



Enhanced cognitive control over task-irrelevant emotional distractors in generalized anxiety disorder versus obsessive-compulsive disorder[☆]



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ABSTRACT

Generalized anxiety disorder (GAD) is defined in part by excessive and uncontrollable worry. However, little is known about cognitive control abilities in adults with GAD. The present study examined cognitive control over negative and neutral material in a mixed clinical sample of adults with GAD and/or obsessive-compulsive disorder and a comparison healthy control sample. Participants completed a novel emotional variant of the AX-CPT (eAX-CPT) to index proactive and reactive cognitive control in the presence of negative and neutral distractor stimuli. Participants with GAD demonstrated enhanced cognitive control specifically over negative emotional distractors, relative to neutral distractors (within-subjects) and relative to OCD and controls (between-subjects). Findings were specific to GAD versus trait worry; however, higher trait worry predicted better cognitive control in GAD only. These findings are inconsistent with deficit-based cognitive models of GAD and may be better accounted for by models that conceptualize worry as an intentional (albeit maladaptive) cognitive control or emotion regulation strategy that is actively maintained by individuals with GAD in order to avoid engaging with more distressing emotional information.

1. Introduction

Generalized anxiety disorder (GAD) is defined in part by the experience that worry is uncontrollable (American Psychiatric Association, 2013). However, relatively little is known about actual cognitive control abilities (i.e., the abilities that underlie top-down, goal-directed control over the focus of one's attention) in adults with GAD, and theoretical accounts differ as to whether and what role cognitive control is proposed to play in maintaining the disorder and pathological worry more generally (Borkovec, Alcaine, & Behar, 2004; Hirsch & Mathews, 2012; Mennin, Heimberg, Turk, & Fresco, 2002; Mennin, Heimberg, Turk, & Fresco, 2005).

A recent cognitive model (Hirsch & Mathews, 2012) suggests that pathological worry is maintained in large part by impairments in cognitive control, specifically in the context of negative emotional information. Specifically, the model proposes that pathological worry is a largely involuntary process that is driven by an interaction between impaired cognitive control and magnified bottom-up processing of salient stimuli (e.g., attentional bias toward threat). This interaction is

proposed to promote detection and subsequent elaborative processing of threat-related information, which is further magnified by misallocation of cognitive control resources toward worry content, resulting in a protracted, uncontrollable worry episode. By contrast, other prominent models (e.g., Borkovec et al., 2004; Mennin et al., 2005; Newman & Llera, 2011) suggest that, for individuals with GAD, worry is a largely voluntary strategy that is engaged in order to avoid more aversive cognitive-emotional experiences. The Contrast Avoidance model (Newman & Llera, 2011), for example, argues that individuals with GAD actively sustain worry episodes in an effort to avoid sudden negative emotional shifts. This model builds on the earlier Cognitive Avoidance model (Borkovec et al., 2004), which conceptualizes worry as a cognitive strategy that individuals with GAD use in order to draw attention away from more distressing emotional material. Similarly, the Emotion Dysregulation model of GAD (2005, Mennin et al., 2002) conceptualizes worry as a cognitive control strategy employed by individuals with GAD in an effort to manage dysregulated negative emotions. Finally, a recent integrative model (Vasey, Chriki, & Toh, 2017) proposes that cognitive control moderates the relationship

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between autonomic arousal and GAD symptoms, such that worriers with good cognitive control are better able to recruit worry as a cognitive-emotional avoidance strategy.

In contrast to a deficit-based account, these emotion regulation models implicitly or explicitly conceptualize prolonged worry episodes in GAD as a *product* of cognitive control, rather than a consequence of failed cognitive control. Within this framework, certain cognitive control abilities (e.g., the ability to selectively attend to or avoid distressing emotional information) might be expected to be intact or even enhanced in individuals with GAD. In line with predictions from these models, an extensive literature suggests GAD is characterized by strong positive (albeit maladaptive) beliefs about worry (e.g., “my worry helps me solve problems”) and that worry episodes are often initiated voluntarily in accordance with those beliefs (Cartwright-Hatton & Wells, 1997; Llera & Newman, 2017).

