Heart Rate Variability at Rest and During Worry in Chronic Worriers

Dissertation

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of The Ohio State University

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The Ohio State University

2019

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Abstract

Pathological worry has been associated with both blunted and heightened autonomic arousal (AA). The cognitive control model posits that individual differences in effortful control (EC) can account for heterogeneity in level of AA among worriers. It suggests that EC conveys ability to effortfully shift attention to a verbal mode of processing, which, unlike imaginal processing, is associated with reduced AA. Despite the widely held view that pathological worry and generalized anxiety disorder (GAD) are characterized by low EC, there are actually substantial individuals differences in such cognitive control capacity.

Initial tests of the cognitive control model have yielded promising results, but they have so far primarily relied on measures that assess worry over relatively longer durations of time (e.g., past six months). The current study sought to extend prior findings by testing the model in the context of a laboratory worry induction in a sample of undergraduate college students (N = 174). It was expected that individual differences in change in percentage of thoughts during worry and change in HRV during worry would be each be accounted for by initial cognitive control capacity. While the results show that both percentage of thoughts and phasic HRV declined on average over time, statistical tests of moderation were not significant. Potential explanations for null findings are discussed.

Acknowledgments

I am full of gratitude for my advisor, Michael Vasey, Ph.D. I am thankful for his support, encouragement, occasional prodding, and for helping me find my way to completing this milestone. But above all, I am thankful that he introduced me to the study of anxiety, a topic near and dear to my heart. I would also like to thank Drs. Joseph DeCola and Lawrence Needleman for furthering my understanding of treatment of anxiety-related disorders. Finally, I would like to thank Drs. Robert Cudeck and Mark Pitt for their thoughtful feedback on this project.

I dedicate this dissertation to my wife, who encouraged me to return to college as an adult, helped prepare me for graduate school, and who is constantly shaping my understanding of psychopathology and empirically supported treatments. She truly is a life optimizer.

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Fields of Study

Major Field: Psychology

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Chapter 1: Introduction

Excessive, uncontrollable worry is common among the anxiety disorders, but it is the hallmark of generalized anxiety disorder (GAD; American Psychiatric Association [APA], 2013). Modern models of such pathological worry have posited that the relation between worry and autonomic arousal (AA) plays a role in the maintenance of worry. For example the Cognitive Avoidance (CognAv) Model (Borkovec, Alcaine, & Behar, 2004) argues that worry becomes pathological in part because it blunts the heightened AA that would otherwise be activated when the worrier experiences intrusive images of possible future threats. It does so because it involves a shift to a verbal mode of processing such future threats, which limits activation of AA (Lang, 1985). Thus, this model postulates that worry is negatively reinforced because it reduces AA. The CognAv Model has received broad research support, including several correlational and experimental studies showing that worry is indeed associated with blunted AA reactivity to distressing stimuli (e.g., Borkovec & Hu, 1990; Vrana, Cuthbert, & Lang, 1986). However, contrary to the CognAv Model, the Contrast Avoidance (ContrAv) Model (Newman & Llera, 2011) characterizes the link between worry and AA in the opposite way. Specifically, the ContrAv model argues that rather than suppressing AA, worry serves to generate and sustain *heightened* AA (and negative emotionality more broadly), which serves to limit the magnitude of unpredictable spikes in AA (and negative emotion) that would otherwise occur if a threatening event were to arise while the worrier was in a euthymic mood state. Indeed, a number of studies have found that worry is indeed associated with heightened AA (e.g., Brosschot, Dijk, &

Thayer, 2007; Knepp & Friedman, 2008; Wetherell & Gatz, 2005). The fact that considerable evidence links excessive and uncontrollable worry to both blunted and heightened AA presents a paradox that neither the CognAv nor the ContrAv Model alone can explain. The current study seeks to advance understanding of why worry is linked to both blunted and heightened levels of AA.

Although DSM-III-R included AA symptoms among the possible symptoms associated with GAD, they were dropped from the definition in DSM-IV based on evidence that many who reported excessive, uncontrollable worry endorsed relatively few AA symptoms (Marten et al., 1993). The de-emphasis of AA symptoms in DSM-IV, which has been retained in DSM-5, is consistent with and reflects the influence of the CognAv model. However, that position has become untenable with increasing evidence that AA symptoms are often elevated in worry and GAD. Indeed, the heterogeneity in level of AA among worriers has been broadly documented at several levels of analysis. At the symptom level, results from several samples suggest that the relation between level of AA symptoms and worry is low or non-existent, despite adequate variability to detect such correlations. In some samples, individuals with GAD report no greater level of AA on average than non-worriers. For example, Leyfer, Ruberg, & Woodruff-borden (2006) found GAD patients were not significantly different from non-anxious controls on the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988), a measure that largely taps AA symptoms. In addition, GAD status/severity and level of worry are often uncorrelated with scores on measures of AA symptoms (e.g., Brown, Chorpita, & Barlow, 1998; Brown & Mcniff, 2009). On the other hand, a similar number of studies have found that worry severity is related to

heightened AA symptoms on average. For example, contrary to Leyfer et al. (2006), Wetherell & Gatz (2005) found that BAI scores among older adults were significantly higher on average for individuals with GAD than non-anxious controls. In addition, GAD is frequently comorbid with panic attacks and panic disorder (Brown & Barlow, 1992), which are predominantly characterized by exaggerated levels of AA. In a large, multi-site study, 48% of those with GAD concurrently met DSM-III-R criteria for panic disorder (Goisman, Goldenberg, Vasile, & Keller, 1995). Moreover, a large epidemiological study found that GAD status prospectively predicts 1-year incidence of panic disorder and vice-versa. Overall, it is thus clear that there is substantial heterogeneity in self-reported AA among worriers and those with GAD; worry severity is positively associated with AA symptoms for some individuals, yet AA symptoms are blunted in others.

Mirroring the heterogeneity in subjectively measured AA, results from several studies have also revealed substantial heterogeneity in objectively measured AA among worriers. Consistent with the CognAv Model, worry has been shown to be unrelated to several biomarkers of AA. For example, some studies have found no difference between worriers and controls in baseline heart rate (e.g., Fisher & Newman, 2013; Pittig et al., 2013), skin conductance (e.g., Delgado et al., 2009), and salivary alpha amylase (e.g., Fisher, Granger, & Newman, 2010). However, these results have not always replicated. Indeed, consistent with the ContrAv Model, other studies have found that worry is positively associated with such biomarkers, including higher resting heart rate (e.g., Brosschot, Dijk, & Thayer, 2007), greater galvanic skin conductance (Kirschner, Hilbert, Hoyer, Lueken, & Beesdo-Baum, 2016; Stapinski, Abbott, & Rapee, 2010) and increased salivary alpha

amylase (e.g., Fisher & Newman, 2013). Taken together, extant evidence suggests that worry is associated with heightened levels of objectively measured AA among some worriers, and blunted levels of AA among others.

For those who experience blunted AA during worry, the Cognitive Avoidance Model offers a compelling explanation. As noted above, this model suggests that worry is negatively reinforced due to its ability to attenuate elevations in levels of AA when individuals cognitively process threat information. A number of experiments support such a theory. In a seminal study (Borkovec & Hu, 1990), participants who feared public speaking were randomly assigned to engage in relaxed, neutral, or worrisome thinking prior to imagining a scene in which they were giving a speech. Compared with the other two conditions, the worry group experienced negligible increases in heart rate in response to the phobic imagery. Peasley-Miklus and Vrana (2000) reported similar findings: Participants exhibited a more exaggerated cardiac response to fearful imagery related to public speaking after a relaxation period than after a worry period. Moreover, heart rate continued increasing across the fearful imagery period when it followed relaxation, but remained flat when it followed worry, suggesting that worry has a sustained dampening effect on AA. These results have been replicated by Borkovec, Lyonfields, Wiser, and Deihl (1993), as well as Fisher & Newman (2013). Taken together, it is clear that worry appears to inhibit cardiovascular response to phobic imagery in some individuals, which may serve to reinforce worry as a maladaptive strategy as posited by the Cognitive Avoidance Model.

A major limitation of the Cognitive Avoidance Model is that it cannot account for the subset of worriers who experience heightened levels of AA during worry. The Contrast

Avoidance Model provides a potential explanation for such worriers. It characterizes worry as an avoidant strategy used to circumvent sudden, aversive contrasts between emotional states. When individuals in a relaxed or euthymic state perceive an unanticipated threat in the environment, they experience a shift to an anxious state in preparation for addressing the threat. According to the model, some individuals (e.g., GAD patients) are uniquely sensitive to such negative emotional contrasts and find them to be especially distressing. The Contrast Avoidance Model suggests pathological worriers use worry to elicit and *maintain* heightened AA and negative emotionality in order to reduce the magnitude of jarring shifts in emotion. The model has logical appeal; if AA is already elevated when a threat is perceived, there is little room for additional physiological and emotional escalation. Thus, whereas the Cognitive Avoidance Model suggests that worry facilitates suppression of AA, the Contrast Avoidance Model posits that worry serves to heighten it, thereby restricting the range of emotional and physiological contrasts. A number of experiments support casting worry in this opposite role. For example, in an ambulatory study (Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004), participants wore a heart monitor during their normal daily tasks and responded to periodic prompts to assess worry, stress, and mood. Worry was related to heightened heart rate during worry episodes and for an average of two hours following such episodes. Furthermore, aversion to emotional contrasts may indeed play an important role in maintaining worry. For example, Crouch, Lewis, Erickson, & Newman (2017) found that those with higher GAD symptoms were more likely to rate emotional contrasts during a stressful event as the worst part of the experience, though it should be noted that the study focused exclusively

on measures of negative emotionality and not AA.

Overall, both models are well supported by empirical evidence, creating a paradox that neither model alone can resolve. To resolve this paradox, Vasey, Chriki, and Toh (2016) recently proposed a model positing that heterogeneity in AA level among worriers reflects a previously unconsidered moderator. Specifically, they argue that this heterogeneity reflects the influence of individual differences among worriers in cognitive control capacity. Specifically, the Cognitive Control Model postulates that the shift to a verbal mode of threat processing posited by the CognAv Model requires adequate cognitive control capacity. In the absence of sufficient top-down control resources to accomplish and maintain this shift, a worrier should process threat possibilities primarily as images, which are potent activators of AA (e.g., Shearer & Tucker, 1981; Vrana et al., 1986). However, worriers with sufficient cognitive control should be able to suppress such images and shift to a verbal mode of processing, which should tend not to activate AA.

Though it is widely believed that low cognitive control capacity is intrinsic to worry/GAD symptoms (Eysenck, Derakshan, Santos, & Calvo, 2007), there are actually considerable individual differences. One way to capture cognitive control capacity is by using self-report measures of effortful control (EC). EC is viewed as a higher order, temperamental construct encompassing a range of top-down control processes. It includes the ability to flexibly focus and sustain attention (i.e., attentional control [AC]), the ability to override prepotent, goal-irrelevant behaviors (i.e., inhibitory control), and the ability to recruit and coordinate cognitive resources to achieve a goal (i.e., activation control). On one hand, deficits in cognitive control capacity, such as diminished AC, might seem obvious

given that worriers and patients with GAD describe their worry as intrusive and uncontrollable. Consistent with that view, some evidence suggests that worry is associated with poor cognitive control (e.g., Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014; Stefanopoulou, Hirsch, Hayes, & Adlam, 2014), and several studies have found that, compared with non-anxious control, worriers and patients with GAD report lower AC (e.g., Armstrong, Zald, & Olatunji, 2011; Borkovec, Robinson, & Pruzinsky, 1983; Moradi, Fata, Abhari, & Abbasi, 2014) and lower EC more broadly (e.g., Vasey et al., 2017) In addition, scores on measures of cognitive control capacity are often negatively correlated with GAD symptoms (e.g., Armstrong et al., 2011; Olatunji, Ciesielski, Armstrong, Zhao, & Zald, 2011; Vasey & Toh, 2017; Toh & Vasey, 2017). On the other hand, it is important to note that such deficits reflect group differences *on average.* In fact, there is often substantial variability in cognitive control capacity among worriers within samples. Moreover, results reflecting deficits have not always replicated. For example, some studies have found non-significant associations between worry/GAD and conscientiousness (e.g., Bienvenu et al., 2004), a personality trait correlated with cognitive control. Indeed, one study found a weak, *positive* association between worry and conscientiousness when controlling for negative affect (Rosellini & Brown, 2011).

