The Role of Verbal Worry in Cognitive Control and Anxious Arousal in Worry and Generalized Anxiety: A Replication and an Extension

DISSERTATION

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Abstract

While some studies find worry and GAD to be associated with low autonomic arousal (AA) symptoms, others find the contrary. Two theoretical models, the Cognitive Avoidance Model (Borkovec, Alcaine, & Behar, 2004) and the Contrast Avoidance Model (Newman & Llera, 2011), one or the other set of findings. Yet, neither theory can account for the full range of AA symptoms linked to worry and GAD. Vasey, Chriki, and Toh (2017) offered initial support for an integrative model in which effortful control (EC) acts as a moderator that may explain the heterogeneous nature of AA symptoms in worry and GAD. A second study (Toh & Vasey, 2017) provided further support for that model and provided preliminary evidence suggesting that the ability to constrain worry to a verbal mode of processing may be the mechanism by which EC impacts AA symptoms.

The current study sought to provide a further replication and extension for the basic interaction between GAD symptom severity and EC in predicting AA symptoms and percentage of verbal thoughts during worry. A further goal was to extend previous global self-report findings through use of a mentation sampling task to assess percentage of verbal worry, objective measures of AA (i.e., heart rate [HR]), and performance-based measures of EC. A sample of 198 individuals in the Psychology 1100 at The Ohio State University completed questionnaire as well as psychophysiological and behavioral measures. Hierarchical linear regression analyses were conducted and interactions were probed using PROCESS, an SPSS tool (Hayes, 2012), while multilevel modeling was used to examine growth curves for AA, percentage of verbal thoughts, and HR during the worry task.

Results showed that the basic interaction between GAD symptom severity and self-reported EC was successfully replicated. However, performance-based EC measures produced mixed results. Further, as expected, the interactions between GAD symptom severity and self-reported and performance-based EC predicted percentage of verbal thoughts during worry. However, the results regarding objectively measured AA did not support the Cognitive Control Model. Implications and future directions are discussed.

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I dedicate this dissertation to my parents, whose sacrifices have brought me to where I am; my sister, who paved the way first; and my wife Annie, who walked alongside me even when I walked in circles.

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Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction

Excessive and uncontrollable worry is the hallmark of generalized anxiety disorder (GAD; American Psychiatric Association, [APA], 2013). This definition was heavily influenced by the cognitive avoidance (CognAv) model of worry offered by Borkovec, Alcaine, & Behar (2004), which posits that worry is, in part, a means to avoid fear-provoking images and the autonomic arousal (AA) they would otherwise provoke. This model is empirically supported by numerous studies finding that worry and GAD are not associated with elevated AA symptoms compared to normal controls. However, while the CognAv model has been highly influential, a considerable body of research suggests that worry and GAD are instead associated with elevated AA symptoms compared to controls. In light of such findings, another model of GAD, the contrast avoidance (ContrAv) model, has been proposed by Newman & Llera (2011). This model suggests that worry does not serve to suppress AA symptoms, but instead serves to increase and maintain heightened negative emotionality (NE), and by extension, AA symptoms, which permits worriers to avoid unpredictable spikes in such aversive emotional states.

Whereas the CognAv model cannot account for the association between worry/GAD and heightened AA symptoms, neither can the ContrAv model easily explain the opposite pattern. To resolve that tension, Vasey, Chriki, and Toh (2016) have proposed and tested an integrative model, the Cognitive Control Model, that can accommodate both perspectives. This model accounts for why pathological worry, that is, excessive and uncontrollable worry, and GAD symptoms have been linked to both heightened and decreased levels of AA symptoms. An initial

study provided clear support for the Cognitive Control Model, such that AA symptoms in worry and GAD vary as a function of individual differences in cognitive control capacity (Vasey et al., 2016). They found that worry and GAD symptom severity conditionally predict low AA symptoms, as expected by the CognAv model, as well as high AA symptoms, as expected by the ContrAv model, and this conditionality is a function of a previously unconsidered moderator, cognitive control or effortful control (EC) capacity. Specifically, this model predicts that higher levels of EC capacity allow the worrier to perform a subtle avoidance maneuver that fosters the suppression of AA symptoms. Specifically, drawing on the CognAv model, this model posits that when a fear provoking image intrudes into awareness, worriers are able to avoid the heightened AA symptoms normally triggered by such images if they have sufficient cognitive control capacity to suppress that image and shift instead to a verbal-linguistic mode of processing as they think about the feared outcome. Thus, in this Cognitive Control Model, worriers who have higher EC capacity should avoid heightened AA symptoms because they are able to engage in and maintain a verbal mode of processing, while worriers who have lower EC capacity may experience heightened AA symptoms because they are unable to do so. A follow-up study replicated the findings from this initial test and extended it to show that as expected, those with higher EC capacity experienced lower AA symptoms as a function of increased verbal thoughts during worry (Toh & Vasey, 2017). The current study builds upon previous findings that EC capacity is an important moderator to consider in the association between worry and GAD and AA symptoms as well as to provide further support for the critical role of verbal-linguistic thoughts during worry. Specifically, this study aims to extend these findings by utilizing

performance measures of cognitive control, an objective measure of AA, and a more precise measure of verbal-linguistic thoughts during worry.

Prior to the fourth edition of the Diagnostic and Statistical Manual (DSM-IV; APA, 1994), AA symptoms were considered to be among the defining features of GAD. For example, in the DSM-III-R (APA, 1987), GAD was defined by unrealistic and excessive anxiety accompanied by at least 6 out of 18 symptoms from three clusters of psychosomatic symptoms. These psychosomatic symptoms included symptoms relating to *autonomic hyperactivity* (e.g. palpitations, nausea, shortness of breath), *motor tension* (e.g. muscle tension, easily fatigued, restlessness), and *vigilance and scanning* (e.g. irritability, feeling keyed up or on edge, exaggerated startle response). However, with the introduction of DSM-IV, only the motor tension and vigilance and scanning clusters were retained, while autonomic hyperactivity (i.e. AA) symptoms were dropped and remain absent in the DSM-5 (APA, 2013). The decision to remove AA symptoms from the diagnostic criteria was based in part on Borkovec's influential CognAv model on worry and in part on empirical evidence that such symptoms were infrequently endorsed by excessive worriers and GAD patients. For example, of the three clusters of psychosomatic symptoms included in the DSM-III-R, symptoms from the AA cluster were least frequently endorsed by GAD patients and were more weakly associated with GAD status than were symptoms from the motor tension or vigilance and scanning cluster (Brown, Marten, & Barlow, 1995; Marten et al., 1993). Other studies have found that measures of AA symptoms such as the anxiety scale of the Depression, Anxiety, and Stress scales (DASS-A; Brown, Chorpita, & Barlow, 1998) and the Beck Anxiety Inventory (BAI; Beck & Steer, 1990) are poor predictors of GAD status/severity (Brown et al., 1998; Brown & McNiff, 2009) and

worry severity (Brown et al., 1998). Self-reported AA symptoms also failed to differentiate GAD patients from controls (Leyfer, Ruberg, & Woodruff-Borden, 2006). Additionally, a factor analysis of the structure of anxiety disorder diagnoses revealed that GAD was unrelated to AA symptoms. Further, when controlling for symptoms of negative affect, GAD was even significantly negatively associated with AA symptoms (Brown et al., 1998).

On the other hand, considerable research has revealed worry and GAD's association to heightened AA symptoms. While it is true that on average, worriers and individuals with GAD report fewer and less frequent AA symptoms compared to symptoms of motor tension and vigilance and scanning, AA symptoms are reported by a substantial percentage (20% - 50%) of patients (Abel & Borkovec, 1995; Marten et al., 1993). Indeed, other studies have found that GAD is highly comorbid with panic disorder (PD; Brown, Antony, & Barlow, 1995; Brown & Barlow, 1992; Tull, Stipelman, Salters-Pedneault, & Gratz, 2009; Wittchen, Zhao, Kessler, & Eaton, 1994), with an even larger group reporting panic attacks (Barlow et al., 1985; Brown, Antony, et al., 1995; Garvey, Cook, & Noyes, 1988; Mohlman et al., 2004; Tull et al., 2009). Along these lines, a GAD diagnosis significantly raises the odds of a PD diagnosis (Grant et al., 2005) and vice versa (Tull et al., 2009). Furthermore, individuals with GAD often report higher AA symptoms relative to controls (Aldao, Mennin, & McLaughlin, 2012; Hoehn-Saric, McLeod, & Zimmerli, 1989; Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004), and similarly elevated AA symptoms as those with PD (Brown, Marten, et al., 1995; Hoehn-Saric et al., 2004). There are also cross-cultural differences in that Asian cultures are more likely to report somatic rather than psychological concerns (Hoge et al., 2006). For example, a study found that in comparing a Nepali and an American sample with GAD, the Nepali sample reported

significantly higher somatic symptoms while the American sample reported higher psychological symptoms using the BAI (Hoge et al., 2006).

Objectively-measured AA also support psychometric studies showing the heterogeneous nature of AA in worriers and GAD samples relative to controls. On the one hand, studies have shown that GAD samples do not differ in AA symptoms relative to controls. This is true for measures of heart rate (HR; Andor, Gerlach, & Rist, 2008; Fisher & Newman, 2013; Lyonfields, Borkovec, & Thayer, 1995), skin conductance level (SCL; Andor et al., 2008; Fisher, Granger, & Newman, 2010), non-specific skin conductance responses (NS-SCRs; Andor et al., 2008; Fisher et al., 2010; Llera & Newman, 2014) and salivary alpha amylase (sAA; Fisher et al., 2010). Similarly, worriers and non-worriers do not differ in HR or SCL (Davis, Montgomery, & Wilson, 2002; Delgado et al., 2009) while a study found worriers to have significantly lower HR than controls (Davis et al., 2002). On the other hand, other studies show that objectively measured AA symptoms are elevated in GAD samples. This is true for measures of HR and NS-SCRs (Pruneti, Fontana, Fante, & Carrozzo, 2010; Pruneti, Lento, Fante, Carrozzo, & Fontana, 2010), as well as sAA (Fisher & Newman, 2013). Similarly, in a general population sample, higher worry frequency, longer worry duration, and higher trait worry was associated with higher HR during waking periods (Brosschot, Dijk, Thayer, & Van Dijk, 2007).

The findings for AA in response to stressors or worry inductions paint a similar picture in that some studies reveal that worriers or individuals with GAD show little or no AA reactivity while others find the contrary. For example, relative to controls, GAD patients showed significantly dampened SCL and HR in response to stress (Hoehn Saric et al., 1989). Other studies examining startle response found that GAD patients tend to show blunted reactivity to

stressors (Grillon et al., 2008; Grillon, Chavis, Covington, & Pine, 2009; Lang & McTeague, 2009) while disorders such as PD show heightened startle responsivity (Grillon et al., 2008). In response to worry inductions, multiple studies have shown that individuals with GAD do not show elevated HR (Hofmann, Schulz, Heering, Muench, & Bufka, 2010; Lyonfields et al., 1995; Stapinski, Abbott, & Rapee, 2010) or NS-SCR amplitude (Llera and Newman, 2014). Nevertheless, other studies have found AA to be elevated in response to stressors or worry inductions. For example, GAD samples had higher SCR and HR compared to controls during a mental arithmetic stress period (Pruneti, Fontana, et al., 2010) and in response to threat words (Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000). Other studies have also found that worry inductions lead to significant increases in reported anxiety compared to neutral or relaxation conditions in GAD samples or GAD analogues (Behar, Zuellig, & Borkovec, 2005; Hofmann et al., 2010; Llera & Newman, 2014), worriers (McLaughlin, Borkovec, & Sibrava, 2007), speech-anxious individuals (Hazlett-Stevens & Borkovec, 2001), and unselected samples (Behar et al., 2005; McLaughlin et al., 2007).

Additionally, neuroimaging studies also reveal such heterogeneity. On the one hand, low responsivity to threat in GAD samples is supported by findings that GAD samples do not differ from controls in level of amygdala activation when viewing threat-related images (McClure et al., 2007; Monk et al., 2006) or they show significantly less amygdala activity than controls (Blair et al., 2012). For example, a pediatric GAD sample showed no significant differences from controls in amygdala activity as well as activation in the amygdala-ventral PFC network when asked to rate how hostile a fearful face appeared or during passive viewing of that fearful face (McClure et al., 2007). Similarly, Hazlett, Stark, & Hoehn-Saric (2012) found that a GAD

sample showed less amygdala activity than controls under conditions of uncertainty. On the other hand, some GAD samples show heightened amygdala activity relative to controls when processing fearful and angry faces (McClure et al., 2007; Monk et al., 2008). For example, in the pediatric GAD sample mentioned earlier, group differences emerged when asked to rate how afraid they were when viewing an angry face. Similarly, other studies have shown that GAD samples show broadly heightened amygdala activation relative to controls across emotional and non-emotional stimuli (Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010; Nitschke et al., 2009).

The heterogeneity of AA symptoms seen in worry and GAD has led to multiple models of worry. First, the predominance of findings that worry and GAD is related to low AA symptoms provided the basis for the CognAv model, which posits that worry functions in part to suppress AA symptoms. Some studies have examined this question directly. For example, Borkovec & Hu (1990) examined the effect of worry on physiological fear response to phobic imagery. They compared heart-rate change in speech-phobic individuals who were asked to listen to and think about relaxation, neutral, or worry-related statements prior to imagining themselves giving a speech and their AA symptoms while doing so (i.e. heart pounding, wobbly legs, dry mouth and throat). Although there were no differences in HR during baseline between those in the worry and relaxation condition, individuals in the worry condition showed a significantly reduced HR response compared to the relaxation condition following exposure to the phobic image. The authors took these findings as evidence that worry is negatively reinforcing because it functions as an avoidance maneuver to suppress the AA reactivity accompanying feared imagery. These findings were replicated in a similar study (Borkovec,

Lyonfields, Wiser, & Deihl, 1993). Such findings provide an explanation for why worry and GAD symptoms are associated with low AA symptoms.

Yet, in contrast to Borkovec and Hu (1990) and Borkovec et al., (1993), other studies have found that worry and GAD symptoms do not suppress AA symptoms in response to fear-provoking stimuli. Instead, whether or not this worry-related reduction in HR response was replicated depended on which period was selected as a baseline for comparison (Peasley-Miklus & Vrana, 2000; Vrana, Cuthbert, & Lang, 1989; Vrana & Lang, 1990). For example, Peasley-Miklus & Vrana (2000) conducted a study in which individuals were first asked to either engage in worry or relaxation, followed by imagination of a feared situation. The results showed that worrying predicted a smaller increase in HR when switching to imagery than did relaxation. This technically replicates Borkovec and Hu (1990). However, the absolute change in HR from the baseline period (prior to the worry or relaxation period) was not significantly different between the worry and relaxation conditions. Instead, it became clear that the worry period led to a significant increase in HR but no additional increase occurred in response to the phobic image. Thus, worrying muted further reactivity to the phobic image but only because the individuals were already in an aroused state due to the worry period. As a result, the authors suggested that, contrary to Borkovec's CognAv model, worry may not protect the worrier from AA symptoms, but rather it may protect the worrier from further increases in AA symptoms when they encounter a fear provoking stimulus because such symptoms are already elevated due to worry. This finding was replicated by others (Llera & Newman, 2010, 2014) leading to the development of the ContrAv model (Newman & Llera, 2011), which asserts that worry is not negatively reinforced by its suppression of AA symptoms. Instead, worry is reinforced because it engenders

and maintains a state of negative emotionality, which includes elevated AA symptoms, that enables worriers to avoid unpredictable spikes in emotion, which they find particularly aversive and which they would otherwise experience if they encounter a fear provoking stimulus while in a relaxed state. This model is at odds with findings that worry and GAD are associated with low AA symptoms and provides an explanation for why some studies find worry and GAD to be associated with elevated AA symptoms.

Taken together, empirical findings of both low and high AA symptoms, theoretically supported by the distinct avoidant functions of worry as proposed by the CognAv and ContrAv models respectively, suggest substantial heterogeneity in AA symptoms across worriers and individuals with GAD. Both the CognAv and ContrAv models offer an important insight into the functions of worry. However, neither alone can account for the observed heterogeneity in AA symptoms seen among high worriers and individuals with GAD. This conflicting pattern of AA symptoms suggests the presence of a moderator. A close examination of the CognAv model suggests a possible path to integrating the two models. As stated by Borkovec et al. (2004), "...when aversive images occur in the process of worry...the shifting of attention to worrisome thinking upon each occurrence...results in escape from or avoidance of the somatic element of the fear response..." (p. 83). Thus, an important feature of the CognAv model is the supposition that worry functions to suppress AA symptoms for those who are proficient enough in the effortful control of attention to suppress images and shift to a verbal mode of processing. For the rest, consistent with the ContrAv model, it may be that worry functions to prevent a further spike in negative emotionality. Therefore, individual differences in cognitive control capacity may account for the two patterns of AA symptoms seen in worriers and individuals with GAD.

One construct that is linked to cognitive control capacity is effortful control (EC), which is a broad self-regulatory construct encompassing the capacities for attentional, inhibitory, and activation control (Rothbart, 2007). Indeed, the literature on executive attention, an especially relevant facet of cognitive control capacity which stems from the neurocognitive field, is beginning to converge with research on EC, which has its roots in temperament and developmental psychology (Rueda, Posner, & Rothbart, 2005). In general, EC overlaps with the executive attention network systems and subsumes higher-order executive function (EF) processes such as working memory, attention, response inhibition, and task switching, which are in turn associated with different areas within the prefrontal cortex (e.g. Welch, 2001; Duckworth & Kern, 2011; Miyake et al., 2000; Snyder, 2012). Below, evidence that suggests heterogeneity in facets of EC or EF in anxiety, worry, and GAD is reviewed. Specifically, attentional control (AC), which has been postulated to be involved in the shifting (switching flexibly between tasks or mental sets) and updating (constant monitoring and rapid addition/deletion of working memory contents) functions of EF; inhibitory control (IC), which maps onto the inhibition function of EF; and working memory capacity (WMC), which maps onto the updating function of EF (Miyake et al., 2000; 2010), will be reviewed. These facets of EF were chosen because they may influence the cognitive control capacity to disengage from imagery and shift instead to a verbal linguistic mode of processing, with recognition that these facets share strong overlaps with EC and executive attention.

AC is the ability to voluntarily sustain focus on a task and flexibly shift attention from one task to another (Derryberry & Rothbart, 1998). Given that the GAD diagnostic criteria include poor control of worry, most would expect that worry and GAD is linked to poor AC.

Indeed, Hirsch & Mathew's (2012) model of worry proposes that pre-existing impairments in AC contribute to the worry process. Additionally, anxiety has been proposed to further diminish AC by tipping the balance such that bottom-up stimulus-driven attentional processes predominate over top-down goal-driven attentional processes (Eysenck, Derakshan, Santos, & Calvo, 2007). This idea is supported by evidence that GAD samples report lower AC (Armstrong, Zald, & Olatunji, 2011; Olatunji, Ciesielski, Armstrong, Zhao, & Zald, 2011), perform poorer in neuropsychological tests of neutral target detection (Olatunji et al., 2011) and attention and cognitive flexibility (Gualtieri & Morgan, 2008), as well as show delayed attentional disengagement from threat cues (Bar-Haim, 2010). For example, in a rapid serial visual presentation task, in which participants were asked to indicate if they saw a rotated neutral target following erotic, fear, disgust, or neutral distractor images, GAD patients demonstrated impaired target detection following fear and neutral distractors (Olatunji et al., 2011). These findings are further supported by imaging studies. For example, trait anxiety (a close correlate of worry) is negatively associated with recruitment of the dorsolateral prefrontal cortex (dlPFC) during trials involving high conflict distractors under low perceptual load (Bishop, 2009), and GAD samples show decreased PFC activity relative to controls during an emotional Stroop task (Price, Eldreth, & Mohlman, 2011).