Despite the centrality of cognitive control to prominent theoretical models of GAD, surprisingly few studies have assessed actual cognitive control abilities in adults with clinician-assessed GAD.¹ One study of “cold” (i.e., non-affective) cognitive control in older adults with GAD (Price & Mohlman, 2007) and two others in younger adults (Hallion, Tolin, Assaf, Goethe, & Diefenbach, 2017; Leonard & Abramovitch, 2019) found generally intact performance across a range of inhibition tasks (i.e., the facet of cognitive control that subserves the overriding of dominant but task-inappropriate responses). In the Hallion et al. (2017) study, adults with GAD performed worse than healthy controls on a computerized Stroop task; however, performance was not objectively impaired relative to published norms, nor was it related to trait worry. By contrast, Price and Mohlman (2007) found a positive relationship between inhibition performance and trait worry in the GAD sample; no relationship was observed in age-matched healthy controls. Two additional studies (Larson, Clawson, Clayson, & Baldwin, 2013; Xiao et al., 2011) did not observe a difference in performance on a “cold” conflict adaptation task in GAD versus controls; however, participants with GAD differed in neural activity (i.e., decreased conflict N2 amplitude, Larson et al.; increased error-related negativity; Xiao et al.). Fewer studies still have investigated the potential impact of stimulus valence on cognitive control in a diagnosed GAD sample. Blair et al. (2012) and Etkin, Prater, Hoeft, Menon, & Schatzberg (2010) each found intact behavioral performance but altered neural activity during cognitive control over negative information, while Price, Eldreth, & Mohlman (2011) found slower responding and reduced activation in prefrontal cortex to negative versus neutral words during an emotional Stroop task in older adults with GAD. However, these studies had some important limitations, such as small sample sizes ($N \leq 17$ for all three studies) and use of a brief structured interview with poor psychometric properties for assessing GAD in clinical populations (i.e., the Mini-International Neuropsychological Inventory; Verhoeven et al., 2017) to assign diagnoses in two of the studies (Blair et al., 2012; Etkin et al., 2010).

In contrast to the sparse clinical literature, a number of studies have investigated the relationship of cognitive control to worry in unselected and analogue samples. One study found that high trait worry was associated with poorer ability to shift between mental representations of negative worry-related word stimuli; performance was intact for neutral words and personally-irrelevant emotional stimuli (Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014). Another study, however, found that higher trait worry was associated with *better*

cognitive control over negative word stimuli (Brown, 2009). This effect remained stable following a worry and a neutral thought induction. Some studies that have experimentally induced worry have found an adverse effect of experimentally-induced worry on various facets of executive functioning, including working memory (Hallion, Ruscio, & Jha, 2014; Hayes, Hirsch, & Mathews, 2008; Leigh & Hirsch, 2011) and inhibition (Hallion et al., 2014). Of these studies, several have found that state worry impairs performance only for individuals with high (but not low) trait worry (e.g., Hayes et al., 2008; Leigh & Hirsch, 2011), but at least one comparably-powered study did not find evidence for moderation by trait worry (Hallion et al., 2014). Other studies still have found a null or even beneficial effect of worrying on performance (e.g., better performance on a working memory task; Walkenhorst & Crowe, 2009).

To date, few studies have investigated cognitive control over negative emotional material and its relationship to worry in a clinical (treatment-seeking) GAD sample. This gap in the literature is important for several reasons. First, cognitive control abilities vary as a function of stimulus valence even in healthy individuals (Pessoa, 2009). For example, there is reliable evidence that the presence of task-irrelevant threatening distractors interferes with cognitive control performance to a greater extent than do neutral distractors (e.g., Bishop, 2007; Pessoa, 2009). All of the theoretical models described above identify the presence or anticipation of negatively-valenced stimuli as central to the development of a worry episode: task-irrelevant negative stimuli are proposed to be excessively processed due to cognitive control failure (Hirsch & Mathews, 2012), and/or excessively avoided via the voluntary maintenance of worry as a cognitive control or emotion regulation strategy (Borkovec et al., 2004; Llera & Newman, 2017; Mennin et al., 2005; Vasey et al., 2017). Consequently, there is reason to believe that GAD may be characterized by alterations in cognitive control specifically over negative, task-irrelevant stimuli.

Second, individuals with GAD have been shown to differ from comparably severe worriers who do not meet diagnostic criteria for GAD in a number of respects, including in the extent to which worry is experienced as uncontrollable (e.g., Ruscio & Borkovec, 2004; Wells, 2005). Non-GAD worriers could reasonably be expected to differ from GAD worriers in the extent to which they can recruit and maintain cognitive control, particularly in the context of negative emotional stimuli. Consistent with this prediction, the models reviewed above tend to make the case that alterations in the way worry is regulated (i.e., impaired cognitive control over worry, Hirsch & Mathews, 2012; or excessive and maladaptive voluntary initiation of worry, Borkovec et al., 2004; Newman & Llera, 2011) are characteristic of pathological worry or GAD.

