THE EFFECT OF ATTENTIONAL CONTROL ON THE RELATIONSHIP BETWEEN WORRY AND STRESS RESPONDING

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Abstract: Worry is a form of negative perseverative thinking and a maladaptive cognitive emotion regulation strategy associated with multiple forms of psychopathology (Nolen-Hoeksema & Watkins, 2011; Cisler et al., 2010). Perseverative worry may be exacerbated by deficits in attentional control (Armstrong et al., 2011). Attentional control is the ability to voluntarily shift and disengage attention while utilizing cognitive resources selectively to inhibit the processing of extraneous or irrelevant stimuli (Derryberry & Reed, 2002; Friedman & Miyake, 2004). Current influential theories propose that individuals high in attentional control are able to use attention to regulate their emotions (Oschner & Gross, 2008). However, low attentional control may be a cognitive vulnerability factor for developing pathological forms of anxiety due to a broad failure to deploy regulatory processes that directly influence changes in physiological stress responding (Armstrong et al., 2011). The current study evaluated whether trait attentional control mediated the relationship between trait worry and cortisol stress response after a psychosocial stressor. Participants (*N*=95) completed several self-report measures, the Trier Social Stress Test, and provided three saliva samples to measure cortisol stress response throughout the experiment. Results indicated that attentional control did not mediate the relationship between trait worry and cortisol stress response. However, exploratory analyses revealed that attentional control did moderate the relationship between cortisol stress response and self-reported acute worry during the stress recovery phase. Specifically, at low levels of attentional control, decreases in cortisol stress response predicted increases in acute worry levels post-stressor. These findings point toward alternative cognitive control measures better explaining the relationship between trait worry and cortisol stress responding (e.g. working memory, attentional biases). These findings also point toward attentional control potentially impacting the relationship between worry and physiological responses to stress. Specifically, worry may contribute to alterations in attentional control and stress, only to perpetuate enhanced negative feedback sensitivity of the HPA-axis and maintain the cycle of cortisol dysregulation—but only at low levels of attentional control.

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CHAPTER I

INTRODUCTION

Worry is a form of negative perseverative thinking and a maladaptive cognitive emotion regulation strategy associated with multiple forms of psychopathology (Nolen-Hoeksema & Watkins, 2011; Cisler, Olantunji, Feldner, & Forsyth, 2010). Perseverative worry may be exacerbated by deficits in voluntary attentional control (Armstrong, Zald, & Olantunji, 2011). Attentional control is the ability to voluntarily shift and disengage attention while utilizing cognitive resources selectively to inhibit the processing of extraneous or irrelevant stimuli (Derryberry & Reed, 2002; Fox, Russo, & Dutton, 2002; Friedman & Miyake, 2004). Current influential theories propose that individuals high in attentional control are able to use attention to regulate their emotions by either orienting away from internal or external threat representations, or orienting toward non-threatening, "safe" stimuli (Ferri, Schmidt, Hajak, & Canli, 2013; Oschner & Gross, 2008). Low attentional control may be a cognitive vulnerability for developing pathological forms of anxiety due to a broad failure to deploy complex regulatory processes that directly influence changes in physiological stress response

(Armstrong et al., 2011). Acute and chronic stress responses have been shown to contribute to long-term detrimental health consequences (Dimsdale, 2008; Schneiderman, Ironson, & Seigel, 2005), and may be greatly influenced by perseverative negative cognitions (e.g., worry) because they potentially prolong the physiological effects of anxiety on the body (Brosschot & Thayer, 1998). However, the effects of worry on physiological changes due to stress response have yielded equivocal findings. Therefore, attentional control may shed light on the causality of the relationship between worry and stress response. The goal of the current study is to assess self-reported attentional control as a mechanism for the effect of trait worry on cortisol stress response after a psychosocial stressor.

Worry is the core component of Generalized Anxiety Disorder (GAD; American Psychological Association, 2013). Recent models of GAD suggest worry may be employed to maintain heightened arousal, relative to a negative affective state, in order to prevent stark contrasts or shifts in the experience of negative emotion (Llera & Newman, 2010; Newman & Llera, 2011). Consistent with these models, worry has been found to be a correlate of autonomic inflexibility seen in individuals with GAD (Thayer, Friedman, & Borkovec, 1996). The Perseverative Cognition Hypothesis (Brosschot, Gerin, & Thayer, 2006) suggests that the representation of cognitive stressors (e.g., worry) creates a "fight-or-flight" action tendency that flows from the brain to peripheral stress responses including blood pressure, release of stress hormones (e.g., cortisol), and heart rate. Worry's ability to activate peripheral stress responses in the absence of a true internal or external stressor may interfere with adaptive threat and safety learning and contribute to psychopathology. Similarly, the Neurovisceral Integration Perspective (Thayer, Hansen, Saus-Rose, & Johnsen, 2009) suggests that top-down cortical control influences sympathetic nervous activity. For example,

this model suggests higher levels of resting heart rate variability or greater vagal tone at rest is evidence of prefrontal inhibitory control over subcortical circuits that directly influence whether an individual responds to environmental challenges in a controlled and adaptive manner (Thayer & Lane, 2009; Thayer et al., 2012; Hansen, Johnsen, and Thayer, 2003). Therefore, poor executive control of attention also may influence adaptive responses to posed or anticipated threat and possibly impair safety learning.

Chronic worry over time has shown to alter secretory patterns in neuroendocrine function. Specifically, chronic worry has resulted in increased or decreased suppression of hypothalamic-pituitary adrenal (HPA) axis function (Henry, 1992; Arborelius, Owens, Plotsky, & Nemeroff, 1999; Faravelli et al., 2012). Altered activity of the HPA-axis, as a result of chronic stress, is widely considered an important factor in the etiology of psychopathology (Miller, Chen, & Zhou, 2007). More specifically, the gradual development of a disconnection between a stressor, and adaptive responses to the stressor, can result in persistent activation of the HPA-axis and sustained elevations in cortisol levels (Deakin & Graeff, 1991; Henry, 1992). Therefore, cortisol reactivity may indicate difficulties in response inhibition, cognitive flexibility, and attentional regulation (Thayer et al., 2009). Unfortunately, little research has examined the neuroendocrine changes underlying GAD, and the available evidence on cortisol stress responding in GAD specifically has yielded inconsistent findings. The first analyses of HPA-axis function in GAD involved using dexamethasone suppression tests to measure whether the adrenocorticotropic hormone (ACTH) secretion by the pituitary could be suppressed. Results indicated elevated non-suppression rates, or reduced negative feedback sensitivity of the HPA-axis, in individuals with GAD (Schweizer, Swenson, Winokur, Rickels, & Maislin, 1986; Tiller, Biddle,

Maguire, & Davies, 1988). These results suggest that individuals with GAD experience a reduced sensitivity in a feedback system designed to decrease HPA-axis function and stabilize the autonomic nervous system. Another study examined baseline cortisol levels among elderly individuals with GAD compared to non-anxious controls and discovered a nearly 50% increase in early morning basal cortisol levels (Mantella et al., 2008). These results suggest elevated cortisol levels may reflect a greater anxious awakening state. Furthermore, greater levels of worry and GAD severity were associated with elevated diurnal cortisol profiles (Mantella, et al., 2008). Deviations in diurnal profiles have be associated with increased difficulty for the HPA-axis' ability to recover from stressors and lead to less than ideal health outcomes. Several other studies also have reported increases in basal cortisol levels, as measured by saliva or plasma, among individuals with GAD (Greaves-Loed et al., 2007; Pomara, Willoughby, Sidtis, Cooper, & Greenblatt, 2005; Tafet, Feder, Abulafia, & Rofman, 2005).

Conversely, several studies have failed to show increased adrenocortical activity in GAD (Rosenbaum et al., 1983; Hoehn-Saric, McLeod, & Zimmerli, 1989; Chaudieu et al., 2008; Steudte et al., 2011). Steudte and colleagues (2011) did not find differences in diurnal cortisol profiles of salivary cortisol, but they did find lower cortisol levels in hair samples of individuals with GAD compared to controls. These researchers hypothesized that initial chronic HPA-axis activation, or *hyper*-cortisolism in GAD, may eventually lead to *hypo*-cortisolism over time (Steudte et al., 2011; Hellhammer and Wade, 1993). Hypo-cortisolism may account for lowered cortisol levels during baseline, reduced cortisol reactivity to stress, and an increased negative feedback sensitivity of the HPA-axis (Heim, Ehlert, & Hellhammer, 2000; Steudte et al., 2011). Increased negative feedback sensitivity of the HPA-

axis may contribute to heightened anxiety sensitivity and enhanced worry only to perpetuate a cycle of emotional and physiological dysregulation (Hellhammer and Wade, 1993; Heim et al., 2000).

