The Pennsylvania State University

The Graduate School

Department of Human Development and Family Studies

ADOLESCENT STRESS REACTIVITY AND RECOVERY: EXAMINING THE RELATIONSHIPS BETWEEN EMOTION REGULATION AND THE STRESS RESPONSE WITH A SCHOOL-BASED GROUP PUBLIC SPEAKING TASK

A Dissertation in

Human Development and Family Studies

by

Deirdre A. Katz

Submitted in Partial Fulfillment
of the requirements
for the Degree of

Doctor of Philosophy

May 2016

The dissertation of Deirdre A. Katz was reviewed and approved* by the following:

Mark Greenberg
Edna Peterson Bennett Endowed Chair in Prevention Research
Professor of Human Development
Dissertation Adviser
Chair of Committee

Scott Gest
Professor of Human Development and Family Studies

David Almeida
Professor of Human Development and Family Studies

Martin Sliwinski
Professor of Human Development and Family Studies

Martha Wadsworth
Associate Professor of Psychology

Suzy Scherf
Assistant Professor of Psychology

Eva Lefkowitiz
Associate Professor of Human Development and Family Studies
Professor-in-Charge, Graduate Program

* Signatures are on file in the Graduate School

Abstract

This dissertation examines the associations between physiological responses to stress and emotion regulation strategies in a group of adolescents. The primary aim of this study is to examine affective chronometry, the temporal dynamics of emotion regulation, in a group of adolescents by exploring the relationships between their self-reported emotion regulation strategies and their hypothalamic-pituitary-axis (HPA) response to a social-evaluative stressor. The second aim of this study is to explore the feasibility and effectiveness of using a group version of the Trier Social Stress Test (GPST-A), a social-evaluative stressor, in a school-based setting. Salivary cortisol was measured in response to a social-evaluative stressor, the GPST-A, at 6 time points. Affective chronometry was examined by comparing participants' salivary cortisol concentration and self-reported measures of 3 emotion regulation strategies – suppression, reappraisal and rumination - before and after the social-evaluative stressor. Results from a discontinuous multilevel model indicate that the protocol was feasible in the school context and effective at eliciting a typical stress response. Results also indicate that rumination, but not suppression or reappraisal, was associated with the reactivity phase of the stress response. Specifically, trait rumination predicts a less steep cortisol reactivity slope and state rumination predicts a steeper cortisol reactivity slope. Suppression and reappraisal had no effect on participants' cortisol response profiles.

The present study provides a better understanding of affective chronometry in a group of adolescents through a unique analytic approach. These findings suggest that it may be efficacious for researchers to utilize a discontinuous multilevel model with landmark registration to examine affective chronometry.

Table of Contents

LIST OF FIGURES

LIST OF TABLES

Acknowledgements

The research reported herein was supported by grant R305B090007, the Training Interdisciplinary Education Scientists Pre-doctoral fellowship, from the Institute of Education Sciences. I would also like to thank Edna Peterson Bennett for her generous financial support. The funds provided by the Peterson Prevention Research Fellowship contributed directly to my professional development and completion of this dissertation. The views expressed here are the author's and do not reflect those of the funders.

I am immensely grateful for so many people in my life who helped me with this dissertation. My adviser, Dr. Mark Greenberg, is an outstanding role model. His vision, guidance and support provided the foundation on which I was able carry out this study. He has been patient and supportive throughout the process. Wayne Osgood supported me with the statistical modeling. Nancy Schuit opened her class to me to recruit students for this project and was a friend throughout the process. Lorna, Rachel, Frani and Cassidy were the best RA's ever for being flexible and awesome at their jobs. Rachel Abenavoli, Alexis Harris and Melissa Peckins provided me with consistent support and encouragement during my doctoral career as I embraced statistics and became a competent researcher. My Lakeside colleagues and students inspired me to be the change I want to see in education and do this research. Most of all I would like to thank my family: My parent's for their unending encouragement and enthusiasm for all my life pursuits, my husband, Garren, for being my biggest fan and never doubting my ability to succeed, even when my confidence falters, and Clodagh, for all the joy and happiness she brings me.

Chapter 1: Introduction

Stressful situations often instigate negative emotions (Lazarus & Folkman, 1984) and these emotions can affect physiological functioning. In turn, how people manage their emotions has implications for physiological reactivity (Gross, 1998; Gross & Levenson, 1993). Emotion regulation consists of cognitive and affective processes involved in monitoring, evaluating and modifying emotional reactions to experience (Eisenberg & Spinrad, 2004). It involves a variety of processes that amplify, attenuate, or maintain the strength of emotional reactions and is an important element of affective development because of its link to psychopathology and wellbeing (Davidson, 1998; Thompson, 1994; Aldao, Nolen-Hoeksema, & Schweizer, 2010). Repeated activation of the stress response and delayed recovery to stress may increase risk for many negative health outcomes (Sapolsky, 1998, 2003). Understanding more about the time-course of emotion regulation to an acute stressor, known as affective chronometry, may help explain the large individual variability in stress responsivity. This dissertation examines affective chronometry in a group of adolescents by exploring the relationships between their self-reported emotion regulation strategies and their hypothalamic-pituitary-axis (HPA) response to an acute stressor.

The stress response

Lazarus (1966) posited that the cognitive appraisal that one's available resources are not sufficient to deal with the situational demands results in the subjective experience of stress and associated emotions. Neuroendocrine research supports this model, demonstrating that stimuli that are perceived as uncontrollable, novel, challenging or threatening contribute to the stress response (Denson, Spanovic, & Miller, 2009).

A typical response to stress triggers a cascade of coordinated biobehavioral responses between the central and peripheral nervous systems. During a normative stress response, the Sympathetic Nervous System (SNS) coordinates immediate changes focused on survival, including the release of the epinephrine and norepinephrine, which increase heart rate, blood pressure, and respiration. The HPA axis is a comparatively slower response geared toward maintaining the initial SNS response to the stressor, acting as a negative feedback loop. When the HPA axis is activated, the hypothalamus releases corticotropin, which in turn releases hormone (CRH) triggering the anterior pituitary to release adrenocorticotropic hormone (ACTH). This results in the release of the corticosteroid cortisol from the adrenal cortex. Cortisol then acts on the hypothalamus and anterior pituitary in a negative feedback loop to suppress the release of CRH and ACTH. HPA axis activation stimulates immune function and increases circulation of stored energy to the muscles in use (for reviews: Chrousos & Gold, 1992; Kudielka & Kirschbaum, 2005; Sapolsky, Romero, & Munck, 2000). Under normal circumstances these changes are advantageous and help prepare a person for confrontation or escape. However, there are negative consequences associated with repeated activation of the stress response such as dampened immune function and atrophy of the hippocampus (for a review see: McEwen, 2000).

Salivary cortisol is considered a reliable and valid measure for assessing acute changes in HPA axis activation from stressors and is a reliable indicator of emotional wellbeing (Hellhammer, Wüst, & Kudielka, 2009; Juster et al., 2011). Short-term increase of cortisol in response to stress reflects a normal physiological response to challenge and is adaptive under most circumstances, but can be detrimental if overproduced or dysregulated (McEwen, 2000). McEwen (1998) has argued that impaired stress recovery may lead to increased wear and tear on

bodily systems because cortisol concentrations remain high rather than returning to resting levels following stressful experiences.

While neuroendocrinology has provided a strong foundation for research explaining *how* the physiological response to a stressor occurs, identifying the *origins* of the hormonal response is less understood. The integrated specificity model of stress (Weiner, 1992) provides a framework with which to understand the complex relationship between thoughts, emotions and the physiological response to stressors. This theory suggests that exposure to a stressful situation and cognitive appraisals of the situation shape the psychobiological response. When stress is perceived, the response is integrated and coordinated across multiple levels - cognitive, emotional and physiological (Figure 1). Specific events and the way a person appraises it can elicit different emotional and physiological experiences. Additionally, emotional responses have specific neural substrates, which can contribute to different alterations in the peripheral physiological system, dismissing the idea that people have uniform stress response (Kemeny, 2003). A meta-analysis of studies including acute laboratory stressors showed support for the integrated specificity model. The meta-analysis included 66 articles in which stress or emotions were manipulated and investigated cortisol responses. Results show that both emotions and appraisals were related to cortisol reactivity (Denson, Spanovic, & Miller, 2009). Specifically, studies that were associated with challenge and threat predicted larger effect sizes in terms of cortisol responses.

There is substantial individual variability in cortisol trajectory during a response to acute stress including individual variation in baseline concentrations, peak levels in response to stress and recovery following stressors (Kudielka et al., 2009). For example, the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) is a performance oriented, social-

evaluative stressor that consists of a speech followed by an arithmetic task in front of an audience. It consistently elicits a mild to moderate HPA response and is a well-validated measure used to examine stress reactivity in children, adolescents and adults (eg. Gunnar, Wewerka, Frenn, Long, & Griggs, 2009b). The physiological response to this task increases over the course of development, peaking in middle adolescence (15 years old) for both boys and girls (Gunnar et al., 2009b; Sumter et al., 2010).

Emotion regulation

There are many strategies for regulating one's emotions. Several emotion regulation strategies have been hypothesized to be risk factors for or protective factors against psychopathology. Theoretical models associate successful emotion regulation with positive outcomes (Brackett & Salovey, 2004; John & Gross, 2004). Research shows adaptive emotion regulation is associated with positive relationships and wellbeing as well as improved academic and work performance (Brotheridge & Grandey, 2002; Graziano, Reavis, Keane, & Calkins, 2007; Gross & John, 2003). Additionally, difficulties with emotion regulation are associated with mental disorders including major depressive disorder (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Rottenberg, Gross, & Gotlib, 2005), generalized anxiety disorder (GAD; Mennin et al., 2007), eating disorders (Bydlowski et al., 2005), and alcohol-related disorders (Sher & Grekin, 2007).

Associations between emotion regulation and stress physiology. Differences in emotion regulation may be related to differences in physiological responses to stress (Lazarus, 1984; Frijda, 1988). A meta-analysis of studies examining HPA axis activity to manipulated stress situations indicated that both emotions and appraisals were related to cortisol reactivity

(Denson et al., 2009). Specifically, studies in which participants appraised stressors as challenging, threatening, and novel predicted larger effect sizes in total cortisol output.

Adaptive and maladaptive emotion regulation strategies. According to their meta-analysis of studies that examine links between emotion regulation strategies and psychopathology, Aldao and colleagues (2010) identify three emotion-regulation strategies that have been widely accepted as adaptive because they are protective against psychopathology – reappraisal, problem solving and acceptance. In contrast, three other strategies have been argued to be maladaptive because they have been shown to be risk factors for psychopathology – suppression, avoidance and rumination. Overall, psychopathology appears to be related to the greater use of maladaptive emotion regulation strategies and lesser use of adaptive emotion regulation strategies (Garber, Braafledt & Weiss, 1995). The present study will examine the impact of 3 of these response-focused emotion regulation strategies – suppression, rumination and reappraisal - on participants' physiological response to a social-evaluative stressor.

Reappraisal. Cognitive reappraisal involves decreasing the emotional impact of a situation by changing how we think about it. This may include reframing or thinking about a stressful or emotional situation in a different way. While most theories predict that using reappraisal as an emotion regulation strategy should decrease physiological reactivity, some empirical studies show no relationship (Egloff et al., 2006; De Veld, Riksen-Walraven, & de Weerth, 2012; Gross, 1998) and others show increases in reactivity with reappraisal in late adolescence (Lam, Dickerson, Zoccola, & Zaldivar, 2009; Mauss et al., 2007). These inconsistent findings between reappraisal and physiological reactivity could be related to poor measurement of the process of reappraisal. For example, it is challenging to measure if a

subject's reappraisal of a situation was complete. They may attempt to reappraise a situation, but not fully do so and actually suppress or ruminate on the emotional content.

Suppression. Expressive suppression consists of inhibiting ongoing emotional expression in order to regulate emotion (Gross, 1998). It refers to the inhibition of external cues to one's internal emotional state (e.g., facial expression). Expressive suppression has been linked to indices of physiological reactivity in previous research. Studies consistently show that suppressing unpleasant emotions (disgust, sadness, embarrassment) leads to heighted sympathetic nervous system (SNS) activity, as indexed by cardiovascular measures (Egloff et al., 2006; Gross & Thompson, 2007; Harris, 2001). Other studies show higher levels of suppression associated with greater HPA activity, as indexed by the total cortisol output concentrations in response to the TSST (Lam et al., 2009; Mauss et al., 2007).

Rumination. Rumination consists of repetitively focusing on the causes and consequences of an experience (Nolen-Hoeksema et al., 2008). Rumination is a common, normal and sometimes functional response that isn't limited to people with psychological disorders (Watkins, 2008). However, trait rumination has been shown to be a precursor of future depression (Nolen-Hoeksema, 2000; Robinson & Alloy, 2003). While people report ruminating to understand and solve their problems (Papageorgiou & Wells, 2003), rumination has been shown to be negatively related to problem solving (Hong, 2007) and interferes with effective problem solving by immobilizing individuals in indecision (Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003).

The Perseverative Cognition Hypothesis (Brosschot, Gerin, & Thayer, 2006) proposes that repetitive, intrusive thoughts may amplify or maintain physiological responses to stressors. Thus, greater rumination can delay the physiological recovery from an acute stressor. Evidence

using cardiovascular measures show that a variety of rumination measures have been positively associated with elevation and/or delayed recovery from experimental stress manipulations (Brosschot et al., 2006; Glynn, Christenfeld, & Gerin, 2002). Increased trait rumination has been shown to predict increased stress reactivity (Gianferante et al., 2014; Zoccola et al., 2008). Rumination may prolong the activation of the negative emotional response leading to an extended HPA response and, over time, dysregulation of the HPA axis (Denson et al., 2009).

Denson and colleagues (2009) found that when they experimentally induced rumination after an insult, participants maintained high levels of cortisol for the duration of the experiment. Stewart and colleagues (2013) found that high trait rumination among adolescents suffering from depression was associated with delayed post-stressor HPA recovery, but high trait distraction and problem solving was associated with more rapid recovery in the same population. These researchers conclude that impaired post-stressor recovery may be a mechanism underlying the pathological effect of rumination on the development and maintenance of depression.

State vs. trait rumination. A recent review of 15 studies examining rumination and cortisol revealed that state and trait measures do not consistently predict the same patterns of HPA activity (Zoccola & Dickerson, 2012). Three studies measured state rumination after an acute stressor. One showed no association between state rumination and cortisol reactivity (Rudolph, Troop-Gordon, & Granger, 2011), in another greater rumination predicted cortisol post stressor only in men (Zoccola, Quas, & Yim, 2010) and the third reported greater state rumination predicted greater cortisol reactivity for all participants (Zoccola et al., 2008). A recent study not included in the 2012 meta-analysis showed that state rumination was positively associated with greater total cortisol output during the response to the stressor (Gianferante et al., 2014). In this study, higher state rumination was associated with both cortisol increases from

baseline to peak as well as sustained high cortisol level, after the stressor and this was independent of participants' trait rumination.

In contrast, trait rumination measures have been correlated with both increases in cortisol stress responses in some studies (Zoccola, Quas, & Yim, 2010) and decreases in others (Zoccola et al., 2008). Other work has failed to find any relationship between trait rumination and acute stress responses (Gianferante et al., 2014; Young & Nolen-Hoeksema, 2001). These inconsistencies in the literature point to a complex relationship between rumination and acute stress responses that warrants further investigation. The association between higher state rumination and cortisol output without an association to trait rumination suggests that it is important to measure both state and trait rumination when examining response and regulatory behaviors to acute stressors.

Overall, there is a need for more research to clarify the relationship between cognitive strategies used to manage the experience of stress and associated physiological responses.

The temporal dynamics of emotion regulation

It is likely that the inconsistency in the literature about the effect of emotion regulation strategies on the stress response are likely due to measurement inconsistencies (Lopez-Duran, Mayer, & Abelson, 2014; Zoccola & Dickerson, 2012). This dissertation seeks to address gaps in the literature by measuring multiple strategies of emotion regulation as well as utilize a unique approach to modeling participants' stress response profiles across time to elucidate how psychological strategies impact specific phases of the participants' stress response.

Affective chronometry. Although the cascade of physiological responses to stress is activated through the HPA in a similar manner across species, the variability among individuals in the intensity and duration of emotional reactions to similar challenges is striking (Davidson,

1998). Davidson (1992) refers to the differences in emotional reactions to similar motivators as *affective style* and argues that differences among humans are associated with temperament, personality and vulnerability to psychopathology. He maintains that *affective chronometry* – the time course of emotional responding - is an important feature of affective style and likely plays a key role in determining vulnerability to psychopathology. Individual differences in prefrontal asymmetry in the brain are associated with time course affective responding, particularly the recovery stage following emotional challenge (Jackson, Malmstadt, Larson, & Davidson, 2000).

Affective chronometry involves examining the temporal dynamics of emotion regulation. Evidence from multiple studies using a variety of physiological indices indicates that the prolonged recovery from a stressful event is more predictive of negative outcomes then the magnitude of the response to the stressor (e.g. Davidson, 2003; Brosschot & Thayer, 1998). While Davidson (2003) suggests time course variables are imperative to understanding individual differences in emotional reactivity and emotion regulation that may reflect vulnerability to psychopathology, studies examining the HPA response to acute stress and emotion regulation strategies have yet to apply this theoretical construct to their analytic approach. In addition to examining baseline levels as well as the peak amplitude of a response, Davidson (1998), proposes decomposing the response into its two components - the rise time to peak (reactivity phase) and the recovery time (recovery phase). Some studies have attempted to make conclusions about the time course of emotional responding from their analyses, but do not model cortisol trajectory appropriately to enable them to examine different phases of the response between individuals, as Davidson suggests. By modeling time in a multilevel model with only a linear and quadric term (Lam et al., 2009; Zoccola et al., 2008) or assuming all individuals reach their peak cortisol values at the same time (Burke, Davis, Otte, & Mohr, 2005;

Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013) does not allow for individual differences in response profiles.