Heterogeneity in cognitive control capacity among worriers is not limited to self-report; scores on performance-based tasks and neuroimaging findings have also yielded conflicting findings. For example, on one hand, Vytal and colleagues (2016) found that during threat (e.g., completing a task while at risk for electric shock), GAD patents showed deficits on an N-back working memory task. Balderston et al., (2017) also reported that

GAD patients exhibited poorer accuracy and longer reaction times on an N-back task compared with controls. Consistent with the performance deficits, they also found that, relative to controls, patients had less task-related activation of the dorsal lateral prefrontal cortex (dlPFC). Other studies have shown reduced activation of the dorsal anterior cingulate cortex in GAD patients (dACC; e.g., Blair et al., 2012), as well as reduced frontal cortical thickness (Veronese et al., 2015). Each of these regions plays a major role in the distributed neural network underpinning cognitive control. On the other hand, however, Osinksy, Gebhardt, Alexander, & Hennig (2012) found no relation between trait anxiety and performance on a modified Stroop task. Moreover, in two separate studies, Yiend et al. (2015) found that patients with GAD were significantly faster at disengaging attention from angry and fearful faces than non-anxious controls. In addition, a recent, large population-based study in the Netherlands (N=82,360) examined executive function deficits among patients diagnosed with one or more anxiety disorders compared with healthy controls. They failed to find a link between GAD status and scores on the Ruff Figural Fluency Test, a performance based measure of executive function.

At the neural level, some evidence suggests that worry is moderately to strongly *positively* correlated with volume of several regions of the prefrontal cortex (PFC; Mohlman et al., 2009), and also positively correlated with connectivity between the amygdala and dlPFC (e.g., Etkin, Prater, Schatzberg, Menon, & Greicius, 2009), a region associated with cognitive control (e.g., Banich et al., 2000; MacDonald, Cohen, Stenger, & Carter, 2000). Connectivity between the midbrain areas like the amygdala and regions of the cortex is noteworthy insofar as it mediates top-down cognitive control. The Etkin et al. (2009) study

is especially noteworthy because, in keeping with the cognitive control model, they found that the strength of the connectivity between the amygdala and dlPFC was negatively associated with scores on the BAI among GAD patients. Overall, the extant body of evidence suggests that, pathological worry and GAD are characterized by heterogeneity in performance on cognitive control capacity tasks as well as task-dependent activation of related brain regions. Some studies show deficits, others do not.

Building on the evidence for heterogeneity in both levels of AA and cognitive control capacity among pathological worriers, the central thesis of the Cognitive Control Model is that the former can be accounted for by the latter. Specifically, to the extent a worrier possess sufficient cognitive control capacity to perform the subtle shift from imaginal to verbal processing of threat, postulated by the CognAv model, that worrier should experience blunted AA. That is, to insofar as AA is a maintaining factor for pathological worry, the manner in which it reinforces worry should operate differently among different worriers as a function of individual differences in cognitive control capacity. This model is an outgrowth of several important research findings. First, evidence suggests that worry content is indeed consciously processed primarily in one of two modes: verbal-linguistically or imaginally (Borkovec and Inz, 1990). Second, verbal-linguistic worry is associated with blunted AA reactivity, while imagery-based worry is associated with heightened AA reactivity (e.g., Vrana, Cuthbert, & Lang, 1986). For example, in one study (Shearer & Tucker, 1981), participants were asked to either inhibit or facilitate emotional responding to aversive stimuli. Though they were not advised on how to do so, participants spontaneously used imaginal processing when instructed to *facilitate*

emotional responding, and verbal-linguistic processing when instructed to *inhibit* it. Thus, it is possible that heterogeneity in AA can be accounted for by heterogeneity in amount of verbal thought during worry. Finally, these different modes require different amounts of cognitive resources. Indeed, shifting attention from imaginal to verbal processing is an effortful maneuver that consumes cognitive control resources (e.g., Mathews, 1990). Therefore, the Cognitive Control Model posits that only those worriers with adequate top-down control capacity are able to constrain worry to verbal thought, thereby blunting AA, which is in keeping with the Cognitive Avoidance Model. Individuals without sufficient capacity to maintain verbal thought are consigned to process threat imaginally, and, in turn, experience elevated AA consistent with the Contrast Avoidance Model.

Initial tests of the Cognitive Control Model have been promising. For example, Vasey, et al. (2017) found that the correlation between worry/GAD symptoms and self-reported AA was moderated by self-reported EC scores in a large sample of college students (N=1,323) who reported a wide range of levels of worry/GAD symptoms, EC, and AA. Supporting the basic interaction underpinning the model, they found that worry and GAD symptoms were more strongly positively associated with AA at lower versus higher levels of EC. The moderating effect of EC was even more pronounced in the subset of participants whose scores on worry measures were relatively high and resembled GAD samples. When examining this GAD analogue group alone, the relation between worry and AA was significantly positive at lower levels of EC, but flat and non-significant at higher levels. This basic interaction has been replicated in a sample of 926 participants (Toh and Vasey, 2017) and in a sample of 362 participants (Free, 2017). Thus, evidence derived

from three large studies suggests that the link between worry/GAD symptoms and self-reported level of AA can be accounted for by self-reported EC, as postulated by the Cognitive Control Model.

A significant limitation of these tests of the Cognitive Control Model is their exclusive focus on trait worry or GAD symptoms experienced over prolonged time intervals (e.g., the last six months). Pivotal to the model, however, is the relation between worry, cognitive control capacity, and level of AA *during* worry. The model posits that individuals with greater cognitive control capacity are spared from aversive AA symptoms because they misuse such capacity to effortfully sustain verbal worry. Recently, Toh (2018) addressed this gap in the literature by testing the model in the context of a laboratory worry induction among a large sample of college students (N=198). For the worry task, participants were asked to worry about a topic of current concern, during which they were periodically prompted to indicate percentage of verbal thoughts, percentage of imagery, and overall distress on a scale from 1 to 7. In addition, participants wore a device that recorded cardiac electrical signals. Toh successfully replicated the basic interaction underpinning the cognitive control model. That is, the relation between self-reported worry and AA symptoms was moderated by EC such that the relation was more positive when EC was low than when it was high. Moreover, worry/GAD symptoms interacted with measures of EC (both self-report and performance based) to predict percentage of verbal thoughts *during worry*. Consistent with the Cognitive Control Model, worry was more positively associated with percentage of verbal thought at lower versus higher levels of EC. While not all of Toh's hypotheses were fully supported by the results in this study, it

nevertheless provides compelling evidence that cognitive control capacity plays an important role in the mode of cognitive processing worriers use during worry.

Subsequent studies have shown that the basic interaction underpinning the Cognitive Control Model is not limited to self-report measures. For example, Free (2017) successfully replicated Vasey et al.'s (2017) findings using mean resting heart rate (HR) as an objectively measured index of AA. As expected, the association between self-reported worry and resting HR was more positive at lower versus higher levels of EC. Free also found support for the model using resting heart rate variability (HRV) as an objectively measured index of cognitive control capacity. Resting HRV was significantly negatively correlated with self-reports of AA symptoms at high levels of worry/GAD symptoms.

HRV refers to variation in the interval between individual heartbeats, which is influenced by the parasympathetic nervous system via the vagus nerve (Thayer & Lane, 2009). It is increasingly viewed as a biomarker of cognitive control capacity (Park & Thayer, 2014). Indeed, several studies have found that HRV is positively correlated with a number of indices of cognitive control capacity. For example, neuroimaging studies show that higher HRV is associated with greater activity in several areas of the PFC related to executive control (see Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). HRV is also related to performance on executive function tasks (e.g., Ramírez, Ortega, & Reyes Del Paso, 2015; Thayer, Hansen, Saus-rose, Psychol, & Johnsen, 2009). For example, Hansen, Johnsen, & Thayer (2003) found that compared with those with lower HRV, participants with higher HRV exhibited greater accuracy and faster response time on an N-back task. In addition, in a sample of individuals with panic disorder (Hovland et al., 2012), HRV was positively

correlated with scores on the Wisconsin Card Sorting Test and with scores on the inhibition task of a color Stroop test, but not color naming or word reading (which require relatively minimal cognitive resources). In addition, HRV is related to scores on self-report measures that tap elements of cognitive control capacity such as EC (e.g., Spangler & Friedman, 2015), and attentional control (AC; e.g., Balle et al., 2013; Healy, 2010). While these findings have not always replicated, it appears that resting HRV can indeed serve as an objective measure of cognitive control capacity (Thayer et al., 2009; Park and Thayer, 2014).

Suggesting that HRV can account for heterogeneity in level of AA during worry may seem at odds with the widely held view that pathological worry (and GAD) is characterized by blunted HRV. Contrary to that position, however, there are in fact substantial individual differences in HRV among worriers, both at rest and during worry. For example, on one hand, some studies have found that low resting HRV is associated with trait worry (Chalmers et al., 2016; Chalmers, Quintana, Abbott, & Kemp, 2014; Thayer, Friedman, & Borkovec, 1996) and state worry (Brosschot et al., 2007; Pieper, Brosschot, van der Leeden, & Thayer, 2010). Likewise, some studies have found that HRV is lower among patients with GAD than non-anxious controls (e.g., Lyonfields, Borkovec, & Thayer, 1995; Pittig et al., 2013). Indeed, Chalmers, Heathers, Abbott, Kemp, & Quintana (2016) have suggested low HRV may be a transdiagnostic biomarker of pathological worry, even in the absence of a diagnosable anxiety disorder. On the other hand, worry's relation to low HRV has not always emerged (e.g., Hammel et al., 2011; Kollai & Kollai, 1992). In fact, in one sample, worriers had significantly *higher* HRV at baseline and during a worry task than non-

anxious controls (Davis, Montgomery, & Wilson, 2002). Moreover, wide-ranging individual differences in resting HRV among worriers are apparent *within* samples as well. For example, Chalmers et al., (2016) suggestion that worry is associated with robust reductions in HRV was based on a correlation of r = -0.31. Less than 10% of the variance of scores on the Penn State Worry Questionnaire was accounted for by HRV. Consistent with that view, a recent meta-analysis shows that while HRV tends to be modestly lower on average among high trait worriers, there is neverthelss heterogeneity between sample (Holley, 2017). Indeed, some samples show no such difference in HRV. Moreover, Holley reported that many samples pointed to substantial overlap in HRV between GAD patients and non-anxious controls, consistent with the observation that some trait worriers have higher HRV than controls.

In addition, experimentally induced worry has been shown to lead to lower resting HRV in some samples (e.g., Gazzellini et al., 2016; Hammel et al., 2011; Hofmann et al., 2005; Levine, Fleming, Piedmont, Cain, & Chen, 2016; Ottaviani et al., 2014; Thayer et al., 1996) but not others (e.g., Davis et al., 2002; Delgado et al., 2009; Fisher & Newman, 2013; Knepp & Friedman, 2008; Lyonfields et al., 1995). Thus, it is plausible that these inconsistent findings have emerged due to an undetected moderator. Indeed, worry inductions may deplete HRV in some individuals and not others. For example, it is possible that high resting HRV reflects a baseline reserve capacity that does not begin to show depletion during typical laboratory worry inductions, which are often less than 10 minutes long. In that case, declines in HRV during relatively shorter worry episodes should only be detectable among worriers who do not have high resting HRV. On the other hand, it is also

possible that those with low resting HRV fail to show declines in HRV across a worry period because there is little room for further decline. Thus, among individuals with low baseline HRV, the relations between worry and HRV over time may be statistically obscured by a simple floor effect.

While several studies have shown that worry depletes HRV, most have only examined pre- to post-worry differences in HRV. No study has examined phasic changes in HRV during worry using repeated measures analyses. Parsing continuously measured HRV into multiple measurement occasions permits examination of an important aspect of the worry process and its consequences. Insofar as HRV reflects cognitive control capacity, do changes in HRV precede changes in percentage of verbal versus imaginal worry? Consistent with the Cognitive Control Model, it is expected that declines in HRV over time will be associated with declines in percentage of verbal worry over time. In other words, as worry depletes cognitive control capacity, participants are expected to be less successful at constraining worrisome thoughts to a verbal mode of processing.

