strategies is an important factor in psychological and health-related outcomes in chronically stressed populations. For example, secondary control coping strategies (i.e., efforts to adapt to rather than change sources of stress) are linked to decreased depressive symptoms and physiological arousal resulting from uncontrollable stress (Compas et al., 2006). While stress is frequently related to interruptions in attentional, biological, and emotional processes, the specific means through which chronic life stress during development, including exposure to high levels of family conflict during childhood, may contribute to poorer psychological and health related outcomes remains poorly understood.

Responses to stress and the physical and emotional consequences of stress may be further modified by gender. For example, considerable gender differences exist in the prevalence of diagnosed affective disorders, with women having a significantly greater risk of affective psychopathology compared to men (Kessler et al., 2005). Further, women's rates of cardiovascular disease, a health issue strongly linked to stress, have steadily climbed to rival the high rates traditionally observed in men (American Heart Association, 2010). Although physiological stress reactivity patterns related to disease have been found to vary by gender (Kirschbaum, Wust, & Hellhammer, 1992; Low, Matthews, Kuller, & Edmundowicz, 2011), little research has looked specifically at the potential cognitive and biological mechanisms linking chronic stress and women's disease risk.

The purpose of the proposed study is to investigate the possible role of chronic stress in alterations in automatic attentional and stress reactivity processes that may affect vulnerability to stress in women. Based on a methodology developed by Luecken and Appelhans (2006), healthy undergraduate participants were classified based on their past exposure to family conflict during childhood. Participants were randomized in a crossover design to complete both a mild

laboratory social stress task and a computerized task assessing attentional bias to socially threatening words. A neuroendocrine marker of HPA activation (salivary cortisol) was measured continuously throughout the study in order to examine how attentional processes may be related to altered biological stress reactivity profiles that may ultimately underlie illness variability. In addition, heart rate was measured during three discreet experimental periods (i.e., during a baseline period, during the acute stress task, and during the attentional bias task) as an indicator of SAM axis activation and a more general marker of stress reactivity. The use of coping and emotion regulation strategies as well as current levels of distress was measured using well-validated measures of these constructs (Achenbach & Rescorla, 2002; Connor-Smith et al., 2001; Gross & John, 2003). Findings from this study provide further information about attentional mechanisms through which chronic life stress during development may contribute to long-term alterations in physiological stress responses and decreased use of adaptive coping strategies that increase the risk of poorer mental and physical health outcomes in young women.

Impact of Stress During Development and Allostatic Load

Prior theory and research suggests that characteristics of the family environment during development may contribute to vulnerability to problems in adulthood in domains of both mental and physical health. For example, exposure to chronic life stress has been linked to increased risks of mood disorders (Keller, Neale, & Kendler, 2007; Kendler, Karkowski, & Prescott, 1999) and cardiovascular disease (Black & Garbutt, 2002). While the exact mechanisms underlying these relationships remain unclear, Luecken et al. (2006) present a combined cognitive-affective model to link characteristics of the family environment during development to alterations in psychological and physiological stress reactivity processes in adulthood, which may ultimately

underlie illness vulnerability (see also Repetti, Robles, & Bridget, 2011; Repetti, Taylor, & Seeman, 2002). According to this model, prolonged exposure to psychosocial stressors during development, such as moderate levels of family conflict, have been found to impact an individual's ability to psychologically and biologically respond adaptively to acute everyday stressors through effects on coping, emotion regulation, and physiological arousal, subsequently increasing an individual's disease risk (Luecken et al., 2006).

Heightened allostatic load during childhood and adolescence is one biological mechanism that may elucidate a causal link between chronic stress exposure and subsequent altered acute stress reactivity (e.g., Flier, Underhill, & McEwen, 1998; Juster, McEwen, & Lupien, 2010; McEwen, 2008; Repetti et al., 2011). Allostatic load is defined as "the wear and tear that results from chronic overactivity or underactivity of allostatic systems," including the physiological stress response systems, and past research has linked this chronic activation with physical diseases as well as several psychiatric disorders (see Repetti, Taylor, & Seeman, 2002, for review). One hypothesis for the mechanism through which allostatic load affects physical and psychological functioning involves a neuroendocrine feedback loop that is interrupted under conditions of chronic stress (McGowan et al., 2009; Miller & Chen, 2006). Once the biological stress response cascade of the HPA axis has been activated by perception of a stressor, glucocorticoid compounds circulate systemically, preparing the individual to address the stressor in a "fight or flight" response. Certain brain areas, including the prefrontal cortex, hippocampus, and more primitive limbic regions, including the amygdala, maintain a high concentration of corticosteroid receptors that serve to regulate biological stress responses through this hormonal messenger system (Lupien & McEwen, 1997; Sapolsky, Meaney, & McEwen, 1985). Through a negative feedback loop, glucocorticoids bind to these receptors signaling a halting of the cascade

once the stressor has been adequately addressed (Sapolsky et al., 1985). However, while this system is finely tuned for acute, physical stressors that maintain an evolutionary significance to survival, it lacks the capacity to respond effectively to prolonged psychological stressors (McEwen, 2004).

Neurocognitive Effects of Allostatic Load

Research using animal models has provided significant insight into the effects of chronic stress on the functioning of this biological cascade and the resulting downstream negative neurocognitive effects. These studies suggest that if the HPA axis remains activated by repeated and prolonged exposure to stress, the system breaks down, leading to neuronal atrophy in the prefrontal cortex (PFC) and hippocampus (e.g., Isgor et al., 2004; Radley et al., 2005) as well as hippocampal cell death (McEwen, 2000). These specific brain regions play a significant role in higher-level cognitive control abilities, such as selective attention, involving reciprocal interactions between prefrontal inhibitory control processes and sensory encoding by both cortical and subcortical structures (Miller & Cohen, 2001). Through selective attention, for example, prefrontal regions may up-regulate focus on a specific representation or stimulus quality and retain goal-relevant information while avoiding environmental noise and interference (Derryberry & Rothbart, 1997). However, medial regions of the PFC are acutely affected by damage due to allostatic load (Diorio, Viau, & Meaney, 1993; Liston, McEwen, & Casey, 2009), and thus selective attention abilities may be particularly affected by chronic stress exposure. Using a rodent model, Liston et al. (2006) found that chronic restraint stress was associated with changes in dendritic morphology in the orbitofrontal cortex, which was related to deficits in attentional set shifting. While logistics and ethical constraints make rigorous, analogous human

research difficult, a study by Liston et al. (2009) employing modern imaging technology provides evidence of a similar process in humans. In this study, medical students studying for an important exam performed a task of selective attention while undergoing fMRI. High levels of stress were related to disruptions in prefrontal networks that underlie attentional processing as well as diminished behavioral performance on attentional tasks.

Stress, Attention, and Attentional Bias

Stress and attention. While aspects of human attention are largely directed by executive guidance networks from moment to moment as one plans, initiates, and carries out essential tasks of daily living, attention may be seized by unexpected, acute environmental stressors. The attention of most healthy humans, for example, will be drawn towards the sight of a snake in the wild, and a concurrent increase in selective attentional abilities would be adaptive for promoting survival. It is thus not surprising that a growing body of research has found evidence for a link between an individual's experience of acute stress and his/her ability to perform tasks of controlled attention (e.g., Rodrigues, LeDoux, & Sapolsky, 2009). For example, Chajut and Algom (2003) used several types of tasks, including noise and difficult or impossible psychometric tests, to induce mild, acute stress in healthy individuals before presenting them with Stroop measures (Stroop, 1935) of selective and divided attention. Results showed that the induction of stress actually improved attentional abilities on the attention tasks presented.

Once a stressor has been adequately identified and managed, lower levels of automatic attentional engagement related to the threat may be overridden in a top-down fashion by more controlled, higher level processes, resulting in an attentional disengagement. However, with insufficient cognitive control resulting from the allostatic load of chronic stress exposure, higher-

level attentional processes may not sufficiently override automatic attentional capture and enhancement. For instance, adolescents who have experienced the chronic stress of the loss of a parent during early childhood have been shown to be particularly biased in attention towards social evaluations, suggesting a lack of top-down cognitive control and subsequent bias to attend to psychological threats (Luecken & Appelhans, 2005). These patterns of attentional bias are also characteristic of individuals affected by anxiety and depression, specific types of psychopathology often related to stress, who exhibit an attentional bias towards various types of threatening environmental stimuli that go unnoticed by an unaffected individual (e.g., Bar-Haim et al., 2007; MacCleod, Mathews, & Tata, 1986; Mathews & MacLeod, 1994; Ohman, Flykt, & Esteves, 2001). In sum, it may stand to reason that acute stress influences automatic elements of selective attention. However, in some cases, perhaps due to chronic stress-related deficits in cognitive control, attention may be too easily engaged and/or become resistant to disengagement, resulting in an attentional bias to environmental threat that provides the basis for symptoms of affective psychopathology.

Stress biology and attention. While increased attentional abilities are congruent with an evolutionarily adaptive response to prepare oneself for "fight or flight," it is also plausible that additional psychobiological or neuroendocrine processes may partly explain these findings. A surge in cortisol released as part of an acute stress response may stimulate medial regions of the PFC responsible for selective attention through binding to prefrontal receptors (Lupien & McEwen, 1997). Relatively few studies have examined the association between glucocorticoid patterns and general selective attention. The existing literature is inconclusive with regards to the relationship between cortisol and attentional control with several studies generating equivocal findings (e.g., Born et al., 1987; 1991; Hinkelmann et al., 2009; Kopell et al., 1970;

Schmidt et al., 1999; Skosnik et al., 2000; Vedhara et al., 2000; Wolkowitz et al., 1990). For example, Skosnik and colleagues (2000) detected deficiencies in the inhibition of attention towards non-relevant information related to higher cortisol levels. In this study, healthy participants completed an attentional priming task broken up by a stressful video game task intended to elicit a cortisol reaction. Although cortisol levels were not significantly affected by the stressful task used in this study, reaction time and priming measures of attention were significantly negatively correlated with cortisol levels. Decreases in several domains of executive functioning and variability in cortisol levels and reactivity are commonly associated with major depressive disorder. To test this association, Hinkelmann et al. (2009) measured diurnal cortisol patterns and executive functioning in both depressed and healthy individuals. This study found a significant negative relationship between diurnal cortisol secretion and selective attention in depressed individuals, suggesting that cortisol may be an important factor in the variability of attention and other cognitive domains.

The potential causal relationship between cortisol and selective attention has been further explored through the manipulation of glucocorticoids through both exogenous administration (Born et al., 1987; 1991; Kopell et al., 1970) and natural HPA responses to stress (Vedhara et al., 2000) and subsequent measurement of attention. For example, Kopell and colleagues (1970) found a decreased pattern of average-evoked-potential amplitudes thought to reflect selective attention after exogenous cortisol administration. These results were replicated by Born and colleagues (1987; 1991), who showed that exogenous administration of glucocorticoids reduced stimulus processing and selective attention in a dichotic listening paradigm, as measured by specific event-related brain potentials thought to reflect these processes in healthy individuals. In contrast, Vedhara and colleagues (2000) measured several domains of cognitive functioning

including attention during a short-term period of acute exam stress in students. During the acute stress pre-exam period, a significant reduction in cortisol levels was associated with deficits on tests of selective attention.

While the findings Vedarha et al. (2000) may be considered inconsistent with current conceptualizations of HPA axis reactivity to acute social stress, they may represent the complexity of this biological system, especially when measured in less controlled, non-laboratory paradigms. Since the study by Vedarha et al. examined a naturally occurring setting of a common life stressor (i.e., an academic exam), instead of through the use of a laboratory paradigm, it is more difficult to link diminished attentional abilities directly to a change in overall cortisol levels. It is possible that other biological and social factors (e.g., change in sleep schedule) may play a more direct causal role in the measured cognitive changes than the cortisol levels per se.

When taken together, these laboratory and naturalistic studies provide some evidence that cortisol modulates short-term selective attentional processing. While their results may initially appear incongruous, in a review of the literature, Lupien and McEwen (1997) suggest that a dose-dependent effect of cortisol on selective attention may account for the findings. A study by Hopper and colleagues (2004) lends support to an inverted U-shaped curve reflecting a low to medium-dose enhancement and high-dose impairment of selective attention by glucocorticoid administration. As such, cortisol release through HPA axis response to stress may be one pathway through which acute stress affects selective attention.

Stress and biases in attention: Too much attention, too little cognitive control? As altered attentional processing of threat has been linked to levels of affective symptoms and psychopathology (e.g., Bar-Haim et al., 2007; Bishop, 2009; Clarke, MacLeod, & Shirazee,

2008; Mathews et al., 1990; Mogg et al., 1995), it is important to examine the growing collection of studies that utilize tasks of attentional bias in relation to measures of stress and cortisol. Several recent studies (e.g., Applehans & Luecken, 2006; Ellenbogen et al., 2002, 2006; Ellenbogen, Carson & Pishva, 2010; McHugh et al., 2010; Pilgrim, Marin, & Lupien, 2010; van Honk et al., 2000) indicate a relationship between selective attention towards emotional stimuli and stress-induced variations in levels of cortisol, and may provide the best initial evidence of a relationship between attentional bias to threat and biological stress reactivity.

Here, I review studies in which data were collected on both attentional bias to threat and individual differences in cortisol variability. Relatively few studies have examined levels of *baseline* cortisol in relation to performance on subsequent attentional bias tasks. In one of the few studies to examine this relationship, Van Honk et al. (1998) found that participants in a high cortisol group, as determined by a median split of baseline measurements in healthy volunteers, compared to a low cortisol group displayed an attentional bias away from angry faces on an emotional Stroop task using angry and neutral faces. However, Bakvis, Spinhoven, and Roelofs (2009) failed to replicate these findings and did not find a consistent association between baseline cortisol and attentional bias to emotional stimuli in healthy individuals. This inconsistency in results may underscore the complex and perhaps non-linear relationship (i.e., an inverted U pattern) between cortisol and attentional bias, as has been hypothesized by dose-dependent effects.

Van Honk and colleagues (2000) examined cortisol *reactivity* (i.e., changes in cortisol levels in response to stress) in healthy subjects completing an emotional Stroop task using angry and neutral faces as stimuli. Results indicated that subjects who displayed a bias towards angry faces also had an increase in cortisol levels from pre- to post-task. These results are consistent

with previous work suggesting that activation of the amygdala in response to threat may stimulate stress-related cortisol secretion through its excitatory effects on several components of the HPA axis (e.g., Herman & Cullinan, 1997). In addition, regions of the prefrontal cortex, especially the ventromedial prefrontal cortex, are thought to exhibit top-down control over the amygdala (Zald & Rauch, 2007). As such, the limbic hyperactivation to threat found by Van Honk et al. (2000) provides initial evidence for a possible deficit in top-down prefrontal control of the amygdala that may influence downstream, biological stress responses.

Ellenbogen and colleagues (2002) built on the findings of Van Honk et al. (2000) by adding a laboratory stress task before administering a selective attention paradigm. The stress task used in this study met the criteria subsequently identified by Dickerson and Kemeny (2004) for activating the HPA-axis by creating an uncontrollable, social evaluative threat in the form of a computer game in which the participant would always lose to a confederate. A comparison group of participants in a non-stress condition was able to win the game. No association was found between task condition and cortisol reactivity in this study. However, participants exposed to the negative stress condition were more likely to disengage from threatening stimuli as indicated by directing attention away from negative stimuli on the attentional task. Further, Ellenbogen et al. (2002) found that attentional bias towards negative stimuli was associated with overall higher cortisol levels during recovery. The type of selective attention task chosen in this study may help explain these results. The paradigm was similar to a flanker task, in which stimuli were presented at a conscious level of awareness, and similarly valenced trials were grouped together. Thus, participants may have been able to consciously employ adaptive coping and emotion regulation strategies and successfully orient towards neutral stimuli, as all were healthy, non-depressed, non-anxious individuals.

In a more recent study, Ellenbogen, Carson, and Pishva (2010) embedded an attentional orienting task within a modified version of the Trier Social Stress Test. The attentional task was thus presented after the public speaking component and in the middle of the mental arithmetic component. Cortisol samples were collected at regular intervals beginning during a baseline rest period, through the tasks, and throughout a recovery period after all tasks were completed. In this study, attentional shifting in response to negative stimuli was related to increased cortisol reactivity to the stress task (Ellenbogen et al., 2010). These results point to a significant association between attention and information processing on HPA axis threat reactivity. However, results of this study still do not fully parse the causal direction of this association because the attentional task occurred during the stressful event, making it impossible to identify the directionality of the relationship between the cognitive and physiological processes.

Pilgrim, Marin, and Lupien (2010) administered an adaptation of Posner's attentional orienting task using socially threatening words prior to a modified Trier social stress task. Both attentional bias to social threat and cortisol reactivity in response to the stress task were measured. Faster attentional engagement towards social threat in the orienting paradigm was related to increased cortisol reactivity during the stress task from pre- to post-measures. Finally, McHugh and colleagues (2010) administered a dot probe task as a measure of attentional bias to threat and collected cortisol before and after the completion of a stress task. In this task, a dot probe using socially threatening words presented at the level of conscious awareness was administered prior to and after a frustrating computer task used as a stressor. No significant relationship was found between changes in the cortisol and attentional bias measures pre- to post-stressor. However, consistent with the results of Van Honk et al. (1998), lower baseline levels of cortisol were associated with higher orienting towards threat prior to the stressful task.

In addition, lower cortisol reactivity to the stress task was also associated with increased attentional bias to threat after the stress task.

A study by Appelhans and Luecken (2006) sheds further light on the relationship between attentional threat processing and cortisol by accounting for trait levels of anxiety. In this study, a dot probe detection task using socially threatening words was administered, and cortisol was measured pre- and post-task. While initial levels of anxiety predicted orienting away from socially threatening stimuli, orienting attention away predicted lower cortisol reactivity only in high trait anxious individuals. In addition, orienting attention away was related to higher cortisol reactivity in low trait anxious individuals. This study thus provides evidence for downstream effects of cognitive processing on physiological arousal and responses that may account for symptoms of anxiety disorders.