However, studies also find that worriers and GAD samples show smaller deficits in AC than one might suspect, suggesting that there is substantial variability in AC in this population. Specifically, GAD samples report comparable levels of AC to controls (Armstrong et al., 2011) and high trait anxious individuals varied in terms of reported AC (Derryberry & Reed, 2002). Additionally, the correlation between scores on the AC scale and State-Trait Anxiety Inventory

trait anxiety scale (STAI-T; Spielberger & Gorsuch, 1983) is typically modest (e.g. r = -.30; Bardeen & Orcutt, 2011). Further, scores on the STAI trait anxiety scale are sometimes not correlated with a performance-based measure of AC (Reinholdt-Dunne, Mogg, & Bradley, 2009). In this lab, worry was found to be only modestly correlated with measures of AC (r=-.24 to -.42). In performance-based tasks, studies have found that anxious individuals are *quicker* to detect negative stimuli over positive or neutral stimuli (Cisler & Koster, 2010; Gole, Köchel, Schäfer, & Schienle, 2012) and that even when negative stimuli are presented subliminally, anxious individuals display physiological responses akin to anxiety (Ohman, 2008). Further, anxious individuals preferentially attend to threatening stimuli (Cisler & Koster, 2010), suggesting not poor control, but misplacement of resources leading to differences in the orienting network compared to controls. This idea is supported by findings that some anxious individuals are able to redirect attention when given sufficient time to do so. For example, during longer latency trials (i.e. 500ms vs 250ms), high trait anxious individuals who reported higher levels of AC did not show an attentional bias towards threat (Derryberry & Reed, 2002). The authors suggest that high trait anxious individuals are able to override a prepotent attentional bias towards threat when their AC is sufficiently high, indicating that some anxious individuals have a strong executive attention network.

Another facet of interest is the inhibition function or inhibitory control (IC), the ability to actively inhibit or delay a dominant response to achieve a goal. On the one hand, some studies have found that IC is impaired in worriers or trait anxious individuals (Ansari & Derakshan, 2010; Gole et al., 2012). For example, in a go/no go task, high worriers compared to low worriers made more mistakes when they had to inhibit prepotent responses to move away from

worry-related words relative to moving toward neutral stimuli (Gole et al., 2012). Furthermore, poorer performance on a Stroop task predicted higher likelihood of meeting criteria for an anxiety disorder (Bardeen et al., 2015). On the other hand, other studies have found that there are smaller deficits in IC than expected. For example, Price and Mohlman (2007) found that higher levels of IC were associated with more severe worry and clinician-rated anxiety within a GAD sample. Neuroimaging studies have also found that medial orbital PFC volume, which is related to emotional decision-making under conditions of uncertainty and suppression of amygdala activity, is positively associated with Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) scores in individuals with GAD as well as controls (Mohlman et al., 2009). Other studies show that trait anxiety is more strongly associated with deficits in IC processing efficiency (i.e. response latency) rather than effectiveness (i.e. accuracy; Eysenck et al., 2007), suggesting that anxiety is not associated with a global deficit in IC, but is instead characterized by a misapplication of IC resources. For example, a neural imaging study found that trait anxious individuals showed stronger neural activation in the dlPFC, an area linked to top-down IC, but reduced functional connectivity between the dlPFC and other task-relevant regions during incongruent compared to congruent trials of a color word Stroop task (Stelzel, Basten, & Fiebach, 2011).

Another construct related to the ability to constrain imagery and shift to a verbal mode of processing is WMC, which is a limited cognitive resource that allows for temporary storage and processing of task-related information (Baddeley, 1986). Previous studies have found anxiety to have a negative impact on WMC (Eysenck, 1979; Eysenck & Calvo, 1992). Indeed, high worriers showed more evidence of restricted WMC than low worriers when worrying (Hayes,

Hirsch, & Mathews, 2008). Along these lines, other studies have found WMC to be more exhausted in worriers after engaging in a period of verbal worry compared to imaginal worry (Hirsch et al., 2013). Some theorists have also suggested that because worry is primarily a verbal-linguistic process, worry differentially affects verbal versus visual working memory. Specifically, worry consumes more verbal working memory resources (Eysenck & Calvo, 1992) and less visuospatial working memory (Rapee, 1993). Another study found that worry specifically restricted visuospatial but not verbal working memory (Shackman et al., 2006). On the other hand, studies have found no differences in performance in a verbal or visuospatial working memory task in high worriers (Moreno, Ávila-souza, Gomes, & Gauer, 2015) and even enhanced performance in high trait anxious individuals on a verbal and visuospatial working memory task (Crowe, Matthews, & Walkenhorst, 2007; Walkenhorst & Crowe, 2009).

Overall, there is substantial heterogeneity in these facets of EC and EF in relation to worry and GAD. This variability in worriers and GAD samples is especially interesting given that there may be a link between EF and AA symptoms in anxiety. For example, GAD patients exhibited stronger functional connectivity between the amygdala and dlPFC (associated with self-regulatory functions) at rest relative to controls, and the strength of this connectivity was negatively associated with scores on the BAI (Etkin, Prater, Schatzberg, Menon, & Greicius, 2009), a measure of AA symptoms. Etkin et al. (2009) cited other studies that have also found heightened vlPFC activation to be negatively correlated with anxiety scores in pediatric samples (McClure et al., 2007; Monk et al., 2006). Based on these findings, Etkin et al., (2009) asserted that some GAD patients exhibit habitual engagement of an executive control system to regulate AA symptoms. Along these lines, when healthy controls engaged in neutral and worrisome

thinking, activity in the left orbito-frontal gyrus was negatively correlated with activity in the amygdala, leading the authors to conclude that "worry-induced prefrontal activity suppresses affect-related subcortical areas" (Hoehn-Saric, Lee, McLeod, & Wong, 2005). Interestingly, higher IC has also been linked to lower AA symptoms. For example, performance on the emotional Stroop task was positively associated with self-report measures of worry and trait anxiety, but *not* AA (Price & Mohlman, 2007). Specifically, in an older adult GAD sample, age-normed performance on the emotional Stroop task were positively correlated with the PSWQ (Meyer et al., 1990), the STAI-T (Spielberger & Gorsuch, 1983), clinician-rated anxiety using the Hamilton Rating Scales for anxiety (HRSA; Hamilton, 1959) but not the BAI (Beck et al., 1990). These associations were not evident in the matched control group.

An initial test of the Cognitive Control Model of worry and GAD provided results that are consistent with this perspective (Vasey, Chriki, & Toh, 2016). In this study, a sample of 1343 undergraduate students completed self-report measures related to symptoms of GAD (Worry and Anxiety Questionnaire [WAQ]; Dugas et al., 2001; Generalized Anxiety Disorder Questionnaire – IV [GADQ-IV]; Newman et al., 2002), worry (PSWQ; Meyer et al., 1990), AA (Depression, Anxiety, and Stress Scales [DASS-A]; Lovibond & Lovibond, 1995), and EC (Effortful Control Scale [ECS]; Lonigan & Vasey, 2002). Further analyses were conducted on an analog GAD group within the full sample, that is, individuals who would likely meet criteria for GAD based on the GADQ-IV and who also scored ≥ 70 on the PSWQ. As expected, there was considerable heterogeneity in AA symptoms and level of EC in the full sample as well as in the analog GAD group. Indeed, DASS-A scores in the GAD group covered the entire range possible (i.e., 0 – 40). Importantly, individual differences in EC in the GAD sample moderated the link between GAD

symptom severity and AA symptoms. As predicted by the Cognitive Control Model, GAD symptom severity was strongly positively associated with AA symptoms when EC was low, but was unrelated to AA symptoms when EC was high. This effect was even more evident in the GAD group.

However, evidence that EC moderates the link between worry and GAD and AA symptoms does not reveal the mechanism by which it does so. Consistent with the CognAv model, the Cognitive Control Model suggests that the extent to which worry suppresses AA symptoms depends on how much verbal processing predominates during worry. Based on Lang's (1985) bioinformational theory of fear and Foa & Kozak's (1986) emotional processing theory, the CognAv model posits that worry functions in part to suppress the AA responses normally triggered by imaginal processing of fear provoking information by maintaining a predominantly verbal thought process. This is supported by findings that verbal thoughts are negatively associated with AA symptoms (Borkovec et al., 1993; Freeston, Dugas, & Ladouceur, 1996). Especially among highly fearful individuals, visual images of feared stimuli are more likely to activate AA responses whereas verbal thoughts about the same fearful stimulus, a process characteristic of pathological worry, predict a decrease in AA responses (e.g., Tucker & Newman, 1981; Vrana, Cuthbert, & Lang, 1986). Furthermore, evidence suggests that people spontaneously shift from imagery to verbalization to reduce AA when processing aversive material (Borkovec, Ray, & Stober, 1998; Tucker & Newman, 1981). A direct test of the role of verbal thoughts in the CognAv model was done in a study that extended but did not fully replicate the findings of Borkovec & Hu (1990). This study showed that only speech-phobic individuals who engaged in verbal worry during a Thought-Worry condition showed lower HR

than those in the Relaxation condition (Borkovec et al., 1993). Speech-phobic individuals who were instead in a General-Worry condition, where individuals were asked to worry the way they normally do, failed to show lower HR than those in the Relaxation condition. Interestingly, in this General-Worry condition, percentage of verbal worry was significantly negatively correlated with HR response whereas in the Relaxation condition, percentage of imagery was significantly positively correlated with HR response. This suggests that worry is not necessarily a predominantly verbal activity nor is it necessarily associated with suppression of AA symptoms. The authors suggest that this is evidence that worry suppresses AA symptoms via maintenance of a verbal mode of processing. This model is built on the CognAv model and suggests that higher EC capacity may be linked to stronger ability to maintain worry in a predominantly verbal mode of processing, which in turn is associated with reduced AA symptoms.

Further, there should be differences in the extent to which verbal thoughts predominate during worry, and the literature shows that this is indeed the case. On the one hand, verbal processes have been shown to predominate over images during worry (Borkovec & Lyonfields, 1993; Freeston, Dugas, & Ladouceur, 1996). For example, in a questionnaire-based study, a large community sample of 900 women and a college sample of 300 students reported a predominance of thought over images during worry (Borkovec & Lyonfields, 1993). Another study examining worry content in normal and excessive worriers found that while both groups reported a predominance of thoughts over images during worry, excessive worriers reported an even greater percentage of thought compared to normal worriers (Freeston et al., 1996). Studies using mentation (i.e., thought) sampling procedures, which reduce reliance on retrospective recall, have also provided evidence that worry content mainly comprises thoughts as opposed to

images for both controls and GAD patients. For example, in a study by Borkovec & Inz (1990), 13 GAD patients and 13 controls were asked to relax for 10 minutes followed by a 10-minute worry period. During the relaxation period, GAD patients reported equal amounts of thought and imagery whereas controls reported a predominance of imagery. On the other hand, during the worry induction, both groups reported a predominance of thoughts over images. An interpretation of this finding is that worry is predominantly verbal and that GAD patients are worrying excessively, even during periods when they are asked to relax. Interestingly, thought/imagery ratios of GAD patients during relaxation normalized after receiving psychotherapy. Similarly, Hirsch et al. (2012) found that GAD patients reported not only fewer images but also briefer ones during worry compared to controls. Worry is also characterized by a predominance of left-frontal cortical activity (Carter, Johnsont, Borkovec, Johnson, & Borkovec, 1986; Heller, Nitschke, Etienne, & Miller, 1997; Hofmann et al., 2005; Smith, Zambrano-Vazquez, & Allen, 2016; Wu et al., 1991), which has been linked to verbal and analytic processes (Pinker, 1994; Tucker, 1981). In an EEG study, the authors found that worriers and GAD patients had more left frontal brain activity while those with high trait anxiety but low worry, had greater right frontal and parietal activity (Smith et al., 2016).

On the other hand, although worry is primarily seen as a verbal mode of cognitive processing, research shows that it is not exclusively verbal-linguistic in nature. For example, Davis et al. (2002) found that worriers did not report more thoughts than controls during an hour in which they anticipated giving a speech even though they reported more worry during that period. In fact, when speech-phobic individuals were instructed to worry in a general way (worry as they normally do) before being presented with a feared stimulus (Borkovec et al., 1993), some

had high and unchanging imagery with mean levels similar to those in the Relaxation group. Further, their HR response did not differ significantly from those who had been in the Relaxation condition even though the percentage of verbal worry reported by individuals in the General-Worry condition was inversely associated with HR reactivity as expected, suggesting that verbal and imaginal processing varies during worry. In another study, a verbal worry manipulation failed to reveal any differences compared to an imaginal worry manipulation in frequency of verbal and imagery-based thoughts, suggesting that worry can involve considerable imagery (Stapinski et al., 2010). Worry has also been shown to produce paradoxical effects - serving as a cognitive avoidance mechanism while concurrently priming catastrophic images of future negative events. For example, individuals who had been asked to worry about a stressful stimulus experienced more intrusive images during the following three days compared to individuals who had been asked to use imagery (Butler, Wells, & Dewick, 1995). Thus, it seems that for some worriers, images may play a bigger role than predicted by the CognAv model. The Cognitive Control Model suggests that there may be a continuum of worriers – with those who successfully avoid images and experience lower levels of AA symptoms at one end, and those who either engage in imaginal worry or fail to avoid images and experience higher levels of AA symptoms on the opposite end.

As an initial test for this aspect of the Cognitive Control Model, a study tested the hypothesis that EC capacity influences the extent to which verbal worry predominates, with the expectation that verbal worry would be negatively related to AA symptoms (Toh & Vasey, 2017). Using self-report measures in a sample of 926 college students, this study provided a fourth replication for the finding that EC capacity moderates the link between worry and GAD

and AA symptoms. Additionally, this study provided an extension to Vasey et al. (2016)'s study to show that EC capacity moderates the link between worry and GAD and AA via the expected mechanism. Specifically, as expected, those with high GAD symptoms who reported higher EC capacity experienced lower AA symptoms by virtue of engaging in more verbal thoughts during worry.

However, both studies testing the Cognitive Control Model were purely cross-sectional in nature and relied exclusively on self-report measures. Thus, due to potential self-report limitations, the current study aimed to replicate these findings using other measures. To extend these findings, a behavioral measure of worry was implemented, which also allowed more precise measurements of amount of verbal thoughts and self-reported AA during worry that does not rely on retrospective report. Further, performance-based measures of EC capacity and a physiological measure of AA was also used. A previous study from this research lab used a cruder measure of thoughts during worry that involved having participants recall how they normally worry and estimate the percentage of verbal thoughts they experienced. In the current study, a worry sampling task adapted from Borkovec & Inz (1990) and Hirsch et al. (2012) was used as a more precise measure of amount of thoughts while worrying. In this worry sampling task, participants were asked to worry as they normally do and report when prompted whether they had been thinking in words or imagery at that moment. The expectation is that on average, higher EC capacity should be associated with more verbal thoughts during worry for high worriers.

Additionally, AA symptoms were measured both objectively (i.e., resting HR and phasic HR change during worry) and subjectively (i.e., self-report). A single-item measure on AA

symptoms using a seven-point Likert scale was also included during the worry sampling task to assess AA symptoms during worry (i.e. AA ratings during worry). The current study also aimed to extend past findings on the role of EC capacity in moderating the link between worry and AA symptoms as well as worry and the predominance of verbal thoughts. Previous studies from this research lab have been limited to self-report measures of EC capacity. The current study utilized behavioral measures assessing attentional control, inhibitory control, and working memory capacity to investigate how performance on cognitive control tasks relate to heterogeneity in AA and the predominance of verbal thoughts in worry and GAD.

In sum, the current study aimed to replicate the basic interaction between GAD symptom severity and EC in predicting AA symptoms and amount of verbal thoughts during worry. Using a worry sampling task, the expectation was to find increased verbal thoughts and decreased AA symptoms during worry when EC capacity is high versus low among high worriers. A further goal was to extend previous self-report findings to objective measures of AA by investigating resting HR and phasic HR during worry. The current study also examined whether the findings regarding self-reported EC could be replicated using performance-based measures of EC. Finally, EC capacity was expected to moderate the link between worry and amount of verbal thoughts, which in turn would be less strongly positively related to AA. However, because the sample was constrained to those high in GAD symptom severity, variance was reduced and could result in insufficient power to detect the interaction. As such, if there was insufficient power to detect the interaction, at the very least, EC was expected to have a main effect on the dependent variables.

The current study tested the following hypotheses:

Hypothesis 1: GAD symptom severity will be less strongly and positively associated with AA and more strongly and positively associated with amount of verbal thoughts when self-reported EC is high than low.

Hypothesis 1A: GAD symptom severity and self-reported EC will interact to predict AA symptoms such that GAD symptom severity will predict lower AA symptoms when EC is high than low. At the very least, EC will have a negative main effect on AA symptoms.

Hypothesis 1B: GAD symptom severity and self-reported EC will interact to predict amount of verbal thoughts. At the very least, self-reported EC will have a positive main effect on verbal thoughts.

Hypothesis 1C: GAD symptom severity and self-reported EC will interact to predict AA ratings during worry. At the very least, self-reported EC will have a negative main effect on AA ratings during worry.

Hypothesis 1D: GAD symptom severity and self-reported EC will interact to predict baseline HR. At the very least, self-reported EC will have a negative main effect on baseline HR.

Hypothesis 1E: GAD symptom severity and self-reported EC will interact to predict phasic HR change during worry. At the very least, self-reported EC will have a negative main effect on phasic HR change during worry.

Hypothesis 2: GAD symptom severity will be less strongly and positively associated with AA and more strongly and positively associated with amount of verbal thoughts when performance-based EC is high than low.

Hypothesis 2A: GAD symptom severity and performance-based EC will interact to predict AA symptoms such that GAD symptom severity will predict lower AA symptoms when EC is high than low. At the very least, EC will have a negative main effect on AA symptoms.

Hypothesis 2B: GAD symptom severity and performance-based EC will interact to predict amount of verbal thoughts. At the very least, EC will have a positive main effect on verbal thoughts.

Hypothesis 2C: GAD symptom severity and performance-based EC will interact to predict AA ratings during worry. At the very least, EC will have a negative main effect on AA ratings during worry.

Hypothesis 2D: GAD symptom severity and performance-based EC will interact to predict baseline HR. At the very least, EC will have a negative main effect on baseline HR.

Hypothesis 2E: GAD symptom severity and performance-based EC will interact to predict phasic HR change during worry. At the very least, EC will have a negative main effect on phasic HR change during worry.

Hypothesis 3: GAD symptom severity will be more strongly and positively associated with amount of verbal thoughts when EC is high than low, which in turn, will be less strongly positively associated with AA.

Chapter 2: Methods

Sample

The sample comprised 198 undergraduates taking Psychology 1100 at The Ohio State University. Participants were recruited through the Ohio State University Psychology Department Research Experience Program. Students who participated in the study received partial course credit for their participation. The mean age of the sample was 19.32 (SD = 3.009) and 64.6% were female. Participants were White (66.2%), with 5.6% African-American, 8.6% Asian, 4.5% Hispanic, 4.0 had mixed ethnic heritage, and 11.1% considered themselves other. Questionnaire responses were collected using Qualtrics, which is a secure, web-based data collection service.

This study recruited participants in two ways. While most of the participants (N=128, 65%) were identified via screening with the Effortful Control Scale – Persistence and Low Distraction subscale (ECS; Lonigan & Phillips, 1998) and having reported worrying at least 50% of the day and considered worry to be a problem for them, some participants (N = 70, 35%) were recruited through an advertisement on a website specifying that the study would be most suitable for those who considered themselves worriers (worry at least 50% of the day and consider worry to be difficult to control). Based on a past large undergraduate sample, ECS ≤ 41 and ECS ≥ 47 were identified as the lower and upper threshold. Following procedures approved by the Institutional Review Board (IRB) of the Ohio State University, participants completed all measures and tasks over the course of two sessions.

