

# Cognitive Control and Anxious Arousal in Worry and Generalized Anxiety: An Initial Test of an Integrative Model

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**Abstract** Pathological worry is thought to persist, in part, because it fosters avoidance of unpleasant internal experience. However, current models disagree as to whether worry serves that function because: (1) it suppresses high levels of autonomic arousal (AA) symptoms or (2) it leads to persistent heightened AA symptoms. In fact, pathological worry and generalized anxiety disorder (GAD) are linked to both high and low levels of AA symptoms. To account for this heterogeneity, we propose an integrative model, which predicts that AA symptoms vary as a function of the worrier's capacity to perform the subtle cognitive maneuver necessary to avoid such arousal. We review evidence supporting this model and report an initial test in a large college sample. Our results provide clear support for the major premise of our model: AA symptoms in worry and GAD vary as a function of individual differences in cognitive control capacity.

**Keywords** Autonomic arousal · Cognitive avoidance · Effortful control · Generalized anxiety disorder · Negative affect intensity · Worry

Excessive and uncontrollable worry is the hallmark of generalized anxiety disorder (GAD; American Psychiatric

Association (APA) 2013). Until recently there was consensus that such worry persists, in part, because it fosters avoidance of unpleasant internal experience (see Behar et al. 2009). This idea was first advanced by Thomas Borkovec in his cognitive avoidance (CognAv) model of pathological worry and GAD (see Borkovec et al. 2004). The CognAv model casts worry, in part, as a strategy for avoiding threatening images and the autonomic arousal (AA) they induce. Although this model has considerable support (see Behar et al. 2009), other evidence links worry and GAD instead to *high levels* of AA. This led Newman and Llera (2011) to formulate their contrast avoidance (ContrAv) model, which casts worry as a way to engender and maintain a chronic state of heightened negative emotionality (NE) and AA. Worriers are thereby reinforced by avoidance of the aversive contrast produced by unpredictable spikes in such emotion that would otherwise result if a feared event were to occur during a euthymic mood state.

Newman and Llera have made a strong case for their model (Llera and Newman 2010, 2014; Newman et al. 2013; Newman and Llera 2011). Nevertheless we believe there remains a place for the CognAv model. In this regard, we contend that it is crucial to distinguish between NE, a dimension capturing general psychological distress, which includes worry (Sylvester et al. 2011), and AA, a dimension capturing the tendency to experience autonomic hyperactivity. As shown by a recent meta-analysis, these dimensions are only modestly positively correlated ( $r = .14$  to  $.32$ ; Sylvester et al. 2011). Thus, consistent with Clark and Watson's (1991) tripartite model, NE and AA are related yet distinct constructs. Indeed, they appear to have distinct neural correlates (Sharp et al. 2015). Moreover, evidence suggests that high levels of NE, including high levels of worry, can inhibit AA reactivity (Craske et al. 2009; Lang

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and McTeague 2009; McTeague and Lang 2012). Nevertheless, that is clearly often not the case (Newman et al. 2013). Thus, there is considerable heterogeneity in the level of AA among those high in worry/GAD symptoms (see below), which requires explanation. In contrast, given that worry is an aspect of NE, relatively little heterogeneity in NE symptoms should be expected among such individuals.

Whereas the CognAv model cannot account for evidence linking worry and GAD to higher levels of AA, neither can the ContrAv model easily accommodate evidence that worry and GAD are characterized by lower levels of such symptoms. In the current study, we provide an initial test of an integrative model, which posits that the observed heterogeneity in AA in worry and GAD reflects the operation of a previously unconsidered moderator. In short, we propose a cognitive control model of AA heterogeneity in worry and GAD, which predicts that heightened AA can be avoided when worriers have sufficient cognitive control capacity to perform the subtle avoidance maneuver emphasized by the CognAv model. Absent such capacity, worry should instead activate AA. In contrast, worry should be associated with similar high levels of NE, regardless of level of cognitive control.

Before the fourth edition of the Diagnostic and Statistical Manual (DSM-IV; APA 1994), AA symptoms were among the diagnostic criteria for GAD. In the revised third edition of the DSM (DSM-III-R; APA 1987) GAD was defined by excessive worry accompanied by at least 6 of 18 symptoms from three clusters, including *autonomic hyperactivity* (e.g. shortness of breath, accelerated heart rate). However, such symptoms were dropped from the diagnosis in DSM-IV (APA 1994) and remain absent in DSM-5 (APA 2013). That decision was based in part on evidence that GAD patients infrequently endorse AA symptoms (e.g., Marten et al. 1993).

Consistent with this view, many studies have found weak associations between GAD diagnostic status and AA symptoms. For example, GAD status/severity and worry are often not correlated with measures of AA symptoms (e.g. Brown et al. 1998; Brown and McNiff 2009) and such symptoms sometimes fail to distinguish GAD patients from controls (e.g., Leyfer et al. 2006). Similarly, in a structural model of anxiety disorder diagnoses, GAD was unrelated to AA symptoms and that correlation became significantly *negative* when controlling for symptoms of NE (Brown et al. 1998). On the other hand, considerable evidence links worry and GAD to heightened AA symptoms. For example, a substantial percentage (20–50 %) of GAD patients report elevated AA symptoms (e.g. Marten et al. 1993; Starcevic and Bogojevic 1999). GAD samples also often score significantly higher than controls on measures of such symptoms (e.g. Aldao et al. 2012; Hoehn-Saric et al. 2004).

Moreover, GAD is sometimes comorbid with panic disorder (PD) and vice versa (e.g. Brown and Barlow 1992; Tull et al. 2009). Indeed, although some studies have found GAD samples to report significantly lower AA symptoms than PD samples (e.g. Leyfer et al. 2006), others have found such symptoms to be similarly elevated in both diagnoses (e.g. Brown et al. 1995).