The results of these studies can be viewed in terms of non-causal relations between attentional processing and HPA activation, as the research designs and measurement techniques used in these studies do not lend themselves to a comprehensive evaluation of possible causal relationships between attentional bias to threat and HPA functioning. Taken together, these studies (Applehans & Luecken, 2006; Ellenbogen et al., 2002, 2006, 2010; McHugh et al., 2010; Pilgrim, Marin, & Lupien, 2010; van Honk et al., 2000) indicate that attentional biases may play a significant role in the maintenance of psychobiological stress processes. The presence of an acute stressor may trigger biases in attention, which in turn contribute to the release of stress hormones that may then further affect attentional processing. To elaborate, while the amygdala and associated limbic structures may be more acutely sensitive to specific environmental threats and events, they are connected through a broad network to both cortical and subcortical regions involved in the regulation of attention that may be subsequently affected. The presentation of an

acute threat may initially trigger the amygdala, but this activation may also help prepare the individual to deal with the threat through downstream activation of several brain structures and regions, including the hypothalamus, stimulating the HPA axis response to stress. The dose-dependent dampening effects of glucocorticoids on prefrontal attention regions may facilitate both an adaptive increase in threat processing, including attentional processes, and a down-regulation in stress reactivity once the stressor has been managed.

Several studies have examined the possible causal link between increased cortisol levels and alterations in attentional bias to threat through the use of both controlled laboratory stress tasks designed to produce an HPA response (e.g., Roelofs et al., 2005; 2007) and the exogenous administration of cortisol (e.g., Putman et al., 2007; 2010; Taylor et al., 2010; van Peer, Spinhoven, & Roelofs, 2010). Roelofs and colleagues (2007) administered a masked emotional Stroop task to healthy participants at rest and after undergoing the Trier Social Stress Test. Using a median split, the authors found that high cortisol responders to the stress test displayed a bias away from threat at rest and a bias towards threat after having undergone the stress test. However, low cortisol responders to the stress test showed no bias in the rest condition but displayed a bias away from threat after the stress test. These findings are similar to those found in an earlier study by Roelofs et al. (2005) in which high and low cortisol responders to a stress task differed in their reactions to an approach-avoidance paradigm in which participants were instructed to conduct arm movements that were either consistent or inconsistent with affect associated with either positive or negative facial stimuli conducted after the stress task. Participants who exhibited high cortisol responsivity to the Trier Social Stress Test also demonstrated a "freezing" reaction, showing increases in reaction times for responses congruent with the presented stimuli. While these findings may not represent a purely attentional change,

but also include a significant motor integration component, they do lend support for an overall change in attentional processing in highly reactive individuals in the context of a stressor.

While there is some evidence that changes in cortisol levels have a causal effect on attentional bias to threat, this relationship may be moderated by trait-like characteristics of individuals that may be similar to anxiety and manifest as cortisol reactivity to stress. Such a hypothesis would be consistent with the results of Ellenbogen et al. (2002) that suggest that depressive symptoms may moderate both attentional processing of threat and cortisol variability in response to an acute laboratory stressor. In the Ellenbogen et al. (2002) study, participants completed a difficult computer game against a confederate, but prior assignment to a positive or negative stress condition determined if they would win or lose. While cortisol did not change significantly in response to the negative stressor (losing), attentional bias to socially threatening stimuli was affected, compared to results of individuals who were able to win the game. However, depression symptoms moderated this effect, with individuals with high depressive symptom levels showing slower disengagement away from negative stimuli. Further, these individuals also showed a blunted cortisol profile, with higher baseline followed by less recovery.

The administration of exogenous cortisol has allowed for more direct exploration of the causal association between cortisol level and attentional bias, as the individual is not subjected to a stressor that may alter the bias through other biological and psychological stress pathways. Putman et al. (2007) conducted the first study that found significantly decreased selective attention to threat on an emotional Stroop paradigm after the exogenous administration of cortisol in healthy volunteers. While these results were most prominent in high-anxious individuals, Putman, Hermans, and van Honk (2010) found similar dampening effects of

exogenously administered cortisol on threat detection, but only in low-anxious individuals. It is noteworthy that the task in the later study employed a spatial orientation paradigm similar to a visual probe, and low-anxious participants performed the task significantly more efficiently, indicating a possible ceiling effect in task performance. Further, Van Peer, Spinhoven, and Roelofs (2010) replicated the dampening effect of exogenous cortisol on attentional bias to threat by examining event-related potentials. In this study employing a Stroop paradigm, the decreasing effect of cortisol on patterns of attentional bias was again most pronounced in individuals with higher levels of social anxiety. Taylor et al. (2010) again replicated these results and also found a dose effect of cortisol on the attenuation of attentional bias.

There is promising evidence to suggest that attentional bias to threat and amygdala activation may be related to psychobiological cascades that regulate human stress responses (e.g., Applehans & Luecken, 2006; Ellenbogen et al., 2002; Pilgrim et al., 2010; Van Honk et al., 2000). Individual differences in expression of prefrontal receptors for stress hormones released as part of the HPA stress reactivity process may play a role in further explaining long-term variability in interrelated patterns of attentional bias, stress reactivity, and affective symptoms. The presence of higher numbers or increased sensitivity of receptors may provide a more effective "brake" to decrease neuroendocrine cascades through the down-regulation of amygdala activity after a stress response has been initiated and run its adaptive course.

Preliminary research has begun to examine the specific genes and polymorphisms that may be implicated in cortisol receptor expression and sensitivity. Wust and colleagues (2004) studied the relation between 3 specific single nucleotide polymorphisms and cortisol reactivity after administration of a Trier Social Stress Test and a dexamethasone suppression test. While no significant results were found as a function of genotype on responses to the dexamethasone

suppression test, genotype was significantly related to cortisol reactivity after the Trier. For example, the presence of specific single nucleotide polymorphisms (363S) was related to increased cortisol reactivity to the acute laboratory stressor. This study suggests a possible genetic basis for individual differences in psychosocial stress reactivity that may be important in the risk for attentional biases and affective disorders. The findings of Wust et al. may be viewed within the context of findings discussed earlier on the relation between attentional bias and cortisol reactivity as well as the causal link between cortisol and decreased sensitivity to threat.

Taken together, these studies suggest the emergence of a negative feedback process in which an acute stressor may adaptively trigger increased attention towards threat-relevant information for a short period that may help facilitate the HPA stress response. Neuroendocrine binding may then regulate the cognitive and HPA response in a dose-dependent fashion, in order to prepare the body to engage in a "fight or flight" response and then effectively turning both off after the stressor has been addressed. However, allostatic load resulting from exposure to chronic stress during development may alter this adaptive feedback loop through damage to prefrontal tissues that modulate limbic activation and desensitization of glucocorticoid receptors.

In such a case as persistent exposure to family conflict, chronic activation of the HPA axis may result in decreased cortical and subcortical glucocorticoid receptor density (McEwen, 2000; Miller & Chen, 2006). As such, self-modulatory capacities of the biological stress response cascades may be reduced, leading to eventual system desensitization and increased levels of circulating glucocorticoids at baseline (Meaney et al., 1996; Mizoguchi, Ishige, Aburada, & Tabira, 2003; Plotsky & Meaney, 1993; Sapolsky, Meaney, & McEwen, 1985). It is predicted that individuals exposed to chronic stress during development would thus have increased difficulty responding to common everyday stressors due to both the decreased

functioning in critical neural regions as well as the desensitization of the neuroendocrine feedback loop (Raison & Miller, 2003). In addition, increased baseline cortisol levels have been linked to greater heart rate reactivity in response to acute social stress (Larson, Ader, & Moynihan, 2001). The resulting decreases in adaptive coping and emotion regulation strategies as well as altered physiological responses due to effects of allostatic load are both related to long-term negative impacts on psychological and physical health characteristic of chronically-stressed populations.

Stress, Emotion Regulation, and Coping

The use of coping and emotion regulation strategies has been found to be an important factor in the psychological and health related outcomes noted in chronically stressed populations. Emotion regulation can be characterized as individuals' efforts to affect the type and timing of their own emotions as well as their personal experience and expression of these internal states (e.g., Gross & Thompson, 2007; Joorman & Gotlib, 2010; Mauss et al., 2007). It consists of the set of processes that allow for the increase, decrease, or maintenance of affective states (Davidson et al., 2000). Conscious, controlled strategies dominate the literature on emotion-regulation and will be the focus of this study. These strategies require effort, and studies have shown that individuals are able to accurately report their own use of such strategies in daily activities (e.g., Gross & Thompson, 2007). Emotion regulation also includes both the up-regulation of positive emotions and the down-regulation of negative emotions (Davidson et al., 2000; Gross, 1998).

Closely related to emotion-regulation, the concept of coping refers to both cognitive and behavioral efforts to manage stress and adversity (Lazarus & Folkman, 1984). The construct has

evolved over time in the psychological literature, and recent conceptualizations view coping as processes of regulation, including the regulation of emotions, in response to stress (e.g., Compas et al., 2001; Eisenberg, 1997). For example, Compas et al. (2001, p. 89) define coping as, "conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances." This definition makes explicit links between coping and processes of regulation, including emotion regulation, and includes the purposeful regulation of emotions in response to stress (Compas, 2009).

Consensus has slowly emerged regarding the various dimensions of coping. Skinner, Edge, Altman, and Sherwood (2003) identified over 400 categories or types of coping that have been represented in research on this construct. Skinner et al. highlight recent theory-based top-down models of coping that capture higher order factors or categories of coping responses. One such model is a three-factor model of coping that has been validated using confirmatory factor analyses in several studies of adolescents and adults (e.g., Benson et al., 2011; Calvete & Connor-Smith; 2005; Compas et al., 2006; Connor-Smith et al., 2000; Wadsworth et al., 2004; Yao et al., 2010). Specifically, coping can be broken down into three domains, each encompassing several subtypes: primary control engagement coping (efforts to act directly on the source of stress or one's emotions, including problem solving, emotional expression, and emotional modulation); secondary control engagement coping (efforts to adapt to the source of stress, including cognitive restructuring, positive thinking, acceptance, and distraction); and disengagement coping (efforts to cognitively or behaviorally withdraw from the source of stress, including wishful thinking, avoidance, and denial).

Although coping and emotion regulation overlap significantly in that both involve volitional efforts to reduce negative emotions associated with stressful experiences and

circumstances, there are salient differences between the fields of emotion regulation and coping. First, in addition to altering emotional reactions to environmental stressors, coping encompasses actions taken to directly act on the environmental source of stress (Compas et al., 2001; 2009; Skinner et al., 2003). Second, whereas coping typically refers to the down-regulation of a negative emotion, emotion regulation also includes the maintenance or augmentation of a positive emotion (Eisenberg, Fabes, & Guthrie, 1997). However, recent research has begun to reconcile this distinction between down-regulating negative emotions and up-regulating positive emotions. For example, Austenfeld and Stanton (2004) have used the term "emotional approach coping," to describe coping that involves acknowledging, expressing, and understanding emotions in response to stressors. Their conceptualization of emotional approach coping provides an alternative to emotion-focused coping, which has been previously associated with poorer psychological and health-related outcomes. In addition, Jaser et al. (2010) and Compas et al. (2011) found that secondary control coping is related to both the down- regulation of negative affect (sadness) and up- regulation of positive affect.

The current study focuses on the use of secondary control coping strategies, which require active engagement with the emotions and cognitions brought on by a stressor. Measurement of the secondary control engagement coping factor includes responses related to cognitive restructuring (efforts to actively reinterpret stressful or negative events in more neutral or positive terms). Cognitive restructuring, as viewed in the context of coping, is similar to the concept of cognitive reappraisal described extensively in the emotion regulation literature as "changing the way one thinks about a potentially emotion-eliciting event" (John & Gross, 2004; p. 1302). Deficits in the use of cognitive reappraisal in response to stress have been tied to significant emotional and behavioral problems in adults, including mood and anxiety (e.g.,

Campbell-Sills & Barlow, 2007; Gross & Levenson, 1997). In addition, deficiency in regulating negative emotions has been linked to depressive symptoms and disorders in children and adolescents (Compas et al., 2009).

The Role of Cognitive Control in Coping and Emotion Regulation

A growing body of research has indicated that cognitive control, and specifically attentional control, is integral to the process of responding to and coping with stress (Compas & Boyer, 2001). This link between attention and coping has been supported by findings from research with several medical populations, including children with recurrent abdominal pain (Boyer et al., 2006; Walker et al., 1997) and adult women with breast cancer (Glinder et al., 2007). For example, results from a study by Walker and colleagues (1997) show that children with recurrent abdominal pain who attend more to pain have increased somatization and pain symptoms. One possible link between attention and the processes of coping and emotion regulation may be the requirement of several executive function abilities that rely heavily on cognitive control to adequately reappraise a stressful situation (Compas, Campbell, Robinson, & Rodriguez, 2009).

Further, studies relying on experimental neuropsychological and neuroimaging techniques have indicated that cognitive reappraisal calls on both selective attention and working memory skills in addition to several other higher-level cognitive functions including inhibition and monitoring of response conflict (Ochsner et al., 2002). Selective attention is necessary to maintain concentration on essential aspects of the stressor without interference from other information that may be emotionally salient, but ultimately irrelevant. Working memory allows for the reframing of the current information in more neutral or positive terms, as the information

being held in mind is manipulated through working memory processes (Baddeley & Hitch, 1974). During this process, ongoing monitoring is necessary to resolve response conflicts that may occur between the top-down reappraisals and the bottom-up processes of emotion generation (Barch et al., 2001). Continuous on-line assessment of one's internal state with respect to the outside environment provides constantly updated information regarding an individual's current emotional state (Paradiso et al., 1999). The interaction of these higher-level PFC functions with other cortical and subcortical emotion-processing regions may thus provide the neural basis for processes integral to emotion regulation (Ochsner et al., 2002).

Adolescence and early adulthood are critical periods for the development of cognitive control (Luna & Sweeney, 2006). During this period, axon pruning and myelination allow for the honing of executive functions as they are applied and practiced in natural settings (Blakemore & Choudhury, 2006). Deficits in neurocognitive functioning, particularly higher order cognitive control processes involved in executive functioning, may thus be related to variability in emotional and interpersonal development during adolescence and early adulthood. As neurocognitive development may provide the basis for the regulation of emotions and coping with stress, impairment of these capabilities may reduce one's ability to utilize adaptive strategies when dealing with stressors (Compas, 2006). As such, deficits in cognitive control may result in impaired social and emotional adjustment during adolescence. For example, in a sample of adolescents, Copeland and Compas (2007) found that deficits in inhibitory control are related to decreased use of problem-solving and emotion-regulation strategies in response to stress. Further, problems with these coping skills mediated the relation between inhibitory control and behavior problems suggesting that cognitive control provides a foundation of skills that can be put into action in response to stress.

Current Study

Although prior theory and research has suggested relationships among chronic stressors during development and alterations in physiological stress response that may contribute to later health problems, little research has tested these relationships in young adulthood, often a period of significant life transitions and increased stress. In addition, attentional bias to threat has been linked to several psychiatric disorders, but few studies have closely examined the development and stability of this cognitive phenomenon and how it may function as a potential mechanism through which the family environment during development may impact long-term psychological and physical health.

The purpose of this study was thus to examine the relationships among attentional bias to threat, stress reactivity profiles, coping/emotion regulation, and cognitive functioning in young adult women. By examining these constructs within the broader context of self-reported past exposure to chronic family conflict, I aimed to assess the extent to which these processes may moderate psychological and physiological outcomes in individuals exposed to chronic stress during development. This study provides further information regarding the long-term significance of the family environment during development and highlights potential points of intervention for the prevention of psychological illness later in life. The specific hypotheses are as follows, and indicated figures illustrate hypotheses as noted.

1. Effects of exposure to acute laboratory stress and high levels of family conflict during development on attentional bias (Figure 1):
    a. Exposure to an interpersonal laboratory stressor will increase bias towards words related to social evaluative threat presented below the level of conscious awareness in the sample overall. See path 1a in Figure 1.

b. Exposure to high levels of family conflict during development will be related to a greater bias towards words related to social evaluative threat presented below the level of conscious awareness. See path 1b in Figure 1.

c. Exposure to high levels of family conflict during development will moderate the relationship between exposure to an interpersonal laboratory stressor and bias towards words related to social evaluative threat. That is, individuals exposed to higher levels of family conflict will display an increased bias to social threat. See path 1c in Figure 1.

2. Effects of exposure to acute laboratory stress and high levels of family conflict during development and attentional bias to threat on stress biology (Figure 1):

   a. Individuals with a greater attentional bias towards socially threatening stimuli presented both below and at the level of conscious awareness will exhibit increased cortisol reactivity (i.e., change from baseline to peak) and heart rate reactivity in response to an interpersonal laboratory stressor. See path 2a in Figure 1.

   b. Exposure to high levels of family conflict during development will be related to altered biological stress reactivity in response to an acute laboratory stressor. Specifically, individuals exposed to high levels of family conflict during development will have a higher baseline level of cortisol, a blunted cortisol reactivity pattern (i.e., smaller change from baseline to peak response due to a ceiling effect) to an interpersonal laboratory stress task, and overall higher levels of cortisol output over the course of the study (i.e., total levels under the curve). Secondarily, individuals exposed to high levels of family conflict during

development will have increased heart rate reactivity to an interpersonal laboratory stress task. See path 2b in Figure 1.

c. Exposure to high levels of family conflict during development will moderate the relation between attentional bias to social threat and biological stress reactivity to acute laboratory stress exposure. Specifically, exposure to high levels of family conflict will be related to decreased cortisol reactivity (i.e., change from baseline to peak response) due to the ceiling effect imposed by high baseline levels of cortisol as well as increased heart rate reactivity. See path 2c in Figure 1.