Measurement

Self-report measures

Demographics:

Demographic Questionnaire: The demographic questionnaire included items concerning the participant's age, gender, year in school, ethnicity, marital status, and primary language.

Measure of GAD symptom severity:

Worry and Anxiety Questionnaire (WAQ). The WAQ (Dugas et al., 2001) consists of 11 items covering DSM-IV diagnostic criteria for GAD. The WAQ has satisfactory test–retest reliability and good known-groups validity (Dugas et al., 2001a). The authors found the WAQ to have 82% specificity and 75% sensitivity. In the current study, the questionnaire demonstrated good internal consistency ($\alpha = .89$).

Measures of Effortful Control:

Effortful Control Scale – Persistence/Low Distractibility subscale (ECS). The ECS (Lonigan & Phillips, 2002) comprises 24 items rated on a 5-point scale from 1 (Not at All) to 5 (Very Much) with regard to how much each describes the individual most of the time. The ECS yields two subscale scores reflecting Persistence/Low Distractibility (ECS-PLD; 12 items) and Impulsivity (ECS-I; 12 items). In this study, scores from the ECS-PLD subscale (hereinafter labeled ECS), which focuses on attentional control and the capacity to persist in activities despite reactive motivation to avoid, were used. The ECS-I focuses on inhibition of impulsive motor responses, which were irrelevant in the current context. Example items from the ECS subscale include, "It's very hard for me to concentrate on a difficult task when there are noises around" and "I can quickly switch from one task to another." The measure has good psychometric

properties in college samples (Vasey et al., 2013; 2014) and demonstrated high internal consistency in the current study (α = .88).

Measures of Autonomic Arousal:

Depression, Anxiety, and Stress Scales (DASS). The DASS (Lovibond & Lovibond, 1995) is a 42-item self-report measure designed to yield three scales measuring the negative emotional states of depression (DASS-D), anxiety (DASS-A), and stress (DASS-S). Participants were asked to rate on a scale of 0 (Did not apply to me at all) to 3 (Applied to me very much, or most of the time) how much the statements applied to them over the past week. The DASS was empirically derived as a measure that would maximally differentiate among symptoms of depression, enduring states of anxiety and fear, and general nervousness/stress (Lovibond & Lovibond, 1993). The DASS-D taps into dimensions of depression including dysphoria, hopelessness, devaluation of life, self-deprecation, lack of interest, anhedonia, and inertia. The DASS-A predominantly assesses symptoms of autonomic arousal. This subscale includes items such as "I had a feeling of faintness" and "I felt I was close to panic". Finally, the DASS-S assesses dimensions that are similar to general distress symptoms associated with worry, such as difficulty relaxing, nervous arousal/tension, agitation, irritability, and impatience. The authors report good psychometric properties for the DASS. Internal reliability for the DASS-D, DASS-A, and DASS-S were found to be high (α = .91, .81, and .89 respectively). In the current study, internal consistency was high (α = .87).

Behavioral Measures

The effect of EC on content of worrisome-mentation:

Worry sampling task. To assess for content of worry, a thought sampling task involving a 5-min period of worrying was adapted from Borkovec & Inz (1990)'s and Hirsch et al. (2012)'s study. First, participants were taught the difference between verbal and imaginal processing. They were read a statement: "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). Next, participants were led through an exercise of imagining vs thinking about cutting a lemon (adapted from Holmes, & Mathews, 2006). They were then asked to imagine a specific topic (eating dinner), and to generate and hold the image for about a minute. Next, they were asked to practice thinking in verbal form about another abstract topic (friendship), which is positive and unlikely to trigger worry, and abstract enough to minimize the chances of spontaneously generating a lot of imagery.

For the worry period, participants were first asked to identify a worry topic and were asked about the negative outcomes anticipated (Vasey & Borkovec, 1992). Then, the experimenter left the room after asking the participants to worry in their usual fashion about a topic of current concern to them. Participants were also told that they would be prompted to rate their mentation content. A computer-generated beep every 30s prompted them to indicate what percentage of their mentation was in verbal thoughts and imagery. They responded to 10 beeps over 5 min. They were also asked to rate the extent to which they felt relaxed, worried, and aroused on 7-point scales, and were asked to continue worrying.

Effortful control tasks:

To assess effortful control capacity and its impact on the ability to maintain a verbal mode of processing as well as AA reactivity to feared imagery, the current study utilized three computer tasks. The three tasks included the attention network task (ANT), the Stroop Color Naming Test (Stroop), and the complex working memory span tasks.

Attention Network Task (ANT). The ANT (Fan, McCandliss, Sommer, Raz, & Posner, 2002) was developed to measure the functioning of three attention networks, namely, alerting, orienting, and executive control, within a single task. Internal reliability for the alerting, orienting, executive control network were found to be moderate to high (α = .52, .61, and .77 respectively). However, note that the ANT has been shown not to be able to isolate these three networks because of interdependences between them (Fan et al., 2002; Macleod et al., 2010). As such, the current study focused on the executive control network because of its high internal reliability and its stronger theoretical overlap with EC (Rueda, Posner, & Rothbart, 2005).

The ANT is a combination of a cued reaction time task (Posner, 1980) and a flanker task (Eriksen & Eriksen, 1974). All participants performed the ANT as described in Fan et al. (2002). Stimuli was presented using E-Prime on a desktop computer. Responses were collected using Chronos, which is a USB-based response and stimulus device with millisecond accuracy. Participants hit the first and fifth button on the Chronos device, corresponding to either a leftward or rightward pointing central arrow target. In each trial, there were five events. First, there was a fixation cross, lasting between 400 and 1600 ms (randomized). Then, the fixation cross was replaced by one of four warning cue types (100ms) that provide increasing levels of information about the forthcoming target. Next, there was a short fixation period for 400 ms after

the warning cue and then the target and flankers appeared simultaneously. The target (left or right pointing middle arrow) was flanked by 4 arrows pointing in the same (congruent; 1/3 trials) or opposite (incongruent; 1/3 trials) direction. In the final third of trials, the target arrow was flanked by dashed lines that formed the neutral condition. The target and flankers remained on screen until a response was recorded, but for no longer than 1700 ms. After participants made a response, the target and flankers disappeared immediately and there was a post-target fixation period for a variable duration which was based on the duration of the first fixation and reaction time (3500 ms minus duration of the first fixation minus RT). After this, the next trial began. A practice block of 24 trials, with feedback on accuracy and speed of response, was followed by three experimental blocks, with no feedback, of 96 trials per block (4 cue conditions x 3 flanker types). Participants rested between blocks. Each block lasted approximately 5 minutes.

Mean reaction time (RT) and number of correct responses (responses made in the same direction to the direction of the target arrow) were measured. All dependent variables were calculated per participant and per condition. Task factors included Cue Type (no, center, double, spatial) and Flanker congruency (congruent, neutral, incongruent). Any RTs that were associated with an incorrect response or were shorter than 100ms were rejected from the RT calculations. The conflict (EC) accuracy score and mean RT was calculated by subtracting the accuracy and mean RT of all congruent flanking conditions, summed across cue types, from the accuracy and mean RT of incongruent flanking conditions.

Inhibitory Control task:

Stroop Color Naming Task (Stroop). The Stroop task was administered on a desktop computer in a quiet room, using E-Prime software and using the Chronos device. A

computerized version of the Stroop test because the Chronos device allows millisecond accuracy, allowing better ability to detect latency effects from incongruent trials. Multiple studies have used and validated computerized versions of this task (McLeod, Hoehn-Saric, & Stefan, 1986; Price, Siegle, & Mohlman, 2012). In this task, participants indicated the color of each word presented on the screen as quickly as possible via button press. Words were printed in red, green, yellow, or blue ink. Participants responded by pressing the appropriate Chronos device button using their left (red) or left (green) third finger or right (yellow) or left (blue) index finger. As described in Price, Siegle, & Mohlman (2012), each trial will begin with a 1000 ms fixation cross hair followed by a word presentation. These words remained on screen until a response was made. Before the task, participants completed 100 practice trials, composed of text strings of colored Xs, with corrective feedback, to overlearn response key mapping. Scores and reaction times from the conflict trial (trials with incongruent color and words) were utilized.

Working Memory Task:

Working Memory Span task. The working memory span task was administered as described by Oswald, McAbee, Redick, & Hambrick (2015). This task was composed of shortened versions of the automated operation (OSpan), symmetry (SSpan), and reading span (RSpan) tasks. The shortened versions have been found to have good reliability. Internal reliability for the Ospan, SSpan, and RSpan were found to be high if the composite of all tasks were used ($\alpha = .76$). In these complex span tasks, individuals were given a sequence of to-be-remembered items (such as sequence of letters while completing a distractor task). In the Ospan task, participants were presented with a set of arithmetic operations and asked to judge whether each equation was true or false (with approximately half being true). After each operation,

participants were presented with a letter for recall at the end of the set. In the Sspan task, participants were presented with a set of 8 x 8 matrices of black and white squares and asked to make a judgment as to whether the matrices are symmetrical down the vertical axis (with approximately half being symmetrical). After each matrix, participants were presented with a red square positioned in a 4 x 4 matrix for recall at the end of the set. In the Rspan task, participants were presented with a set of sentences of approximately 10-15 words in length and were asked the judge whether the sentence were sensible. After each sentence, participants were presented with an element (a letter) for recall at the end of the set.

For each task, participants received two overall scores. First, they received an absolute score, which are the number of trials in which the participant recalled the letter or position of the red square correctly. Participants receive partial-credit scores for correctly recalling some elements within each trial. As suggested by Oswald et al. (2015), partial-credit scores from the three working memory tasks were grouped to represent a domain-general working memory score.

Physiological Measures

Heart rate was measured using a Firstbeat Bodyguard 2 device. This device is a simple non-invasive electrocardiographic (ECG) monitoring system developed for short and long-term measurements of HR. This system consists of two chest electrodes attached directly to the skin on the chest. The ECG data were used to calculate summary scores for heart rate. ECG data using this device were collected throughout the entire session. However, only ECG data during the first 5-minute resting period, the thought sampling task, and the second 5-minute resting period in session 1 and 2 were analyzed in the current study. ECG data during the resting periods

was examined to establish resting HR. Phasic HR change was measured using change in HR during the worry sampling tasks relative to HR before the worry sampling task.

Data Analytic Strategy

Multiple linear regression

Study hypotheses involving single time point assessments of the dependent variables were tested using multiple linear regression (MLR) analyses. All non-dichotomous predictors were mean-centered by z-transformation in these analyses (Aiken & West, 1991). All product terms used in these analyses to test interactions were computed from the standardized predictor variables. Additionally, dependent variables without readily interpretable scales (i.e. DASS-A) were also standardized while those with readily interpretable scales (i.e. percentage of verbal thought, HR) were kept in their original scales. For the MLR analyses, regression diagnostics were examined for each model to determine if extreme data points were present that might be exerting excessive influence on overall model fit or on individual regression coefficients. Specifically, for each model the standardized Dffits and Dfbeta values using ±1.0 as a cutoff were examined (Cohen, Cohen, West, & Aiken, 2002). With the exception of one data point for one of the analyses, no other cases exerted high influence on the fit of the model (Dffits < 0.94), nor on the coefficient for the interaction term (Dfbeta < .75). The high influence data point was reported with the model in the results section, and analyses was run with and without that data point. Furthermore, a data point with a *SD* > 4 was subject to closer examination. There were 2 data points which fell more than 4 *SD* from the mean on accuracy and 1 data point which fell more than 4 *SD* from the mean on reaction time of the performance-based EC tasks. Given that the study recruited those with low and high EC, it was difficult to ascertain if these points were

erroneous or not. As such, models that included accuracy and reaction time of EC tasks were run with and without these data points.

To test Hypothesis 1A, a MLR analysis was used to investigate whether GAD symptom severity interacted with EC to predict AA symptoms. The interaction term is represented by path c_3 in Figure 1. Specifically, WAQ and ECS were entered in the first step, the product term representing the WAQ x ECS interaction in the second step, and DASS-A as the dependent variable. Finally, if the c_3 path in Figure 1 was significant, the interaction was probed using PROCESS, which is a computational tool for SPSS for estimating and probing interactions and conditional process effects in moderation and mediation models (Hayes, 2012). Specifically, simple slopes were probed using bootstrapped (1000 resamples) tests of each predictors' effects on the dependent variable at high (90th percentile) and low (10th percentile) levels of the moderator, and examined regions of significance. Hypotheses 1D, 2A, and 2D were tested using the same approach.

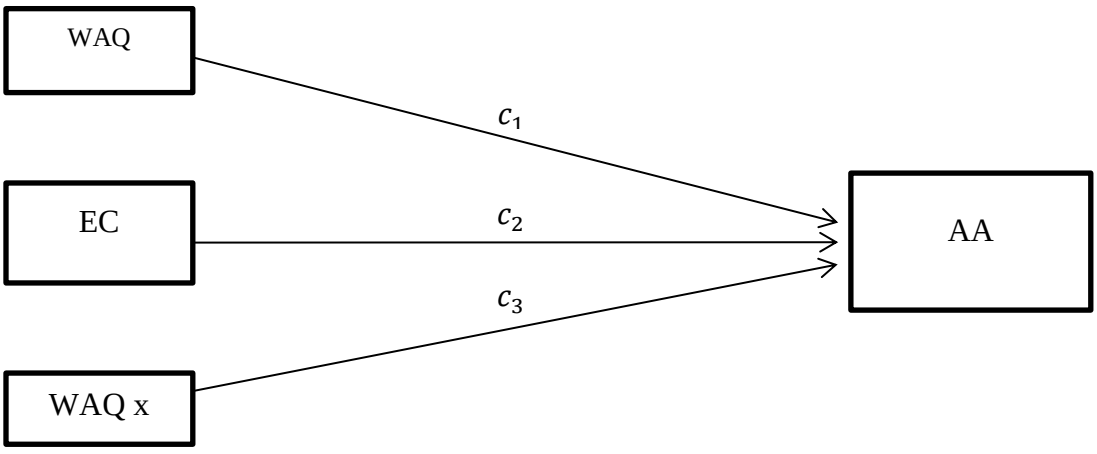

Figure 1. WAQ x ECS predicting outcome variables including DASS-A and baseline HR.

Multilevel modeling

Multilevel modeling (MLM) or linear mixed-effects regression analyses for repeated measures (also known as hierarchical linear model, multilevel model, random effects model) were utilized to assess hypotheses with dependent variables with multiple time point assessments. The strength of MLM analyses is the ability to account for correlations among repeated observations within individuals by estimating individual growth parameters (i.e. random effects; Peugh, 2010). In addition, by utilizing full information maximum-likelihood estimation, MLM provides unbiased estimates in the presence of missing data (Peugh, 2010). In the current study, MLM analyses using SPSS were utilized to investigate individual differences in change over time or growth curves for variables that were measured repeatedly. Specifically, percentage of verbal thoughts, AA ratings, and HR were assessed over ten trials during a worry sampling task for each individual. As such, before hypothesis testing, the best fitting growth curves were first estimated for each of these variables, with percentage of verbal thoughts during worry, AA ratings during worry, and change in HR during worry (phasic HR) as the outcome variable, and trial as the independent variable. Furthermore, MLM was also used to investigate predictors of such individual differences to determine whether characteristics of individuals (level-2 units) such as GAD symptom severity and EC help predict these differences.

Centering variables

All predictors were mean-centered by z-transformation in these analyses to prevent extrapolation to values that are not in the data (e.g. zero baseline HR; Aiken & West, 1991; Singer & Willet, 2003). Such extrapolation would likely lead to uninterpretable or unreliable estimates as there would be no participants or moments with these values in the data. Outcome

variables with interpretable scales were left uncentered (e.g. percentage of verbal thought, HR). Trial was centered to 0, indicating that the intercept is the first point of assessment.

Growth curve modelling

Growth curve modelling is a multi-step process that involves comparing goodness-of-fit indices. Goodness-of-fit indices provide a quantitative measure of the degree of correspondence that each model has to the sample data (i.e., how well the model explained the data). Specifically, the -2 Log-likelihood (-2LL) was tested for significance to compare each model with the previous model. A statistically significant -2LL difference between the two models suggests that the new parameter contributes significantly to the fit of the model (Field, 2014). The smallest -2LL statistic indicated the best fitting model. For the current study, the following models were examined sequentially to determine the best fitting growth curve for each dependent variable: an unconditional means model, an unconditional growth model, and a conditional growth model. The bootstrapped (1000 resamples) estimates of standard errors and p-values were reported.

Unconditional means model

The first model examined was an unconditional means model where only the intercept was allowed to vary and change over time was not modelled. This model primarily served to determine the significance and degree of variation observed between and within participants for an outcome measure without any predictors.

Unconditional growth model

The next model tested was an unconditional growth model where the intercept was allowed to vary and trial was included as a time-varying covariate. This model provided information on whether additional variance in a particular outcome measure could be explained by adding time into the model. If no additional variance could be accounted for by the addition of time as a covariate, then further growth curve model testing cannot be justified and the unconditional means model was judged to be the best fitting model. However, if time was significant as a predictor, it indicated that the outcome measure changed over time. Determining whether this model was an improvement over the unconditional means model was achieved by calculating statistical significance of the deviance in -2LL statistic. If the difference between the -2LL was more than the critical value of 3.84, the model with the smallest -2LL was judged to be better fitting than the other.

Several different growth curves were constructed to attempt to model different shapes of growth trajectories. The simplest model constructed was to model a linear growth trajectory. Additionally, quadratic polynomial functions of time were also incorporated to model curvilinear growth trajectories. Finally, these models also varied in terms of whether the slopes of the models were set to be fixed or randomly varying. A significant fixed slope would suggest that participants varied significantly with respect to the first measurement (i.e. intercepts), and that all participants shared the same slope. A significant random slope allowed participants to vary in their growth rates over time. If any model failed to converge, suggesting that the sample data was highly unbalanced or that there was too much missing data, the slopes were set as fixed (Nakamoto, Lindsey, & Manis, 2007).

Conditional growth model with moderation

After growth curves were estimated, relevant predictors of interest (i.e. GAD symptom severity [as measured by the WAQ], EC measures) were added to the model to examine main effects and their interactions. These models were used to detect additional variance in the outcome measures that could be accounted for by including additional predictors of interest. As such, the current study tested GAD symptom severity, EC, and their interactions as time-invariant covariates at Level-2.

All predictors were mean-centered by z-transformation in these analyses. A significant effect of any of these predictors (i.e. WAQ, EC measures) should be interpreted as a main effect of the predictor on the outcome variable at the first trial when all other predictors were average in the sample. A significant two-way interaction between the two predictors (i.e. WAQ x ECS) indicated that ECS moderated the link between WAQ and the outcome variable at the first trial. Additionally, the conditional growth models also included GAD symptom severity's and ECS's interactions with trial. A significant WAQ x trial interaction should be interpreted as WAQ's effect on the outcome variable over the ten trials when ECS was average. Similarly, a significant ECS x trial interaction should be interpreted as ECS's effect on the outcome variable over the ten trials when WAQ was average. Lastly, a three-way interaction, trial x WAQ x ECS, was also tested. A significant three-way interaction suggested that EC moderated the relationship between WAQ and the outcome measure over the ten trials.

Significant interactions were probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). All continuous predictor and moderator variables were plotted at the 10^{th} and 90^{th} percentile

(1.28 *SD* below and above the mean). In sum, the hypotheses sought to understand whether GAD symptom severity, EC, their interactions with each other and with time could explain the growth curves displayed by participants in the outcome measures (e.g. AA and amount of verbal thoughts during worry).