Extant literature has shown that the prefrontal cortex (PFC) is especially important for regulating aversive emotion and conflict through top-down attentional control (Bishop, Duncan, Brett, & Lawrence, 2004; Ohman, 2005). Research suggests attentional control may be a mechanism for facilitated attention, difficulties disengaging from threatening stimuli, and attentional avoidance (Cisler & Koster, 2010; Cisler, Olatunji, & Lohr, 2009; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005; Fox, et al., 2002; Mogg, Bradley, Miles, & Dixon, 2004). Furthermore, recent research suggests low attentional control may serve as a mechanism for relationships between repetitive negative thinking and associated symptomology (Mills et al., 2016). As previously mentioned, worry is postulated to be a primary mechanism by which an individual maintains the physiological effects of a stressor through cognitive representation (Brosschot et al., 2006; Brosschot, Pieper, & Thayer, 2005). Whereas few studies have shown that homeostatic recovery after emotional distress may be slowed due to repetitive negative thinking (Glynn, Christenfeld, & Gerin, 2002; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006), several studies also have shown that state and trait worry are associated with increased physiological activation (see for review: Brosschot et al., 2006). Diminished attention control may then confer risk for preservative worry by impairing an individual's ability to prevent or inhibit intrusive, unwanted thoughts (Brown, Moras, Zinbarg, & Barlow, 1993). Thus, deficits in attentional control may facilitate excessive negative cognition by allowing invasive thoughts to gain entry into working memory despite attempts to inhibit them (Rosen and Engle, 1998). If worry prolongs the

cognitive representation of physiological activation related to stress, and levels of attentional control determine whether these intrusive thoughts are suppressed, then properties of attentional control may make it a likely candidate as a mediator of stress responding and worry. Therefore, the current study seeks to assess self-reported trait attentional control in order to examine the relationship between trait worry and cortisol stress responding after a psychosocial stressor. Trait attentional control will be examined as a mediator of the relationship between trait worry and stress response. For this mediational analysis, trait attentional control was assessed using the Attention Control Scale (ACS; Derryberry & Reed, 2002) total score and trait worry was assessed using the Penn-state Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) total score. Finally, cortisol stress response was measured by calculating area under the curve with respect to increase (AUCi), as detailed by Preussner et al., (2003). It was hypothesized that the relationship between trait worry and cortisol stress response will be mediated by trait attentional control. Specifically, it was hypothesized that trait worry will significantly predict stress response (path c), trait worry will significantly predict trait attentional control (path a), and trait attentional control will significantly predict stress response when controlling for trait worry (path b), indicating an indirect effect of trait worry on cortisol stress responding through trait attentional control (see Figure 1).

CHAPTER II

METHODOLOGY

Participants

Participants included 95 individuals (56% female; 68% Caucasian) with a mean age of 19.62 (*SD* = 1.92) recruited from a large mid-western university. Participants completed informed consent prior to participating in the study. The study was approved by the university's Institutional Review Board prior to recruitment and participants were offered course credit for their time.

Measures

Demographics form. Demographics included questions regarding age, sex, race, ethnicity, and year in school. This form also asked health questions to assess factors that could potentially affect cortisol levels, including health-related behaviors within 2-hours of testing (caffeine consumption, digestion, smoking, physical activity), disease states (asthma, allergies, other health conditions), body mass index (BMI), and medication use (Kirschbaum & Hellhammer, 1992; Kirschbaum, Pirke, & Hellhammer, 1995).

Penn-State Worry Questionnaire (PSWQ). The PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report questionnaire designed to assess trait worry and to capture the generality, excessiveness, and uncontrollability characteristics of pathological worry. Items are rated on a five-point Likert scale ranging from 1 (*Not at all typical of me*) to 5 (*Very typical of me*). Higher scores indicate more severe worry. In the current study, the PSWQ demonstrated good internal consistency ($\alpha = .95$).

Attention Control Scale (ACS). The ACS (Derryberry & Reed, 2002) is a 20-item self-report measure of attention across two domains: focusing, the ability to maintain attention on a given task, and shifting, the ability to reallocate attention to a new task or to engage attention on multiple tasks. Each item is rated on a 4-point Likert scale ranging from 1 (*almost never*) to 4 (*always*) with higher scores indicative of better attentional control. This questionnaire has shown to be a valid measure of attentional regulation (Judah, Grant, Mills, & Lechner, 2014) and has demonstrated good internal consistency in the present sample ($\alpha = .91$). It is important to note that the ACS does not contain questions related to affectively valenced situations, and as such, attempts to capture a general information-processing trait uncontaminated by reactions to emotional stimuli or cognitions.

Subjective Units of Distress Scale (SUDS). In order to ensure stress inductions were successful and monitor state worry levels throughout the experiment, participants were administered a SUDS consisting of a 9-point Likert scale assessing levels of state worry. The manipulation check measure was modeled after those used in other studies (e.g., Behar Zuellig, & Borkovec, 2005; Oathes et al., 2008). Participants were asked to rate the extent to which they felt "worried" on a scale that ranges from 0 (*not at all*) to 8

(*extremely*). SUDS data were collected at 5 time points during the current study and subsequently converted to Z-scores prior to data analyses. Five time points were selected to evaluate basal levels of state worry (S1), after the 5-minute TSST prep period (S2), immediately after the stress test (S3), after cortisol collection 2 (S4), and during the recovery period at cortisol collection 3 (S5). These SUDS ratings at 5 time points provided manipulation check data and state self-report data during the experiment.

Tasks

Trier Social Stress Test (TSST). The TSST was applied as described by Kirschbaum and colleagues (1993). Participants sat in a chair with a video camera and tape recorder in front of them and pointed in their direction. Participants were instructed to prepare a 5-minute speech where they are asked to introduce themselves and convince two research confederates that they are an excellent applicant for a leadership position. The participants were instructed that the two confederates were specially trained to monitor non-verbal behavior. Furthermore, the participants were told that a voice frequency analysis and a video analysis would be performed to analyze their non-verbal behavior. The confederates were both male and female. Following these instructions, the subjects were given 5 minutes to prepare their free speech. They were given paper and pencils to outline their talks, however, they were instructed they would not be able to use the written concept for their speech. After the 5-minute prep period, participants were instructed to deliver their speeches. Upon completion of the speech, participants were instructed to complete a serial subtraction task. Participants were asked to subtract the number 13 from 1,022 as quickly and as accurately as possible.

Data recording and reduction

Salivary Cortisol (CORT). Three saliva samples were taken from each participant over the course of the experiment using the Salimetrics saliva collection aid (Salimetrics, LLC, Carlsbad, USA). One sample was taken at baseline (T1: 10 minutes upon arrival to laboratory), a second sample was taken approximately 30-minutes after the stress test to detect any changes in cortisol levels after exposure to stress (T2), and a third sample was taken during the post-stress period (T3: ~60 minutes post-stressor). Saliva samples were frozen at −20 degrees Celsius. Before analysis, samples were thawed and centrifuged at 4 degrees Celsius at 3000 rpm for 15 min to remove particulate matter. Samples were then assayed in duplicate by immunoassay (Salimetrics, LLC, Carlsbad, USA). The average of the duplicates for each sample was used in the analyses. The sensitivity of the saliva assay is 0.03 μg/dL. Intra- and interassay coefficients of variation ranged from 2% to 9%. Cortisol stress response was measured by calculating area under the curve with respect to increase (AUCi), as detailed by Preussner et al., (2003) in order to limit the number of statistical comparisons needed. AUCi is a parameter more related to sensitivity of the HPA-axis system and emphasizes cortisol changes over time (Preussner et al., (2003).

Procedure

Experimental sessions were run between 11 a.m. – 6 p.m., respectively. Participants were provided written informed consent approved by Oklahoma State University's Institutional Review Board. Participants were seated in a chair and instructed to complete questionnaire measures online. When participants arrived to the laboratory, research assistants began a timer that prompted them to collect a baseline saliva sample (T1) 10 minutes post-laboratory arrival. Additionally, SUDS rating scale

(S1) was administered after T1 saliva collection. Upon completion of online questionnaire data, T1 saliva and S1 self-report participants were guided to the experimental room. Once participants were attached to peripheral measuring devices, they were introduced to the TSST Protocol (Kirschbaum et al., 1992; Kirschbaum et al., 1993). Participants were instructed to use 5-minutes to prepare a 5-minute speech on how they would be an ideal candidate for a leadership position. After completing a 5-minute speech prep, participants were administered a SUDS (S2) and then instructed to deliver their speeches. If participants exhibited difficulty maintaining free-flowing speech, research confederates had a list of questions they could ask to prompt the participant to continue speaking. After participants delivered 5-minutes of free speech, they were instructed to complete a serial subtraction task for 5 minutes total. After completing the serial subtraction task, participants were administered a SUDS (S3) and asked to provide a second saliva sample (T2: ~30 minutes after the beginning of the TSST). After T2 collection another SUDS (S4) was administered. Finally, participants were administered a SUDS (S5) and asked to provide a final saliva sample (T3) 60 minutes after the start of the TSST protocol.

Analytic Approach

Identification of covariates. Variations in characteristics of the sample, including sex, smoking, digestion, caffeine intake, physical activity, body mass index (BMI), illness, and medications affect cortisol levels. Failure to account for these variables as covariates in analyses could affect the consistency of observed associations. Therefore, the variables listed above were controlled for in the current analyses.