Because Toh (2017) collected heart rate data in such a way as to permit estimation of HRV both at rest and during a worry induction, that study provided a suitable context to clarify the relation between HRV and level of AA during worry. Toh did not examine the relation between worry and HRV. However, the Cognitive Control Model would predict that phasic changes in EC, as measured by resting HRV, should be related to changes in modes of processing threat (e.g., verbally vs. imaginally). Specifically, it is expected that lower phasic HRV during worry will be related to less verbal-linguistic processing of threat. While all of the data in the current study were collected by Toh (2017), the current study

seeks to extend her findings by examining the role resting HRV plays in worry during a worry induction, as well as what happens to HRV during worry.

Overall, the current study (and the Cognitive Control Model more broadly) seeks to identify moderators that explain the heterogeneity among worriers in AA (hypotheses 1a and 1b), resting HRV (hypothesis 3), phasic HRV during worry (hypotheses 4 and 5) and verbal processing of threat (hypothesis 2). According to Andrew Hayes (2013), a variable is considerate a "moderator" if predicts the size, direction, or strength of an independent variable's (X) effect on a dependent variable (Y). In that case, the two variables interact in their influence on Y. In other words, the relation between X and Y is conditioned on the value of a moderating variable. In the current paper, interactions will be tested by comparing the parameter estimates of their product terms to their respective standard errors (i.e., null hypothesis testing). Statistically significant interactions will be "probed" to determine at what values of the moderator the relation between X and Y is significant.

In summary, the current study will test the following hypotheses:

Hypothesis 1: Self-reported worry/GAD symptom severity is expected to interact with self-reported EC in prediction of level of AA as measured by self-reports and an objective index, specifically resting HR at baseline. This replication of prior findings serves as a test of the setting conditions for the study. Toh (2017) already successfully replicated this pattern in her study, but it is being repeated since some participants from the original sample were excluded in the current study due to missing or unusable HRV data. Although this interaction is expected to achieve statistical significance, it should be noted that even

in the absence of a significant interaction, the central tenet of the Cognitive Control Model will be supported if indices of cognitive control are significantly negatively correlated with AA at higher levels of worry.

Study Hypotheses

Hypothesis 1A: The pattern of the interaction predicting self-reported AA will be such that worry/GAD symptom severity will be more positively associated with self-reported AA when EC is lower compared with when EC is higher. From the perspective of EC's association with AA, EC will be significantly negatively associative with AA when worry/GAD symptoms are higher. Importantly, for this and all similar hypotheses, the latter prediction will be tested even in the absence of a significant interaction since the pivotal prediction of the Cognitive Control Model is that indices of cognitive control will be negatively associated with AA at higher levels of worry/GAD symptoms. Specifically, I tested the simple slope for cognitive control predicting AA symptoms at the 90th percentile of worry and GAD symptoms. Furthermore, I will probe the region of significance for the association between cognitive control and AA to determine if there is any region of high worry and GAD symptoms in which the slope becomes significantly negative. As noted above, this hypothesis establishes the "setting conditions" for subsequent analyses. If the predicted pattern is not replicated, it may indicate that the current sample does not provide a good context in which to answer other research questions of interest.

Hypothesis 1B: Worry/GAD symptom severity will be more positively associated with average HR during baseline at lower versus higher levels of EC. HR provides a

physiological measure of AA. EC will be negatively associated with baseline HR when worry/GAD symptoms are higher. That prediction will be tested by examining the simple slope for worry/GAD symptoms and by examining the region of significance to determine if EC's association with AA is significantly negative at higher levels of worry/GAD symptoms. Free (2017) found that worry interacted with self-reported EC in prediction of mean HR. This hypothesis seeks to reproduce that pattern in hopes that a successful replication will put the finding on firmer footing.

Hypothesis 2: Self-reported worry/GAD symptom severity is expected to interact with measures of cognitive control in predicting percentage of verbal thought during worry.

***Hypothesis 2A*:** Replicate Toh's (2017) finding in the current sample: Worry/GAD symptom severity will be more positively associated with percentage of verbal thought during worry when EC is higher than when EC is lower. In turn, self-reported EC is expected to be negatively associated with percentage of verbal thoughts during worry when worry/GAD symptoms are higher. Similar to above, this hypothesis attempts to establish the setting conditions for hypothesis 2B. For example, if worry and EC do not interact to predict percentage of verbal thoughts during worry as they did in Toh's study, it may indicate that the subset of participants used in the current study is different from the full sample in ways that make it hard to interpret null findings.

Hypothesis 2B: Worry/GAD symptom severity will be more positively associated with percentage of verbal thought during worry when resting HRV is higher than

when resting HRV is lower. In turn, resting HRV is expected to be negatively associated with percentage of verbal thoughts during worry when worry/GAD symptoms are higher. This hypothesis attempts to extend prior findings by testing whether resting HRV can account for individual differences in percentage of verbal thought during worry. As reviewed above, adequate cognitive control capacity is necessary to shift worry to a verbal mode of processing. In addition, verbal worry has been shown to deplete such capacity. Thus, it is reasonable to expect that individual with higher initial capacity will be more successful at maintaining verbal worry compared with those lowering in cognitive control capacity.

Hypothesis 3: Self-reported worry/GAD symptom severity is expected to interact with self-reported EC in predicting resting HRV. This prediction reflects the view that high levels of worry and GAD symptoms will be associated with higher top-down control capacity among individuals who report higher versus lower levels of EC.

Hypothesis 4: Phasic HRV is expected to decline across the five-minute worry period. The hypotheses below are exploratory, as no prior study has attempted to account for the heterogeneity in HRV among worriers during a worry induction. Thus, as described below competing hypotheses will be tested regarding the pattern of this expected decline:

Hypothesis 4A: Phasic HRV will decline and that decline will be similar regardless of a participant's level of resting HRV.

Hypothesis 4B: Phasic HRV will decline, but the pattern of decline will vary as a function of resting HRV such that:

Hypothesis 4B1: The magnitude of the decline will be greatest or the

downward slope will be strongest over time at lower versus higher levels of resting HRV. This hypothesis reflects the possibility that individuals with low HRV during baseline are those who are most susceptible to the impact of worry due to a lack of reserve capacity.

Hypothesis 4B2: The magnitude of the decline will be greatest or the downward slope will be the strongest over time at moderate levels of resting HRV. This hypothesis reflects the possibility that individuals with low HRV during baseline are constrained regarding further declines whereas those with higher resting HRV are not. However, to the extent that higher resting HRV reflects reserve capacity, individuals at higher levels of resting HRV may show little decline in phasic HRV. Thus, it is the mid-range of resting HRV in which phasic declines in HRV due to a worry induction should be strongest

Hypothesis 4B3: The magnitude of the decline will be greatest or the downward slope will be the strongest over time at higher versus lower levels of resting HRV. This hypothesis reflects the possibility that individuals with low HRV during baseline are constrained regarding further declines whereas those with higher resting HRV are not.

Hypothesis 5: The decline in HRV across the worry period will also be examined as a function of self-reported EC at baseline. Mirroring hypothesis 4, the below hypotheses are exploratory, as no prior study has attempted to account for the heterogeneity in HRV among worriers during a worry induction. Thus, similar to above, competing hypotheses will be tested regarding the pattern of this expected decline.

Hypothesis 5A: Phasic HRV will decline and that decline will be similar regardless of a participant's baseline EC.

Hypothesis 5B: Phasic HRV will decline, but the pattern of decline will vary as a function of baseline EC such that:

Hypothesis 5B1: The magnitude of the decline will be greatest or the downward slope will be strongest over time at lower versus higher levels of self-reported EC. This hypothesis reflects the possibility that individuals with lower EC during baseline are those who are most susceptible to the impact of worry due to a lack of reserve capacity.

Hypothesis 5B2: The magnitude of the decline will be greatest or the downward slope will be the strongest over time at moderate levels of self-reported EC. This hypothesis reflects the possibility that individuals with lower EC during baseline are constrained regarding further declines whereas those with higher EC are not. However, to the extent that higher baseline EC reflects reserve capacity, individuals reporting higher baseline EC may show little decline in phasic HRV. Thus, it is the mid-range of baseline EC in which phasic declines in HRV due to a worry induction should be strongest

Hypothesis 5B3: The magnitude of the decline will be greatest or the downward slope will be the strongest over time at higher versus lower levels of baseline EC. This hypothesis reflects the possibility that individuals with lower EC during baseline are constrained regarding further declines whereas those with higher EC are not.

Chapter 2: Methods

Sample

The sample will comprise a subset of subjects from a previous study (Toh, 2017), which included 198 undergraduate students who were enrolled in Introduction to Psychology at The Ohio State University (OSU). Participants were recruited through the Psychology Department's Research Experience Program (REP), which allows students aged 18 years or older to earn course credit by participating in departmental research. All of the original subjects with suitable HRV data will be included in the current study.

Participants in the original study were recruited in two ways. At the beginning of each semester, all adult students enrolled in Introduction to Psychology are invited to complete optional pre-screening questionnaires for extra credit in the course. A subset of students who opted to complete the prescreening were invited by email to participate if they 1) reported worrying at least 50% of the day and characterized their worry as a problem and 2) had relatively higher or lower scores on the Effortful Control Scale – Persistence and Low Distractibility subscale (ECS; Lonigan & Phillips, 2001). Worriers with ECS scores ≤ 41 and ≥ 47 were invited to participate and constituted the majority of subjects in the original study (N=128, 65%). The remaining subjects (N=70, 35%) opted to enroll in the study in response to an advertisement on the REP webpage that was visible to all REP students. The description of the study indicated that it would be well suited for students who considered themselves worriers (i.e., "worry at least 50% of the day and consider worry difficult to control). Oversampling participants reporting high worry was done to yield a sample that provided a context to test hypotheses relevant to pathological

worry. Screening subjects on the basis of EC allowed for oversampling the tails of the construct in order to increase statistical power for tests of the moderating effects of EC. Self-report measures

Measurement

Demographics.

Demographics. A brief demographics questionnaire was used to gather information about age, gender, race, class rank (e.g., freshman), ethnicity, martial status, and primary language spoken.

Measures of GAD symptoms and worry.

Worry and Anxiety Questionnaire (WAQ). The WAQ (Dugas et al., 2001) is an 11-item self-report measure designed to assess severity of GAD symptoms according to DSM-IV diagnostic criteria. The authors found the WAQ to have 82% specificity and 75% sensitivity. The WAQ has satisfactory test–retest reliability and good known-groups validity (Dugas et al., 2001).

Penn State Worry Questionnaire (PSWQ). The PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report measure that assesses trait-like worry. Respondents rate how typical each item is of them (e.g., I know I should not worry about things, but I just can't help it) using a five-point scale (1 = Not at all typical, 5 = Very typical). This measure has demonstrated good internal consistency ($\alpha = 0.93$) and high test-re-test reliability (0.92) over an 8 – 10 week period.

Autonomic arousal measures.

Depression, Anxiety, and Stress Scales (DASS). The DASS (Lovibond & Lovibond, 1995) is a 42 item self-report measure that comprises three subscales (14 items each) that measure level of depression, anxiety, and stress. Respondents are instructed to indicate how much each item applied to them during the past week using a 4-point scale (0 = Did not apply to me at all, 3 = Applied to me very much, or most of the time). The current study will use the anxiety subscale (DASS-A), which includes items related to AA symptoms such as dry mouth, perspiration, and increased heart rate. High scores indicate greater symptom severity.

Electrocardiography. Ambulatory heart rate recordings were collected throughout each laboratory session using Firstbeat Bodyguard 2 devices, which are non-invasive electrocardiographic (ECG) monitoring systems. The devices have a heart beat sampling rate of 1000 Hz. Two pre-gelled, disposable electrodes were attached directly to the skin, one on the upper right side of chest, below the clavicle, the other on the left side of the chest, on the lower rib cage. ECG data from the device were used to calculate baseline resting heart rate as well as phasic heart rate during the worry periods.

To obtain estimates of HRV, the interbeat interval time series from the Firstbeat Bodyguard 2 output will be analyzed using the Kubios HRV analysis package 3.0 (Tarvainen, Lipponen, & Karjalainen, 2009). Kubios will be used for error correction of ectopic beats and to calculate autoregressive estimates of high frequency power (HF-HRV; 0.15-.40 Hz, ms2), a commonly used index of vagally-mediated HRV. As noted above, higher values of HF-HRV are interpreted as reflecting greater cognitive control capacity.