In addition to the theoretical implications of understanding cognitive control over emotional material in GAD, the increasing popularity of computerized cognitive training and other training-based interventions suggests that there may also be treatment implications. If GAD is characterized by impaired cognitive control, either broadly or specifically with respect to negative emotional contexts, it would suggest that cognitive remediation could be valuable for improving the ability to disengage from pathological worry. By contrast, if GAD is characterized by intact or even enhanced cognitive control, it might instead suggest that more traditional cognitive and metacognitive interventions may be more fruitful.

The goal of the present study was therefore to investigate alterations in cognitive control as a function of stimulus valence, worry severity, and GAD status specifically. In keeping with a prominent contemporary model of cognitive control (i.e., Dual Mechanisms of Control theory [DMC]; Braver, 2012), we investigated two behaviorally- and neurally-dissociable facets of cognitive control, termed *proactive control* and *reactive control*. Proactive control is defined as a sustained, effortful process in which task goals are actively maintained at the forefront of one’s mind in order to bias attention. Successful recruitment of proactive control promotes sustained focus on the selected task and prevents

¹ A distinction is drawn between cognitive control and attentional bias, the latter of which has received more research attention. A recent systematic review concluded that GAD is characterized by attentional bias toward threatening stimuli, particularly verbal-linguistic threat (i.e., threatening words; Goodwin et al., 2017). However, the presence of a “bottom-up” (automatic) attentional bias toward threat does not require or preclude an impairment in “top-down” (effortful) cognitive control over threatening or other emotional material.

task-irrelevant distracting stimuli from entering awareness. By contrast, reactive control is a late-stage correction mechanism that is mobilized only after a conflict is detected (e.g., the realization that one is off-task). Successful recruitment of reactive control allows an individual to re-focus attention on the task after attention has wandered.

Participants included a mixed clinical sample of treatment-seeking adults with GAD and/or obsessive-compulsive disorder (OCD) and healthy controls. Participants were recruited as part of a larger study aimed at investigating various facets of cognitive control and their relationship to worry and other forms of perseverative thought (e.g., obsessions). Because the larger study was developed within a trans-diagnostic framework and was not designed as an explicit comparison of GAD and OCD, no effort was made either to recruit or exclude participants with comorbid GAD–OCD. Consequently, the final sample included 7 participants with both disorders.

OCD was identified as an appropriate comparison group for several reasons. First, OCD is independently associated with elevated worry (Gladstone et al., 2005), which creates an opportunity to dissociate the relative contributions of severe worry, clinical and treatment-seeking status, and GAD status specifically. OCD is also characterized by obsessions, which are similar to worry in repetitiveness and valence but which are by definition involuntary (American Psychiatric Association, 2013). Some theoretical models of OCD predict that obsessions should be associated with a failure of one or more facets of cognitive control (e.g., Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005); however, meta-analytic studies find limited support for this relationship (Abramovitch, McCormack, Brunner, Johnson, & Wofford; Abramovitch, Abramowitz, & Mittelman, 2013; Bragdon, Gibb, & Coles, 2018; Snyder, Kaiser, Warren, & Heller, 2015). The relationship of cognitive control to OCD and obsessions was therefore of secondary interest in its own right, beyond its usefulness as a comparison to GAD and worry.

2. Method

2.1. Participants

Participants included 44 treatment-seeking adults with DSM-5 GAD and/or OCD ($n = 22$ GAD; $n = 29$ OCD; both figures include 7 participants diagnosed with both GAD and OCD) and an age-matched comparison sample of adults with no current or past psychological disorders ($n = 38$).² Two healthy controls were excluded; one was determined to have neurological disease shortly after participating and one talked throughout the task. Additional inclusion criteria were normal or corrected-to-normal vision and English language fluency. Exclusion criteria included a history of psychotic disorder, mania, traumatic brain injury, organic brain illness, acute suicidality, or current (past 12 months) substance use disorder. To maximize ecological validity, psychotropic medications were permitted in clinical participants except for current benzodiazepine or stimulant medications, which were identified as exclusion criteria for participants at the study outset. Approximately one-third (34%) of clinical participants endorsed medication use.

Demographic and clinical characteristics are provided in Table 1. Participants were 58% female, 1% transgender, M age = 40.21, $SD = 16.62$; 80% White, 13% Black or African American, 4% Asian, 3% American Indian or Alaska Native, 3% missing or other (race categories

²The original version of this manuscript included $n = 28$ healthy control participants. During peer review, we were advised to recruit additional healthy control participants to address a significant age difference between the clinical and healthy groups (i.e., clinical participants were significantly younger than healthy controls). We therefore recruited 10 additional healthy control participants who were between ages 18–30. The 10-participant target was chosen *a priori*.

were non-mutually-exclusive), and 6% Hispanic or Latinx. The healthy control group was more racially representative compared to both clinical groups (28% versus 0% Black or African-American; 67% versus 91% White, respectively, $ps < .05$) but less ethnically representative compared to the combined clinical group only (0% versus 11% Hispanic or Latinx, respectively; $p = .042$). There were no group differences on any other demographic variable (i.e., age; gender; education).