Correlation analyses. Zero-order correlations were computed to assess the associations between the independent variable (PSWQ), the proposed mediator (ACS), the primary dependent variable (AUCi), and covariates.

Manipulation check. In order to evaluate whether worry was induced during the experiment, SUD ratings (S1-S5) were entered into a repeated measures analysis of variance (ANOVA). Post-hoc comparisons were used to follow-up analyses.

Mediation analysis. In order to test the hypothesis that attention control (ACS) mediates the relationship between trait worry (PSWQ) and cortisol stress response (AUCi), a simple mediation analysis was conducted using PROCESS in SPSS (Hayes, 2012). To estimate the total effect, *c*, was conducted with PSWQ entered as the predictor variable, AUCi entered as the outcome variable, and demographic and health related behaviors variables entered as covariates. The direct effect, c', was estimated with PSWQ entered as the predictor variable and AUCi as the outcome variable, controlling for ACS and the covariates. The effect of PSWQ on ACS, *a*, was estimated with the covariates entered at step one and AUCi entered at step two. In order to estimate the effect of ACS on AUCi, *b*, ACS was entered as the predictor variable and AUCi entered as the outcome variable, controlling for PSWQ and the covariates. The indirect effect (*ab*) was calculated by multiplying the coefficient *a* by the coefficient *b*. In order to determine whether the indirect effect was significant (i.e., whether ACS mediated the relationship between PSWQ and AUCi), bootstrapping, an alternative to the Sobel test that also directly tests the indirect effect yet does not assume normality of the sampling distribution, was conducted. Bootstrapping involves generating a number of samples (*k*) from the original sample in order to estimate an effect from each of the *k* samples. These estimated effects

are then used to generate confidence intervals, which are then used to test whether the indirect effect is significantly different from zero. Given the nature of bootstrapping, there is the potential for skew or bias in the distribution of the estimated effects and resulting confidence intervals compared to that of the original sample. One way to reduce this potential bias is to conduct bias-corrected bootstrapping, a type of bootstrapping that accounts for the bias in the bootstrapped samples (Steck & Jaakkola, 2004). A bias-corrected bootstrap-confidence interval (CI) for the product of these paths that does not include zero provides evidence of a significant indirect effect of PSWQ on AUCi through ACS (Preacher & Hayes, 2008; Hayes, 2012). For the present study, bias-corrected bootstrapping (k = 5,000) was conducted in order to generate a 95% confidence interval (CI) to test the significance of the indirect effect of trait attentional control in the relationship between trait worry and cortisol stress responding.

CHAPTER III

FINDINGS

Results

Manipulation check. In order to evaluate the effects of time on self-reported worry ratings through the duration of the experiment, SUDS ratings (S1-S5) were entered into a repeated-measures ANOVA. Normality checks were carried out on the residuals, which were approximately normally distributed. Results showed a significant within-subjects effect, $F(4,91) = 31.29$, $p = .000$. Post-hoc pairwise comparisons using a Bonferroni correction revealed significant differences between S1 ($M = .89$, $SD = 1.62$) and S2 ($M = 2.11$, $SD = 2.14$)[$r = -1.22$, $p = .000$], S1 and S3 ($M = 2.42$, $SD = 2.33$)[$r = -1.53$, $p = .000$], S1 and S4 ($M = 1.37$, $SD = 1.82$) [$r = -.48$, $p = .005$], S2 and S4 [$r = .74$, $p = .001$], S2 and S5 ($M = .74$, $SD = 1.3$) [$r = 1.37$, $p = .000$], S3 and S4 [$r = 1.05$, $p = .000$], S3 and S5 [$r = 1.7$, $p = .000$], S4 and S5 [$r = .63$, $p = .002$] (see Figure 2), such that worry scores significantly increased from baseline, to after speech prep, to after the stress test, and to cortisol collection 2. There was no significant differences between S2 (after

speech prep) and S3 (after stress test) [r = -.32, p = 1.00], indicating worry levels remained elevated both during preparation for and during the stress test. Similarly, there was no significant difference between baseline worry (S1) and worry during the recovery period (S5) [r = .152, p = 1.00], indicating self-report worry levels returned back to baseline by the end of the experiment.

Outliers. Data were screened for outliers via Mahalanobis distance, Cook's distance, and centered leverage values. Three cases had a combination of at least two extremely large Mahalanobis, Cook's, or centered leverage values and were removed from the data set. Based on removal of outliers, the final sample included 92 subjects.

Correlations. Zero-order correlations among the dependent variable, the proposed mediator, the independent variable, and covariates are presented in Table 1. Cortisol stress response (AUCi) was significantly correlated with attentional control (ACS, r = .236, p = .012) and marginally associated with trait worry (PSWQ, r = -.159, p = .064). PSWQ (r = .428, p = .000) and ACS (r = -.190, p = .035) were each significantly correlated with sex. AUCi showed a marginal association with sex (r = -.161, p = .063) and caffeine intake (r = .156, p = .068). ACS showed a marginal association with nicotine intake (r = .164, p = .059).

Mediation Analyses. Using PROCESS (Hayes, 2012) Model 4 in SPSS, the total, direct, and indirect effects of trait worry on cortisol stress response through attentional control were evaluated. Covariates for these analyses included sex, digestion, nicotine intake, caffeine intake, physical activity, BMI, illness, and medications and these were entered in the models simultaneously with predictor variables.

We hypothesized that attention control (ACS) would mediate the association between trait worry (PSWQ) and cortisol stress reactivity (AUCi). Following the procedure outlined by Preacher and Hayes (2008), we tested the products of (1) the independent variable (α path: unstandardized beta = -0.24, *SE* = 0.05, $p < .001$), and (2) the mediator to the dependent variable (AUCi) when the independent variable is taken into account (β path: unstandardized beta = 0.28, *SE* = 0.13, $p = 0.03$). Caffeine (unstandardized beta = 5.55, *SE* = 2.6, $p < .034$) and nicotine intake (unstandardized beta = 4.4, *SE* = 2.3, $p < .056$) also appeared to significantly predict AUCi on the β path. A variation on the Sobel (1982) test that accounts for the non-normal distribution of the αβ path through bootstrapping procedures (number of resamplings = 5000) revealed that the mediator (ACS), controlling for covariates, was not significant (unstandardized beta = -0.06, *SE* = .06, $p = 0.33$). Examination of the 95% confidence interval of the indirect path (αβ) overlapped with zero for AUCi (lower limit = -0.16, upper limit = 0.01), indicating no mediation effect. Thus, attention control did not mediate the relationship between trait worry and cortisol stress response. Complete results of these analyses are provided in Table 2 and the outcome model provided in Figure 3.

Exploratory Analyses. Acute self-report worry throughout the experiment, as measured by SUD (S1-S5), was examined to determine its relationship with attentional control and cortisol stress response Correlational analyses showed that attentional control (ACS) was significantly associated with acute worry at S1 ($r = -.438$, $p = .000$), S2 ($r = -.456$, $p = .000$), S3 ($r = -.441$, $p = .000$), S4 ($r = -.490$, $p = .000$), and S5 ($r = -.373$, $p = .000$), such that higher scores of ACS were significantly correlated with lower levels of self-reported acute worry throughout the experiment. Similarly, AUCi was significantly

associated with acute worry scores at cortisol collection 2 (S4) after the stressor (r = -.201, p = .052) and during the recovery period (S5, r = -.273, p = .008), such that higher levels of cortisol stress response were associated with lower levels of self-reported acute worry after the stressor and during recovery. AUCi was not associated with acute worry at S1 (p = .074), S2 (p = .518), or S3 (p = .327). Regarding covariates, acute worry at S4 (r = .248, p = .016) was significantly associated with sex. Acute worry levels at S5 (r = .194, p = .059) was marginally associated with sex. Similarly, AUCi was marginally correlated with physical activity (r = .198, p = .056).