Conditional growth model with moderated mediation

To justify conducting tests of moderated mediation (Hypothesis 3), which sought to explain why high levels of EC were associated with lower AA symptoms among those high in GAD symptom severity, there first needed to be demonstrations that GAD symptom severity and EC interact to predict AA symptoms (Hypothesis 1A, 1C, 1D, 1E or 2A, 2C, 2D, or 2E) as well as percentage of verbal thoughts during worry (Hypothesis 1B or 2B), which were expected to mediate that effect. These interactions must be significant for the hypothesized moderated mediation to occur (Preacher, Rucker, & Hayes, 2007). To test the conditional growth or conditional means model with moderated mediation, a multilevel structural equation modeling (MSEM) approach was utilized. Specifically, the relationship between GAD symptom severity and percentage of verbal thoughts during worry (*a* paths), and between percentage of verbal thoughts during worry and AA (*b* paths) would differ as a function of EC capacity (included as a fixed effect predictor). Thus, Hypothesis 3 was tested only when specific measures of EC (e.g. self-report and performance) significantly interacted with GAD symptom severity to predict specific measures of AA (i.e. self-report, objective) as well as percentage of verbal thoughts during worry.

As depicted in Figure 2, the predictor (X), moderator (W), and outcome (Y) variables were one-time assessments and were conceptualized as Level 2 variables. The mediator variable,

percentage of verbal thoughts, was measured ten times over the worry period, and was conceptualized as a Level 1 variable (i.e. 2-1-2 mediation model; Lachowitz et al., 2015). The mediator was allowed to vary across individuals with a random intercept and slope (as reported in the results section of Hypothesis 1B). To account for the multilevel structure of the data, R lavaan package was utilized. A latent growth modeling technique was used to capture the random effects of the mediator in this model. The first latent factor was labeled as "Intercept", which is the constant percentage of verbal thoughts for any individual across the ten trials, and the factor loadings were fixed to 1. The second factor, labelled "Slope", represented the slope of an individual's percentage of verbal thoughts trajectory. In this case, it is the slope of the straight line determined by the ten repeated measures and was fixed from 0 to 9, representing linear growth. The two factors, Intercept and Slope, were allowed to covary. The indirect effect was quantified as $a_{1i}b_1$ and $a_{1s}b_2$ (Preacher & Hayes, 2008).

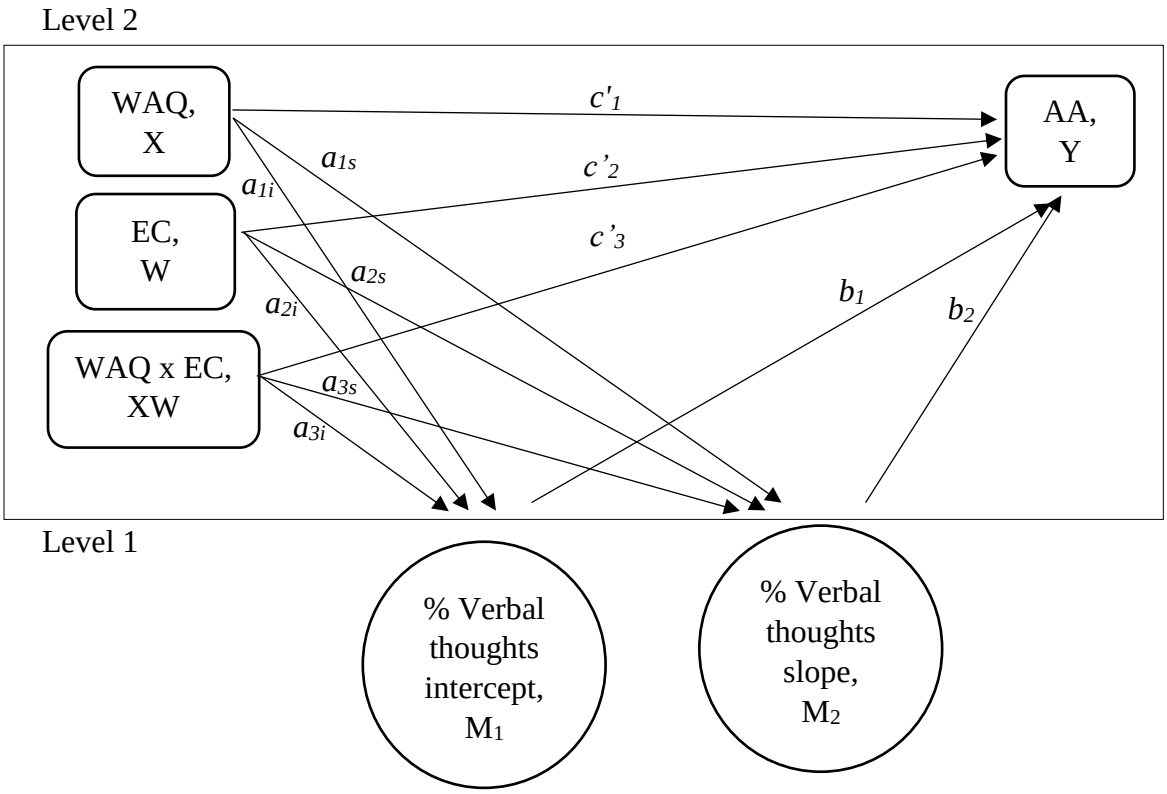

Figure 2. Depiction of multilevel structural equation model with latent growth curve for the mediating effect of verbal thoughts during worry. GAD symptom severity, EC, and their interaction predict Level 1 mediator. Nested frames indicate levels of sampling, boxes indicate variables, circles indicate latent factors.

Summary of data analytic strategy

The current study utilized multiple linear regression for testing hypotheses without a multi-level structure. PROCESS was used to probe interactions at the 10th and 90th percentile of the moderator. Further, multi-level modelling was used for testing hypotheses with multiple time point measurements of the outcome variables to profile growth curves over time. A series of growth curve models were built for outcomes with multiple measurements (i.e. AA ratings

during worry, phasic HR, and percentage of verbal thoughts during worry). Trials were the default time and centered on 0, which represented the first measurement of the variable. The series of growth curve models tested were as follows: (1) unconditional means model (randomly varying intercepts), (2) unconditional growth models (including fixed vs. randomly varying slopes), and (3) conditional means or conditional growth models (depending on the best fitting model based on previous tests, and including all potential main effects and interactions of WAQ, EC measures, and trial). -2LL statistic was used to determine the best fitting model. Significant interactions were probed and all predictor and moderator variables were plotted at the 10^{th} and 90^{th} percentile (1.28 *SD* below and above the mean).

Chapter 3: Results

Preliminary analyses

Data from 198 participants are reported. Incomplete items and missing data were handled using a two-step process. First, for participants with missing items within a questionnaire, their individual means were used to compute their total score. Individual mean substitution when internal consistency of a questionnaire is strong does not produce substantial bias and is more desirable than discarding individuals from the dataset (Osbourne, 2013). Hotdeck imputation in SPSS was used when total scores were missing (Myers, 2011; 1 case [0.5%] had 1 missing total score; 11 cases [5.5%] were missing the worry sampling period; 15 cases [7.5%] were missing the ANT and Stroop task; and 6 cases [3.0%] were missing the WM tasks).

Structural equation modeling

Composite scores for working memory. As planned, scores from the three working memory tasks were entered into a principal components analysis (PCA) with a Varimax (orthogonal) rotation to produce a single score for working memory. Specifically, partial scores from the OSpan, RSpan, and SSpan tests were included. The Kaiser-Meyer-Olkin (KMO) measures of sampling adequacy was used to determine if the scores were factorable. The KMO measure of sampling adequacy is a ratio of the "squared correlation between variables to the squared partial correlations between variables" (Field, 2013) and bound between 0 and 1, with values closer to 1 representing more factorable data. An examination of the KMO measure of sampling adequacy revealed that the sample was factorable (KMO = .624). As presented in Table 1, the items loaded on one principal component which accounted for 55.89% of the total variance. This component will heretofore be referred to as WMPCA.

	Component 1
OSpan	.783
RSpan	.777
SSpan	.678

Table 1. Loadings on principal components.

Data reduction for EC tasks. Because there were multiple behavioral measures of EC, another PCA with a Varimax (orthogonal) rotation was conducted to reduce redundant tests of scores from each EC task that would inflate Type 1 error. Examination of the KMO measure of sampling adequacy for the five scores (i.e. WMPCA, ANT-EC accuracy, ANT-EC reaction time, Stroop Color-Word task accuracy, and Stroop Color-Word task reaction time) was factorable (KMO = .567), albeit poor (Dziuban & Shirkey, 1974). As such, the current study proceeded with the PCA but the interpretations of these results are tentative.

A PCA with Varimax rotation was conducted on the five scores from the EC behavioral tasks with 198 complete cases. The two-components solution, which explain 54.3% of the variance, was preferred for two reasons: (a) acceptable primary score loadings on each component (>.4) and no cross-loadings above .3 (see Table 2), (b) ease of interpretation of the two components. Specifically, ANT-EC and Stroop C-W tasks accuracy scores and WM scores loaded on one component and ANT-EC and Stroop C-W reaction times loaded on a second component. As such, the two components will be referred to as EC-accuracy (EC-acc) and EC-reaction time (EC-rt) respectively. High scores on the EC-acc component refers to better

performance on EC tasks while high scores on the EC-rt component indicates slower reaction time.

	Component 1	Component 2
WM	**.412**	-.220
ANT-EC accuracy	**.745**	-.267
ANT-EC reaction time	-.432	**.643**
Stroop C-W accuracy	**.749**	.249
Stroop C-W reaction time	.053	**.804**

Table 2. Loadings on principal components.

Descriptive Statistics

Mean scores, standard deviations, and internal consistency reliabilities of all measures (i.e. Cronbach's coefficient alpha) are presented in Table 3. For the WAQ, internal consistency estimates were calculated using only continuously and dichotomously scaled items. Kurtosis and skewness did not exceed the suggested cut-off values of 3.0 for skewness and 10.0 for kurtosis (Kline, 2015) for all measures. Table 4 show the means and *SDs* for variables with multiple time point assessments. Zero-order correlations are presented in Table 5.

	M	***SD***	**α**	**N**
WAQ	44.79	16.35	.89	198
ECS	44.00	8.56	.88	198
EC-accuracy	0	1.00	-	198
EC-reaction time	0	1.00	-	198
DASS-A	10.42	7.70	.87	198
Mean AA ratings during worry	3.75	1.62	-	197
Baseline HR	78.58	9.79	-	198
Mean HR during worry	79.02	10.85	-	198
Mean percentage of verbal thoughts during worry	68.50	20.20	-	198

Table 3. Descriptive statistics

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)	M (*SD*)
AA ratings	3.84 (1.77)	3.91 (1.74)	3.87 (1.85)	3.70 (1.84)	3.63 (1.86)	3.75 (1.88)	3.64 (1.89)	3.82 (1.95)	3.70 (1.94)	3.61 (1.99)
HR	78.04 (11.78)	78.44 (11.26)	79.00 (11.48)	79.87 (11.48)	78.65 (11.45)	79.82 (12.09)	79.59 (11.91)	79.05 (12.10)	78.86 (11.91)	78.87 (11.67)
Percentage of verbal thoughts	69.98 (28.35)	71.64 (27.68)	71.19 (27.79)	71.48 (27.54)	68.21 (29.19)	66.65 (28.87)	67.18 (29.10)	66.49 (29.37)	66.37 (28.95)	65.82 (29.95)

Table 4. Means and *SD*s of outcome measures at each worry trial.

	1	2	3	4	5	6	7	8
1. WAQ								
2. ECS	-.46**							
3. EC-accuracy	.06	-.05						
4. EC-reaction time	.03	-.06	.00					
5. DASS-A	.65**	-.43**	.04	.07				
6. Mean AA ratings during worry	.20**	.04	-.05	-.06	-.01			
7. Baseline HR	.05	-.09	.18*	-.13	.06	-.03		
8. Mean HR during worry	-.01	-.03	.14	-.15*	-.01	-.01	.83**	
9. Mean percentage of verbal thoughts during worry	.05	-.01	.05	-.08	.03	.11	-.07	-.08

Table 5. Zero-order correlations.

Primary Analyses

Hypothesis 1 – Did GAD symptom severity and self-reported EC interact to predict AA or verbal thoughts?

Hypothesis 1A - WAQ x ECS predicting DASS-A.

As shown in Table 6, the regression analysis revealed a significant effect of WAQ (B = .57, p < .001) and ECS (B = -.17, p = .01) in Step 1. Furthermore, the addition of the WAQ x ECS interaction (B = -.14, p = .034) produced a significant increment in R^2 in Step 2. Consistent with expectation and as depicted in Figure 3, WAQ was less strongly positively associated with DASS-A when ECS was high (i.e. 90th percentile; B = .46, p < .001) than when ECS was low (i.e. 10th percentile; B = .81, p < .001). Examination of the region of significance revealed that the simple slope for WAQ was significant for all observed values of ECS.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.442	.442	<.001
Intercept		- .01	.05		1.00			
WAQ		.57	.06	.51	<.001			
ECS		-.17	.06	-.15	.010			
	Step 2					.458	.016	.017
Intercept		-.06	.06		.270			
WAQ		.62	.07	.52	.001			
ECS		-.13	.06	-.11	.028			
WAQ x ECS		-.14	.06	-.13	.034			

Table 6. Multiple regression analysis predicting DASS-A with WAQ and ECS.

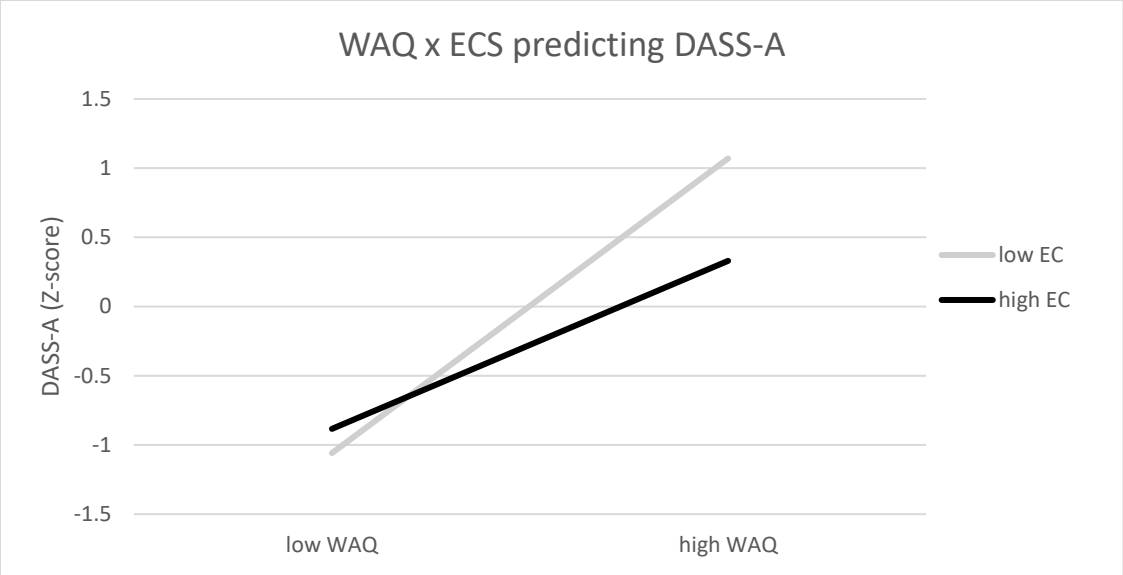

Figure 3. WAQ x ECS interaction predicting DASS-A. Low and high WAQ and EC at the 10th and 90th percentile.

Hypothesis 1B - WAQ x ECS predicting amount of verbal thoughts during worry.

Unconditional growth curve model testing. Because percentage of verbal thoughts during worry was assessed at ten time points, MLM was utilized to examine this hypothesis. First, an unconditional means model was fitted without considering time or any other predictors. This model resulted in significant within-person residual variability to be explained at Level-1 (σ_ε^2 = 464.27, $p < .001$), suggesting that there is significant variability within individuals. Next, time (i.e. trial) was added as a covariate at Level-1 in an unconditional growth model. A fixed linear growth model was first examined along with a fixed quadratic growth model, and then a growth model randomly varying slopes. Table 7 shows the comparisons of the fixed and random slopes for the models tested. The best fitting model was a random intercept with random linear slopes model (-2LL = 18154.976). Percentage of verbal thoughts during worry was found to decrease linearly throughout the ten trials ($\beta = -.68$, $df = 265$; $p < .001$) and the variance explained by the

random slope term was significant ($\sigma_j^2 = 3.18$, p = .027). The model with randomly varying quadratic slopes did not converge. Thus, the final unconditional growth model used allowed participants to have different starting points as well as different linear growth rates.

Conditional growth model testing. Next, to determine if GAD symptom severity, EC, and their interactions with each other and with trial was predictive of the amount of verbal thoughts during worry, a model including percentage of verbal thoughts as the outcome variable and trial, WAQ, ECS, and all combinations of their interactions (i.e. WAQ x ECS, WAQ x trial, ECS x trial, and WAQ x ECS x trial) was analyzed. In this model, all independent variables were z-transformed such that the intercept is interpreted as the average percentage of verbal thought at the first trial for an individual with average GAD symptom severity and EC.

Because the three-way interaction between WAQ, ECS, and trial was not significant, and other terms approached significance, the model was rerun without the three-way interaction. The final model is presented in Table 8. Results did not reveal significant main effects of WAQ or ECS. Further, the results indicated that WAQ significantly predicted increases in percentage of verbal thoughts during worry over the ten trials (β = .47, *df* = 237; *p* = .003). Similarly, ECS also significantly predicted increases in percentage of verbal thoughts during worry over the ten trials (β = .35, *df* = 267; *p* = .035). Additionally, the WAQ x ECS interaction was significant (β = 2.23, *df* = 196; *p* < .001), suggesting that WAQ and ECS interacted to predict percentage of verbal thoughts during worry at the first trial. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 248.41, 407.81) as well as significant individual heterogeneity in slope of percentage of verbal thoughts over time (p < .001, CI = 1.86, 4.77).

The WAQ x ECS interaction was probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). As depicted in Figure 4, all continuous predictive and moderator variables were plotted at the 10^{th} and 90^{th} percentile (1.28 SD below and above the respective mean). Examination of simple slopes were not significant at the points selected. However, the directions of the slopes were informative. Simple slopes analyses revealed that as expected, WAQ negatively predicted percentage of verbal thoughts during worry at the first trial when ECS was low (i.e. 10^{th} percentile; $\beta = -3.31$, $p = .26$) but positively predicted percentage of verbal thoughts when ECS was high (i.e. 90^{th} percentile; $\beta = 2.37$, $p = .27$).

	df	-2LL	Difference in -2LL (critical value)
Random intercept only	3	18206.504	
Random intercept with fixed linear slope	4	18190.109	16.395 (3.84)
Random intercept with fixed quadratic slope	5	18190.092	.17 (3.84)
Random intercept with random linear slope	**6**	**18154.976**	**35.116 (3.84)**
Random intercept with random quadratic slope	NC	NC	NC

Table 7. Comparisons of fixed and randomly varying slopes for unconditional growth models of percentage of verbal thoughts during worry. NC: Non-converging model.