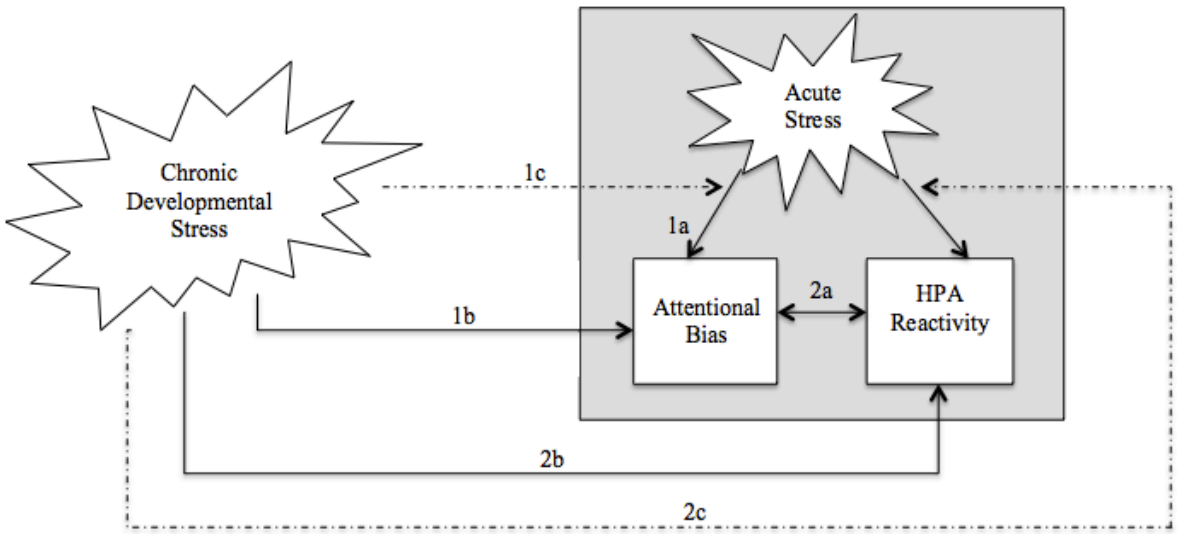

Figure 1. Illustration of Hypotheses 1 and 2

3. Coping, emotion regulation, and neurocognitive as well as psychological sequelae (Figure 2):
    a. Individuals exposed to high levels of family conflict during development will report using lower levels of secondary control coping and cognitive reappraisal emotion regulation strategies with regards to current social stressors compared to individuals without exposure to high levels of family conflict. See path 3a in Figure 2.
    b. Diminished cognitive control, as measured by standardized neurocognitive measures of selective attention and working memory, and attentional bias to words related to social threat presented both below (subliminally) and at the level of conscious awareness (supraliminally) will be negatively related to self-reported use of secondary control coping and cognitive reappraisal emotion regulation strategies and positively related to stress reactivity in response to social stressors. See path 3b in Figure 2.
    c. Individuals exposed to high levels of family conflict during development will show relative deficits in domains of attention and working memory compared to individuals without exposure to high levels of family conflict. See path 3c in Figure 2.
    d. Individuals exposed to high levels of family conflict during development will report higher levels of anxiety and depression symptoms compared to individuals without exposure to high levels of family conflict. See path 3d in Figure 2.

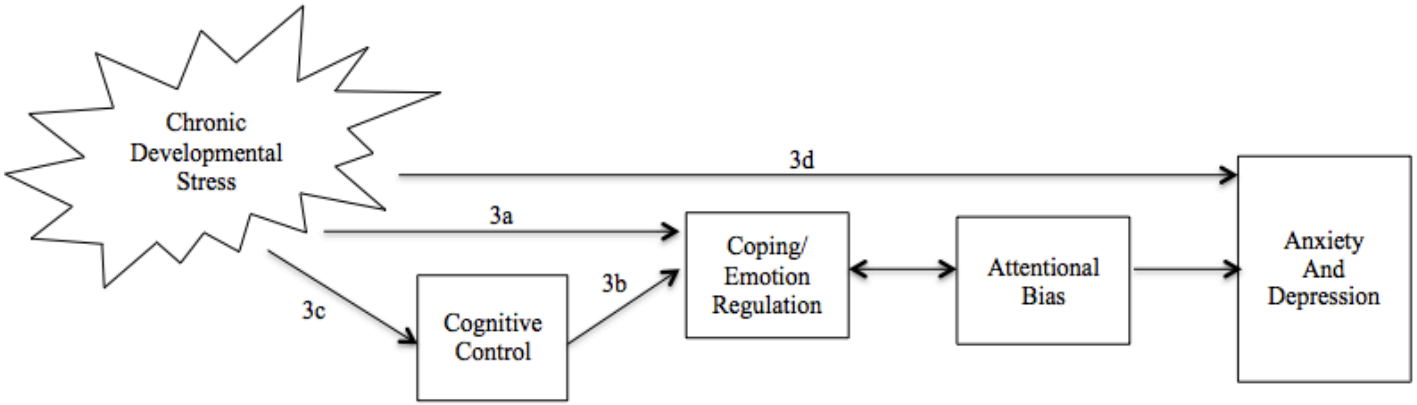

Figure 2. Illustration of Hypothesis 3

Exploratory Analyses*:* Conflict, attentional bias, stress reactivity, and psychological sequelae. See path 4 in Figure 3.

a. Attentional biases towards social threat stimuli presented below the level of conscious awareness will be related to increased levels of depression and anxiety symptoms. This relation will be moderated by exposure to family conflict during development in that increased conflict will lessen the association.
b. Cortisol reactivity will be related to increased levels of depression and anxiety symptoms. This relation will be moderated by exposure to family conflict during development in that increased conflict will lessen the association.

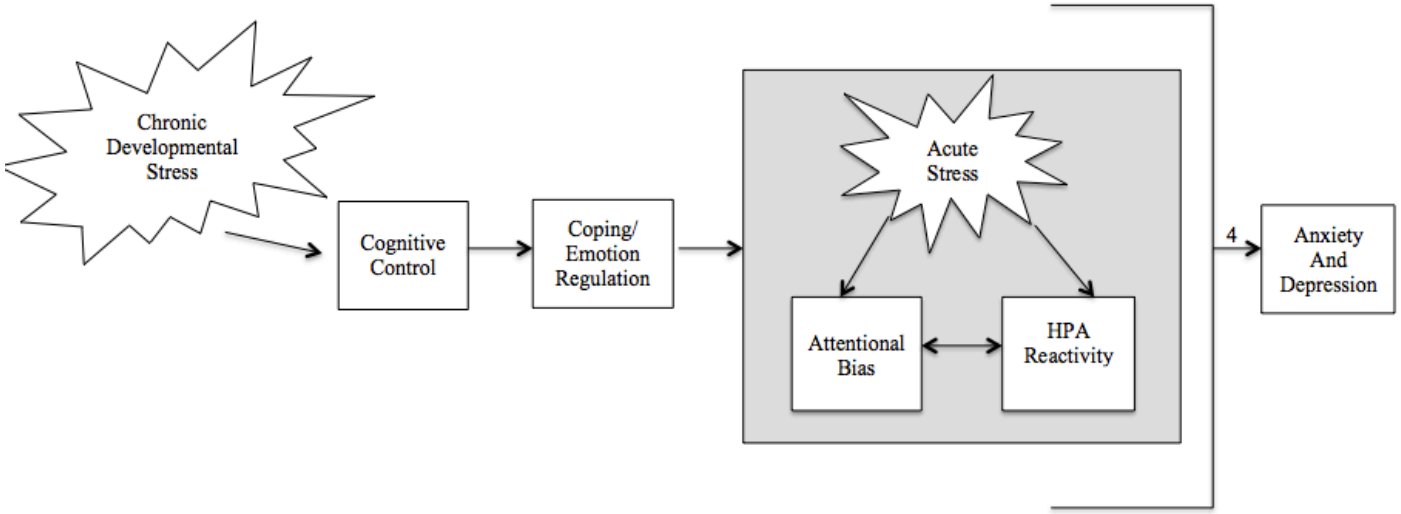

Figure 3. Illustration of exploratory hypotheses

CHAPTER II

METHOD

Participants

Participants for this study include 116 female undergraduate students currently enrolled at Vanderbilt University. The mean age of the sample was 18.96 years (*SD* = 1.13), range 18 to 22 years. The sample was 78.6% Caucasian, 8.5% African American, 6.8% Asian American, 4.3% Hispanic/Latina, and .9% endorsed more than one ethnicity. All participants were recruited through the on-line SONA subject pool management system, which allows students to receive credits for completing on-campus research studies required for many undergraduate behavioral science courses. The only requirement for inclusion in the study was current full-time enrollment status as an undergraduate student at Vanderbilt and self-identified gender as female. The study was approved by the Institutional Review Board at Vanderbilt University.

Measures

Demographics. All participants completed a demographics questionnaire to collect information on family structure, annual family income, parent education level, and non-academic extracurricular or work activities.

Salivary cortisol. Saliva samples were collected at baseline after the participant has been in the laboratory for approximately 25 minutes, *between* the two tasks, immediately after the second task, and at two 15 minute follow-up intervals after the tasks for use in analyses of salivary cortisol (5 samples total). The five data points allow for analyses of both reactivity to stress (as reflected in increases in from pre- to post-stress) and recovery from stress (as reflected in the rate

of decrease in cortisol). To control for diurnal fluctuations in cortisol, all appointments were scheduled for the afternoon (2-6pm). Participants were instructed to refrain from eating, alcohol use, smoking, exercise, or prescription drugs for at least two hours prior to participation.

Salivary cortisol concentrations are independent of flow rate, and reflect unbound "free" levels in plasma. Saliva samples were obtained with the Salivette sampling device (Sarstedt, Rommelsdorf, Germany). Participants were instructed to place a small cotton swab in their mouths and chew on it for 2 minutes. The swabs was immediately frozen and stored at -30 °C for 1-3 months prior to analysis. Saliva samples were frozen and later assayed by the laboratory of Dr. Clemens Kirschbaum (Dresden, Germany). Analyses of cortisol were conducted in duplicate and the mean level of the two tests was used in all analyses.

Stress reactivity task. The Noisy Neighbor Task was used instrumentally as an acute laboratory stressor and as a measure of biological and psychological stress reactivity. This task has been used previously by Luecken and colleagues and it has been found to induce a mild physiological stress response due to the social interaction and feelings of social evaluation induced by the task (Luecken, Kraft, & Hagan, 2009). Stress responses to this task have previously been measured by Luecken and colleagues by self-report as well as biological (i.e. salivary cortisol and heart rate) data. No harmful side effects have been noted. Participants interacted with a gender-matched research assistant. The prompt given to participants instructed them:

> "You are trying to study for an important exam. You really need to do well on this exam, but you can't concentrate because your neighbor is playing his/her music to loud. You decide to ask him/her to turn down his/her music so you can study."

The gender-matched research assistant entered the room, and the participant stood facing the research assistant, approximately 30 inches away. Participants interacted during a conversation with the research assistant for 10 minutes and were videotaped during the role-play. The research assistant responded to the participant with an ordered list of memorized prompts. If the participant stopped responding in the conversation, she was prompted by the research assistant to continue until 10 minutes have elapsed. This conversation continued for approximately 10 minutes. The research assistant utilized the following list of memorized prompts:

"Why"

"Don't you like this music?"

"I like it like this"

"I'll think about it"

"I don't think it's too loud"

"It's my apartment"

"No one else has a problem with it"

"Hey, we're having a party"

"I have my rights too"

"I've never asked you to turn down your music'

"I don't know"

"I don't want to"

"I don't see why I should turn it down"

"This is my favorite song"

"It isn't that loud"

"It's still early"

"It hasn't been playing that long"

"You can study with it on"

"Come on, we won't be playing it that much longer, only a couple of hours"

"Get some ear plugs. I'll be glad to get you some cotton if you don't have any."

“Why does it matter…..(insert main subject of their question here)?

e.g. Why does it matter what song it is?

Why does it matter who is here?

If all of the prompts were delivered before 10 minutes had elapsed, the research assistant began again with the list in the same order. Below is an example of a conversation that might take place during the task and the list of prompts used by the research assistant as responses during the task.

Participant: “Hey. I was wondering if you might be able to turn your music down a bit.”

Research Assistant: “Why?”

Participant: “Well, I have a big test tomorrow, and I really need to study.”

Research Assistant: “Don’t you like this music?”

Participant: “Usually I do. But today I really need to study.”

Research Assistant: “I like it like this.”

Attentional bias. Attentional bias to negative words was assessed using a modified dot-probe task. For the dot-probe task, participants sat at a computer terminal. A white fixation cross was displayed in the center of a black screen. Participants were presented with two words in the center of the screen in white uppercase letters, with one word one centimeter above and one word one centimeter below the former location of the central fixation cross. A white dot then appeared in place of one of the words. Participants were then asked to indicate the location of the

dot by pressing keys on a keyboard. The "C" key was relabeled with the word "UP" and was used to indicate that the dot had replaced the top word, and the "M" key was relabeled with the word "down" and used to indicate that the dot had replaced the bottom word. One negatively valenced word and one neutral word (e.g., shelf, curtain) or two neutral words were used in each pair. All valenced and neutral words were matched for length and frequency of use in the English language. Valenced words included those related to social threat (e.g., criticized, loner) or interpersonal conflict (e.g., argument, bully).

Selection and validation of valence or neutrality of stimulus words was conducted with a questionnaire presented to a sample of 24 undergraduate female students reflecting the experimental sample. The validation required each individual to rate a collection of words on negativity scales ranging from 0 to 5 on dimensions of both social threat and interpersonal conflict. Social threat words were chosen for the dot-probe if they had an average rating of 3 or more as relevant to social threat and less than 3 as relevant to conflict. Conflict words were chosen if they had an average rating of 3 or more as relevant to interpersonal conflict and less than 3 as relevant to social threat. Overall ratings were not significantly different for each group of negative words. Neutral words all received scores of 0 on both social threat and interpersonal conflict scales and were all related to objects in the home.

Participants were presented with 6 learning trials in which the experimenter demonstrated the procedure, 12 practice trials, and 180 total test trials. Ninety trials included unmasked stimuli presented at the conscious level of awareness for 120ms, and the remaining 90 included masked stimuli presented below the level of conscious awareness for 50ms and then covered with a string of nonsensical letters (e.g., SPTUZKT) to prevent participants from being able to view the shadow of previously presented stimuli. Each of the two groups of stimulus display durations

included 30 word pairs reflecting a neutral word and social threat word, 30 word pairs reflecting a neutral word and conflict word, and 30 word pairs of two neutral words. A 90 second rest break was provided midway through the task, with random selection of display time (i.e., masked vs. unmasked) and trial type (i.e., social threat/neutral, conflict/neutral, neutral/neutral) throughout.

E-Prime 2.0 experimental presentation software (Psychology Software Tools, Pittsburgh, PA) was used to conduct these tasks and record responses as well as response latencies for each trial. Incorrect responses were discarded in the analyses, and response latencies were used as a measure of bias in automatic selective attention in this study. Shorter response latencies on trials in which threat words were replaced by dot probes and longer response latencies on trials in which neutral words were replaced by dot probes indicated an attentional bias towards negative words. The reverse pattern indicated an attentional bias away from negative words.

A standard lexical decision-making task (e.g., Mogg, Bradley, & Hallowell, 1994) was subsequently conducted in order to assess whether participants were able to consciously perceive masked stimuli. In this task, participants were presented with stimuli for 50ms reflecting either a real (e.g, cooler) or nonsense word (e.g., blorky), which was subsequently replaced with a string of nonsensical letters. Exactly half of the stimuli were real words. Participants were then asked to indicate by pressing the "C" key that had been relabeled "YES" or the "M" key that had been relabeled "NO" whether the stimulus was a real or non-real word. Fifty percent overall accuracy indicated that the participant was guessing and unable to consciously perceive the stimuli.

Family conflict. All participants completed the frequency (6 items) and intensity (7 items) subscales of the Children's Perception of Interparental Conflict Scale (Grych, Seid, & Fincham, 1992). This scale is designed to measure various aspects of conflict occurring in the home from a child's perspective. Each item is rated on a 3-point scale (true/sort of true/false).

This scale has demonstrated adequate validity and internal consistency in samples of older adolescents.

Emotion regulation. All participants completed the Emotion Regulation Questionnaire (ERQ). The ERQ (Gross & John, 2003) is a self-report measure of emotion regulation strategies pertaining to cognitive reappraisal and suppression of emotions. The ERQ has shown adequate internal consistency and test-re-test reliability. Further, it has demonstrated adequate convergent and discriminant validity against measures of coping, mood state, rumination, and personality (Gross & John, 2003).

Coping. Data for this study was collected on a revised version of the Responses to Stress Questionnaire, a coping measure designed and previously validated by Compas and colleagues (Connor-Smith et al., 2000). All participants completed a revised version of the RSQ (i.e., RSQ-II), which also includes items measuring volitional coping responses and involuntary responses to stress. Engagement and disengagement responses are included for volitional and involuntary responses, with the volitional engagement responses further divided into primary and secondary control coping. This study analyzed responses to secondary control engagement coping items, which include cognitive restructuring (e.g., I tell myself that things could be worse), positive thinking (e.g., I tell myself that everything will be alright), acceptance (e.g., I just take things the way they are, I go with the flow), and distraction (e.g., I imagine something really fun or exciting happening in my life). The 6 items assessing cognitive reappraisal from the Secondary Control Coping Scale were also included separately in this analysis. This "cognitive reappraisal parcel" was used as a direct and more specific comparison to the ERQ Reappraisal scale. The RSQ uses proportional scoring. Proportion scores are thus reported for the RSQ-II Secondary Control Coping scale and reappraisal parcel. These scores take into account the total number of items

endorsed when reporting the factor statistics. Because of changes made in current version of the RSQ-II (i.e., an increase in the number of items on the measure), these scores cannot be compared to proportion scores from previous studies.