Although subjective reports of AA may not show concordance with physiological measures of AA (Lang 1985),<sup>1</sup> this heterogeneity can also be found in studies using such measures. On one hand, many studies have found that physiological measures of AA are not elevated in GAD samples relative to controls at baseline (e.g., Fisher et al. 2010; Fisher and Newman 2013; Llera and Newman 2014) and in worriers relative to non-worriers (e.g. Davis et al. 2002; Delgado et al. 2009). Nevertheless, GAD samples in other studies score significantly higher than controls on such measures at baseline (e.g., Fisher and Newman 2013; Pruneti et al. 2010; Thayer et al. 1996). Similarly, in a general population sample worry was positively associated with heart rate (HR; Brosschot et al. 2007). This heterogeneity is also seen in neuroimaging studies. GAD samples either show significantly less or do not differ from controls in amygdala activation in response to threat stimuli (e.g. Blair et al. 2012; Monk et al. 2006). Yet, other studies have found that GAD samples show significantly *higher* amygdala activity than controls when processing threat cues (e.g., McClure et al. 2007; Monk et al. 2008).

Some GAD samples also show either no more AA in response to emotional provocation than controls or they show less (e.g. Grillon et al. 2009; Hoehn-Saric et al. 1989). Lang and McTeague (2009) reported that GAD patients show blunted startle response compared to the heightened startle seen in other anxiety disorders. The same is true for high versus low worriers (Delgado et al. 2009). Yet, in other studies GAD samples exhibit higher AA symptoms in response to emotion provoking stimuli than controls (e.g. Pruneti et al. 2010; Thayer et al. 2000). Similarly, worry inductions have sometimes not led to significant increases in AA in those with GAD (e.g. Llera and Newman 2014) and in high and low worriers (e.g. Delgado et al. 2009). Nevertheless, such inductions in other studies have significantly increased objective measures of AA (e.g., Thayer et al. 1996).

Of particular importance, several studies have found that worry blunts AA in response to fear-provoking imagery (Borkovec and Hu 1990; Borkovec et al. 1993). However, other studies have found that worry did not suppress AA in response to fearful stimuli, at least not in absolute terms

<sup>1</sup> However, evidence suggests that these response domains show higher concordance among individuals with high trait anxiety, which is a close correlate of worry (Calvo and Miguel-Tobal 1998).

(e.g. Llera and Newman 2014; Peasley-Miklus and Vrana 2000). Rather, fear provoking-imagery only failed to produce an increase in HR in comparison to the induction period in which worry had already significantly increased HR from baseline.

In summary, there is a striking pattern of heterogeneity in levels of AA at baseline and in response to worry and fear-provoking imagery among high worriers and in GAD samples. Indeed, even in studies finding elevated AA symptoms on average in GAD samples, the range of scores often includes very low levels (e.g. Hoehn-Saric et al. 2004; Leyfer et al. 2006). Similarly in studies finding low levels of AA symptoms in GAD samples, the range frequently includes very high levels (e.g. Fisher et al. 2010). We are not the first to note this heterogeneity. Hoehn-Saric and Masek (1981) reported findings they interpreted as “indicating the existence of a subgroup of generalized anxiety patients with low autonomic reactivity” (p. 1041). Similarly, Fisher et al. (2010) posited “the existence of separate GAD taxons, one with and one without accompanying physiological arousal symptomatology” (p. 191) and suggested that such heterogeneity could “create rival statistical conclusions resulting from drawing from one subset versus another” (p. 192). Based on our review of the evidence we too believe there are two GAD phenotypes, which differ markedly in their level of AA. Unfortunately, although the CognAv model and the ContrAv model each offer critical insight into the functions of worry, it is unclear how either model alone can account for both phenotypes. However, closer consideration of the CognAv model suggests a possible path to an integrative model.

Drawing on Lang’s (1985) bioinformational theory of fear, the CognAv model views worry as a predominantly verbal thought process, which serves to suppress the AA normally triggered by imaginal processing of fear-provoking possibilities. Visual images of feared stimuli are more likely to activate AA responses than verbal thoughts about such stimuli (e.g., Tucker and Newman 1981; Vrana et al. 1986). Furthermore, evidence suggests that people spontaneously shift from imagery to verbalization to reduce AA when processing aversive material (Borkovec et al. 1998; Tucker and Newman 1981). There is also evidence that verbal thoughts predominate over imagery during worry (Borkovec et al. 1993; Freeston et al. 1996), especially in GAD patients (Hirsch and Mathews 2012). Worry is also characterized by a predominance of left-frontal cortical activity (e.g. Hofmann et al. 2005; Wu et al. 1991), which has been linked to verbal thought (Pinker 1994; Tucker 1981). Nevertheless, other studies have failed to find that verbal processing predominates over imagery during worry (e.g. Borkovec et al. 1993; Stapinski et al. 2010). Furthermore, it appears that differences in the extent to which verbal worry predominates can account for

differences in the extent of AA experienced. For example, Borkovec et al. (1993) found that the percentage of verbal worry reported by participants in their worry condition was significantly negatively correlated with HR response whereas in the relaxation condition, percentage of imagery was significantly positively correlated with HR response. Thus, it appears that heterogeneity in AA symptoms may depend on the extent to which verbal or imaginal processing predominates during worry.

As stated by Borkovec et al. (2004), “...when aversive images occur in the process of worry...the shifting of attention to [verbal] worrisome thinking upon each occurrence...results in escape from or avoidance of the somatic element of the fear response...” (p. 83). As noted by Price and Mohlman (2007), to perform this maneuver, worriers and those with GAD must have “a degree of cognitive dexterity in order to direct attention narrowly” (p. 2629). Similarly, although Hirsch and Mathews (2012) postulate that deficient capacity for top-down control contributes to risk for pathological worry, they also explicitly acknowledge that top-down attentional control resources can be misdirected, fostering protracted worry in verbal form. Thus, the AA avoidance through verbal worry emphasized by the CognAv model requires some degree of proficiency in the effortful control of attention. This may seem surprising since worry and GAD are generally thought to be associated with deficits in attentional control (AC), working memory and other aspects of executive control (e.g. Armstrong et al. 2011; Borkovec et al. 1983). However, as discussed below, evidence points to considerable heterogeneity among worriers and GAD patients in their capacity for such cognitive control. We propose that this heterogeneity may account for the two patterns of AA symptoms seen in worry and GAD. In short, a worrier with high cognitive control capacity should have greater success in making and maintaining the shift to a verbal mode of threat processing posited by the CognAv model, thereby limiting activation of AA. In contrast, a worrier low in such capacity should have difficulty performing or maintaining such a shift, instead processing threat possibilities predominantly as images, resulting in heightened AA, in keeping with the ContrAv model.