To address this gap in the research, this study examines affective chronometry by testing whether emotion regulation strategies impact the reactivity or recovery phase of the HPA response to stress with a discontinuous multilevel model.

Adolescents

The current study investigates the relationship between three emotion regulation strategies and the stress response in a sample of mid-adolescent participants (mean age= 16.6 years). Adolescence is a time of transitions – biological, social and cognitive. It is a unique developmental period where changing social roles as well as cognitive and biological changes create a variety of possibilities and challenges for this group. Adolescence is also a period in which the long-lasting effects of earlier exposures to stress become evident as novel challenges multiply (Boyce & Ellis, 2005). Adolescents are at heightened risk for psychopathology such as mental illness (depression, anxiety, eating disorders) and substance abuse. However, unique characteristics including their heightened responsivity to stress and increasing susceptibility to peer influence contribute to the potential for competence building and wellbeing promotion.

The evidence is mounting that, after early childhood, adolescence is a second sensitive period for neural reorganization (Nelson, Leibenluft, McClure, & Pine, 2005; Gogtay et al., 2004) and since exposure to heightened stress during developmentally sensitive periods may alter physiological processes (Boyce & Ellis, 2005) and the stress system is constantly adapting to changes in the environment (Del Guidice, Ellis, & Shirtcliff), promoting skills to manage stress during this time could greatly promote resilience and long term mental health. However,

little is known about the associations between physiological stress response and the emotion regulation strategies that adolescents use to deal with the every-day stressors they encounter.

The stress response in adolescence

There are substantial shifts in stress reactivity during adolescence. Although adolescence is marked by many neuroendocrine changes, the shifts in the HPA function are more subtle then the increases in gonadal hormones associated with puberty. While basal levels of hormones of the HPA (e.g. ACTH and cortisol) remain fairly stable in adolescence and are similar to those of adults, the amount and duration of the release of these hormones during times of stress show significant changes from childhood into adolescence. Studies on rats show that males and females on the cusp of adolescence show longer responses, sometimes 45-60 minutes longer, than adults following stressors (Romeo, Bellani, Karatsoreos, Chhua, Vernov, Conrad, & McEwen, 2006). Recent studies in human adolescents also show changes in HPA responsiveness. Boys and girls in later stages of adolescence (15-17 years old) display greater peak cortisol levels after stress compared to children or early adolescents (9-13 years old) that are similar to adults HPA reactivity patterns (Gunnar, Wewerka, Frenn, Long & Griggs, 2009; Stroud et al., 2009).

The mechanisms that mediate these changes in hormonal responsiveness to stress in adolescence are not clear. However, it appears to include both the activation and the feedback phases of the HPA response different in adolescence as compared to children (Romeo et al., 2006). Researchers suggest that these changes are likely related to a number of factors including asymmetric development between limbic and frontal regions of the brain and increased sensitivity to pubertal hormones in the amygdala (Nelson et al., 2005; Scherf, Smyth, & Delgado, 2013). The prefrontal cortex and the hippocampus exert inhibitory control over the

stress system and activate it (Gunnar & Quevedo, 2007). Changes in these structures, under the influence of gonadal hormones, may contribute to the increasing sensitivity and responding of the stress system to social evaluation (Van den Bos, de Rooij, Miers, Bokhorst, & Westenberg, 2014). This peak may result from temporary imbalances in the system due to difference in the maturation rate between structures (Spear, 2009). Scherf et al. (2013) suggest that the increase in sensitivity to stress observed in adolescents may be amplified by amygdalar changes that instigate new connections with other areas of the brain thereby increasing the salience of social information.

Stress and psychopathology in adolescence. Although social, cognitive and biological changes are all typical in adolescence, many adolescents show great resilience in the face of challenges, while others show maladaptive coping (Masten, 2001). Nearly half of adolescents report difficulty coping with the stress they face (Gans, 2009). Spear (2009) proposed that the typical increase in stress response in adolescence is adaptive to support individuals as they face the significant cognitive and emotional stressors associated with this period. However, stress has also been linked to the onset of many psychological disorders in adolescence (Dahl & Gunnar, 2009; McClure & Pine, 2007; Paus, Keshavan & Giedd, 2008). Stress can affect individuals in different ways; the same types and levels of stress do not lead to dysfunction or psychopathology in all adolescents (Steinberg & Avenevoli, 2000). Preexisting genetic or other vulnerability can lead to brain development that is made more susceptible to disorder under conditions of stress (Alloy & Abramson, 2009).

Research shows a link between dysregulated stress response patterns and the emergence of psychopathology in adolescence. Dysregulated patterns of stress responsivity have been consistently observed in adolescents who demonstrate depression and other psychopathology

(Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Lopez-Duran, Kovacs, & George, 2009). Adolescents with moderate/severe depression exhibit a blunted HPA response following the TSST compared to adolescents with low levels of depressive symptoms (Harkeness, Stewart, & Wynne-Edwards, 2011). The link between psychopathology and dysregulated stress responsivity could be related to preexisting biological characteristics associated with risk of developing a disorder or consequences of the disorder (Adam, Sutton, Doane, & Mineka, 2008). In turn, it is challenging to determine the causal sequence of the emergence of increased psychopathology in adolescence.

While there is a much interest in preventing the mental health disorders that emerge in adolescence, it is challenging to determine when typical features of healthy development might become pathological. Alloy and Abramson (2009) suggest that increased brain maturation and cognitive competencies (metacognition, hypothetical thinking) in adolescence, may raise their risk for depression and anxiety because it is likely to lead to escalating rumination in challenging situations. On the other hand these increased cognitive competencies may also contribute to adolescents' ability to use adaptive emotion regulation skills, such as reappraisal (Steinberg, 2008). Reappraisal is thought to serve as important strategy for stress management and serve as a protective factor, helping adolescents cope with the new stressors that emerge during this period and contribute to their resilience (Masten & Coatsworth, 1998). Emerging research suggests that rumination and suppression increase the risks for negative health outcomes, including psychopathology, but the mechanisms for these relationships are not well understood (Gillespie & Nameroff, 2005; Thomsen, Mehlsen, Christensen, & Zachariae, 2003; Thomsen et al., 2004). While it is hypothesized that the use of maladaptive coping strategies in response to acute stressors might increase stress system dysregulation and contribute to the accumulation of

chronic stress over time, this has yet to be addressed in the literature with any population. Numerous studies have examined adolescent's stress reactivity (the difference between their peak physiological response and their baseline) or the total physiological output over the course of the response (area under the curve, AUC) with neuroendocrine measures. Very few studies have examined what occurs physiologically and psychologically after an acute stressor (e.g. Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013; Zoccola et al., 2010). This study is unique in examining reappraisal and suppression as well as state and trait rumination during a response to an acute stressor with adolescents.

Measuring the stress response in adolescents. Stressor paradigms that include a public speaking task such as the Trier Social Stress Test (TSST; Buske-Kirschbaum et al., 1997) are a common method of examining the physiological response to stress. The TSST is a performance oriented, social-evaluative stressor in which participants give a mock speech and perform an arithmetic task. It is a well-validated measure used to examine the physiological response to stress in children, adolescents and adults (e.g. Gunnar et al., 2009b). A typical pattern of response to the TSST includes a peak concentration of cortisol between 20-30 minutes after first learning about the task, then participants show a decline in concentrations of after their peak and some participants recover completely to their baseline concentration of cortisol (or sometimes lower) within an hour of the task (Nater et al., 2005; Dickerson & Kemeny, 2004).

The magnitude of the physiological response to an acute social-evaluative stressor increases over the course of development, peaking in middle adolescence (14-15 years old) for both boys and girls with adult like HPA responses continuing after that (Gunnar et al., 2009b; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010). Van den Bos and colleagues (2014) found that this increase in social-evaluative threat was related to the overall increases in cortisol

during adolescence. There are only a handful of studies that utilize social-evaluative stressors to examine the relationship between reappraisal, suppression, or rumination, and cortisol levels (e.g. Lam et al., 2009; Zoccola, 2008; Zoccola & Dickerson, 2012).

The GPST-A. The Group Public Speaking Task for Adolescents, GPST-A, a group version of the TSST was developed to elicit a physiological response to stress in adolescents that is less time-consuming and resource-intensive compared to the single-subject version of the task (Hostinar, McQuillan, Mirous, Grant, & Adam, 2014). A group TSST has been validated with adult populations (TSST-G; Von Dawans et al., 2011) but the GPST-A is a newly validated measure with adolescent populations. The GPST-A is carried out in groups of 5-8. Participants are in the same room, but separated by opaque dividers and called on randomly to present a speech about themselves to two judges. They are told that their performance will be evaluated and the judges are instructed to give no feedback and remain neutral during the task. When participants stand to speak, they present to the mock judges, but their group members are watching them from behind. Hostinar and colleagues (2014) found that the task elicited a significant increase in cortisol production in participants (approximately 60% above baseline) and in self-reported negative affect with no gender differences in reactivity. Researchers showed that this task is an effective and efficient way to examine adolescents' response to stress and concluded that five participants is the ideal group size to produce a uniform HPA response across individuals with the GPST-A (Hostinar et al., 2014).

This Study

The aims of this dissertation study are twofold: (1) To examine the feasibility and effectiveness of utilizing the GPST-A in a school setting to elicit a mild to moderate stress response in a group of adolescents, (2) Explore the relationships between adolescents' self-

reported emotion regulation strategies and their physiological responses to acute stress. Although affective chronometry may be a useful theoretical construct to guide research examining associations between self-reported emotion regulation strategies and the physiological response to an acute stressor, no studies have used this theoretical framework to create an analytic approach that allows researchers to separately examine the reactivity phase from the recovery phase after an acute stressor. In turn, I will examine the affective chronometry, (the temporal dynamics of emotion regulation), by analyzing the relationship between the concentration of cortisol and 3 self-reported regulation strategies – Suppression, Reappraisal and Rumination - separately during the reactivity and recovery phases of the stress response with the use of a discontinuous multilevel model.

Below are the hypotheses for the present study:

Reactivity Phase - Participants who report higher levels of trait rumination and suppression will have steeper cortisol reactivity slopes including higher peak values of cortisol.

Recovery Phase - Participants who report higher levels of state rumination will show less steep recovery slopes during the recovery phase of the stress response.

Overall Response - Participants who report higher levels of reappraisal will have lower baseline, peak and ending values of cortisol.

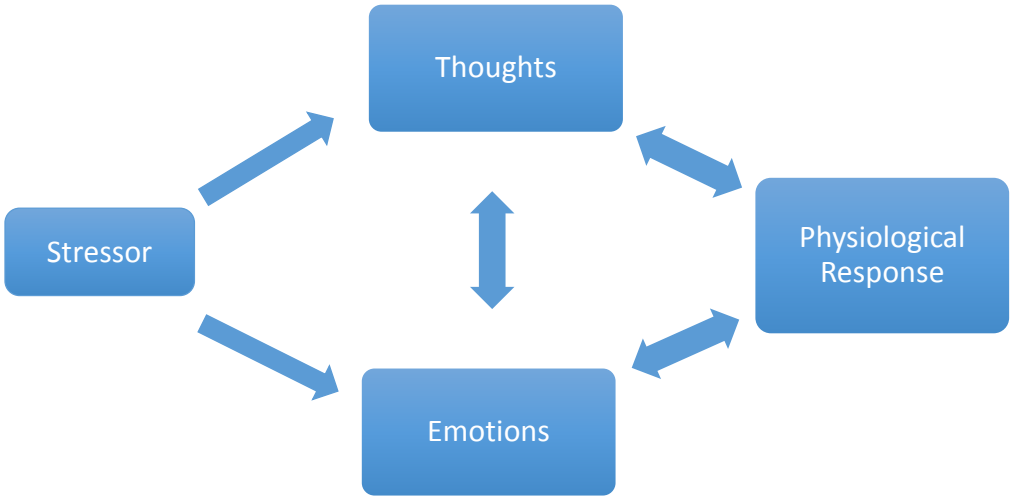

Figure 1. Psychological and physiological responses to stress. Stressful situations and cognitive appraisals of the situation shape the psychobiological response. The integrated specificity model posits that when stress is perceived, the response is integrated and coordinated across multiple levels - cognitive, emotional and physiological.

Chapter 2: Method

Recruitment and Participants

Full-time public high school students in the Central Dauphin School District in Pennsylvania currently enrolled in a regularly offered health class for students in grade 11 were recruited for this study. All participants spoke English fluently, had written consent from their parents, and provided assent to participate in the current study. No students were excluded from the study. Fifty-four adolescents (female=40, male=14, mean age=16.6) from two high schools participated in the study.

Procedure

All data collection procedures were conducted following a protocol approved by the Pennsylvania State University's Institutional Review Board**.** Data were collected through two methods: self-report questionnaires and in-person physiological assessment. The Group Public Speaking Task for Adolescents (GPST-A; Hostinar et al., 2014) was conducted after-school in a classroom in the school. Participants attended one 90-minute appointment and were part of 3-5 person groups. Data collection took place over a four-week period after school hours (2:30-4pm) to control for the normative diurnal decline in cortisol.

Each of the group members sat at a desk with opaque dividers between them (Figure 2). After giving assent, participants were instructed how to provide sample of saliva and gave a practice sample (Time 0). Then participants completed computer based questionnaires consisting of demographic information as well as cognitive and emotion measures during a 35-minute baseline period. At the end of this period, the baseline saliva sample (Time 1) was collected. Participants then heard the task instructions for the public speaking task.

The Group Public Speaking Task for Adolescents. This study utilized The Group Public Speaking Task for Adolescents (GPST-A; Hostinar et al., 2014). Participants were asked

to give a 1.5 minute speech and to imagine they were introducing themselves to new classmates. Participants were told that they would be called on randomly to present their speech to two judges who would be assessing their speech and video recording them. Then, the judges would leave the room, score them compared to their peers and return later to give them feedback. Research assistants acting as judges were trained to have serious looks and to provide no feedback during the speech task. When participants stepped forward to give their speeches to the judges, they stepped in front of their desks and beyond the opaque dividers where they could be seen by but not see, their peers (see Figure 2). Immediately after the speeches were completed, participants provided a saliva sample and then completed self-report measures assessing cognitive and emotional responses to the stressors (i.e., current emotion ratings, current thoughts). These measures took approximately two minutes to complete. Once finished participants were instructed to sit quietly to wait for the judges' feedback. After ten minutes participants provided another saliva sample and filled out the same self-report measures and then were told the judges were still deliberating. This occurred five times total (see Figure 3 for experimental timeline). After the final saliva sample and self-report, a trained research assistant told the participants that the judges were fake and that they were not actually scoring their speeches. Participants were allowed to ask questions about the study, were given $20 gift certificates and pizza at the end of the appointment. Participants were also entered in a drawing for an iPad mini.

Salivary Samples. Salivary cortisol was collected at seven points via passive drool before and after the GPST-A. Sampling procedure included 2 samples prior to GPST-A, and 5 additional samples at ~10 minute increments post-stressor to capture peak reactivity values and recovery (Figure 3). Participants were given one minute to complete their saliva samples with a

goal of 1ml of saliva. Studies utilizing a similar paradigm show clear effects of the task on hormone trajectories with sample sizes of N=20-60 (Dickerson & Kemeny, 2004). SAS Proc Power indicates that, assuming a modest effect size (*r*=0.35, Cohen, 1988), to power this study at 80% requires a sample size of N=52.

Cortisol assays were completed by the Biomarker Core Lab at the Pennsylvania State University (Dr. Laura Klein). Hormone concentrations in each of the six salivary samples (Time 1-Time 6) were determined using Salimetrics assay kits. The first sample (Time 0) was not assayed because it reflects cortisol response to a novel experience (coming into the appointment for the experiement) and not cortisol at rest. Duplicate cortisol values were averaged and then converted to nmol/L to produce values for analysis. For salivary assays, inter-assay covariances were less than 10%, and intra-assay covariances were less than 5%. To correct for skew and kurtosis, a log transformation was applied to cortisol values, and those log-transformed variables are used in analyses. All specimens collected were coded numerically and by color and de-identified so that Dr. Klein and her lab personnel did not have access to any identifying information.

Self-report Measures

Emotion regulation (ERQ-CA). The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is comprised of 10 items forming two subscales that assess the habitual use of two emotion regulation strategies: cognitive reappraisal (6 items, e.g. “When I'm faced with a stressful situation, I make myself think about it in a way that helps me stay calm”) and expressive suppression (4 items, e.g. “I keep my emotions to myself”). Participants rate the extent to which they agree with each statement on a Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree). The Emotion Regulation Questionnaire for Children and

Adolescents (ERQ-CA; Gullone & Taffe, 2012), is a revision of the adult measure. The ERQ-CA is a valid age-appropriate measure for investigating the use of 2 specific strategies of emotion regulation – suppression and reappraisal - during the childhood and adolescence developmental periods. The ERQ-CA was evaluated with a sample of 827 participants aged between 10 and 18 years (Gullone & Taffe, 2012). Results indicate sound internal consistency (Chronbach's α: suppression = 0.69, reappraisal = 0.65).

Rumination. Two types of rumination were measured in this study: trait rumination (an individual's general tendency to ruminate) and state rumination (the ruminative thoughts elicited by the stressor in the current study)(Table 1). Both trait and state rumination measures are related to heightened cortisol responses to a variety stressors (Zoccola & Dickerson, 2004). State rumination was measured two ways in this study: with the Thoughts Questionnaire (Zoccola et al., 2008) and with a new measure created for this study, the Momentary Rumination questionnaire.

State rumination. The Thoughts Questionnaire (Zoccola et al., 2008) was used to measure state rumination post stressor at Time 6, ~40 minutes post stressor. This stressor specific measure has been used in previous studies to assess state rumination after a social evaluative stressor (Edwards, Rapee, & Franklin 2003, Zoccola et al., 2008). The original version of the Thoughts Questionnaire was designed to assess rumination after a laboratory speech task during two subsequent weeks (Edwards et al., 2003). I used a version created by Zoccola and colleagues (2008) that modified the questionnaire by asking participants to indicate how much they thought about the following statements in the time since their speech ended, using a 5-point scale ranging from never (0) to very often (4). The Thoughts Questionnaire contains both negative and positive thoughts subscales. The 14-item negative thoughts subscale

was used for analyses in this study. Examples of rated statements included: "How often did you think about how bad your speech was?" and "How often did you think that you must have looked stupid?". A higher score indicates more rumination and there was high internal consistency for this scale (α =0.93).