Symptoms of psychopathology. Symptoms of internalizing and externalizing problems were assessed by the Adult Self Report, a widely used self-report measure assessing emotional and behavioral problems, as well as social competence that has been normed on a nationally representative sample (Achenbach & Rescorla, 2001). It includes 113 items scored on a three-point scale indicating how descriptive the items are of the individual during the preceding six months. The measure produces individual profiles for empirically based syndromes as well as DSM-oriented scales. The measure includes both Borderline and Clinical cutoffs that can be used to describe an individual's responses with respect to the normative sample, taking into account the participant's gender. For the narrow-band scales (anxiety/depression, affective problems), the Borderline range includes *T* scores ranging from 65-69, and *T* scores of 70 (98^{th} %ile) and above fall in the Clinical range. For broad-band scales (internalizing, externalizing), the Borderline cut-off is a *T* score of 63, and the Clinical cut-off is a *T* score of 67 (95^{th} %ile). The measure maintains high test-retest reliabilities and internal consistency scores for all subscales in a nationally representative sample. The current analyses utilized the Affective Problems scale as an index of depressive symptoms (items include lack of enjoyment, sleep disruption, appetite disturbance, sadness, suicidal ideation, under-activity, feelings of worthlessness).

Executive function and cognitive control. The Wechsler Adult Intelligence Scale 4^{th} Edition (WAIS-IV; Wechsler, 2008) is a standardized measure of cognitive ability for adolescents and adults between the ages of 16 and 89 years. The IQ and index scores are both presented as age-based standard scores with a mean of 100 and a standard deviation of 15.

Percentile ranks (PR) indicate an individual's performance relative to a national sample and indicate the percentage of test takers in the national sample who had lower scores than the individual. The range for each individual subtest scaled scores is from 1 to 19; scores from 8 to 12 are considered average.

Graduate research assistants administered the Digit Span and Arithmetic subtests of the WAIS-IV to all participants to measure overall attentional control and obtain an index of working memory abilities. The Digit Span subtest measures executive attention, auditory short-term memory, and sequential processing. It is comprised of three parts. In the first, the participant must repeat a string of digits as stated. In the second, the participant must repeat a string of digits in reverse order. In the final part, the participant must state the digits in numerical order. The Arithmetic subtest requires participants to solve numerical reasoning problems that are read aloud by the test administrator without the use of paper and pencil. The Working Memory Index (WMI) combines these two subtest scores to assess ability to attend to new information, hold it in short-term memory, concentrate, and manipulate that information to produce some result or reasoning processes. It is important in higher-order thinking, learning, and achievement as well as cognitive flexibility and planning ability, learning and the ability to self-monitor.

The Delis-Kaplan Executive Functioning System (D-KEFS; Delis, Kaplan, & Kramer, 2001) is a comprehensive battery of tests designed to measure higher-level cognitive functions in individuals from 8 to 89 years-old. The range for each individual subtest scaled scores is from 1 to 19; scores from 8 to 12 are considered average. The Color Word Interference Test was administered to all participants as a measure of cognitive control, divided attention, mental flexibility, processing speed, and inhibition. This test is based largely on the original Stroop test

(Stroop, 1935), but relies on a process analysis approach. Participants complete four conditions total, with each condition assessing a specific component of the overall task. In the first condition, participants state the colors of a series of printed boxes as quickly and accurately as possible. In the second condition, participants read a series of color words all printed in black ink as quickly as possible. These conditions assess processing speed, taking into account color-perception and reading abilities. In the third condition, participants perform the classic Stroop task, stating the ink color of words printed in incongruously colored ink. This task is traditionally conceptualized as a measure of cognitive control and inhibition as well as divided attention. The final condition builds on the Stroop task by asking participants to switch the applied rule and read the read the presented word when it is presented inside a box. This task adds an additional component of mental flexibility and set shifting.

As part of the packet of questionnaires given during the assessment session, all participants completed the Behavior Rating Inventory of Executive Function –Adult Version (BRIEF; Gioia et al., 2000). The BRIEF is a 75-item assessment of impairment in several domains of executive functioning. Participants age 18 to 90 years rate behavior frequency on a 3-point Likert scale (0 to 2). The questionnaire contains 75 items covering 9 non-overlappping clinical scales and 3 validity scales. These theoretically and statistically derived scales comprise two broader indices of Behavioral Regulation (Inhibit, Shift, Emotional Control) and Metacognition (Initiate, Working Memory, Plan/Organize, Organization of Materials, Self-Monitor, Task Monitor). The three validity scales identify unlikely response patterns in the domains of Negativity, Inconsistency, and Infrequency. The BRIEF has demonstrated satisfactory internal consistency reliability and has been normed on appropriate census populations in the US (Roth, Isquith, & Gioia, 2005).

Procedure

Recruitment of participants was performed using the SONA Experiment Management System advertising to all current Vanderbilt undergraduates. When participants arrived for their scheduled appointment, they were given the consent form. A trained graduate student research assistant also verbally explained the study and consenting process to the participant in a quiet room.

Prior to commencing with the study, the participant was connected to an electronic heart rate monitor. For the heart rate monitor, the participant was told to attach one electrode on the skin just below her right clavicle and another electrode on the skin on her left lower thorax on the ribcage. She was then shown a diagram of these locations. The research assistant briefly left the room while the participant did this. These electrodes remained in place until after both the Noisy Neighbor Stress Task and dot-probe attentional bias task were complete.

The participant then sat in a quiet room where magazines were provided for 15 minutes in order to measure baseline cardiovascular function and allow the participant to adjust to her surroundings. At the end of this 15-minute period, the first saliva sample was collected, and the participant completed the PANAS Questionnaire. The participant then completed either the Noisy Neighbor Stress Task or the dot-probe attentional bias task, depending on randomization status. After completing the first task, another saliva sample was collected, and the participant again filled out the PANAS questionnaire. The task that was not already completed was then performed. After completing the second task, another saliva sample was collected, and the participant again filled out the PANAS questionnaire. The participant was then escorted to a quiet room where she filled out the questionnaires discussed above. The participant remained in the room for 30 minutes. Saliva samples were collected every 15 minutes during this time.

After this 30-minute recovery period, a trained research assistant administered the Color Word Interference Subtest from the DKEFS as a standardized measure of selective attention and the Digit Span and Arithmetic subtests from the WAIS-IV as a measure of working memory abilities. Participants were then free to leave the laboratory. They were compensated four SONA credits, which were credited to their account within 48 hours.

If the research assistant learned through any statements made verbally or in writing during the experiment that the participant was an immediate threat to herself or another identified individual, she provided referral information for emergency mental health services available to Vanderbilt students. A visual flow chart representation of the procedure is shown below in Figure 4.

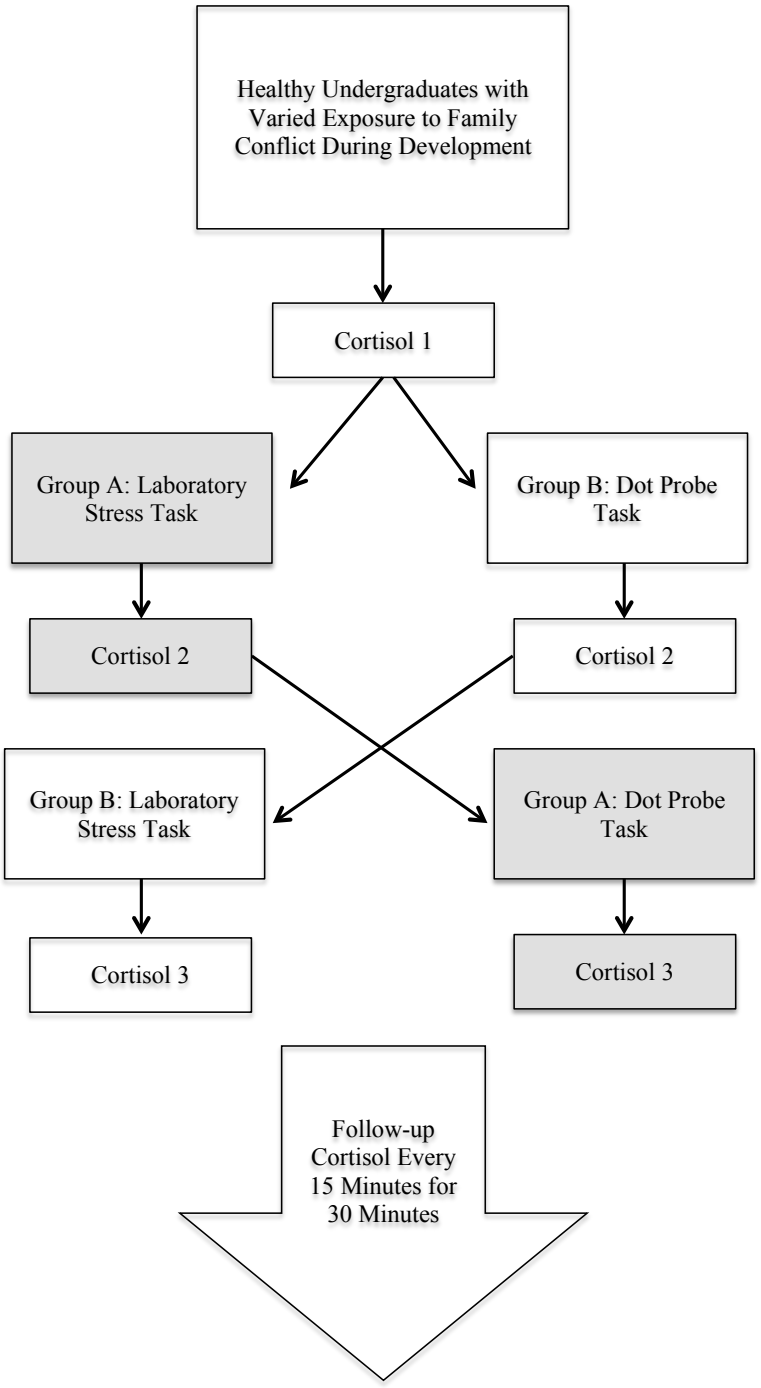

Figure 4. Schematic illustration of study design and procedure

Data Analytic Strategy

Attentional bias. Data from the attention bias task were cleaned and bias scores calculated according to the following steps. For each participant, incorrect responses as well as responses greater than 3-seconds or with latencies greater or less than 3 *SD*s from both the sample mean as well as each participant's mean response time were removed. This ranged from 0 to 6 trials per participant, representing 2.7% of the overall data. Attentional bias scores were able to be calculated for 108 study participants. Data for one participant was saved incorrectly and not analyzable, and 10 participants provided insufficient correct responses to allow the calculation of bias scores, indicating a lack of understanding of the task or lack of motivation to perform it correctly. Bias scores were calculated separately for threat and conflict by subtracting the average response time when the dot replaced a threat or loss word from the average response time when the dot replaced a neutral word according to the following equation (Boyer et al., 2006):

$$\text{Attentional bias score} = \tfrac{1}{2}[\,(\text{UpLt} - \text{UpUt}) + (\text{LPUt} - \text{LpLt})]$$

According to this equation, U = upper position, L = lower position, p = probe, t = negative word. For example, UpLt defines the response latency when the probe is in the upper position and the threat word is in the lower position. Positive scores indicate a bias toward threat or conflict, whereas negative scores indicate avoidance.

Hypothesis testing. First, descriptive statistics were calculated to examine variable distributions, and outliers three or more standard deviations above or below sample means were removed for biological, questionnaire, and behavioral testing measures. Next, parametric and non-parametric tests were used to test the hypotheses listed below. The details of the analytic procedure for each hypothesis is detailed below:

Hypothesis #1: Exposure to an acute laboratory stressor and high levels of family conflict during development will be related to attentional bias to social threat. Independent samples *t* tests were used to ascertain significant differences in attentional biases between randomization groups (Hypothesis 1a). Pearson correlations as well as one-way analyses of variance were used to examine relations between conflict exposure and attentional bias and test main effects of randomization group, history of exposure to family conflict during development using the CPIC score (Hypothesis 1b), and their two-way interaction (Hypothesis 1c) with mean bias scores (conflict and social threat) as the dependent variables.

Hypothesis #2: Attentional bias to social threat and exposure to high levels of family conflict during development will be related to baseline cortisol level and reactivity patterns during the study. Pearson correlations as well as a series of linear multiple regression analyses were used to test main effects of attentional bias to conflict and social threat using the computed social threat bias score (Hypothesis 2a), history of stress exposure during development using the CPIC family stress score (Hypothesis 2b), and their two-way interaction (Hypothesis 2c) with cortisol baseline level and reactivity (i.e. change from baseline to peak) as the dependent variables.

Hypothesis #3: Exposure to high levels of family conflict during development will be related to neurocognitive function, coping and emotion-regulation, and symptoms of anxiety and depression. Pearson correlations were used to examine relations among exposure to family conflict using the CPIC score, coping using the RSQ-II secondary control coping and involuntary engagement scales, emotion regulation using the ERQ reappraisal scale, the BRIEF self-report Metacognition Index and WAIS-IV Working Memory Index as measures of cognitive control, and symptoms of anxiety and depression on the Achenbach Adult Self Report.

Exploratory Analyses: Investigating Interactions of Conflict Exposure, Attentional Bias, and Cortisol Reactivity on Symptoms of Psychopathology. A series of linear multiple regression analyses were used to explore the moderating effect of exposure to family conflict during development on the relation between laboratory measures (biological stress reactivity and attentional bias) and symptoms of psychopathology.

Statistical Power

Power analyses were conducted using G*Power (Faul et al., 2009) to determine the appropriate sample size needed to test the proposed hypotheses. For between-groups comparisons of order randomization, power estimates were based on a power of .80, an alpha coefficient of .05, and a sample of 120 (approximately 60 per group for the randomization of the order of the lab stress task and the attentional bias task). With approximately 60 participants per group, I was able to detect a main effects and interactions of $d \geq .52$.

For regression analyses, Green's (1991) rule of thumb requires a total sample size of approximately 50 + 8m (where m is the number of predictors) was used. With up to three predictors in the proposed regression models, a minimum of 74 participants would be required. Therefore, the sample of 116 is sufficient to test the proposed regression models. Further, in regression models with 3 predictors, the sample of 116 is adequate to detect a critical R^2 of .088, with alpha at .05 and power at .80.

CHAPTER III

RESULTS

Hypothesis 1: *Effects of acute laboratory stress and high levels of family conflict during development on attentional bias*

Descriptive statistics for attentional bias scores. Descriptive statistics for attentional bias scores for masked and unmasked social threat and conflict stimuli, including means, standard deviations, minimums, and maximums for the sample as a whole and broken down by randomization condition, are presented below in Table 1. Positive values reflect a shorter reaction time when the affectively valenced word was probed compared to neutral stimuli, and negative values reflect a longer reaction time when the affectively valenced word was probed compared to neutral stimuli.

First, the data were examined for overall patterns of bias independent of the experimental conditions. No significant biases in attention (i.e., bias scores differing significantly from zero) were found for the sample as a whole or either experimental group.

Second, within condition analyses were performed to detect differences in biases in each experimental condition. For those participants in the acute stress first condition, a significant difference was found between biases to masked as compared with unmasked social threat, t (100) = 2.04, p = .045. Individuals were biased to look towards social threat stimuli presented below the level of conscious awareness but look away when stimuli were presented at the level of conscious awareness. Further, for those participants in the dot probe first condition, significant differences were found between biases to masked and unmasked conflict, t (100) = 2.10, p =

.038, and between masked social threat and unmasked conflict, t (100) = 2.29, p = .024. Individuals were biased to look towards conflict stimuli presented at the level of conscious awareness compared but away from conflict and social threat stimuli presented below the level of conscious awareness.

In testing the effect of acute stress exposure on attentional bias, significant differences between experimental conditions were found for masked social threat, t (98) = -2.04, p = .045, and also unmasked conflict stimuli, t (100) = 2.02, p = .046. Thus, these findings indicate that exposure to a laboratory stress task caused an increase in bias towards social threat stimuli presented below the level of conscious awareness, consistent with predictions in Hypothesis 1a, and away from conflict stimuli presented at the level of conscious awareness.

Table 1

Descriptive Statistics for Attentional Bias Scores to Social Threat and Conflict Stimuli

	Full Sample				Noisy Neighbor/Dot Probe				Dot Probe/Noisy Neighbor			
Bias Condition	*N*	*M (SD)*	Min	Max	*N*	*M (SD)*	Min	Max	*N*	*M (SD)*	Min	Max
Masked Social Threat	102	-1.56 (38.83)	-92.88	115.88	50	6.26 (31.57)	-77.62	71.71	52	-9.08 (43.72)	-92.88	115.88
Masked Conflict	102	-6.75 (40.49)	-102.21	108.33	51	-6.17 (38.50)	-98.63	93.54	51	-7.35 (42.77)	-102.21	108.33
Unmasked Social Threat	100	-5.18 (35.47)	-80.25	100.63	50	-6.72 (32.20)	-80.25	74.83	50	-3.65 (38.74)	-69.13	100.63
Unmasked Conflict	100	2.05 (42.38)	-126.38	127.83	49	-6.62 (41.15)	-126.38	74.67	51	10.37 (42.27)	-69.29	127.83

Note. All scores reflect attentional bias scores calculated according to the explanation and equation on page 39 comparing response latencies of probed negative words to latencies of probed neutral words.

Descriptive statistics for family conflict and relationship to attentional bias. Scores on the CPIC used to measure family conflict prior to 16 years of age ranged from 14 to 40, with a mean of 24.22 and a standard deviation of 7.33. These results are consistent with those found by Reese-Weber and Hesson-McInnis (2008) in a sample of late adolescents (age 17 to 21 years-old) who reported means of 11.23 and 12.54 and standard deviations of 3.54 and 4.24 for the Frequency and Intensity subscales of the measure, respectively, which were combined for the current study.