2.2. Measures

2.2.1. Clinical measures

2.2.1.1. Diagnostic Interview for Anxiety, Mood, OCD, and Related Neuropsychiatric Disorders (DIAMOND; Tolin et al., 2018). Diagnostic status was determined using the DIAMOND, a semi-structured diagnostic interview for a wide range of psychological disorders. The DIAMOND has generally strong psychometric properties, including good inter-rater reliability across separate administrations completed by independent interviewers (GAD $\kappa = 0.71$; OCD $\kappa = 0.62$), good test-retest reliability across administrations timed one week apart (GAD $\kappa = 0.68$; OCD $\kappa = 0.83$), and strong convergent and discriminant validity vis-à-vis leading self-report measures (e.g., higher Penn State Worry Questionnaire [PSWQ] in GAD versus other diagnoses; higher Obsessive-Compulsive Inventory – Revised (Foa et al., 2002) in OCD versus other diagnoses; Tolin et al., 2018).

2.2.1.2. Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). Trait worry was assessed using the PSWQ, a widely-used 16-item self-report measure of worry severity in daily life. The PSWQ has strong psychometric properties, including strong internal consistency, test-retest reliability, and convergent and discriminant validity vis-à-vis semi-structured diagnostic interview and other self-report measures, in clinical and healthy samples (Fresco, Mennin, Heimberg, & Turk, 2003; Meyer et al., 1990). Internal consistency was excellent in the present sample ($\alpha = 0.97$).

2.2.1.3. Yale-Brown Obsessive-Compulsive Scale – Self-Report Version (Y-BOCS-SR; Baer, Brown-Beasley, Sorce, & Henriques, 1993). Obsession severity was assessed using the self-report version of the Y-BOCS. Obsession-related items (items 1–5) were summed to create an overall obsession severity score. The Y-BOCS-SR and the obsession subscale have generally strong psychometric properties in both clinical and healthy samples, including good internal consistency, test-retest reliability, and discriminative validity for OCD versus non-OCD clinical samples (Steketee, Frost, & Bogart, 1996). Internal consistency for the obsession severity score in the present sample was strong ($\alpha = 0.91$).

2.2.2. Cognitive control task

To investigate emotion-related alterations in cognitive control, we developed a novel emotional variant of the AX-Continuous Performance Task (AX-CPT; Cohen, Barch, Carter, & Servan-Schreiber, 1999; Edwards, Barch, & Braver, 2010). The AX-CPT is a well-established, widely-used computerized measure of proactive and reactive cognitive control.

In the standard AX-CPT, participants view a series of sequentially-presented letters on a computer screen (A, B, X, Y), which are organized as cue-probe pairs. Participants are instructed to press the “target” button when they see the letter X, but only if the X was immediately preceded by an A (A–X trial type). In all other cases, participants press the “non-target” button. Each “trial” consists of a cue (300 ms), a fixation cross (5000 ms), a probe (300 ms), a second fixation cross (1000 ms), and a feedback screen (2000 ms; stimulus timing modeled after Edwards et al. (2010) and MacDonald (2008)). Target (A–X) trials are presented with high frequency (70%). Other trial types (A–Y, B–X, B–Y) occur with a frequency of 10% each throughout the experiment. The high frequency of A–X trials creates a strong dominant tendency to

Table 1
Clinical and Demographic Characteristics.

	GAD $n = 20$ -22 ^a	OCD $n = 28$ -29 ^a	Clinical $M (SD) n = 43$ -44	Controls $M (SD) n = 37$ -38	p (Clinical vs. Control)
Age	42.15 (16.44)	35.486 (15.94)	39.95(16.68)	40.51 (16.80)	.884
Education					.641
High school or less	32%	31%	32%	31%	
Some college	36%	35%	32%	31%	
Bachelor's degree or more	32%	39%	36%	39%	
PSWQ	65.41 (8.55)	65.00 (10.02)	64.57 (9.24)	36.67 (12.20)	< .001
Y-BOCS-SR total	15.14 (6.70)	20.90 (5.24)	18.18 (6.47)	3.29 (2.72)	< .001
Y-BOCS-SR obsessions	8.45 (3.19)	11.45 (3.05)	10.18 (3.45)	2.22 (1.99)	< .001

Note. GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; PSWQ = Penn State Worry Questionnaire; Y-BOCS-SR = Yale-Brown Obsessive-Compulsive Scale – Self-Report Version.