***Momentary rumination*.** The Momentary Rumination questionnaire was developed for this study to test a state measure that was stressor specific like the Thoughts Questionnaire, but even more 'in the moment'. This measure was used as a repeated measure at the same time that saliva samples were collected post-stressor (every 10 minutes, see yellow arrows, Figure 3). This stressor specific scale included items about what the participant was thinking at the moment with neutral, positive and negative valences. Statements began with 'Right now I am…' and the scale ranged from Not at all (0) to A Lot (5). Two statements had a positive valence (e.g. '…thinking about how confident I was during my speech.') and 4 had a negative valence (e.g. '…thinking how bad my speech was'.) and one had a neutral valence ('…replaying how I acted during the speech'). Based on the definition of rumination put forth by Nolen-Hoeksema and colleagues (2003) as 'perseverative thoughts about past oriented, unwanted emotions', the momentary rumination measure consists of the following items (one neutral and five negative) summed with higher scores indicating more state rumination (Cronbach's alpha ranged from 0.76-0.91for each of the five times of assessment):

Replaying how I acted during the speech
Thinking about how bad my speech was
Thinking that I didn't make a good impression
Thinking about how self-conscious I felt during the speech
Dwelling on things I could have done better/differently during the speech

The two positive items created another subscale called 'positive thinking' and had alphas ranging form 0.66-.93 at each of the five times of measurement.

Trait rumination. The rumination subscale of the Responses to Stress Questionnaire (RSQ; Connor-Smith, Compas, Wadsworth, Thomsen & Saltzman, 2000) was used to measure participants' trait rumination. RSQ assesses both involuntary and voluntary responses to stress. It begins with a checklist of stressors that pertain to a specific stressful situation involving one's peers, which the participant rates in terms of how often each stressor has occurred in the recent past. This checklist is the perceived stress subscale. For the rest of the questionnaire, participants rate how often they use each coping method or experience each type of involuntary stress response on a scale of 1 (Not at all) to 4 (A lot). Items on this subscale included: 'When problems with my peers come up, I can't stop thinking about how I am feeling', 'When I have problems with my peers I can't stop thinking about what I did or said.' and 'When I have problems getting along, I can't stop thinking about why they happened to me'. A proportion score was created for the rumination subscale (item/RSQ total) to control for individual differences in rates of endorsing items. Cronbach's alpha for this 3-item scale was 0.58.

Positive and Negative Affect. Each time a saliva sample was taken post-stressor, participants were asked how nervous, excited, angry, happy, embarrassed, confused, and sad, they were feeling at that moment on a scale from 1 (not a all) to 5 (very). This measure was used to verify that the stress paradigm elicited an emotional response from participants and to provide a general measure of participants' affect during each phase of their response to the stressor. Negative and positive items were aggregated together and averaged to create negative and positive affect scales. The internal consistency for the negative affect scale ranged from α=0.6-0.8 and the positive affect scale ranged from α =0.6-0.8 at each time point it was collected.

Depression - PHQ-8 Depression Severity (Spitzer, Kroenke, & Williams, 1999) was used to assess depressive symptoms. Participants were asked how often they have been bothered by

the following problems in the last 2 weeks. Participants rated how much they have been bothered by each problem: 0 (not at all), 1 (several days), 2 (more than half the days), 3 (nearly every day). PHQ-8 total score is calculated to determine a depression severity score. A sum score of 10 or greater suggests Major Depressive Disorder (MDD). The internal consistency for this scale was $\alpha = .82$.

Anxiety. The GAD-7 Anxiety Severity (Löwe, Decker, Müller, Brähler, Schellberg, Herzog, & Herzberg, 2008; Spitzer, Kroenke, Williams, & Löwe, 2006) was used to assess symptoms of general anxiety among participants. Participants were asked how often they have been bothered by the following problems in the last 2 weeks. Participants rated how much they have been bothered by each problem: 0 (not at all), 1 (several days), 2 (more than half the days), 3 (nearly every day). GAD-7 is a screening and severity measure for generalized anxiety disorder. GAD-7 total score for the seven items ranges from 0 to 21. Sum scores of 10 or above are used as a cut point for identifying an anxiety disorder (Spitzer, Kroenke, Williams, & Löwe. 2006). The internal consistency for this scale in this study was $\alpha = .91$.

Perceived Stress. The perceived stress subscale of the Responses to Stress Questionnaire (RSQ; Connor-Smith, Compas, Wadsworth, Thomsen & Saltzman, 2000) was used to measure participants' perception of stress. This scale consists of a checklist of 7 stressors. Participants identified how stressful those items have been for them in the past 6 months on a scale from 1 (Not at all) to 4 (Very). A total score was created for this scale and the internal consistency was $\alpha = .75$.

Adolescent Stress Questionnaire. Two subscales of the Adolescent Stress Questionnaire (ASQ, Byrne, Davenport & Mazanov, 2007) were used to assess participants' perceived levels of stress: school stress and peer stress. Participants rated how stressful 6 school items were to them

on a scale from 1 (Not at All Stressful for me) to 5 (Very Stressful for me). Some of the school items included: 'Keeping up with schoolwork' and 'Teachers expecting too much from you'. The peer items utilized the same scale and included 'Being hassled for not fitting in' and 'Disagreements between you and your peers'. Items for each scale were summed and their internal consistencies were high (α=.89 & .92).

Medication Survey. Participants were asked to report any medications that they are currently taking including the dosage and the most recent administration. Oral contraceptives and steroid medications can alter the levels of circulating stress hormones and therefore need to be controlled for in analyses.

Statistical Analyses

Because cortisol samples were nested within persons, who were nested within GPST-A groups, a three-level hierarchical growth curve analysis was used to adjust for the non-independence of observations associated with nesting. This approach permits the simultaneous modeling of the shape of the cortisol response profile for each individual along with person-level and group-level factors predicting differences in these rhythms.

Previous studies have utilized *Time* and $Time^2$ variables at level 1 to capture the curvilinear nature of the cortisol response (e.g., Hostinar et al., 2014). However, recent literature indicates that this approach ignores a critical source of variability in salivary cortisol responses: individual and group differences in the time latency of post-stress peak concentrations (Lopez-Duran, et al., 2009; Lopez-Duran et al., 2014). To address this *peak latency* among participants as well as examine affective chronometry in response to stress, I used a discontinuous multilevel model with landmark registration (Singer & Willet, 2003; Lopez-Duran et al., 2014). This approach allows one to examine how independent variables affect the different phases of the

stress response (i.e., baseline, reactivity and recovery) separately while accounting for peak latency. The models were fitted using HLM software, version 7 (Raudenbush & Bryk, 2002).

Creation of variables for landmark registration. To carry out the discontinuous multilevel model with landmark registration, first I identified each participant's peak cortisol value in the data. Then, based on Singer and Willet's (2003) recommendation, I created a 'CortInflect' variable that was dummy coded as 0 when the participant was in their reactivity phase and at their peak and coded as 1 once they had reached their peak and were in their recovery phase. From there, I created the adjusted time variables for each participant. The **Reactivity** variable is the total time in minutes up until and including the participants' peak cortisol value. **Recovery** is the total time in minutes after the peak cortisol value (Table 2).

Modeling. The level 1 model represents individual change in cortisol as a function of time. Cortisol values were log transformed in order to reach normal distribution. Cortisol concentration, $\boldsymbol{LgCortisol_{ijk}}$, is the outcome at time *t* for Person *i* in group *j*. π_{0ij} , the intercept, represents an estimate of each person's average cortisol level at baseline. $\boldsymbol{Reactivity_{ijk}}$, is an estimate of linear change in a person's cortisol reactivity up until and including when they reach their peak cortisol value. $\boldsymbol{Recovery_{ijk}}$, is an estimate of linear change from a person's peak cortisol value to the end of the appointment.

Level 1 Model. The level 1 model was first fit across all subjects to demonstrate a cortisol response pattern:

$$\boldsymbol{LgCortisol}_{ijk} = \pi_{0jk} + \pi_{1jk}*(\boldsymbol{Reactivity}_{ijk}) + \pi_{2jk}*(\boldsymbol{Recovery}_{ijk}) + e_{ijk}$$

The variance components for the intercept were significantly different from zero (intercept= 0.18, $p<.001$; Reactivity=0.00022, $p<.001$; Recovery= 0.00005, $p<.001$) indicating that adding predictor variables could explain some of the variance.

In an one alternative model, momentary rumination values were used as level 1 variables to investigate the changes in state rumination at the same time as cortisol samples were taken to examine if participants' state rumination profile might be associated with participants' cortisol response profiles. Measuring momentary rumination at the same time as salivary samples were taken allows me to use it as a level 1 variable and represents individual change in cortisol concentration as a function of state rumination. However, initial analyses revealed that participants' state rumination profiles were not associated with changes in cortisol over time and so momentary rumination was not used as a level 1 variable in final models.

In two other alternative models, the *Reactivity* (total minutes up to and including peak cortisol value) and *Recovery (*Total minutes post-peak cortisol value) variables were centered at zero so that the intercept in these models could be interpreted as either the peak or the end cortisol value (instead of the baseline value). Centering the Level-1 variables in this way allowed me to examine if the predictors were associated with participants' peak and ending values of cortisol. In these models, the intercept could be interpreted as the peak cortisol value and the ending cortisol values when each was centered at zero.

Level-2 Model. The level two model represents the between subject differences in the cortisol response patterns as predicted by independent variables (e.g., rumination, suppression, reappraisal):

$$\pi_{0jk} = \beta_{00k} + \beta_{01k}*(Sex_{jk}) + \beta_{02k}*(Variable\ of\ Interest_{jk}) + r_{0jk}$$
$$\pi_{1jk} = \beta_{10k} + \beta_{11k}*(Sex_{jk}) + \beta_{12k}*(Variable\ of\ Interest_{jk}) + r_{1jk}$$
$$\pi_{2jk} = \beta_{20k} + \beta_{21k}*(Sex_{jk}) + \beta_{22k}*(Variable\ of\ Interest_{jk}) + r_{2jk}$$

Initial analyses tested whether speech order, steroidal medication use, symptoms of depression, symptoms of anxiety or somatic symptoms were associated with cortisol response patterns. Previous research has demonstrated that sex is a significant predictor of baseline cortisol and was therefore initially included in all models (Kajante & Phillips, 2006; Kirschbaum

& Hellhammer, 1989; Kudielka & Kirschbaum, 2005). Initial analyses also tested whether speech order, race, group size, steroidal medication use, anxiety or depressive symptoms were associated with cortisol response patterns. Speech order, race, group size (3, 4 or 5), and steroidal medication use were not significant predictors of baseline cortisol levels or cortisol response patterns and, therefore, were not included in the final models. Sex had a significant influence on cortisol secretion and was therefore included as a covariate in all models. Depressive symptoms and anxiety also revealed significant associations with cortisol response patterns and, therefore, are included as predictors in the final models. To test hypotheses about emotions regulation strategies effecting participants' cortisol response profiles, rumination, suppression and reappraisal scores were entered at level two with sex. All variables included in the interactions were grand mean centered to reduce the possibility of collinearity and to facilitate interpretation of the results.

Level-3 Model. The third level of the model tested to see if group membership had an effect on cortisol response patterns:

$\beta_{00k} = \gamma_{000} + u_{00k}$

While there were no predictors at this level, including it is important because participants are nested in peer groups and it is important to control for this aspect of the study.

Table 1.
Rumination Measures

State Rumination	A. **Momentary Rumination** *Momentary Rumination Scale* - 5 items about what the participant is thinking at that moment. Scale made for this study.
	B. **State Rumination** *Thoughts Questionnaire* (Zoccola et al., 2008) 24 items scale asking about thoughts since speech ~45 minutes after speech task.
Trait Rumination	Rumination Subscale of RSQ 3 items, Responses to Stress Questionnaire (Compas et al., 2001)

Table 2.
Example of data in long format with adjusted time variables

Participant	Sample	lgCort	Time	CortInflect	Recovery	Reactivity
2	2.1	1.94	0	0	0	0
2	2.2	**2.15**	13	0	0	13
2	2.3	2.02	22	1	9	13
2	2.4	1.88	32	1	19	13
2	2.5	2.04	41	1	28	13
2	2.6	1.84	51	1	38	13
3	3.1	1.38	0	0	0	0
3	3.2	1.52	13	0	0	13
3	3.3	**1.74**	22	0	0	22
3	3.4	1.64	32	1	10	22
3	3.5	1.56	41	1	19	22
3	3.6	1.5	51	1	29	22

Note. Time = minutes since baseline sample (S1), lgCort = log transformed cortisol value in nmol/L units, **Bolded** value = Peak cortisol, CortInflect= the inflection point of the participant's cortisol response, 0=samples up until their peak cortisol and including the peak, 1= recovery period. CortRecover = the minutes since peak, the recovery period of the response participant X; CortReact = the minutes to the peak, the reactivity period of the response for participant X.

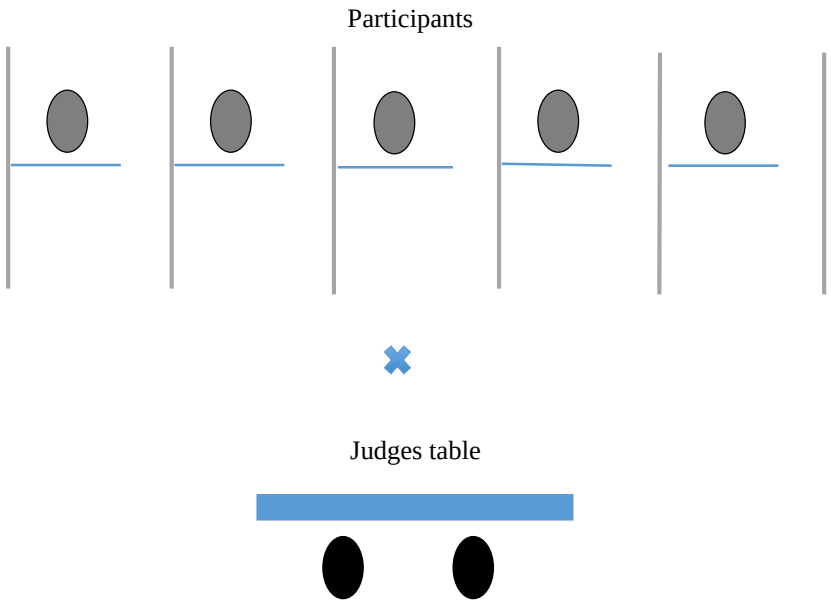

Figure 2. GPST-A room layout.

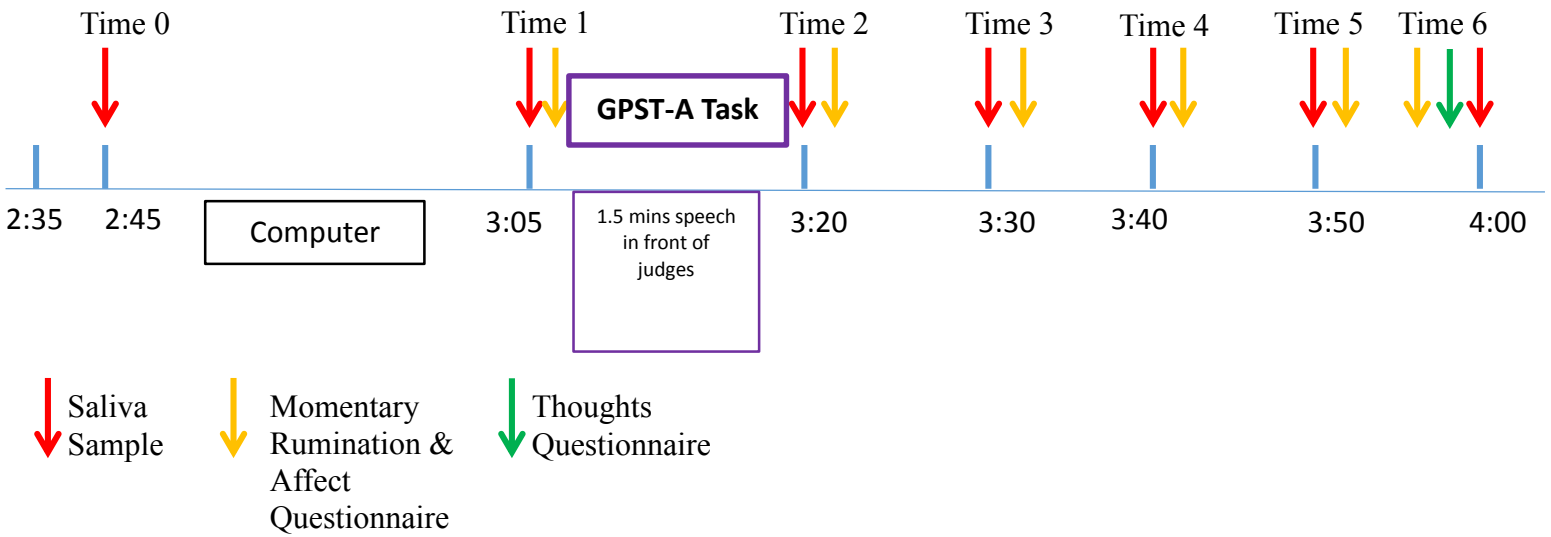

Figure 3. Experimental timeline. Times are +/- 10 minutes depending on arrival time.

Chapter 3: Results

Descriptive statistics

There was a small amount of data missing in this study. In addition to 2 participants not completing the Generalized Anxiety Disorder (GAD) questionnaire, one group was given incomplete questionnaire packets that resulted in 5 participants missing GAD data and 3 participants missing Adolescent Stress Questionnaire (ASQ) data.