One construct that may capture this type of cognitive control capacity is the temperament dimension of effortful control (EC), which is a broad self-regulatory construct encompassing the capacities for attentional, inhibitory, and activation control (Rothbart 2007). A variety of evidence suggests that worriers and GAD patients vary widely in their self-reported levels of EC. For example, whereas some studies have found moderately strong negative correlations between GAD status and self-reported AC (e.g., Armstrong et al. 2011; Olatunji et al. 2011), other studies have found non-significant associations (e.g., Bienvenu

et al. 2004) or even positive association between worry/GAD status and EC or similar constructs. In a large clinical sample, Rosellini and Brown (2011) found that a structural model of symptoms revealed that GAD symptom severity was significantly *positively* correlated ( $r = .12$ ) with conscientiousness (C), a construct which is closely related to EC (Roberts et al. 2014).

Studies using performance-based measures of cognitive control have also yielded a mixed picture. For example, GAD patients are significantly less accurate in target detection in general (Olatunji et al. 2011), and perform worse than controls on neuropsychological tests of attention and cognitive flexibility (Gualtieri and Morgan 2008). On the other hand, Hoehn-Saric et al. (1989) found no difference between GAD patients and controls in performance on a divided attention task and Yiend et al. (2014) found GAD patients to show significantly *faster* disengagement from angry and fearful faces than controls in two separate studies. Although Yiend and colleagues did not measure EC, other evidence suggests their findings may reflect that construct's influence. Derryberry and Reed (2002) found high trait-anxiety (a close correlate of worry) was linked to delayed disengagement from threat cues only among individuals reporting low AC. Others have reported similar results (e.g., Lonigan and Vasey 2009).

Heterogeneity in cognitive control capacity in worriers and GAD samples is also supported by neuroimaging and psychophysiological studies. Price et al. (2011) found GAD patients to show hypoactivity in the prefrontal cortex (PFC) compared to controls during an emotional Stroop task. However, other studies have found no effect of GAD status on such activity or even that worry or GAD status are *positively* associated with cognitive control. For example, Etkin et al. (2009) found that, unlike controls, GAD patients exhibited an atypical heightened functional connectivity at rest between the amygdala and the dorsolateral (dl)PFC, a region that is associated with self-regulatory functions. Similarly, Mohlman et al. (2009) found medial orbital PFC volume to be *positively* associated with PSWQ scores in older adults with GAD and controls. Moreover, whereas some studies have found heart rate variability (HRV) at rest (a physiological index of the capacity for top-down control [Thayer et al. 2009]) to be lower as a function of worry or GAD status (e.g., Brosschot et al. 2007; Thayer et al. 1996), other studies have not (e.g. Aldao and Mennin 2012; Knepp and Friedman 2008). Indeed, Davis et al. (2002) found that high worriers had significantly higher HRV at rest than controls (study 2). Similarly, whereas several studies have found that a worry induction significantly reduces HRV (e.g., Delgado et al. 2009; Thayer et al. 1996) others have not (e.g. Knepp and Friedman 2008; Lyonfields et al. 1995).

Such individual differences in cognitive control capacity in GAD and worry are noteworthy because executive function and level of AA appear to be related. For example, in a study of healthy older adults, Beaudreau and O'Hara (2009) found that scores on the Beck Anxiety Inventory (BAI; Beck and Steer 1990), which predominantly taps AA symptoms (Cox et al. 1996; Leyfer et al. 2006), were negatively correlated with performance on executive function measures. Furthermore, in addition to finding an atypical pattern of functional connectivity between the amygdala and the dlPFC in GAD patients, Etkin et al. (2009) found that the strength of that connectivity was significantly *negatively* associated with scores on the BAI. Consequently, they concluded that at least some GAD patients exhibit habitual engagement of an executive control system to regulate AA. Consistent with this view, recent evidence suggests that individual differences in AC moderate the level of AA experienced by high trait-anxious individuals in response to a CO<sub>2</sub> inhalation challenge (Richey et al. 2012). Specifically, this study found that trait anxiety was significantly positively associated with fear during such a challenge when AC was low but that association reversed to become significantly negative when AC was high.

In summary, we propose that individual differences in cognitive control capacity can account for the heterogeneity in level of AA among pathological worriers. Specifically, our model predicts that the positive association between worry/GAD symptoms and AA should be weakest among individuals with good capacity for cognitive control (specifically EC in the current study). Absent such capacity, worry/GAD symptoms should be strongly positively correlated with AA. Providing an initial test of this prediction was the primary aim of this study, which was based on a large sample combining two archival samples in which measures of all relevant constructs were available and in which we were able to identify an analog GAD subsample. First, we predicted that EC should moderate the positive association between worry/GAD symptom severity and AA such that it is strongest when EC is low and weakest (and perhaps even reversed) when EC is high. That should be true in a broad college sample and in a subsample meeting criteria for GAD. We focused on worry as well as GAD symptom severity because one can endorse high worry without meeting GAD diagnostic criteria or endorsing high levels of GAD symptoms severity (Ruscio 2002). Second, we predicted that, despite its potential to be misallocated in service of worry, high EC should, on average, also protect against worry becoming excessive and uncontrollable (see Hirsch and Mathews 2012). Specifically, EC should moderate the association between worry and both GAD symptom severity and GAD status.

Also, as reported in the online supplement, we conducted several ancillary analyses. First, EC's moderating influence should be specific to AA symptoms. Thus, EC should *not* moderate the association between worry/GAD symptom severity and NE in either the full sample or in the GAD subsample. Second, we conducted exploratory analyses to determine if EC also moderates the positive association between worry/GAD symptom severity and depressive symptoms in both the full sample and the GAD subsample. Finally, because all variables were collected concurrently and the direction of effects was unclear, we considered alternate models in which worry conditionally predicts EC as a function of AA symptoms and AA symptoms conditionally predict worry as a function of EC.

