Testing the Cognitive Control Model of Pathological Worry Using Objective Measures of Cognitive Control and Autonomic Arousal

Thesis

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By

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Abstract

Pathological worry has been associated with both blunted and heightened autonomic arousal (AA). The role AA plays in the maintenance of pathological worry has been emphasized by two different models. Accounting for individuals who experience reduced AA during worry, the Cognitive Avoidance Model suggests that such a reduction in AA negatively reinforces the use of worry as an avoidant strategy. For those who experience heightened AA during worry, the Contrast Avoidance Model suggests that generating and maintaining elevated AA protects against sudden shifts in emotions, which some individuals find aversive. In turn, worry is negatively reinforced. Neither model alone can account for the well-documented heterogeneity in level of AA among pathological worriers. A new, integrative model posits that individual differences in effortful control (EC) can account for such heterogeneity. 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.

Initial tests of that model have yielded promising results, but they have so far exclusively relied on subjective EC and AA. The current study sought to extend prior findings by testing the model using mean heart rate (HR) as an objective measure of AA, and an index of vagally-mediated heart rate variability (HRV) as an objective measure of EC. We did so in a large, non-clinical sample of undergraduate students

(N = 286). As predicted, worry and GAD symptom severity interacted with EC in prediction of mean HR. In addition, EC was negatively associated with HR when worry/GAD symptoms were high. Results provide partial support for the model using HRV as a proxy for EC. This is the first study to test the Cognitive Control Model using objective measures of AA and EC; while results are promising, replication is clearly needed.

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Chapter 1: Introduction

Pathological worry is a common symptom of anxiety-related disorders and the hallmark of generalized anxiety disorder (GAD; American Psychiatric Association [APA], 2013). Several models of pathological worry have emphasized the role worry plays in either heightening or dampening autonomic arousal (AA) symptoms. For example, the Cognitive Avoidance Model (see Borkovec, Alcaine, & Behar, 2004), suggests that worry persists, in part, because it is characterized by verbal-linguistic processing of threat, which inhibits the AA symptoms that imaginally processing such threats would otherwise induce. On the other hand, Newman and Llera's (2011) Contrast Avoidance Model casts worry as a maladaptive strategy employed to foster and maintain *elevated* AA (and negative emotion), which spare worriers from aversive contrasts between euthymic and anxious states when anticipated threats actually occur. These opposing perspectives reflect the fact that there is substantial heterogeneity in AA among worriers, for which neither model alone can account. However, a recently proposed integrative model of pathological worry posits that heterogeneity in AA symptoms can be accounted for by individual differences in cognitive control capacity (Vasey, Chriki, & Toh, in press). While extant tests of this model suggest that worry's association with AA does indeed vary as a function of individual differences in cognitive control capacity, initial studies have relied exclusively on self-reported AA. Prior research has also

relied on self-report measures of cognitive control capacity. The current study sought to extend these findings by testing the primary tenant of this integrative model (i.e., pathological worry's relation to AA symptoms varies as a function of cognitive control capacity) using heart rate (HR) as an objectively measured index of AA. The model was also tested using resting heart rate variability (HRV) as an objectively measured index of cognitive control capacity.

As noted, there is substantial evidence of variability in AA among worriers. On one hand, many studies have shown that worry is not associated with heightened AA. In some samples, individuals with GAD resemble non-anxious controls on self-report measures of AA. For example, Leyfer, Ruberg, & Woodruff-borden (2006) found GAD patients were not significantly different from controls on the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) which 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 substantial number of studies have found that worry is instead related to *heightened* AA symptoms. For example, Wetherell & Gatz, (2005) found that BAI scores among older adults were significantly higher on average for individuals with GAD than controls. Consistent with such findings, GAD is frequently comorbid with panic attacks and panic disorder (Brown & Barlow, 1992), which is predominantly characterized by AA symptoms. For example, 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).

Such conflicting findings are not limited to self-reports of AA. Indeed, results from several studies have found that worry/GAD symptoms are positively associated with objective measures of AA. For example, worry has been related to heightened heart rate (e.g., Brosschot, Dijk, & Thayer, 2007), skin conductance (e.g., Kirschner, Hilbert, Hoyer, Lueken, & Beesdo-Baum, 2016), and salivary alpha amylase (e.g., Fisher & Newman, 2013). However, these results have not always replicated. Indeed, some studies have found no difference between worriers and controls in heart rate (e.g., Fisher & Newman, 2013; Pittig et al., 2013), skin conductance (e.g., Delgado et al., 2009), or salivary alpha amylase (e.g., Fisher, Granger, & Newman, 2010).

The first model to emphasize the importance of AA in the maintenance of GAD was the Cognitive Avoidance Model by Thomas Borkovec (see Borkovec et al., 2004). The Cognitive Avoidance Model casts worry, in part, as an avoidant strategy used to shield the worrier from aversive somatic symptoms of AA. Pivotal to that aspect of the theory is the manner in which individuals worry. Worry often takes the form of verbal-linguistic thought, but sometimes may take the form of mental imagery. Several studies have demonstrated that imaginal processing of fear-related stimuli often provokes physiological arousal, whereas verbal-linguistic processing does not (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, but they were not instructed on how to do so. Overall, participants tended to spontaneously use imaginal processing when instructed to *facilitate* emotional responding, and verbal-linguistic processing when instructed to *inhibit* it. The Cognitive Avoidance Model suggests that when worriers imagine a threatening event in the future, they shift attention to verbal processing and are therefore spared from the aversive AA symptoms that would otherwise occur. In turn, suppression of AA symptoms negatively reinforces verbal worry as an avoidant strategy.

Consistent with this view, evidence suggests that verbal-linguistic thought predominates over imagery during worry (Borkovec & Inz, 1990; Freeston, Dugas, & Ladouceur, 1996), especially among GAD patients (Hirsch, Hayes, Mathews, & Borkovec, 2012; Hoyer, Becker, & Roth, 2001) and high worriers (East & Watts, 1994). Moreover, during relaxation, GAD patients report significantly more verbal thought and less imagery than controls (Borkovec & Inz, 1990). Thus, it is possible that heterogeneity in AA can be accounted for by heterogeneity in amount of verbal thought during worry.

One limitation of the Cognitive Avoidance Model is that it is unable to account for the subset of individuals for whom worry is instead associated with heightened symptoms of AA. Similarly, it is unable to account for the heterogeneity in the amount of verbal worry across worriers. A recent model by Newman and Llera (2011) provides a plausible explanation for how worry is reinforced for such individuals. Drawing on early findings in cognitive psychology and the Affective

Contrast Theory (e.g., Bacon, Rood, & Washburn, 1914), the Contrast Avoidance Model casts worry as a maladaptive avoidant strategy used to circumvent sudden negative contrasts between emotional states. When individuals perceive an unanticipated threat in the environment, the contrast between euthymic and anxious states is sometimes distressing. The Contrast Avoidance Model suggests that persistent worry primarily serves to *maintain* heightened AA symptoms and negative emotionality in order to reduce the magnitude of such aversive contrasts. If AA is already elevated when a threat is detected, there is little room for greater physiological and emotional responding. Thus, whereas the Cognitive Avoidance Model suggests that worry facilitates avoidance of AA, the Contrast Avoidance Model posits that worry serves to heighten AA, thereby restricting the range of emotional contrasts.

The Contrast Avoidance Model has made an important contribution to understanding a possible path for worry's maintenance in the subset of individuals for whom worry is an autonomically arousing process. However, like Borkovec's Cognitive Avoidance Model, the Contrast Avoidance Model is limited by its inability to account for the well-documented heterogeneity in AA. It appears that for some, worry is a verbal process that reduces AA symptoms, and for others, worry is an imaginal process that heightens AA symptoms. Investigation of factors that underpin the predominance of either thought or imaginal processing may lead to better understanding of how worry's modulation of AA symptoms contributes to maintenance of GAD.

A recent model of pathological worry and GAD offers a compelling explanation for the heterogeneity in AA symptoms among worriers and integrates these competing models (Vasey et al., in press). This Cognitive Control Model suggests that heterogeneity in AA symptoms among pathological worriers can be accounted for by individual differences in cognitive control capacity. Foundational to this model is the well-supported view that shifting attention from imaginal to verbal processing is an effortful maneuver that requires adequate cognitive resources (e.g., Mathews, 1990). Therefore, the Cognitive Control Model posits that worriers with adequate top-down control capacity maladaptively use that capacity to constrain worry to verbal thought, suppressing AA, in keeping with the Cognitive Avoidance Model. Individuals unable to successfully shift attention to or maintain verbal thought are instead left to process threat imaginally, and, in turn, experience AA consistent with the Contrast Avoidance Model.

Such an integrative model seems at odds with the view that worry and GAD is characterized by deficits in cognitive control capacity (Eysenck, Derakshan, Santos, & Calvo, 2007). One construct thought to reflect such capacity is the temperament dimension of effortful control (EC), which broadly encompasses attentional, inhibitory, and activation control (Rothbart, 2007). Indeed, poor self-regulatory capacity, especially diminished attentional control (AC), appears to be in line with the uncontrollable nature of pathological worry. Consistent with that view, some studies have found that self-reported AC is lower in GAD patients than in non-anxious controls (e.g., Armstrong, Zald, & Olatunji, 2011; Borkovec, Robinson, &

Pruzinsky, 1983; Moradi, Fata, Abhari, & Abbasi, 2014) and that AC is negatively correlated with GAD symptoms (e.g., Armstrong et al., 2011; Olatunji, Ciesielski, Armstrong, Zhao, & Zald, 2011). In addition, evidence suggests that worry is associated with reduced cognitive control (e.g., Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014; Stefanopoulou, Hirsch, Hayes, & Adlam, 2014).