Non-Repeated Measures. Descriptive analyses were performed on all variables and showed normal distributions for all self-reported measures. Table 3 shows means and standard deviations for all non-repeated, self-reported measures. Mean anxiety and depression scores ($M_{anxiety}$=7.3, SD=5.8; $M_{depression}$=7.2, SD= 4.7) were within typical ranges for this population and well below the clinical cutoff points for GAD or MDD (<10, Richardson et al., 2010; Löwe et al., 2008). Mean reappraisal and suppression scores (M_{reap}=3.1, SD=0.9; M_{supp}=3.9, SD 1.1) were also typical for this age group (Gullone & Taffe, 2012). Mean scores on the peer stress and school stress subscales were different from those reported by the ASQ scale makers. In this sample, the mean peer stress score was ~5 points lower (M=10.2, SD=5.5) and the mean score for the school stress was ~4 points higher (M=21.9, SD =5.8) than the means reported by the creators of the scale (Byrne, Davenport & Mazanov, 2007). This indicates that participants in the current study reported experiencing less peer stress and greater school stress then participants in the study by Byrne and colleagues (2007). The mean for the state rumination in this study was 30.3 (SD=13.8), which was considerably higher then the mean reported in the one previous study that utilized this questionnaire (M=18.1, SD=12.9, Zoccola et al., 2008) which could be attributed to difference in age between the two studies (16.8 years vs. 20 years) or the context of the protocol (school setting vs. laboratory setting).

There were many significant associations between self-reported variables. Females (dummy coded: female=1, males=0) reported more symptoms of depression (r= 0.28, p<.05) and higher school stress (r=0.29, p<.05) than boys. Table 4 presents partial correlations between non-repeated, self-reported measures, partialing out sex. As expected, depression and anxiety scores were significantly, positively correlated (r=0.78, p<.01) and all the stress measures were positively correlated with one another. Self-reported symptoms of anxiety were positively associated with all the measures of stress (perceived stress: r=0.37, p<.05, peer stress: r=0.46, p<.01, school stress: r=0.57, p<.01 ,) as well as suppression (r=-0.37, p<.05*)* and state rumination (r=0.48, p<.01) but not trait rumination (r=-0.001, p>.05). Higher reports of depressive symptoms were also positively associated with higher scores on all the measures of stress (perceived stress: $r=0.37$, p<.01, peer stress: $r=0.47$, p<.05, school stress: $r=0.57$, p<.01) as well as suppression (r=-0.28, $p<0.1$*)* and state rumination (r=0.48, p<.05) but not trait rumination (r=-0.001, p>.05). The lack of association between the trait rumination measure and depression and anxiety measures is unexpected and raises the question of the construct validity of the trait rumination measure. Higher reports of school stress was associated with higher state rumination (r=0.38, p<.01). Contrary to previous studies that show positive correlations between suppression and trait rumination (Moulds, Kandris, Starr, & Wong, 2007; Nolen-Hoeksema & Morrow, 1993; Wenzlaff & Luxton, 2003), suppression was negatively associated with both state and trait rumination measures in this sample (state rumination scores & suppression: r= -0.45, p<.05; trait rumination scores & suppression: r= -0.29, p<.05*).* Lastly, and surprisingly, reappraisal was not significantly associated with any measure.

Repeated measures. There were 3 measures that were repeated at multiple time points in this study: affect, momentary rumination, and cortisol concentration. Table 5 presents the

means and standard deviations for the self-reported repeated measures – momentary rumination and affect and Table 6 included correlations between momentary rumination and non-repeated self-report measures. Cortisol values were positively skewed at each time point so data was log transformed and transformed values were used in all analyses. Table 7 includes raw and log transformed cortisol means and standard deviations for the entire sample and for males and females separately because previous literature indicates gender differences in cortisol responses (Gunnar et al., 2009; Kudeilka & Kirschbaum, 2005). Momentary rumination is positively associated with state rumination at each measurement time (rang from r=0.41 to r=0.75). Suppression is negatively correlated with momentary rumination at each time point except Time 5.

The associations between cortisol and time varying self-reported variables are analyzed below in Aim 1.

AIM 1: Feasibility and Effectiveness of the GPST-A

The first aim of this study was to examine the feasibility and effectiveness of utilizing the GPST-A in a school setting. To test this, I performed a repeated measures ANCOVA to examine if the task elicited a mild to moderate cortisol response, then examined participants' change in momentary rumination over time to see if the task was successful at eliciting rumination, and finally examined participants' change in affect over time to see if participants' revealed psychological discomfort with the task. Results show that the stressor was effective at eliciting a physiological response and rumination as well as a change in a change in affect.

Group cortisol response. A repeated measures ANCOVA revealed a significant change in cortisol concentration over Time *($F_{5,49}=12.6$; $p<.001$;* Figure 4). The average cortisol increase post stressor was 50% over baseline levels, which is within the range typical of single-subject

TSST studies with adolescents (Gunnar et al., 2009). There are no clear guidelines for determining a definite cortisol response, but 10% increase has been previously used as a meaningful cut-off (Gordis et al., 2006) and 78% (N = 42) of the sample exceeded this threshold. Twelve of the 54 participants did not show a 10% increase. Three of those 12 participants started high at baseline and declined to a lower ending level, one participant started high, declined, and then went back up (U shaped), and the other eight showed an increase in cortisol post stressor but did not meet the 10% increase from baseline to cut-point. Males had significantly higher mean levels of cortisol than females at every time point, which is consistent with cortisol research in adults (Nicolson, Storms, Ponds, & Sulon, 1997) (Figure 5).

On average, people reach peak cortisol level 21-30 minutes post stressor (see Dickerson & Kemeny, 2004 for a review). In this sample, 65% percent of participants peaked within the typical time course of peak HPA axis activation (21-30 minutes post stress, at Time 3 or Time 4 in this study, see Figure 6). However, 18 of the 54 participants (35%) did not peak during that time period.

Twenty-four of the 54 participants (44%) returned to their baseline levels of cortisol by the final sample and 30 participants (56%) did not (Figure 7). Whether participants' cortisol recovered to their baseline was not associated with any of the predictor variables (e.g., positive and negative affect, sex, emotion regulation strategies). The only trend that emerged was intuitive: individuals who peaked early were more likely to recover (r=0.41, p<0.01) and those who peaked late were less likely to recover (r= -0.46, p<.01).

Momentary rumination. Participants reported their level of ruminative thoughts repeatedly, at 10 minute increments post stressor from Time 2 to Time 6. A repeated measures ANCOVA showed that the task was successful at eliciting rumination and that, on average,

ruminative thoughts decreased across the five post-stressor time periods ($F_{2.6,129.8}$= 75.8, p<.001; see Figure 4). Group means indicate that momentary rumination was highest immediately following the GPST-A (M=17.8, SD=4.6) and decreased by more than 50% by the end of the appointment (M=8.4, SD=4.1). Momentary rumination steadily decreased following the GPST-A (Time 2- Time 6; M=17.8 to 7.3).

State rumination and momentary rumination were positively correlated at each time point (Time 2- Time 6: r=0.41 to 0.75, p<.05). However, trait rumination was only correlated with a participants' momentary rumination score immediately following their speech (Time 2, r=0.33, p<.05) and not with state rumination.

Affect. Figure 8 shows participants' average change in positive and negative affect from Time 1 to Time 6. Repeated measure ANCOVAs reveal there was a significant change in both positive and negative affect over time (Positive Affect: $F_{5,48}$= 9.6, p<.001 Negative Affect: $F_{5,46}$= 33.2, p<.001). Participants reported a significant decrease in positive affect, t(112)=-1.46, p<0.01, from Time 1 to Time 2) and stayed low for the rest of the appointment (Figure 8). Participants reported a significant increase in negative affect, $t(112)=4.97$, p<0. 01 from Time 1 to Time 2 (Figure 4). Negative affect declined after Time 2 and ended significantly lower then baseline levels (Time 6, 1.2 ±.06, p<.001).

In summary, the school-based data collection protocol was successful at eliciting a cortisol response, rumination and change in affect for participants. Together, these results indicate that the GPST-A was both feasible and effective in this study.

AIM 2: Explore Affective Chronometry

The second aim was to examine the affective chronometry of the stress response by examining the relationship between 3 phases of the response – baseline, reactivity and recovery

and 3 emotion regulation strategies – reappraisal, suppression and rumination. First, change scores were calculated with cortisol values (participants' peak cortisol value – baseline cortisol, peak cortisol - end cortisol, and end cortisol – baseline cortisol) and those were correlated with self-reported measures. The three participants who did not peak post stressor were not included in these analyses.

Partial correlations controlling for sex revealed three significant correlations: increased suppression was associated with a smaller change in cortisol concentration from their peak to their ending value (r=-0.29, p<.05) indicating a less efficient recovery post peak. Participants with higher trait rumination scores and higher reported stress associated with school were more likely to return to their baseline cortisol values (r=-0.39, p<.05; r=-0.32, p<.05).

Next, to examine the time course of the stress response, a discontinuous multilevel model was employed to test the hypotheses that emotion regulation strategies – suppression, reappraisal and rumination – may affect different phases of the stress response. To address my 3 hypotheses, the results from the discontinuous multilevel model are divided into the three major phases of the stress response - baseline, reactivity and recovery. **Baseline** is a participants' starting cortisol value before the public speaking task (Time 1). The **Reactivity Phase** includes the slope between each participant's baseline value (Time 1) and their peak (referred to as *reactivity slope*) and their peak values of cortisol. The **Recovery Phase** includes the slope from a participant's peak value to their ending cortisol value (referred to as *recovery slope*) and participants' ending cortisol values (Time 6).

The following variables were explored in the discontinuous multilevel model:

Level 1: Minutes to peak (Reactivity), Minutes post peak (Recovery)
Level 2: Sex, Speech Order, Depression, Anxiety, State Rumination, Trait Rumination, Average Momentary Rumination, Perceived Stress, School Stress, Peer Stress .

Level 3: Group membership

Null model. $\text{LgCortisol}_{ijk} = \pi_{0jk} + \pi_{1jk}*(Reactivity_{ijk}) + \pi_{2jk}*(Recovery_{ijk}) + e_{ijk}$

Echoing the repeated measures ANCOVA analyses, results from the null model indicated that the GPST-A protocol elicited a typical cortisol response pattern in participants. There was a significant rise from baseline (γ_{000}= 1.44, SE= 0.07, p<0.001) to peak (γ_{100}= 0.015, SE=0.002, p<0.001) and a decline post peak (γ_{200}= -0.011, SE= 0.001, p<0.001)(Table 7).

Sex. Previous literature and descriptive analyses above indicate that males have higher cortisol values overall compared to females. Therefore, sex was used as the first level 2 predictor variable following the test of the null model (Table 8). Results show that females had lower baseline levels of cortisol (β=-0.46, SE=0.12, p<0.001), lower peak values (β=-0.60, SE=0.15, p<0.001) and lower ending values (β=-0.56, SE=0.15, p<.001) compared to males. Females also had less steep recovery slope post stressor (β=0.01, SE=0.003, p<.001), which was likely driven by their lower peak values. Since multilevel analyses indicated that sex had a significant effect on participants' stress response profiles, it was controlled for in all subsequent models.

Next, to test my hypotheses related to affective chronometry, I examined the role of self-reported measures of stress (i.e., perceived stress, school stress, peer stress) and emotion regulation strategies (i.e., suppression, reappraisal and rumination) in predicting cortisol response profiles. Due to the small sample size, each self-reported measure was entered as a Level 2 variable in separate models along with sex in order to control for the possible effects of sex on each of the predictors. In addition to examining associations between baseline levels and predictor variables, associations with peak and ending levels of cortisol were assessed by

centering the Level 1 Time variables in subsequent models. All models included random effects for each term at Level 2 and random intercepts for Level 3.

Level 1 equation:

$$\text{LgCortisol}_{ijk} = \pi_{0jk} + \pi_{1jk}*(\textbf{\textit{Reactivity}}_{ijk}) + \pi_{2jk}*(\textbf{\textit{Recovery}}_{ijk}) + e_{ijk}$$

Level 2 equations:

$$\pi_{0jk} = \beta_{00k} + \beta_{01k}*(Sex_{jk}) + \beta_{02k}*(Predictor\ Variable_{jk}) + r_{0jk}$$

$$\pi_{1jk} = \beta_{10k} + \beta_{11k}*(Sex_{jk}) + \beta_{12k}*(Predictor\ Variable_{jk}) + r_{1jk}$$

$$\pi_{2jk} = \beta_{20k} + \beta_{21k}*(Sex_{jk}) + \beta_{22k}*(Predictor\ Variable_{jk}) + r_{2jk}$$

The significant results of these analyses are reported by phase to explore the affective chronometry of participants' responses. Table 9 contains a complete summary of the results from these analyses.

Baseline cortisol. Higher reports of depressive symptoms were associated with lower baseline levels of cortisol (β= -0.03, SE=0.01, $p<.05$). Higher momentary rumination measured at Time 3 (≈10 minutes post GPST-A), Time 4 (≈20 minutes port GPST-A) and Time 5 (≈30 minutes post GPST-A) were all associated with higher baseline levels of cortisol. Time 3 results were significant and Times 4 and 5 were trend level (Time 3: β=0.02, SE=0.01, $p<.05$; Time 4: β=0.02, SE=0.01, $p<0.1$; Time 5: β=0.03, SE=0.01, $p<0.1$).

Reactivity phase. Higher rates of depressive symptoms and symptoms of anxiety were associated with lower peak values of cortisol (β= -0.04, SE=0.01, $p<.05$; β= -0.02, SE=0.01, $p<0.1$). Higher reported levels of stress associated with school was predictive of lower peak levels of cortisol (β= -0.03, SE=0.01, $p<.05$). State and trait rumination had opposite effects on the cortisol reactivity slope than hypothesized. Higher state rumination (≈40 minutes post speech) was associated with a steeper cortisol reactivity slope (β=0.0004, SE=0.03, $p<0.05$).

Figure 9 shows the steeper slope of cortisol reactivity associated with higher state rumination. In contrast, higher trait rumination was associated with a less steep reactivity slope (β=-0.51, SE=0.2, $p<.05$). There was also a positive association between momentary rumination at Time 3 (≈20 minutes post stressor) and the reactivity slope (β=0.001, SE=0.0005 $p<.05$). Contrary to my first hypothesis, suppression was not associated with the cortisol reactivity slope (β=-0.003, SE=0.0002 $p>0.1$).

Recovery phase. Two self-reported measures of stress showed trend level associations with the recovery phase. Higher perceived stress and higher school stress were both associated with less steep recovery slope (Perceived Stress: β=0.0006, SE=0.0002, $p<0.1$; School Stress: β=0.0004, SE=0.0002, $p<0.1$), indicating a less efficient or delayed physiological recovery from the GPST-A. Increased reports of depressive symptoms were associated with lower ending values of cortisol (β=-0.03, SE=0.01 $p<.05$). Higher levels of trait rumination were associated with lower ending values of cortisol (β=-11.42, SE=5.4, $p<.05$). Higher momentary rumination reported immediately after the GPST-A (Time 2) showed a trend level association with lower ending cortisol values (β=-0.02, SE=0.01, $p<0.1$). However, neither measure of state rumination was associated with a delayed cortisol recovery (momentary rumination: β=-0.0001 to -0.00006, SE=0.0003 to 0.0004, $p>0.1$; state rumination: β=-0.0001, SE=0.13, $p>0.1$).

Summary

While there were interesting findings, none of them support the original hypotheses:

Hypothesis 1 - Reactivity Phase: *Participants who report higher levels of trait rumination and suppression will have steeper cortisol reactivity slopes including higher peak values of cortisol.*

In this study, the cortisol reactivity slope was operationalized by the rate of increase in cortisol from participants' baseline to peak value of cortisol post-stressor in a multilevel level

model. Trait rumination was associated with a less steep reactivity slope and also higher baseline cortisol. Reports of suppression were not associated with the cortisol reactivity slope. Suppression was negatively correlated with the change in cortisol concentration from peak to end and negatively correlated with momentary rumination, state rumination and trait rumination.

Hypothesis 2 - Recovery Phase: *Participants who report higher levels of state rumination will show less steep recovery slopes during the recovery phase of the stress response.*

The recovery slope was operationalized by the rate of decline from the participants' peak cortisol value (which occurred at different times) to the last sample of cortisol taken at the end of the appointment (Time 6). Both state measures of rumination predicted elevated cortisol reactivity, and showed no association with the recovery slope. Momentary rumination ~20 minutes post speech was predictive of a steeper cortisol reactivity slope. Higher state rumination (~40 minutes post speech, Time 6) was also associated with a steeper cortisol reactivity slope. Both measures of state rumination were positively associated with the reactivity slope, but not the recovery slope as predicted.

Hypothesis 3 - Overall Cortisol Response: *Participants who report higher levels of reappraisal will have lower baseline, peak and ending values of cortisol.*

Reappraisal was not associated with baseline, peak or ending values of cortisol nor the reactivity or recovery slopes. Therefore, this hypothesis was not supported by the data.

Table 3.
Means and standard deviations for non-repeated, self-reported measures.

	N	Mean	SD	Min	Max
Anxiety	47	7.3	5.8	0	19
Depression	54	7.2	4.7	0	20
Reappraisal	54	3.1	0.9	1	5
Suppression	54	3.9	1.1	2	7
Perceived Stress	54	12	3.2	7	25
Peer Stress	51	10.2	5.5	5	25
School Stress	51	21.9	5.8	8	30
State Rumination	54	30.3	13.8	3	56
Trait rumination	54	0.06	0.01	0.03	0.07

Note. Means calculated from untransformed cortisol measured in nmol/L. Measures: Anxiety = GAD-7, Depression = PHQ-8, Reappraisal & Suppression = ERQ-CA, Perceived Stress = RSQ, Peer & School Stress = ASQ, State Rumination = Thoughts Questionnaire, Trait Rumination = RSQ.

Table 4.
Partial correlations between non-repeated, self-reported measures partialing out sex.