Correlations of CPIC and attentional bias scores for the entire sample as well for each randomization group are presented in Table 2. In testing Hypothesis 1b, this relationship was examined for the group who performed the dot probe task first. Exposure to high levels of family conflict during development as measured by the CPIC was related to a greater bias away from social threat stimuli presented below the level of conscious awareness (r = -.48, p < .001). In the whole sample as well as in the group performing the acute stress task first, exposure to family conflict was related to greater bias towards stimuli related to conflict presented at the level of conscious awareness (r = .27, p = .006; r = .36, p = .01, respectively) and also a greater bias away from stimuli related to social threat presented below conscious awareness (r = -.39, p < .001; r = -.29, p = .043, respectively).

Table 2

Correlations of CPIC and Attentional Bias Scores

Sample Group	Masked Social Threat	Masked Conflict	Unmasked Social Threat	Unmasked Conflict
Whole Sample	-.39***	-.02	.16	.27**
Noisy Neighbor/Dot Probe	-.29*	.15	.19	.36**
Dot Probe/Noisy Neighbor	-.48***	-.17	.14	.18

Note. *p<.05, **p<.01, ***p<.00

Interaction of level of family conflict exposure and acute stress exposure on attentional bias. High (n = 26) and low (n = 24) groups were selected for exposure to family conflict during development based on CPIC total score. One-way analyses of variance were performed testing main effects of conflict exposure, task order, and their interaction on attentional bias scores. Significant main effects of conflict, F (1, 41) = 12.61, p = .001, and order, F (1, 41) = 4.10, p = .049, as well as an interaction, F (1, 41) = 4.14, p = .048, were found in predicting bias to masked social threat stimuli.

As shown in Figure 5, individuals exposed to low levels of family conflict were significantly biased towards social threat stimuli presented below the level of conscious awareness in the acute stress first condition, t (11) = 4.85, p = .001. In addition, individuals exposed to high levels of family conflict during development were biased away from these stimuli in the dot probe first condition, t (11) = -5.78, p < .001, and a significant difference was found between low and high conflict groups in attentional bias towards social threat stimuli presented below the level of conscious awareness when the dot probe was presented first, t (21) = 4.08, p < .001, with individuals in the high conflict group tending to display an attentional bias away from these stimuli.

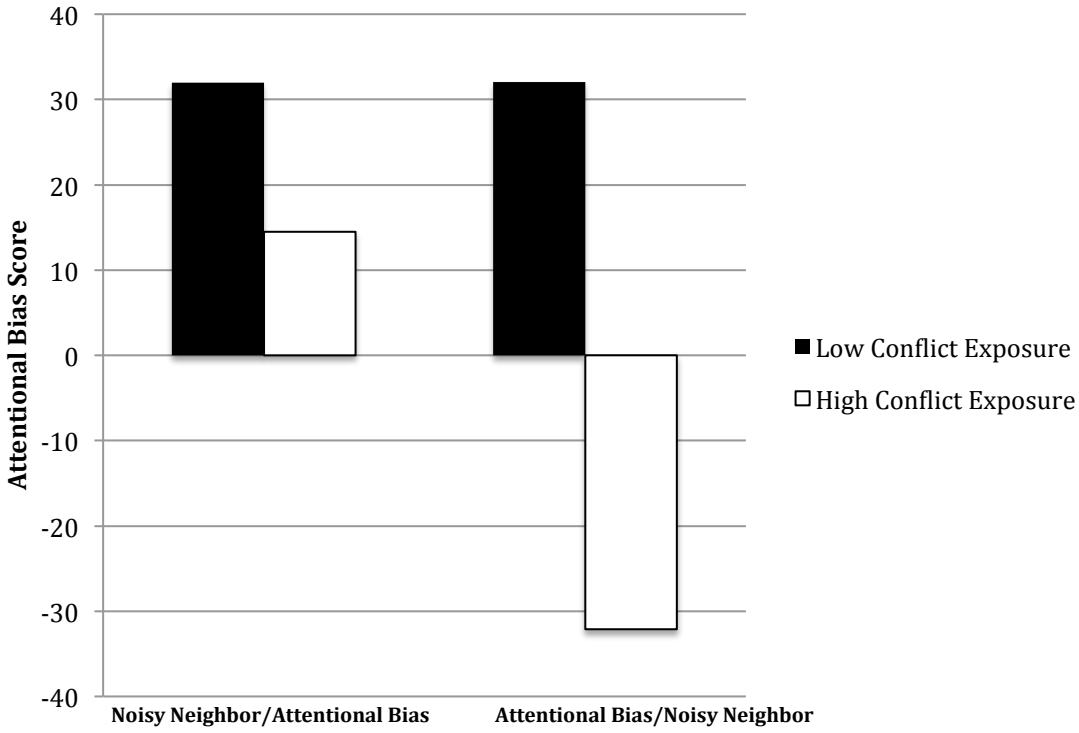

Figure 5. Interaction of conflict and randomization group on masked social threat bias

Hypothesis 2: *Effects of exposure to acute laboratory stress and high levels of family conflict during development and attentional bias to threat on stress biology.*

Descriptive statistics for measures of stress biology.

Cortisol. Levels of salivary cortisol (ug/dL) obtained for 116 participants at 5 time points as well as cortisol reactivity, defined as change in level pre- to post-stress task, and total area under the curve for cortisol output over the course of the study are shown below in Table 3. As shown in Table 3, both experimental groups exhibited an overall linear effect of time over the course of the study on cortisol levels. This is consistent with the typical diurnal decline in cortisol levels during the late afternoon. Mauchly's Test of Sphericity indicated that the assumption of sphericity had been violated, $\chi 2\ (9) = 271.50\ p < .001$, and, therefore, a Greenhouse-Geisser correction was used. There was a significant effect of time on cortisol level, $F\ (2.11, 234.22) = 13.34, p < .001$. Post-hoc comparisons indicate no significant differences between groups at any of the 5 time points. In addition, no significant between-groups differences were found for cortisol reactivity or AUC measures.

Table 3

Descriptive Statistics for Cortisol, Reactivity, and Area Under the Curve (AUC)

Randomization	Baseline	Post-Task 1	Post-Task 2	Follow-up 1	Follow-up 2	Stress Reactivity	AUC
Noisy Neighbor/Dot Probe	7.22 (4.22)	7.27 (4.32)	7.01 (4.68)	6.33 (3.63)	5.75 (3.11)	.047 (2.02)	410.38 (227.85)
Dot Probe/Noisy Neighbor	7.87 (4.35)	7.38 (3.71)	7.16 (4.10)	6.77 (3.77)	6.02 (2.91)	-.28 (2.03)	429.19 (208.08)

Note. Cortisol values reflect ug/dL as assayed from saliva samples.

In spite of these lack of between-group differences in measures of cortisol, relations among measures of cortisol (baseline, reactivity, and AUC) and attentional bias scores are displayed in Table 4 for the group that performed the dot probe first, to account for significant between-groups order effects observed for attentional bias scores. Significant relations were found among cortisol baseline, reactivity, and AUC. Individuals with higher levels of baseline cortisol had significantly lower levels of reactivity pre- to post-stress task and produced greater total cortisol over the course of the study, suggesting a possible ceiling effect for reactivity (see Table 4). In testing Hypothesis 2a regarding the relations between cortisol levels and measures of attentional bias, correlations between unmasked bias to conflict and baseline cortisol level (r = -.40, p = .004) as well as AUC (r = -.38, p = .007) were significant. Even after controlling for baseline level of cortisol, no additional significant correlations were found between cortisol reactivity or AUC and attentional bias measures. As such, individuals with higher levels of cortisol at baseline as well as those who produced more total cortisol over the course of the study tended to display a bias away from conflict stimuli presented at the level of conscious awareness.

Table 4

Correlations Among Measures of Cortisol, Attentional Bias, and Conflict Exposure for Group Performing Dot Probe Task First

	1.	2.	3.	4.	5.	6.
1. Baseline Cortisol	-					
2. Cortisol Reactivity	-.34*	-				
3. Cortisol Total AUC	.83***	.13	-			
4. Masked Social Threat	.17	-.20	.08	-		
5. Masked Conflict	-.10	.07	.09	.20	-	
6. Unmasked Social Threat	.02	-.18	-.10	-.02	-.10	-
7. Unmasked Conflict	-.40**	.09	-.38**	-.02	-.01	.20

Note. *p<.05, **p<.01, ***p<.001

Correlations of CPIC and measures of cortisol for the entire sample as well for each experimental condition are presented in Table 5. As shown in Table 5, exposure to family conflict during development as measured by the CPIC scale was significantly positively correlated with baseline cortisol in the sample as a whole ($r = .35$, $p < .001$), as well as individually in both experimental groups ($r = .35$, $p = .008$; $r = .30$, $p = .026$). In addition, prior exposure to family conflict was significantly positively correlated with cortisol AUC in the sample as a whole ($r = .26$, $p = .005$) and in the group that performed the dot probe first ($r = .28$, $p = .032$).

Table 5

Correlations of CPIC and Measures of Cortisol (ug/dL)

	Baseline	Reactivity	AUC
Whole Sample	.34***	-.11	.26*
Noisy Neighbor/Dot Probe	.35**	.19	.24
Dot Probe/Noisy Neighbor	.30*	-.01	.28*

Note. *p<.05, **p<.01, ***p<.001

Testing main effects and interaction of conflict exposure and attentional bias on cortisol reactivity. Finally, as shown in Figure 6, a linear regression analysis indicated a negative main effect of conflict exposure, β (3, 45) = -.36, p = .022, and a significant interaction of the effects of conflict exposure and unmasked bias to conflict on cortisol reactivity in the group performing the acute stress task first, β (3, 45) = -.30, p = .42. As shown in Figure 6, attentional bias to conflict stimuli presented at the level of conscious awareness was positively related to cortisol reactivity to an acute stressor, but only for individuals exposed to lower levels of family conflict during development.

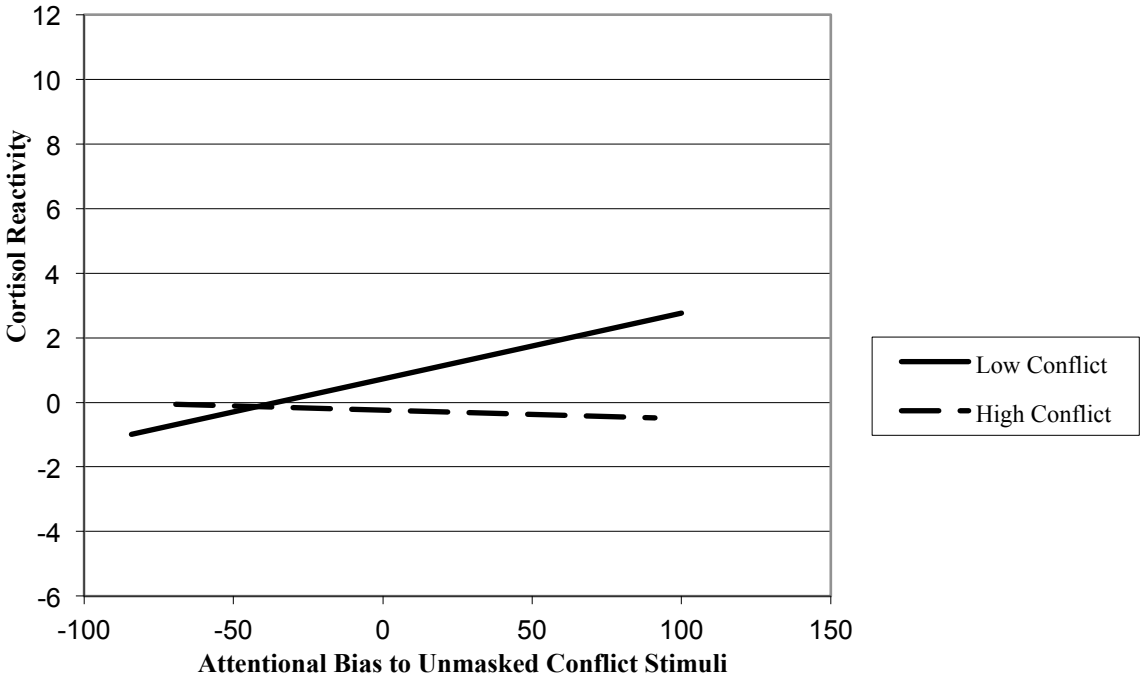

Figure 6. Interaction of conflict and unmasked conflict bias on cortisol reactivity.

Heart rate. Mean heart rate in beats per minute obtained for 115 participants during the baseline, Noisy Neighbor, and dot probe task periods, as well as heart rate reactivity, defined as the difference in average heart rate between the baseline and stress task periods, are shown below in Table 6. As seen in Table 6, both groups displayed a significant increase between baseline mean heart rate and mean heart rate during the Noisy Neighbor stress task, t (56) = -13.54, p < .001; t (57) = -11.74, p < .001. Post-hoc analyses indicate no significant between-groups differences during any of the three time periods. However, the group who performed the stress task first displayed significantly greater reactivity than the group who performed the dot probe task first, t (113) = 2.76, p < .01.

Table 6

Descriptive Statistics for Heart Rate (bpm) during Baseline, Experimental Task Periods, and Reactivity

Sample Group	Baseline	Task 1	Task 2	Reactivity
Noisy Neighbor/Dot Probe	80.13 (12.77)	96.62 (17.47)	78.83 (12.30)	16.49 (9.19)
Dot Probe/Noisy Neighbor	81.01 (15.55)	81.34 (9.61)	93.10 (17.95)	12.10 (7.85)

Relations among heart rate measures (baseline, task periods, reactivity) and attentional bias scores are displayed below in Table 7 for individuals for the group that performed the dot probe first only. Significant relations were found among baseline heart rate and heart rate during the two experimental tasks. In addition, average heart rate during the Noisy Neighbor stress task was significantly related to heart rate reactivity (Table 7). After controlling for baseline heart rate, no additional significant correlations were found among measures of heart rate, reactivity, and attentional bias.

Table 7

Correlations Among Measures of Heart Rate and Attentional Bias for the Group that Performed the Dot Probe First

	1.	2.	3.	4.	5.	6.	7.
1. Baseline Heart Rate	-						
2. Dot Probe Heart Rate	.71***	-					
3. Noisy Neighbor Heart Rate	.90***	.62**	-				
4. Heart Rate Reactivity	.08	.01	.50***	-			
5. Masked Social Threat	.11	.27	.03	-.16	-		
6. Masked Conflict	-.01	.23	-.02	-.02	.21	-	
7. Unmasked Social Threat	-.11	-.18	-.11	-.04	-.03	-.10	-
8. Unmasked Conflict	-.13	-.30*	-.08	-.07	-.02	-.01	-.10

Note. *p<.05, **p<.01, ***p<.001

Correlations of CPIC and measures of heart rate for the entire sample as well for each experimental group are presented in Table 8. As shown in Table 8, exposure to family conflict during development as measured by the CPIC was significantly negatively correlated with heart rate reactivity in the sample as a whole (r = -.18, p = .049), as well as in individuals who performed the stress task first (r = -.33, p = .013), providing evidence that family conflict exposure is related to blunted cortisol and heart rate responses.

Table 8

Correlations of CPIC and Measures of Heart Rate (bpm)

	Baseline	Noisy Neighbor	Dot Probe	Reactivity
Whole Sample	.08	-.01	-.05	-.18*
Noisy Neighbor/Dot Probe	.02	-.17	-.04	-.33*
Dot Probe/Noisy Neighbor	.13	.16	-.09	.01

Note. *p<.05, **p<.01, ***p<.001

Testing main effects and interaction of conflict exposure and attentional bias on heart rate reactivity. Finally, a linear regression analysis indicated a main effect of conflict exposure in predicting heart rate reactivity to acute stress, β (3, 45) = -.36, p = .024. Individuals exposed to higher levels of family conflict during development had significantly decreased reactivity to an acute stress task.

Hypothesis 3: *Exposure to high levels of family conflict during development will be related to neurocognitive function, coping and emotion-regulation, and symptoms of anxiety and depression*

Descriptive statistics, including means, standard deviations, minimums, and maxima, for measures of coping, emotion regulation, and symptoms of anxiety and depression are shown in Table 9. As noted above, proportions are used in scoring the RSQ-II. The distributions of scores in the current study for measures of coping and emotion regulation were not highly skewed (all standard deviations were less than the means), and sufficient variability was present to examine the relationships between these variables and other measures of executive functioning and psychopathology used in this study. Scores for the ERQ Reappraisal Scale represent an average of the responses given on the six Reappraisal items of the ERQ, which are each scored from 1 to 7. In this study, the mean score on the ERQ was found to be 4.79 with a standard deviation of .93. These results were similar to those found by Gross and John (2003) in a sample of nearly 800 undergraduates (M = 4.60, SD = .94 for males; M = 4.61, SD = 1.02 for females).

The DSM Depression (M = 57.0, SD = 8.0) and Anxiety T scores (M = 55.1, SD = 6.9) on the ASR all reflect a moderate elevation of approximately one-half standard deviation above the normative mean for symptoms of psychopathology. A total of 5.0% of the current sample fell

above T = 70 (98%ile) for both DSM depression and anxiety symptoms, respectively. Therefore, the rate of individuals above the clinical sample is between approximately 2.5 times what would be expected in a normal sample, indicating that this sample was exhibiting mild to moderate levels of distress.

Table 9

Descriptive Statistics for Measures of Coping, Emotion Regulation, and Psychological Symptoms

Measure	*N*	*M (SD)*	Min	Max
RSQ-2 Secondary Control Coping	116	.17 (.04)	.03	.26
RSQ-2 Involuntary Engagement	116	.16 (.07)	.02	.85
ERQ Reappraisal	116	4.79 (.93)	1.33	6.83
ASR DSM Depression T Score	116	55.53 (7.09)	50	77
ASR DSM Anxiety T Score	116	57.87 (8.54)	50	83

Descriptive statistics for measures of cognitive control and executive function, including means, standard deviations, minimums, and maximums, are shown in Table 10. WAIS-IV subtest scores as well as the WMI scores (M = 111.48, SD = 11.27) indicate that the sample is significantly above the normative mean and exhibits decreased variability on this cognitive measure. Similar results were found for the two D-KEFS tests administered. Results on the Metacognition Index of the BRIEF (M = 52.29, SD = 11.20), which includes items related to working memory ability, indicate that this sample is similar to the normative sample.