Previous literature suggests that cortisol levels and stress induced cortisol responses predict psychological traits reflecting stress sensitivity and differences in vigilance processing during stress exposure (Everaerd et al., 2015; Laceulle et al., 2015; Henckens et al., 2016). AUCi takes into account the vertical distances of each cortisol measurement from baseline, while ignoring the distance from zero, and measures patterns or rates of change in repeated measures over time (Fekedulegn et al., 2007). Therefore, the current study determined S5 would best fit the model as an outcome variable, as it is the very last measure of acute worry in the experiment. In order to explore whether attentional control (ACS) moderated the relationship between stress induced cortisol changes (AUCi) and self-reported acute worry levels after stress while controlling for covariates, Model 1 in PROCESS (Hayes, 2012) was conducted. Before the analysis, data (N = 95) were screened for outliers via Mahalanobis distance, Cook's distance, and centered leverage values. One case had a combination of two extremely large Mahalanobis and centered leverage values and was removed from the data set. Based on removal of the outlier, the final sample included 94 subjects. Whereas correlational

analyses indicated acute worry levels after the stressor (S4) and during recovery (S5) were associated with cortisol stress response and attentional control, the current exploratory analyses focused on predictors of acute worry after stress. Changes in cortisol stress response (AUCi) was entered as the predictor variable, attentional control the predicted moderator, acute worry scores during recovery as the dependent variable, and covariates (i.e., see mediation analysis) entered simultaneously in the model. The model was significant, $F(9,84) = 3.88$, $p = .000$, $R^2 = .294$. As shown in Table 3, there was no significant main effect of AUCi ($b = -.563$) [$t(93) = -.170$, $p = .098$], indicating cortisol stress response was not a significant predictor of self-reported acute worry during the recovery period (S5). However, there was a significant main effect of ACS ($b = -.350$) [$t(93) = -3.62$, $p = .000$], indicating attentional control significantly predicted acute worry levels after stress (S5). There also was a significant interaction between AUCi and ACS ($b = .190$)[$t(94) = 2.24$, $p = .03$]. The significant interaction between AUCi and ACS was probed by testing the conditional effects of AUCi at three levels of ACS, one standard deviation below the mean, at the mean, and one standard deviation above the mean. As shown in Table 4, AUCi was significantly related to S5 when ACS was one standard deviation below the mean ($t(93) = 3.13$, $b = -.35$, $p = .002$), but not when ACS was at the mean ($b = -.17$, $p = .098$) or one standard deviation above the mean ($b = .02$, $p = .88$). The Johnson-Neyman technique showed that the relationship between AUCi and S5 was significant when attentional control was less than -0.15 standard deviations below the mean but not significant with higher values of attentional control. This interaction is illustrated in Figure 4.

CHAPTER IV

CONCLUSION

Discussion

The goal of the current study was to determine whether trait attentional control mediates the relationship between trait worry and cortisol stress response. Results indicated that trait attentional control did not mediate this relationship. Whereas studies have shown perseverative worry to be exacerbated by deficits in attentional control or the control of information processing (Derryberry and Reed, 2002), the lack of mediation in the current study indicates that self-reported, general information processing does not explain the relationship between trait worry and cortisol stress response. It could be that alternative cognitive control measures better explain this relationship. For example, LeMoult and colleagues (2019) examined individual differences in working memory capacity (WMC), a cognitive control mechanism associated with attentional control, and cortisol responses to stress among individuals with GAD and normal controls.

To measure WMC after a stressor (e.g., TSST), these authors used an adapted reading span task that simultaneously presented emotionally valanced distractors. Results showed that the relationship between WMC and cortisol stress responding was specific to individuals with GAD. Specifically, individuals with GAD, and lower WMC, exhibited greater cortisol levels in the presence of neutral distractors. Lower WMC in the presence of negative distractors resulted in attenuated cortisol recovery from the stressor. WMC was unrelated to cortisol stress responding and recovery in the control group. These results suggest individual differences in WMC are associated with individual differences in GAD-related cortisol stress responses. Therefore, measuring components of attentional control may be important for understanding the relationship between worry and cortisol stress responding.

It could be speculated that prolonged cortisol recovery in the presence of negative distractors is due to difficulties disengaging from threatening stimuli. Working memory has been linked to attentional control difficulties with shifting attention among anxious individuals, specifically for threat-related information (Judah, Grant, Mills, and Lechner, 2014). Similarly, several studies have shown that baseline, early stage attentional biases predict cortisol responses to stress (Fox et al., 2001; Pilgrim et al., 2010). Furthermore, randomized controlled trials experimentally modifying attention have shown that attentional training has been effective in reducing symptoms of anxiety (Bar-Haim, 2010; Li et al., 2008; Hakamata et al., 2010), as well as emotional reactivity to stress (MacLeod et al., 2002; Amir et al., 2008). Taken together with the current study findings, exploring alternative cognitive control mechanisms (e.g., attentional biases; WMC) may assist in

elucidating cortisol dysregulation found in GAD and the relationship between worry and stress response.

As mentioned above, results of the current study suggest general self-report indicators of information processing do not explain the relationship between trait worry and changes in cortisol stress response. However, results from exploratory analyses indicated that attentional control does moderate the relationship between cortisol stress response and acute worry after stress. Interestingly, at lower levels of attentional control, decreases in cortisol over time predicted higher levels of acute worry after stress. This finding is consistent with the Neurovisceral Integration model (Thayer and Lane, 2009) that suggests impaired cognitive processes (e.g., attention) can promote perseveration (e.g., worry), hypervigilance, and continued system activation which may limit resource availability for other adaptive processes. Specifically, lower attentional control may impair information processing (e.g., stimulus, context), which may inhibit flexible up and down regulation of acute stress responses (McEwen, 2008, 2000). This inflexibility may result in the experience of increased negative affect or emotions. Increased worry may be employed as a maladaptive cognitive coping strategy that interferes with emotional processing of information, the integration of information incompatible with the 'fear' or feared outcome, and the subsequent formation of new memories that can evoke emotional change (Foa and Kozak, 1986; Verkuil, Brosschot, Gebhardt, & Thayer, 2010). Worry then may contribute to alterations in attentional control and physiological responses to stress through perpetuating enhanced negative feedback sensitivity and maintaining a cycle of cortisol dysregulation—but only at low levels of attentional control.

Several limitations are worth mentioning. First, whereas the manipulation check revealed significant changes in self-reported state worry levels across time in the experiment, the peak worry level means indicated participants experienced only mild-moderate levels of state worry. Previous research suggests that subjective distress and/or negative affect in response to stress is not specifically related to cortisol changes (Dickerson and Kemeny, 2004). In a meta-analysis on acute stressors and cortisol responses, Dickerson and Kemeny (2004) revealed that specific characteristics of a stress test (e.g., social-evaluation, uncontrollability) are associated with cortisol elevations despite producing negative affective states. Whereas social-evaluative threat and uncontrollability was associated with greater cortisol reactivity, it was not associated with greater increases in self-reports of distress, and these more emotional states were not correlated with cortisol responses. (Dickerson and Kemeny, 2004). Therefore, the current study's parameters were adequate in eliciting a salient stress response and changes in state worry levels, though these state worry levels averaged mild to moderate.

Second, our study utilized a time-limited, acute laboratory stressor. Cognitive and affective processes that lead to cortisol changes may be more likely extended in naturalistic stressors. It is unclear the extent to which cortisol responses to acute laboratory stressors mirror those to naturalistic stress. Future studies may choose to adapt experimental parameters to incorporate more naturalistic contexts, which could lead to evaluating greater persistence in cortisol elevations or decreases over time.

Third, a predominantly female and college-aged analogue sample was examined in the current study. Whereas some empirical studies have found small differences in cortisol responses in men and women or young and elderly participants, gender and

average age do not appear to explain cortisol stress response variability (Dickerson and Kemeny, 2004). Future studies may choose to utilize a more representative clinical sample while controlling for lifetime history of chronic worry to improve generalizability of the current findings. Finally, our sample was relatively low considering the number of covariates controlled for in the analyses. Increasing the sample size could allow for improved determination of the mediating effect of attentional control on worry and stress reactivity. Similarly, additional statistical procedures such as growth models may assist in investigating whether experimental variables indicate unobservable constructs. These latent constructs may help with considering a more dynamic approach to understanding attentional control (Preacher et al., 2008).

In sum, that current study found that trait attentional control does not mediate the relationship between trait worry and cortisol stress response. However, attentional control did moderate the relationship between stress responsivity and acute worry levels post-stressor. Future research should continue to explore alternative attentional or cognitive control mechanisms that may elucidate the relationship between worry and cortisol stress responding.

CHAPTER V

REVIEW OF THE LITERATURE

Generalized Anxiety Disorder

Generalized Anxiety Disorder (GAD) is a persistent mental disorder characterized by excessive and chronic worrying, anxiety, tension, and other somatic complaints that occur more days than not for a period of at least 6 months (APA, 2013). GAD has an estimated lifetime prevalence rate of 5.7% (Kessler et al., 2005), and is associated with a significant burden of cost due to an over-utilization of health care resources, social disability (Wittchen, Kessler, Pfister, Hofler, & Lieb, 2000), and low remission rates (Kessler, Keller, & Wittchen, 2001; Maier et al., 2000). GAD is the most common disorder in primary care (Ormel et al., 1994) and is highly comorbid with other disorders (e.g., Major Depressive Disorder) (Stein, 2001). Due to the high comorbidity of GAD with other mental disorders, there has been less focus on GAD as a discrete disorder, which has interfered with improving the recognition, diagnosis, and treatment of this condition (Wittchen, 2002). Therefore, examining the core symptoms of GAD may help

identify treatment targets and promote the reduction of economic and social burdens relative to this common and disabling disorder.