While some studies have found deficits in cognitive control capacity among worriers, it is important to note that such deficits reflect differences relative to controls *on average.* There is often substantial variability in cognitive control capacity among pathological worriers within samples. In addition, these deficits have not always been found. For example, some studies have found non-significant negative associations between worry/GAD and AC (e.g., Bienvenu et al., 2004), while others have found weak, *positive* associations between worry at conscientiousness (Rosellini & Brown, 2011), a construct related to EC.

Heterogeneity in cognitive control capacity among worriers is not limited to self-report; performance-based measures have also yielded conflicting findings. For examples, GAD patients show deficits on neuropsychological tests compared with controls (Gualtieri & Morgan, 2008). 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 from angry and fearful faces than non-anxious controls.

Evidence for heterogeneity in cognitive control capacity in worry and GAD can also be found in neuroimaging and psychophysiological studies. Some studies have found reduced activation of the dorsal anterior cingulate cortex in GAD patients (e.g., Blair et al., 2012), as well as reduced frontal cortical thickness (Veronese et al., 2015); these areas are thought to largely underpin EC. However, other studies have found that worry is moderately to strongly *positively* correlated with volume of several regions of the prefrontal cortex (Mohlman et al., 2009), and also positively correlated with connectivity between the amygdala and dorsolateral (dl)PFC (e.g., Etkin, Prater, Schatzberg, Menon, & Greicius, 2009), a region thought to play a role in self-regulation (e.g., Banich et al., 2000; MacDonald, Cohen, Stenger, & Carter, 2000). Individual differences in cognitive and neural indices of self-regulatory capacity in GAD and worry are noteworthy because such differences appear to be related to individual differences in AA. For example, Beaudreau and O'Hara (2009) found that scores on the BAI were negatively correlated with performance on executive function tasks. Etkin et al. (2009) found that the strength of functional connectivity between the dlPFC and amygdala in GAD patients was also negatively correlated with scores on the BAI.

As predicted by the Cognitive Control Model, individual differences in cognitive control capacity appear to moderate the relation between worry and AA symptoms. In an initial test of the model (Vasey et al., in press), scores on a measure of GAD symptom severity interacted with levels of EC to predict AA symptoms. Among high worriers, those with relatively low EC reported greater AA symptoms

than those with relatively higher EC. In keeping with the view that greater EC permits constraining worry to verbal processing, Toh and Vasey (2016) found that scores on the GADQ-IV interacted with level of EC in prediction of percent of verbal thoughts during worry. Among high worriers, those with higher EC reported more verbal thoughts than those with lower EC. Moreover, worry's relation to AA symptoms was mediated by percentage of thoughts during worry, but the strength of that indirect path varied as a function of individual differences in EC. Thus, early tests of the model have provided evidence consistent with the view that cognitive control capacity plays an important role in determining the extent to which an individual engages in verbal processing during worry, which in turn accounts for the heterogeneity in AA symptoms among individuals with GAD.

One limitation of the evidence supporting this integrative model is that prior studies have exclusively relied on self-reported AA. The current study sought to extend prior findings by testing the model using mean resting HR as an objective measure of AA, a frequently used index of such arousal (e.g., Gonçalves et al., 2015). Another limitation is that prior studies have also relied exclusively on self-reported levels of EC. The current study sought to test the moderating effects EC with an objective index of cognitive control capacity. Resting vagally-mediated HRV appears to be such an index. HRV is a measure of the variation of beat-to-beat intervals. Variability in resting heart rate is largely mediated by the vagus nerve, and is thought to reflect a flexible interplay between sympathetic and parasympathetic influences on the heart (Thayer & Lane, 2009). Evidence suggests that high HRV is a

reflection of parasympathetic predominance, which is achieved through top-down inhibitory control over sympathetic pathways.

Several studies have shown 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 prefrontal cortex (PFC) that are thought to play a major role in the neural network underpinning cognitive control capacity (see Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). HRV is also related to performance on executive function tasks (e.g., Hansen, Johnsen, & Thayer, 2003; Ramírez, Ortega, & Reyes Del Paso, 2015). For example, 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 results have not always replicated, HRV is increasingly viewed as a biomarker of cognitive control capacity.

It is widely believed that pathological worry is associated with autonomic rigidity and, in turn, low HRV (Chalmers, Quintana, Abbott, & Kemp, 2014; e.g., Tully, Cosh, & Baune, 2013). Consistent with that view, several studies have found that low resting HRV is associated with trait worry (Chalmers et al., 2014; Thayer, Friedman, & Borkovec, 1996) and state worry (Brosschot et al., 2007; Pieper,

Brosschot, van der Leeden, & Thayer, 2010). In addition, some studies have found that HRV is lower among patients with GAD than non-anxious controls (e.g., Pittig et al., 2013). Mirroring the findings of worry's relation to AA symptoms, however, there is actually substantial heterogeneity in HRV among worriers. It is important to note that the findings above reflect differences in HRV between group means. Yet, among those samples, there is substantial variability in HRV among worriers, albeit lower on average compared with non-worriers. Indeed, there is often a wide range of HRV scores among worriers; many who experience severe pathological worry also have relatively high resting HRV (e.g., Mankus, Aldao, Kerns, Mayville, & Mennin, 2013). In addition, worry's relation to low HRV has not always been found (e.g., Hammel et al., 2011; Kollai & Kollai, 1992). In fact, in one sample, high worriers had significantly *higher* HRV than non-anxious controls (Davis, Montgomery, & Wilson, 2002). In addition, experimentally induced worry has been shown to lead to lower resting HRV in some samples (e.g., Delgado et al., 2009; Fisher & Newman, 2013; Hofmann et al., 2005; Thayer et al., 1996), but not others (e.g., Knepp & Friedman, 2008; Lyonfields, Borkovec, & Thayer, 1995). Taken together, heterogeneity in HRV among worriers is evident within and between samples.

Insofar as HRV is an index of cognitive control capacity, heterogeneity in HRV is entirely consistent with Cognitive Control Model. Based on that model, higher resting HRV should permit individuals to effortfully suppress imaginal processing of threating stimuli during worry. Thus, similar to EC, it should moderate worry's

relation to AA symptoms such that worriers with high HRV should experience fewer AA symptoms. The current study tested this hypothesis in a sample of college students.

In summary, the current study tested the following hypothesis:

Hypothesis 1: Worry/GAD symptom severity interacts with EC in prediction of self-reported AA. Specifically, worry and GAD symptom severity are expected to be more positively associated with AA symptoms when EC is lower compared with when EC is higher. In addition, EC is expected to be negatively associated with AA symptoms among those high in worry/GAD symptom severity.

Hypothesis 2: Worry/GAD symptom severity interacts with EC in prediction of resting mean HR. Specifically, worry and GAD symptom severity are expected to be more positively associated with mean HR when EC is lower compared with when EC is higher. In addition, EC is expected to be negatively associated with mean HR those high in worry/GAD symptom severity.

Hypothesis 3: Worry/GAD symptom severity interacts with resting HRV in prediction of self-reported AA. Specifically, worry and GAD symptom severity are expected to be more positively associated with AA symptoms when HRV is lower compared with when HRV is higher. In addition, HRV is expected to be negatively associated with AA symptoms those high in worry/GAD symptom severity.

Chapter 2: Methods

Sample

The sample comprised 362 undergraduate students taking Introduction to Psychology at The Ohio State University. Students enrolled in the class participated in research for course credit through the psychology department's Research Experience Program (REP). A description of the current study was posted on the REP website, where students over the age of 18 could enroll on their own. In addition, at the beginning of the semester, students had the opportunity to earn extra credit by completing a battery of prescreening questionnaires that included the Penn State Worry Questionnaires (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), which is described below. Students with a PSWQ score of 56 or higher were invited by email to participate. In a large, non-clinical college sample (Ruscio, 2002), a PSWQ score of 56 or greater represented 28% of the sample and captured the majority of participants who met DSM-IV diagnostic criteria for GAD. A total of 192 students received invitations. Those who did not respond received a second invitation two weeks later. Thirty-eight students enrolled in the study after receiving an invitation.

Procedure

Participation in the study entailed completing two sessions. The first was an online session in which participants completed a battery of self-report questionnaires. Upon enrollment, participants were emailed a unique link to SurveyMonkey, a secure online questionnaire website, where they provided informed consent and completed the questionnaires. The demographics questionnaire (described below) was presented first, and the remaining 15 in random order. The online session lasted approximately 30 minutes, for which participants received 0.5 hours of REP credit.

The second session was a one-hour in-person laboratory session. Upon arrival, participants were given a brief verbal description of the tasks they would be completing, and asked to sign a paper copy of the consent form. Next, participants were fitted with three electrodes to measure electrocardiogram (ECG) signal and asked to sit quietly alone in the experiment room for five minutes while baseline heart rate data were collected. After the resting period, participants continued the laboratory session by completing computer tasks and questionnaires as part of another study. For the current study, resting heart rate was the only data used from the laboratory session. Students received one hour of REP credit upon completion of the laboratory session.

Material

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

GAD diagnostic and symptom measures.

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). As shown in Table 1, the measure had good internal consistence in the current sample (Cronbach's alpha was .92).