^a Participants with a diagnosis of both GAD and OCD ($n = 7$) are represented in both columns. Variation in the sample size reflects missing demographic or questionnaire data.

press the “target” button any time X is presented. It also creates a strong dominant tendency to press the “target” button on the following trial any time A is presented.

Proactive control is assessed by examining performance on B–X trials. B–X trials provide a useful index of proactive control because the presence of the B stimulus on trial $n - 1$ means that the participant must actively maintain the goal of making the non-dominant response in order to override the strong dominant response tendency elicited by the X (target) on trial n . Conversely, *reactive control* is assessed by performance on A–Y trials. Because A–X trials occur with high frequency, the presence of an A stimulus on trial $n - 1$ potentiates the already dominant tendency to make the “target” (X) response on trial n . Participants must react to the unexpected presentation of a Y stimulus on trial n by overriding the dominant response tendency to make the less-dominant “non-target” response.

For the present study, we adapted the AX-CPT to include negative emotional and neutral distractor word stimuli (eAX-CPT; see Appendix for the complete list of distractor words). Word stimuli were used as distractors because individuals with GAD show more interference from negative distractor words versus pictures, consistent with the verbal-linguistic nature of worry (Goodwin, Yiend, & Hirsch, 2017). Parameters for the eAX-CPT were identical to those described for the standard task, except that each fixation period was replaced by either a neutral or a negative emotional distractor word (counterbalanced; distractor selection criteria are described below). Consequently, performance on proactive control (B–X) trials indexed the extent to which negative stimuli interfered with the maintenance of task information, whereas performance on A–Y trials indexed the extent to which salient emotional (versus neutral) distractors interfered with subsequent late-stage recruitment of cognitive resources.

2.2.2.1. Selection of distractor stimuli. A list of possible distractor stimuli was generated using the standardized Affective Norms for English Words (ANEW; Bradley & Lang, 1999) database. In order to maximize the salience of the distractor stimuli, candidate negative emotional distractor words were rated for potential relevance to GAD and OCD by a team of clinical psychologists with extensive clinical experience in the diagnosis and treatment of these disorders. The final word list was determined by the first author and consisted of 40 negative words that were rated as especially likely to be salient for adults with GAD or OCD and 40 neutral words that were matched for length and English language frequency (see Appendix).

2.3. Procedure

Diagnostic interviews were conducted by trained graduate students or postdoctoral fellows as part of the routine clinical intake for clinical participants and immediately prior to completion of the experimental procedures for healthy controls. Final diagnoses were determined by

the licensed clinical psychologist who supervised each intake, following a discussion of the case between the diagnostician and supervisor and a feedback session that included the supervisor, diagnostician, and participant. The intakes and diagnoses occurred in the context of a specialty outpatient anxiety and OCD clinic. All licensed supervisors had extensive experience (10+ years) with differential diagnosis of GAD and OCD.

The experimental session took place in a private room. Participants completed all cognitive and self-report measures during a single session. Written informed consent was obtained for all participants. Participants completed the standard AX-CPT as well as the eAX-CPT (administration order was counterbalanced); findings for the standard task will be reported elsewhere.

2.4. Analytic plan

Accuracy approached ceiling and did not differ between groups, both $M = 0.95$, $SD = 0.06 - 0.08$, $t = 0.15$, $p = .885$, and was not examined further. Mean reaction time (RT) was calculated for each trial type (i.e., emotional AX, AY, BX; neutral AX, AY, BX; BY trials were not examined) by averaging performance across all trials of that type. Individual trials with RT $\geq 3SD$ greater or less than that participant's mean RT were considered outliers and were excluded. The primary analytic approach was a single repeated-measures analysis of covariance (ANCOVA) with two between-subjects factors (GAD diagnostic status; OCD diagnostic status), two within-subjects factors (distractor valence: negative vs. neutral; trial type: proactive vs. reactive control), and one covariate (A–X trial performance; i.e., to control for overall speed of responding, including potential effects of fatigue; A–X response times did not differ between groups, $t = 0.83$, $p = .408$). To probe the relative contributions of trait worry versus diagnostic status, we repeated the main analysis first with trait worry and obsession severity included as covariates, and second, with the further addition of a trait worry x GAD status interaction term. Due to the complexity of the design, we also ran two additional sensitivity analyses to probe: 1) the extent to which the main GAD findings replicated when participants with OCD were excluded (i.e., GAD versus healthy control); and 2) the extent to which the main GAD findings replicated when healthy control participants were excluded (i.e., GAD versus OCD). For ease of interpretation, findings from the sensitivity analyses are presented alongside the main findings.