Worry

GAD is characterized by chronic, uncontrollable worry that is verbal-linguistic in nature (Borkovec & Inz, 1990). Worry is a form of negative perseverative thinking and a maladaptive cognitive emotion-regulation strategy associated with multiple forms of psychopathology (Nolen-Hoeksema & Watkins, 2011; Cisler et al., 2010). Worry is a fundamental characteristic in social anxiety, panic, agoraphobia, posttraumatic stress disorder, anorexia nervosa, bipolar disorder, and psychosis, suggesting it is a transdiagnostic phenomenon (Ottaviani et al., 2016; Nolen-Hoeksema & Watkins, 2011). As a result, several frameworks have been developed to describe the effects of worry on emotional and physiological processing. For example, Brosschot and colleagues (2006) proposed the Perseverative Cognition Hypothesis, which suggests that the representation of cognitive stressors (e.g., worry) creates a "fight-or-flight" action tendency that cascades from the brain to peripheral stress responses including the release of stress hormones (e.g., cortisol), blood pressure, and heart rate. Furthermore, perseverative cognitions prolong this state of action readiness, representing a highly vigilant state, which may produce chronic increases in cardiovascular, adrenal, and immunological activation (Brosschot, Gerin, & Thayer, 2006).

Thayer and colleagues (1996) evaluated cardiac autonomic activity between individuals diagnosed with Generalized Anxiety Disorder (GAD) and non-anxious controls during a worry and relaxation task. Subjects were asked to determine a worry topic to be used during the worry condition. Electrocardiogram (ECG) and respiration

were recorded throughout the experiment. A 5-minute baseline recording proceeded each of the experimental sessions. Three 3.5-minute recordings were obtained during the worry and relaxation sessions. Several measures of parasympathetic functioning were calculated including the average cardiac inter-beat interval (IBI), a variance of average IBI's in milliseconds, and the average of the absolute values of successive differences in R-spike intervals in milliseconds (ms). Results demonstrated that individuals with GAD exhibited decreased cardiac parasympathetic activity compared to non-anxious controls across baseline, relaxation, and worry tasks. These results indicate that individuals with GAD exhibit lower cardiac parasympathetic activity regardless of condition. Moreover, worrying also was associated with less cardiac parasympathetic activity compared to relaxation across subjects. Overall, the results of this study support the process of worry as a behavioral correlate of autonomic inflexibility (Thayer et al., 1996; Thayer & Lane, 2000; Hansen, Johnson, and Thayer, 2003; Thayer and Brosschot, 2005; Thayer and Sternberg, 2006).

Fisher and Newman (2013) evaluated the effects of state and trait worry, and GAD on stress responding during and following a worry or relaxation induction. Heart rate (HR), respiratory sinus arrhythmia (RSA), and salivary alpha-amylase (sAA) were measured in GAD worriers, non-GAD high worriers, and healthy controls to assess activation of the autonomic nervous system. Participants were assigned randomly to worry or relaxation conditions. Upon completion of the worry or relaxation induction, participants were prompted to report the degree to which they felt worried and relaxed. Two iterations of the Paced Auditory Serial Addition Task (PASAT; Gronwall, 1977), used to assess indices of information processing speed, divided attention, and working

memory capacity, were completed after the worry or relaxation inductions and participants were asked to report their level of worry after each PASAT. sAA samples were collected at baseline and 5 minutes after the last PASAT was administered.

Results demonstrated that HR significantly increased and RSA significantly decreased during the stress period across all groups and conditions. Baseline levels of sAA and state worry significantly moderated RSA response during the stress period, with higher baseline sympathetic arousal predicting greater reductions in RSA and increases in HR, respectively. State worry was found to decrease HR during the stressor. Lastly, in individuals with GAD, higher baseline arousal significantly predicted decreased sAA over the stress period. In contrast, higher baseline arousal at baseline predicted increased sAA in control groups. Taken together, these results highlight the effect of worry on diminished HR stress response and also propose that baseline adrenergic tone plays a significant role in GAD (Fisher & Newman, 2013). These findings also add to a growing body of literature supporting the Avoidance Theory of Worry (Borkovec, Alcaine, & Behar, 2004) and the role of worry in inhibiting sympathetic reactivity (Borkovec & Hu, 1990; Borkovec & Costello, 1993; Llera & Newman, 2010).

The Avoidance Theory of Worry suggests that the persistent nature of worry is due to the perceived notion that worry helps avoid catastrophe (Borkovec et al., 2004). Specifically, individuals with GAD use worry to suppress emotional and physiological responses to aversive imagery (Borkovec et al., 2004; Mineka & Zinbarg, 2006). More recent literature suggests worry may be used to maintain a negative affective state in order to avoid stark shifts in negative emotionality (Llera & Newman, 2010; Newman & Llera, 2011; Llera & Newman, 2014; Newman, Llera, Erickson, & Przeworski, 2014).

Llera and Newman (2010) evaluated physiological and subjective responses to negative and positive emotional stimuli with a variation of emotive contexts using film clips (e.g., happy, calm, sadness). Individuals with GAD and healthy controls were instructed to engage in a worry, relaxation, or a neutral thinking induction for 2 minutes prior to viewing each film clip (e.g., Borkovec & Inz, 1990; Behar, Zuellig, & Borkovec, 2005). Participants also were asked to complete the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) after each clip to assess extent of subjective emotional reactivity to the different film clips.

Results revealed no resting baseline difference in vagal tone between GAD and healthy controls. Consistent with the avoidance theory of worry and the contrast avoidance model (Newman & Llera, 2011), worrying compared to relaxing resulted in reduced vagal withdrawal and reduced subjective reactivity for all subjects relative to the fearful clip. Those with GAD in the neutral induction condition did not exhibit vagal withdrawal upon exposure to the fearful clip, whereas non-anxious controls did. Both individuals with GAD and healthy controls receiving the neutral induction reported greater subjective responding to the fear clip. All inductions led to increased vagal activity to sad clips, however, prior worry led to reductions in negative affect in response to the sad clip. Taken together, these results suggest that the process of worry may diminish anxious responding and facilitate the avoidance of processing negative emotions (Llera & Newman, 2010; Borkovec et al., 2004).

Llera and Newman (2014) conducted a similar study that tracked changes in negative emotionality from baseline to worry inductions following exposure to emotive film clips. Results indicated that worrying resulted in an upward shift in negative emotion

from baseline. Negative emotionality persisted across negative exposures after the worry induction. Lower emotionality was observed during neutral and positive inductions, thus resulting in greater increases in response to negative exposures. When asked which induction was more helpful in coping with negative exposures, healthy controls reported worry was unhelpful, whereas individuals with GAD reported worry helped with coping (Llera & Newman, 2014). Overall, these studies suggest that worry diminishes sympathetic reactivity and that worry may be maintained by this diminishing effect due to the perception that it serves to evade further increases in anxiety or negative emotionality (Thayer et al., 1996; Thayer & Lane, 2000; Hansen, Johnson, and Thayer, 2003; Thayer and Brosschot, 2005; Thayer and Sternberg, 2006; Fisher & Newman, 2013; Llera & Newman, 2010; Llera & Newman, 2014). Furthermore, the process of worry may point toward potential cognitive or attentional factors that mediate homeostatic function relative to stress responding.

Stress

Allostasis is the ability for an organism to achieve stability through change (Sterling & Eyer, 1988; Sterling, 2004). Allostasis utilizes the hypothalamic-pituitary-adrenal (HPA) axis, autonomic, cardiovascular metabolic, and immune systems to protect the body by responding to internal and external stress (Sterling & Eyer, 1988). Both acute stress and chronic stress have been shown to contribute to long-term detrimental health consequences including cardiovascular disease or poor immune function (Dimsdale, 2008; Schneiderman, 2005). In the body's attempt to accommodate stress through adaptation, abnormal over- or under-activity of allostatic systems may develop (McEwen & Stellar, 1993). More specifically, when the brain perceives an experience as stressful,

physiological and behavioral responses are activated in order to accommodate the stress through adaptation (McEwen & Stellar, 1993). However, over time, the accumulation of the body's attempts to achieve allostasis, or allostatic load, can adversely affect organ systems through over- or under-activation of neural, endocrine, and immune functions and result in disease (McEwen & Stellar, 1993).

For example, Brosschot and Thayer (1998) proposed a model linking hostility and cardiovascular disease. These researchers highlighted that the unfinished action tendencies of provoked anger, or postponed angry behavior, more often results in transient angry cognitions that prolong the physiological effects of anger in the body (e.g., low vagal tone) (Brosschot & Thayer, 1998). Brosschot and Thayer (1998) emphasized the role of the speed of cardiovascular recovery, as opposed to reactivity, in the risk for developing cardiovascular diseases (e.g., atherosclerosis; hypertension). More specifically, better heart rate regulation or flexibility, via the vagus, reduces the amount of time it takes for the heart to recover from increased activation through increased parasympathetic activity. Low parasympathetic activity, or low vagal tone, is associated with a hypervigilant and inflexible state, both increasing attention to external threat and decreasing internal "noise" or awareness of bodily states (Porges et al., 1994; Porges, 1992). Whereas low vagal tone may be initially adaptive within high stress or threatening contexts, low vagal tone also can lead to chronic pathogenic states whereby heart rate recovery decreases, resulting in reductions in heart rate variability, reactivity, sensitivity, and blood pressure regulation (Amarena & Julius, 1995).