***Generalized Anxiety Disorders Questionnaire IV (GADQ-IV)*.** The GADQ-IV (Newman et al., 2002) is a 9-item self-report measure that assesses the presence and degree of distress and interference of GAD symptoms. Using yes/no questions, the GADQ-IV assesses pathological worry (e.g., Is your worry excessive in intensity, frequency, or amount of distress it causes?) and GAD-related symptoms in the past six months (e.g., difficulty falling/staying asleep, muscle tension, fatigue). The measure also includes a section in which respondents are asked to list their most frequent worry topics. Finally, respondents use a 9-point scale (0 = None, 8 = Very Severe) to indicate how much distress and interference worry and physical symptoms cause. The GADQ-IV was designed with a skip-out rule that instructs respondents to skip the last eight items if they do not endorse experiencing

pathological worry (item 6). Consistent with the recommendation of Newman and colleagues (2002), the skip-out rule was removed, allowing the measure to be used to assess GAD symptom severity as a continuous dimension. In the current study, GADQ-IV scores reflect all nine items, irrespective of endorsement of item 6. Scored this way, the measure had good internal consistency. As shown in Table 1, Cronbach's alpha was .89.

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). In the current sample, the measure had good internal consistency. As shown in Table 1, Cronbach's alpha was .94.

Autonomic arousal measures.

Depression, Anxiety, and Stress Scales (DASS). The DASS (Lovibond & Lovibond, 1995) is a 42 item self-report measure composed of three subscales (14 items each) used to measure level of depression, anxiety, and stress. The current study used the anxiety subscale (DASS-A), which includes items that tap AA symptoms such as dry mouth, perspiration, and increased heart rate. Respondents were 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). High scores indicate greater symptom severity. In the

current sample, the anxiety subscale had good internal consistency. As shown in Table 1, Cronbach's alpha was .90.

Resting heart rate. Heart rate was collected using a three-lead setup. The electrocardiogram (ECG) signal was sampled at 1000 Hz using a high-pass filter of .5 Hz and was passed through Mindware Technology's BioNex two-slot mainframe (Mindware Technology, Gahanna, OH) to a personal computer. The ECG signal was processed using Mindware Technology's HRV 2.51 software. This software provides automated R-peak detection and allows for visual inspection and editing of the ECG signal. Artifact correction was performed for any irregular and ectopic beats.

Cognitive capacity measures.

Adult Temperament Questionnaire Short Form – Effortful Control (ATQ-EC; Evans & Rothbart, 2007). The ATQ-EC includes 19 items of the full ATQ. The effortful control domain of the ATQ comprises the Attentional Control, Inhibitory Control, and Activation Control subscales. The ATQ-EC has deomonstrated strong internal consistency and good test-retest reliability (Evans & Rothbart, 2007). In the current sample, the measure had acceptable reliability. As shown in Table 1, Cronbach's alpha was .76.

High-frequency heart rate variability. To obtain estimates of HRV, the interbeat interval time series was extracted from the ECG data using Mindware Tchnology's HRV 2.51 software. It was then written to a single text file and analyzed using the Kubios HRV analysis package 2.2 (Tarvainen, Lipponen, & Karjalainen, 2009). The Kubios software program was used to compute autoregressive

estimates of high frequency power (HF-HRV; 0.15-.40 Hz, ms2). As noted, higher values of HF-HRV are thought to reflect stronger cognitive control capacity. Values of HF-HRV were natural log transformed to better approximate a normal distribution.

Analytic Strategy

Study hypothesis were testing using hierarchical linear regression analyses. With the exception of mean HR, all continuous variables in the regression models were transformed to Z-scores. Thus, parameter estimates of the effects of predictors are at the average levels of the other variables in the model. To address the primary research questions (Hypotheses 2 and 3), which sought to test the basic interaction underpinning the Cognitive Control Model using objective measures of AA symptoms and EC, we first needed to establish that worry/GAD symptoms interacted with EC in prediction of AA symptoms in the current sample (Hypthothesis 1). To do so, we conducted a regression analysis with WAQ and ATQ-EC entered in the first step, and their product in the second step, with DASS-A as the dependent variable. We then repeated that analysis with the other two measures of GAD symptoms: GADQ-IV and PSWQ. We examined each model in three ways. First, we tested whether worry's relation to DASS-A differed as a function ATQ-EC scores (i.e., significant interaction). Second, because we were primarily interested in those with high worry/GAD symptoms, we examined EC's effect on DASS-A among high worriers by testing the simple slopes of ATQ-EC predicting DASS-A at the 90th percentile of worry/GAD. Finally, interactions were probed to identify regions of

significance using PROCESS, a statistical tool for SPSS that includes features for estimating and probing interactions at multiple levels of the moderator (Hayes, 2013). Specifically, we examined simple slopes of the predictors' effects on the dependent variable, which we illustrated at the 90th and 10th percentiles of the moderator.

To test hypothesis 2, we examined whether worry/GAD symptoms interacted with ATQ-EC to predict mean resting HR. As with Hypothesis 1, we examined the interaction term, simple slopes of ATQ-EC at the 90th percentile of worry, and the regions of significance for each model. As noted above, resting HR is influenced by sympathetic and parasympathetic inputs. To narrow resting HR to an index of sympathetically-mediated AA, we partialed out parasympathetic influence by statistically controlling for resting HF-HRV. Thus, estimates of ATQ-EC and worry (and their product term) reflect their relation to the variance in mean HR that is unaccounted for by HF-HRV. Similar to Hypothesis 1, we examined the interaction terms, simple slopes of HF-HRV at the 90th percentile of worry, and the regions of significance for each model.

To test hypothesis 3, we examined whether worry/GAD symptoms interacted with HF-HRV to predict DASS-A. Mirroring hypotheses 1 and 2, we examined the interaction terms, simple slopes of HF-HRV at the 90th percentile of worry, and the regions of significance for each model.

Chapter 3: Results

Data from 286 of 362 participants were analyzed. A total of 76 participants were excluded from the study. Data from 15 participants were excluded due to repetitive patterns of responding on questionnaires, and data from 4 participants were excluded because they spent less than 10 minutes completing the online questionnaires, raising doubt about their validity. Data from 57 participants were excluded because of missing data due to failure to complete both study sessions (N = 52), or incomplete or missing questionnaire data (N = 5). Missing items on a questionnaire were replaced with the mean of that questionnaire in cases in which less than 25% of items were missing. If more than 25% of items were missing, the questionnaire was not scored and the participant was excluded. Mean substitution was not used when calculating Cronbach's alpha, as doing so artificially inflates estimates of internal consistency.

Excluded participants were compared with included participants on all measures for which data were available. Independent samples t-tests revealed a significant difference between the two groups on scores on the ATQ-EC. Excluded participants had lower scores on the ATQ-EC (M = 78.8) compared with included participants (M = 83.0), $t(329) = 2.22$, $p = .03$. The two groups did not differ on any other variable.

The current sample was composed primarily of Caucasian (72%), Asian (12.6), and African American (8%) undergraduate students. Participant ages ranged from 18 to 59 years, with a mean age of 19.9 years (*SD* = 3.8 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. Self-report measures demonstrated acceptable internal consistency; α's ranged from .76 to .94. For the GADQ-IV, internal consistency estimates were calculated using all nine items, ignoring "skip out" instructions.

Table 1:

Descriptive Statistics

	M	SD	α	N (alpha)*
DASS-A	7.95	7.71	.90	279
ATQ-EC	82.75	13.10	.76	266
WAQ	39.31	18.40	.92	274
GADQ-IV	5.24	3.37	.89	276
PSWQ	50.36	14.38	.94	274
Heart rate	76.92	11.99		
RMSSD (ms)	45.45	27.16		
HF-HRV	6.56	1.13		

*Missing 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, GADQ-IV, and PSWQ were all significantly positively correlated with each other (*r*'s ranged

from .72 to .80), and each was significantly negatively correlated with ATQ-EC (r = -.39, -.31, and -.32, respectively). In addition, WAQ, GADQ-IV, and PSWQ were significantly positively correlated with DASS-Anxiety (r = .64, .57, and .47, respectively).

Table 2:
Zero order correlations

	1	2	3	4	5	6	7
1. DASS-A							
2. Mean HR	.10						
3. ATQ-EC	-.36**	-.04					
4. HF-HRV	-.07	-.58**	.01				
5. WAQ	.64	.06	-.39**	-.06			
6. GADQ-IV	.60	.03	-.37**	-.01	.85**		
7. PSWQ	.47	-.04	-.32**	.03	.72**	.79**	

Hypothesis 1: Worry/GAD symptoms x ATQ-EC predicting AA symptoms

WAQ x ATQ-EC predicting DASS-A. As shown in Table 3, regression analysis revealed a significant effect of WAQ (B = .59, $p < .001$) and ATQ-EC (B = -.14, $p < .01$) predicting DASS-A in Step 1. The addition of the WAQ x ATQ-EC term in Step 2 yielded a significant interaction ($p < .001$) 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 ATQ-EC was low (i.e., 10th percentile; B = .78, $p <$

.001) than when ATQ-EC was high (i.e., 90th percentile; B = .35 $p < .001$). Probing the region of significance of the interaction revealed that the simple slope for WAQ was significantly positive at all Z-score values of ATQ-EC below 2.22, which was 98% of observed values. As expected, examination of the simple slopes from the perspective of the effect of ATQ-EC on DASS-A, depicted in Figure 2, revealed that when WAQ scores were high (i.e., 90th percentile), ATQ-EC was significantly negatively correlated with DASS-A scores (B = -.38, $p < .001$).. Probing the region of significance of the interaction in this direction revealed that the simple slope of ATQ-EC was significantly negative at all Z-score values of WAQ above -.37, which was 62% of observed values.