Values of HF-HRV will be natural log transformed to better approximate a normal distribution. ECG data from the device were used to calculate baseline HRV as well as phasic HRV the worry periods.

Cognitive capacity measures.

Effortful Control Scale – Persistence/Low Distractibility subscale (ECS). The ECS (Lonigan & Phillips, 2001) is a 24-item scale that measures effortful control. Respondents rate how well each item describes them the majority of the time using a 5-point scale (1 = Not at all, 5 = Very much). The full ECS comprises two subscales: Persistence/Low Distractibility (ECS-PLD; 12 items) and Impulsivity (ECS-I; 12 items). In the current study, however, the ECS-I items were not included because they tap a dimension of EC that is not relevant to they study's hypotheses (e.g., inhibition of impulsive motor responses). The measure has good psychometric properties in college samples (Vasey, 2012).

Study Procedures

Participation in the study entailed two 150-minute laboratory sessions. At the beginning of the first session, participants were provided with a description of the study, an overview of their rights, and asked to sign an informed consent form. Next, they were given instructions on how to affix the FirstBeat Bodyguard 2 electrodes. After the device was properly attached, participants were asked to sit quietly alone in the experiment room for five minutes while baseline resting ECG data were collected.

Next participants were randomly assigned to engage in one of two thought sampling tasks, which were adapted from Brokovec and Inz (1990), and Hirsch an Mathews (2012).

In the first session, thoughts sampling occurred during a 5-minute self-relaxation period or a 5-minute worry period. In the second session, participants completed the other task (task order was counterbalanced to control for order effects). Prior to the task, participants were taught to differentiate between verbal and imaginal processing. They were told the following: "Images are when you are generating a picture in your mind and really concentrating on what you can see, feel, smell, hear, and taste in the image. Images are often very vivid because you're tuning into all of your senses. Verbal thoughts are when you're thinking using words and silently talking to yourself, like an internal running commentary or dialogue. When you're thinking in verbal thoughts you are thinking in words and sentences" (Leigh & Hirsch, 2011). Participants were then asked to practice each mode of processing by imagining vs. thinking about cutting a lemon, an exercise adapted from Holmes, Mathews, Dalgleish, and Mackintosh (2006). They were then asked to imagine a specific topic (eating dinner), and to generate and hold the image in their mind for one minute. Next, they were asked to practice thinking in verbal form about another abstract topic (friendship). Friendship was selected as a practice topic because it is positively valenced, and thus unlikely to prompt worry, and it is sufficiently abstract to minimize spontaneous switching to imaginal processing.

At the beginning of the worry period, participants were asked to identify a personal worry topic and then asked about the potential negative outcomes, a worry catastrophizing task previously used by Vasey and Borkovec (1992). Next, they were left alone in the room and asked to worry in their usual manner about a topic of current concern to them. Every 30 seconds during the worry period, a short computer-generated tone was played. Prior to

the task, participants were that, upon hearing the tone, they were to indicate on a computer what percentage of their worry was verbal thought and what percentage was imagery. They were also asked to indicate the degree to which they felt relaxed, worried, and aroused on a 7-point scale (these data will not be used in the current study). They responded to a total of 10 tones. Data from the relaxation period were collected in a similar fashion, but a different session, and not included in the current study.

After completing the session's thought sampling task, participants completed a battery of questionnaires (described above) on Qualtrics, a secure, online data collection service. Finally, participants completed executive function tasks that were not used in the current study.

Data Analytic Strategy

Hypothesis 1A-1B. Multiple Linear Regression (MLR) will be used to test the hypotheses that self-reported worry/GAD symptom severity interacts with self-reported EC in prediction of level of self-reported AA (Hypothesis 1A) and resting HR at baseline (Hypothesis 1B).

Hypothesis 2A-2B. SAS's Repeated Measures Modeling will be used to test all sub-hypotheses concerning whether change in percentage of verbal thought during worry varies as a function of cognitive control capacity, as measure by self-reported EC and resting HRV.

Hypothesis 3: MLR will be used to test the hypothesis that worry/GAD symptoms interacts with EC in prediction of resting HRV.

Hypotheses 4A-4B2. SAS's Repeated Measures Modeling will be used to test all sub-hypotheses concerning whether change in phasic HRV during the worry period is a function of resting HRV.

Hypotheses 5A-4B2. SAS's Repeated Measures Modeling will be used to test all sub-hypotheses concerning whether change in phasic HRV during the worry period is a function of baseline self-reported EC.

Chapter 3: Results

Data from 174 of the original 199 participants included in Toh (2018) were analyzed in the current study. The bulk of excluded participants were removed because of missing cardiac data due to participant drop out (N=22). Two subjects were mistakenly assigned that same identification number; they were excluded because it was not possible to match their questionnaire and cardiac data. One subject was excluded due to missing all questionnaire data. Missing data was handled in two ways. Missed items on questionnaires were replaced with participants' respective means on that questionnaire. When entire questionnaires were missing, scores were imputed using EM maximum likelihood estimates. Questionnaire data were imputed for two participants; one participants was missing the PSWQ, and another was missing the PSWQ and DASS-A. In addition, resting cardiac data (i.e., HR and HRV) were imputed for two participants. Since the correlations between resting cardiac values and cardiac values during the first worry period were high (*r*'s ranged from .75 to .89), these values were entered as auxiliary variables to improve estimation of imputed values.

Descriptive Statistics. The sample was composed of undergraduate students. Participants were aged 18 and 43 years, with a mean age of 19.4 years (*SD* = 3.2 years). Mean scores, standard deviations, and internal consistency (i.e., Cronbach's alpha) are presented in Table 1. Means and standard deviations were within expected ranges and consistent with prior samples of undergraduate students. Self-report measures demonstrated acceptable internal consistency; α's ranged from .87 to .93.

Excluded participants were compared with included participants using independent

t-tests on all measures for which data were available. Included participants did not significantly differ from excluded participants in terms of scores on the WAQ, PSWQ, ECS or DASS-A.

Table 1: Descriptive Statistics

	M	SD	α	N (alpha)*
DASS-A	10.67	7.85	.87	173
WAQ	45.14	16.51	.90	172
PSWQ	59.19	13.37	.93	171
ECS	43.74	8.74	.89	172
Heart rate	78.84	11.73		
RMSSD (ms)	40.24	21.37		
HF-HRV (nat. log.)	6.33	1.13		

*Missing questionnaire items were not imputed; only those with complete data were included in internal consistency analyses.

Zero-order correlations, reported in Table 2, show that all variables were correlated as expected. For example, consistent with prior findings, WAQ and PSWQ were significantly positively correlated with each other (r = .68), and each was significantly negatively correlated with ECS scores (r = -.48 and -.30, respectively). In addition, WAQ and PSWQ were significantly positively correlated with DASS-Anxiety (r = .66 and .52, respectively). As expected, ECS was significantly negatively correlated with DASS-A (r = -.43).

Table 2: Zero order correlations

	1	2	3	4	5	6	7
1. DASS-A							
2. WAQ	.66*						
3. PSWQ	.52*	.68*					
4. ECS	-.43*	-.49*	-.30				
5. Heart rate[1]	.05	.04	-.01	-.06			
6. RMSSD (ms)[1]	.03	.01	-.04	.01	.66*		
7. HF-HRV (nat. log.)[1]	.03	-.05	-.05	.04	-.53*	.86*	

[1]Computed from 5-minute resting period

*$p < .0001$

Primary Analyses

Hypothesis 1a: Worry/GAD symptoms x Effortful Control predicting AA symptoms

WAQ x ECS predicting DASS-A. As shown in Table 3, regression analysis revealed significant effects of WAQ (B = .59, $p < .001$) and ECS (B = -.14, $p = .03$) predicting DASS-A in Step 1. The addition of the WAQ x ECS term in Step 2 yielded a significant interaction ($p = .017$) that was consistent with expectation. As depicted in Figure 1, higher scores on the WAQ were more positively associated with scores on the DASS-A when ECS was low (i.e., 10th percentile; B = .85, $p < .001$) than when ECS was high (i.e., 90th percentile; B = .45 $p < .001$). Probing the region of significance of the interaction revealed that the simple slope for WAQ was significantly positive at all observed Z-score values of ECS. As expected,

examination of the simple slopes from the perspective of the effect of ECS on DASS-A, depicted in Figure 2, revealed that when WAQ scores were high (i.e., 90th percentile), ECS was significantly negatively correlated with DASS-A scores (B = -.27, $p < .01$). Probing the region of significance of the interaction in this direction revealed that the simple slope of ECS was significantly negative at all Z-score values of WAQ above .15, which represented 51% of the sample.

Table 3: Multiple regression analysis predicting DASS-A from WAQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.450	.450	<.001
Intercept	.00	.06		1.00			
WAQ	.59	.07	.51	<.001			
ECS	-.14	.05	-.13	<.03			
Step 2					.468	.018	.017
Intercept	-.07	.06		.27			
WAQ	.64	.07	.53	<.001			
ECS	-.11	.07	-.09	.11			
WAQ x ECS	-.14	.06	-.18	.017			

Figure 1. Predicting DASS-A from WAQ as a function of ECS

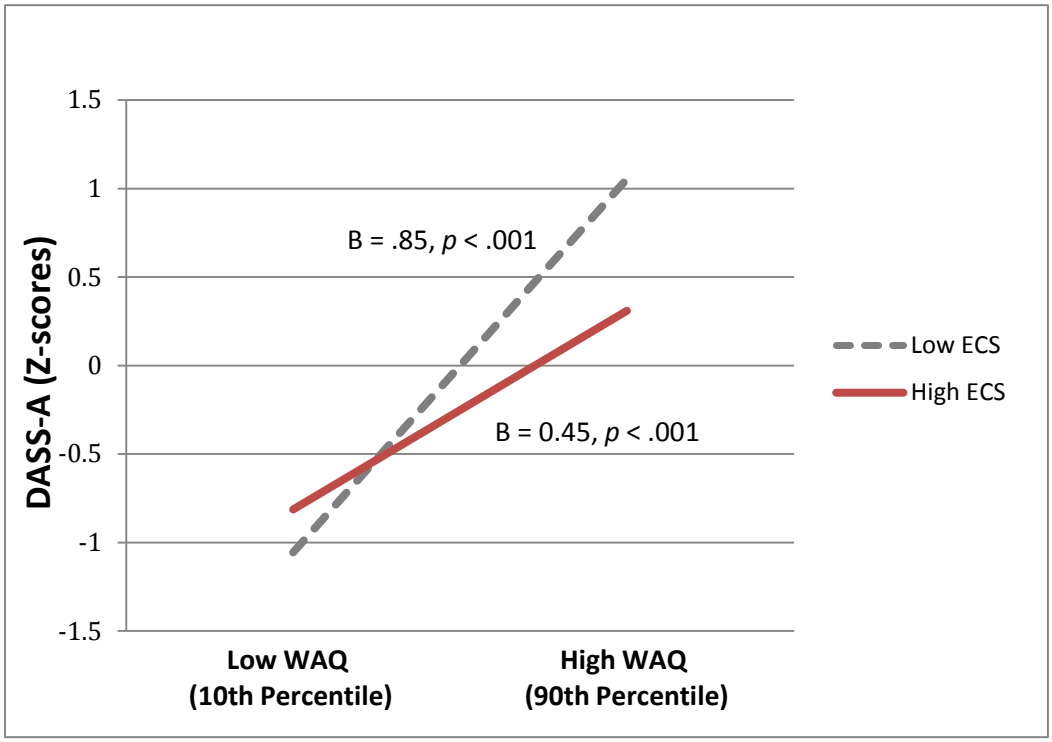
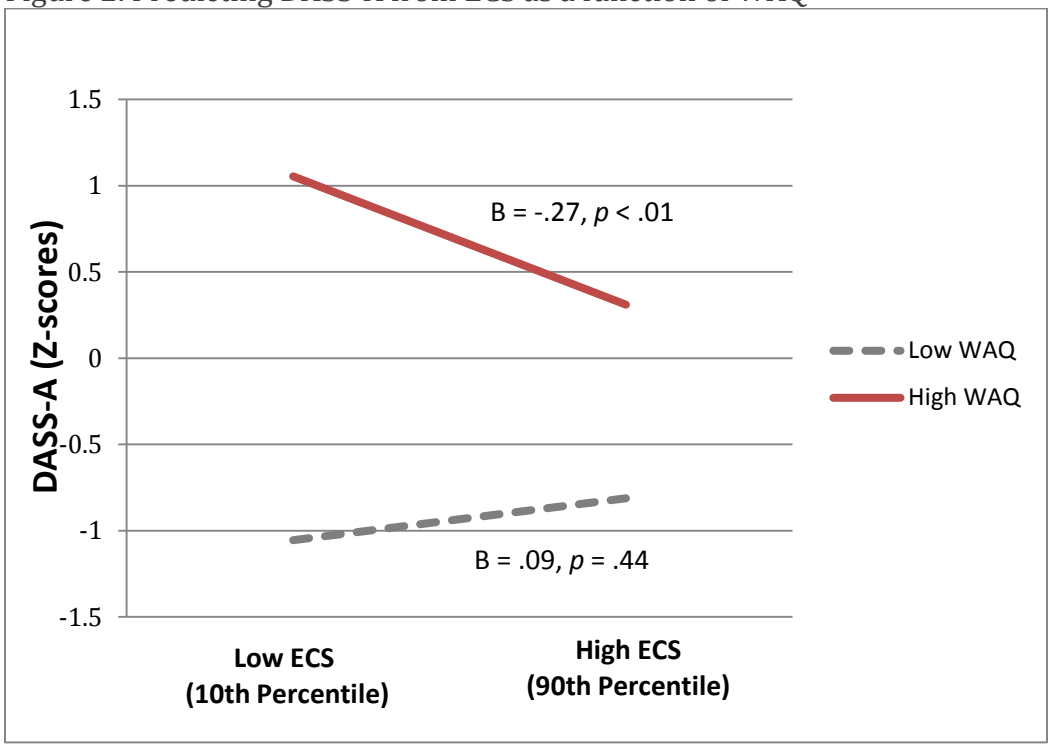