Table 10

Descriptive Statistics for Behavioral and Self-Report Measures of Cognitive Control and Executive Function

Measure	*n*	*M (SD)*	Min	Max
WAIS-IV Digit Span Scaled Score	107	12.31 (2.83)	7	19
WAIS-IV Arithmetic Scaled Score	106	11.93 (1.87)	7	17
WAIS-IV Working Memory Index	106	111.48 (11.27)	86	142
WAIS-IV Letter Number Sequencing Scaled Score	99	12.21 (2.85)	8	19
WAIS-IV Coding Scaled Score	106	12.37 (2.47)	6	18
D-KEFS Color-Word Inhibition Scaled Score	102	11.77 (2.26)	4	16
D-KEFS Color-Word Switching Scaled Score	102	11.28 (1.91)	6	15
BRIEF Metacognition Index T Score	116	52.29 (11.20)	36	88

Correlations among all measures described above and their relationship to exposure to family conflict are shown in Table 11. As shown in Table 11, exposure to family conflict during development was significantly negatively related to both secondary control coping ability (r = -.21, p = .024) and self-reported executive function ability (r = .25, p = .008) and positively related to DSM anxiety symptoms (r = .23, p = .011). Also of note, secondary control coping abilities were significantly positively related to cognitive control abilities as measured by both self-report on the BRIEF (r = -.29, p = .003) and behavioral on the WAIS-IV WMI (r = .23, p = .018) indices of working memory and significantly negatively related to DSM symptoms of depression (r = -.35, p < .001) and anxiety (r = -.33, p < .001). No additional significant correlations were found among measures listed above, cortisol reactivity, or attentional bias.

Table 11

Correlations Among Measures of Family Conflict, Coping, Emotion Regulation, Cognitive Control, and Symptoms of Psychopathology

	1.	2.	3.	4.	5.	6.	7.
1. CPIC Family Conflict	-						
2. RSQ-II Secondary Control Coping	-.21*	-					
3. RSQ-II Involuntary Engagement	.15+	-.41***	-				
4. ERQ Reappraisal	.07	.42***	-.20*	-			
5. WAIS-IV WMI	-.06	.23*	-.01	.10	-		
6. BRIEF MI	.25**	-.29**	.31**	-.14	-.1	-	
7. ASR DSM Depression	0.08	-.35***	.1	-.32***	-.07	.42***	-
8. ASR DSM Anxiety	.23*	-.33***	.17+	-.13	-.04	.37**	.66***

Note. +p<.10, *p<.05, **p<.01, ***p<.001

Exploratory Analyses: Testing Interactions of Conflict Exposure, Attentional Bias, and Cortisol Reactivity on Symptoms of Psychopathology.

Since cortisol reactivity and attentional bias scores were not directly related to symptoms of anxiety and depression, their interactions with past conflict exposure were tested as predictors of symptoms. While no main effects were detected, a significant interaction of conflict exposure and cortisol reactivity was found in the prediction of depression symptoms, β (3, 53) = -.33, p = .028. As shown in Figure 7, cortisol reactivity was associated with depression symptoms in individuals exposed to lower levels of conflict. Individuals exposed to higher levels of conflict displayed the opposite pattern, with lower reactivity associated with higher levels of symptoms, providing further evidence for family conflict exposure related to blunted biological stress reactivity.

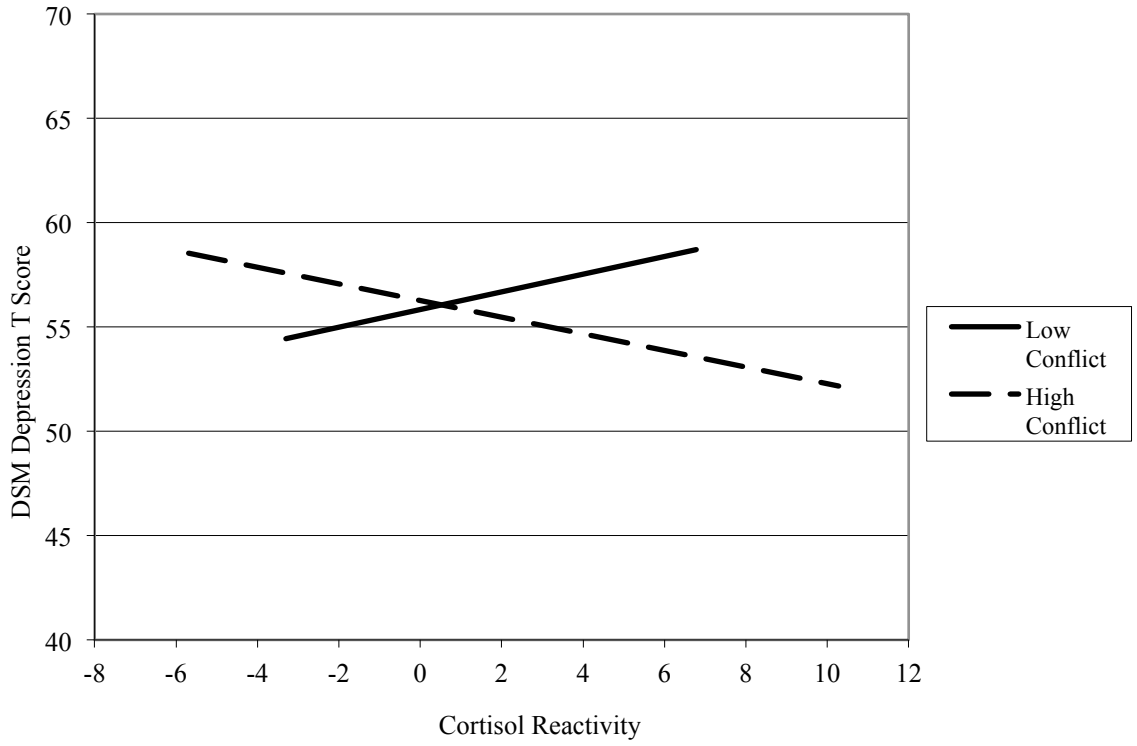

Figure 7. Interaction of conflict and cortisol reactivity in prediction of depression symptoms.

In addition, no main effects were found, but a significant interaction of conflict exposure and attentional bias to masked social threat was found in the prediction of anxiety symptoms, β (3, 48) = -.434, p = .009. As shown in Figure 8, attentional bias towards social threat stimuli presented below the level of conscious awareness was associated with anxiety symptoms in individuals exposed to lower levels of conflict. Individuals exposed to higher levels of conflict displayed the opposite pattern, with attentional bias away from social threat stimuli presented below the level of conscious awareness associated with higher levels of symptoms.

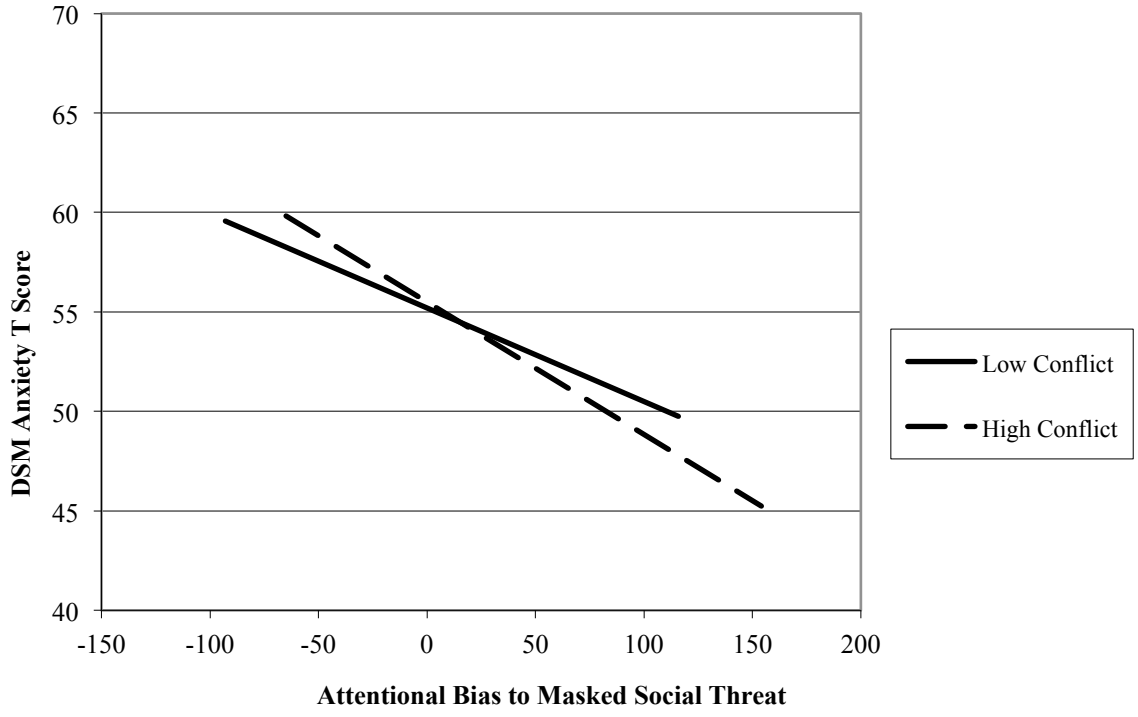

Figure 8. Interaction of conflict and attentional bias to masked social threat in prediction of anxiety symptoms.

CHAPTER IV

DISCUSSION

The current study experimentally examined the reciprocal relations between acute stress reactivity and attentional bias to environmental threat. By examining these constructs within the broader context of self-reported past exposure to family conflict during childhood, I aimed to assess the extent to which these processes may underlie psychological and physiological outcomes in individuals exposed to chronic stress during development. This study thus provides further information regarding the long-term significance of the family environment during development. By also assessing individual differences in coping and emotion regulation strategies and cognitive control abilities, the current study highlights potential points of intervention for the prevention of psychological problems later in young adulthood. Study hypotheses and support are presented in the following sections and summarized in Table 12.

Effects of Stress on Attentional Bias

In examining the relation between stress and attentional bias to social threat and interpersonal conflict, this study focused on both acute stress reactivity in the laboratory and self-reported exposure to chronic stress earlier in development. No significant attentional biases either towards or away from threatening stimuli for the whole sample were found in this study. While calculated bias scores did not differ significantly from zero, due at least in part to the large variances characteristic of these scores, significant effects of the order of the stress and attentional bias tasks were found for two categories of threat stimuli. As predicted, exposure to

an analogue of acute interpersonal stress in the laboratory increased attentional bias towards stimuli related to social threat presented below the level of conscious awareness (see Table 12). This pattern was hypothesized based on expected adaptive increases in automatic selective attention following identification of an environmental threat (e.g., Chajut & Algom, 2003) as well as surges in glucocorticoid production and circulation stimulating medial prefrontal regions which play a role selective attention abilities (Lupien & McEwen, 1997). Further, the acute stressor, designed to be socially threatening, presented immediately preceding the dot probe task may have heightened emotional sensitivity to words related to social threat. Previous studies (e.g., MacLeod & Rutherford, 1992) suggest that individuals may be less sensitive to distinctions among similarly valenced words of differing semantic categories that are presented below the level of conscious awareness compared to presentations at the level of conscious awareness (MacLeod, 1999). In the current study, the bias scores for social threat and interpersonal conflict stimuli presented below the level of conscious awareness were not significantly different, supporting these previous findings. In addition, acute stress exposure increased attentional bias away from conflict stimuli presented at the level of conscious awareness. While not initially hypothesized, this finding suggests that individuals directed attention away from words related to interpersonal conflict shortly after being involved in such a situation.

In examining these biases within the context of self-reported exposure to earlier chronic family conflict, a significant negative relation was observed between attentional bias to social threat stimuli presented below the level of conscious awareness and chronic family conflict exposure. In other words, individuals with higher levels of prior family conflict tended to focus their attention away from socially threatening words that they were not consciously aware of (Table 12). Similarly, Glinder et al. (2007) found that women newly diagnosed with breast

cancer tended to orient their attention away from stimuli related to cancer treatment that were presented below the level of conscious awareness in a dot-probe paradigm. Such self-protective behavior may help individuals to cope and regulate their emotions when presented with stimuli maintaining personal relevance.

A significant moderating effect of family stress was confirmed for the relation between acute stress and attentional bias (Table 12). Individuals exposed to high levels of family conflict were biased away from social threat stimuli presented below the level of conscious awareness when the dot probe task was presented prior to the acute stress task. Also, a significant difference was found between low and high conflict groups in attentional bias to these stimuli. In the acute stress first group, individuals exposed to low levels of chronic family conflict were significantly biased towards social threat stimuli presented below the level of conscious awareness, but no significant bias was found for the high conflict group. These results may signify general cognitive changes and enhanced short-term malleability of automatic attentional processing possibly resulting from chronic stress-related changes in prefrontal function.

Alternatively, an emerging literature on attentional retraining suggests that using a dot probe paradigm in which individuals are reinforced to focus attention away from threatening stimuli and towards neutral stimuli may be an adaptive coping strategy and translate into decreased psychological and physiological stress reactivity (e.g., Amir et al., 2009; MacLeod et al., 2002; Schmidt et al., 2009). If the tendency to focus attention away from threatening stimuli does in fact promote emotional and physical well-being as has been hypothesized (Koster et al., 2010), early exposure to moderate levels of family conflict may have acted as a "stress inoculation" of sorts, facilitating more adaptive attentional response styles later in life.

However, a positive impact of chronic stress appears less probable when interpreted alongside dot probe results for stimuli presented at the conscious level of awareness. When individuals were presented with an acute stressor before performing the dot probe task, a significant, positive relation was observed between chronic stress exposure during development and attentional bias toward stimuli related to interpersonal conflict presented at the conscious level of awareness. These results extend previous findings by Marin and colleagues (2009) who found that a history of chronic family stress was related to increased levels of inflammatory markers measured after a single acutely stressful event. Thus, past experience appears to continue shaping an individual's well-being through enduring changes in automatic biological reactivity systems that come online in response to daily life stressors and subsequently influence long-term health outcomes. In the current experiment, while no significant differences related to prior exposure to family stress were found for attentional biases to threat in the dot probe first condition which served as a control for the task, an acute stressor selectively influenced automatic attentional processes in individuals with a history of chronic family stress. These findings may be initial evidence of co-occurring changes in both cognitive and biological acute stress reactivity systems following chronic family stress exposure. Such changes may represent more proximal contributors to mental and physical health disorders in chronically stressed populations.

Relations Among Stress, Attentional Bias, and Stress Biology

Cortisol patterns as measured over the course of the study (baseline, reactivity, AUC) were examined within the context of the acute laboratory stressor and history of chronic stress exposure. Regardless of experimental group, participants tended to show an overall decline in

cortisol levels over the course of the study. One interpretation of this finding is that the interpersonal laboratory stress task failed to produce HPA axis activation. However, the typical diurnal pattern of cortisol fluctuation that peaks in the early morning and declines steadily over the course of the day may also have concealed any mild to moderate surges that occurred as a result of the task.

No significant relations were found between cortisol reactivity and measures of attentional bias, but baseline cortisol and total AUC were significantly negatively related to attentional bias to conflict stimuli presented at the level of conscious awareness (Table 12). Therefore, individuals with higher levels of baseline cortisol at the start of the study and those who produced more cortisol throughout the study tended to focus attention away from conflict stimuli. These results are consistent with several studies suggesting dampening effects of exogenously administered cortisol on selective attention and attentional bias to threat (e.g., Putman et al., 2007; Putman, Hermans, & van Honk, 2010; Van Peer, Spinhoven, & Roelofs, 2010).

More systematic evaluation of these biases within the context of self-reported exposure to chronic stress revealed that exposure to family conflict was significantly positively correlated with baseline cortisol and related to total AUC over the course of the study (Table 12). In accordance with allostatic load theory, repeated and prolonged activation of the HPA axis promotes altered acute stress reactivity, including increased ambient levels of cortisol (Miller, Chen, & Zhou, 2007). As individuals exposed to higher levels of family conflict tended to have increased baseline cortisol levels, the positive relation between baseline cortisol level and attentional bias away from conflict stimuli provides evidence for a mechanism underlying altered attentional processing in these individuals.

In examining the relation between attentional biases and cortisol reactivity, a significant moderating effect of family conflict was also confirmed (Table 12). Attentional bias to conflict stimuli presented at the level of conscious awareness was positively related to cortisol reactivity, but only for individuals exposed to lower levels of chronic stress during development. The specificity of findings for lower stress individuals may lend support to allostatic load related changes in HPA axis functioning (Juster, McEwen, & Lupien, 2010; McEwen, 2008; Repetti et al., 2011). If the HPA axis does in fact become dysregulated due to prolonged activation by psychosocial threat during early developmental periods, the resulting higher levels of ambient circulating cortisol may prevent typical and adaptive reactivity.

Individuals exposed to higher levels of family conflict also tended to have decreased heart rate reactivity to the acute stress task. While these results run counter to the hypothesized outcome (Table 12), they may represent inadequacies in the methods used for measurement of cardiovascular reactivity. For this study, simple heart rate (beats per minute) was recorded and compared for each participant during three discreet experimental periods (i.e., baseline, stress task, attentional bias task). However, standing vs. sitting as well as the performance of a controlled attention task vs. a talking task are known factors in influencing heart rate (American Heart Association, 2012). Future studies may include continuous blood pressure monitoring or measures of vagal tone in order to more adequately control for these factors.