## Methods

### Participants and Procedure

Participants were 1343 undergraduate students at a large Midwestern university ages 18–41 years (mean age = 19.1 [SD = 1.9]; 56 % female) who received partial course credit for participation. Most participants were Caucasian (82.2 %), with 5.7 % African–American, 6.5 % Asian/Pacific Islander, 3.3 % Hispanic, .2 % Native American, and 2.1 % other. Data were collected across multiple academic quarters. This study combines two samples of introductory psychology student and participants were recruited in two ways. The majority (1200) were unselected volunteers, whereas the remaining 143 were identified via screening with the Effortful Control Scale—Persistence and Low Distraction subscale (ECS-PLD; Lonigan and Phillips 1998) and the Positive and Negative Affect Schedule (PANAS—Trait Version (Clark and Watson 1988) as described for sample 2 in Vasey et al. (2014).<sup>2</sup> Twenty participants were dropped due to missing data. According to Little's MCAR test, these data were missing completely at random ( $\chi^2 = 85.71$ ,  $p = .166$ ). This resulted in a final sample of 1323 individuals.

Following procedures approved by the Institutional Review Board (IRB) of the Ohio State University, participants completed all measures as part of a larger, randomly ordered set of measures. The majority of participants (i.e.,

<sup>2</sup> Because individuals scoring above the median on NA and in the upper or lower quartile on the ECS-PLD scale were disproportionately invited to participate in this subsample, our total sample may have had somewhat more individuals than is typical having both high NE and either high or low EC. Thus, we likely had somewhat higher statistical power to detect an interaction involving variables associated with heightened NE (such as worry) and varying levels of EC than would be typical (see McClelland and Judd 1993). Consistent with this, 5.67 % of Sample 1 qualified for membership in our analog GAD group versus 10.64 % of Sample 2.

1200 [89 %]) completed the measures in groups of 30–40. The remainder completed the measures individually during a laboratory session.

### Measures

*Penn State Worry Questionnaire (PSWQ;* Meyer et al. 1990). The PSWQ is a self-report measure of pathological worry, which comprises 16 items rated on a Likert scale ranging from 1 (Not at All Typical) to 5 (Very Typical). The PSWQ has excellent psychometric properties (Meyer et al. 1990).

*Affect Intensity Measure (AIM;* Larsen 1984). We used the 29-item version of the AIM developed by Weinfurt et al. (1994), which yields four scales measuring positive and negative emotional reactivity and intensity. Participants rate the extent to which each item describes them on a 5-point Likert scale ranging from 1 (Never) to 5 (Always). In this study we considered only the 6-item negative intensity scale (AIM-NI), which has been used in past studies of NE intensity in GAD (e.g. Mennin et al. 2005; Mennin et al. 2009). The AIM-NI has good psychometric properties (Bryant et al. 1996; Weinfurt et al. 1994).

*Effortful Control Scale (ECS;* Lonigan and Phillips 1998). The ECS 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 respondent 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 we focused on the ECS-PLD subscale (hereinafter labeled EC), which focuses on attention control and the capacity to persist in activities despite reactive motivation to avoid. The ECS-I focuses on inhibition of impulsive motor responses, which were irrelevant in the current context. Example items from the ECS-PLD 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 2014).

*The Depression, Anxiety, and Stress Scales (DASS;* Lovibond and Lovibond 1995). The DASS is a 42-item questionnaire comprising three 14-item subscales measuring symptoms of anxiety (DASS-A), stress and depression. Participants rate each item on a 4-point Likert scale ranging from 0 (Did not apply to me at all) to 3 (Applied to me very much, or most of the time) regarding how much the item applied to them over the past week. The current study focused on the DASS-A because it predominantly measures AA symptoms (Brown et al. 1998).<sup>3</sup> Example DASS-

<sup>3</sup> As described in the supplementary material, we also considered DASS depression (DASS-D) scores.

A items include “I felt I was close to panic” and “I was aware of dryness of my mouth”. The DASS scales have good psychometric properties (Lovibond and Lovibond 1995).

The *Generalized Anxiety Disorder Questionnaire for DSM-IV (GADQ-IV)*; Newman et al. 2002). The GADQ-IV uses items that closely follow *DSM-IV* (APA 1990) diagnostic criteria to screen for GAD. The GADQ-IV consists of five yes/no questions assessing the occurrence of excessive and uncontrollable worry and its duration, a question requesting a list of one’s most frequent topics of worry, a checklist of associated symptoms (e.g. muscle tension, irritability), and two questions regarding the severity of interference and distress resulting from worry and physical symptoms (see Newman et al. 2002). It has good test–retest reliability, convergent and discriminant validity, and good agreement with diagnostic interviews (Moore et al. 2014; Newman et al. 2002).

There are several ways to score the GADQ-IV. In its original form, participants were instructed to skip items 7–9 if their excessive worry has not lasted for at least 6-months (item 6). This approach results in scores varying from 0 to 7 for those who skip items 7–9 and from 0 to 13 for those completing those items. However, based on the findings of Rodebaugh et al. (2008), we chose to have all participants answer all of the questions and to include them in the total score even if the 6-month duration criterion was not met.<sup>4</sup> We otherwise scored the GADQ-IV according to Newman et al. (2002). We used the resulting continuous GADQ-IV score as a measure of GAD symptom severity (Rodebaugh et al. 2008).

## Data Analytic Strategy

Study hypotheses were tested through multiple linear regression (MLR) analyses. All continuous variables were standardized in the relevant sample or subsample. Thus, all main effects tested the effect of a predictor at average levels of the other variables in the model (Hayes 2013). Interaction terms were computed after the relevant variables were standardized.