Table 3:

Multiple regression analysis predicting DASS-A from WAQ and ATQ-EC

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.424	.424	<.001
Intercept	.00	.05		1.00			
WAQ	.59	.05	.54	<.001			
ATQ-EC	-.14	.05	-.13	<.01			
Step 2					.453	.029	<.001
Intercept	-.06	.05		.17			
WAQ	.58	.05	.54	<.001			
ATQ-EC	-.16	.05	-.14	<.01			
WAQ x ATQ-EC	-.17	.04	-.17	<.001			

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

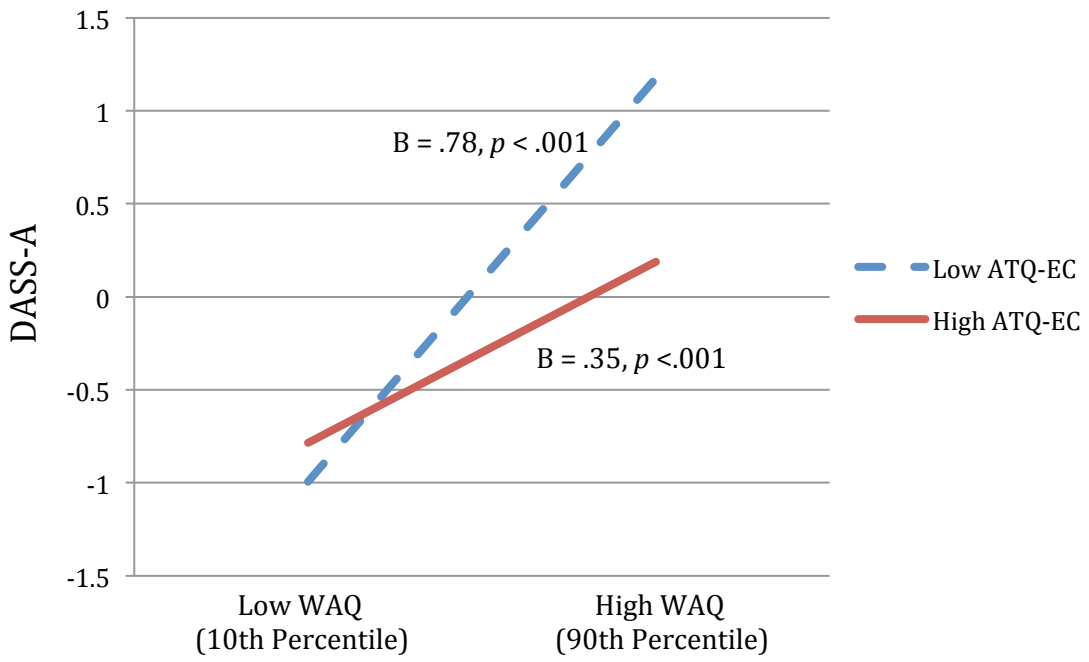
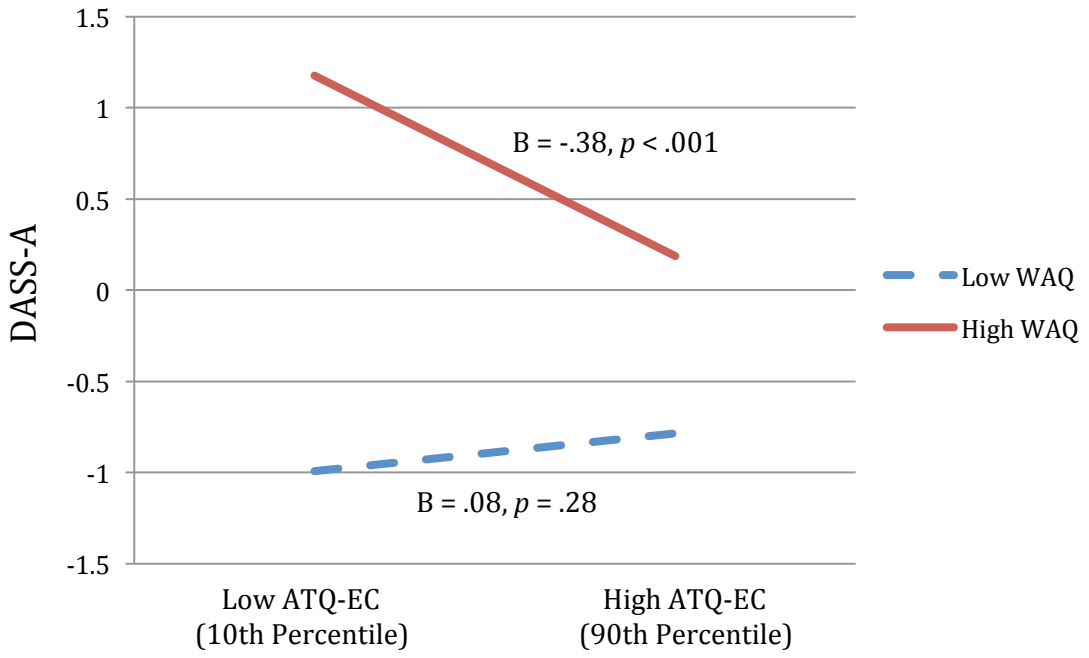


Figure 2. Predicting DASS-A from ATQ-EC as funciton of WAQ



GADQ-IV x ATQ-EC predicting DASS-A. As shown in Table 4, regression analysis revealed a significant effect of GADQ-IV (B = .54, $p < .001$) and ATQ-EC (B = -.16, $p < .01$) predicting DASS-A in Step 1. The addition of the GADQ-IV x ATQ-EC term in Step 2 yielded a significant interaction ($p < .01$) that was consistent with expectation. As depicted in Figure 3, higher GADQ-IV scores were more positively associated with scores on the DASS-A when ATQ-EC was low (i.e., 10th percentile; B = .70 $p < .001$) than when ATQ-EC was high (i.e., 90th percentile; B = .36, $p < .001$). Probing the region of significance of the interaction revealed that the simple slope for GADQ-IV was significant at all Z-score values of ATQ-EC below 2.31, which was 99% of observed values. Examination of the simple slopes from the perspective of the effect of ATQ-EC on DASS-A, depicted in Figure 4, revealed that when GADQ-IV scores were low (i.e., 10th percentile), ATQ-EC was unrelated to DASS-A scores (B = .00, $p = 1.0$). When GADQ-IV scores were high (i.e., 90th percentile), however, ATQ-EC was significantly negatively correlated with DASS-A scores (B = -.35, $p < .001$). Probing the region of significance of the interaction in this direction revealed that the simple slope of ATQ-EC was significantly negative at all Z-score values of GADQ-IV above -.47, which was 62% of observed values.

Table 4

Hierarchical multiple regression analysis predicting DASS-A from GADQ-IV and ATQ-EC

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.387	.387	<.001
Intercept	.00	.05		1.00			
GADQ-IV	.54	.05	.51	<.001			
ATQ-EC	-.16	.05	-.15	<.01			
Step 2					.404	.017	<.01
Intercept	-.05	.05		.33			
GADQ-IV	.54	.05	.50	<.001			
ATQ-EC	-.17	.05	-.16	<.01			
GADQ-IV x ATQ-EC	-.13	.05	-.13	<.01			

Figure 3. Predicting DASS-A from GADQ-IV as a function of ATQ-EC

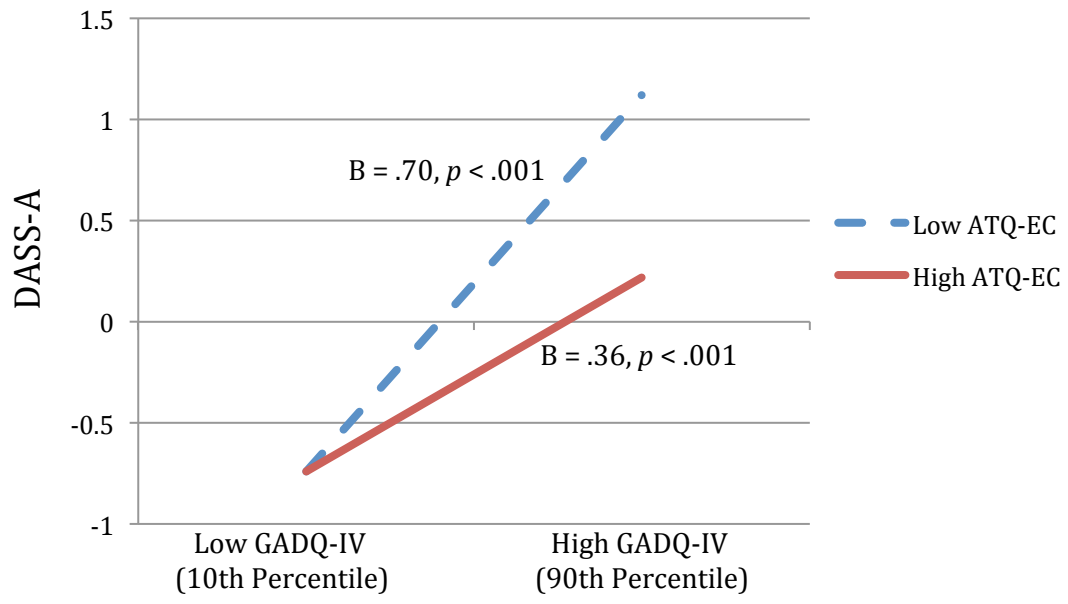
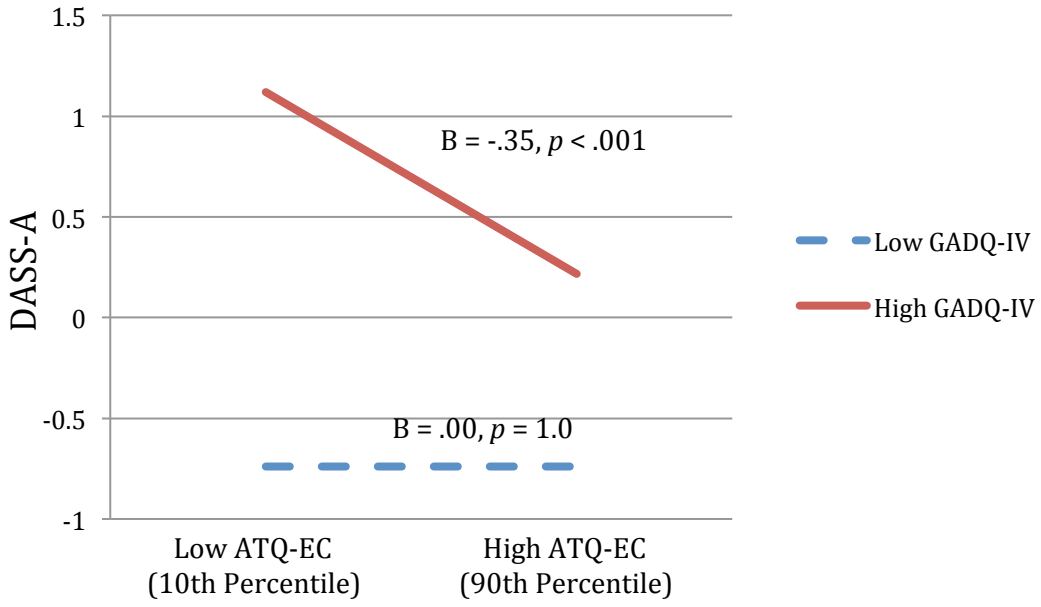