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	72.59	.88	**<.001**	**70.82**	**74.30**
Trial	-.68	.16	**<.001**	**-1.02**	**-.37**
WAQ	-1.80	1.02	.061	-3.60	.37
ECS	-1.79	1.01	.052	-3.61	.32
WAQ * ECS	2.23	.73	**<.001**	**.62**	**3.51**
Trial * WAQ	.47	.18	**.003**	**.10**	**.80**
Trial * ECS	.35	.20	**.035**	-.04	.73
Random effects					
Variance of intercept	318.29	40.25	**<.001**	**248.41**	**407.81**
Variance of slope	2.98	.72	**<.001**	**1.86**	**4.77**

Table 8. Multilevel model predicting percentage of verbal thoughts during worry without the three-way interaction. Fixed effects parameters were bootstrapped based on 1000 resamples.

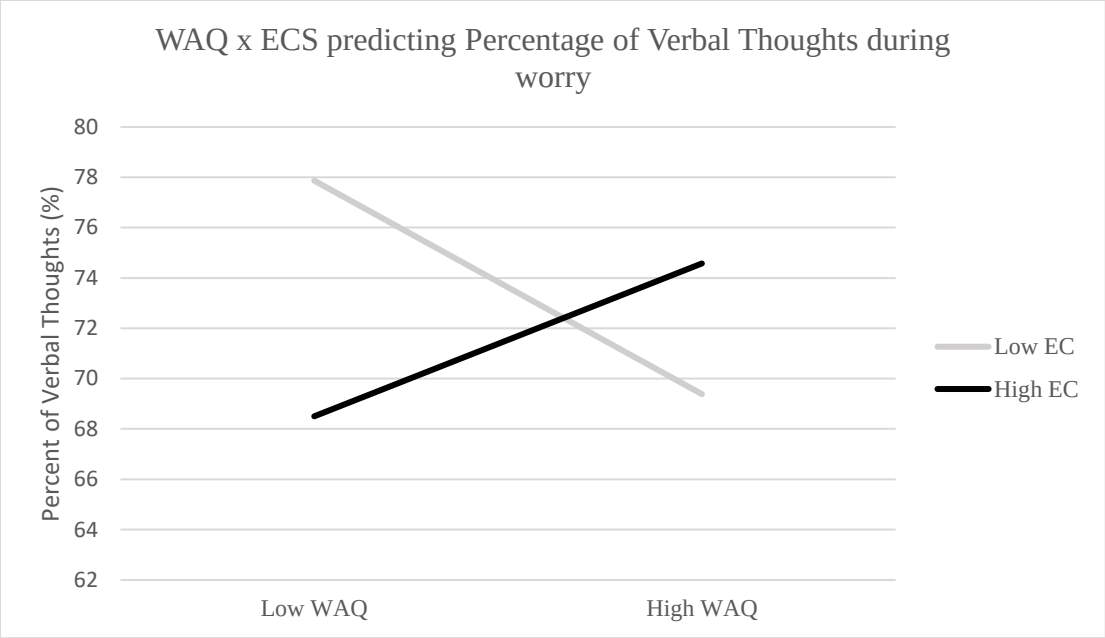

Figure 4. WAQ x ECS interaction predicting percentage of verbal thoughts at the first trial of the worry period. Low and high WAQ and EC at the 10th and 90th percentile.

Hypothesis 1C - WAQ x ECS predicting AA ratings during worry.

Unconditional growth curve model testing. Because AA ratings during worry was assessed at ten time points, MLM was utilized to examine this hypothesis. First, an unconditional means model was fitted without considering time or any other predictors. This model resulted in significant within-person residual variability to be explained at Level-1 ($\sigma_\varepsilon^2 = .99$, $p = .001$), suggesting that there is significant variability within individuals. Next, time was added as a covariate at Level-1 in an unconditional growth model. A fixed linear growth model as well as a fixed quadratic growth model was examined, followed by a growth model with randomly varying slopes. Table 9 shows the comparisons of the fixed and random slopes for the models tested. The best fitting model was a random intercept with fixed linear slopes model (-2LL =

6209.243). AA ratings during worry were found to decrease linearly over the ten trials (β = -0.02, *df* = 235; *p* < .001). The model with randomly varying linear slopes did not converge. Similarly, when a fixed or random quadratic term was included, the model also failed to converge. Thus, the final unconditional growth model used required participants to have the same negative linear growth rate, but allowed participants to vary in their starting points.

Conditional growth model testing. Next, to determine if GAD symptom severity, EC, and their interaction was predictive of self-reported arousal during worry, a model including AA ratings during worry as the outcome variable and trial, WAQ, ECS, and all combinations of their interactions was analyzed. In this model, all independent variables were z-transformed such that the intercept should be interpreted as the average AA rating at the first trial for an individual with average GAD symptom severity and EC. As shown in Table 10, results showed that once these predictors were entered into the model, trial was no longer a predictor of AA ratings (β = -0.01, *df* = 235; *p* = .183). Further, results indicated that WAQ significantly positively predicted AA ratings during worry at the first trial (β = .48, *df* = 238; *p* < .001). Unexpectedly, ECS also significantly positively predicted AA ratings during worry at the first trial (β = .25, *df* = 238; *p* < .001). Additionally, the trial x WAQ interaction was significant (β = -.02, *df* = 1773; *p* < .034), suggesting that WAQ significantly negatively predicted AA ratings over the ten trials of the worry task. The WAQ x ECS interaction was not significant, suggesting that ECS did not moderate WAQ's effect on AA ratings at the first trial. Finally, trial x WAQ x ECS significantly predicted AA ratings during worry. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 1.89, 2.85).

The trial x WAQ x ECS interaction was probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). All continuous predictive and moderator variables were plotted at the 10^{th} and 90^{th} percentile (1.28 SD below and above the respective mean). For those with high WAQ (i.e. 90^{th} percentile), simple slopes analyses revealed that unexpectedly, WAQ significantly negatively predicted AA ratings over the worry task when ECS was low (i.e. 10^{th} percentile; $\beta = -.05$, $p < .001$), but did not significantly predict AA ratings over the worry task when ECS was high (i.e. 90^{th} percentile; $\beta = -.01$, $p = .43$). Furthermore, as shown in Figure 5, while WAQ did not significantly predict AA ratings over time when ECS was high, unexpectedly, those high in GAD symptom severity with high ECS reported overall higher levels of AA than those with low ECS.

	df	-2LL	Difference in -2LL (critical value)
Random intercept only	3	8056.880	
Random intercept with fixed linear slope	4	6209.243	1847.637 (3.84)
Random intercept with fixed quadratic slope	NC	NC	NC
Random intercept with random linear slope	NC	NC	NC
Random intercept with random quadratic slope	NC	NC	NC

Table 9. Comparisons of fixed and randomly varying slopes for unconditional growth models of AA ratings during worry. NC: Non-converging model.

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	3.88	.05	**<.001**	**3.79**	**3.97**
Trial	-.01	.01	.183	-.03	.01
WAQ	.48	.05	**<.001**	**.39**	**.58**
ECS	.25	.05	**<.001**	**.14**	**.34**
WAQ * ECS	.06	.05	.253	-.04	.17
Trial * WAQ	-.02	.01	**.034**	**-.04**	**-.01**
Trial * ECS	-.01	.01	.754	-.02	.01
Trial * WAQ * ECS	.02	.01	**.025**	**.01**	**.04**
Random effects					
Variance of intercept	2.32	.24	**<.001**	**1.89**	**2.85**

Table 10. Multilevel model predicting AA ratings during worry. Fixed effects parameters were bootstrapped based on 1000 resamples.

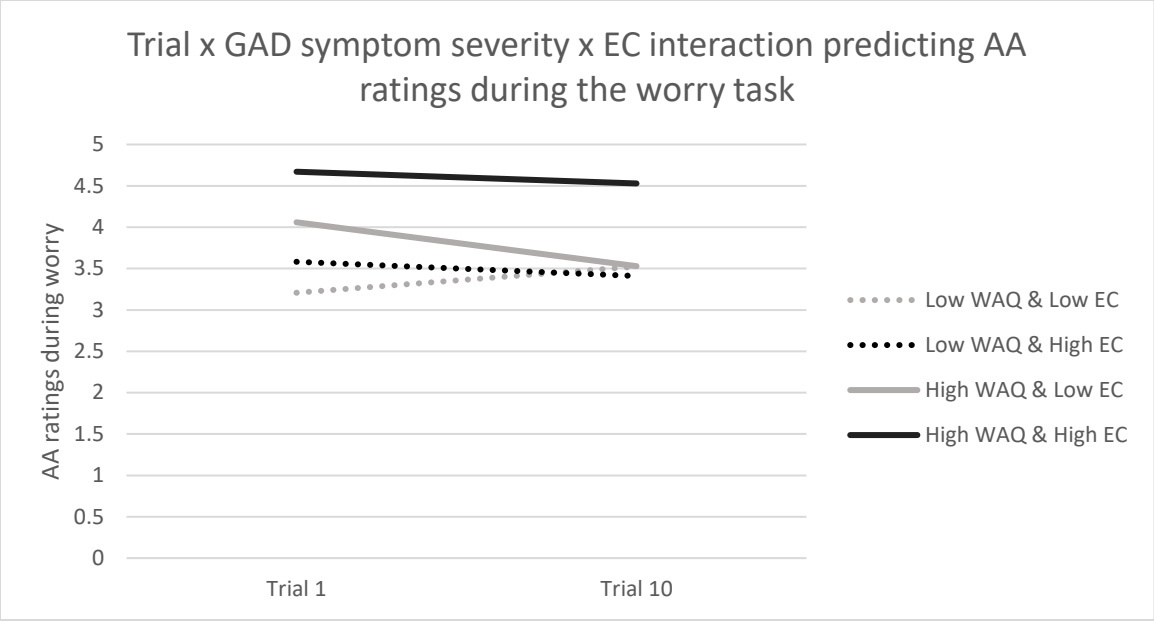

Figure 5. Trial x WAQ x ECS interaction predicting AA ratings during worry. Low and high WAQ and EC at the 10th and 90th percentile.

Hypothesis 1D - WAQ x ECS predicting resting HR.

As shown in Table 11, the regression analysis revealed no significant effects of WAQ (B = .11, p < .886) or ECS (B = -.81, p = .303) in Step 1. Furthermore, the addition of the WAQ x ECS interaction (B = -.21, p = .783) did not produce a significant increment in R^2 in Step 2.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.008	.008	.463
Intercept		78.58	.70		<.001			
WAQ		.11	.79	.01	.886			
ECS		-.81	.79	-.07	.303			
	Step 2					.008	.000	.783
Intercept		78.48	.78		<.001			
WAQ		.18	.83	.02	.825			
ECS		-.76	.81	-.07	.352			
WAQ x ECS		-.21	.74	-.02	.783			

Table 11. Multiple regression analysis predicting resting HR with WAQ and ECS.

Hypothesis 1E - WAQ x ECS predicting phasic HR during worry.

Unconditional growth curve model testing. Because HR was collected continuously over the ten trials of the worry task, MLM was utilized to examine this hypothesis. First, an unconditional means model was fitted without considering time or any other predictors except pre-worry HR as a covariate. Note that pre-worry HR was mean-centered for ease of interpretation. When pre-worry HR was included as a random factor, the model could not converge and as such, this term was left out in subsequent analyses. The unconditional growth model with pre-worry HR as a fixed covariate resulted in significant within-person residual variability to be explained at Level-1 ($\sigma_\varepsilon^2 = 21.6$, $p < .001$), suggesting that there was significant variability within individuals. Next, time was added as a covariate at Level-1 in an unconditional growth model. A fixed linear growth model as well as a fixed quadratic growth model was examined, followed by a growth model with randomly varying slopes. Table 12 shows the

comparisons of the fixed and random slopes for the models tested. The best fitting model was an unconditional means (random intercept only) model (-2LL = 12162.180). While the model with random intercepts and fixed linear slope converged, the difference in -2LL did not reach the critical level. As such, that model was judged not to be a superior fit to the unconditional means model. Thus, the final unconditional means model used allowed participants to vary in HR at the first worry trial, but did not require any change over time.

Next, to determine if GAD symptom severity, EC, and their interaction was predictive of phasic HR during worry, a model including phasic HR as the outcome variable, WAQ, ECS, and their interaction was analyzed (see Table 13). Again, pre-worry HR was entered as a covariate. In this model, all predictors (except pre-worry HR, which was mean-centered) were z-transformed such that the intercept is interpreted as the average HR during the worry period for a participant with average GAD symptom severity, ECS, and pre-worry HR. Consistent with the CognAv model, results indicated that after controlling for pre-worry HR, WAQ significantly negatively predicted phasic HR ($\beta = -.40$, $df = 198$; $p < .001$). Additionally, the WAQ x ECS interaction was significant ($\beta = .21$, $df = 198$; $p < .031$), suggesting that ECS moderated the relationship between WAQ and phasic HR. Moreover, there was significant individual heterogeneity at the start of the worry period ($p < .001$; 95% CI = 15.76, 24.41).

The WAQ x ECS interaction was probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). Examination of simple slopes showed that the points selected were not significantly different from 0. However, the steepness of the slopes was informative. As depicted in Figure 6 and contrary to expectations, simple slopes analyses revealed that WAQ more strongly negatively

predicted HR change during the worry period when ECS was low (i.e. 10th percentile; β = -.66, p = .33) but less strongly negatively predicted HR when ECS was high (i.e. 90th percentile; β = -.13, p = .79).

	df	-2LL	Difference in -2LL (critical value)
Random intercept only	4	12162.180	
Random intercept with fixed linear slope	5	12158.839	3.341 (3.84)
Random intercept with fixed quadratic slope	NC	NC	NC
Random intercept with random linear slope	NC	NC	NC
Random intercept with random quadratic slope	NC	NC	NC

Table 12. Comparisons of fixed and randomly varying slopes for unconditional growth models of phasic HR during worry. NC: Non-converging model.

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	79.11	.12	**<.001**	**78.86**	**79.35**
Pre-worry HR	.87	.01	**<.001**	**.85**	**.88**
WAQ	-.40	.12	**<.001**	**-.62**	**-.17**
ECS	.05	.13	.695	-.21	.30
WAQ * ECS	.21	.10	**.027**	**.01**	**.39**
Random effects					
Variance of intercept	19.61	2.19	**<.001**	**15.76**	**24.41**

Table 13. Multilevel level model predicting phasic HR during worry, controlling for pre-worry HR. Fixed effects parameters were bootstrapped based on 1000 resamples.

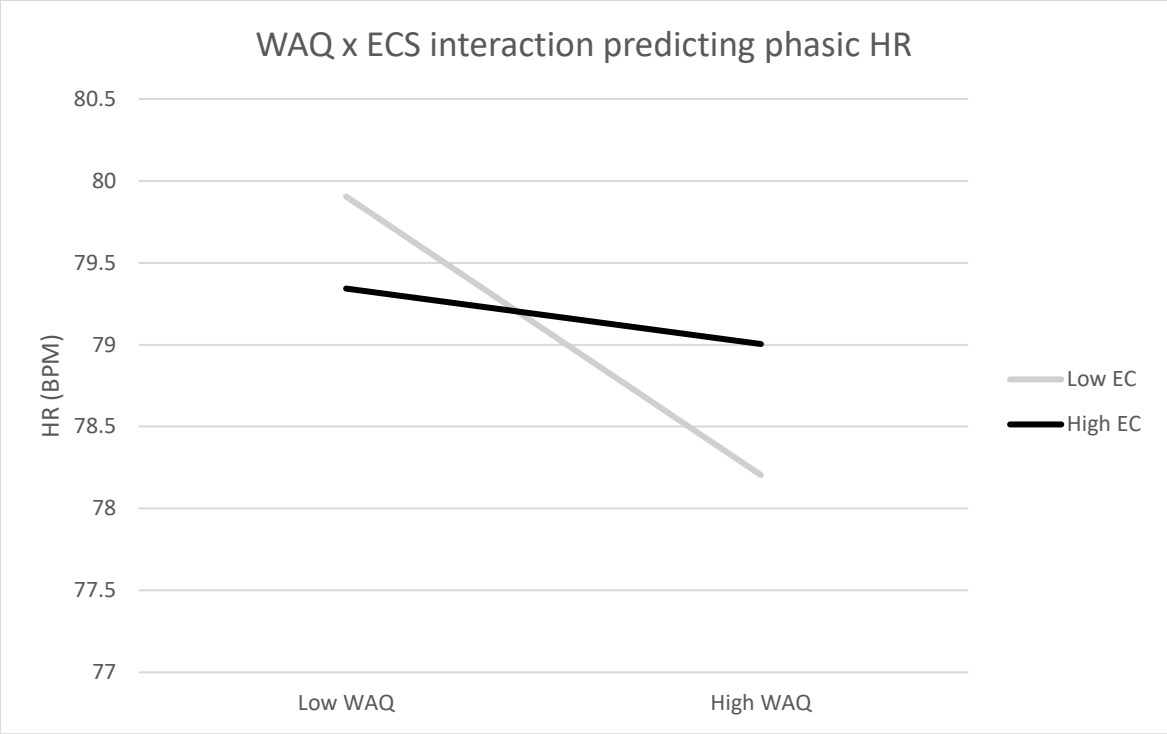

Figure 6. WAQ x ECS interaction predicting phasic HR during worry. Low and high WAQ and EC at the 10th and 90th percentile.

Hypothesis 2 – Did GAD symptom severity and performance-based EC interact to predict AA or verbal thoughts?

Hypothesis 2A - WAQ x performance-based EC predicting DASS-A.

EC-acc. As shown in Table 14, the regression analysis revealed only a significant main effect of WAQ (B = .65, p < .001) in Step 1. Unexpectedly, there was no main effect of EC-acc. Furthermore, the addition of the interaction did not produce a significant increment in R^2 in Step A high influence case was identified when regression diagnostics were examined (Dffits = -1.95, with the next closest value being .99; Dfbeta interaction term = -1.76 with the next closest value

being -.31). The model was run with and without that data point however there was no change in significant findings. As such, the point was included in the model reported.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.420	.420	<.001
Intercept		- .01	.05		1.00			
WAQ		.65	.06	.65	<.001			
EC-acc		.01	.06	.01	.944			
	Step 2					.425	.005	.208
Intercept		-.01	.05		.941			
WAQ		.65	.06	.65	<.001			
EC-acc		-.01	.05	-.01	.845			
WAQ x EC-acc		.07	.06	.07	.153			

Table 14. Multiple regression analysis predicting DASS-A with WAQ and EC-acc.

EC-rt. As shown in Table 15, the regression analysis revealed only a significant main effect of WAQ (B = .65, p < .001) in Step 1. However, the addition of the WAQ x EC-rt interaction (B = .14, p = .029) produced a significant increment in R^2 in Step 2. As depicted in Figure 7. WAQ x EC-rt predicting DASS-A. and as expected, WAQ was more strongly significantly associated with DASS-A when EC-rt was slow (B = .81, p < .001) than when EC-rt was fast (B = .48, p < .001). Examination of the region of significance revealed that the simple slope for WAQ was significant for all observed values of EC-rt.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.422	.422	<.001
Intercept		- .01	.05		1.00			
WAQ		.65	.06	.65	<.001			
EC-rt		.05	.06	.05	.365			
	Step 2					.440	.018	.014
Intercept		-.01	.05		.935			
WAQ		.65	.06	.65	<.001			
EC-rt		.09	.06	.08	.141			
WAQ x EC-rt		.14	.07	.13	.029			

Table 15. Multiple regression analysis predicting DASS-A with WAQ and EC-rt.