Neurocognitive Function, Coping Skills, and Psychological Sequelae

Hypothesized allostatic load related neurocogntive deficits that impact long-term health were subsequently investigated. All measures of cognitive control, coping, emotion regulation, and symptoms of psychopathology functioned as expected in this sample. While the mean score

on the WAIS-IV measures of working memory were higher than the normative population, this elevation is consistent with the use of a sample drawn from the undergraduate introductory psychology courses at a highly selective university. Further, the variance on the WAIS-IV WMI was more constrained than in the normative population. The BRIEF-MI mean for this sample was, however, similar to the normative population. In addition, results on measures of emotion regulation were comparable to those described in the literature for college students. Finally, participants' self-reports of symptoms on the ASR indicate that the sample was experiencing mild to moderate distress in domains of depression and anxiety compared to a normative sample. Therefore, although the sample analyzed was one of convenience, meaningful variation was found for measures of executive functioning, emotion regulation, and coping in addition to internalizing symptoms of psychopathology. Such results allowed the construction of correlation and regression models in order to examine individual differences in coping and emotion regulation strategies and cognitive control abilities as they relate to chronic family conflict exposure and current acute stress reactivity in the laboratory. Based on these results, the current study highlights potential points of intervention for the prevention of psychological illness later in life.

Neurocognitive deficits posited as a primary outcome of allostatic load and related difficulties in coping and emotion regulation were supported in this study (Table 12). Individuals with higher levels of chronic stress during development reported significantly higher levels of executive dysfunction, lower levels of secondary control coping, a trend towards higher levels of disengagement coping, and higher levels of anxiety symptoms. Further, as reported by Andreotti et al. (2012), both self-report and behavioral measures of executive function abilities were significantly positively related to secondary control coping efforts and significantly negatively

related to symptoms of depression and anxiety. Such a pattern of findings supports a means through which chronic stress related deficits in cognitive control may underlie problems of mental and physical health by limiting an individual's ability to engage in adaptive coping techniques. In addition to a dysregulation of physiological stress response systems, compromised abilities to cope with daily stress creates a "double hit" for those with a history of chronic stress exposure (Compas, 2006).

In exploring the links between laboratory measures collected in this study (attentional bias and biological stress reactivity) and symptoms of depression and anxiety, chronic stress exposure during development was found to be a moderator of these relations (Table 12). Attentional bias towards social threat stimuli presented below the level of conscious awareness was associated with anxiety symptoms in individuals exposed to lower levels of family conflict. Individuals exposed to higher levels of family conflict displayed the opposite pattern, with attentional bias away from social threat stimuli presented below the level of conscious awareness associated with higher levels of symptoms. Appelhans and Luecken (2006) similarly found links among anxiety, the tendency to focus attention away from social threat stimuli, and diminished cortisol reactivity to the dot probe task. As such, decreased reactivity may be a reflection of elevated levels of ambient cortisol characteristic of chronically stressed individuals known to be at high risk for affective psychopathology. In the current study, cortisol reactivity was associated with depression symptoms in individuals exposed to lower levels of family conflict. Individuals exposed to higher levels of family conflict displayed the opposite pattern, with lower reactivity associated with higher levels of symptoms, supporting the elevated ambient cortisol levels and HPA axis "burnout" of allostatic load. Altered HPA axis function (i.e., high baseline, low reactivity) and the automatic attentional processes (i.e., attentional bias away from threat) to

which it has been routinely linked (e.g., Putman et al., 2007; Putman, Hermans, & van Honk, 2010; Taylor et al., 2010; Van Peer, Spinhoven, & Roelofs, 2010) therefore may indicate a combined cognitive-biological-affective framework for deleterious long-term outcomes related to chronic stress.

Strengths and Limitations of the Current Study

Several limitations of the current study are of note. Chiefly, it relied on a cross-sectional design conducted over the course of a single laboratory session with a fairly homogeneous sample of college students from a selective university. However, the current study employed multiple methods in order to further examine the interrelationships among constructs analyzed in this study. First, an experimental behavioral paradigm (the dot probe) was utilized to measure automatic aspects of cognitive control that have been previously linked to the processes of coping and emotion regulation (see Compas & Boyer, 2001 for review) and symptoms of psychopathology (see Bar-Heim et al., 2007 for review). As an indicator of attentional bias and automatic selective attention, the measurement of dot probe reaction times provides insight into non-conscious, non-effortful, and uncontrolled processes of cognition that are integral to the development of hypervigilance and deficits in attentional disengagement that underlie psychological disorders. Current research has however begun to utilize eye tracking technology to begin to parse more specific behavioral components of attentional bias (e.g., Armstrong, Sarawgi, & Olatunji, 2012) which may be helpful in future work.

Second, a laboratory stress task was used alongside questionnaires in learning about how individuals cope with stress and regulate their emotions. The Trier Social Stress Task has historically been used in order to effectively affect acute stress in a controlled environment

(Kirschbaum, Pirke, & Hellhamer, 1993). However, the Noisy Neighbor task used in this study has been found to be an alternative and potentially more ecologically valid example of stress in this population (Luecken, Kraft, & Hagan, 2009) and satisfies the criteria put forth by Dickerson and Kemeny (2004) of including social-evaluative, motivated performance, interpersonal aspects that define successful laboratory stress induction paradigms.

Third, physiological measures of both the effects of chronic stress and acute stress reactivity collected throughout the crossover experimental design further elucidate the psychological and biological processes related to coping and emotion regulation that may impact attention to environmental threat. For example, salivary cortisol and heart rate reactivity measures were obtained during the laboratory stress task and experimental neuropsychological measure. As previously mentioned, however, heart rate reactivity was measured through the recording of simple heart rate (bpm). As activity and position are known to affect heart rate (American Heart Association, 2012), future studies may consider continuous blood pressure monitoring or measures of vagal tone in order to more accurately represent SAM axis activation.

This crossover, multi-method approach sheds further light on the psychobiology of both acute and chronic stress and how individual differences in physiological reactions and coping strategies may influence both psychological and health-related outcomes uniquely and through alterations in attentional processing.

Directions for Future Research

The current study investigated the role of chronic stress in changes in automatic attentional and stress reactivity processes that may affect vulnerability to stress-related mental and physical disorders over the long-term through hypothesized transformations in neural

structure and function. As such, the more direct investigation of the neural bases of these cognitive processes may be valuable. Several studies employing neuroimaging techniques while individuals perform dot probe type tasks have provided evidence of prefrontal and anterior cingulate involvement in automatic attentional processes to threat (e.g., Bishop et al., 2004). Increased anxiety has been routinely linked to altered prefrontal activation in response to threat in similar regions (Monk et al., 2006, van den Heuvel et al., 2005). Further investigation may thus examine specific patterns of neural activation that are related to a history of chronic stress exposure during tasks of automatic attention to threat. Confirmation of functional changes would provide evidence for a link between long-term effects of psychobiological stress reactivity processes and neurocognitive underpinnings of psychological symptoms.

Impairments in coping and emotion regulation related to chronic stress were hypothesized to partly account for this link in the current study. Work by Oschner and colleagues (2002) lends support to this idea in a neuroimaging study that examines regional activation during a coping/emotion regulation task. Results of the study indicated increased activation in the lateral and medial PFC and decreased activation in the amygdala and vmPFC during a cognitive reappraisal task. These findings suggest that the process of cognitive reapprsaisal may influence emotion generation and evaluation functions of the amygdala and vmPFC, regions that may be affected by chronic stress exposure.

The dense concentration of stress-sensitive glucocoricoid receptors in the vmPFC whose chronic stimulation is believed to affect structural and functional neural development may be a primary source of neurocognitive deficits. Structurally, chronic stress has been linked to reduced dendritic morphology in animal models (Radley et al., 2006). Excess glucocorticoid signaling in prefrontal regions was shown to down-regulate the negative feedback system through decreased

cytosolic receptor levels, supporting a mechanism for blunted neuroendocrine responsitivity and behavioral changes characteristic of affective psychopathology (Mizoguchi, Ishige, Aburada, & Tabira, 2003). A line of research by Miller, Chen, and colleagues suggests an epigenetic pathway for long-term deleterious health outcomes related to early chronic stress exposure (Miller et al., 2008; Miller et al., 2009). Excessive circulation of glucocorticoids reduces receptor transcription, promoting functional glucocorticoid resistance and simultaneous up-regulation of inflammatory pathways. Heightened inflammation may be a direct causal factor in chronic stress-related diseases. Future work should build on current understanding of the association of glucocorticoid receptor gene polymorphisms in receptor expression, function, and neural development (Wust et al., 2004). This research may provide evidence for a mechanism for individual differences in mental and physical health outcomes in those exposed to chronic stress during development and thus serve as a potential source of resilience.

Examining these processes in young adults from clinical high-stress samples and those in exceptional circumstances may aid in further understanding the development of these basic human processes. For example, future work focusing on childhood survivors of acute lymphocytic leukemia and pediatric brain tumors could be used to compare the effects of an exogenous insult to the prefrontal cortex in the form of intrathecal dexamethasone (a drug that is biochemically similar to cortisol) used as treatment for leukemia and brain tumors to endogenous stress processes (e.g., Campbell et al., 2009). It is hypothesized that this treatment regimen involving a large dose of corticosteroids may produce similar long-term deficits in prefrontal functioning to chronic endogenous stress processes. Such work would provide insight into both endogenous and exogenous factors that may affect development of the prefrontal cortex and how these factors may affect long-term outcomes in cognitive control. In addition, this work will

further elucidate the role of the reciprocal connectivity between prefrontal and limbic regions in emotion-cognition interactions as well as the contributions of these interactions to emotion regulation and coping processes that combine elements of both emotion and cognition. These studies of children whose brain development may be affected by either chronic stress or medical treatments will provide insights into the role of the prefrontal regions in the development of the ability to regulate emotions and manage life stress through combined psychobiological and cognitive-affective processes.

Conclusions

The current study focused on attention as a gateway process integral in the perception and interpretation of environmental cues as stressful and subsequently influencing emotional and behavioral responses. Results from this study replicate past findings of the effects of early chronic life stress on biological reactivity axes. While previous work has supported a role for chronic stress exposure in influencing acute biological stress reactivity, this work provides initial insight into how both prior chronic stress and current acute stress both concurrently influence the attentional gateway. Further, these attentional control processes are related to both cognitive and physiological stress reactivity systems shown to contribute to mental and physical health.

Table 12

Summary of Hypotheses

I. Effects of Stress on Attentional Bias	
A. Greater attentional bias to social threat as a function of acute	Full Support
B. Greater attentional bias to social threat as a function of early	Moderate Support
C. Interaction of early family conflict exposure and acute lab stress on attentional bias to social threat	Moderate Support
II. Relations Among Stress, Attentional Bias, and Stress Biology	
A1. Greater attentional bias to social threat related to increased	No Support
A2. Greater attentional bias to social threat related to increased	No Support
B1. Early family conflict exposure related to blunted cortisol	Full Support
B2. Early family conflict exposure related to increased heart rate	No Support
C1. Interaction of early family conflict exposure and attentional bias to social threat on cortisol reactivity	Full Support
C2. Interaction of early family conflict exposure and attentional bias to social threat on heart rate reactivity	No Support
III. Neurocognitive Deficits, Diminished Coping Skills, and	
A. Early family conflict exposure related to decreased coping and	Moderate Support
B. Diminished cognitive control related to decreased coping and	Full Support
C. Early family conflict exposure related to decreased attention	Moderate Support
D. Early family conflict exposure related to increased depression	Moderate Support
Exploratory Analyses: Conflict, attentional bias, stress reactivity,	
A. Interaction of early family conflict exposure and attentional bias to social threat on psychological symptoms	Moderate Support
B. Interaction of early family conflict exposure and cortisol reactivity on psychological symptoms	Moderate Support

REFERENCES

Achenbach, T. M. & Rescorla, L. A. (2003). *Manual for the ASEBA Adult Forms & Profiles*. Burlington, VT*:* University of Vermont, Research Center for Children, Youth, and Families.

American Heart Association, Health Equities-Advocacy. (2010). *Women and Cardiovascular Disease.* Retrieved from http://www.heart.org/HEARTORG/Advocate/IssuesandCampaigns/QualityCare/Health - Equities---Advocacy_UCM_318105_Article.jsp.

American Heart Association, Physical Activity and Blood Pressure. (2012). Retrieved from http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/PreventionTreatmentofHighBloodPressure/Physical-Activity-and-Blood-Pressure_UCM_301882_Article.jsp.

Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention Modification Program in Individuals With Generalized Anxiety Disorder. *Journal of Abnormal Psychology, 118,* 28-33.

Andreotti, C., Champion, J. E., Dunn, M. J., Watson, K., Potts, J., Reising, M. M., et al. (2012). Coping, emotion regulation, and executive function in older adolescents, *Anxiety, Stress, & Coping,* in press.

Applehans, B. M., & Luecken, L. J. (2006). Attentional processes, anxiety, and the regulation of cortisol reactivity. *Anxiety, Stress & Coping: An International Journal, 19,* 81-92.

Armstrong, T., Sarawgi, S., & Olatunji, B. O. (2012). Attentional bias toward threat in contamination fear: Overt components and behavioral correlates. *Journal of Abnormal Psychology, 121,* 232-237.

Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: a new look at emotion, coping, and health-related outcomes. *Journal of Personality*, *72*, 1335-1363.

Baddeley, A. D., & Hitch, J. (1974). Working Memory. *The Psychology of Learning and Motivation* (Vol. 8, pp. 47-89). New York: Academic Press.

Bakvis, P., Spinhoven, P., & Roelofs, K. (2009). Basal cortisol is positively correlated to threat vigilance in patients with psychogenic nonepileptic seizures. *Epilepsy & Behavior: E&B*, *16*, 558-560.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychological Bulletin*, *133*, 1-24.

Barch, D. M., Carter, C. S., Braver, T. S., Sabb, F. W., MacDonald III, A., Noll, D. C., & Cohen,

J. D. (2001). Selective deficits in prefrontal cortex function in medication-naive patients with schizophrenia. *Archives of General Psychiatry*, *58*(3), 280-288.

Benson, M. A., Compas, B. E., Layne, C. M., Vandergrift, N., Pasalic, H., Katalinksi, R., et al. (2011). Measurement of post-war coping and stress responses: A study of Bosnian adolescents. *Journal of Applied Developmental Psychology, 32,* 323-335.

Bishop, S. J. (2009). Trait anxiety and impoverished prefrontal control of attention. *Nature Neuroscience*, *12*, 92-98.

Black, P. H., & Garbutt, L. D. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research*, *52*, 1-23.

Blakemore, S., & Choudhury, S. (2006). Development of the adolescent brain: implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*, *47*, 296-312.

Born, J., Seidel, E., Pietrowsky, R., & Fehm, H L. (1991). Brain evoked responses, a bioassay for central actions of adrenocorticotropin (ACTH 1-39) and corticotropin releasing hormone (CRH) in humans. *Hormone and Metabolic Research*, *23*, 126-130.

Born, J., Kern, W., Fehm-Wolfsdorf, G., & Fehm, Horst L. (1987). Cortisol effects on attentional processes in man as indicated by event-related potentials. *Psychophysiology*, *24*, 286-292.

Boyer, M. C., Compas, B. E., Stanger, C., Colletti, R. B., Konik, B. S., Morrow, S. B., & Thomsen, A. H. (2006). Attentional biases to pain and social threat in children with recurrent abdominal pain. *Journal of Pediatric Psychology*, *31*, 209-220.

Calvete, E., & Connor-Smith, Jennifer K. (2005). Automatic thoughts and psychological symptoms: A cross-cultural comparison of American and Spanish students. *Cognitive Therapy and Research*, *29*, 201-217.

Campbell, L. K., Scaduto, M., Van Slyke, D., Niarhos, F., Whitlock, J. A., & Compas, B. E. (2009). Executive function, coping, and behavior in survivors of childhood acute lymphocytic leukemia. *Journal of Pediatric Psychology, 34,* 317-327.

Campbell-Sills, L., & Barlow, D. H. (2007). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J.J. Gross (Ed., pp. 542-560) *Handbook of Emotion Regulation.* NY, NY: Guilford Press.

Chajut, E., & Algom, D. (2003). Selective attention improves under stress: Implications for theories of social cognition. *Journal of Personality and Social Psychology*, *85*, 231-248.

Clarke, P., MacLeod, C., & Shirazee, N. (2008). Prepared for the worst: Readiness to acquire threat bias and susceptibility to elevate trait anxiety. *Emotion*, *8*, 47-57.

Compas, B E, & Boyer, M C. (2001). Coping and attention: implications for child health and

pediatric conditions. *Journal of Developmental and Behavioral Pediatrics: JDBP*, *22*, 323-333.

Compas, B. E, Boyer, M. C, Stanger, C., Colletti, R. B, Thomsen, A. H, Dufton, L. M., & Cole, D. A. (2006). Latent variable analysis of coping, anxiety/depression, and somatic symptoms in adolescents with chronic pain. *Journal of Consulting and Clinical Psychology*, *74*, 1132.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: problems, progress, and potential in theory and research. *Psychological Bulletin*, *127*, 87-127.

Compas, B. E. (2009). Coping, regulation, and development during childhood and adolescence. *New Directions for Child and Adolescent Development*, *2009*, 87-99.

Compas, B. E., Campbell, L. K., Robinson, K. E., & Rodriguez, E. M. (2009). Coping and Memory. In (pp. 121-142) *Emotion in Memory and Development*. Oxford Scholarship Online Monographs.

Compas, B. E. Potts, J. E., Reising, M. M., Reeslund, K. L., Williamson, J. A., Garai, E. et al. (2011). Resilience in children of parents with a history of depression: Coping and the regulation of positive and negative affect. Manuscript under review.

Connor-Smith, J. K., Compas, B. E., Wadsworth, M. E., Thomsen, A H, & Saltzman, H. (2001). Responses to stress in adolescence: Measurement of coping and involuntary sresponses to stress. *Journal of Consulting and Clinical Psychology*, *68*, 976-992.

Copeland, W. E., & Compas, B. E. (2007). *Neuropsychological correlates of coping: The role of executive inhibition*. Unpublished manuscript.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the Neural Circuitry of Emotion Regulation--A Possible Prelude to Violence. *Science, 289,* 591-594.

Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *The Delis- Kaplan Executive Function System: Examiner's Manual.* San Antonio, TX: The Psychological Corporation.

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Development and Psychopathology*, *9*, 633-652.

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.

Diorio, D., Viau, V., & Meaney, M. (1993). The role of the medial prefrontal cortex (cingulate gyrus) in the regulation of hypothalamic-pituitary-adrenal responses to stress. *The Journal of Neuroscience*, *13*, 3839 -3847.

Eisenberg, N., Fabes, R. A., & Guthrie, I. (1997). Coping with stress: The roles of regulation and

development. *Handbook of children's coping* (pp. 41-72). New York: Plenum Press.

Ellenbogen, M. A., Carson, R. J., & Pishva, R. (2010). Automatic emotional information processing and the cortisol response to acute psychosocial stress. *Cognitive, Affective & Behavioral Neuroscience*, *10*, 71-82.

Ellenbogen, M. A., Schwartzman, A. E., Stewart, J., & Walker, C.-D. (2002). Stress and selective attention: The interplay of mood, cortisol levels, and emotional information processing. *Psychophysiology*, *39*, 723-732.

Ellenbogen, M. A., Schwartzman, A. E., Stewart, J., & Walker, C.-D. (2006). Automatic and effortful emotional information processing regulates different aspects of the stress response. *Psychoneuroendocrinology, 31,* 373-387.

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.

Flier, J. S., Underhill, L. H., & McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, *338*, 171-179.

Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *The Behavior Rating Inventory of Executive Function professional manual.* Florida: Psychological Assessment Resources.

Glinder, J., Beckjord, E., Kaiser, C., & Compas, B.E. (2007). Psychological adjustment to breast cancer: Automatic and controlled responses to stress. *Psychology and Health*, *22*, 337-359.

Green, S. B. (1991). How many subjects does it take to do a regression analysis. *Multivariate Behavioral Research*, *26*, 499.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2,* 271-299.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: the acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology*, *106*, 95-103.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. *Handbook of Emotion Regulation* (pp. 3-26). New York: Guilford Press.

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.

Grych, J. H., Seid, M., & Fincham, F. D. (1992). Assessing marital conflict from the child's perspective: The Children's Perception of Interparental Conflict Scale. *Child*

Development, *63*, 558-572.

Herman, J. P., & Cullinan, W. E. (1997). Neurocircuitry of stress: central control of the hypothalamo-pituitary-adrenocortical axis. *Trends in Neurosciences*, *20*, 78-84.

van den Heuvel, O. A., Veltman, D. J., Groenewegen, H. J., Witter, M. P., Merkelbach, J., Cath, D. C., van Balkom, A. J. L. M., et al. (2005). Disorder-specific neuroanatomical correlates of attentional bias in obsessive-compulsive disorder, panic disorder, and hypochondriasis. *Archives of General Psychiatry, 62,* 922-933.

Hinkelmann, K., Moritz, S., Botzenhardt, J., Riedesel, K., Wiedemann, K., Kellner, M., & Otte, C. (2009). Cognitive impairment in major depression: association with salivary cortisol. *Biological Psychiatry*, *66*, 879-885.

van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., & Verbaten, R. (1998). Baseline salivary cortisol levels and preconscious selective attention for threat: a pilot study. *Psychoneuroendocrinology*, *23*, 741-747.

van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., & Verbaten, R. (2000). Conscious and preconscious selective attention to social threat: different neuroendocrine response patterns. *Psychoneuroendocrinology*, *25*, 577-591.

Hopper, J. W., Karlsgodt, K. H., Adler, C. M., Macklin, E. A., Lukas, S. E., & Elman, I. (2004). Effects of acute cortisol and cocaine administration on attention, recall and recognition task performance in individuals with cocaine dependence. *Human Psychopharmacology*, *19*, 511-516.

Isgor, C., Kabbaj, M., Akil, H., & Watson, S. J. (2004). Delayed effects of chronic variable stress during peripubertal-juvenile period on hippocampal morphology and on cognitive and stress axis functions in rats. *Hippocampus*, *14*, 636-648.

Jaser, S. S., Champion, J. E., Dharamsi, K. R., Riesing, M. M., & Compas, B. E. (2010). Coping and positive affect in adolescents of mothers with and without a history of depression. *Journal of Child and Family Studies*, *20*, 353-360.

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality*, *72*, 1301-1334.

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition & Emotion*, *24*, 281.

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*, 2-16.

Keller, M. C., Neale, M. C., & Kendler, K. S. (2007). Association of different adverse life events with distinct patterns of depressive symptoms. *The American Journal of Psychiatry*, *164*,

1521-1529.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry*, *156*, 837-841.

Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication.

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.

Kopell, B. S., Wittner, W. K., Lunde, D., Warrick, G., & Edwards, D. (1970). Cortisol effects on averaged evoked potential, alpha-rhythm, time estimation, and two-flash fusion threshold. *Psychosomatic Medicine*, *32*, 39-50.

Koster, E. H., De Lissnyder, E., Derakhshan, N., & De Raedt, R. (2010). Understanding depressive rumination from a cognitive science perspective: The impaired disengagement hypothesis. *Clinical Psychology Review, 31,* 138-145.

Larson, M. R., Ader, R., & Moynihan, J. A. (2001). Heart Rate, Neuroendocrine, and Immunological Reactivity in Response to an Acute Laboratory Stressor. *Psychosomatic Medicine, 63,* 493-501.

Lazarus, R. S., & Folkman, S. (1984). *Stress, Appraisal, and Coping*. New York: Springer.

Liston, C., Miller, L. M., Goldwater, D. S., Radley, J. J., Rocher, A. B., Hof, P. R., et al. (2006). Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. *Journal of Neuroscience, 26,* 7870-7874.

Liston, C., McEwen, B S, & Casey, B. J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 912-917.

Low, C. A., Matthews, K. A., Kuller, L. H., & Edmundowicz, D. (2011). Psychosocial predictors of coronary artery calcification progression in postmenopausal women. *Psychosomatic Medicine, 73,* 789-794.

Luecken, L. J., & Appelhans, B. M. (2006). Early parental loss and salivary cortisol in young adulthood: the moderating role of family environment. *Development and Psychopathology*, *18*, 295-308.

Luecken, L.J., Appelhans, B. M., Kraft, A., & Brown, A. (2006). Never far from home: A cognitive-affective model of the impact of early-life family relationships on physiological stress responses in adulthood. *Journal of Social and Personal Relationships, 23,* 189-203.

Luecken, L. J., Kraft, A., & Hagan, M. J. (2009). Negative relationships in the family-of-origin predict attenuated cortisol in emerging adults. *Hormones and Behavior*, *55*, 412-417.

Luecken, L. J., & Appelhans, B. (2005). Information-processing biases in young adults from bereaved and divorced families. *Journal of Abnormal Psychology*, *114*, 309-313.

Luna, B., & Sweeney, J. A. (2004). The emergence of collaborative brain function: fMRI studies of the development of response inhibition. *Annals of the New York Academy of Sciences*, *1021*, 296-309.

Lupien, S. J., & McEwen, Bruce S. (1997). The acute effects of corticosteroids on cognition: integration of animal and human model studies. *Brain Research Reviews*, *24*, 1-27.

MacLeod, C. (1999). Anxiety and anxiety disorders. In *The handbook of cognition and emotion,* Dalgleish, T. & Powers, M. (Eds.), Chichester: Wiley.

MacLeod, C, Mathews, A, & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*, 15-20.

MacLeod, C., & Rutherford, E. M. (1992). Anxiety and the selective processing of emotional information: Mediating roles of awareness, trait and state variables, and personal relevance of stimulus materials. *Behaviour Research and Therapy, 30,* 479–491.

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology, 111,* 107-123.

Marin, T., Chen, E. Munch, J. A., & Miller, G. (2009). Double-exposure to acute stress and chronic family stress is associated with immune changes in children with asthma. *Psychosomatic Medicine, 71,* 378-384.

Mathews, A., & MacLeod, C. (1994). Cognitive approaches to emotion and emotional disorders. *Annual Review of Psychology*, *45*, 25-50.

Mathews, A., May, J., Mogg, K., & Eysenck, M. (1990). Attentional bias in anxiety: Selective search or defective filtering? *Journal of Abnormal Psychology*, *99*, 166-173.

Mauss, I. B., Bunge, S. A., & Gross, J. J. (2007). Automatic emotion regulation. *Social and Personality Psychology Compass*, *1*, 1-22.

McEwen, B. S. (2000). Allostasis, allostatic load, and the aging nervous system: Role of excitatory amino acids and excitotoxicity. *Neurochemical Research, 25,* 1219-1231.

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences*, *1032*, 1-7.

McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology*, *583*, 174-185.

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., Turecki, G., et al. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature neuroscience*, *12*, 342-348.

McHugh, R. K., Behar, E., Gutner, C. A., Geem, D., & Otto, M. W. (2010). Cortisol, stress, and attentional bias toward threat. *Anxiety, Stress, and Coping*, *23*, 529-545.

Meaney, M J, Diorio, J., Francis, D., Widdowson, J., LaPlante, P., Caldji, C., Sharma, S., et al. (1996). Early environmental regulation of forebrain glucocorticoid receptor gene expression: Implications for adrenocortical responses to stress. *Developmental Neuroscience*, *18*, 49-72.

Miller, E. K., & Cohen, J D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, *24*, 167-202.

Miller, G. E., & Chen, E. (2006). Life stress and diminished expression of genes encoding glucocorticoid receptor and β2-adrenergic receptor in children with asthma. *Proceedings of the National Academy of Sciences*, *103*, 5496 -5501.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60,* 501-524.

Miller, G., Chen, E., Sze, J., Marin, T., Arevalo, J.M.G., Doll, R., et al. (2008). A functional genomic fingerprint of chronic stress in humans: Blunted glucocorticoid and increased NF-κB signaling. *Biological Psychiatry, 64,* 266-272.

Miller, G., 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.*

Mizoguchi, K., Ishige, A., Aburada, M., & Tabira, T. (2003). Chronic stress attenuates glucocorticoid negative feedback: involvement of the prefrontal cortex and hippocampus. *Neuroscience, 119,* 887-897.

Mogg, K., Bradley, B. P., & Hallowell, N. (1994). Attentional bias to threat: Roles of trait anxiety, stressful events, and awareness. *The Quarterly Journal of Experimental Psychology, 47A,* 841-864.

Mogg, K., Bradley, B. P., & Williams, R. (1995). Attentional bias in anxiety and depression: the role of awareness. *The British Journal of Clinical Psychology / the British Psychological Society*, *34*, 17-36.

Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology*, *113*, 160-165.

Monk, C. S., Telzer, E. H., Mogg, K., Bradley, B. P., Mai, X., Louro, H. M. C., Chen, G., et al. (2008). Amygdala and ventrolateral prefrontal cortex activation to masked angry faces in children and adolescents with generalized anxiety disorder. *Archives of General Psychiatry, 65,* 568-576.

Ochsner, K. N., Bunge, Silvia A., Gross, James J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, *14*, 1215-1229.

Ohman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology. General*, *130*, 466-478.

Pace, T. W. W., Mletzko, T. C., Alagbe, O., Musselman, D. L., Nemeroff, C. B., Miller, A. H., & Heim, C. M. (2006). Increased stress-induced inflammatory responses in male patients with major depression and increased early life stress. *The American Journal of Psychiatry*, *163*, 1630-1633.

Paradiso, S., Johnson, D. L., Andreasen, N. C., O'Leary, D. S., Watkins, G. L., Boles Ponto, L. L., & Hichwa, R. D. (1999). Cerebral blood flow changes associated with attribution of emotional valence to pleasant, unpleasant, and neutral visual stimuli in a PET study of normal subjects. *American Journal of Psychiatry*, *156*, 1618-1629.

van Peer, J. M., Spinhoven, P., & Roelofs, K. (2010). Psychophysiological evidence for cortisol-induced reduction in early bias for implicit social threat in social phobia. *Psychoneuroendocrinology*, *35*, 21-32.

Pilgrim, K., Marin, M.-F., & Lupien, S. J. (2010). Attentional orienting toward social stress stimuli predicts increased cortisol responsivity to psychosocial stress irrespective of the early socioeconomic status. *Psychoneuroendocrinology*, *35*, 588-595.

Pine, D. S., Mogg, K., Bradley, B.P., Montgomery, L., Monk, C. S., McClure, E., Guyer, A. E., et al. (2005). Attention bias to threat in maltreated children: Implications for vulnerability to stress-related psychopathology. *American Journal of Psychiatry*, *162*, 291-296.

Plotsky, Paul M., & Meaney, Michael J. (1993). Early, postnatal experience alters hypothalamic corticotropin-releasing factor (CRF) mRNA, median eminence CRF content and stress-induced release in adult rats. *Molecular Brain Research*, *18*, 195-200.

Putman, P., Hermans, E. J., & van Honk, J. (2010). Cortisol administration acutely reduces

threat-selective spatial attention in healthy young men. *Physiology & Behavior*, *99*, 294-300.

Putman, P., Hermans, E. J., Koppeschaar, H., van Schijndel, A., & van Honk, J. (2007). A single administration of cortisol acutely reduces preconscious attention for fear in anxious young men. *Psychoneuroendocrinology*, *32*, 793-802.

Radley, J. J., Rocher, A. B., Janssen, W. G. M., Hof, P. R., McEwen, Bruce S, & Morrison, J. H. (2005). Reversibility of apical dendritic retraction in the rat medial prefrontal cortex following repeated stress. *Experimental Neurology*, *196*, 199-203.

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. *The American Journal of Psychiatry*, *160*, 1554-1565.

Reese-Weber, M. & Hesson-McInnis. (2008). The Children's Perception of Interparental Conflict Scale: Comparing factor structure between developmental periods. *Educational and Psychological Measurement, 68,* 1008-1023.

Repetti, R. S., Robles, T. F., & Reynolds, B. R. (2011). Allostatic processes in the family. *Development and Psychopathology, 23,* 921-938.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin,* 128, 330-366.

Rodrigues, S. M., LeDoux, J. E., & Sapolsky, R. M. (2009). The influence of stress hormones on fear circuitry. *Annual Review of Neuroscience*, *32*, 289-313.

Roelofs, K., Bakvis, P., Hermans, E. J., van Pelt, J., & van Honk, J. (2007). The effects of social stress and cortisol responses on the preconscious selective attention to social threat. *Biological Psychology*, *75*, 1-7.

Roelofs, K., Elzinga, B. M., & Rotteveel, M. (2005). The effects of stress-induced cortisol responses on approach-avoidance behavior. *Psychoneuroendocrinology*, *30*, 665-677.

Roth, R. M., Isquith, P. K., & Gioia, G. A. (2005). Behavioral Rating Inventory of Executive Function—Adult version, *Psychological Assessment Resources, Inc.,* Lutz, FL.

Sapolsky, R. M., Meaney, M. J., & McEwen, B. S. (1985). The development of the glucocorticoid receptor system in the rat limbic brain. III. Negative-feedback regulation. *Developmental Brain Research*, *18*, 169-173.

Schmidt, L. A., Fox, N. A., Goldberg, M. C., Smith, C. C., & Schulkin, J. (1999). Effects of acute prednisone administration on memory, attention and emotion in healthy human adults. *Psychoneuroendocrinology*, *24*, 461-483.

Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology, 118,* 5-14.

Skinner, E. A., Edge, K., Altman, J., & Sherwood, H. (2003). Searching for the structure of coping: a review and critique of category systems for classifying ways of coping. *Psychological Bulletin*, *129*, 216-269.

Skosnik, P. D., Chatterton, R. T. J, Swisher, T. & Park, S. (2000). Modulation of attentional inhibition by norephephrine and cortisol after psychological stress. *International Journal of Psychophysiology, 36,* 59–68.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*, 643-662.

Taylor, V. A., Ellenbogen, M. A., Washburn, D., & Joober, R. (2011). The effects of glucocorticoids on the inhibition of emotional information: A dose-response study. *Biological Psychology*, *86*, 17-25.

Vedhara, K., Hyde, J., Gilchrist, I. D., Tytherleigh, M., & Plummer, S. (2000). Acute stress, memory, attention and cortisol. *Psychoneuroendocrinology*, *25*, 535-549.

Wadsworth, M. E., Rieckmann, T., Benson, M. A., & Compas, Bruce E. (2004). Coping and responses to stress in Navajo adolescents: Psychometric properties of the Responses to Stress Questionnaire. *Journal of Community Psychology*, *32*, 391-411.

Walker, L. S., Smith, C. A., Garber, J., & Van Slyke, D. A. (1997). Development and validation of the Pain Response Inventory for Children. *Psychological Assessment, 9*, 392–405.

Wechsler, D. (2008). *Wechsler Adult Intelligence Scale-Fourth Edition.* San Antonio, TX: Pearson.

Wolkowitz, O., Reus, V., Weingartner, H., Thompson, K., Breier, A., Doran, A., Rubinow, D., et al. (1990). Cognitive effects of corticosteroids. *American Journal of Psychiatry*, *147*, 1297-1303.

Wust, S., Federenko, I. S., Rossum, E. F. C., Koper, J. W., Kumsta, R., Entringer, S., & Hellhammer, D. H. (2004). A psychobiological perspective on genetic determinants of hypothalamus‐pituitary‐adrenal axis activity. *Annals of the New York Academy of Sciences*, *1032*, 52-62.

Yao, S., Xiao, J., Zhu, X., Zhang, C., Auerbach, R. P., Mcwhinnie, C. M., Abela, J. R. Z., et al. (2010). Coping and involuntary responses to stress in Chinese university students: Psychometric properties of the Responses to Stress Questionnaire. *Journal of Personality Assessment*, *92*, 356.

Zald, D. H., & Rauch, S. L. (2006). *The Orbitofrontal Cortex*. Oxford: Oxford University Press UK.