Figure 4. Predicting DASS-A from GADQ-IVas a function of ATQ-EC



PSWQ x ATQ-EC predicting DASS-A. As shown in Table 5, regression analysis revealed a significant effect of PSWQ (B = .39, $p < .001$) and ATQ-EC (B = -.24, $p < .001$) predicting DASS-A in Step 1. The addition of the PSWQ x ATQ-EC term in Step 2 produced the expected pattern of results, but the interaction was only marginally significant ($p = .07$). As depicted in Figure 5, higher PSWQ scores were more positively associated with scores on the DASS-A when ATQ-EC was low (i.e., 10th percentile; B = .50 $p < .001$) than when ATQ-EC was high (i.e., 90th percentile; B = .28, $p < .001$). Probing the region of significance of the interaction revealed that the simple slope for PSWQ was significantly positive at all Z-score values of ATQ-EC below than 2.15, which was 98% of observed values. As expected examination of

the simple slopes from the perspective of the effect of ATQ-EC on DASS-A, depicted in Figure 6, revealed that when PSWQ scores were high (i.e., 90th percentile), ATQ-EC was significantly negatively correlated with DASS-A scores (B = -.36, $p < .001$). Probing the region of significance of the interaction in this direction revealed that the simple slope of ATQ-EC was significantly negative at all Z-score values of PSWQ above -1.14, which was 84% of observed values.

Table 5

Hierarchical multiple regression analysis predicting DASS-A from PSWQ and ATQ-EC

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.269	.269	<.001
Intercept	.00	.05		1.00			
PSWQ	.39	.05	.37	<.001			
ATQ-EC	-.24	.05	-.23	<.001			
Step 2					.278	.009	.072
Intercept	-.03	.05		.61			
PSWQ	.40	.05	.38	<.001			
ATQ-EC	-.24	.05	-.23	<.001			
PSWQ x ATQ-EC	-.08	.05	-.09	.07			

Figure 5. Predicting DASS-A from PSWQ as a function of ATQ-EC

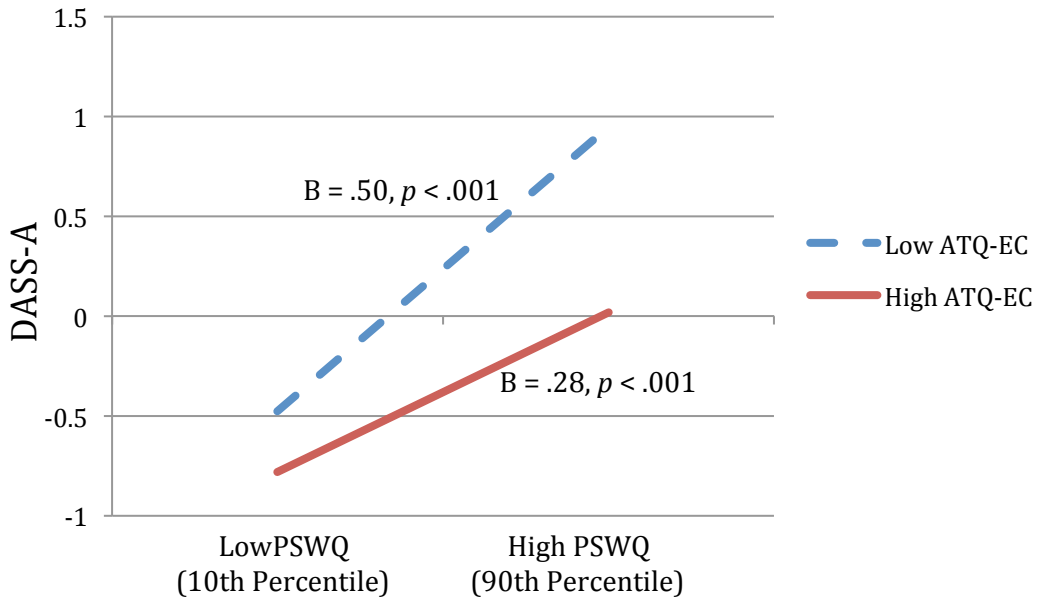
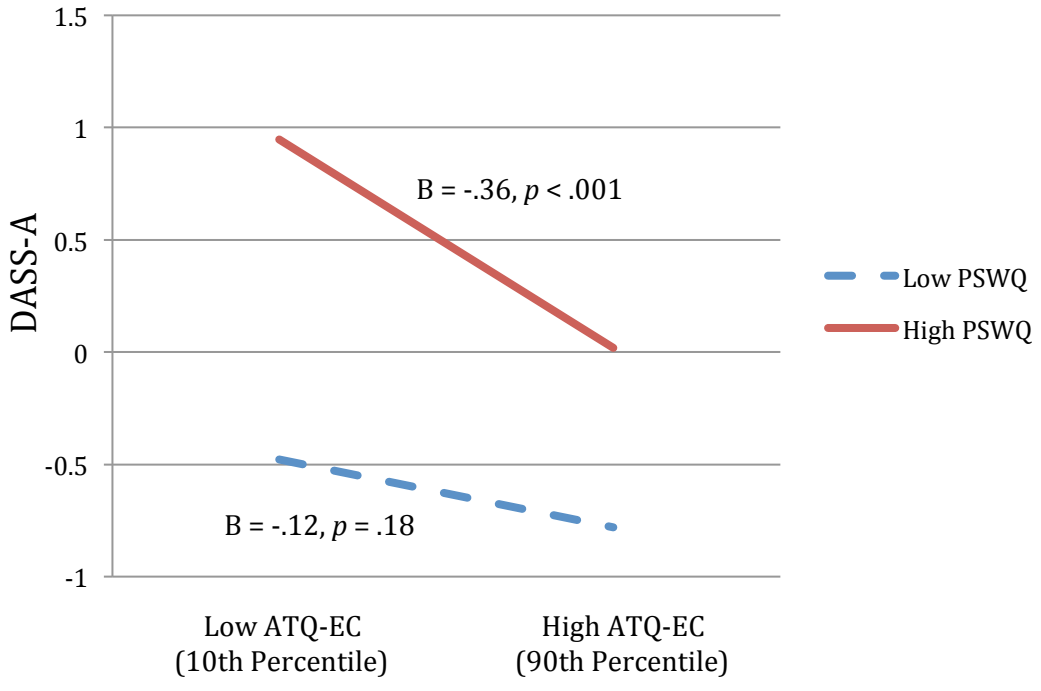


Figure 6. Predicting DASS-A from ATQ-EC as a function of PSWQ



Hypothesis 2: Worry/GAD symptoms x ATQ-EC predicting mean HR

Because Hypothesis 1 was supported and the pattern of results was consistent with expectation, there is sufficient evidence that the current sample provides an adequate context in which to test Hypothesis 2: Worry/GAD symptoms interact in prediction of mean HR.

WAQ x ATQ-EC predicting mean HR. As shown in Table 6, regression analysis in Step 1 revealed a significant effect of HF-HRV (B = -6.93, $p < .001$), but not WAQ or ATQ-EC. Addition of the WAQ x ATQ-EC term in Step 2 marginally improved R^2 ($p = .06$). As depicted in Figure 7, higher scores on the WAQ were not associated with mean HR when ATQ-EC scores were low (i.e., 10th percentile, B = 1.48, $p = .11$), or when ATQ-EC scores were high (i.e., 90th percentile, B = -1.28, $p = .21$). Probing the region of significance of the interaction revealed that the simple slope for WAQ was not significant at any observed Z-score value of ATQ-EC. Examination of the simple slopes from the perspective of the effect of ATQ-EC on DASS-A, depicted in Figure 8, revealed that when WAQ scores were high (i.e., 90th percentile), ATQ-EC was marginally negatively correlated with mean HR scores (B = -1.91, $p = .07$). Probing the region of significance of the interaction in this direction revealed that the simple slope of ATQ-EC was not significant at any level of WAQ.