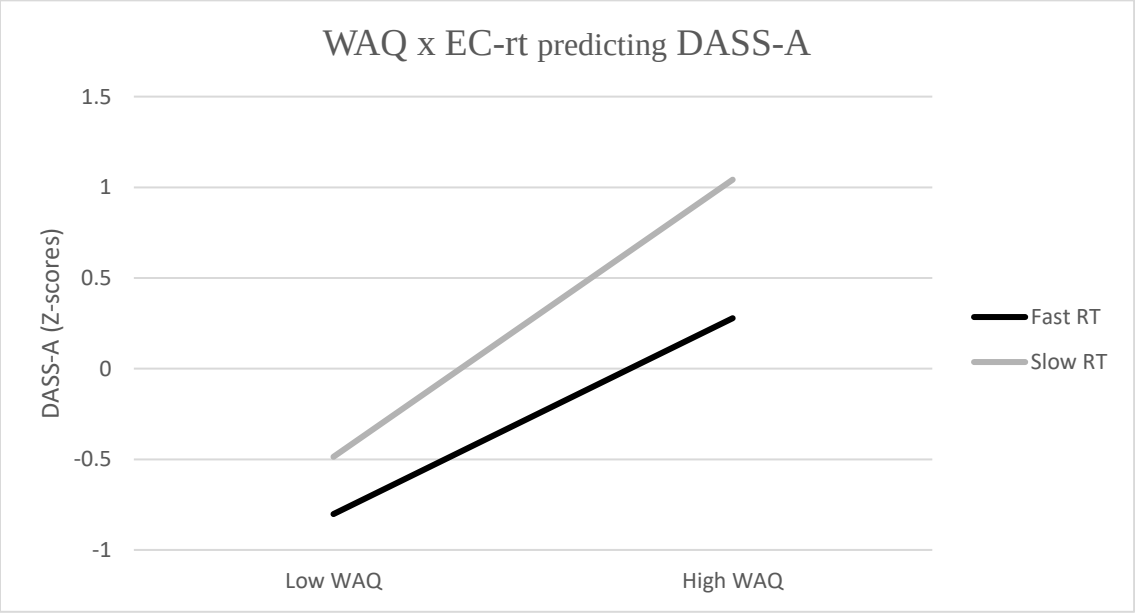

Figure 7. WAQ x EC-rt predicting DASS-A.

Hypothesis 2B - WAQ x performance-based EC predicting amount of verbal thoughts during worry.

EC-acc - Conditional growth model testing. Given that the best fitting growth curve for percentage of verbal thoughts during worry was already determined to be a random intercept and fixed linear growth curve in Hypothesis 1E, the current hypothesis test proceeded with conditional growth model testing. The predictors in this model were WAQ, EC-acc, their interactions with each other and with trial and the outcome variable was percentage of verbal thoughts during worry. As shown in Table 16, results indicated that as expected, there was a significant positive main effect of EC-acc on percentage of verbal thought ($\beta = 2.98$, $df = 264$; $p = .003$). However, this finding did not achieve significance when 2 outliers > 4*SD* were excluded from the analysis. As mentioned earlier, given that the study recruited those with low and high EC, it was difficult to ascertain if these points were erroneous or not. As such, caution should be used when interpreting this effect. Additionally, the WAQ x trial interaction predicted increases in verbal thoughts over the ten trials ($\beta = .34$, $df = 264$; $p = .019$). There were no other significant interactions. Furthermore, dropping the non-significant 3-way interaction did not change the findings. Moreover, there was significant individual heterogeneity at the start of the worry period ($p < .001$; 95% CI = 245.79, 405.66) and changes in percentage of verbal thought over time ($p = < .001$, 95% CI = 1.97, 4.96).

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	71.18	.87	**<.001**	**69.42**	**72.95**
Trial	-.67	.17	**<.001**	**-1.00**	**-.35**
WAQ	-.76	.92	.374	-2.50	1.15
EC-acc	2.98	.96	**.003**	**1.11**	**5.00**
WAQ * EC-acc	.68	.98	.484	-1.25	2.56
Trial * WAQ	.34	.18	**.019**	-.01	.69
Trial * EC-acc	-.14	.18	.341	-.49	.20
Trial * WAQ * EC-acc	1.27	.18	.379	-.22	.50
Random effects					
Variance of intercept	315.77	40.36	**<.001**	**245.79**	**405.66**
Variance of slope	3.13	.74	**<.001**	**1.97**	**4.96**

Table 16. Multilevel model with percentage of verbal thoughts during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

EC-rt - Conditional growth model testing. As shown in Table 17, results indicated that percentage of verbal thoughts decreased over time (β = -.70, *df* = 265; *p* < .001). Additionally, there were no main effects of WAQ or EC-rt. WAQ also positively predicted percentage of verbal thoughts over time (β = .35, *df* = 265; *p* = .013). In line with expectations, the trial x EC-rt interaction significantly negatively predicted increases in percentage of verbal thought during worry over the ten trials (β = -.57, *df* = 237; *p* <.001), such that slower reaction time was associated with decreases in percentage of verbal thoughts over the ten trials. No other two-way or three-way interactions were significant. Furthermore, when this model included a participant

who was more than 6 *SD*s from the mean in terms of their reaction time, there was a marginal interaction between WAQ and EC-rt predicting percentage of verbal thoughts at the first trial (β = 1.63, *df* = 237; *p* = .079). However, that this marginal interaction disappeared without the outlier suggests that this marginal interaction was highly influenced by this single data point and as such should be interpreted with caution. In addition, a main effect of EC-rt was uncovered (β = 1.95, *df* = 265; *p* = .037), suggesting that slower reaction time was associated with higher percentage of verbal thoughts at the first trial. Furthermore, dropping the non-significant 3-way interaction did not change the findings and as such, this term was included in the final model. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 248.89, 408.95) and marginally significant individual heterogeneity in changes in percentage of verbal thought over time (p < .001, 95% CI = 1.86, 4.77).

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	71.79	.88	**<.001**	**70.11**	**73.54**
Trial	-.70	.17	**<.001**	**-1.04**	**-.38**
WAQ	-.71	.90	.405	-2.45	1.05
EC-rt	1.95	.95	**.037**	-.01	3.81
WAQ * EC-rt	.79	.99	.398	-1.04	2.73
Trial * WAQ	.35	.17	**.013**	-.01	.67
Trial * EC-rt	-.57	.18	**<.001**	**-.95**	**-.22**
Trial * WAQ * EC-rt	-.18	.18	.247	-.57	.17
Random effects					
Variance of intercept	319.04	40.42	**<.001**	**248.89**	**408.95**
Variance of slope	2.98	.71	**<.001**	**1.86**	**4.77**

Table 17. Multilevel model with percentage of verbal thoughts during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

Hypothesis 2C - WAQ x performance-based EC predicting AA ratings during worry.

EC-acc - Conditional growth model testing. Given that the best fitting growth curve for self-reported arousal during worry was already determined to be a random intercept and fixed linear growth curve in Hypothesis 1B, the current hypothesis test proceeded with conditional growth model testing. The predictors in this model were WAQ, EC-acc, their interactions with each other and with trial and the outcome variable was tswArousal. As seen in Table 18, because the three-way interaction was not significant, and other terms approached significance, the model was rerun without the three-way interaction.

As shown in Table 19, results indicated that AA ratings were dropping over time (β = -.02, *df* = 1755; *p* = .008), suggesting that participants habituated to the worry task. WAQ significantly positively predicted AA ratings during worry at the first trial (β = .39, *df* = 236; *p* < .001). As expected, EC-acc significantly negatively predicted AA ratings during worry at the first trial (β = -.08, *df* = 236; *p* = .045). Additionally, there was a significant WAQ x EC-acc interaction (β = -.05, *df* = 197; *p* = .008). No other interaction significantly predicted AA ratings during worry. Furthermore, when this model was rerun without 2 outliers who were more than 4 *SD*s from the mean, EC-acc's effect was no longer significant (p = .405). Thus, caution should be used when interpreting EC-acc's effect. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 1.94, 2.94).

The WAQ x EC-acc interaction was probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). As depicted in Figure 8 and as expected, simple slopes analyses revealed that WAQ significantly positively predicted AA ratings at the first trial when EC-acc was low (i.e. 10^{th} percentile; β = .48, p = .015), but did not significantly predict AA ratings at the first trial when EC-acc was high (i.e. 90^{th} percentile; β = .29, p = .161).

Parameter	Estimate	*SE*	*P*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	3.85	.04	**<.001**	**3.77**	**3.94**
Trial	-.02	.01	**.008**	**-.04**	**-.01**
WAQ	.39	.05	**<.001**	**.30**	**.48**
EC-acc	-.08	.04	**.045**	**-.15**	**.01**
WAQ * EC-acc	-.05	.02	**.008**	**-.09**	**-.01**
Trial * WAQ	-.01	.01	.112	-.03	.01
Trial * EC-acc	-.01	.01	.787	-.02	.01
Random effects					
Variance of intercept	2.39	.25	**<.001**	**1.94**	**2.94**

Table 18. Multilevel model with self-reported arousal during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

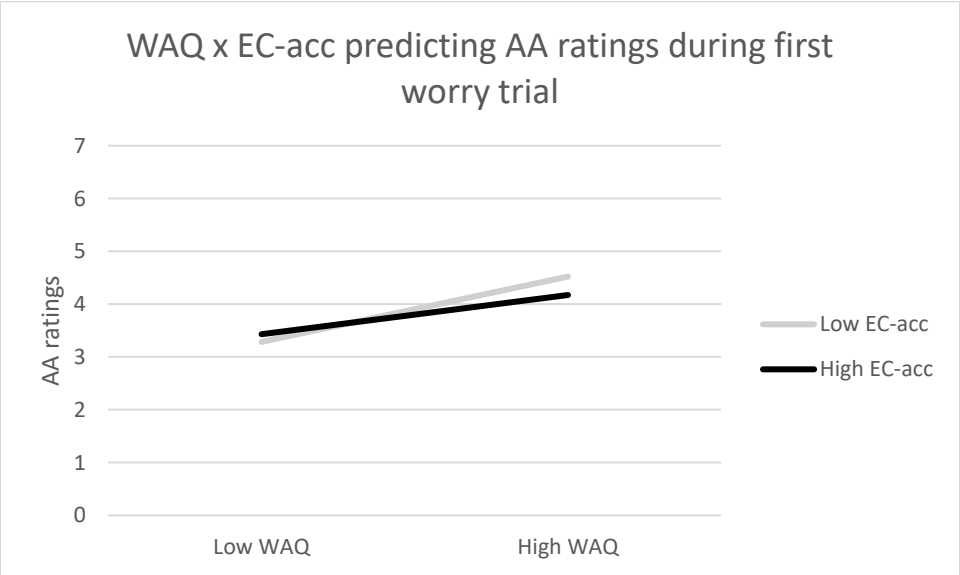

Figure 8. WAQ x EC-acc predicting AA ratings during the first worry trial. Low and high WAQ and EC at the 10th and 90th percentile.

EC-rt - Conditional growth model testing. As shown in Table 19, results indicated that WAQ significantly positively predicted AA ratings during worry at the first trial (β = .39, *df* = 237; *p* < .001). Unexpectedly, EC-rt significantly negatively predicted AA ratings at the first trial (β = -.13, *df* = 237; *p* = .007), such that slower reaction time was associated with lower AA ratings at the first trial. Furthermore, the two-way interaction between WAQ and EC-rt produced a significant effect on AA ratings at the first trial (β = -.15, *df* = 237; *p* = .002). No other two-way or three-way interaction was significant. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 1.93, 2.92). Furthermore, dropping the non-significant 3-way interaction did not change the findings. As such, the term was included in the final model.

The WAQ x EC-rt interaction was probed using an online tool designed for evaluating interactions in multilevel models (Preacher, Curran, & Bauer, 2006; http://www.quantpsy.org). As depicted in Figure 9 and contrary to expectation, simple slopes analyses revealed that WAQ significantly positively predicted AA ratings at the first trial when EC-rt was fast (i.e. 10^{th} percentile; β = .57, p < .001), but did not significantly predict AA ratings at the first trial when EC-rt was slow (i.e. 90^{th} percentile; β = .22, p = .28).

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	3.85	.04	**<.001**	**3.78**	**3.94**
Trial	-.02	.01	**.006**	**-.04**	**-.01**
WAQ	.39	.05	**<.001**	**.29**	**.48**
EC-rt	-.13	.05	**.007**	**-.22**	**-.02**
WAQ * EC-rt	-.15	.05	**.002**	**-.25**	**-.05**
Trial * WAQ	-.01	.01	.110	-.03	.01
Trial * EC-rt	-.01	.01	.912	-.02	.02
Trial * WAQ * EC-rt	.01	.01	.719	-.02	.02
Random effects					
Variance of intercept	2.37	.25	**<.001**	**1.93**	**2.92**

Table 19. Multilevel model with self-reported arousal during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

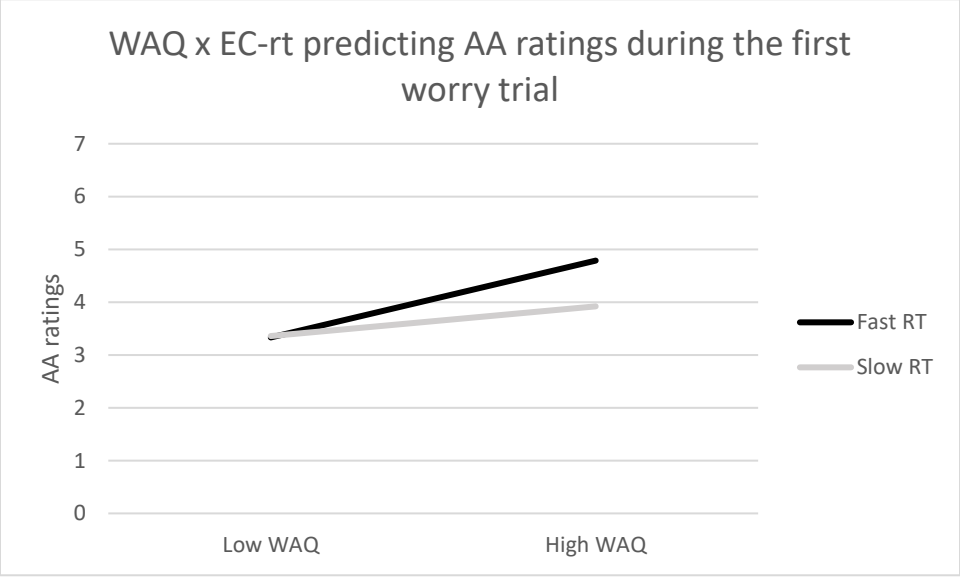

Figure 9. WAQ x EC-rt interaction predicting self-reported arousal during worry. Low and high WAQ and EC at the 10th and 90th percentile.

Hypothesis 2D- WAQ x performance-based EC predicting resting HR.

EC-acc. As shown in Table 20, the regression analysis revealed only a significant effect of EC-acc (B = 1.73, p = .019) in Step 1. However, the direction of the effect was in the opposite direction predicted. Furthermore, the addition of the WAQ x ECS interaction (B = 1.05, p = .106) did not produce a significant increment in R^2 in Step 2.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.034	.034	.036
Intercept		78.58	.66		<.001			
WAQ		.38	.74	.04	.598			
EC-acc		1.73	.73	.18	.019			
	Step 2					.044	.010	.155
Intercept		78.52	.66		<.001			
WAQ		.47	.75	.05	.519			
EC-acc		1.56	.73	.16	.022			
WAQ x EC-acc		1.05	.74	.10	.106			

Table 20. Multiple regression analysis predicting baseline HR with WAQ and EC-acc.

EC-rt. As shown in Table 21, the regression analysis revealed no significant effects of WAQ (B = .52, p = .494) or EC-rt (B = -1.26, p = .106) in Step 1. Furthermore, the addition of

the WAQ x EC-rt interaction (B = .81, p = .395) did not produce a significant increment in R^2 in Step 2.

		B	SE	*sr*	*p*	R^2	ΔR^2	*p*
	Step 1					.019	.019	.153
Intercept		78.58	.69		<.001			
WAQ		.52	.76	.05	.494			
EC-rt		-1.26	.81	-.13	.106			
	Step 2					.025	.006	.274
Intercept		78.55	.69		<.001			
WAQ		.52	.75	.05	.491			
EC-rt		-1.04	.83	-.10	.201			
WAQ x EC-rt		.81	.98	.08	.395			

Table 21. Multiple regression analysis predicting baseline HR with WAQ and EC-rt.

Hypothesis 2E - WAQ x performance-based EC predicting phasic HR during worry.

EC-acc - Conditional growth model testing. Given that the best fitting growth curve for phasic HR during worry was already determined to be a random intercept only model in Hypothesis 1D, the current hypothesis testing proceeded with a conditional means model testing. As shown in Table 22, the predictors in this model were WAQ, EC-acc, and their interactions, with pre-worry HR as a covariate, and the outcome variable was phasic HR during worry. In support of the CognAv, results indicated that after controlling for pre-worry HR, WAQ

significantly negatively predicted phasic HR during worry (β = -.38, *df* = 198; *p* < .001). However, unexpectedly, EC-acc did not impact phasic HR during worry. The interaction between WAQ and EC-acc also did not significantly predict phasic HR during worry. Moreover, there was significant individual heterogeneity at the start of the worry period (p < .001; 95% CI = 15.79, 24.45).

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	79.02	.10	**<.001**	**78.82**	**79.23**
Pre-Worry HR	.86	.01	**<.001**	**.85**	**.88**
WAQ	-.38	.10	**<.001**	**-.58**	**-.20**
EC-acc	.10	.13	.427	-.15	.34
WAQ * EC-acc	-.10	.13	.444	-.33	.16
Random effects					
Variance of intercept	19.65	2.19	**<.001**	**15.79**	**24.45**

Table 22. Multilevel model with phasic HR during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

EC-rt - Conditional growth model testing. As shown in Table 23 and consistent with the CognAv model, results indicated that after controlling for preworry HR, WAQ significantly negatively predicted phasic HR during worry (β = .34, *df* = 198; *p* < .001). Unexpectedly, EC-rt significantly negatively predicted phasic HR during worry (β = -.85, *df* = 198; *p* < .001), such that slower reaction time was associated with lower HR during worry relative to the pre-worry

period. Furthermore, the two-way interaction between WAQ and EC-rt did not produce a significant effect on HR during worry. Moreover, there was significant individual heterogeneity at the start of the worry period ($p < .001$; 95% CI = 18.13, 24.75).

Parameter	Estimate	*SE*	*p*	Lower 95% CI	Upper 95% CI
Fixed effects					
Intercept	79.02	.11	**<.001**	**78.82**	**79.24**
Pre-Worry HR	.86	.01	**<.001**	**.84**	**.88**
WAQ	-.34	.10	**<.001**	**-.54**	**-.14**
EC-rt	-.85	.13	**<.001**	**-1.12**	**-.58**
WAQ * EC-rt	-.14	.15	.346	-.42	.17
Random effects					
Variance of intercept	18.99	1.62	**<.001**	**18.13**	**24.75**

Table 23. Multilevel model with phasic HR during worry as the dependent variable. Fixed effects parameters were bootstrapped based on 1000 resamples.

Hypothesis 3: GAD symptom severity will be more strongly and positively associated with amount of verbal thoughts when EC is high than low, which in turn, will be less strongly positively associated with AA.

Hypothesis 3A: GAD symptom severity x self-reported EC predicting AA through verbal thoughts.

To test for moderated mediation, the hypothesis of whether percentage of verbal thoughts during worry could mediate the effect of GAD symptom severity on AA, and whether the mediated effect differed as a function of EC capacity was explored. In other words, the relationship between GAD symptom severity and percentage of verbal thoughts during worry (a_1 path), and between percentage of verbal thoughts during worry and AA (*b* paths) would differ as a function of effortful control capacity (a_3 path). Specifically, when ECS is high, WAQ should be positively associated with the percentage of thoughts during worry (a_1 paths), which in turn should be negatively associated with DASS-A (*b* paths). Thus, when ECS is high, the indirect path should be significantly *negative*, reflecting the product of the positive a_1-paths and negative *b*-paths. In contrast, when ECS is low, the positive association between GAD symptom severity and percentage of verbal thoughts during worry should be attenuated.