3. Results

Missing data represented less than 1% of values and were determined to be Missing Completely at Random (MCAR) using Little's (1982) MCAR test ($\chi^2 (34) = 29.86$, $p = .671$). Missing data were therefore addressed using pairwise deletion.

Table 2
Repeated measures ANCOVA evaluating effects of diagnostic group, distractor valence, and trial type (proactive versus reactive) on task performance.

	F	p	η_p^2
Main effects			
Distractor valence (emotional vs. neutral)	0.10	.753	.001
Trial type (proactive vs. reactive)	17.46	.000	.20
GAD diagnosis	5.02	.028	.07
OCD diagnosis	0.01	.923	.000
Two-Way Interactions			
GAD x distractor valence	11.02	.001	.13
OCD x distractor valence	0.57	.451	.01
GAD x trial type	1.95	.167	.03
OCD x trial type	2.04	.158	.03
Distractor valence x trial type	0.53	.468	.01
Three-Way Interactions			
Distractor valence x trial type x GAD	1.56	.215	.02
Distractor valence x trial type x OCD	0.03	.859	.000

Note. GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder. AX trial response time was included as a covariate of non-interest and is not reported here.

3.1. Emotional and neutral proactive and reactive control

A repeated-measures analysis of covariance (ANCOVA) with two between-subjects factors (GAD yes/no; OCD yes/no), two within-subjects factors (distractor valence: neutral vs. negative; trial type: proactive vs. reactive), and one covariate (RT for A–X trials) revealed a significant main effect of trial type (i.e., performance was faster on proactive vs. reactive trials, $p < .001$, $\eta_p^2 = 0.20$) and a significant main effect of GAD diagnosis (i.e., participants with GAD responded faster than those without GAD across proactive and reactive control trials; $p = .028$, $\eta_p^2 = 0.07$). This main effect was qualified by a significant GAD by distractor valence interaction ($p = .001$, $\eta_p^2 = 0.13$). Post-hoc tests of simple effects were used to deconstruct the interactions. Findings are presented in Table 2.

Table 3
Marginal means and standard errors for follow-up tests of simple effects for all two-way interactions between GAD diagnostic status, distractor valence, and trial type.

			Within-Subjects Contrasts		
	F	p	η_p^2		
GAD x distractor valence					
	Neutral	Emotional			
GAD	572 (17)	543 (19)	3.69	.059	.05
Non-GAD	585 (10)	614 (11)	9.90	.002	.12
Between-Subjects Contrasts	$p = .536$; $\eta_p^2 = .01$	$p = .001$; $\eta_p^2 = .13$			
GAD x trial type					
	Proactive	Reactive			
GAD	428 (25)	688 (20)	121.84	< .001	.49
Non-GAD	495 (15)	704 (12)	68.72	< .001	.63
Between-Subjects Contrasts	$p = .024$; $\eta_p^2 = .07$	$p = .463$; $\eta_p^2 = .01$			

Note. GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder. Non-GAD participants include healthy controls and clinical participants without GAD (OCD-only). There were no interactions between valence and trial type; therefore, marginal means and standard errors are collapsed across the non-relevant category (i.e., valence marginal means are collapsed across trial type; trial type marginal means are collapsed across valence) for ease of interpretation. Marginal means are reported with AX reaction time included as a covariate of non-interest and evaluated at 555ms.

3.1.1. Interaction of GAD diagnosis and distractor valence

Two major patterns were identified. For ease of interpretation we have organized the results into differences that were significant at the group-level (between-subjects) and differences that were significant at the valence-level (within-subjects) (Table 3).

3.1.1.1. Group differences. GAD participants responded significantly faster than non-GAD participants on emotional trials (marginal mean difference [M_{diff}] = -72, $p = .001$, $\eta_p^2 = .13$), with no group differences observed for neutral trials ($M_{diff} = -12$, $p = .536$, $\eta_p^2 = .01$). This effect was magnified in sensitivity analyses that excluded participants with an OCD diagnosis (i.e., such that the comparison was restricted to GAD versus healthy controls), $M_{diff} = -64$, $p = .017$, $\eta_p^2 = .12$; overall interaction term $p = .005$, $\eta_p^2 = .16$. The univariate effect was also preserved in sensitivity analyses that excluded healthy controls (i.e., such that the comparison was restricted to within the clinical sample), $M_{diff} = -90$, $p = .013$, $\eta_p^2 = .15$; however, the overall interaction term did not reach significance ($p = .168$, $\eta_p^2 = .05$).