Figure 2. Predicting DASS-A from ECS as a function of WAQ



PSWQ x ECS predicting DASS-A. As shown in Table 4, regression analysis revealed significant effects of PSWQ (B = .43, $p < .001$) and ECS (B = -.30, $p < .001$) predicting DASS-A in Step 1. The addition of the PSWQ x ECS term in Step 2 yielded a significant interaction ($p < .05$) that was consistent with expectation. As depicted in Figure 3, higher scores on the PSWQ were more positively associated with scores on the DASS-A when ECS was low (i.e., 10th percentile; B = .62, $p < .001$) than when ECS was high (i.e., 90th percentile; B = .30 $p < .01$). Probing the region of significance of the interaction revealed that the simple slope for PSWQ was significantly positive at all observed Z-score values of ECS. As expected, examination of the simple slopes from the perspective of the effect of ECS on DASS-A, depicted in Figure 4, revealed that when PSWQ scores were high (i.e., 90th percentile), ECS was significantly negatively correlated with DASS-A scores (B = -.44, $p < .001$). Probing the region of significance of the interaction in this direction revealed that the simple slope of ECS was significantly negative at Z-score values of PSWQ above -1.08, which represented 84% of the sample.

Table 4: Multiple regression analysis predicting DASS-A from PSWQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.354	.354	<.001
Intercept	.00	.06		1.00			
PSWQ	.43	.06	.41	<.001			
ECS	-.30	.06	-.29	<.001			
Step 2					.369	.015	<.05
Intercept	-.03	.06		.59			
PSWQ	.45	.07	.42	<.001			
ECS	-.30	.06	-.29	<.001			
PSWQ x ECS	-.12	.06	-.12	.0496			

Figure 3. Predicting DASS-A from PSWQ as a function of ECS

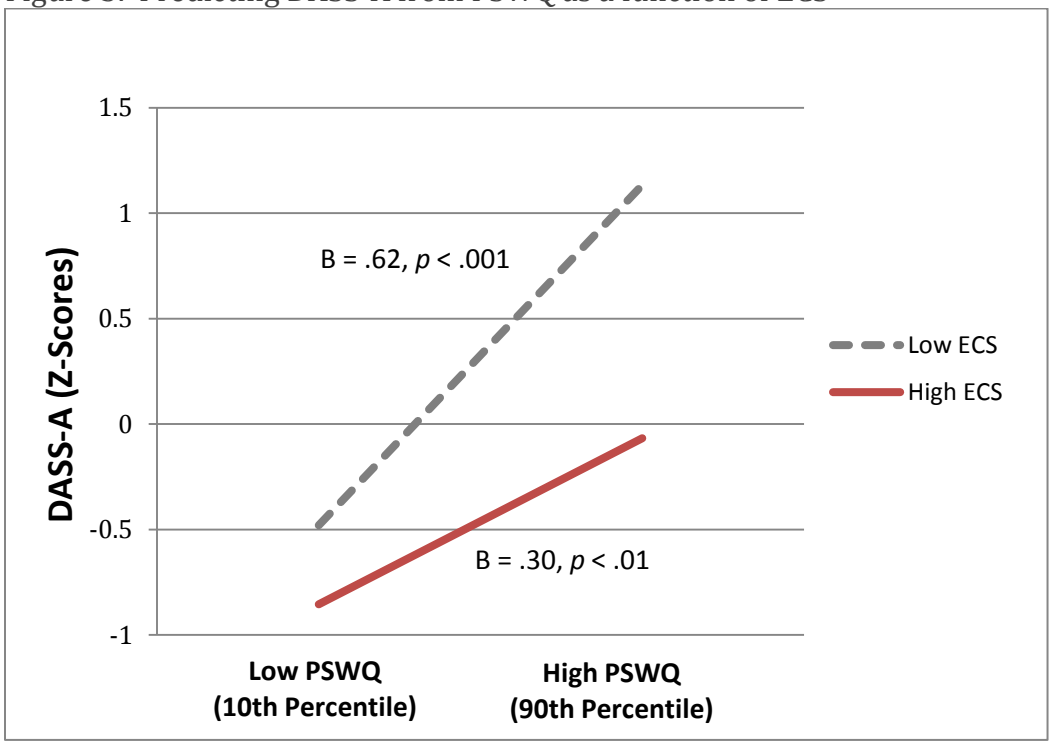
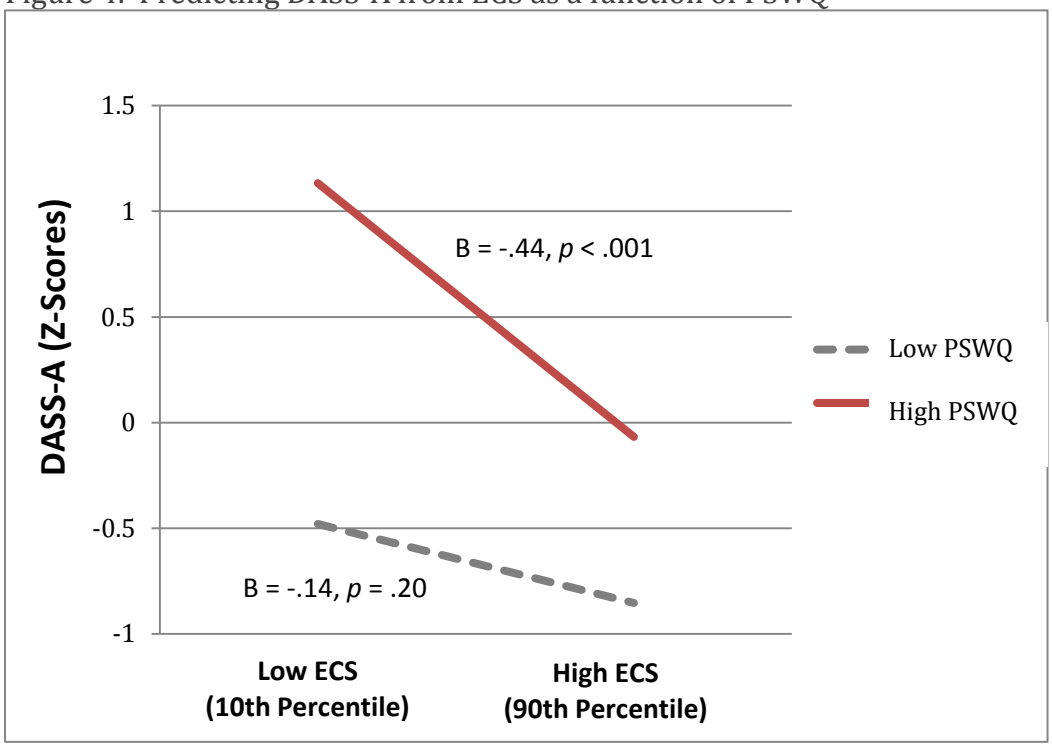


Figure 4. Predicting DASS-A from ECS as a function of PSWQ



Hypothesis 1b: Worry/GAD symptoms x Effortful Control predicting resting HR

WAQ x ECS predicting resting HR. As shown in Table 5, regression analysis revealed significant effects of HF-HRV (B = .59, $p < .001$) predicting mean resting HR in Step 1. Contrary to expectation, the addition of the WAQ x ECS interaction term in Step 2 did not significantly improve the model's fit, though the pattern of the interaction was consistent with expectation. Probing the interaction revealed that the simple slope for WAQ was not significant at any level of ECS in the sample. Likewise, the simple slope of ECS was not significant at any observed score on the WAQ.

Table 5: Multiple regression analysis predicting resting HR from WAQ and ECS, controlling for HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.281	.281	<.001
Intercept	.00	.07		1.00			
WAQ	.00	.08	.00	.99			
ECS	-.04	.08	-.03	.62			
HF-HRV	-.53	.07	-.53	<.001			
Step 2					.285	.004	.304
Intercept	-.04	.06		.63			
WAQ	.25	.07	.02	.75			
ECS	-.02	.06	-.02	.81			
HF-HRV	-.53	.07	-.53	<.001			
WAQ x ECS	-.07	.07	-.07	.30			

PSWQ x ECS predicting resting HR. As shown in Table 6, regression analysis revealed significant effects of HF-HRV (B = .53, $p < .001$) predicting mean resting HR in Step 1. Contrary to expectation, the addition of the PSWQ x ECS interaction term in Step 2 did not significantly improve the model's fit, though the pattern of the interaction was consistent

with expectation. Probing the interaction revealed that the simple slope for PSWQ was not significant at any level of ECS in the sample. Likewise, the simple slope of ECS was not significant at any level of observed PSWQ scores.

Table 6: Multiple regression analysis predicting resting HR from PSWQ and ECS, controlling for HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.281	.281	<.001
Intercept	.00	.07		1.00			
PSWQ	.01	.07	.01	.94			
ECS	-.04	.07	-.03	.60			
HF-HRV	-.53	.07	-.53	<.001			
Step 2					.288	.007	.198
Intercept	-.02	.07		.73			
PSWQ	.02	.07	.02	.79			
ECS	-.04	.07	-.03	.60			
HF-HRV	-.53	.07	-.53	<.001			
PSWQ x ECS	-.08	.06	-.08	.20			

Hypothesis 2: Change in percentage of verbal thoughts during worry as a function of EC.

As shown in Table 7, linear mixed models analysis revealed a significant average decline in percentage of verbal thoughts during worry (B = -0.60 *p* =.011, Figure 5), which was consistent with prediction. As illustrated in Appendix A there were substantial individual differences in change in percentage of thoughts across the worry period. Contrary to expectation, however, the heterogeneity in individual slopes was not accounted for by scores on the ECS (Table 8), resting HF-HRV (Table 9), or resting RMSSD (Table 10).