To account for the multilevel structure of the data, a latent growth modeling technique was used to capture the random effects of the mediator in this model. The first latent factor was labeled “Intercept”, which is the constant percentage of verbal thoughts for any individual across the ten trials, and the second factor, was labelled “Slope”, which represented the slope of an individual’s percentage of verbal thoughts trajectory. In this case, it is the slope of the straight line determined by the ten repeated measures and was fixed from 0 to 9, representing linear

growth. The indirect effect was quantified as $a_{1i}b_1$ and $a_{1s}b_2$ (Preacher & Hayes, 2008). See Figure 2 for a graphical depiction of the model.

This model was run only when GAD symptom severity and EC interacted significantly to predict both percentage of verbal thoughts during worry and AA in the direction expected. As reported above, WAQ x ECS did interact to predict DASS-A as well as percentage of verbal thoughts, such that higher GAD symptom severity and higher EC was associated with lower AA and higher percentage of verbal thoughts over the worry period compared to lower EC. Thus, a moderated mediation model was tested.

Table 24 shows the results of the multilevel structural equation model. None of the independent variables significantly predicted the intercept or slope of verbal thoughts during worry. More importantly and contrary to prediction, the intercept and slope of verbal thoughts were also not significantly associated with DASS-A. The indirect effect of the WAQ on DASS-A through the intercept of verbal thoughts was estimated to be B = -.002 (SE = .008, p = .773) while the indirect effect of WAQ on DASS-A through the slope of verbal thoughts was estimated to be B = -.006 (SE = .025, p = .801), showing no evidence of a mediation. When interpreting model fit for structural equation modeling analyses, a comparative fit index (CFI) and Tucker-Lewis Index (TLI) around .95, a root mean square error of approximation (RMSEA) around 0.05 suggest excellent model fit (Kline, 2005). The current model's fit statistics indicated that it showed good fit to the data (CFI = 0.95; TLI = 0.94; RMSEA = 0.056).

DV/Predictor	B	SE	*p*
DV: Intercept of Verbal thoughts			
Constant	72.53	1.81	<.001
WAQ	-1.42	2.06	.492
ECS	-1.59	2.02	.432
WAQ x ECS	1.77	1.98	.372
DV: Slope of Verbal thoughts			
Constant	-.60	.22	.007
WAQ	.34	.24	.150
ECS	.27	.23	.238
WAQ x ECS	.23	.26	.369
DV: DASS-A			
Constant	-.19	.25	.445
Intercept of verbal thoughts	.01	.01	.606
Slope of verbal thoughts	-.02	.06	.744
WAQ	.63	.08	<.001
ECS	-.12	.06	.057
WAQ x ECS	-.13	.07	.053
Random effects			
Variance of intercept	374.12	**56.91**	**<.001**
Variance of slope	3.80	**1.12**	**<.001**

Table 24. Moderated mediation results involving percentage of thoughts.

Hypothesis 3B: GAD symptom severity x reaction time on EC tasks predicting AA through verbal thoughts.

A second moderated mediation model was tested to evaluate whether percentage of verbal thoughts during worry could mediate the effect of GAD symptom severity on AA, and whether the mediated effect differed as a function of reaction time on EC tasks. In other words, the relationship between GAD symptom severity and percentage of verbal thoughts during worry (a_1 paths), and between percentage of verbal thoughts during worry and AA (*b* paths) would differ as a function of reaction time on EC tasks (a_3 path). Specifically, when EC reaction time is fast, WAQ should be positively associated with the percentage of thoughts during worry (a_1 paths), which in turn should be negatively associated with DASS-A (*b* paths). Thus, when EC reaction time is fast, the indirect path should be significantly *negative,* reflecting the product of the positive a_1-path and negative *b*-paths. In contrast, when EC reaction time is slow, the positive association between GAD symptom severity and percentage of thoughts during worry should be attenuated.

This model was run only when GAD symptom severity and EC reaction time interacted significantly to predict both percentage of verbal thoughts during worry and AA in the direction expected. As reported above, WAQ x EC-rt did interact to predict DASS-A as well as percentage of verbal thoughts, such that higher GAD symptom severity and faster EC reaction time was associated with lower AA and higher percentage of verbal thoughts over the worry period compared to lower EC. Thus, a moderated mediation model was tested.

Table 25 shows the results of the multilevel structural equation model. As shown in Table X, EC-rt marginally significantly predicted the slope of verbal thoughts (B = -.42, SE = .23, p = .067). No other independent variables significantly predicted the intercept or slope of verbal thoughts during worry. More importantly and contrary to prediction, the intercept and slope of verbal thoughts were also not significantly associated with DASS-A. The indirect effect of the WAQ on DASS-A through the intercept of verbal thoughts was estimated to be B = -.001 (SE = .007, p = .974) while the indirect effect of WAQ on DASS-A through the slope of verbal thoughts was estimated to be B = -.007 (SE = .024, p = .754), showing no evidence of a mediation. The current model's fit statistics indicated that it showed good fit to the data (CFI = 0.95; TLI = 0.95; RMSEA = 0.051).

DV/Predictor	B	SE	*p*
DV: Intercept of Verbal thoughts			
Constant	71.67	1.65	<.001
WAQ	-.30	1.88	.871
EC-rt	.39	1.92	.839
WAQ x EC-rt	1.66	1.85	.368
DV: Slope of Verbal thoughts			
Constant	-.70	.22	<.001
WAQ	.29	.24	.241
EC-rt	-.42	.23	.067
WAQ x EC-rt	-.27	.23	.240
DV: DASS-A			
Constant	-.07	.27	.787
Intercept of verbal thoughts	.01	.01	.833
Slope of verbal thoughts	-.03	.06	.673
WAQ	.65	.06	<.001
EC-rt	.08	.07	.271
WAQ x EC-rt	.13	.07	.053
Random effects			
Variance of intercept	374.12	**56.91**	**<.001**
Variance of slope	3.80	**1.12**	**<.001**

Table 25. Moderated mediation results involving percentage of thoughts.

Chapter 4: Discussion

The current study first aimed to replicate the basic tenet of the Cognitive Control Model (Vasey et al., 2016). Namely, that GAD symptom severity and self-reported EC interact to predict self-reported AA symptoms (Hypothesis 1A), such that high levels of GAD symptom severity are less strongly positively associated with AA symptoms when EC is high versus low. At the very least, EC should be largely independent of level of worry and have a negative association with AA symptoms that is in opposition to the positive association between such symptoms and GAD symptom severity. Further, this study was designed to extend and replicate the next major aspect of the Cognitive Control Model regarding verbal thoughts (Toh & Vasey, 2017). That is, GAD symptom severity and self-reported EC will interact to predict amount of verbal thoughts during worry (Hypothesis 1B). High levels of GAD symptom severity should be more strongly positively associated with percentage of verbal thoughts during worry when EC is high versus low. At the very least, EC should have a positive main effect on verbal thoughts during worry. As discussed below, results were largely consistent with both these predictions.

The current study emphasizes the use of methods other than self-report. First, to obtain a more valid self-report measure of amount of verbal thoughts during worry and AA symptoms, a worry sampling task was utilized. Participants were asked to report the percentage of verbal thoughts and the level of arousal they were experiencing ten times throughout the worry sampling task. The expectation was that the GAD symptom severity by self-reported EC interaction would predict percentage of verbal thoughts (Hypothesis 1B) as well as AA ratings (Hypothesis 1C). Second, the basic interaction was tested when AA was assessed objectively and operationalized as resting HR (Hypothesis 1D) as well as phasic HR change during worry

(Hypothesis 1E). As discussed below, while the results with regards to the predominance of verbal thoughts during worry were in line with predictions, the predictions regarding AA ratings during worry and objectively measured AA were not supported.

The current research also aimed to extend the Cognitive Control Model by assessing EC using behavioral measures, including the Attention Network Task, Stroop Color-Word task, as well as working memory span tasks. Thus, Hypotheses 2 investigated whether GAD symptom severity and performance-based EC interact to predict AA symptoms (Hypothesis 2A, 2C, 2D, and 2E) and amount of verbal thoughts during worry (Hypotheses 2B). As discussed further below, the results provided mixed support for the generalization of self-reports of EC to behavioral measures of EC.

Finally, Hypothesis 3 was a test of moderated mediation and tested only when GAD symptom severity interacted with specific measures of EC to predict percentage of verbal thoughts as well as specific measures of AA. Specifically, the prediction was that at high versus low EC, GAD symptom severity will be more strongly and positively associated with amount of verbal thoughts, which in turn, will be less strongly positively associated with AA. In the current study, these criteria were met twice and as such two tests of moderated mediation were conducted. First, GAD symptom severity interacted with self-reported EC to predict verbal thoughts during worry as well as self-reported AA symptoms. Second, GAD symptom severity also interacted with reaction time during EC tasks to predict verbal thoughts during worry and self-reported AA symptoms. As discussed below, the findings failed to support Hypotheses 3.

Performance-based measures of EC

The current study aimed to investigate if findings based on self-reported EC can be extended using performance-based EC tasks. Specifically, the Attention Network Task, Stroop Color-Word task, as well as working memory span tasks were utilized as performance-based measures of EC. These tasks were selected for several reasons. First, the literature on executive attention, which stems from the neurocognitive field, is converging with research on EC, which has its roots in temperament and developmental psychology (Rueda, Posner, & Rothbart, 2005). Executive attention is especially relevant to the ability to shift attention from imagery to verbal modes of processing. Both executive attention and EC share strong overlaps with higher-order executive function (EF) processes (Friedman et al., 2007; Snyder, Miyake, & Hankin, 2015), which subsumes attentional control, working memory, and inhibitory control (Welch, 2001). Second, because there is heterogeneity in level of these different facets of EF in anxiety, worry, as well as GAD, the current study ultimately attempted to capture the unitary component associated with the different facets of EF by utilizing principal components analyses on the three performance-based cognitive control tasks. This was done primarily to prevent redundant tests for each EC task, which will inflate Type 1 error. As such, the study instead takes the perspective that heterogeneity in different facets of EF reflects a common component of EF that varies between individuals, which may influence the top-down cognitive control capacity to disengage from imagery and shift instead to a verbal linguistic mode of processing while worrying.

First, data from the EC tasks were reduced. First, a component score for the three working memory tasks was derived. The decision to create a component score for the working

memory tasks was based on greater research support for a domain-general rather than domain-specific perspective of working memory (Oswald et al., 2014). A domain-general perspective assumes that processes underlying specific working memory tasks are common across different tasks, and that this commonality is what is most strongly influencing correlations between working memory and outcomes of interest. For example, verbal and spatial working memory is highly correlated and share about 70-85% of their variance (Kane et al., 2004). Next, the working memory component score was entered into another principal components analysis with scores and reaction times of the executive attention subtests of the Attention Network task and the Stroop Color-Word task. The final two component scores were related to accuracy in the WM, Attention Network task, and Stroop Color-Word task, as well as the reaction times in the Attention Network task and the Stroop Color-Word task.

Dependent variable: Self-reported AA

Hypothesis 1A

Hypothesis 1A examined the basic tenet of the Cognitive Control Model, specifically to replicate the interaction between GAD symptom severity and self-reported EC predicting self-reported AA symptoms over the past week. The data fully supported replication of this effect. Consistent with prior studies, individuals with higher GAD symptom severity reported lower levels of AA symptoms when EC was high versus low (Vasey et al., 2016). One standard deviation of increase in GAD symptoms was associated with a .48 standard deviation increase in AA symptoms when EC was high, and an even larger .76 standard deviation increase in AA symptoms when EC was low. Thus, the basic setting condition for the study, the replication of the interaction between GAD symptom severity and self-reported EC to predict AA symptoms,

was fulfilled. Furthermore, examination of the overall levels of self-reported AA showed that high GAD symptom severity was related to overall higher levels of AA than low GAD symptom severity. This finding is consistent with some studies showing that individuals with GAD report higher levels of AA symptoms than controls (Hoehn-Saric et al., 2004). However, it is also important to note that this characteristic in the current sample may have constrained the ability to find the interaction effects predicted by other hypotheses of the study. For example, compared to a previous study (Toh & Vasey, 2017), the current study revealed a larger correlation between the WAQ and DASS-A (r = .65 vs r = .58).

Hypothesis 2A

While accuracy during the performance-based EC tasks did not influence self-reported arousal, reaction time during these tasks interacted with GAD symptom severity to predict self-reported AA symptoms. As expected, GAD symptom severity was more strongly and significantly associated with self-reported AA symptoms when reaction time was slow than when it was fast. Furthermore, in line with predictions, among those with high GAD symptom severity, those with fast reaction times experienced AA symptoms close to the mean of the sample, while those with slow reaction times experienced AA symptoms one standard deviation above the mean.

These findings on accuracy and reaction time during performance-based EC tasks suggests that accuracy during these cognitive tasks alone may not be sufficient to parse out the relationship between GAD symptom severity and AA symptoms. Other models of attention in anxiety suggest that reaction time more strongly distinguishes between those with problematic worry versus controls. Specifically, Eysenck's Attentional Control Theory (Eysenck, Derakshan,

Santos, & Calvo, 2007) posits that anxiety leads to use of compensatory strategies that may not lead to differences performance effectiveness (i.e. accuracy) but to differences in performance efficiency (i.e. reaction time). Our findings suggest that decreases in performance efficiency may be related to increased AA symptoms.

Summary

Overall, the findings on self-reported EC and reaction time during EC tasks support the Cognitive Control Model in that higher levels of EC interacted with GAD symptom severity to predict lower AA symptoms over the past week. Further, failure to find significant results when using accuracy during EC tasks suggests perhaps that it is important for future studies to distinguish between performance effectiveness versus efficiency. Additionally, a meta-analysis found that GAD patients show the strongest attentional bias when confronted with emotional stimuli that are presented in a verbal format (Goodwin, Yiend, & Hirsch, 2017). As such, future studies should include tasks with such stimuli to be better positioned to assess EC and uncover significant effects.

Dependent variable: Amount of verbal thoughts during worry

Hypothesis 1B

The next main hypothesis postulated that GAD symptom severity and self-reported EC would have positive associations with the amount of verbal thought experienced during worry. In the worry sampling task, percentage of verbal thoughts was assessed after every 30-second worry period, which occurred over ten trials. To account for the possibility of individual growth parameters, a multilevel modeling approach was utilized. The model which provided the best fit

for the data was one that allowed individuals to start at different points in their amount of verbal thoughts reported, with individual differences in change over time which is best represented by a linear slope. The pattern suggests that everyone starts with different percentages of verbal thoughts, with significant heterogeneity in change in amount of verbal thoughts over time. Consistent with our prediction, GAD symptom severity and self-reported EC interacted to predict percentage of verbal thoughts over time. This finding is supported by a past study utilizing retrospective self-reports that found that GAD symptoms interact with EC to predict percentage of thoughts during worry (Toh & Vasey, 2017). The current study's replication of this effect also supports the use of worry inductions as a viable method to investigate the relationship between GAD symptom severity, EC, and percentage of thoughts.

The pattern of the three-way interaction was such that high levels of GAD symptom severity in combination with high EC was associated with *increasing* levels of verbal thoughts throughout the worry induction, while high levels of GAD symptom severity coupled with lower EC was instead associated with *decreasing* levels of verbal thoughts during the worry induction. Further, at the first trial, there was no evidence of a moderation effect of EC on verbal thoughts, suggesting that a predominantly verbal-linguistic form of worry is typical when worry is first initiated, however the likelihood of maintaining such form of worry decreases over time. The pattern of this three-way interaction supports the notion that constraining worry to a verbal mode of processing depletes cognitive resources (Leigh & Hirsch, 2011), and depth in cognitive control resources may be crucial to the ability to maintain such a mode of processing. This pattern of verbal-linguistic worry raises the possibility that had the worry induction been for a longer duration, or if the individuals were pre-stressed, even those with high EC may experience

declining levels of verbal thoughts during worry. Future studies should include experimental manipulations on state EC to investigate its effect on the amount of verbal thoughts during worry.

Interestingly, there was a trial main effect, such that for the individual with average GAD symptom severity and EC in the current sample, percentage of verbal thoughts *decreased* significantly over the worry task. There was also a significant GAD symptom severity by trial interaction on percentage of verbal thoughts during worry. At higher levels of GAD symptom severity and average EC, percentage of thoughts increased over time during the worry task. This provides evidence for Borkovec's CognAv model, and suggests that those with high GAD symptom severity engage more strongly in verbal worry, perhaps partially in efforts to suppress AA symptoms. This finding also lends support to other studies that have found that worry is primarily verbal in nature (Borkovec & Inz, 1990; Hirsch et al., 2012) and adds to those findings because it suggests that for those with high GAD symptom severity with average EC, worry becomes more verbal as one continues to engage in worry while for an individual with average GAD symptom severity, worry tends to become less verbal over time. There was also a positive trend of EC, suggesting that stronger cognitive control capacity allowed more engagement in verbal thoughts during worry.

Hypothesis 2B

When EC was measured using different performance-based tasks, results showed that as predicted, performance during these tasks was significantly positively associated with percentage of verbal thoughts during worry. Specifically, higher accuracy during the EC tasks predicted more verbal thoughts at the first trial of the worry period. It should be noted that this main effect

was uncovered only when two outliers who were more than 4 SDs away (-6.24 and -4.04 SDs respectively) below the mean in terms of accuracy were removed from the analysis. Given their extreme scores, it is probable that these participants failed to complete the EC tasks due to inattention or failure to understand the tasks. In any case, these findings should be considered tentative and interpreted with caution.

In addition, the interaction between GAD symptom severity and accuracy during EC tasks did not achieve significance. However, the fact that it did not is perhaps not surprising. This study's sample was predominantly selected to be high in GAD symptom severity. As such, the range of GAD symptom severity in the current sample was constrained, which reduces variance in the product term representing the GAD Symptom Severity x EC interaction. That, in turn, reduces statistical power to find an effect of the interaction (see McClelland & Judd, 1993). In essence, the interaction term becomes redundant with the EC main effect because it will not vary much beyond the variance produced by EC because the range of GAD symptom severity has been severely restricted. Instead, at high levels of GAD symptom severity, the Cognitive Control Model can be evaluated in terms of the EC main effect (controlling for remaining variance in GAD symptom severity). Provided that the EC main effect is significantly positive, the Cognitive Control Model would be supported. As such, the finding that there is a positive association between accuracy during the performance-based EC tasks and amount of verbal thoughts during worry supports the Cognitive Control Model.

Further, while reaction time during EC tasks did not have a significant average effect on percentage of verbal thoughts during worry, reaction time did interact with trial significantly. As mentioned before, while there was a negative trial main effect on percentage of verbal thoughts,

when trial was considered in interaction with reaction time during the EC tasks, the pattern of that interaction was as expected, faster reaction times were associated with *increasing* percentage of verbal thoughts over the worry task and vice versa. Further, while there was a marginally significant interaction between GAD symptom severity and reaction time in predicting percentage of verbal thoughts at the first trial, it should be noted that this marginal interaction became non-significant when a participant who was more than 6 SDs from the mean in terms of their reaction time was removed from the analysis. These findings provide further support for the Cognitive Control Model, and show that performance-based EC concord with self-reported EC in predicting increasing levels of verbal thought during worry.

Summary

In sum, the current findings provide support for the Cognitive Control Model. Self-reported EC as well as performance-based EC showed findings that were consistent with the idea that higher levels of EC should predict increased amount of verbal thoughts during worry. The general negative slope of amount of verbal thoughts over the worry period also suggests that while worry is predominantly verbal in nature, it may be an effortful process to maintain such a form of worry.