	Depression	Anxiety	Reappraisal	Suppression	Perceived Stress	Peer Stress	School Stress	State Rum
Depression								
Anxiety	**0.78****							
Reappraisal	0.14	-0.03						
Suppression	**-0.28$^{\Delta}$**	**-0.30***	-0.06					
Perceived Stress	**0.37****	**0.37***	0.21	-0.13				
Peer Stress	**0.47****	**0.46****	-0.004	-0.18	**0.35***			
School Stress	**0.57****	**0.57****	-0.07	0.04	**0.35***	**0.31***		
State Rumination	**0.48****	**0.48****	0.17	**-.45****	0.19	**0.30***	**0.38****	
Trait Rumination -	-0.001	-0.001	0.03	**-.29***	0.06	0.18	0.002	0.21

Note. Measures: Anxiety = GAD-7, Depression = PHQ-8, Reappraisal & Suppression = ERQ-CA, Perceived Stress = RSQ, Peer & School Stress = ASQ, State Rumination = Thoughts Questionnaire, Trait Rumination = RSQ. $^{\Delta}p<.1$ level*;* * $p< 0.05$ level; ** $p<$ 0.01 level (2-tailed).

Table 5.
Means and standard deviations for self-reported repeated measures

	Time 1	Time 2	Time 3	Time 4	Time 5	Time 6
Positive Affect	2.8	2.1	2.1	2.0	2.0	2.2
	(0.8)	(0.9)	(0.9)	(1.0)	(1.0)	(1.0)
Negative Affect	1.6	2.2	1.7	1.4	1.3	1.2
	(0.5)	(0.7)	(0.7)	(0.6)	(0.4)	(0.4)
Momentary		17.8	12.1	8.7	7.3	8.4
Rumination		(4.6)	(4.9)	(4.5)	(3.6)	(4.1)

Note. N = 52-54. standard deviations (in parentheses). Time 1= Baseline, before the GPST-A, Time 2-Time 6 = Following the GPST-A in ~10 min increments.

Table 6

Correlations between non-repeated self-reported measures and momentary rumination

	Momentary Rumination Score				
	Time 2	Time 3	Time 4	Time 5	Time 6
Sex	0.09	-0.04	**-0.23**$^{\Delta}$	-0.18	-0.09
Depression	0.19	-0.01	0.09	0.06	**0.25**$^{\Delta}$
Anxiety	0.19	-0.03	0.01	0.11	0.13
Reappraisal	0.003	0.07	0.15	0.13	0.11
Suppression	**-0.33***	**-.28***	**-0.26**$^{\Delta}$	-0.19	**-0.31***
Perceived Stress	0.21	0.18	0.13	0.19	0.20
Peer Stress	-0.002	0.12	0.09	0.18	**0.26**$^{\Delta}$
School Stress	**0.30***	0.19	0.19	0.13	0.22
State Rumination	**0.75****	**0.58****	**0.52****	**0.44****	**0.41****
Trait Rumination	**0.33***	0.20	0.16	0.15	0.10

Note. Measures: Anxiety = GAD-7, Depression = PHQ-8, Reappraisal & Suppression = ERQ-CA, Perceived Stress = RSQ, Peer & School Stress = ASQ, State Rumination = Thoughts Questionnaire, Trait Rumination = RSQ. $^{\Delta}p<.1$ level; * $p< 0.05$ level; ** $p< 0.01$ level (2-tailed).

Table 7.

Means, standard deviations and ranges of raw cortisol values

	Raw Cortisol values						Log Transformed Cortisol Values					
	Time 1	Time 2	Time 3	Time 4	Time 5	Time 6	Time 1	Time 2	Time 3	Time 4	Time 5	Time 6
Cortisol (N=54)	3.9	4.5	5.9	6.1	5.3	4.5	1.5	1.6	1.8	1.8	1.7	1.6
Range	0.8-20.4	1.1-19.6	1.3-22.0	.9-28.4	1.3-21.9	.97-14.2	.60-3.1	.73-3.0	.81-3.1	.65-3.4	.82-3.1	.68-2.7
SD	3.4	3.4	4.3	5.0	4.1	3.1	0.47	0.46	0.55	0.59	0.55	0.52
Males (N=14)	6.6	7.2	9.4	9.6	7.9	6.4	1.85	1.93	2.18	2.19	2.05	1.89
Range	1.6-20.4	1.5-19.6	1.2-22.0	.9-28.4	1.7-21.9	1.5-14.2	.97-3.1	.92-3.0	.81-3.1	.65-3.4	.98-3.1	.91-2.7
SD	5.5	5.5	6	5.1	5.1	3.6	0.58	0.59	0.62	0.64	0.55	0.49
Females (N=40)	3	3.6	4.6	4.9	4.4	3.8	1.3	1.5	1.6	1.6	1.5	1.4
Range	0.8-6.7	1.1-7.6	1.3-12.7	1.3-16.3	1.3-14.3	.97-13.6	.60-2.0	.73-2.2	.82-2.6	.84-2.9	.82-2.7	.68-2.7
SD	1.4	1.5	2.7	3.6	3.3	2.7	0.34	0.33	0.45	0.51	0.49	0.49

Note. Cortisol concentration measured in nmol/L. Time 1= Baseline before the GPST-A, Time 2-Time 6 = Following the GPST-A in ~10 min increments.

Table 8.

Linear Growth Model

	Model 1		Model 2	
Fixed effects				
Intercept, γ_{000}	1.44**	(0.07)	1.45**	(0.06)
Reactivity slope, γ_{100}	0.015**	(0.002)	0.015**	(0.002)
Recovery slope, γ_{200}	-0.011**	(0.001)	-0.010**	(0.003)
Sex, γ_{010}			-0.46**	(0.12)
Reactivity slope*Sex, γ_{110}			-0.003	(0.01)
Recovery slope*Sex, γ_{210}			0.01**	(0.002)
Random effects				
Variance intercept, σ^2_{r0}	0.18**	(0.43)	0.14**	(0.38)
Variance Reactivity slope, $\sigma^2 r_1$	0.0002**	(0.02)	0.0002**	(0.01)
Variance Recovery slope, $\sigma^2 r_2$	0.001**	(0.01)	0.00003**	(0.01)
Covariance,Intercept1/Intercept2, σ_{u00}	0.01	(0.10)	0.01	(0.10)
Residual variance, σ^2_e	0.02**	(0.12)	0.02**	(0.12)

Note. . Unstandardized estimates and standard errors (in parentheses). Models based on up to 6 occasions nested within 54 participants nested with 12 groups for a total of 324 observations. Dependent variable is log transformed cortisol concentration, nmol/L. Model 1 includes intercept, minutes from baseline through peak cortisol (***Reactivity)***, minutes from Peak to appointment end (***Recovery)*** at Level 1. Model 2 includes intercept, *Reactivity* and *Recovery* at Level 1 and sex as an independent variable at Level 2 as well as other specified variable. ***p<.01,* **p<.05,* $^{\Delta}$*p<.1.*

Table 9.
HLM estimates. Level 1 included two time terms to model variation across samples (time from baseline to peak and time after peak). The level 2 variables are person-level variables. Level 3 modeled nesting of participants within GPST-A groups and only included a random intercept.

	β	Std. Error
Intercept π_0		
Estimates for cortisol concentration at baseline, before GPST-A		
Intercept, γ_{000}	**1.44****	(0.06)
Sex, γ_{010}	**-0.46****	(0.12)
Anxiety	-0.01	(0.01)
Depression	**-0.03***	(0.01)
Reappraisal	0.02	(0.06)
Suppression	-0.04	(0.05)
Perceived Stress	-0.01	(0.02)
Peer Stress	-0.005	(0.01)
School Stress	-0.01	(0.01)
Trait Rumination	**8.96**$^{\Delta}$	(5.09)
State Rumination	-0.003	(0.004)
Reactivity π_1		
Estimates for slope from Baseline to Peak		
Intercept, γ_{100}	**0.02****	(0.002)
Sex, γ_{110}	-0.003	(0.005)
Anxiety	-0.00005	(0.0004)
Depression	-0.00001	(0.001)
Reappraisal	0.0001	(0.003)
Suppression	-0.003	(0.002)
Perceived Stress	-0.0004	(0.001)
Peer Stress	0.00001	(0.0004)
School Stress	-0.0004	(0.0004)
Trait Rumination	**-0.51***	(0.20)
State Rumination	**0.0004***	(0.03)
Recovery, π_2		
Estimates for slope from Peak to End		
Intercept , γ_{200}	**-0.01****	**(0.001)**
Sex, γ_{210}	**0.01****	(0.003)
Anxiety	0.0001	(0.0002)
Depression	0.0002	(0.0002)
Reappraisal	-0.002	(0.001)
Suppression	0.001	(0.001)
Perceived Stress	**0.0006**$^{\Delta}$	(0.0003)
Peer Stress	0.0002	(0.0002)
School Stress	0.0004	(0.0002)
Trait Rumination	0.03	(0.11)
State Rumination	-0.0001	(0.13)

Note. Unstandardized estimates and standard errors (in parentheses). Models based on up to 6 occasions nested within 54 participants nested with 12 groups for a total of 324 observations. Dependent variable is log transformed cortisol concentration, nmol/L. ***p<.01,* **p<.05,* $^{\Delta}$*p<.1,*.

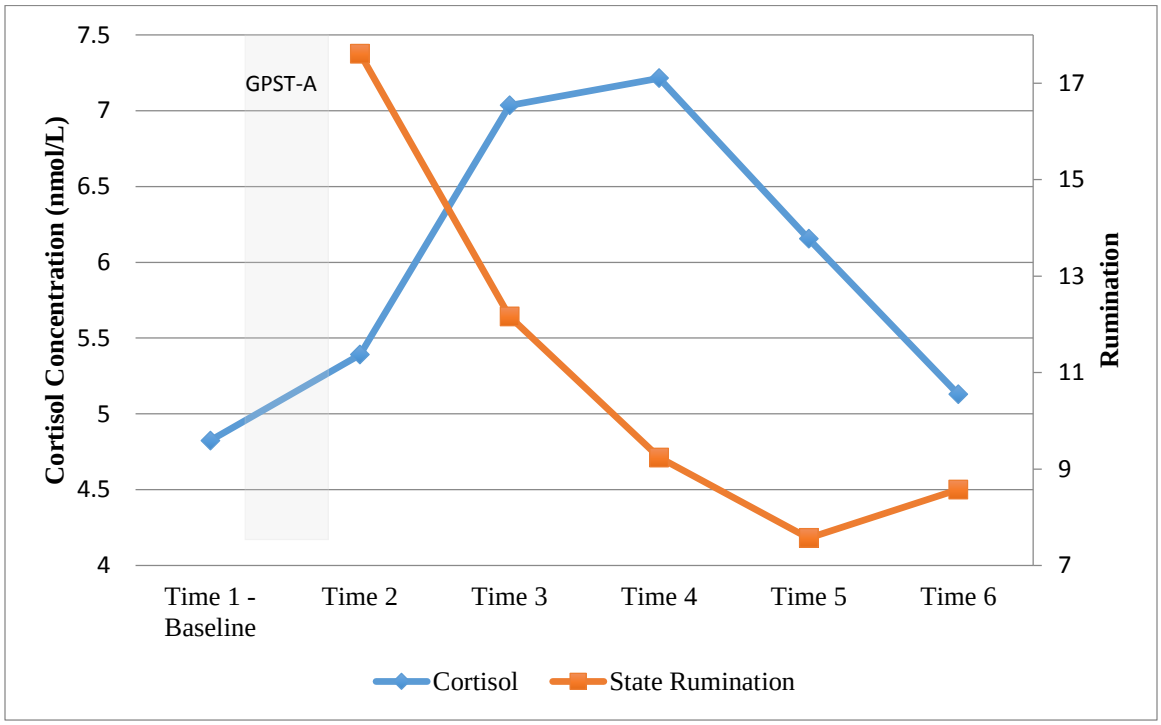

Figure 4. Group means of cortisol concentration and momentary rumination. Shaded area indicates the GPST-A. Cortisol values are untransformed for graphing purposes.

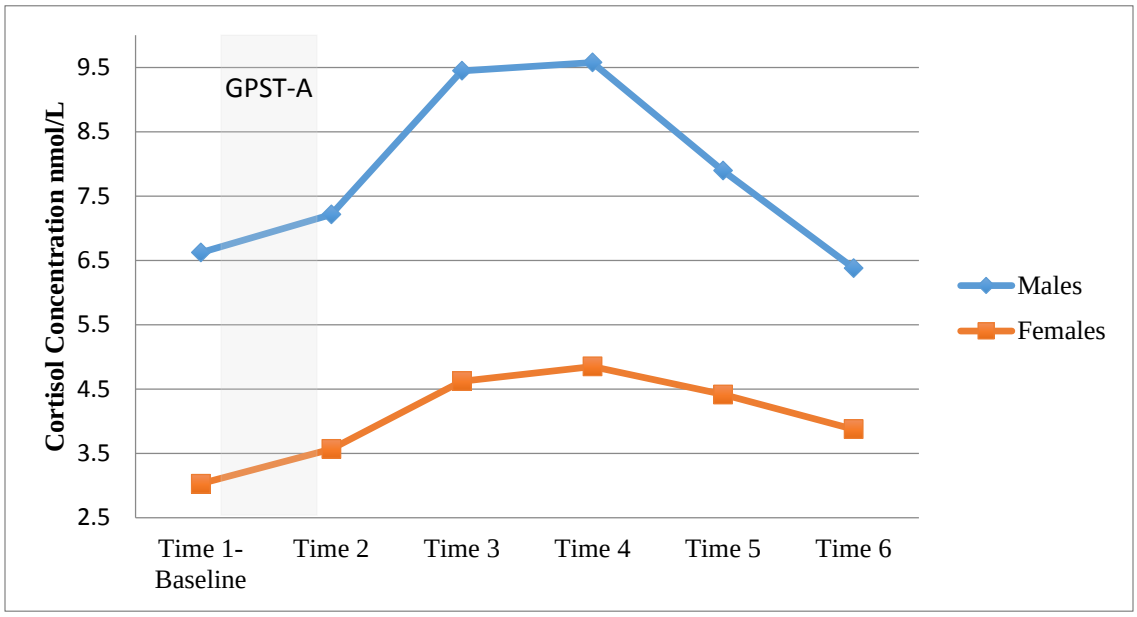

Figure 5. Mean cortisol concentration at each time point during appointment for males and females. Cortisol values are untransformed.

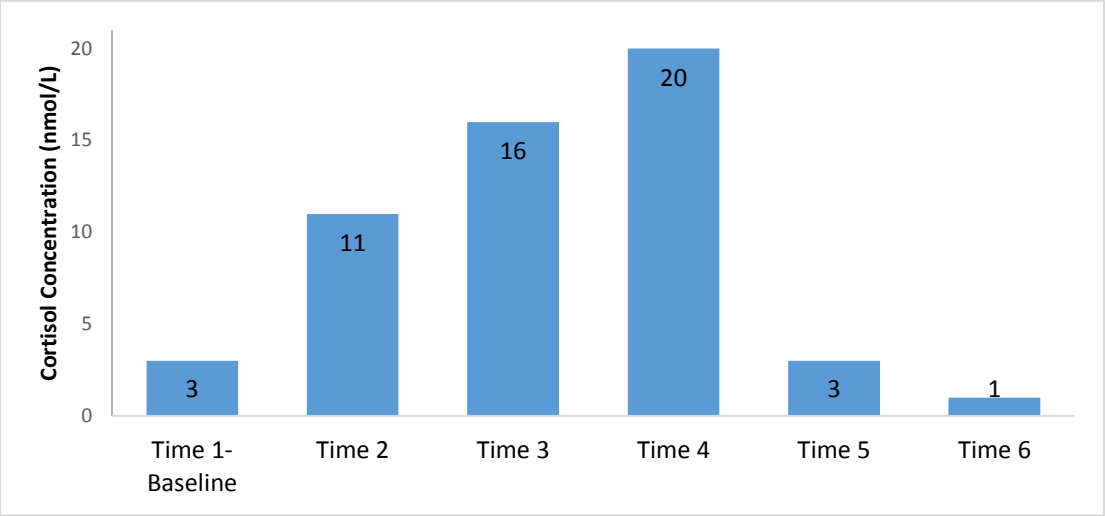

Figure 6. Peak cortisol timing. The timing of when participants' reached their peak cortisol value varied.

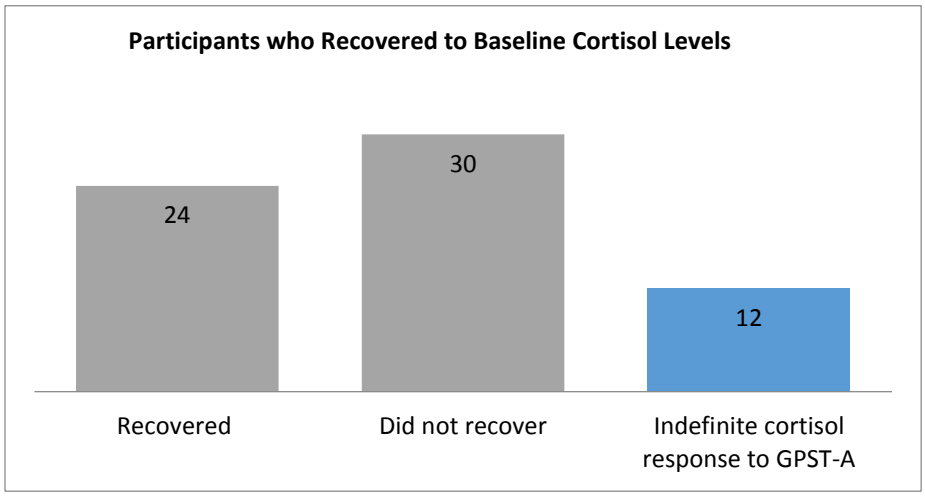

Figure 7. Cortisol recovery and indefinite responses. The *recovered* category includes participants whose ending cortisol value was the same or lower then their baseline cortisol value. A definite cortisol response is defined as a 10% change in cortisol concentration from baseline post-stressors (Gordis et al., 2006). Some participants may have experienced a definite cortisol response but not have recovered to their baseline levels. Twelve of the 54 participants did not show a 10% increase. Eight of those 12 showed an increase in cortisol post stressor but did not meet the 10% cut-point. The other 4 participants showed an atypical response to the stressor.)