The HPA-axis is activated following high intensity acute or chronic durations of stress (Herman et al., 2003). The physiological response to stress is largely mediated by

increases in corticotrophin releasing hormone (CRH), a peptide, which is released by the periventricular nucleus (PVN) of the hypothalamus (Ulrich-Lai and Herman, 2009). Thus, the anterior pituitary gland releases adrenocorticotropic hormone (ATCH), which activates the release of cortisol within the adrenal gland. Cortisol follows a circadian rhythm (Ulrich-Lai and Herman, 2009), in which concentrations tend to be disproportionately high in the morning time, upon awakening, compared to mid-afternoon and later evening times (Weitzman, Clark, & Tellegen, 1971). This is due to cortisol's ability to bind to glucocorticoid receptors, with less receptor sensitivity following a diurnal decline. These changes in levels of cortisol from morning until evening are called diurnal cortisol slopes. The amount and accessibility of glucocorticoid receptors modulate overall HPA-axis function (Bamberger, Schulte, & Chrousos, 1996; Sapolsky, Romero, & Munck, 2000), with flatter diurnal slopes implicated in psychosocial stress response and poor mental and physical health outcomes (Adam et al., 2010). Altered activity of the HPA-axis, as a result of chronic stress, is widely considered as an important factor in the etiology of psychopathology (Miller et al., 2007). Specifically, persistent non-specific stress may compromise behavioral and physical adaptive responses and result in pathological changes of the endocrine and immune system (Yehuda et al., 1993; Leonard and Myint, 2009)

Features of GAD (e.g., worry), specifically, may lead to chronic activation of the HPA-axis, resulting in an increase in the sensitivity or number of glucocorticoid receptors, ultimately affecting suppression of HPA-axis function (Henry, 1992; Arborelius et al., 1999; Faravelli et al., 2012). However, the available evidence on cortisol stress responding in GAD has yielded inconsistent results. Several studies

support an upregulation of HPA-axis activity, or hyper-cortisolism, in individuals with GAD (Hood et al., 2011; Mantella et al., 2008; Terlevic et al., 2013; Tafet et al., 2005). For example, Mantella and colleagues (2008) examined alterations in HPA-axis functioning in elderly individuals with GAD compared to healthy controls by evaluating alterations in free salivary cortisol levels. Seventy-one individuals over 60 years of age and 40 non-anxious controls completed several clinical measures and provided six saliva samples over a two-day period. Results revealed individuals with GAD had increased cortisol concentration levels compared to non-anxious controls. Furthermore, whereas both groups exhibited a diurnal curve with peak cortisol levels occurring shortly after wakening, morning and evening cortisol concentrations were significantly larger in GAD subjects (Mantella et al., 2008). Taken together, these results suggest older adults with GAD experience early morning elevations in cortisol concentrations. Furthermore, these findings also suggest that elevated cortisol levels reflect an anxious affective state that is not dependent on the duration or age of onset of the disorder. However, other researchers have shown decreased cortisol levels in elderly samples (Hek et al., 2012; Heaney, Phillips, & Carroll, 2010; Chaudiue et al., 2008).

Hoehn-Saric and colleagues (1991) evaluated plasma cortisol levels in female GAD patients, with a mean age of 36, compared to non-anxious controls. Specifically, they measured cortisol levels before and after a stress-based laboratory task, before and after a single dose of a benzodiazepine (e.g., Valium) or placebo, and before and after 6 weeks of treatment with Valium or placebo. Results found significant differences between baseline cortisol levels in week 0 and week 6, as well as between baseline cortisol levels and administration of the laboratory stress task at week 0 and week 6.

Cortisol exhibited diurnal changes comparable in magnitude across baseline, week 0, week 6, and after the lab stressor. However, there were no significant differences in plasma cortisol levels between GAD female patients and normal controls, in contrast to results reported above. Other researchers also have shown no differences in diurnal profiles for cortisol levels in adults (Rosenbaum et al., 1983; Chaudieu et al., 2008; Steudte et al., 2011).

Some researchers suggest that the initial chronic HPA-axis activation, or hyper-corticolism in GAD, may actually lead to *hypo*-corticolism over time (Steudte et al., 2011; Hellhammer and Wade, 1993). Furthermore, hypo-cortisolism may account for lowered cortisol levels during baseline, reduced adrenocortisol reactivity to stressors, and an increased negative feedback sensitivity of the HPA-axis (Heim et al., 2000; Steudte et al., 2011). Increased negative feedback sensitivity of the HPA-axis may contribute to heightened anxiety sensitivity and enhanced worry (Hellhammer and Wade, 1993; Heim et al., 2000), only to perpetuate the cycle of physiological dysregulation. Whereas, the literature points to some form of abnormality related to cortisol secretion in GAD, the specific nature of these changes are largely still unknown.

Attentional Control

Research has linked cortisol regulation to neural structures including the amygdala, hippocampus, prefrontal cortex (PFC) and anterior cingulate cortex (ACC), with a prominent negative association between PFC activation and cortisol secretion (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009). Therefore, cortisol concentration levels may be linked to difficulties in response inhibition, cognitive flexibility, and attentional regulation, which are necessary for health and optimal

performance in complex environments (Thayer & Lane, 2009; Thayer et al., 2009). Extant literature has shown that the PFC is especially important for regulating aversive emotion and conflict through top-down attentional control (Bishop et al., 2004; Ohman, 2005). Attentional control is an individual's ability to voluntarily shift and disengage attention and utilize cognitive control to selectively inhibit the processing of extraneous or irrelevant stimuli (Derryberry & Reed, 2002; Fox et al., 2002; Friedman & Miyake, 2004). Reliable individual differences have emerged on measures of attentional control, thus suggesting it may be conceptualized as a trait capturing the control of information processing (Derryberry & Reed, 2002). Research also suggests attentional control may be a mediating mechanism for facilitated attention, difficulties disengaging from threatening stimuli, and attentional avoidance (Cisler & Koster, 2010; Cisler et al., 2009; Koster et al., 2005; Fox, Russo, Bowles, & Dutton, 2001; Mogg et al., 2004). Impaired attentional control has been implicated in poor emotion regulation and linked to pathological affective conditions including trait anxiety (Bishop, 2009) and excessive worry (Hirsch & Mathews, 2012). Attention Control Theory (ACT; Eysenck, Derakshan, Santos & Calvo, 2007) hypothesizes that anxiety is associated with deficits in quick and accurate cognitive processing. Specifically, ACT posits that anxiety affects an individual's ability to inhibit task-irrelevant information, shift attention flexibly, and update working memory. A wide body of literature suggests that attentional deficits, relative to cognitive control, are a result of anxiety (Derakshan, Smyth, & Eysenck, 2009; Derakshan & Eysenck, 2009; Ansari & Derkashan, 2011a, 2011b; Bishop et al., 2004). However, an inability to integrate information from both inside and outside of the body may contribute to

maladaptive regulation of cognition, emotion, perception, action, and physiological responsivity.

As mentioned above, the Neurovisceral Integration Perspective (Thayer et al., 2009; Thayer & Lane, 2009) suggests that top-down cortical control influences autonomic nervous system activity. Heart rate variability (HRV) is the fluctuation in the time intervals between adjacent heartbeats. HRV is proposed to index a system that integrates perceptual, motor, memory, and interoceptive information in order to facilitate successful adaptations to physiological and environmental changes (Thayer, Ahs, Fredrikson, Sollers, & Wagner, 2012). HRV is comprised of several measures relative to sympathetic and parasympathetic activity. By breaking down HRV into frequency components, it is possible to isolate a specific range of frequencies that are thought to reflect parasympathetic nervous activity (Shaffer and Ginsberg, 2017). These range of frequencies are known as high frequency HRV (HF-HRV), which is associated with respiratory sinus arrhythmia (RSA). In this model, higher levels of resting HRV (i.e., greater vagal tone) at rest are considered an exhibition of prefrontal inhibitory control over subcortical circuits that allow an individual to respond to environmental challenges in a controlled and adaptive manner (Thayer & Lane, 2009; Thayer et al., 2012; Hansen, Johnsen, and Thayer, 2003). In contrast, lower HRV at rest is associated with limited PFC activity and poor self-regulatory functioning (Thayer & Lane, 2000; Thayer et al., 2009). Several studies have found associations between individual differences in HRV and performance on tasks that require attentional control (Park, Van Bavel, Vasey, & Thayer, 2012; Park, Vasey, Van Bavel, & Thayer, 2013), motor-response inhibition (Krypotos, Jahfari, van Ast, Kindt, & Forstmann, 2011; Hovland et al., 2012), and

working memory (Hansen, Johnsen, & Thayer, 2009; Hansen et al., 2003). Given that resting HRV has been shown to index important aspects of prefrontal neural function, it follows that individual differences in cognitive control may be a useful predictor of HRV. For this reason, examining the parasympathetic influence on the heart via HRV may provide an index of an individual's capacity to effectively function in a complex and challenging environment.