Dependent variable: AA ratings during worry

Hypothesis 1C

AA ratings were also assessed ten times during the worry task. Using a growth curve modeling approach, results revealed that the model that best represented how individuals rated their AA symptoms during worry was one that allowed individuals to start at different points in

their AA ratings, with the same negative linear growth rate. The pattern suggests that while everyone may rate their level of AA differently in the first worry trial, most individuals habituated to the worry induction and reported decreasing AA symptoms over time. The main goal of Hypothesis 1C was to investigate whether individual differences in GAD symptom severity and EC would predict individual differences in AA ratings throughout the worry period. This hypothesis postulated that GAD symptom severity would have a positive association with AA ratings while EC would have the opposite effect, and both may interact to predict AA ratings during worry. Consistent with our prediction, for the typical individual in the current sample, higher levels of GAD symptom severity were positively associated with AA ratings. This finding is also consistent with other studies showing that worry inductions lead to significant increases in reported anxiety in GAD samples or GAD analogues (Behar, Zuellig, & Borkovec, 2005; Hofmann et al., 2010; Llera & Newman, 2014). This result also accords with the finding that GAD symptom severity positively predicted self-reported AA over the past week (using the DASS-A), suggesting that a worry task may be a viable behavioral measure for assessing subjective AA.

Unexpectedly, while EC was associated with AA ratings during worry, this association was in the opposite direction expected. The current finding shows that EC is positively associated with AA ratings during worry. Furthermore, there was a three-way interaction between GAD symptom severity, EC, and trial. For individuals in the 90th percentile of GAD symptom severity, those with low EC reported significantly decreasing AA ratings over time, yet those with high EC reported overall higher AA ratings during worry which did not decline over time. The pattern for those with high GAD symptom severity and high EC is puzzling. AA

ratings show a pattern of habituation over time for the rest of the individuals in the study, while those with higher EC reported higher AA ratings at the start, and maintained higher levels of AA ratings over the ten trials. One possible explanation for this contrary finding is that individuals with higher EC showed more engagement with the worry task, and higher EC allowed them to stay engaged with the task over a longer period. Post-hoc analyses revealed that indeed, those with higher GAD symptom severity and higher EC reported they were more worried in contrast to those with lower EC who reported significantly declining worry ratings throughout the worry induction. Another possibility for this contrary finding is that AA ratings as assessed during the worry period do not overlap with AA symptoms as captured by the DASS-A either in content or duration. Initial analyses show that AA ratings during worry did not correlate significantly with the total score nor any individual item in the DASS-A. Further, AA ratings were assessed during a worry induction while self-reported AA symptoms as measured by the DASS-A targeted AA symptoms experienced in the past week. It could be that higher EC allowed participants with high GAD symptom severity to maintain worry as instructed by the experimenter, which may have increased their experience of AA symptoms. At the same time, higher EC may also allow those with high GAD symptom severity to terminate bouts of worry relatively quickly in their everyday life, and consequently experience lower AA symptoms in general. Indeed, it has been found that worry duration is a strong predictor of somatic symptoms (Brosschot & Van den Doef, 2006). Future studies should utilize ecological momentary assessments to obtain a more naturalistic assessment of the worry process, with a focus on assessing AA symptoms that overlap with the DASS-A as well as on the duration of worry.

Hypothesis 2C

The next hypothesis investigated whether performance-based EC and GAD symptom severity predicted associations with AA ratings during worry. Again, GAD symptom severity positively predicted AA ratings at the first trial. Further, as expected, accuracy during the performance-based EC tasks also negatively predicted AA ratings at the first trial. However, when two outliers who were more than 4 SDs from the mean were excluded from the model, accuracy during the performance-based EC tasks no longer negatively predicted AA ratings. As such, this finding should be interpreted with caution and other studies are needed to replicate this effect. Furthermore, the interaction between GAD symptom severity and accuracy achieved significance. The pattern of the interaction was in line with the model, such that GAD symptom severity positively predicted AA ratings at the first trial when accuracy during EC tasks was low, but was not associated with AA ratings when accuracy was high. The finding that there is a negative association between accuracy during the performance-based EC tasks and AA ratings also supports the Cognitive Control model.

On the other hand, reaction time during performance-based EC tasks negatively predicted AA ratings during worry at the first trial. Specifically, contrary to expectation, faster reaction time during these tasks was associated with higher AA ratings during worry at the start of the task. Further, the interaction between GAD symptom severity and reaction time during performance-based EC tasks significantly predicted AA ratings at the first worry trial, such that at high levels of GAD symptom severity, faster reaction time significantly positively predicted AA ratings, whereas slower reaction time did not significantly predict AA ratings. The discordance between the findings on accuracy versus reaction time on EC tasks in predicting AA

ratings again lend support to Eysenck's Attentional Control Theory (Eysenck et al., 2007), and highlight the importance of examining both in relation to anxiety, worry, and GAD. Such a finding again implies that higher EC may also increase engagement in the worry task, which result in increased AA ratings.

Summary

Overall, the best fitting model was one where individuals reported different levels of AA at the first trial and showed differing linear changes over the worry period. GAD symptom severity positively predicted AA ratings at the first trial. Further, the findings on self-reported EC and performance-based EC on AA ratings during worry are mixed. On the one hand, as expected, accuracy during the EC tasks negatively predicted AA ratings, and yet, in contrast to the Cognitive Control Model, higher self-reported EC as well faster reaction times during the EC tasks were positively associated with AA ratings during worry. The convergence of the findings on self-reported EC and reaction time during the EC tasks suggest more strongly that individuals with higher EC showed more engagement with the task, and higher EC allowed them to stay engaged with the task, thereby increasing AA ratings.

Dependent variable: Resting HR

Hypothesis 1D

The next hypothesis examined whether GAD symptom severity and EC interact to predict physiologically-measured AA, which was operationalized as resting HR. However, the results did not show any effect of GAD symptom severity, EC, nor their interaction, on resting HR. That GAD symptom severity is not related to resting HR is perhaps not surprising given that there

have been heterogeneity in the literature, with some showing that dispositional measures of worry are associated with high HR at rest or at waking (e.g. Brosschot, Van Dijk, & Thayer, 2007; Verkuil, Brosschot, Borkovec, & Thayer, 2009), and others showing that trait worry is not related to high HR (e.g. Dua & King, 1987; Lyonfields et al., 1995). However, the null findings in regard to EC's main effect on resting HR fails to provide support for the Cognitive Control model.

Hypothesis 2D

The next hypothesis examined whether GAD symptom severity and performance-based EC interact to predict resting HR. While accuracy during the EC tasks was significantly associated with resting HR, it was in the opposite direction expected, such that better performance in the EC tasks was associated with higher resting HR. Furthermore, reaction time during the EC tasks was not associated with resting HR nor was the interaction between GAD symptom severity and reaction time. These findings are difficult to explain given that low resting HR is generally associated with higher heart rate variability (HRV), which is an index of top-down cognitive control (Beauchaine & Thayer, 2016). Studies have shown that manipulating HRV influences cognitive performance (Thayer et al., 2009). In the context of this study, those findings suggest that higher cognitive performance should be associated with higher HRV, by extension, lower HR, but the results were the opposite of this prediction. That said, HR has been shown to correlate differently with different indices of HRV. While HR has been shown to correlate moderately with root mean square of successive differences (RMSSD), it showed weaker correlations with low frequency/high frequency ratio (LF/HF; Agelink et al., 2001). As

such, while examining HRV is beyond the scope of this study, further analyses should be done using HRV instead of HR.

Summary

In sum, the findings on resting HR suggests that subjective reports of AA symptoms do not concord with HR measured at rest. In the current study, correlational analyses showed no evidence of an association between the DASS-A and resting HR. This is perhaps to be expected as subjective reports of AA have been known not to concord with physiological measures of AA (Lang, 1985), which may explain why the interaction between GAD symptom severity and EC predicted subjective reports of AA but not resting HR. Other research suggest that GAD patients are especially attuned to fluctuations in objective AA such as HR and as such may over-report many symptoms ignored by healthy individuals (Andor et al., 2008). Thus, it is expected that there should be a de-coupling of objective and subjective measures of AA in GAD patients. Even so, given that there is heterogeneity in objectively-measured AA (e.g. Fisher, Granger, & Newman; Fisher & Newman, 2013; Vasey, Chriki, & Toh, 2016), the Cognitive Control Model predicts that GAD symptom severity and EC can still account for this heterogeneity. Yet, the current findings do not support this assertion. It is possible that resting HR collected in a lab setting may not be optimal for several reasons. First, resting HR may not be the best physiological measure that correlates with the experience of AA symptoms. Future studies should include other indices of AA, such as skin conductance level, salivary alpha-amylase, and blood pressure. Another possibility for the failure to uncover a significant effect of GAD symptom severity or EC is that perhaps trait measures of these constructs are not good predictors of resting HR. Additionally, future studies may instead want to examine state levels of these

constructs and their impact on resting HR. Other possibilities for the null findings include the fact that GAD symptom severity's effect on resting HR is best detected when HR is assessed during waking hours (Brosschot, Van Djik, & Thayer, 2007).

Dependent variable: Phasic HR

Hypothesis 1E

Hypothesis 1E investigated GAD symptom severity and EC's effects on phasic HR during worry. As with other outcomes assessed during the worry task, HR was measured during the 30-second worry trials. Exploratory findings using growth curve modeling revealed that the best fit for phasic HR trajectory after controlling for the 5-minute pre-worry resting HR was one that allowed individuals to vary in HR at the first trial, with no changes in HR over time during the worry task. As such, individual differences in overall mean HR during the worry period relative to the average mean HR during the pre-worry period was the best fit for the data.

Interestingly, a one standard deviation increase in GAD symptom severity was significantly associated with a .40 beats per minute (BPM) *decrease* in phasic HR for the typical individual with average pre-worry HR and EC in the sample. This finding is consistent with Borkovec's CognAv model which posits that worry has a suppressive effect on AA. When examining the overall levels of phasic HR during worry, high GAD symptom severity was associated with lower HR compared to low GAD symptom severity. Again, such a pattern is in line with Borkovec's CognAv model, which postulates that worry's suppressive effect on AA is negatively reinforcing to those with high GAD symptom severity. This finding also concords

with Borkovec & Hu's (1990) study where a worry induction was shown to produce reduced HR response relative to baseline compared to a relax condition.

Contrary to expectations, there was no negative main effect of EC. Further, while there was an interaction between GAD symptom severity and EC in predicting phasic HR, the interaction was in the opposite direction predicted. High GAD symptom severity was less strongly negatively related to HR when EC was high than when EC was low after controlling for pre-worry HR. However, the simple slopes were not significant, suggesting perhaps that these findings are tenuous and the error terms may be too large. It suggests also that the differences between low and high EC is negligible at high GAD symptom severity given that neither points are significantly different from zero.

Hypothesis 2E

While accuracy during the performance-based EC tasks did not influence phasic HR, reaction time was associated with phasic HR during worry at the first trial. However, the pattern of the effect was again in the opposite direction expected, such that faster reaction time was related to higher phasic HR. This indicates that relative to those with slower reaction time, when an individual had faster reaction time during the EC tasks, they also showed a bigger increase in HR from baseline to the worry task.

Summary

In sum, the findings did not show a negative main effect of self-reported or performance-based EC on phasic HR. The prediction was that higher levels of EC will be associated with lower phasic HR, however there was no evidence of such. In fact, significant interactions

between GAD symptom severity and self-reported or performance-based EC showed opposite patterns. Such findings are in direct contrast to the prediction of the Cognitive Control Model. Failure to find the expected effects may be the result of multiple possibilities discussed earlier regarding the use of HR: the discordance between subjective and objective AA, especially in the recall period length, the need to use other objective measures of AA, or the use of trait measures to predict state levels of objective AA. Additionally, given that GAD symptom severity has a suppressive effect on phasic HR, but in combination with high EC produces positive effects on phasic HR, suggests that higher EC allowed participants with high GAD symptom severity to engage more strongly in the worry task as instructed by the experimenter, which may have increased phasic HR. However, such an explanation is contraindicated by the CognAv model. Furthermore, contrary to other studies that found a negative correlation between percentage of verbal thoughts and HR during a worry induction (Borkovec et al., 1993), the current study did not uncover such a correlation.

Further, while in opposition to the Cognitive Control Model, these findings concord with the abovementioned results that GAD symptom severity and EC interact to predict AA ratings during worry. Individuals with high GAD symptom severity and high EC are reporting higher AA ratings as well as showing increased HR, suggesting that concordance between subjective and objective AA can be achieved when recall is close enough in time.

Hypothesis 3 Moderated mediation tests

Since GAD symptom severity was found to interact with self-reported EC as well as reaction time during performance-based EC tasks to predict AA symptoms (Hypothesis 1A and 2A) and percentage of thoughts during worry (Hypothesis 1E and 2E), two tests of moderated

mediation were conducted. The prediction was that the relationship between GAD symptom severity and AA symptoms would differ as a function of EC through percentage of verbal thoughts. However, there was no evidence of a moderated mediation when self-reported EC nor reaction time on EC tasks were used as the moderator variable. It should be noted that this finding is in contrast with findings from a self-reported study where higher GAD symptom severity was less strongly related to AA symptoms when EC was higher by virtue of increased percentage of thoughts during worry (Toh & Vasey, 2017). There are several notable differences between that study and the current study. First, that study utilized a large unselected sample (N ~ 1000), while the current study had a sample size of 198. Second, the current study selected for individuals high in worry and attempted to oversample for those with high and low EC. Interactions are best uncovered when there are many individuals with both high and low worry with high and low EC. Consequently, the interaction in the current study cannot account for much variance because there were much fewer individuals in the low worry quadrant. Finally, the use of trait-level variables (i.e. GAD symptom severity, self-reported EC, self-reported AA) in the same model as state-level variables (i.e. performance-based EC, percentage of verbal thoughts during the worry task) may have posed difficulties in uncovering an effect in the current study. Future studies should attempt to keep the variables consistent at the state or trait level.

Overall summary of results

In conclusion, the current study sought to provide evidence for the Cognitive Control Model. The model posits that there is heterogeneity in level of AA symptoms in worry and GAD, and that this heterogeneity is accounted for by individual differences in amount of verbal thoughts during worry, which in turn is supported by cognitive control capacity. The current

study aimed to build on the model by utilizing methods beyond self-report. The findings provide mixed support for the model.

Results that supported the model include GAD symptom severity's positive and self-reported EC's negative effect on self-reported arousal over the past week. However, these findings could not be extended to behavioral measures of EC. Second, the findings regarding amount of verbal thoughts during worry support the Cognitive Control Model, with self-reported and behavioral measures of EC showing positive associations with percentage of verbal thoughts during worry. Third, while accuracy during the EC tasks negatively predicted AA ratings during worry as expected, self-reported EC and reaction time during the EC tasks provided evidence contrary to the Cognitive Control Model, showing instead positive associations with AA ratings during worry. Other results that contradicted the model include those examining resting and phasic HR during worry. The findings reveal that self-reported or behavioral measures of EC either failed to significantly predict resting and phasic HR or significantly predicted resting and phasic HR in a direction that contradicts the model's predictions. Furthermore, the current study failed to uncover significant moderated mediations in the relation between GAD symptom severity, EC, amount of verbal thoughts, and AA.

Limitations and future directions

Findings from the current study must be viewed in light of several limitations. Mixed results regarding AA show two problems that need to be addressed. First, the discordance in results when using self-reported AA symptoms in the past week and during the worry task suggest that there may be recall effects or that the worry task failed to capture the way in which worry occurs in everyday life (especially in the duration of worry). As such, future studies

should utilize ecological momentary assessments to obtain a better understanding of the worry process.

Secondly, objective AA was operationalized only as HR. In fact, the autonomic nervous system is multiplicitous in nature with many central nervous system efferents, and generalization of one form of measurement to the ANS is difficult (Berntson, Cacioppo, & Quigley, 1991). Other indices of ANS activity have been used in studies of worry and GAD, including electrodermal activity in the form of skin conductance level or non-specific skin conductance responses, salivary alpha-amylase (Fisher et al., 2010), and future studies may want to include these indices. That being said, the current study's failure to find significant effects on HR is contraindicated by other studies finding that HR is reliably linked to trait or experimentally-induced worry (Borkovec & Inz; Lyonfields et al., 1995). Further, a meta-analysis showed that perseverative cognition (i.e. worry and rumination) is in general associated with increased sympathetic nervous system activity, which includes HR (Brosschot et al., 2006). Another meta-analysis with 60 studies found that perseverative cognition was associated with higher HR ($g = .28$; Ottaviani et al., 2016), although it looked only at healthy subjects.

Furthermore, HR is under the influence of both the sympathetic and parasympathetic nervous system (S/PNS). Both activation of the SNS and withdrawal of the PNS can result in increase in HR. The most well-supported index of PNS activity is heart rate variability (HRV). Because HRV was not examined in the current study, one possibility for the null findings regarding objectively-measured AA is that autonomic activation during worry is more strongly facilitated by PNS withdrawal. Further, PNS withdrawal acts much more quickly than SNS activation (Thayer & Lane, 2002), and given the structure of data collection in the current study

(i.e. HR measured in 30-second blocks after 30 seconds of worrying), it is plausible that HRV would have been a better measure of AA. Given the current study's failure to find significant effects using HR, future studies should aim to utilize other physiological measures of AA such as HRV. Alternatively, studies should include assessment of resting HR during waking hours (Brosschot, Van Djik, & Thayer, 2007) instead of in the lab. Other possibilities also point to use of state rather than trait measures of worry and EC in examining their effect on HR.

Third, while the current study oversampled for individuals with high GAD symptom severity who fall in the two corners of the dimensions of EC, the next step is to generalize these findings to a clinical populations such as GAD patients. On the other hand, the current state of the literature indicates that there are no clear boundaries between the normal range of worry/GAD symptoms and the clinical range. Taxometric studies seeking evidence for discontinuity consistently suggest instead that worry and GAD symptoms are continuously distributed in the population (Olatunji et al., 2010; Ruscio, Borkovec, & Ruscio, 2001; Ruscio, 2002). Excessive and uncontrollable worry still poses some level of impairment in their lives for many who may not (ever or usually) meet DSM criteria for GAD. As such, while it is important to use a clinical sample, a sample including those with high levels of worry/GAD symptom severity is still of value.

Fourth, the cross-sectional nature of our data precluded testing predictions the model makes over time. Future studies should include prospective data to permit tests of the impact of changes in AA symptoms on later worry, the impact of worry on cognitive control resources over time, and the impact of depletion of such resources due to stress on the level of AA resulting from worry. For example, a study looking at fourteen-year-olds collected data at multiple points

within a day, and found that worry predicted decreases in working memory, which predicted increases in worry (Trezise & Reeve, 2016). Such a study design would allow examination of the effect of worry and cognitive control capacity on the amount of verbal thoughts, as well as on AA over time.

This study is also limited by its correlational design and that it will be important for future research to include laboratory-based paradigms in which cognitive control resources are experimentally manipulated. As such, another important avenue of research would be to examine the causal predictions the model makes by experimentally manipulating worry (through a worry induction) or cognitive control resources (measured under load or stress) to examine their effects on verbal thoughts as well as objective and subjective AA. Other studies have done this to good effect and found that when asked to worry verbally, high worriers performed more poorly on a working memory task (Leigh & Hirsch, 2011).

The next studies to extend this line of research should also endeavor to use other behavioral measures of EC. The mixed results from using performance-based EC measures in the current study suggests that other behavioral tasks should be included. Further, it might be useful to include measures that include emotional stimuli that are in verbal form given that GAD patients evidence the strongest bias to threat using this form of threat (Goodwin, Yiend, & Hirsch, 2017). Another important avenue for future research is to parse out differences in performance effectiveness versus efficiency, as suggested by Eysenck's Attentional Control Theory (Eysenck et al., 2007).

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