Table 6

Hierarchical multiple regression analysis predicting mean HR from WAQ and ATQ-EC, controlling for HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.337	.337	<.001
Intercept	76.92	.58		<.001			
WAQ	.23	.63	.02	.72			
ATQ-EC	-.36	.63	-.03	.57			
HF-HRV	-6.93	.58	-.58	<.001			
Step 2					.345	.008	.063
Intercept	76.52	.62		<.001			
WAQ	.20	.63	.02	.75			
ATQ-EC	-.49	.63	-.04	.44			
HF-HRV	-6.94	.58	-.58	<.001			
WAQ x ATQ-EC	-1.06	.57	-.09	.06			

Figure 7. Predicting mean HR from WAQ as a function of ATQ-EC

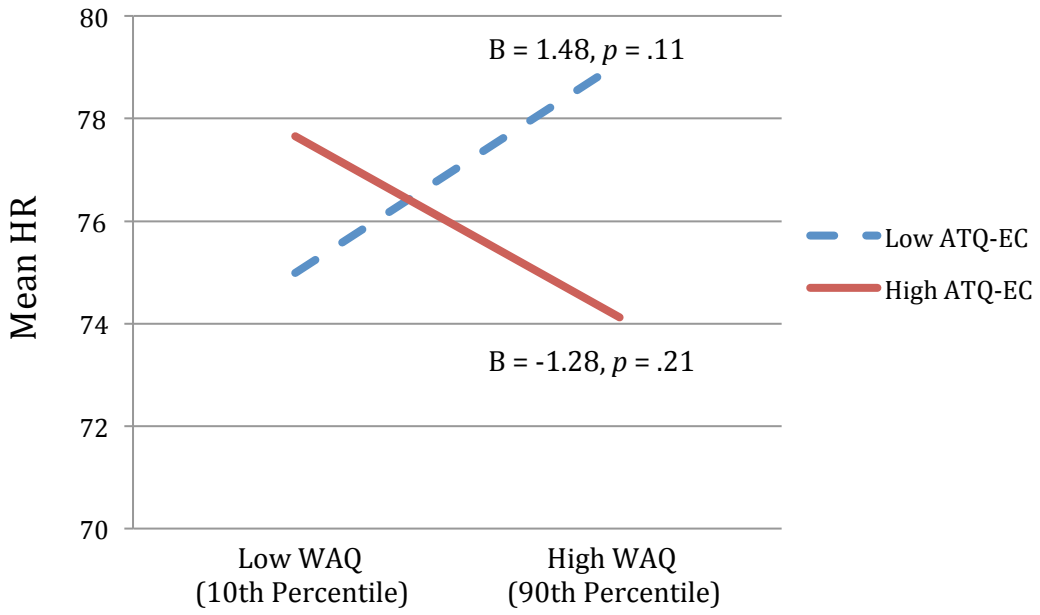
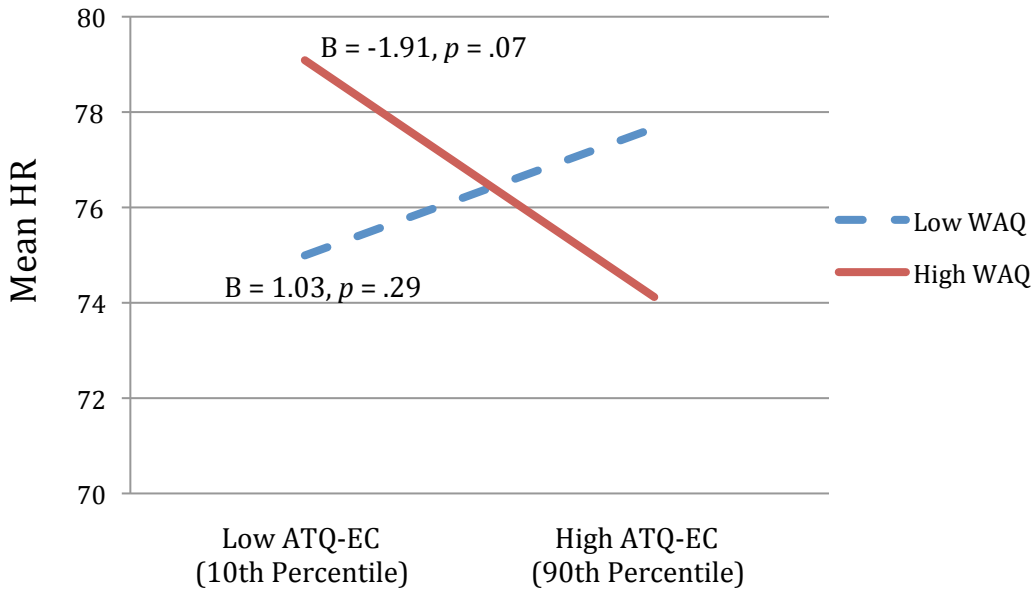


Figure 8. Predicting mean HR from ATQ-EC as a function of WAQ



GADQ-IV x ATQ-EC predicting mean HR. As shown in Table 7, regression analysis in Step 1 revealed a significant effect of HF-HRV (B = -6.94, $p < .001$), but not GADQ-IV or ATQ-EC. Addition of the GADQ-IV x ATQ-EC term in Step 2 significantly improved R^2 ($p = .02$). As depicted in Figure 9, higher GADQ-IV scores were marginally positively associated with mean HR when ATQ-EC scores were low (i.e., 10th percentile; B = 1.71, $p = .07$) and marginally negatively associated with mean HR when ATQ-EC scores were high (i.e., 90th percentile; B = -1.88, $p = .07$). Probing the region of significance of the interaction revealed that the simple slope for GADQ-IV was significantly positive for Z-score values of ATQ-EC below -1.46 (6% of observed values) and significantly negative for ATQ-EC scores above 1.83 (3% of observed values). Examination of the simple slopes from the perspective of the

effect of ATQ-EC on mean HR, depicted in Figure 10, revealed that when GADQ-IV were high (i.e., 90th percentile), ATQ-EC was significantly negatively correlated with mean HR (B = -2.38, *p* = .02). Probing the region of significance of the interaction in this direction revealed that the simple slope of ATQ-EC was significantly negative at all Z-score values of GADQ-IV above 1.48, which was 8% of observed values.

Table 7

Hierarchical multiple regression analysis predicting mean HR from GADQ-IV and ATQ-EC, controlling for HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.337	.337	<.001
Intercept	76.92	.58		<.001			
GADQ-IV	.09	.63	.01	.89			
ATQ-EC	-.42	.63	-.03	.51			
HF-HRV	-6.94	.58	-.58	<.001			
Step 2					.349	.012	.020
Intercept	76.52	.62		<.001			
GADQ-IV	.05	.62	.00	.94			
ATQ-EC	-.46	.62	-.04	.46			
HF-HRV	-6.93	.58	-.58	<.001			
GADQ-IV x ATQ-EC	-1.38	.59	-.11	.02			

Figure 9. Predicting mean HR from GADQ-IV as a function of ATQ-EC

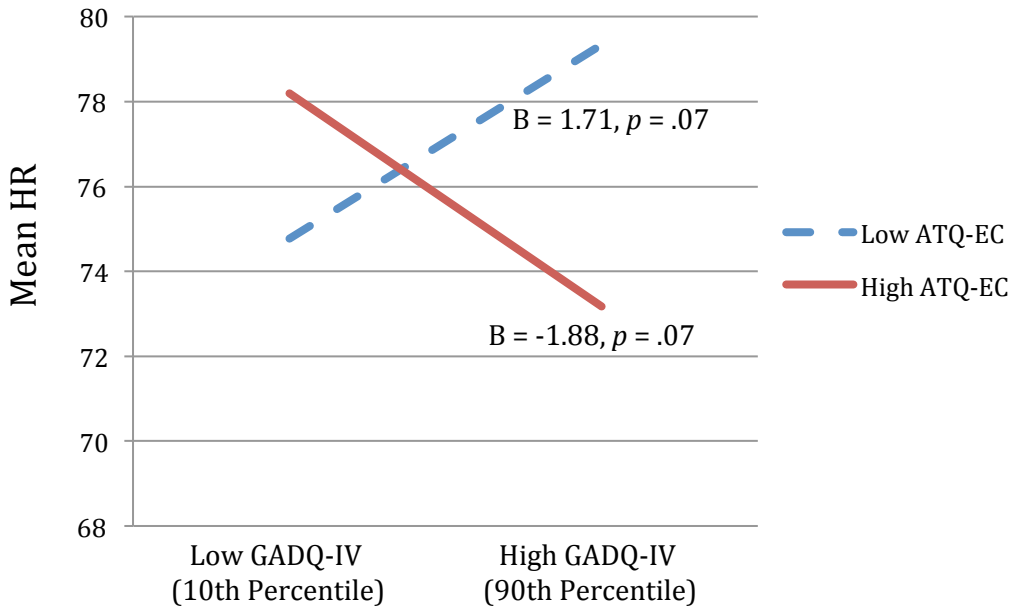
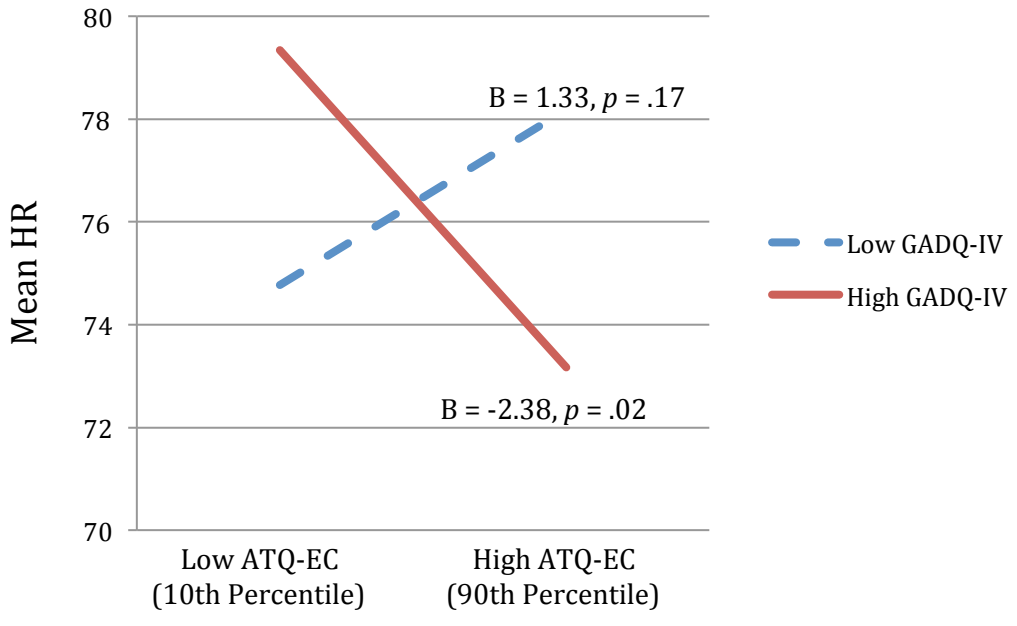