Table 7: Linear mixed model analysis predicting change in percentage of verbal thought during worry

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	71.75	1.69	<.0001	(68.41 ≥ β_0 ≥ 75.06)
Time (b_1)	-0.60	.23	.011	(-1.05 ≥ β_1 ≥ -0.14)
Variances of random effects				
φ_{00} = V(b_{i0})	356.51	53.76	<.0001	(270.8 ≥ φ_{00} ≥ 490.7)
φ_{11} = V(b_{i1})	4.30	1.02	<.0001	(2.84 ≥ φ_{11} ≥ 7.26)
σ^2 = V(e_{ij})	412.19	15.63	<.0001	(383.2 ≥ σ^2 ≥ 444.6)

Figure 5: Change in percentage of verbal thoughts during worry (noise added to reduce overplotting)

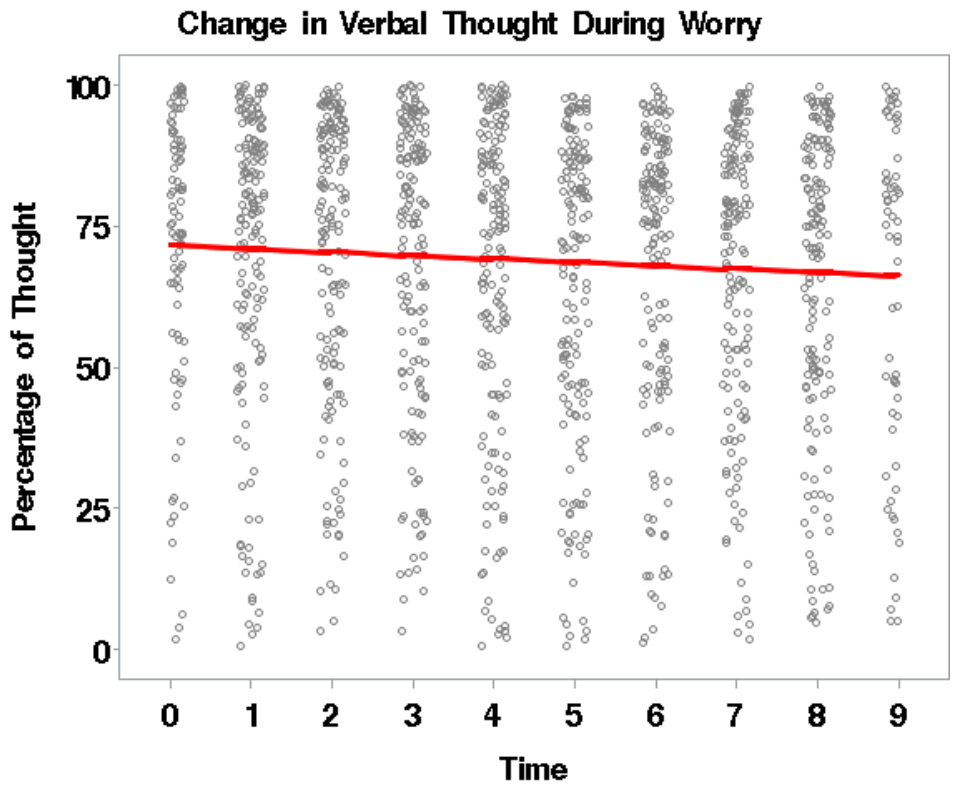


Table 8: Linear mixed model analysis predicting change in percentage of verbal thought during worry as a function of ECS.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	71.75	1.69	<.0001	(68.41 ≥ β_0 ≥ 75.06)
Time (b_1)	-0.60	0.23	0.01	(-1.05 ≥ β_1 ≥ -0.14)
ECS (b_2)	-0.32	1.70	0.85	(-3.65 ≥ β_2 ≥ 3.01)
Time x ECS (b_3)	0.23	0.66	0.51	(-0.30 ≥ β_3 ≥ 0.60)
Variances of random effects				
φ_{00} = V(b_{i0})	356.11	53.75	<.0001	(270.7 ≥ φ_{00} ≥ 490.6)
φ_{11} = V(b_{i1})	4.28	1.01	<.0001	(2.83 ≥ φ_{11} ≥ 7.24)
σ^2 = V(e_{ij})	412.19	15.63	<.0001	(383.2 ≥ σ^2 ≥ 444.6)

Table 9: Linear mixed model analysis predicting change in percentage of verbal thought during worry as a function of resting HF-HRV.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	71.75	1.69	<.0001	(68.42 ≥ β_0 ≥ 75.09)
Time (b_1)	-0.60	0.23	0.01	(-1.05 ≥ β_1 ≥ -0.14)
HF-HRV (b_2)	1.38	1.70	0.42	(-1.95 ≥ β_2 ≥ 4.70)
Time x HF-HRV (b_3)	0.01	0.23	0.97	(-0.44 ≥ β_3 ≥ 0.46)
Variances of random effects				
φ_{00} = V(b_{i0})	354.63	53.56	<.0001	(269.3 ≥ φ_{00} ≥ 488.4)
φ_{11} = V(b_{i1})	4.30	1.02	<.0001	(2.83 ≥ φ_{11} ≥ 7.24)
σ^2 = V(e_{ij})	412.19	15.63	<.0001	(383.2 ≥ σ^2 ≥ 444.6)

Table 10: Linear mixed model analysis predicting change in percentage of verbal thought during worry as a function of resting RMSSD.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	71.75	1.69	<.0001	(68.41 ≥ β_0 ≥ 75.10)
Time (b_1)	-0.60	0.23	0.01	(-1.05 ≥ β_1 ≥ -0.14)
RMSSD (b_2)	0.39	1.70	0.82	(-2.94 ≥ β_2 ≥ 3.72)
Time x RMSSD (b_3)	0.01	0.23	0.67	(-0.36 ≥ β_3 ≥ 0.55)
Variances of random effects				
φ_{00} = V(b_{i0})	356.36	53.74	<.0001	(270.7 ≥ φ_{00} ≥ 490.6)
φ_{11} = V(b_{i1})	4.30	1.02	<.0001	(2.84 ≥ φ_{11} ≥ 7.25)
σ^2 = V(e_{ij})	412.19	15.63	<.0001	(383.2 ≥ σ^2 ≥ 444.6)

Hypothesis 3: Worry/GAD symptoms x Effortful Control predicting resting HRV

***WAQ x ECS predicting resting HF-HRV*.** As shown in Table 11, regression analysis revealed no significant main effects in Step 1. Contrary to expectation, the addition of the WAQ x ECS interaction term in Step 2 did not significantly improve the model's fit. In addition, probing the interaction revealed that the simple slope for WAQ was not significant at any level of ECS in the sample. Likewise, the simple slope of ECS was not significant at any observed score of WAQ.

Table 11: Multiple regression analysis predicting resting HF-HRV from WAQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.003	.003	.791
Intercept	.00	.08		1.00			
WAQ	-.04	.09	-.03	.67			
ECS	.02	.09	.02	.26			
Step 2					.003	.000	.847
Intercept	.01	.09		.93			
WAQ	-.04	.09	-.04	.64			
ECS	.02	.09	.02	.84			
WAQ x ECS	.02	.08	.02	.85			

PSWQ x ECS predicting resting HF-HRV. As shown in Table 12, regression analysis revealed no significant main effects in Step 1. Contrary to expectation, the addition of the PSWQ x ECS interaction term in Step 2 did not significantly improve the model's fit. In addition, probing the interaction revealed that the simple slope for PSWQ was not significant at any level of ECS in the sample. Likewise, the simple slope of ECS was not significant at any observed score of PSWQ.

Table 12: Multiple regression analysis predicting resting HF-HRV from PSWQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.006	.006	.600
Intercept	.00	.08		1.00			
PSWQ	.07	.08	.07	.39			
ECS	.06	.08	.06	.44			
Step 2					.003	.000	.847
Intercept	-.00	.08		.97			
PSWQ	.07	.08	.07	.39			
ECS	.06	.08	.06	.44			
PSWQ x ECS	-.01	.07	-.01	.89			

WAQ x ECS predicting resting RMSSD. As shown in Table 13, regression analysis revealed no significant main effects in Step 1. Contrary to expectation, the addition of the WAQ x ECS interaction term in Step 2 did not significantly improve the model's fit. In addition, probing the interaction revealed that the simple slope for WAQ was not significant at any level of ECS in the sample. Likewise, the simple slope of ECS was not significant at any observed score of WAQ.

Table 13: Multiple regression analysis predicting resting RMSSD from WAQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.000	.000	.992
Intercept	.00	.08		1.00			
WAQ	-.01	.09	-.01	.92			
ECS	.02	.09	.00	.98			
Step 2					.001	.001	.993
Intercept	-.01	.09		.90			
WAQ	-.00	.09	-.00	.99			
ECS	.01	.09	.01	.93			
WAQ x ECS	-.02	.08	.02	.78			

***PSWQ x ECS predicting resting RMSSD*.** As shown in Table 14, regression analysis no significant main effects in Step 1. Contrary to expectation, the addition of the WAQ x ECS interaction term in Step 2 did not significantly improve the model's fit. In addition, probing the interaction revealed that the simple slope for PSWQ was not significant at any level of PSWQ in the sample. Likewise, the simple slope of ECS was not significant at any observed score of WAQ.

Table 14: Multiple regression analysis predicting resting RMSSD from PSWQ and ECS

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.002	.002	.870
Intercept	.00	.08		1.00			
PSWQ	.04	.08	.04	.60			
ECS	.02	.08	.02	.82			
Step 2					.002	.000	.949
Intercept	-.01	.08		.94			
PSWQ	.05	.08	.04	.58			
ECS	.02	.08	.02	.81			
PSWQ x ECS	-.02	.07	-.02	.78			

Hypothesis 4: Change in Phasic HRV as a function of resting HRV

Linear mixed models analysis was used to test three competing hypotheses regarding change in phasic HRV over time during worry. Results indicate that HRV indeed significantly declined across the worry period as indexed by HF-HRV (B = -0.03. p =<.0001; Table 15) and RMSSD (B = -.02, p <.0001; Table 16). The decline in HRV during worry is consistent with expectation and is depicted in Figures 6 and 7. As illustrated in Appendices B and C there were substantial individual differences in change in phasic HRV across the worry period. The heterogeneity in individual slopes, however, was not accounted for by individual differences in resting HF-HRV (Table 17), or resting RMSSD (Table 18).

Table 15: Linear mixed models analysis predicting change phasic HF-HRV during worry

Fixed effects				
	Est.	SE	p	95% C.I.
Intercept (b_0)	0.05	.09	0.58	(-0.012 ≥ β_0 ≥ 0.218)
Time (b_1)	-0.03	.007	<.0001	(-.0433 ≥ β_1 ≥ -0.159)
Variances of random effects				
φ_{00} = V(b_{i0})	1.131	0.138	<.0001	(0.903 ≥ φ_{00} ≥ 1.460)
φ_{11} = V(b_{i1})	0.003	0.001	<.001	(0.002 ≥ φ_{11} ≥ 0.006)
σ^2 = V(e_{ij})	0.454	0.017	<.0001	(0.422 ≥ σ^2 ≥ 0.490)

Figure 6: Change in phasic HF-HRV over time during worry (first 25 cases; line of best fit calculated from full sample)

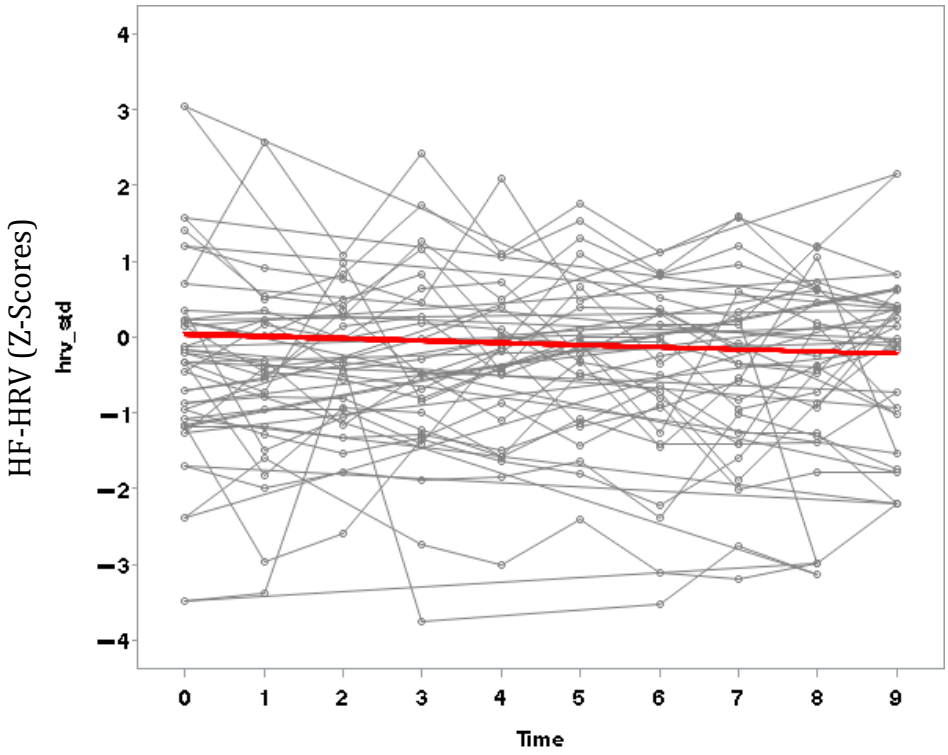


Table 16: Linear mixed models analysis predicting change phasic RMSSD during worry