3.1.1.2. Valence differences. Non-GAD participants were significantly faster to respond to neutral trials versus emotional trials ($M_{diff} = -29$, $p = .002$, $\eta_p^2 = .12$), while GAD participants were marginally faster to respond to emotional trials versus neutral trials ($M_{diff} = -30$, $p = .059$, $\eta_p^2 = .05$). This pattern was magnified in sensitivity analyses that excluded participants with an OCD diagnosis (healthy control $M_{diff} = -27$, $p = .039$, $\eta_p^2 = .09$; GAD $M_{diff} = 45$, $p = .037$, $\eta_p^2 = .09$), but was diminished and marginally or non-significant in sensitivity analyses that excluded healthy control participants (OCD $M_{diff} = -12$, $p = .560$, $\eta_p^2 = .01$; GAD $M_{diff} = 27$, $p = .076$, $\eta_p^2 = .01$).

3.2. Trait worry analyses

To assess whether trait worry was incrementally related to performance beyond variance accounted for by diagnostic status and obsession severity, we replicated the main analysis with trait worry and obsession severity entered as covariates. There was no main effect of trait worry ($p = .372$, $\eta_p^2 = .01$) or obsession severity ($p = .273$, $\eta_p^2 = .02$), nor did trait worry or obsession severity interact with trial type (worry $p = .11$, $\eta_p^2 = .04$; obsession $p = .820$, $\eta_p^2 = .001$), distractor type (worry $p = .451$, $\eta_p^2 = .01$; obsession $p = .832$, $\eta_p^2 = .001$), nor their interaction (worry $p = .924$, $\eta_p^2 < .001$; obsession $p = .729$, $\eta_p^2 = .002$). Moreover, the interaction between GAD status and distractor valence was preserved ($p = .028$, $\eta_p^2 = .07$) and a significant interaction between GAD status and trial type emerged ($p = .025$, $\eta_p^2 = .07$) such that GAD participants responded faster than non-GAD participants on proactive control trials only ($M_{diff} = -88$, $p = .016$, $\eta_p^2 = .08$). This interaction superseded the main effect of GAD from the previous analysis, which was no longer significant ($p = .105$, $\eta_p^2 = .04$). All other contrasts remained unchanged.

Finally, we further probed these relationships in a final sensitivity analysis that included all terms described above, with the addition of an interaction term for the relationship between GAD and trait worry. The interaction between GAD and trait worry was significant ($p = .042$, $\eta_p^2 = .06$). To decompose this interaction, we repeated the analysis separately for participants with and without GAD. This follow-up analysis revealed a main effect of trial type in both groups (i.e., faster performance on proactive versus reactive control trials, both $p \leq .033$) and a main effect of trait worry in the GAD group only ($p = .020$, $\eta_p^2 = .31$), such that higher trait worry predicted better (faster) performance irrespective of trial type or distractor valence in GAD participants only.

4. Discussion

The primary aim of the present study was to characterize cognitive control over neutral and emotional distractors in clinically significant GAD and as a function of trait worry. Contrary to predictions of

impairment-based models (e.g., Hirsch & Mathews, 2012), GAD was associated with enhanced cognitive control in the context of emotional distraction, with preliminary evidence of specificity for proactive control specifically. This enhancement was observed relative to neutral distraction (within-subjects) and relative to healthy and non-GAD (OCD) clinical controls (between-subjects). Enhancement was specific to GAD versus trait worry, although there was some evidence linking higher trait worry to better cognitive control specifically in participants with GAD.

Bearing the study's limitations in mind, the present findings fail to support predictions of deficit-based cognitive models of pathological worry (e.g., Hirsch & Mathews, 2012). Instead, these findings are more consistent with predictions of emotion regulation models of pathological worry, which propose that worry is recruited at least semi-voluntarily, usually as a means of displacing more distressing cognitive content (e.g. imagery) or buffering against more aversive emotional experiences (e.g., negative emotional contrasts; Borkovec et al., 2004; Mennin, 2002; Mennin et al., 2002, 2005; Llera & Newman, 2017; Vasey et al., 2017). The finding that GAD would be associated with enhanced cognitive control in negative emotional contexts aligns with the idea that individuals with GAD voluntarily initiate and maintain worry specifically to distract from negative emotional information (Borkovec et al., 2004; Mennin, 2002; Mennin et al., 2005; Vasey et al., 2017) or when negative emotional experiences are anticipated (Llera & Newman, 2017).