Each MLR analysis involved two hierarchical steps. In Step 1, we included Sex along with the predictor (e.g., PSWQ) and moderator (e.g., EC) variables. All models included the AIM-NI as a covariate.<sup>5</sup> Step 2 added the interaction between the predictor and the moderator (e.g.,

PSWQ  $\times$  EC). Regression diagnostics were examined for each model to check for high influence cases. Specifically, for each model we examined the standardized DFFITS and DFBETA values using  $\pm 1.0$  as a cutoff (Cohen et al. 2002). No high influence cases were identified in any analysis.

Interaction effects were probed using the PROCESS utility for SPSS (Hayes 2013; freely available at <http://www.afhayes.com>). PROCESS implements the Johnson-Neyman technique for deriving regions of significance for the simple slope of the predictor at all observed values of the moderator (see Hayes 2013, pp. 307–315). For each significant interaction we report the region of significance in terms of standard deviations from the mean of the moderator, along with the percentile of the distribution corresponding to the point demarking the region of significance. PROCESS also estimates simple slopes at specific values of the moderator. Given the large size of the sample, we chose to illustrate all interactions by depicting simple slopes for each predictor at high (90th percentile) and low (10th percentile) levels of the moderator.

There are several approaches for identifying those likely to meet criteria for GAD based on the GADQ-IV (Moore et al. 2014). We chose to use the most stringent of these approaches, which does not rely on a score threshold but rather requires that DSM-IV criteria be met with at least moderate severity (see Moore et al. 2014). We chose to utilize this DSM-based approach because Moore et al. (2014) concluded it provides the best balance between sensitivity and specificity. To further increase specificity, we also required PSWQ  $\geq 70$  for membership in our GAD group. Various PSWQ scores have been proposed for identifying pathological worriers (e.g., 56 [Ruscio 2002] and 65 [Fresco et al. 2003]). However, in the current study we chose a cutoff score of 70 because it fell at the 90th percentile in our large sample. In contrast, cutoff scores of 56 and 65 identified 32.1 % and 15 % of the sample respectively and were deemed over-inclusive. Notably, a PSWQ score of 70 is higher than the typical average PSWQ score reported for GAD patient samples. As shown in Table 1, this approach resulted in an analog GAD group comprising 6.2 % of the total sample, in which the average PSWQ score was 75.5, which is well above the mean for analog GAD samples (i.e., 64.6) and clinical GAD samples (i.e., 67.16) as reported by Startup and Erickson (2006).

Membership in our non-anxious control group required a score below the threshold (i.e., 5.7) identified by Newman et al. (2002) when the GADQ-IV was scored according to their original skip-out rule. As shown in Table 1, this approach identified 72.0 % of the sample, resulting in a control group in which the average PSWQ score was 42.6, which falls within the range for non-anxious groups as described by Startup and Erickson (2006).

<sup>4</sup> The duration criterion has been called into question (e.g., see Andrews et al. 2010).

<sup>5</sup> The pattern of all results was unchanged if the covariate was omitted.

**Table 1** Descriptive statistics

Variable	Coefficient $\alpha$	Full sample N = 1323		Non-anxious sample N = 952 (72.0 %)		GAD sample N = 82 (6.2 %)	
		Mean	SD	Mean	SD	Mean	SD
PSWQ	.94	48.0	15.1	42.6	12.5	75.5	3.1
AIM-NI	.76	17.4	4.8	16.0	4.3	23.6	3.8
GADQ-IV score	.88	5.3	3.2	3.8	2.2	10.9	1.0
EC	.81	45.5	6.9	46.5	6.4	41.1	7.4
DASS-A	.88	6.9	7.0	4.9	5.2	16.5	9.4
DASS-D	.94	6.6	7.8	4.6	6.0	16.0	10.2
Sex	–	56.0 % female		50.0 % female		79.3 % female	

All variables, including sex, differ between the GAD and non-GAD samples at  $p < .001$

## Results

### Preliminary Analyses

Table 1 shows the mean and standard deviation (SD) for all variables for the full sample as well as for the GAD and control groups. Table 1 also shows Cronbach's coefficient alpha for all variables in the full sample. The GAD and control groups differed significantly on all variables, including the DASS-A and EC. Given the importance of heterogeneity in AA symptoms and EC in this study, it is notable that DASS-A and EC scores in the GAD group had higher SDs than were seen in the full sample. Most notably, DASS-A scores in the GAD group ranged from 0 to 40. Table 2 shows correlation coefficients in the full sample.

### Did EC Moderate the Link Between Worry/GAD Symptom Severity and AA Symptoms?<sup>6</sup>

*PSWQ.* As Table 3 shows, on average DASS-A scores were significantly positively predicted by the PSWQ and AIM-NI and significantly negatively predicted by Sex and EC. The PSWQ  $\times$  EC interaction was also significant. Examining the region of significance revealed that PSWQ scores significantly positively predicted DASS-A scores for EC  $< 1.71$  SDs (percentile = 97.8). That association was stronger when EC was low (simple slope [B] = .41,  $p < .001$ ) versus high (B = .14,  $p < .001$ ; see Fig. 1). From the opposite viewpoint, EC scores significantly negatively predicted DASS-A scores for PSWQ  $> -1.57$  SDs (percentile = 4.91). That association was stronger when PSWQ was high (B = -.41,  $p < .001$ ) versus low (B = -.11,  $p = .004$ ).

*GADQ-IV Score.* Table 3 shows the GADQ-IV score produced the same pattern of results as the PSWQ, with Sex, the GADQ-IV score, AIM-NI, EC, and the GADQ-IV  $\times$  EC interaction all achieving significance. GADQ-IV scores significantly positively predicted DASS-A scores across all

levels of EC. However, that association was stronger when EC was low (B = .49,  $p < .001$ ) versus high (B = .28,  $p < .001$ ; see Fig. 1). From the reverse perspective, EC scores significantly negatively predicted DASS-A scores for GADQ-IV Score  $> -1.36$  SDs (percentile = 5.21). That association was stronger when the GADQ-IV Score was high (B = -.32,  $p < .001$ ) versus low (B = -.09,  $p = .015$ ).