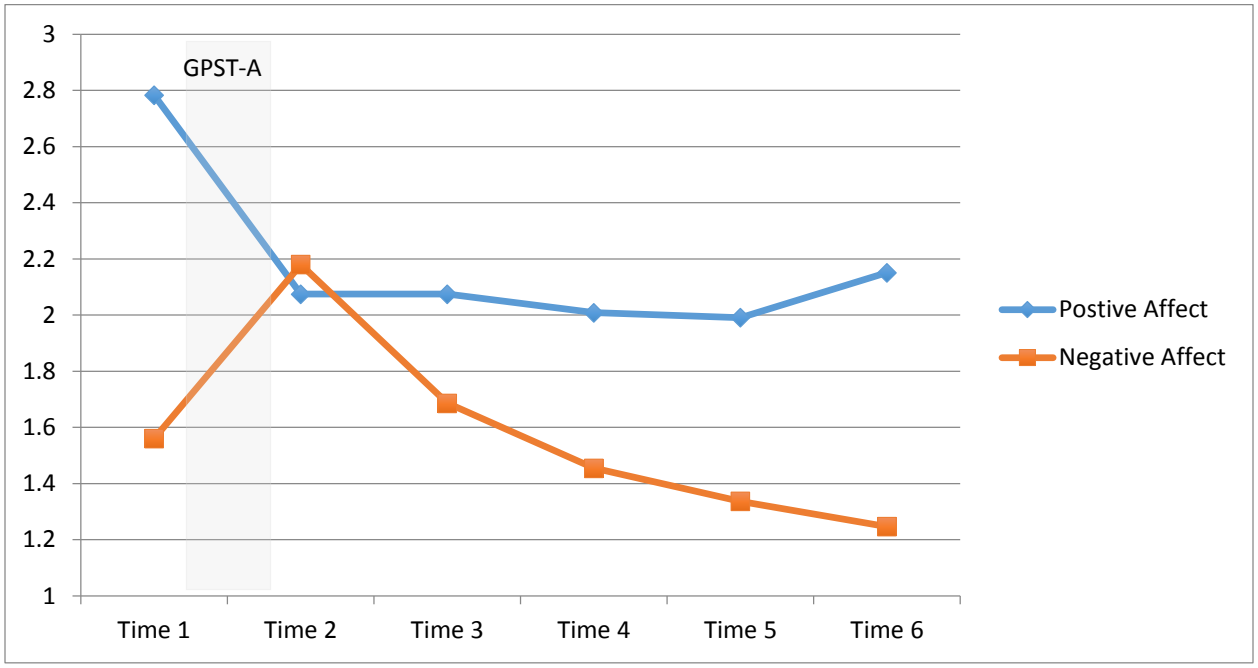

Figure 8. Self-reported affect before and after the GPST-A. Positive affect scores were an average of 2 items; negative affect scores were an average of 5 items.

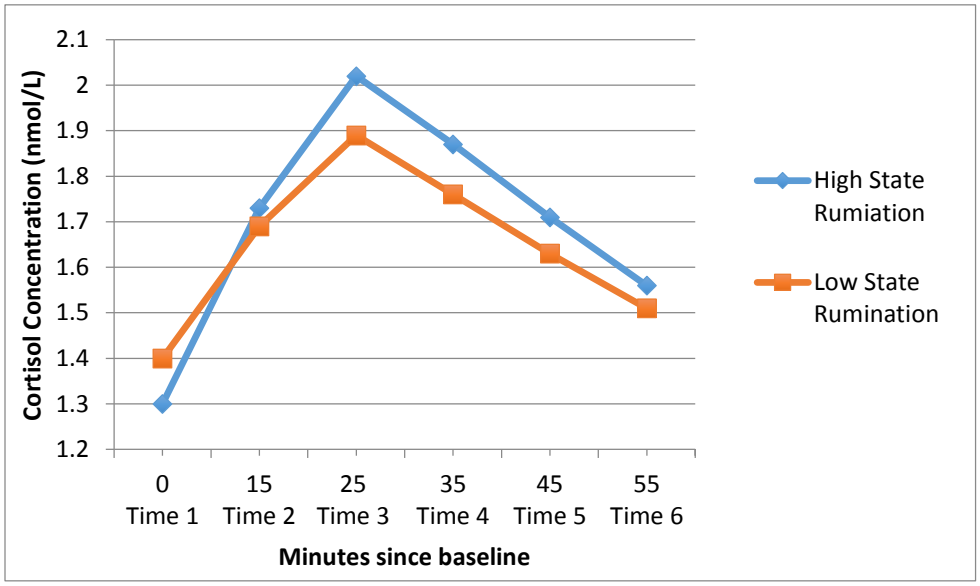

Figure 9. Level of state rumination by cortisol concentration over time. HLM Analyses revealed that higher State Rumination reported 45 minutes post-task predicted steeper cortisol reactivity slopes. Cortisol values were log transformed for analyses. Time and state rumination measure was a continuous variable in these analyses. Predicted levels of cortisol were plotted at each sampling time point for participants scoring 1 SD above and below the mean on the Thoughts Questionnaire (measuring state for illustrative purposes.)

Chapter 4: Discussion

The first aim of the present study was to examine the feasibility and effectiveness of a group social-evaluative stressor, the GPST-A, in a school setting. The second aim was to examine affective chronometry in a group of adolescents by exploring the relationships between their self-reported emotion regulation strategies and their HPA response to a laboratory stressor. Results indicate that the protocol was feasible in the school context and effective at eliciting a typical stress response. Results also indicate that rumination, but not suppression or reappraisal, was associated with the reactivity phase of the stress response.

Aim 1: Feasibility and Effectiveness of GPST-A

The first aim of this study was to test the feasibility and effectiveness the GPST-A with a group of adolescents in a school setting. This task is designed to elicit mild to moderate cortisol reactivity. Previous reports employing group social stress paradigms have been effective with adults (Von Dawans et al., 2011, Von Dawans, Fischbacher, Kirschbaum, Fehr, & Heinrichs, 2012) and with adolescents in a laboratory setting (Hostinar et al., 2014). Given the potential for group testing to be more efficient and ecologically valid, I examined whether the group laboratory stressor would be both feasible and effective with groups of adolescents who are also classmates. The ecological validity of the protocol used in this study was two fold – the task was performed in the school building and the group members were classmates. Even though the task was shorter compared to the original lab-based TSST, I hypothesized that the increased social sensitivity indicative of adolescence (Somerville, 2013) would induce sufficient social-evaluative threat to elicit a mild to moderate stress response in participants. To test this, I examined participants' change in affect as well as changes in their cortisol concentration during the session.

Change in affect. Subjective ratings of positive and negative affect changed in the expected directions, indicating that the participants experienced some discomfort and stress as a result of the GPST-A. Also, even though all participants were free to withdraw from the study at any time, none refused to perform the task or provide saliva samples, suggesting that this protocol is not too extreme and does not contribute to attrition. Together, these results alleviate concern that the group testing with classmates may have been too upsetting or stressful due to adolescents' enhanced sensitivity to social evaluation by peers (Somerville, 2013).

Cortisol response. The protocol was effective at significantly increasing cortisol production. The average cortisol peak was approximately 50% over baseline levels and 50 out of 54 participants, about 93%, exhibited a nonzero increase in cortisol (comparable to the rate of response in studies using single-subject TSST paradigms with adolescents, e.g. Gordis et al., 2006). The size of the average increase in cortisol is also within the range noted in single subject TSST studies conducted with adolescents (Gunnar, Talge, & Herrera, 2009a; Gunnar et al., 2009b). On average, participants showed a decline after they reached their peak cortisol concentration, indicating that the stressor was mild to moderate and not so extreme that participants were unable to recover.

This protocol proved an effective way to study individual differences in the psychological and physiological response to stress because it was mild to moderate. Mild to moderate stressors allow individual differences in stress reactivity to be expressed and avoid ceiling effects (Hostinar et al., 2014). It was also a developmentally appropriate and ecologically valid stressor, since introducing oneself and speaking in front of groups of adults and peers are typical scenarios in school settings and public speaking is commonly stressful.

Sex differences. Although both males and females elicited typical responses to the GPST-A, males' cortisol concentration was significantly higher than females' during each phase. Previous studies show mixed results with respect to gender differences in adolescent cortisol reactivity. Some studies show there are no differences between genders at age 15 (Gunnar et al., 2009b). Others show higher cortisol concentration at baseline and peak in adolescent and adult males (Bouma et al., 2009; Zijlmans et al., 2013; Zoccola, Quas, & Yim, 2010). Hostinar and colleagues (2014) found that males were marginally higher at baseline and showed no other gender differences. The analytic approach used in the present study differs from previous work and allowed me to examine different phases of the response and revealed gender effects at each aspect of the response. Males had higher baseline cortisol, final cortisol levels, and higher peak values of cortisol than females. Females had less steep recovery slopes, likely driven by a less steep peak cortisol value.

In summary, results from Aim 1 indicate that the GPST-A task was a feasible and effective social-evaluative stressor in a school-based setting. It proved feasible and more efficient compared to the single-subject version of the TSST.

Aim 2: Explore Affective Chronometry

The second aim of this study was to examine the affective chronometry in a group of adolescents by exploring the associations between HPA responses to stress at three different phases of the response – baseline, reactivity and recovery. I examined these three phases in relation to three common emotion regulation strategies – suppression, reappraisal and rumination.

Accounting for peak latency. In order to explore affective chronometry in participants by examining each phase of the stress response, I needed to account for differences in

participants' peaks. Consistent with prior research (Lopez-Duran, et al., 2009), participants in this study reached peak cortisol concentrations at different times. The majority of participants peaked between 20 and 30 minutes post-stressor, but 35% of participants did not peak in that time period.

A discontinuous multilevel model with landmark registration (Lopez-Duran et al., 2014), allowed me to examine how self-reported measures were associated with different phases (baseline, reactivity, and recovery) of the stress response, and also test my hypotheses that participants' use of common emotional regulation strategies – suppression, reappraisal and rumination – would be associated with differences in their cortisol response profiles.

Reappraisal. Analyses revealed that there were no significant associations between self-reported reappraisal and cortisol concentration at any of the three phases. Findings from this study are consistent with other studies that show no relationship between reappraisal and physiological reactivity (Egloff et al., 2006; De Veld et al., 2012; Gross, 1998), but it is surprising that reappraisal was not correlated with any other measure.

Suppression. While suppression was not predictive of the change in cortisol over time in any of the three phases – baseline, reactivity nor recovery - indicated by results from the multilevel model, suppression was associated with a change in cortisol concentration from peak to end. Suppression was negatively correlated with the change in magnitude of cortisol from peak to baseline as well as ending levels of cortisol, indicating that suppression may be inhibiting HPA recovery from the acute stressor. This finding is not supported by previous research. Suppression has previously been related to both heighted sympathetic nervous system (SNS) activity in studies using cardiovascular measures (Egloff et al., 2006; Gross & Thompson, 2007; Harris, 2001), and HPA increase in another study (Lam et al., 2009). These inconsistent results

indicate that more research is needed to determine how suppression is associated with HPA activity.

Notably, my findings for suppression and reappraisal conflict with those of Lam and colleagues (2009) in their study that utilized a similar protocol and measures. Lam and colleagues (2009) used the TSST and the ERQ with a sample of young adults (N=128; mean age= 20.3) and found that increased reports of both suppression and reappraisal were associated with increased cortisol reactivity. The conflicting results could be due to several methodological differences between the two studies. Lam and colleagues (2009) used a single-participant TSST whereas the present study administered the TSST in a group setting. They also utilized a different modeling technique. While they did use multilevel modeling like the present study, their level one variables were Time and Time^2. This approach captures the curve of the cortisol response but researchers are unable to compare reactivity or recovery phases nor compare participants' peak or ending levels of cortisol with this type of modeling. Lastly, and arguably most importantly, Lam and colleagues (2009) did not control for gender in their analyses despite substantial literature indicating that males and females differ in their cortisol responses to stress. Evidence shows that adult men respond to psychological stress from the TSST with greater increases in cortisol then adult women (Kudielka & Kirschbaum, 2005) and that sex differences in HPA axis function become apparent as adolescents transition through pubertal development (Gunnar et al., 2009b; Stroud, Papandonatos, Williamson, & Dahl, 2004). In the present study, when models were run with just suppression or reappraisal as predictors at level 2, but without sex as a predictor, suppression had a significant effect on the recovery phase. When sex was included as a predictor variable, this finding became non-significant. Despite the methodological differences and contrasting findings, the findings from the present study are noteworthy and

indicate that more studies measuring emotion regulation strategies and cortisol response are needed.

Even though the ERQ has been validated with adolescents (Gullone & Taffe, 2012), it may not be adequate for measuring emotion regulation strategies and future studies could consider using additional measures. For example, the ERQ uses only 9 items to capture suppression and reappraisal, other measures attempt to capture more dimensions of emotion regulation. For example, the Difficulties in Emotion Regulation Scale (DERS, Gratz, & Roemer, 2004) may be more comprehensive than the ERQ. The DERS attempts to capture difficulties within 6 dimensions of emotion regulation.

Another limitation may be related to using questionnaires that ask people to recollect how they handled situations in the past. Using questionnaires like the ERQ to measure emotion regulation in this broad manner may not provide a valid assessment of their actual emotion regulation strategies in the moment. Work by Robinson and Clore (2002) indicates that when people recall their emotions from previous periods, they rely on what they believe, rather than on how they felt or what they did. This indicates that future studies exploring emotion regulation should utilize measures that capture subject's state regulation strategies rather than measures asking them to recall the strategies they used in past situations. While this study assessed both emotions and state rumination in 10 minute increments, other emotion regulation strategies like reappraisal and suppression were not measured in the moment. In turn, future studies may benefit from creating stressor-specific questionnaires similar to the momentary rumination scale or the state rumination measure used in the present study to capture more dimensions of emotion regulation in the moment. These state emotion regulation measures could include the items from

the ERQ or other emotion regulation scales so that they could be compared with trait measures of the same constructs.

Rumination. Several trends emerged in the data with respect to rumination. Based on previous studies revealing that state and trait rumination have different associations with cortisol release (Zoccola et al., 2008; Zoccola & Dickerson, 2012), I used 3 measures of rumination– momentary rumination, state rumination and trait rumination - to examine the differential effects that state and trait rumination appear to have on the stress response after a social-evaluative stressor. My goal in measuring state rumination in two ways was to test a previous approach to measuring state rumination (using the Thoughts Questionnaire to measures state rumination 40 minutes post stressor; Zoccola et al., 2008) and to try a repeated measure of state rumination after the stress task to examine the affective chronometry.

My hypotheses that higher trait rumination would be associated with a steeper cortisol reactivity slope and that higher state rumination would be associated with a prolonged recovery were not supported by the data. Interestingly, state and trait rumination had opposite effects on the reactivity phase of the stress response: increased *state* rumination predicted steeper cortisol reactivity whereas higher *trait* rumination predicted *less* steep cortisol reactivity. In fact, both the state measures of rumination - state rumination (at Time 6, ~40 minutes after the GPST-A) and the first momentary rumination measure (at Time 2, immediately following the stressor) - were associated with steeper cortisol reactivity.

Results from the current study are in partial support of the Perseverative Cognition Hypothesis (Brosschot et al., 2006). The finding that both state rumination measures were associated with steeper cortisol reactivity supports the hypothesis's claim that repetitive, intrusive thoughts may amplify physiological responses to stressors. However, there were no

results from this study that support the part of the hypothesis that posits that thoughts might maintain physiological responses to stressors.

Findings from this study are also consistent with Zoccola and colleagues (2008) study showing higher trait rumination associated with decreased reactivity, but higher state rumination measured at the end of the session (measured with the same Thoughts Questionnaire to measuring state rumination) associated with steeper reactivity. Gianferante and colleagues (2014) also showed that state rumination (measured 10 minutes post stressor) was predictive of higher cortisol reactivity, but found no relationship between cortisol and trait rumination.

Though the precise mechanisms mediating the relationship between rumination and the HPA response were not the focus of the current study, I offer some possibilities of why certain relationships may have emerged that could be the focus of future research.

Why might higher trait rumination predict less steep cortisol reactivity slope? The findings that trait rumination is associated with a less steep cortisol reactivity slope is consistent with Zoccola et al.'s (2008) finding with a similar protocol. They suggest that trait and state rumination measures may be tapping into different dimensions of rumination. Zoccola and colleagues (2008) point out that trait rumination measures are often designed to measure depressive rumination and that depression is associated with blunted cortisol responses (Burke et al., 2005; Harkness et al., 2011), but they did not include depression measures in their study to test this and previous literature does not support this hypothesis. Results from both adult and adolescent studies (Burke et al., 2005; Harkness et al., 2011), as well as the present study indicate that those who report more depressive symptoms have a lower overall cortisol response including lower baseline, peak and ending values of cortisol, but not a less steep reactivity slope. In the present study, higher baseline cortisol is associated with a less steep reactivity slope (the

random effects of the intercept are negatively correlated with the random effects of the time since baseline (the reactivity slope); $r = -0.31$). It is unclear from Zoccola and colleagues (2008) study if this is also true for their sample. Results from the current study indicate that trait rumination has its own unique effect on the stress response outside of depressive symptoms.