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	-0.07	.07	0.29	(-0.218 ≥ β_0 ≥ 0.065)
Time (b_1)	-0.02	.005	<.0001	(-.029 ≥ β_1 ≥ -0.010)
Variances of random effects				
φ_{00} = V(b_{i0})	0.835	0.096	<.0001	(0.675 ≥ φ_{00} ≥ 1.060)
φ_{11} = V(b_{i1})	0.002	0.000	<.0001	(0.001 ≥ φ_{11} ≥ 0.003)
σ^2 = V(e_{ij})	0.166	0.006	<.0001	(0.154 ≥ σ^2 ≥ 0.179)

Figure 7: Change in phasic RMSSD over time during worry (first 25 cases; line of best fit calculated from full sample)

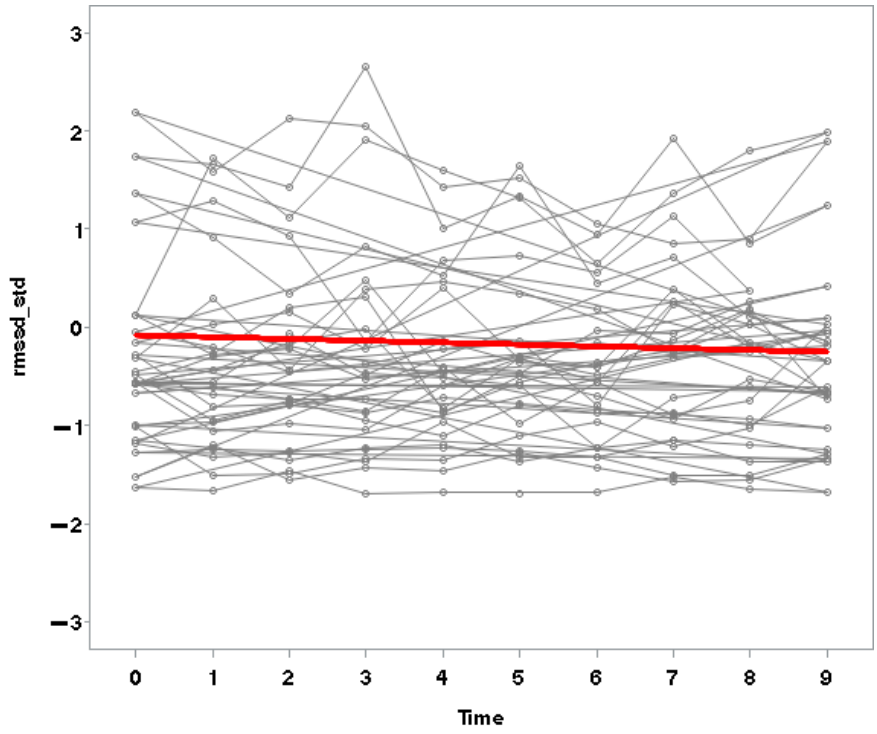


Table 17: Linear mixed models analysis predicting change in phasic HF-HRV during worry as a function of resting HF-HRV.

Fixed effects				
	Est.	SE	*p*	95% C.I.
Intercept (b_0)	0.048	.052	0.36	(-.055 ≥ β_0 ≥ .151)
Time (b_1)	-0.030	.007	<.0001	(-.043 ≥ β_1 ≥ .016)
HF-HRV (b_2)	0.905	.052	<.0001	(.803 ≥ β_2 ≥ 1.008)
Time x HF-HRV (b_3)	-0.008	.007	0.26	(-.021 ≥ β_3 ≥ 0.006)
Variances of random effects				
φ_{00} = V(b_{i0})	.317	.051	<.0001	(.236 ≥ φ_{00} ≥ .447)
φ_{11} = V(b_{i1})	.003	.001	.001	(.002 ≥ φ_{11} ≥ .006)
σ^2 = V(e_{ij})	.454	.017	<.0001	(.422 ≥ σ^2 ≥ .490)

Table 18: Linear mixed models analysis predicting change in phasic RMSSD during worry as a function of resting RMSSD.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	-0.076	.037	0.04	(-.150 ≥ β_0 ≥ -.003)
Time (b_1)	-0.019	.005	<.0001	(-.029 ≥ β_1 ≥ .010)
HF-HRV (b_2)	0.808	.037	<.0001	(.735 ≥ β_2 ≥ .882)
Time x HF-HRV (b_3)	-0.007	.005	0.17	(-.016 ≥ β_3 ≥ 0.003)
Variances of random effects				
φ_{00} = V(b_{i0})	.185	.026	<.0001	(.236 ≥ φ_{00} ≥ .447)
φ_{11} = V(b_{i1})	.002	.000	<.0001	(.002 ≥ φ_{11} ≥ .006)
σ^2 = V(e_{ij})	.166	.006	<.0001	(.422 ≥ σ^2 ≥ .490)

Hypothesis 5: Change in Phasic HRV as a function of self-reported EC.

Linear mixed models analysis was used to test three competing hypotheses regarding change in phasic HRV over time during worry. As noted above, results indicate that HRV significantly declined across the worry period. As shown in Tables 19 (HF-HRV) and 20 (RMSSD), the heterogeneity in individual slopes was not accounted for by individual differences in scores on the ECS..

Table 19: Linear mixed models analysis predicting change in phasic HF-HRV during worry as a function of ECS.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	0.048	.086	0.58	(-.122 ≥ β_0 ≥ .218)
Time (b_1)	-0.030	.007	<.0001	(-.043 ≥ β_1 ≥ -.016)
ECS (b_2)	-0.019	.086	.827	(-.188 ≥ β_2 ≥ .150)
Time x ECS (b_3)	0.004	.007	0.574	(-.010 ≥ β_3 ≥ 0.018)
Variances of random effects				
φ_{00} = V(b_{i0})	1.13	.138	<.0001	(.902 ≥ φ_{00} ≥ 1.460)
φ_{11} = V(b_{i1})	.003	.009	.001	(-.037 ≥ φ_{11} ≥ .003)
σ^2 = V(e_{ij})	.454	.017	<.0001	(.422 ≥ σ^2 ≥ .490)

Table 20: Linear mixed models analysis predicting change in phasic RMSSD during worry as a function of ECS.

	Est.	SE	*p*	95% C.I.
Fixed effects				
Intercept (b_0)	-0.076	.072	0.28	(-.218 ≥ β_0 ≥ .065)
Time (b_1)	-0.019	.005	<.0001	(-.029 ≥ β_1 ≥ .010)
ECS (b_2)	0.018	.072	.80	(-.123 ≥ β_2 ≥ .159)
Time x ECS (b_3)	-0.003	.005	0.55	(-.012 ≥ β_3 ≥ 0.006)
Variances of random effects				
φ_{00} = V(b_{i0})	.835	.096	<.0001	(.675 ≥ φ_{00} ≥ 1.059)
φ_{11} = V(b_{i1})	.002	.000	<.0001	(-.025 ≥ φ_{11} ≥ -.006)
σ^2 = V(e_{ij})	.166	.006	<.0001	(.154 ≥ σ^2 ≥ .179)

Chapter 4: Discussion

This study sought to extend prior findings supporting the Cognitive Control Model (Vasey et al., 2016) by examining HRV and verbal-linguistic processing during a worry induction. The first aim (hypothesis 1a) was to replicate the basic interaction underpinning the Cognitive Control Model to establish that the sample was an appropriate context in which to test the current study's novel hypotheses. As expected, self-reported worry/GAD symptoms interacted with self-reported EC in prediction of AA symptoms. Mirroring prior research, worry/GAD symptoms were less strongly positively associated with self-reported AA when EC was higher versus lower. From the standpoint of EC, its association with AA was significantly negative when worry/GAD symptoms were high. As noted above, Toh (2018) successfully replicated this interaction, but the analyses were repeated here because the current study used only a subset of Toh's original sample.

This study also sought to replicate Free's (2018) finding that worry/GAD symptoms interact with EC in prediction of resting heart rate, an objective measure of AA (hypothesis 1b). While the pattern was in the expected direction, the interaction was not statistically significant. As in Free's previous study, resting HRV was added to the model as a covariate in order to control for the parasympathetic nervous system's influence on heart rate. Thus, heart rate could be interpreted as a closer proxy to sympathetic activation (i.e., AA). Addition of HRV as a covariate, however, provided little improvement to the model's fit; the interaction term did not reach significance. The inability to fully replicate Free's finding may be attributable to differences in sample sizes. That study was based on data from 286 participants; the current study had only 174, which limited statistical power.

Hypothesis 2: The Cognitive Control model posits that worriers with high cognitive control capacity misuse it in service of constraining worry to a verbal mode of processing, sparing them from aversive levels of AA. As described earlier, there is reason to believe that verbal worry both requires and depletes cognitive resources. Consistent with that view, verbal worry declined on average across the five-minute worry induction. Despite this pattern on average, there were substantial individual differences in change in verbal worry over time. Some individuals reported a steady decline in verbal thought, while others reported an increase. It is possible that the former reflects diminishing cognitive control capacity, and the latter an exertion of such capacity. That is, to the extent that verbal worry is especially depleting among those with low cognitive control capacity, such individuals should have a diminishing resources to sustain verbal worry. On the other hand, it is possible that people with greater cognitive control capacity spontaneously increase verbal processing in an effort to manage AA. However, the current study failed to find support for that hypothesis. Specifically, this study tested whether the observed heterogeneity in change in amount of verbal thought could be explained by individual differences in cognitive control capacity, as indexed by resting HRV. Contrary to expectation, however, resting HRV did not moderate change in verbal worry over time. Post-hoc analyses further revealed that change in verbal thought was also not moderated by worry/GAD symptoms or self-reported EC, which conflicts which Toh's findings in the larger sample.

Assuming that change in proportion of verbal processing during worry is dependent on level of cognitive control capacity, there are several reasons why such a pattern was not

evident in the current sample. First, it appears that the subset of participants included in the current study differed from the full sample in regard to the relation between percentage of thoughts during worry and EC. As noted above, Toh (2018) found that change in percentage of verbal thoughts over time was moderated by self-reported EC. Since that finding was not replicated in the current sample, it is difficult to interpret the null findings with HRV as the moderator. Indeed, the fact that HRV did not moderate the relationship may reflect an usual characteristic of sample. Second, it is possible that resting HRV is less predictive of the fluctuations in amount of verbal thought during worry than previously thought. Instead, phasic HRV may be more related to change in amount of verbal worry. As discussed below, participants experienced a small decline in HRV on average during the worry induction. It is possible that change in amount of thought during worry occurs in tandem with changes in HRV during worry. It is beyond the scope of this study, but it would be helpful to know whether change in verbal thought tracks HRV during a worry episode. Are steeper declines in phasic HRV related to steeper declines in amount of verbal thought? Third, it is possible that the sample did not include enough of the kind of participants necessary to detect the predicted fan-shaped interaction (e.g., ample high and low HRV individuals who reported high levels of worry). Many participants were prescreened and invited to participate based on scores on worry and EC measures in order to overcome this limitation, but participants were not prescreened for HRV. It is unfeasible to screen for level of HRV due to the large number of participants it would require to yield an adequate sample.

Hypothesis 3: Worry/GAD symptom severity was expected to be associated with

higher top-down control capacity (i.e., resting HRV) among individuals who reported higher versus lower levels of EC. The correlation between worry and resting HRV is often low, and in the current study was virtually non-existent (r = -.05). As described earlier, this is consistent with the fact that there is substantially heterogeneity in resting HRV among worriers. Attempting to account for such heterogeneity, the current study predicted that high worriers would be more likely to exhibit high HRV if they reported high effortful control. That prediction, however, was not supported by the data. There is typically very weak to non-existent correlation between self-reported EC and resting HRV (e.g., Free, 2018, Vasey & Toh, 2018). It is possible that EC is tapping something more enduring than resting HRV; both are viewed as trait variables, but resting HRV is dependent on the experimental situation more than the EC. On the other hand, subjective reports of EC may be less valid measures of actual capacity.