### Did EC Moderate the Association Between GAD Symptom Severity and AA Symptoms<sup>7</sup> in the GAD Group?<sup>8</sup>

As shown in Table 3, when the sample was limited to those individuals in the GAD group, on average DASS-A scores were significantly positively predicted by the GADQ-IV score, with a non-significant trend for them to be negatively predicted by EC. However, as predicted, the GADQ-IV Score  $\times$  EC interaction was also significant. Specifically, GADQ-IV scores significantly positively predicted DASS-A scores only for EC  $< .36$  SDs (percentile = 59.8 in the GAD group [equivalent to EC  $< -.25$  and percentile = 41.0 in the overall group]). As shown in Fig. 2, GADQ-IV scores significantly positively predicted DASS-A scores when EC was low (B = .93,  $p < .001$ ) but not when it was high (B = .01,  $p = .939$ ). From the opposite perspective, EC significantly negatively predicted DASS-A scores for GADQ-IV scores  $> .10$  SDs (percentile = 48.8 in the GAD group [equivalent to GADQ-IV Score  $> 1.76$  and percentile = 94.5 in the overall group]). That association was significantly negative when GADQ-IV scores were high (B = -.67,  $p = .002$ ) but not low (B = .38,  $p = .192$ ).

<sup>7</sup> See the online supplement for a test of the GADQ-IV Score  $\times$  EC interaction predicting DASS-D scores in the GAD group. The interaction was not significant.

<sup>8</sup> The PSWQ  $\times$  EC interaction was not tested in the GAD group due to the restriction of range on the PSWQ imposed by our approach to identifying members of the GAD group (i.e., one requirement was PSWQ  $\geq 70$ ).

<sup>6</sup> See the online supplement for consideration of alternate models.

**Table 2** Zero-order correlations

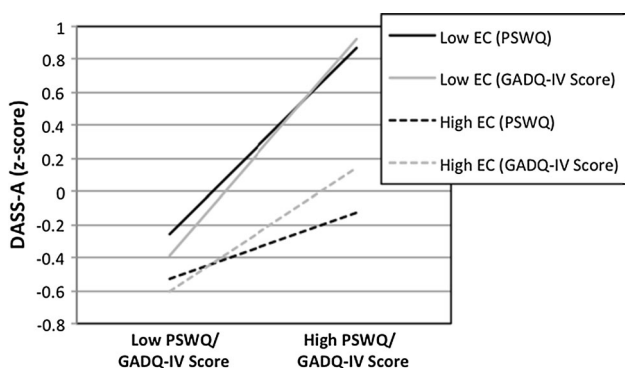
Sample/variable	PSWQ	AIM-NI	GADQ-IV score	EC	DASS-A	DASS-D	Sex
AIM-NI	<b>.65</b>	–					
GADQ-IV score	<b>.78</b>	<b>.61</b>	–				
EC	<b>–.26</b>	<b>–.28</b>	<b>–.35</b>	–			
DASS-A	<b>.49</b>	<b>.49</b>	<b>.57</b>	<b>–.40</b>	–		
Sex	<b>.32</b>	<b>.25</b>	<b>.27</b>	.02	<b>.10</b>	–	
DASS-D	<b>.42</b>	<b>.38</b>	<b>.53</b>	<b>–.46</b>	<b>.67</b>	.02	–
GAD status	<b>.47</b>	<b>.33</b>	<b>.45</b>	<b>–.17</b>	<b>.35</b>	<b>.31</b>	<b>.12</b>

N = 1323; sex: 0 = male, 1 = female; All bolded correlations significant at  $p < .01$

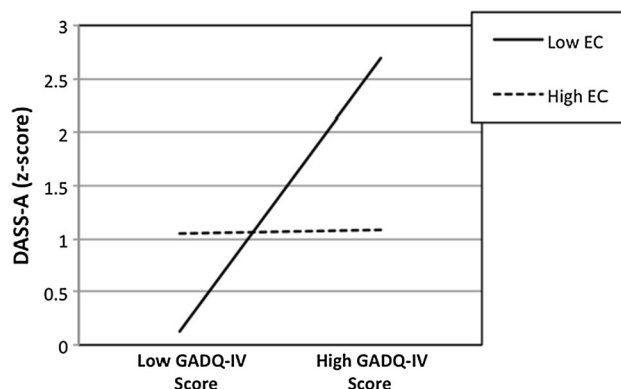
**Table 3** Regression model testing PSWQ/GADQ-IV score × EC predicting DASS-A in the full sample and in the GAD group

Step/variable	Full sample						GAD group		
	Predictor: PSWQ			Predictor: GADQ-IV score			Predictor: GADQ-IV score		
	R <sup>2</sup> /B	ΔR <sup>2</sup> /SE	Sr	R <sup>2</sup> /B	ΔR <sup>2</sup> /SE	Sr	R <sup>2</sup> /B	ΔR <sup>2</sup> /SE	Sr
Step 1	.354***	–		.402***	–		.165**	–	
Step 2	.367***	.012***		.409***	.008***		.229***	.064*	
Intercept	.03	.04		.04	.03		1.58***	.30	
Sex	–.10*	.05	–.05*	–.12**	.04	–.06**	–.33	.34	–.10
PSWQ/GADQ-IV score	.27***	.03	.20***	.38***	.03	.29***	.42**	.14	.30**
EC	–.25***	.02	–.24***	–.19***	.02	–.18***	–.24 <sup>†</sup>	.14	–.17 <sup>†</sup>
AIM-NI	.26***	.03	.19***	.21***	.03	.16***	.23	.14	.17
PSWQ/GADQ-IV score × EC	–.11***	.02	–.11***	–.09***	.02	–.09***	–.38*	.15	–.25**

N = 1323 for full sample; N = 82 for GAD subgroup. Sex: 0 = male, 1 = female; \*\*\*  $p < .001$ ; \*\*  $p < .01$ ; \*  $p < .05$ ; <sup>†</sup>  $p < .10$