Why might higher state rumination predict steeper cortisol reactivity? It is theorized that when stressed, some people ruminate and negatively dwell on the stressor itself (Simonson, Sánchez, Arger, & Mezulis, 2012). Participants who were unable to stop thinking about the stressor may have prolonged their state of arousal. One explanation offered as to why state rumination might be positively associated with cortisol reactivity is that state rumination is inhibiting other coping mechanisms that would otherwise allow an individual to adapt to a stressor and dampen their reactivity to an acute stressor (Gianferante et al., 2014). Another possibility is that the state rumination measure is capturing adaptive coping strategies like problem solving or cognitive restructuring as opposed to rumination. People report ruminating to understand or solve their problem (Hong, 2007) and it is unclear when reflecting on or mulling over a problem turns into rumination. Further investigation into the relationships between state rumination and other coping strategies could support or reject this hypothesis. Lastly, it is also possible that the way one appraises the stressor could affect post-stress rumination. The integrated specificity model (Weiner, 1992, see Figure 1) suggests that people's cognitive appraisal of the situation shapes their psychobiological response. For example, those who feel threatened may also ruminate more after a stressful experience. Feeling threatened has been shown to be related to increased cortisol reactivity (Denson et al., 2009; Scholtz et al., 2011). This study did not fully explore participants' appraisal of the GPST-A other than their momentary emotional state. In turn, future research should investigate appraisal mechanisms

that may mediate the relationship between state rumination and cortisol response. This could be investigated by seeing if those who are higher in state rumination show certain emotions at higher rates. Additional measures asking about how participants felt about the stressor could examine the role that appraisal of the stressor may play in participants' response and more thoroughly explore the integrated specificity model of stress.

In their review of the 15 studies that have examined the association between rumination and cortisol, Zoccola and Dickerson (2012) conclude that the way that researchers conceptualize and assess rumination influences the statistical significance and direction of associations between rumination and cortisol that they find. However, it should be noted that it may also be due to differences in how researchers model the cortisol response. None of the studies that Zoccola and Dickerson (2012) reviewed took into consideration participants' peak latency within their analytic strategies nor did they attempt to examine the reactivity phase separate from the recovery phase. The most common modeling practices – using repeated measures ANCOVA or multilevel modeling with linear and quadratic time variables – do not allow for conclusions to be made about individual differences in each phase of the cortisol response or affective chronometry. Additionally, there is a need for greater consistency and specificity when measuring any phase of the cortisol response.

Previous work has found thought suppression and rumination to be positively correlated (Nolen-Hoeksema & Morrow, 1993), with evidence suggesting thought suppression fuels rumination (Wenzlaff & Luxton, 2003). Other work shows no correlation between expressive suppression measured with the ERQ and trait rumination (Gortner, Rude & Pennebaker, 2006; Ioannidis & Siegling, 2015; Moore, Zoellner & Mollenholt, 2008). Findings from the present study conflict with past work and suggest the direction of the association may vary, as evidenced

by a negative correlation between suppression and both state and trait rumination. This may be related to the fact that the present study measured expressive suppression with the ERQ and not thought suppression and that this negative correlation is related to that difference. Additionally, in the studies that utilized the ERQ measure, different trait rumination measures were used (Rumination–*Reflection Questionnaire* and the *Ruminative Response Scales*).

A critical challenge that may be at the heart of the inconsistent findings with regard to rumination relates to Zoccola and Dickerson's (2002) point about measuring rumination. It is unclear how to determine what it is in fact rumination and when rumination might be another coping strategy. This determination may not be currently possible but may be achievable when rumination and problem solving are measured repeatedly over time. In this study, I did not explore problem solving strategies that participants might have used after the GPST-A so it is difficult to determine what type of problem solving may have been occurring after the stressor.

It is worth noting that in this study that suppression was associated with less of a change in cortisol from peak to end (not the slope) and two measures of perceived stress had positive, trend level associations with the recovery slope of the stress response in the multilevel model. The fact that these measures were associated with less recovery from peak levels, may support the allostatic load model. The allostatic load model suggests that greater cumulative stress burden can influence stress-responsive physiology, which can lead to a variety of long-term health issues (McEwen, 1998; Seeman, McEwen, Rowe, & Singer, 2001). Participants in this study who are reporting higher levels of stress (general perceived stress and stress associated with school) are not recovering as efficiently from the stressor compared to participants who report less stress. These results are also important to note because these participants are mid-adolescence and adolescence may be another sensitive period (Nelson et al., 2005; Gogtay et al.,

2004). Exposure to heightened stress during developmentally sensitive periods may permanently alter physiological processes and ultimately long-term health functioning (Boyce & Ellis, 2005). Previous research shows that dysregulated patterns of stress responsivity have been observed in early and middle adolescents who demonstrate depression (Harkness et al., 2011; Klimes-Dougan et al., 2001). These findings indicate that further research into the possible stress system dysregulation that may be occurring for adolescents who report high levels of day-to-day stress and using suppression as an emotion regulation strategy is warranted.

Limitations

This study has several limitations worth noting. The small sample size limits the ability to generalize the findings to all adolescents. As such, the findings from this study are representative of adolescents living in suburban United States. However, the present study is the first of its kind to use the GPST-A in a school and utilize discontinuous multilevel models to examine affective chronometry, so the goal of this study is to be a launching point for more studies that may be more generalizable. Similarly, the findings of gender differences in cortisol secretion and reactivity indicate separate models for males and females are necessary yet due to the imbalance between genders (females=40, males=14) and small sample size, gender stratified models were not run. In the future, studies should collect a larger, more gender-balanced sample in order to appropriately examine each phase of the stress response in males and females.

One possible study confound was that the administration of the momentary rumination measure could inflate rumination scores by asking participants if they are thinking about their speech performance. Simply asking them if they are thinking about their experience may lead to them thinking about it more. Additionally, during the period post speech, participants were told that the judges were still deliberating over their evaluation of the speeches, which may have kept

participants wondering and inflated rumination. The confounding effect of asking participants if they are ruminating and reminding them during their recovery that judges were still analyzing their speeches, should be considered when interpreting the findings from the present study. However, at this time there are few state rumination measures and, therefore, none more optimal for exploring this phenomena. Additionally, because both state rumination and the momentary rumination have not been measured in many studies, there are no established cut points for scores that would be considered higher then expected and indicate possible inflation of rumination. Future research could examine this possible confound more closely by seeing if scores on the state rumination measure changed based on if the momentary rumination measure was used or not. If the momentary rumination measure inflates participants' rumination post stressor, then we would expect to see higher scores of state rumination when it was used then when it was not used.

There is also a concern in this study with the validity of the trait rumination measure. The trait rumination measure was only 3 items and had low reliability. It is concerning that the trait rumination scores in this study does not correlate with depression and anxiety scores, as would be expected. However, in previous research the rumination subscale of the RSQ has been part of a composite variable called 'involuntary engagement'. This composite variable is composed of 15 items and 5 subscales – emotional arousal, impulsivity, intrusive thoughts, physical arousal and rumination. This composite variable is correlated positively with depression in the current study (r=0.28, p<.05). Future research that seeks to explore trait rumination more thoroughly should consider using another measure. There are other trait rumination measures that are longer, may have better reliability and better capture trait rumination such as the 22-item rumination scale within the Ruminative Response Scale (RRS,

Nolen-Hokesma & Morrow, 1991) which assesses the tendency to ruminate or repetitively think about events after they occur.

It should be noted that the social context of this GPST-A remains unexplored in this study. Even though the third level of the multilevel model controls for differences related to group membership, there are many other elements of this stress task that may have influenced participants' responses. First, the while judges were trained to be stern and provide no response to participants and they were different ages and races (Caucasian 25 years old and African American 56 years old), the two judges were both female in this study. This may have influenced the participants' reaction to the task. Ideally, judges should include one male and one female to control for gender biases participants may have (Kirschbaum, Pirke, & Hellhammer, 1993).

Second, participants' response to the stressor may be influenced by their relationship with the other participants in the group. Previous literature suggests that both social support and negative relationships influence participants' responses to the TSST (Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Knack, Jensen-Cambell, & Baum 2011). Because participants in this study are classmates with one another, their previous social interactions or social status within the school may contribute to the social evaluative nature of the stressor and influence their HPA response.

Lastly, there was a small amount of missing data in this study. Future studies should ensure that data collection is more complete.

Strengths

The present study has two major strengths: its ecological validity and its statistical modeling. Successfully conducting the GPST-A in a school context demonstrates that public

speaking tasks typically performed in laboratory settings are both portable and more efficient in the group setting than in the single subject version. This group protocol has good ecological validity and may increase the ability for studies utilizing neuroendocrine measures to recruit larger and more diverse samples by performing them in a school rather than bringing participants to a lab.

The analytic approach in the current study highlights different phases of the response and takes into consideration peak latency. While a recent study validated this group social-evaluative stressor with adolescents (Hostinar et al., 2014), the present study is unique in that it explores predictors of the cortisol response, examining the reactivity and recovery of the response profile separately. Even though findings from this study merely add to the quagmire of inconsistent findings about the associations between emotion regulation strategies and HPA responses to stress, it offers a unique modeling approach that future research of this kind could use. The steps for using a discontinuous multilevel model with landmark registration to account for peak latency and examine the reactivity and recovery phase of the stress response separately, are carefully outline in the present study so that future studies might replicate this analytic approach to examine affective chronometry.

Future Directions

An unexplored aspect of this group protocol is how the relationships between the group members may have affected their reaction to, and ability to cope after, the stressor. For some adolescents, having classmates and/or friends in the group may have made the experience less stressful, and for others it may have made it more so. Recent literature suggests (Hostinar & Gunnar, 2013) that social connections can affect people's physiological response to stress. Given their increased social sensitivity (Somerville, 2009), it is logical to hypothesize that

adolescents' physiological response and coping strategies may be effected by positive or negative relationships in their GPST-A group. Future experiments utilizing the GPST-A should explore the relationship between group members, especially when settings such as this experiment take place when students are all in the same school and likely know one another.

Additionally, future prevention science research may benefit from utilizing the GPST-A as a pre- and post- measure during intervention studies. For example, many school-based social emotional learning interventions teach adaptive emotion regulation strategies and stress management skills with the hope that students will be able to utilize them during stressors they encounter on a day-to-day basis. Researchers would expect students to use regulatory skills they acquired from the intervention during the speaking task in the GPST-A. The GPST-A measure could help researchers determine if students are utilizing the skills they learned through the intervention and if utilizing those skills affects their physiological response to stressors.

Lastly, future experiments examining affective chronometry and utilizing the GPST-A should also employ measures from multiple physiological systems. When exploring individual differences in affective behavior the same pattern of individual differences may not be found across response systems and it could be the relationship between systems that is the key to understanding individual differences. For example, Gordis and colleagues (2009) found asymmetry between cortisol and salivary alpha amylase was related to aggressive behavior in adolescents but not to cortisol or salivary alpha amylase trajectory alone. Additionally, the GPST-A is a social-evaluative stressor. If the GPST-A is performed in a school and with peers present, it has the potential to activate pathways in addition to the HPA. Specifically, the SAM, indexed by salivary alpha amylase, has been shown to be more sensitive to tasks involving peers then to the traditional TSST, which does not have peers present (Stroud et al., 2009).

Conclusions

The present study revealed that the GPST-A was successful at eliciting significant cortisol reactivity and was feasible and effective in the school context. Results indicated that state and trait rumination influence adolescents' physiological response to a social-evaluative stressor differently. Overall, these findings suggest that rumination may have significant impacts on physiological functioning and, thus, could affect future health. These findings for rumination are particularly noteworthy in light of Aldao and colleagues' (2010) findings that the presence of maladaptive emotion regulation strategies is more deleterious in the absence of adaptive emotion regulation strategies and that rumination is the strongest predictor of overall psychopathology as compared to the other maladaptive strategies. Interventions seeking to reduce the onset of psychopathology and promote mental health often target adolescents' emotion regulation strategies. Advances in intervention may develop as a result of a deeper understanding of the relationship between emotion regulation strategies that adolescents use and their physiological responses to acute stressors. Modeling affective chronometry with discontinuous multilevel modeling may lead to a more fine-grained understanding of how biological and cognitive factors work in concert with developmental factors to understand normative development and health as well as the development of psychopathology.

References

Adam, E. K., Sutton, J. M., Doane, L. D., & Mineka, S. (2008). Incorporating hypothalamic-pituitary-adrenal axis measures into preventive interventions for adolescent depression: Are we there yet? *Development and Psychopathology*, *20*(3), 975–1001. doi:10.1017/S0954579408000461

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategiesacross psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 217–237. doi:10.1016/j.cpr.2009.11.00

Alloy, L. B., & Abramson, L. Y. (2009). The adolescent surge in depression and emergence of gender differences: A biocognitive vulnerability-stress model in developmental context. In D. Romer, E. F. Walker, (Eds.), *Adolescent psychopathology and the developing brain: Integrating brain and prevention science* (pp. 441-462). New York, NY US: Oxford University Press

Boyce, W.T., & Ellis, B.J., (2005). Biological sensitivity to context: I. An evolutionary developmental theory of the origins and functions of stress reactivity. *Developmental Psychopatholgy.* 17, 271–301.

Brackett, M.A., & Salovey,P. (2004). Measuring emotional intelligence as a mental Ability with the Mayer–Salovey–Caruso Emotional Intelligence Test. In G. Geher (Ed.), Measurement of emotional intelligence (pp. 179−194). Hauppauge, NY: Nova Science Publishers. Brady,

Brosschot, J. F., & Thayer, J. F. (1998). Anger inhibition, cardiovascular recovery, and vagal function: A model of the link between hostility and cardiovascular disease. *Annals of Behavioral Medicine*, *20*(4), 326–332. doi:10.1007/BF02886382

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition

hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*(2), 113–124. doi:10.1016/j.jpsychores.2005.06.074

Brotheridge, C. M., & Grandey, A. A. (2002). Emotional labor and burnout: Comparing two perspectives of "People work".*Journal of Vocational Behavior,60*(1), 17-39.

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: A meta-analysis. *Psychoneuroendocrinology*, 30, 846–856. doi:10.1016/j.psyneuen.2005.02.010

Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine, 59*, 419–426.

Bydlowski, S., Corcos, M., Jeammet, P., Paterniti, S., Berthoz, S., Laurier, C., et al. (2005). Emotional-processing deficits in eating disorders. *International Journal of Eating Disorders*, 37, 321−329.

Byrne, D. G., Davenport, S. C., & Mazanov, J. (2007). Profiles of adolescent stress: The development of the adolescent stress questionnaire (ASQ). *Journal of Adolescence*, 30(3), 393-416.

Chrousos, G.P. & Gold, P.W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *The Journal of the American Medical Association, 267*, 1244-1252.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E.

(2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*(1), 87–127. http://doi.org/10.1037//0033-2909.127.1.87

Dahl, R. E., & Gunnar, M. R. (2009). Heightened stress responsiveness and emotional reactivity during pubertal maturation: implications for psychopathology. *Development and Psychopathology, 21*(1), 1–6. doi:10.1017/S0954579409000017

Davidson, R. J. (1998). Affective Style and Affective Disorders: Perspectives from Affective Neuroscience. *Cognition and Emotion* 12(3): 307–330.

Davidson, R. J. (2003). Affective neuroscience and psychophysiology: Toward a synthesis. *Psychophysiology*, *40*(5), 655–65.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience and Biobehavioral Reviews*, *35*(7), 1562–92. doi:10.1016/j.neubiorev.2010.11.007

Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin*, *135*(6), 823–53. doi:10.1037/a0016909

De Veld, D. M. J., Riksen-Walraven, J. M., & de Weerth, C. (2012). The relation between emotion regulation strategies and physiological stress responses in middle childhood. *Psychoneuroendocrinology*, *37*(8), 1309–19. doi:10.1016/j.psyneuen.2012.01.004

Edwards, S. L., Rapee, R. M., & Franklin, J. (2003). Postevent Rumination and Recall

Bias for a Social Performance Event in High and Low Socially Anxious Individuals. *Cognitive Therapy and Research, 27*(6), 603–617.

Egloff, B., Schmukle, S.C., Burns, L.R., Schwerdtfeger, A., (2006). Spontaneous emotion regulation during evaluated speaking tasks: associations with negative affect, anxiety expression, memory, and physiological responding. *Emotion* 6, 356—366.

Eisenberg, N., & Spinrad, T. L. (2004). Emotion-related regulation: sharpening the definition. *Child Development, 75*(2), 334–9. doi:10.1111/j.1467-8624.2004.00674.x

Frijda, N. H. (1988). The laws of emotion. *The American Psychologist*, *43*(5), 349–58. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3389582

Gans, J. (2009) *America's adolescents: How healthy are they?* Chicago: American Medical Association.

Garber, J., Braafladt, N., & Weiss, B. (1995). Affect regulation in depressed and Nondepressed children and young adolescents. *Development & Psychopathology. Special Issue: Emotions in developmental psychopathology, 7*, 93–115.

Gianferante, D., Thoma, M. V, Hanlin, L., Chen, X., Breines, J. G., Zoccola, P. M., & Rohleder, N. (2014). Post-stress rumination predicts HPA axis responses to repeated acute stress. *Psychoneuroendocrinology*, *49*, 244–52. doi:10.1016/j.psyneuen.2014.07.021

Gillespie, C. F., & Nemeroff, C. B. (2005). Hypercortisolemia and depression. *Psychosomatic Medicine*, 67(Suppl 1), S26–S28. doi:10.1097/01.psy.0000163456.22154.d2.

Glynn, L.M., Christenfeld, N., & Gerin,W., (2002). The role of rumination in recovery

from reactivity: Cardiovascular consequences of emotional states. *Psychosomatic Medicine* 64(5):714–726.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, a C., … Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(21), 8174–9. doi:10.1073/pnas.0402680101

Gortner, E. M., Rude, S. S., & Pennebaker, J. W. (2006). Benefits of expressive writing in lowering rumination and depressive symptoms. *Behavior Therapy*, *37*(3), 292-303.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment, 26*(1), 41-54. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1023/B:JOBA.0000007455.08539.94

Graziano, P. A., Reavis, R. D., Keane, S. P., & Calkins, S. D. (2007). The role of emotion regulation in children's early academic success. *Journal of School Psychology, 45*(1), 3-19. doi:10.1016/j.jsp.2006.09.002

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74, 224-237.

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.

Gross, J.J., & Levenson, R.W., (1993). Emotional suppression: physiology, self-report,

and expressive behavior. *Journal of Personality and Social Psychology*, 64, 970—986.

Gross, J., & Thompson, R. A. (2007). Conceptual foundations for the field. In J. Gross (Ed.), Handbook of emotion regulation (pp. 3-24). New York: Guilford.