Hypotheses 4 and 5: One of the primary aims of this study was to try to account for the heterogeneity in HRV during worry across past studies. As described earlier, the majority of prior studies have found that worry reduces phasic HRV, but some studies have found no such effect. In addition, there is often variability within samples. Inconsistent findings in the literature sometimes indicate the influence of a moderating variable. With that in mind, the current study predicted that resting HRV (hypothesis 4) or self-reported level of EC (hypothesis 5) would moderate change in phasic HRV during worry. For each hypothesis, three competing patterns of interactions were presented as possibilities. The results showed that HRV declined on average across the worry period, and that the rate of decline was unrelated to resting HRV or self-reported EC. Post-hoc analyses indicated that

the change in HRV during worry was also not moderated by worry/GAD symptom severity.

It is possible that failure to identify a moderator was due to lower than expected variation in slopes of HRV across the worry period. As shown in appendices 9 and 10, the differences in slopes were rather small (though the variances of the slope terms in the mixed models were statistically significant). It should be noted that the heterogeneity in *resting* HRV was consistent with other samples.

Assuming that change in HRV during worry is a function of initial capacity (i.e., resting HRV or self-reported EC), there are at least two reasons why such patterns did not emerge in the current sample. First, it is possible that a five-minute worry induction is too brief to deferentially deplete HRV. Indeed, the overall decline in HRV was rather small. It is conceivable that in a longer worry induction individuals with high HRV (or EC) would continue to experience little or no decline in HRV, whereas those with lower HRV would eventually experience steeper declines in HRV. Second, it is possible that the laboratory worry induction was not adequately representative of how worriers actually worry. For example, participants were interrupted every 30 seconds so that they could respond to questions about their experience (e.g., report percentage of verbal thoughts). It is possible that longer segments would allow worriers to engage in more typical catastrophic thinking that may be more depleting to HRV.

General Discussion: The primary aim of the current study was to account for heterogeneity in percentage of thoughts as well as phasic HRV during worry. Contrary to expectation, however, the proposed moderators were not supported by the data. Nevertheless, this study still makes a contribution to the literature, as it is the first to

examine change in HRV during a worry induction using a mixed models design. The results suggest that HRV declines linearly during a relatively short episode of worry.

Limitations: This study had several design limitations that may have made it more difficult to find support for the proposed hypotheses. As noted above, the worry induction may have been too short to differentially deplete phasic HRV. In addition, prompting participants to provide information about their worry every 30 seconds may have interrupted the worry process. Sampling characteristics may have also posed problems. For example, there might not have been enough participants with high or low HRV who also reported high worry. In addition, it is unknown how many participants, if any, met diagnostic criteria for generalized anxiety disorder. Finally, the study was limited by relying on a non-clinical convenience sample of college students, who may have higher cognitive control capacity compared with other populations.

Future Direction: Although many studies have sought to better understand the worry processes by inducing worry in a laboratory setting, no studies have been conducted on optimizing such worry inductions for research. For example, some studies solicit topics from participants and then ask them to worry about the topic in their "usual way" (e.g., Fisher & Newman, 2013; Karim et al., 2017), while other studies have provided worry-provoking statements for participants to read and think about (e.g., York, Borkovec, Vasey, & Stern, 1987). A number of other procedures have been used to induce worry, but no studies have been carried out to clarify which methods are most effective at eliciting worry. In addition, there is no empirical evidence indicating how long a worry induction should last. The current study found that HRV declined in a mostly linear fashion across the worry

period, but it may take more than five minutes of worrying to begin to see differences in HRV's rate of change at different levels of cognitive control capacity. A well-designed study evaluating change in HRV using a repeated measures design and longer worry inductions could potentially inform future research about when during a worry induction changes in phasic HRV begin to emerge.

Future studies should also compare verbal and imaginable worry inductions. Since Hirsch and colleagues (2012) have shown that only verbal worry depletes working memory capacity, perhaps a similar approach is needed to deplete HRV. This study let participants worry in their usual fashion, but perhaps the biggest reductions are seen when worriers with low EC or resting HRV are instructed to worry in words. On the other hand, those instructed to worry in imagery should exhibit no depletion. Since participants in the current studies worried in whatever way they normally do, this study's design was not optimal for seeing the effects of verbal worry on phasic HRV.

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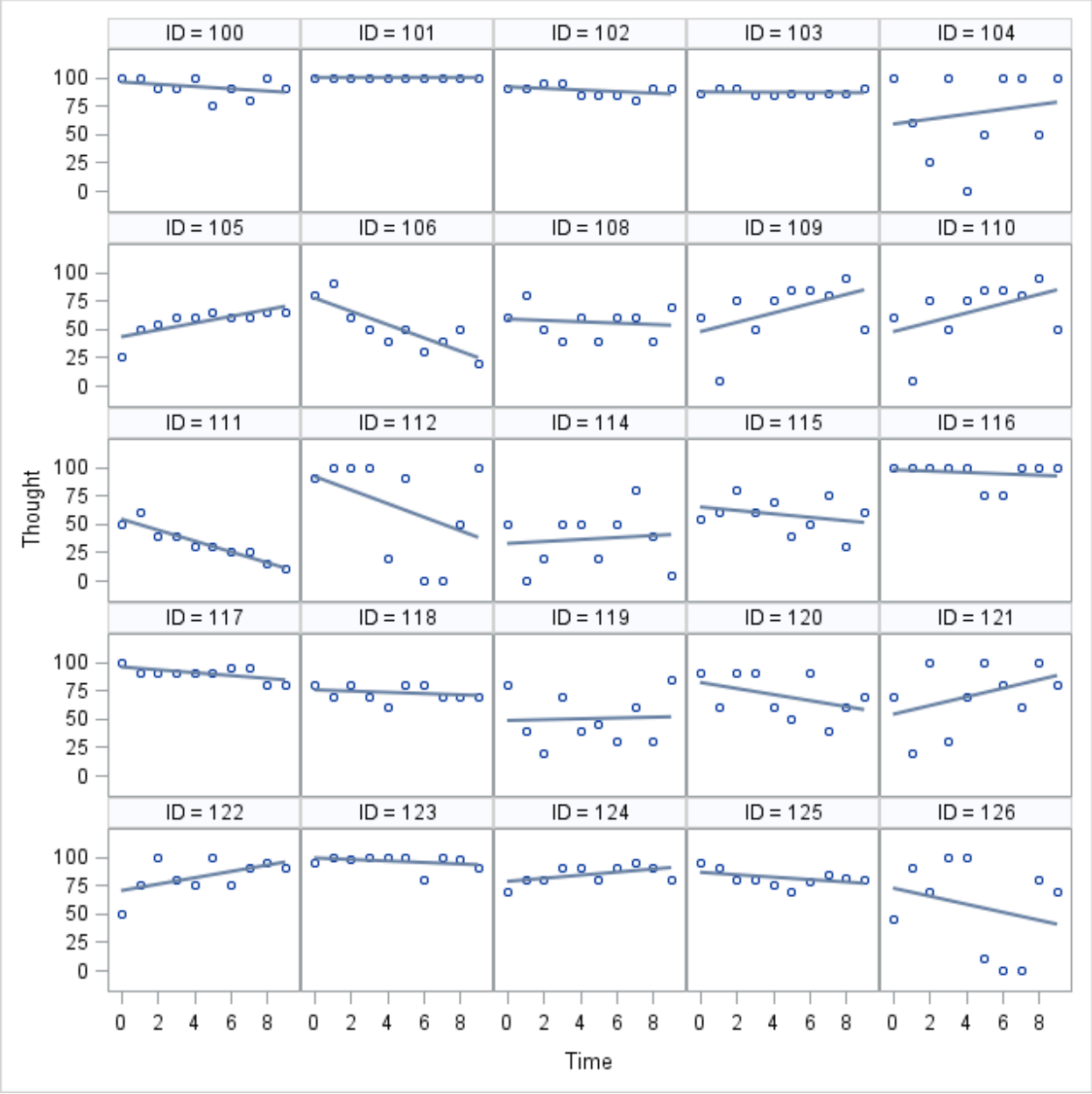
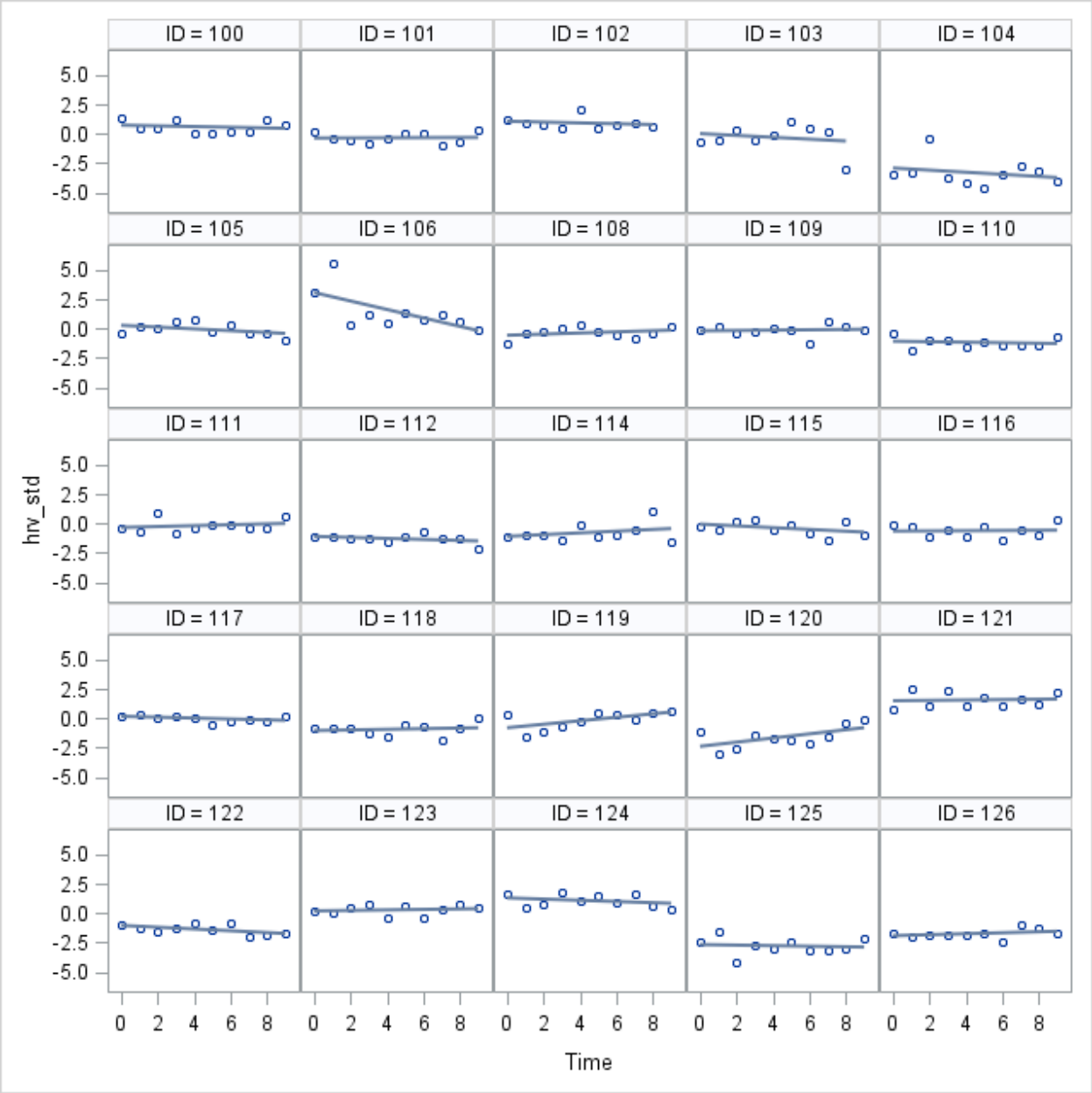
Appendices

Appendix A: Individual plots of change in percentage of thoughts during worry (first 25 cases)



Appendix B: Individual plots showing change in phasic HF-HRV (Z-Scores) during worry (first 25 cases)



Appendix C: Individual plots showing change in phasic RMSSD (Z-Scores) during worry (first 25 cases)