**Fig. 1** Graph of the PSWQ/GADQ-IV score × EC interaction predicting DASS-A



**Fig. 2** Graph of the PSWQ/GADQ-IV score × EC interaction predicting DASS-A in the GAD group

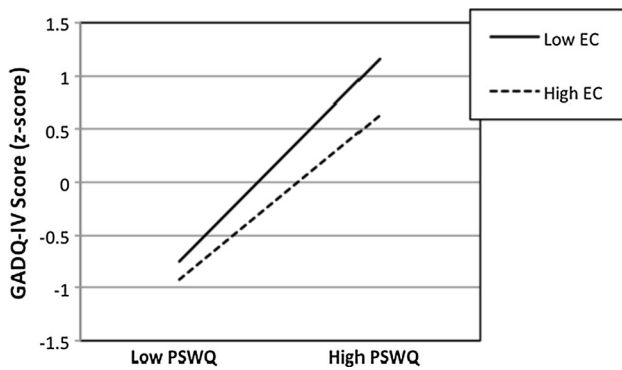
**Did EC Moderate the Association Between Worry and GAD Symptom Severity/GAD Status?<sup>9</sup>**

As shown in Table 4, on average GADQ-IV scores were significantly positively predicted by Sex, PSWQ and AIM-

NI and significantly negatively predicted by EC. The PSWQ × EC interaction was also significant. Examination of the region of significance revealed that PSWQ scores significantly positively predicted GADQ-IV scores at all levels of EC. However, that association was stronger when EC was low ( $B = .69, p < .001$ ) versus high ( $B = .56, p < .001$ ; see Fig. 3). From the reverse perspective, EC significantly negatively predicted GADQ-IV scores for  $PSWQ > -1.56$  SDs (percentile = 4.9). That association

<sup>9</sup> See the online supplement for analyses predicting DASS-D scores. EC significantly moderated the association between PSWQ/GAD symptom severity and DASS-D scores.





**Fig. 3** Graph of the PSWQ × EC interaction predicting GADQ-IV scores

was stronger when PSWQ was high ( $B = -.21, p < .001$ ) versus low ( $B = -.07, p = .009$ ).

Table 4 also shows that GAD status was significantly positively related to PSWQ scores and significantly negatively related to EC. However, as expected the PSWQ × EC interaction was also significant. Examination of the region of significance showed that PSWQ scores were significantly predictive of GAD status for  $EC < 1.74$  (percentile = 97.3 in the GAD/non-anxious subsample [equivalent to  $EC < 1.69$  and percentile = 97.3 in the overall group]). The predicted likelihood of GAD group membership was 31.5 % when PSWQ was high and EC was low whereas it was 15.6 % when both PSWQ and EC were high. From the opposite perspective, EC was significantly negatively associated with GAD status for  $PSWQ > 1.48$  (percentile = 91.6 in the GAD/non-anxious subsample [these values were the same in the overall group]).

### Discussion

This study’s goal was to provide an initial test of our cognitive control model of worry and GAD, which posits that cognitive control capacity acts as a moderator that

explains the well-documented heterogeneity in the level of AA linked to worry and GAD. Our results provide clear support for this model. First, as required by our model, our analog GAD group showed considerable heterogeneity in both AA symptoms and level of EC. Although the GAD group scored significantly higher on AA symptoms and lower on EC than the control group, their scores on both variables covered a wide range and had larger SDs than in the full sample. Indeed, DASS-A scores in the GAD group covered the entire range possible (i.e., 0–40). Second and most importantly, individual differences in EC in our GAD sample moderated the link between GAD symptom severity and AA symptoms. Specifically, as our model predicts, GAD symptom severity was strongly positively associated with AA symptoms when EC was low. Indeed, in that case GAD symptom severity at the 90th percentile predicted a level of AA symptoms falling nearly 2.75 SDs above the mean in the full sample. In contrast, GAD symptom severity was unrelated to AA symptoms when EC was high; the predicted level of AA symptoms was approximately one SD above the mean in the full sample regardless of GAD symptom severity level. In contrast, as reported in the online supplement, such a moderating effect was not found in our GAD group in the case of either NE intensity or depressive symptoms. Thus, the moderating effect of individual differences in cognitive control appears to be specific to AA.

The moderating effect of cognitive control capacity on the strength of the link between worry/GAD symptom severity and AA symptoms was also observed in the full sample. In the case of PSWQ scores, that association became non-significant when EC was very high (i.e.,  $EC > 1.71$  SDs). Furthermore, from the opposite perspective, EC’s negative correlation with AA symptoms was strongest when worry and GAD symptoms were most severe.

Despite its potential to be misallocated in service of worry, high capacity for top-down control should also, on

**Table 4** Regression table for model testing PSWQ × EC predicting GADQ-IV score and GAD status

Step/variable	DV: GADQ-IV score			DV: GAD status		
	R <sup>2</sup> /B	ΔR <sup>2</sup> /SE	Sr	Nagelkerke R <sup>2a</sup> /B	ΔR <sup>2</sup> /SE	Exp(B)
Step 1	.639***	–		.885***		
Step 2	.642***	.003**		.892***	.007*	
Intercept	-.05†	.03		-15.35***	2.57	.00***
Sex	.07†	.04	.03†	-.03	.71	.97
PSWQ	.62***	.02	.46***	8.52***	1.50	5010.14***
EC	-.14***	.02	-.13***	4.96*	2.22	142.20*
AIM-NI	.16***	.02	.12***	.24	.40	1.27
PSWQ × EC	-.05**	.02	-.05**	-15.35***	1.30	.04***

N = 1323 for GADQ-IV score; N = 1034 for GAD status. Sex: 0 = male, 1 = female

DV Dependent variable; \*\*\*  $p < .001$ ; \*\*  $p < .01$ ; \*  $p < .05$ ; †  $p < .10$

average, protect against worry becoming excessive and uncontrollable (see Hirsch and Mathews 2012). Consistent with that view, we found that EC significantly moderated the associations between worry and both GAD symptom severity and GAD status. Although worry was significantly positively predictive of GAD symptom severity across all levels of EC, that association was weakest when EC was high. Consistent with that pattern, an individual at the 90th percentile in worry was nearly twice as likely to be a member of the GAD group when EC was low versus high (i.e., 31.5 versus 15.6 %). Indeed, at very high levels of EC (i.e.,  $EC \geq 97.3$ rd percentile) worry scores were not significantly related to GAD status. As reported in the online supplement, the protective value of EC was also seen in relation to worry's association with depressive symptoms in the overall sample.