Gullone, E., & Taffe, J. (2012). The Emotion Regulation Questionnaire for Children and Adolescents (ERQ–CA): A psychometric evaluation. *Psychological Assessment*, *24*(2), 409–417. http://doi.org/10.1037/a0025777

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–73. doi:10.1146/annurev.psych.58.110405.085605

Gunnar, M.R., Talge, N.M., & Herrera, A. (2009a). Stressor paradigms in developmental studies: what does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology* 34, 953—967, http://dx.doi.org/10. 1016/j.psyneuen.2009.02.010.

Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009b). Developmental changes in hypothalamus-pituitary-adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, *21*(1), 69–85. doi:10.1017/S0954579409000054

Harkness, K. L., Stewart, J. G., & Wynne-Edwards, K. E. (2011). Cortisol reactivity to social stress in adolescents: Role of depression severity and child maltreatment. *Psychoneuroendocrinology*, *36*(2), 173–181. doi:10.1016/j.psyneuen.2010.07.006

Harris, C.R. (2001) Cardiovascular responses of embarrassment and effects of emotional suppression in a social setting. *Journal of Personality and Social Psychology*. 81, 886—897.

Hellhammer, D. H., Wüst, S., & Kudielka, B. M. (2009). Salivary cortisol as a biomarker

in stress research. *Psychoneuroendocrinology*, *34*(2), 163–171. doi:http://dx.doi.org/10.1016/j.psyneuen.2008.10.026

Hong, R. Y. (2007). Worry and rumination: Differential associations with anxious and depressive symptoms and coping behaviors. *Behavior Research and Therapy*, 45, 277−290

Hostinar, C. E., & Gunnar, M. R. (2013). Future directions in the study of social relationships as regulators of the HPA axis across development. *Journal of Clinical Child and Adolescent Psychology : The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53*, *42*(4), 564–75. doi:10.1080/15374416.2013.804387

Hostinar, C. E., McQuillan, M. T., Mirous, H. J., Grant, K. E., & Adam, E. K. (2014). Cortisol Responses to a Group Public Speaking Task for Adolescents: Variations by Age, Gender, and Race. *Psychoneuroendocrinology*, *50*, 155–166. doi:10.1016/j.psyneuen.2014.08.015

Ioannidis, C. A., & Siegling, A. B. (2015). Criterion and incremental validity of the emotion regulation questionnaire. *Frontiers in Psychology*, *6*, 247. http://doi.org/10.3389/fpsyg.2015.00247

Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology*, *37*(4), 515-522.

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

Juster, R.-P., Bizik, G., Picard, M., Arsenault-Lapierre, G., Sindi, S., Trepanier, L., … Lupien, S. J. (2011). A transdisciplinary perspective of chronic stress in relation to psychopathology throughout life span development. *Development and Psychopathology*, *23*(Special Issue 03), 725–776. doi:10.1017/S0954579411000289

Kajante, E., & Phillips, D.I. (2006). The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology* 31:151–78.

Kemeny, M. E. (2003). The psychobiology of stress. Current Directions in Psychological Science, 12, 124–129.

Kirschbaum, C. & Hellhammer, D.H. (1989). Salivary cortisol in psychobiological research: an overview. *Neuropsychobiology* 22:150–69.

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

Klimes-Dougan, B., Hastings, P. D., Granger, D. A., Usher, B. A., & Zahn-Waxler, C. (2001). Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. *Development and Psychopathology*, *13*(3), 695–719. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11523855

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology*, *69*, 113–132. doi:10.1016/j.biopsycho.2004.11.009

Kudielka, B.M., Hellhammer, D.H., & Wüst, S. (2009) Why do we respond so

differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology* 34, 2—18, http://dx.doi.org/10.1016/j.psyneuen.2008.10.004.

Lam, S., Dickerson, S. S., Zoccola, P. M., & Zaldivar, F. (2009). Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology*, *34*(9), 1355–62. doi:10.1016/j.psyneuen.2009.04.006

Lazarus, R.S. (1966) Psychological Stress and the Coping Process. New York: McGraw Hill.

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

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: a meta-analysis. *Psychoneuroendocrinology*, *34*(9), 1272–83. doi:10.1016/j.psyneuen.2009.03.016

Lopez-Duran, N. L., Mayer, S. E., & Abelson, J. L. (2014). Modeling neuroendocrine stress reactivity in salivary cortisol: Adjusting for peak latency variability. *Stress*, *3890*, 1–11. doi:10.3109/10253890.2014.915517

Löwe, B., Decker, O., Müller, S., Brähler, E., Schellberg, D., Herzog, W., & Herzberg, P. Y.. (2008). Validation and Standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the General Population. *Medical Care*, *46*(3), 266–274.

Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist*, *56*(3), 227–238. doi:10.1037/0003-066X.56.3.227

Masten, A. S., & Coatsworth, J. D. (1998). The development of competence in favorable and unfavorable environments. *American Psychologist, 53*, 205–220Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist*, *56*(3), 227–238. doi:10.1037/0003-066X.56.3.227

Mauss, I.B., Wilhelm, F.H., Gross, J.J., (2004). Is there less to social anxiety than meets the eye? Emotion experience, expression, and bodily responding. *Cognition and Emotion* .18, 631—662.

McClure, E. B., & Pine, D. S. (2007). Social stress, affect and neural function in adolescence. In D. Romer & E. F. Walker (Eds.), *Adolescent psychopathology and the developing brain: Integrating brain and prevention science* (pp. 441–462). New York: Oxford University Press.

McEwen, B.S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine.* 338:171-179.

McEwen, B.S. (2000). The neurobiology of stress: From serendipity to clinical relevance. *Brain Research, 886,* 172-189.

Mennin, D. S., Holoway, R. M., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (2007). Delineating components of emotion and its dysregulation in anxiety and mood psychopathology. *Behavior Therapy,* 38, 284−302

Moore, S. A., Zoellner, L. A., & Mollenholt, N. (2008). Are Expressive Suppression and Cognitive Reappraisal Associated with Stress-Related Symptoms? *Behaviour Research and Therapy, 46*(9), 993–1000. http://doi.org/10.1016/j.brat.2008.05.001

Moulds, M.L., Kandris, E., Starr, S., & Wong, A.C.M. (2007). The relationship between rumination, avoidance and depression in a non-clinical sample. *Behaviour Research and Therapy, 45,* 251-261.

Nater, U. M., Rohleder, N., Gaab, J., Berger, S., Jud, A., Kirschbaum, C., & Ehlert, U.

(2005). Human salivary alpha-amylase reactivity in a psychosocial stress paradigm. *International Journal of Psychophysiology*, *55*(3), 333–342. doi:http://dx.doi.org/10.1016/j.ijpsycho.2004.09.009

Nicolson, N., Storms, C., Ponds, R., & Sulon, J. (1997). Salivary cortisol levels and stress reactivity in human aging. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, *52*(2), M68–M75. doi:10.1093/gerona/52A.2.M68

Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social re orientation of adolescence: A neuroscience perspective on the process and its r relation to psychopathology. *Psychological Medicine*, *35*(2), 163–74.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. Journal of Abnormal Psychology, 100, 569−582.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, 109, 504−511.

Nolen-Hoeksema, S., & Morrow, J. (1993). Effects of rumination and distraction on naturally occurring depressed mood. *Cognition and Emotion*, *7,* 561-570

Nolen-hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking Rumination. *Perspectives on Psychological Science*, *3*(5), 400–424.

Papageorgiou, C., & Wells, A. (2003). An empirical test of a clinical metacognitive model of rumination and depression. *Cognitive Therapy and Research*, 27, 261−273. Pardo,

Paus, T., Keshavan, M., & Giedd, J. N. (2008). Why do many psychiatric disorders emerge during adolescence? *Nature Reviews: Neuroscience*, *9*(December), 947–958.

Richardson, L. P., Mccauley, E., Grossman, D. C., Mccarty, C. A, Richards, J., Russo, J.

E., & Katon, W. (2010). NIH Public Access. *Pediatrics*, *126*(6), 1117–1123. doi:10.1542/peds.2010-0852.

Robinson, M.S., & Alloy, L.B. (2003). Negative cognitive styles and stress- reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy Res* 27:275–292

Robinson, M. D., & Clore, G. L. (2002). Belief and feeling: Evidence for an accessibility model of emotional self-report. *Psychological Bulletin, 128*(6), 934-960. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1037/0033-2909.128.6.934

Romeo, R.D., Bellani, R., Karatsoreos, I.N., Chhua, N., Vernov, M., Conrad, C.D., & McEwen, B.S. (2006). Stress history and pubertal development interact to shape hypothalamic pituitary adrenal axis plasticity. Endocrinology147:*1664–1674*.

Rottenberg, J., Gross, J. J., & Gotlib, I. H. (2005). Emotion context insensitivity in major depressive disorder. *Journal of Abnormal Psychology*, 114, 627−639.

Rudolph, K. D., Troop-Gordon, W., & Granger, D. A. (2010). Peer victimization and aggression: moderation by individual differences in salivary cortisol and alpha-amylase. *Journal of Abnormal Child Psychology*, *38*(6), 843–56. doi:10.1007/s10802-010-9412-3

Sapolsky, R.M., 1998. Why Zebras Don't Get Ulcers: An Updated Guide to Stress, Stress-related Diseases, and Coping. Freeman, New York.

Sapolsky, R. M. (2003). Stress and plasticity in the limbic system. *Neurochemical research*, *28*(11), 1735-1742.

Sapolsky, R. M. (2004). Social Status and Health in Humans and Other Animals. *Annual Review of Anthropology*, *33*, 393–418. doi:10.2307/25064859

Sapolsky, R.M., Romero, L.M., & Munck, A.U. (2000). How do glucocorticoids

influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews, 21,* 55-89.

Scherf, K. S., Smyth, J. M., & Delgado, M. R. (2013). The amygdala: An agent of change in adolescent neural networks. *Hormones and Behavior*, *64*(2), 298–313. doi:10.1016/j.yhbeh.2013.05.011

Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. Proceedings of the National Academy of Sciences, 98, 4770–4775.

Sher, K. J., & Grekin, E. R. (2007). Alcohol and affect regulation. In J. J. Gross (Ed.), Handbook of emotion regulation (pp. 560−580). New York, NY: Guilford Press.

Simonson, J., Sánchez, O., Arger, C., & Mezulis, A.H., (2012) Integrating affective and cognitive vulnerabilities to depression: examining individual differences in cognitive responses to induced stress. *Cognitive Therapy Research.* 36 (5), 474—482, http://dx.doi.org/10.1007/s10608-011-9383-x. Strauss,

Somerville, L. H. (2013). Special issue on the teenage brain: Sensitivity to social evaluation. *Current Directions in Psychological Science*, *22*(2), 121–127. doi:10.1177/0963721413476512

Spear, L. P. (2009). Heightened stress responsivity and emotional reactivity during pubertal maturation: Implications for psychopathology. *Development and Psychopathology*, *21*(1), 87–97. doi:10.1017/S0954579409000066

Spitzer, R., Kroenke, K., & Williams, J. (1999) Validation and utility of a self-report version of PRIME-MD: the PHQ primary care study. Primary care evaluation of mental disorders patient health questionnaire. *JAMA*, 282, 1737–44.

Spitzer, R.L. , Williams, J.W., & Löwe, K. K. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine,* 166, 1092–1097.

Stewart, J. G., Mazurka, R., Bond, L., Wynne-Edwards, K. E., & Harkness, K. L. (2013). Rumination and impaired cortisol recovery following a social stressor in adolescent depression. *Journal of Abnormal Child Psychology*, *41*(7), 1015–26. doi:10.1007/s10802-013-9740-1

Steinberg, L. (2008). A Social Neuroscience Perspective on Adolescent Risk-Taking. *Developmental Review : DR*, *28*(1), 78–106. doi:10.1016/j.dr.2007.08.002

Steinberg, L., & Avenevoli, S. (2000). The role of context in development of psychopathology: A conceptual framework and some speculative propositions. *Child Development. 71,* 66-74.

Sumter, S. R., Bokhorst, C. L., Miers, a C., Van Pelt, J., & Westenberg, P. M. (2010). Age and puberty differences in stress responses during a public speaking task: do adolescents grow more sensitive to social evaluation? *Psychoneuroendocrinology*, *35*(10), 1510–6. doi:10.1016/j.psyneuen.2010.05.004

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59,25−52.

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination relationship with negative mood and sleep quality. *Personality and Individual Differences*, 34, 1293–1301. doi:10.1016/S0191-8869(02)00120-4.

Thomsen, D. K., Mehlsen, M. Y., Olesen, F., Hokland, M., Viidik, A., Avlund, K., et al.

(2004). Is there an association between rumination and self-reported physical health? A one-year follow-up in a young and an elderly sample. *Journal of Behavioral Medicine,* 27, 215–231

Van den Bos, E., de Rooij, M., Miers, A. C., Bokhorst, C. L., & Westenberg, P. M. (2014). Adolescents' Increasing Stress Response to Social Evaluation: Pubertal Effects on Cortisol and Alpha-Amylase During Public Speaking. *Child Development*, *85*(1), 220–236. doi:10.1111/cdev.12118

Von Dawans, B., Fischbacher, U., Kirschbaum, C., Fehr, E., & Heinrichs, M., (2012). The social dimension of stress reactivity: acute stress increases prosocial behavior in humans. *Psychol. Sci*. 23, 651—660, http://dx.doi.org/10.1177/0956797611431576.

Von Dawans, B., Kirschbaum, C., & Heinrichs, M., (2011). The trier social stress test for groups (TSST-G): a new research tool for controlled simultaneous social stress exposure in a group format. *Psychoneuroendocrinology* 36, 514—522, http://dx.doi.org/10.1016/j.psyneuen.2010.08.004.

Ward, A., Lyubomirsky, L., Sousa, L., & Nolen-Hoeksema, S. (2003). Can't quite commit: Rumination and uncertainty. *Personality and Social Psychology Bulletin*, 29,96−107

Watkins ER. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin* 134(2):163–206.

Weiner, H. (1992). *Perturbing the organism: The biology of stressful experience*. Chicago: University of Chicago Press.

Wenzlaff, R.M., & Luxton, D.D. (2003). The role of thought suppression in depressive rumination. *Cognitive Therapy and Research.* 22, 293-308.

Young, E.A., & Nolen-Hoeksema, S. (2001) Effect of rumination on the saliva cortisol response to a social stressor. *Psychoneuroendocrinology* 26, 319-329

Zoccola, P. M., Dickerson, S. S., & Zaldivar, F. P. (2008). Rumination and cortisol responses to laboratory stressors. *Psychosomatic Medicine, 70*(6), 661–7. doi:10.1097/PSY.0b013e31817bbc77

Zoccola, P. M., Quas, J. A, & Yim, I. S. (2010). Salivary cortisol responses to a psychosocial laboratory stressor and later verbal recall of the stressor: The role of trait and state rumination. *Stress (Amsterdam, Netherlands), 13*(5), 435–43. doi:10.3109/10253891003713765

Deirdre Ann Katz

deirdreannhon@gmail.com deirdreannkatz.weebly.com

Education

2016 Pennsylvania State University, Doctor of Philosophy in
HUMAN DEVELOPMENT & FAMILY STUDIES

2011 Harvard Graduate School of Education, Master of Education in
MIND, BRAIN AND EDUCATION

2002 Loyola University Chicago, Bachelor of Science in
BIOLOGY & SECONDARY EDUCATION

Research Experience

2011-2015 Training Interdisciplinary Education Scientists (TIES) Predoctoral Research Fellowship (Funded by the Institute of Education Sciences, research arm of the US Department of Education)

2015-2016 *School-based Adolescent Stress Study* (Co-PIs Mark Greenberg & Deirdre Katz, Penn State, funded by Institute of Education Sciences Dissertation Research Grant)

2012-2014 *Comprehensive Approach to Learning Mindfulness (CALM) Study of Teacher Health and Wellbeing* (Co-PIs Patricia Jennings and Mark Greenberg, Penn State; funded by the 1440 Foundation and the Penn State Children, Youth and Families Consortium)

2011-2012 *Excellence in Social Emotional Learning (ExSEL) for Middle Schools*

2011-2013 *PATHS to Success* (PI Mark Greenberg) Pennsylvania Department of Health

2010-2011 Research Assistant *MIT Brain and Cognitive Sciences Department* (PI Rebecca Saxe)

2003-2005 Research Assistant *Marine & Environmental Research Institute of Pohnpei* (PI Simon Ellis)

Selected Publications

Abenavoli, R.M., Jennings, P.A., Greenberg, M.T., Harris, A.R., & **Katz, D.A**. (2013). The protective effects of mindfulness against burnout among educators. *Psychology of Education Review, 37*(2), 57-69.

Greenberg, M.T., **Katz, D.A.,** & Klein, L.C. (2015). The potential effects of social and emotional learning on biomarkers and health outcomes: A promissory note. J. A. Durlak, T. Gullotta, C. Domitrovich, P. Goren, and R. P. Weissberg (Eds), the *Handbook of Social and Emotional Learning. New York: Guilford.*

Katz, D.A., Greenberg, M.T., Klein, L.C. & Jennings, P.A., (2016). Associations between salivary α-amylase, cortisol and self-report indicators of health and wellbeing among educators. *Journal of Teacher and Teacher Education.*

Harris, A.R., Jennings, P.A., **Katz, D.A.,** Abenavoli, R.M., & Greenberg, M.T. (2015). Promoting stress management and well-being in educators: Outcomes of the CALM intervention. *Mindfulness.*

Katz, D. A., Harris, A.R., Jennings, P.A., & Greenberg, M.T., (Under review) Educators' emotion regulation strategies and their physiological indicators of chronic stress over the course of one school year

TEACHING EXPERIENCE

HDFS 433 *DEVELOPMENTAL TRANSITION TO ADULTHOOD* (ONLINE), INSTRUCTOR, PENN STATE

HDFS 495 *ADVANCED INTERNSHIP EXPERIENCE,* TEACHING ASSISTANT, PENN STATE

HDFS 239 *ADOLESCENT DEVELOPMENT*, INSTRUCTOR, PENN STATE