Figure 10. Predicting mean HR from ATQ-EC as a function of GADQ-IV



PSWQ x ATQ-EC predicting mean HR. As shown in Table 8, regression analysis in Step 1 revealed a significant effect of HF-HRV (B = -6.92, $p < .001$), but not PSWQ or ATQ-EC. Addition of the PSWQ x ATQ-EC term in Step 2 did not significantly improve R^2 ($p = .47$). Probing the region of significance of the interaction revealed that the simple slope for PSWQ was significant was not significant at any level of ATQ-EC. Examination of the simple slopes from the perspective of the effect of ATQ-EC on mean HR revealed that when PSWQ scores were high (i.e., 90th percentile) and low (i.e., 10th percentile), ATQ-EC was not significantly related to mean HR.

Table 8

Hierarchical multiple regression analysis predicting mean HR from PSWQ and ATQ-EC, controlling for HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.338	.338	<.001
Intercept	76.92	.58		<.001			
PSWQ	-.44	.61	-.03	.48			
ATQ-EC	-.57	.61	-.04	.34			
HF-HRV	-6.92	.58	-.58	<.001			
Step 2					.339	.001	.468
Intercept	76.80	.61		<.001			
PSWQ	-.40	.62	-.03	.52			
ATQ-EC	-.60	.61	-.05	.33			
HF-HRV	-.6.92	.58	-.58	<.001			
PSWQ x ATQ-EC	-.39	.54	-.04	.47			

Hypothesis 3: Worry/GAD symptoms x HRV predicting DASS-A

Because Hypothesis 1 was supported and the pattern of results was consistent with expectation, the current sample provides an adequate context in

which to test resting HRV as a moderator of the relation between worry/GAD symptoms and DASS-A.

WAQ x HF-HRV predicting DASS-A. As shown in Table 9, regression analysis in Step 1 revealed a significant effect of WAQ (B = .64, $p < .001$) but not HF-HRV. The addition of the WAQ x HF-HRV term in Step 2 did not significantly improve R^2 ($p = .27$). Probing the region of significance of the interaction revealed that the simple slope for WAQ was not significant for any observed Z-score values of HF-HRV. Examination of the simple slopes from the perspective of HF-HRV revealed that when WAQ scores were high (i.e., 90th percentile), HF-HRV was not significantly related to to DASS-A scores (B = -.11, $p = .17$).

Table 9

Hierarchical multiple regression analysis predicting DASS-A from WAQ and HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.410	.410	<.001
Intercept	-.00	.05		1.00			
WAQ	.64	.05	.64	<.001			
HF-HRV	-.04	.05	-.04	.41			
Step 2					.412	.002	.273
Intercept	.00	.05		.95			
WAQ	.64	.05	.64	<.001			
HF-HRV	-.03	.05	-.03	.49			
WAQ x HF-HRV	-.06	.05	-.05	.27			

GADQ-IV x HF-HRV predicting DASS-A. As shown in Table 10, regression analysis in Step 1 revealed a significant effect of GADQ-IV (B = .60, $p < .001$) but not

HF-HRV. The addition of the GADQ-IV x HF-HRV term in Step 2 marginally improved R^2 (p = .08). As depicted in Figure 11, higher GADQ-IV scores were positively associated with scores on the DASS-A when ATQ-EC was low (i.e., 10th percentile; B = .71 p < .001) and somewhat less so when ATQ-EC was high (i.e., 90th percentile; B = .48, p < .001), though the interaction was not significant. Probing the region of significance of the interaction revealed that the simple slope for GADQ-IV was not significant at any value of HF-HRV. Examination of the simple slopes from the perspective of the effect of HF-HRV on DASS-A, depicted in Figure 12, revealed that when GADQ-IV scores were high (i.e., 90th percentile), HF-HRV was significantly negatively correlated with DASS-A scores (B = -.19, p = .02). Probing the region of significance of the interaction in this direction revealed that the simple slope of HF-HRV was significantly negative at all Z-score values of GADQ-IV above .43, which was 37% of observed values.

Table 10

Hierarchical multiple regression analysis predicting DASS-A from GADQ-IV and HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.369	.369	<.001
Intercept	.00	.05		1.00			
GADQ-IV	.60	.05	.60	<.001			
HF-HRV	-.07	.05	-.07	.17			
Step 2					.376	.007	.077
Intercept	.00	.05		.98			
GADQ-IV	.60	.05	.60	<.001			
HF-HRV	-.06	.05	-.06	.19			
GADQ-IV x HF-HRV	-.09	.05	-.11	.08			

Figure 11. Predicting DASS-A from GADQ-IVas a function of HF-HRV

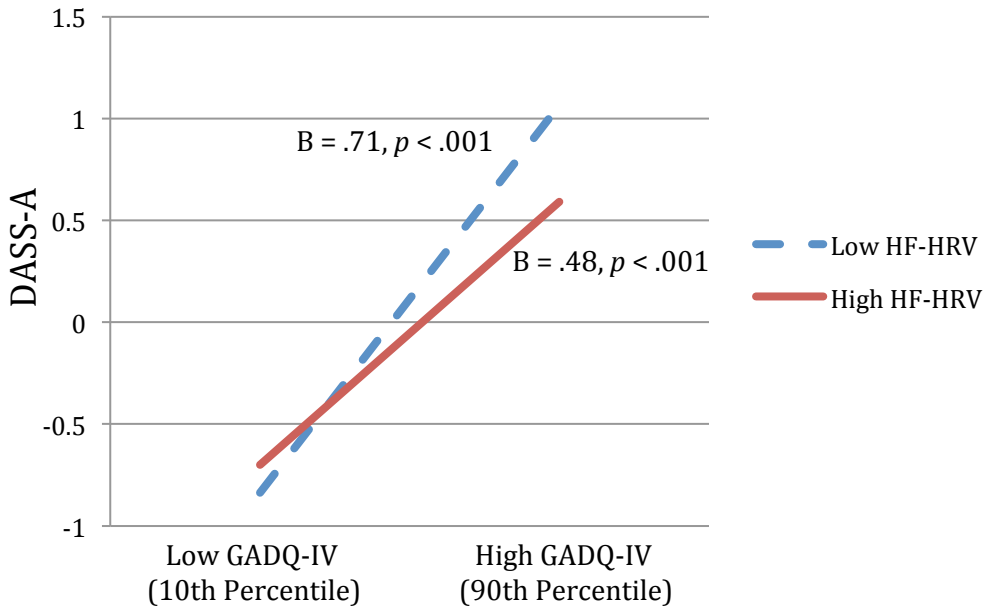
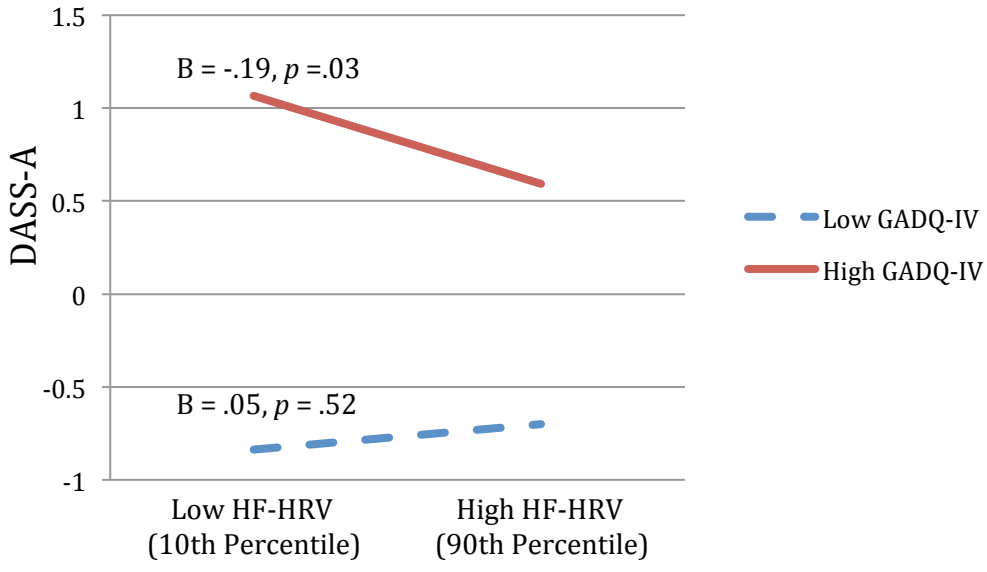


Figure 12. Predicting DASS-A from HF-HRV as a function of GADQ-IV



PSWQ x HF-HRV predicting DASS-A. As shown in Table 11, regression analysis in Step 1 revealed a significant effect of PSWQ (B = .47, $p < .001$) but not HF-HRV. The addition of the PSWQ x HF-HRV term in Step 2 did not significantly improve R^2 ($p = .26$). Probing the region of significance of the interaction revealed that the simple slope for PSWQ was not significant at any observed value of HF-HRV. Examination of the simple slopes from the perspective of the effect of HF-HRV on DASS-A revealed that for both low (i.e., 10th percentile) and high (i.e., 90th percentile) PSWQ scores, HF-HRV was not significantly related to to DASS-A scores.