Worry is postulated to be a primary mechanism by which an individual maintains the physiological effects of a stressor through cognitive representation (Brosschot, Pieper, & Thayer, 2005; Brosschot, Gerin, & Thayer, 2006). A few studies have shown that homeostatic recovery after emotional distress was slowed as a result of repetitive negative thinking (Glynn et al., 2002; Gerin et al., 2006). Similarly, several studies have shown that state and trait worry are associated with increased physiological activation (see for review: Brosschot et al., 2006). Diminished attention control may confer risk for preservative worry by impairing an individual's ability to prevent or inhibit intrusive, unwanted thoughts (Brown et al., 1993). Thus, deficits in attentional control may facilitate excessive negative cognition by allowing unwanted thoughts to gain entry into working memory despite attempts to suppress them (Rosen & Engle, 1998). Recent research suggests that low attentional control may serve as a mechanism for relationships between repetitive negative thinking and associated symptomology (Mills et al., 2016). Poor attentional control may increase difficulty for an individual to disengage from negative internal stimuli (e.g., worries; Hirsch, Clark, & Mathews, 2006; Hirsch & Mathews, 2012) and affect an individual's ability to deploy complex and effortful regulatory processes that directly influence changes in stress responding. If worry

prolongs the cognitive representation of stress, then properties of attentional control may make it a likely candidate as a mediator of physiological activity related to stress and anxiety.

Robinson, Ode, and Hilmert (2014) evaluated the effect of attentional control on individual differences in cortisol reactivity. Fifty participants completed the TSST (Kirschbaum, Pirke, & Hellhammer, 1993), provided basal and post-stressor salivary cortisol samples, and reported their daily affective states and experiences for 15 consecutive days. Multi-level analysis revealed the higher levels of daily mindfulness supported higher levels of attentional control at low levels of cortisol reactivity (Robinson et al., 2014). For individuals with low cortisol reactivity, daily attentional control was an inverse predictor of self-reported awareness of making errors or mistakes throughout the day; whereas high cortisol reactors appeared to be less effective in using attentional control to mitigate their reactivity to errors and mistakes (Robinson et al., 2014). At low levels of cortisol reactivity, attentional control was inversely related to worry, indicating that attentional control appeared to be less effective as cortisol reactivity increased (Robinson et al., 2014). Baseline levels of cortisol did not interact with attentional control to predict daily outcome measures, which potentially implicates processes involving reactivity of the HPA-axis (Robinson et al., 2014). Taken together, these results suggest that higher levels of attentional control are associated with lower levels of worry, error reactivity, and conflicting thoughts. Furthermore, with respect to individual differences, these inverse relationships were present at lower levels of cortisol reactivity and greatly attenuated at higher levels of reactivity. Therefore, we might expect

attentional control to directly influence the relationship between worry and cortisol reactivity during a stressor.

Pilgrim, Marin, and Lupien (2010) evaluated whether attentional engagement toward or disengagement away from social stress-related stimuli during a resting state could predict cortisol responsivity produced after a stress task. Twenty-five healthy subjects completed a selective attention task, followed by the Trier Social Stress Tests (TSST). Attention index scores were calculated using mean reaction times (RTs) of responses to a spatial orienting task using stressful, neutral, and positive social word stimuli that were presented on either the same side as the target (valid trial) or the opposite side of the target (invalid trial). For attentional disengagement scores, RTs for invalid neutral trials (i.e., where cue and target appeared on opposite sides of the display), were subtracted from invalid stress or invalid positive trials, respectively. Attentional engagement scores, RTs for valid neutral trials were computed in the same manner using valid trials (i.e., where cue and target appeared on the same side of the computer screen). Results revealed that individuals with larger attentional engagement difference scores, who slowly engaged in stress-related stimuli, exhibited smaller cortisol response after a stressor compared to those who engaged in stress-related stimuli at a faster rate (Pilgrim et al., 2010). These results suggest that distinct aspects of attentional biases toward threat-related stimuli may have an impact on cortisol stress response and HPA-axis reactivity to stressors. Moreover, maladaptive attentional patterns appear to directly impact cortisol responding. However, reduced cortisol responding in individuals slower to engage in threatening stimuli could either be a healthy, adaptive response, or conversely be indicative of the deleterious effects caused by allostatic load. Blunted cortisol effects in

response to acute stressors could reflect dysregulated HPA-axis functioning (McEwin, 1998).

Pilgrim, Ellenbogen, and Paquin (2014) evaluated the effects of attentional training on stress responding in healthy participants. Specifically, 56 healthy participants were randomly assigned to three attentional training task conditions. Upon attentional training, participants underwent the TSST. Self-report mood measures and saliva were collected at multiple time points, with cortisol and salivary alpha-amylase extracted from the saliva. Interestingly, participants in the attention training task conditions, compared to the control condition, exhibited greater cortisol stress response to the TSST (Pilgrim et al, 2014). Furthermore, those with high attentional control exhibited a more robust cortisol response to the TSST compared to those with low attentional control (Pilgrim et al, 2014). Taken together, these results provide evidence that attentional training, especially for those with high attentional control, experience greater cortisol stress responsivity post stressor. These results seem paradoxical in nature. We would expect individuals with lower attentional control to exhibit greater cortisol responses, comparatively. It could be that individual differences in attention control, due to a stronger or weaker ability to disengage attention from threat, are linked to a greater or lessened ability to respond to a stressor with effortful regulation.

Richey and colleagues (2016) evaluated whether attention control moderated or mediated the effects of a stressor and whether attention control was predictive of state-like fear over an extended period of time between basal and stress measure points. In Study 1, 219 participants were administered the Attention Control Scale (ACS; Derryberry & Reed, 2002) and the State-Trait Anxiety Inventory (STAI; Spielberger,

Gorsuch & Lushene, 1970) at Time 1 (T1: baseline) and asked to complete the Worry-Emotionality Scale-Revised (WES-R; Morris & Liebert, 1970), a self-report measure of subjective test anxiety, immediately before a college examination 3 weeks after T1 baseline measures (T2: stressor). Results from Study 1 indicated the ACS did not moderate the effect of trait anxiety on fearful responding; instead, attention control mediated the effect of trait anxiety on fearful responding to the test stressor. In Study 2, 217 participants completed the ACS and STAI-Trait measures at T1 and were asked to complete the Test Anxiety Scale (TAS; Spielberger, Anton, & Bedell, 1976), and the Achievement Anxiety Test (AAT; Alpert & Haber, 1960) immediately before a college examination 3 weeks later. Results for Study 2 replicated the findings of Study 1 and supported a mediational role for attention control in the relationship between trait anxiety and stress response. Furthermore, results pointed toward debilitating facets of attentional control, compared to facilitative facets, on the basis of the mediation model. Taken together, these results suggest a role for attentional control in mediating the relationship between trait anxiety and state anxiety. However, these researchers posited that due to the stress response measures being administered *prior* to the college examination, the mediating effect of attention control may be more of an effect on anticipatory anxiety, rather than a direct effect on the experience of state anxiety.

In a study that evaluated the effect of attention control on trait anxiety and response to inhaling a CO2-enriched gas mixture, attentional control moderated the relationship between trait anxiety and stress response (Richey et al., 2012). More specifically, individuals high in trait anxiety and high in attentional control reported less fear in response to the CO2 stressor than individuals high in trait anxiety with low

attentional control. Self-reported stress response was completed *after* the stressor as opposed to before the stressor (Richey et al., 2012). Taken together, these results suggest that attentional control may have moderating or mediating effects on stress responding, depending on temporal aspects of when exactly stress responses are measured and the type of anxiety experienced. It also is important to note that attentional control was measured via self-report, as opposed to behavioral indices. Therefore, it may be useful to examine the effects of trait and behavioral measures of attentional control on stress responding before, during, and after a stressor.

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APPENDICES

Table 1. Correlations among independent variable, dependent variable, mediator, and covariates

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Cortisol (AUCi)	___										
2. Worry (PSWQ)	-0.159*	___									
3. Attention Control (ACS)	0.236*	-0.492*	___								
4. Sex	-0.161*	0.428*	-0.19*	___							
5. Digestion	0.137	-0.056	0.099	0.008	___						
6. Caffiene	0.156*	0.039	-0.054	-0.009	0.186*	___					
7. Physical Activity	0.149	0.003	-0.104	0.168*	0.127	-0.035	___				
8. Nicotine	-0.028	-0.069	0.164*	0.065	0.111	0.479*	0.003	___			
9. BMI	0.003	0.074	-0.011	-0.088	-0.119	0.181*	-0.022	0.142	___		
10. Illnes	0.059	-0.085	0.011	-0.097	0.133	0.071	-0.121	0.028	-0.13	___	
11. Medications	0.151	-0.137	0.164	-0.169*	0.195*	-0.027	0.008	-0.047	-0.249*	0.31*	___

*p < .05

Table 2. Summary of mediation results for attentional control and cortisol stress response with trait worry as independent variable.

DV	M	Effect of IV on M	Effect of M on DV	Direct Effect	Indirect Effect	Total Effect
AUCi	ACS	-0.24*	0.28*	-0.06	-0.067	-0.058

*$p < .05$

Table 3. Acute worry levels predicted from cortisol stress response and attentional control.

Predictor	β	*p*	95% CI
AUCi	-0.165	0.098	-.362, .032
ACS*	-0.349	0.001	-.542, -.158
AUCi x ACS*	0.186	0.028	.021, .652

*$p < .05$

Table 4. Conditional effects of cortisol stress response at three levels of attentional control.