Taken together, these findings suggest that high levels of cognitive control capacity both protect against pathological worry and contribute to its persistence. Thus, there appears to be a subset of individuals who develop pathological worry and GAD despite—and perhaps due to—having good capacity for cognitive control. Based on their tendency to report low levels of AA symptoms, such individuals appear to misdirect such resources, as suggested by Hirsch and Mathews (2012), in service of worrying in verbal manner. Although we did not test this prediction in the current study, in a second study we recently found that the effect of the interaction between GAD symptom severity and EC on AA symptoms was indeed mediated by individual differences in the percentage of verbal thoughts reported during worry (Toh and Vasey 2016). Clearly, how and why such individuals develop uncontrollable and excessive worry despite having good cognitive control capacity are important topics for future consideration.

To this point we have focused on the role of trait-level differences in cognitive control capacity in producing inter-individual differences in the level of AA symptoms experienced during worry. However, our model also implies the potential for intra-individual differences in both cognitive control capacity and AA symptoms. There are at least two possible paths to such differences. First, there is evidence that constraining worry to a verbal mode of processing depletes cognitive resources (Leigh and Hirsch 2011), which can lead to increased negative intrusions (Stokes and Hirsch 2010) and promote attention to threat (Williams et al. 2013). This suggests that worrying in a verbal manner may lead to depletion of the very resources needed to maintain such a verbal mode of processing.<sup>10</sup> If so, even

worriers and those with GAD who have high trait-level capacity for cognitive control may experience increasing AA symptoms during prolonged periods of worry, as their ability to suppress images and shift to a verbal mode of processing wanes. Second, during periods of stress, such individuals may experience heightened AA symptoms during bouts of worry because their capacity for cognitive control has been depleted (Steinhauser Williams et al. 2007). Furthermore, such heightened arousal may lead to an upward spiral in worry and AA symptoms because perceptions of arousal appear to maintain worry among those high in GAD symptoms.<sup>11</sup> Andor et al. (2008) instructed GAD patients and controls to relax following a worry induction but provided false arousal feedback to some and relaxation feedback to others. Their GAD group maintained high levels of worry in response to arousal feedback compared to controls and compared to GAD patients given relaxation feedback. This suggests that during periods of prolonged stress individuals whose worry usually functions to limit AA symptoms may find it engenders them instead as a function of stress-related cognitive control resource depletion.

### Limitations and Future Directions

Our study had several noteworthy limitations. First, we tested only the major premise of our cognitive control model and did so in a single sample. It will be important for future studies to replicate our findings and to extend this line of research by testing other predictions made by our model. First among these is the prediction that the moderation of the link between GAD symptom severity and AA by individual differences in cognitive control is carried by individual differences in the percentage of verbal thoughts experienced during worry. As noted previously, we recently replicated the findings of the current study and showed that percentage of verbal thoughts during worry was indeed a significant conditional mediator of the link between GAD symptom severity and AA symptoms (Toh and Vasey 2016).

Second, because we lacked diagnostic information we cannot be sure how many members of our analog GAD group actually met *DSM* criteria for GAD. Although that group likely included false positives, there is considerable disagreement as to where to draw the line between normal and pathological worry (Ruscio 2002) and evidence suggests that even those with “sub-syndromal” or so-called “minor” GAD suffer significant impairment (e.g., Kessler

<sup>10</sup> This implies that worry and AA symptoms may interact to predict declines in cognitive control over time. As discussed in the online supplement, the current study did not provide an appropriate context in which to test such a prediction.

<sup>11</sup> As discussed in the online supplement, we tested an alternate model in which AA symptoms interacted with EC to predict level of worry. The DASS-A  $\times$  EC interaction was significant and produced a pattern consistent with expectation.

et al. 2005; Lawrence and Brown 2009). Moreover, our analog GAD group's average PSWQ score ( $M = 75.5$ ,  $SD = 3.1$ ) was substantially higher than Startup and Erickson (2006) reported for either analog GAD samples ( $M = 63.6$ ,  $SD = 10.8$ ) or clinical GAD samples ( $M = 67.2$ ,  $SD = 9.2$ ). Nevertheless, replication in clinical GAD samples is needed. However, it should be noted that in such studies it will be essential to establish that the sample includes sufficient variability in both AA symptoms and cognitive control to permit adequate statistical power to detect the interaction in question.

Third, 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. 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.

Fourth, our study did not consider other aspects of the CognAv and ContrAv models. For example, it remains to be seen whether individual differences in cognitive control capacity are associated with differences in worrier's beliefs regarding the functions served by their worries. Nor did we consider other variables having established importance for understanding pathological worry, such as metacognitive beliefs about worry (see Behar et al. 2009).

Finally, our study was limited by exclusive reliance on self-reports. Future studies are needed to replicate our findings with objective measures of AA and EC. However, with regard to such measures of AA it is important to note that our model does not require that subjective and objective measures be concordant. Rather, subjective experience should be of primary importance, even if it is not concordant with physiological measures. It should also be noted that future studies should also consider clinician ratings of GAD severity.

## Summary and Conclusions

In summary, the results of this study support the view that there is important heterogeneity in AA symptoms in GAD, which appears to be a function of individual differences in cognitive control capacity. This has clear implications for the CognAv and ContrAv models. The negative reinforcement mechanism for worry postulated by the CognAv model appears to require sufficient cognitive control capacity to constrain worry to a verbal mode of processing. Consequently, that negative reinforcement mechanism

cannot explain the persistence of worry among individuals who lack such capacity. However, insofar as worry in such individuals is associated with heightened AA symptoms, the ContrAv model offers a plausible negative reinforcement mechanism to explain its persistence.

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## Compliance with Ethical Standards

**Conflict of Interest** Michael W. Vasey, Lyvia Chriki, and Gim Y. Toh declare that they have no conflict of interest.

**Informed Consent** All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

**Animal Rights** No animal studies were carried out by the authors for this article.

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