Table 11

Hierarchical multiple regression analysis predicting DASS-A from PSWQ and HF-HRV

	B	SE	*sr*	*p*	R^2	ΔR^2	*p*
Step 1					.225	.225	<.001
Intercept	.00	.05		1.00			
PSWQ	.47	.05	.47	<.001			
HF-HRV	-.09	.05	-.19	.10			
Step 2					.229	.004	.256
Intercept	.00	.05		.97			
PSWQ	.47	.05	.47	<.001			
HF-HRV	-.09	.05	-.09	.11			
PSWQ x HF-HRV	-.06	.06	-.06	.26			

Chapter 4: Discussion

Accounting for the heterogeneity in level of AA has been an obstacle for models that emphasize AA's role as a maintaining factor for pathological worry. Although some evidence supports the theory that worry is negatively reinforced by its ability to dampen aversive AA, there is other evidence that supports a theory that casts worry in an opposite role. Indeed, for some, worry appears to heighten AA, and may be reinforced by attenuating the magnitude of aversive contrasts in mood. The current study sought to further test a new, integrative model of pathological worry and GAD (Vasey, Chriki, & Toh, in press), which posits that individual differences in cognitive control capacity can account for the heterogeneity in AA among those high in GAD symptoms. Initial tests of that model indicated that high level of GAD symptom severity is more strongly positively associated with elevated AA symptoms when EC is low than when EC is high. While promising, these studies have relied exclusively on self-reported AA (e.g., DASS-A) and self-reported levels of EC. As noted, however, there is substantial evidence showing that worriers also differ in *objectively* measured AA and EC.

After successfully replicating the basic interaction underpinning the Cognitive Control Model (Hypothesis 1), we tested the hypothesis that worry/GAD symptoms interact with EC to predict an objective measure of AA (Hypothesis 2), as they do with subjective measures. As predicted, worry interacted with EC to predict

mean baseline HR. Specifically, higher worry/GAD symptoms were less strongly positively associated with HR when EC was high than when EC was low.

The Cognitive Control Model suggests that higher levels of EC should permit individuals to more effectively constrain worry to a verbal mode of processing, thereby reducing activation of AA during worry. That is, such a pattern of results is consistent with the view that individuals high in GAD symptoms tended to worry even during the resting baseline period but the level of AA activated depended on the manner in which they did so. However, as processing mode (e.g., verbal, imaginal) was not assessed in the current study, that remains supposition. However, consistent with this view, Toh (2015) found that worry's relation to AA was mediated by percentage of verbal thoughts reported during a typical worry bout. Importantly, that mediation was moderated by level of EC. When worry was relatively high, higher EC was positively correlated with percentage of thoughts during worry, which, as expected, was related to lower AA symptoms. Lower EC, however, was related to fewer thoughts during worry, which was, in turn, related to higher self-reported AA. It is plausible that that model holds for objectively measured AA, but additional research is needed to confirm such a theory. Furthermore, future research should also test the alternative possibility that individuals high in GAD symptoms but high in EC may be less prone to worrying during baseline.

These results may seem surprising given the widely held view that GAD is characterized by heightened AA. As previously discussed, prior research has been

mixed in regard to heart rate and worry/GAD symptoms. Whereas some studies have found that those with GAD have higher mean HR than non-anxious controls, others have not. Taken together, it is clear that level of objectively measured AA is heterogeneous among those who experience pathological worry. The current study is the first to show that the Cognitive Control Model can account for such heterogeneity. Moreover, it now seems less likely that prior support for the Cognitive Control Model is limited to subjective, self-reports.

Caution toward self-report data may seem more warranted when its concordance with objectively measured data is low, as was the case in the current study. For example, the correlation between DASS-A scores and mean HR was weak (r = .10). It is important to recall, however, that the DASS-A reflects subjective AA over a one-week period, while mean HR reflects AA over a five-minute period. A low correlation between two markedly different time spans is not unexpected.

It is worth noting that caution toward self-report data may be especially *un*warranted in the context of the Cognitive Control Model, which posits that individuals with adequate cognitive control capacity misuse it to dampen AA. Indeed, subjective perception of AA appears to play a central role in worry becoming pathological. Insofar as it is distinct from objective AA, the role objective AA plays in the etiology and maintenance of pathological worry is still unclear. Nevertheless, the current study makes two important contributions. First, as noted above, it rules out the possibility that findings using subjective measures were due to reporting biases. And second, it positions the Cognitive Control Model as a

parsimonious theory that can account for heterogeneity in subjective *and* objective AA.

Prior tests of the Cognitive Control Model have also been limited by exclusive reliance on subjective measures of EC. The current study also sought to extend the literature by testing the model using vagally-mediated HRV as an objective proxy to EC (Hypothesis 3). As described above, resting HRV is increasingly viewed as an objective biomarker of cogitative control capacity, including EC. In the current study, HRV was significantly negatively correlated with AA when worry was higher, a pattern that mirrored the prior findings using self-reported EC. This is the first study to test the Cognitive Control Model using an objective measure of EC. Though effect sizes were not as robust as expected, the pattern of findings was in the predicted direction, which provides promising support.

As with hypothesis 2, these results may seem surprising given the widely held view that GAD is characterized by low HRV. Mirroring AA, findings regarding HRV are mixed; some studies report lower HRV among worriers, others find no difference. Not only was variability in HRV evident at all levels of worry in the current study, it accounted for some variability in AA symptoms, similar to that of self-reported EC. This finding makes it less likely that prior tests of the Cognitive Control Model reflect biases in self-reported EC. Similar to measures of AA, self-reported EC was not concordant with objectively measured EC. For example, ATQ-EC scores were unrelated to HRV (r = .01). This may be due to the fact that the ATQ-

EC does not specify to time period (asking respondents to indicate what is generally true for them), whereas HRV reflected a five-minute measurement period.

A particular strength of the current study is that it included three different measures of worry/GAD symptoms. The PSWQ, GADQ-IV, and WAQ are among the most common measures used in research to tap worry. Thus, their inclusion here allows the results to make contact with the extant literature. It is noteworthy that each of the measures yielded somewhat different results. Despite its wide use, the PSWQ performed the worst. It is possible that its relatively poor performance is due to the fact that it measures worry more broadly, whereas the GADQ-IV and WAQ specifically target features more indicative of *pathological* worry and GAD symptoms. The GADQ-IV and WAQ are both based on DSM-IV diagnostic criteria for GAD. As previously discussed, they differ in one important way. The GADQ-IV uses several dichotomous questions, whereas the WAQ solely uses continuously scored questions. We predicted that the WAQ would outperform the other measures because continuously scored items permit greater variability, which is an important component in detecting interactions. The WAQ produced modestly larger effect sizes in Hypotheses 1 and 2, but not 3. Though the pattern was similar with all measures, none of the models using the PSWQ produced statistically significant results.

Limitations.

Although this study has several strengths, the results should be considered in the context of several limitations. First, the sample likely included relatively few

individuals who were experiencing clinical levels of pathological worry. Our efforts to oversample for high worry fell short, as only 38 students enrolled via prescreening. Students who report symptoms of psychopathology represent a minority of those who complete prescreening questionnaires, and as a result, they often receive multiple invitations to participate research. As associations between EC and AA were strongest among high worriers, it is likely that relatively low representation of worriers limited power to detect moderation effects. Second, resting heart rate was the only objective measure of AA. As noted, heterogeneity among worriers has been documented using several other measures of AA. Finally, the current study used only one objective measure of EC.

Future directions

In the two hypotheses central to the current study (2 and 3), objective measures (e.g., HRV, HR) were collected at a different occasion and represented different periods of time than subjective measures (e.g., WAQ, GADQ-IV, ATQ-EC, DASS-A). A natural next step would be to replicate these findings in studies designed to collect data during the same time period. For example, worry inductions could be employed in the lab while objective AA is measured. In addition, EC could be objectively measured in the same session. Alternatively, advances in ambulatory heart rate monitors have made it possible to collect heart rate and HRV data outside of the lab over long periods of time. Thus, future studies could feasibly collect HR and HRV data that corresponds to questionnaire time

periods. Such a study design would also increase the ecological validity of the findings.

The Cognitive Control Model posits that higher EC permits verbal-linguistic worry, which, in turn, reduces AA. Such a moderated mediation model has been tested (Toh and Vasey, 2016), but it too relied on subjective reports of AA and EC. It is reasonable to expect that a similar causal chain can explain the present study's findings, but additional research is needed to fill in this gap. Future studies should also include additional objective measures of AA, such as skin conductance, salivary alpha amylase, or cortisol levels. Likewise, findings can be extended by testing the Cognitive Control Model using additional objective measures of EC, such as performance tasks related to executive function. Nevertheless, although replication is undoubtedly needed, the current study provides promising support for the models ability to account for heterogeneity in objectively measured AA among pathological worriers.

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