Attentional Control (ACS)	β	*p*	95% CI
One SD below Mean*	-0.352	0.002	-.576, -.129
At the Mean	-0.165	0.098	-.362, .032
One SD above Mean	0.022	0.879	-.265, .309

*$p < .05$

Figure 1. Predicted mediation model.

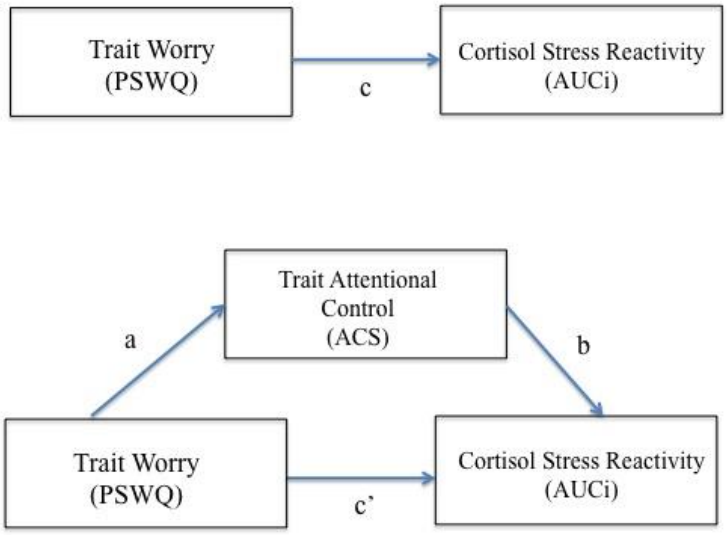


Figure 2. Subjective units of distress scale measures of worry (S1-S5).

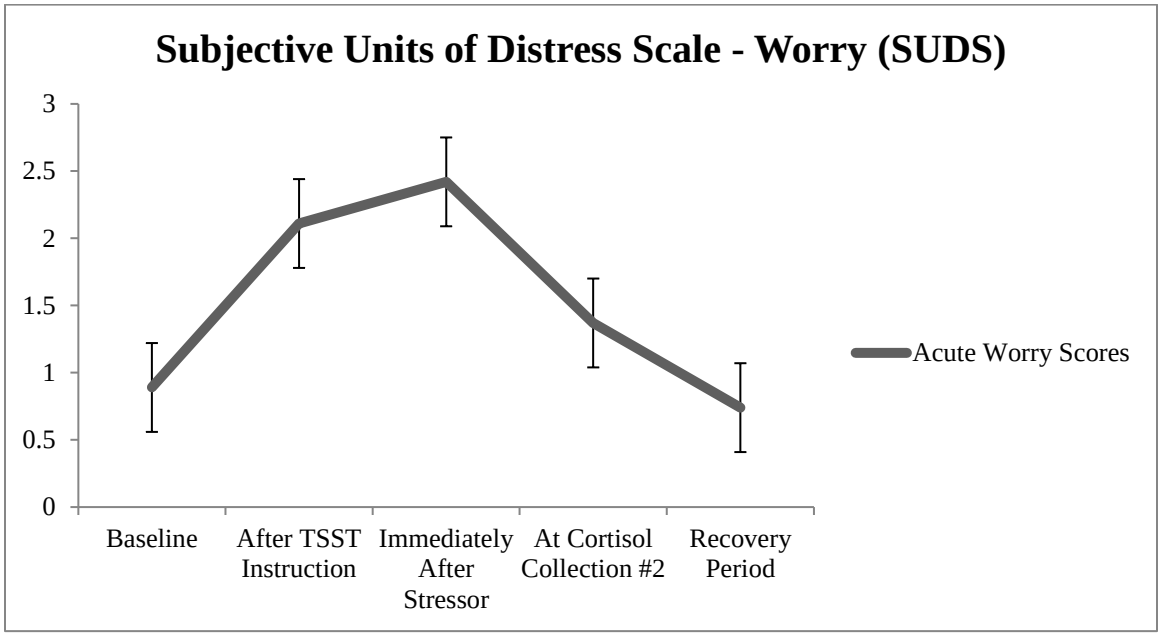


Figure 3. Outcome mediation model.

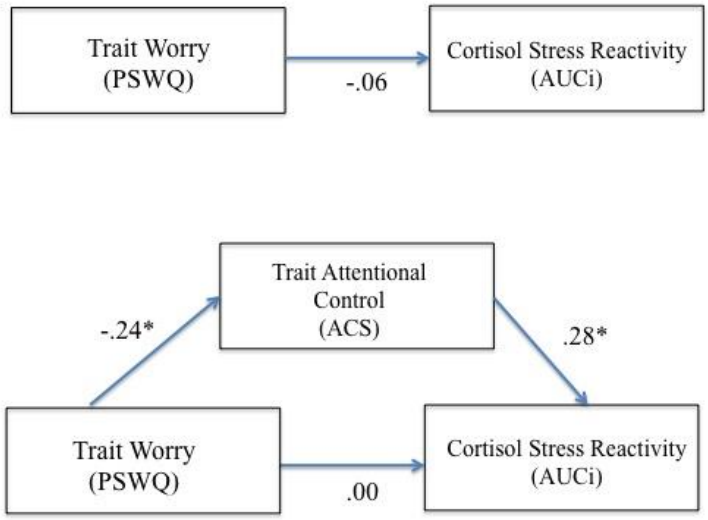
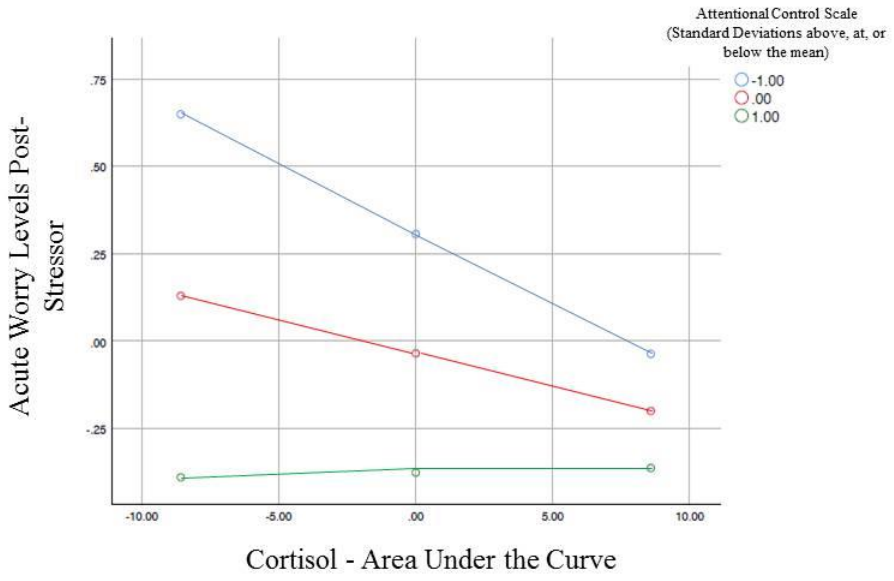


Figure 4. Conditional effects of cortisol stress sensitivity at three levels of attentional control.



Oklahoma State University Institutional Review Board

Date: 11/06/2018
Application Number: AS-18-135
Proposal Title: Worry, Attentional Control, & Stress Response

Principal Investigator: Kristen Frosio
Co-Investigator(s): Faculty Adviser: Demond Grant
Project Coordinator:
Research Assistant(s):

Processed as: Expedited
Status Recommended by Reviewer(s): Approved
Approval Date: 10/29/2018
Expiration Date: 10/28/2019

The IRB application referenced above has been approved. It is the judgment of the reviewers that the rights and welfare of individuals who may be asked to participate in this study will be respected, and that the research will be conducted in a manner consistent with the IRB requirements as outlined in section 45 CFR 46.

The final versions of any recruitment, consent and assent documents bearing the IRB approval stamp are available for download from IRBManager. These are the versions that must be used during the study.

As Principal Investigator, it is your responsibility to do the following:
1. Conduct this study exactly as it has been approved. Any modifications to the research protocol must be approved by the IRB. Protocol modifications requiring approval may include changes to the title, PI, adviser, other research personnel, funding status or sponsor, subject population composition or size, recruitment, inclusion/exclusion criteria, research site, research procedures and consent/assent process or forms.
2. Submit a request for continuation if the study extends beyond the approval period. This continuation must receive IRB review and approval before the research can continue.
3. Report any unanticipated and/or adverse events to the IRB Office promptly.
4. Notify the IRB office when your research project is complete or when you are no longer affiliated with Oklahoma State University.

Please note that approved protocols are subject to monitoring by the IRB and that the IRB office has the authority to inspect research records associated with this protocol at any time. If you have questions about the IRB procedures or need any assistance from the Board, please contact the IRB Office at 223 Scott Hall (phone: 405-744-3377, irb@okstate.edu).

Sincerely, Oklahoma State University IRB

VITA

Kristen Elizabeth Frosio

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