The specificity of the findings to a diagnosis of GAD, versus elevated worry or clinically-significant anxiety more generally, is worth exploring. Specifically, neither trait worry nor obsession severity offered incremental utility beyond diagnostic status for predicting cognitive control, except for a positive relationship between trait worry and cognitive control performance that emerged in the GAD sample only. Although this pattern is somewhat surprising when considered from a transdiagnostic process perspective, there are several converging sources of evidence that argue in favor of the veracity of the finding. The first (and least interesting) reason is purely methodological: from a statistical standpoint, a continuous measure with strong psychometric properties and good variability (i.e., the PSWQ) would typically be expected to outperform a dichotomous predictor (i.e., diagnostic status) on psychometric grounds alone. That this pattern did not emerge speaks to the utility of GAD diagnostic status as a predictor in this particular set of analyses.

More substantively, there is a robust literature that describes reliable differences between comparably severe “high worriers” who do versus do not meet diagnostic criteria for GAD. Compared to “high worriers” without GAD, worriers who meet criteria for GAD have more difficulty disengaging from worry, hold more negative views of their worry, and report worrying about a wider range of topics (Hirsch, Mathews, Lequartier, Perman, & Hayes, 2013; Ruscio & Borkovec, 2004; Ruscio, 2002). These distinctions do not speak to the present findings directly, but they are broadly consistent with a dissociation between GAD diagnostic status and worry severity, specifically with respect to cognitive variables. Finally, the finding that trait worry predicted better cognitive control only in the context of GAD provides further support for a role of GAD *per se*, while also hinting at a potentially more nuanced relationship. This finding in particular aligns with previous findings of a positive relationship between trait worry and cognitive control in older adults with GAD (Price & Mohlman, 2007), although it should also be noted that at least one other study in younger adults with GAD did not find any such relationship (Hallion et al., 2017). Additional research with larger samples in this area would be fruitful, especially because GAD is under-studied relative to lower base-rate disorders, including OCD (Asmundson & Asmundson, 2018; Newman & Przeworski, 2018).

The extent to which these findings align with theoretical predictions from emotion regulation or worry-as-volitional models is less clear. Despite the broad conceptual overlap in their conceptualizations of

worry as a semi-volitional process, the models vary in the extent to which their language and predictions center on GAD, pathological worry, or “worry” at its most broadly construed (i.e., including “normal” worry). At one extreme, Hirsch and Mathews (2012) argue that differences in cognitive control play a major role in the progression of “normal” versus “pathological” worry. At the other extreme, Borkovec et al. (2004) explicitly argue that their cognitive avoidance model of worry is broadly applicable to emotional disorders beyond GAD. Between these extremes, the Contrast Avoidance (Newman & Llera, 2011) and Emotion Dysregulation (Mennin et al., 2005) models focus their discussion and predictions primarily around GAD and pathological worry, but do not make strong claims about the applicability of these predictions to “normal” or non-GAD worry. Similarly, the Integrative Model (Vasey et al., 2017) considers GAD and non-GAD worry separately, but ultimately does not draw strong conclusions about the extent to which cognitive control operates differently for GAD versus non-GAD worriers. This lack of a firm conclusion by Vasey et al. (2017) is appropriate in light of the limitations of that study, which include reliance on an analogue (non-clinical) GAD sample and the use of a self-report measure with unknown convergent validity vis-à-vis actual task performance to assess cognitive control. Nevertheless, to the extent that these models propose to account for the development and maintenance of GAD and related forms of psychopathology, it seems important that the core process proposed therein (i.e., the volitional engagement of worry for emotion regulatory purposes) should be conceptualized as at least pathogenic, if not pathological in its own right.

The present findings may also shed some light on previous findings of cognitive control impairments following a worry induction (e.g., Hayes et al., 2008; Hallion et al., 2014; Leigh & Hirsch, 2011). Each of these studies used analogue samples (e.g., unselected participants or high worriers) rather than a clinical sample. The present findings suggest an important distinction between GAD and severe worry, even when the severe worry occurs in the context of another anxiety-related disorder (i.e., OCD). We believe severe worry is worthy of investigation in its own right; however, caution is warranted when generalizing those findings to clinically significant GAD.

Although characterizing cognitive control functioning in OCD was not a primary goal of this paper, the pattern of results warrants a brief consideration. Specifically, OCD and obsession severity did not show significant independent effects within their respective analyses, nor did those variables interact with distractor valence or trial type to predict performance. The null results for the relationship between obsession severity and cognitive control generally align with those of previous meta-analyses that have attempted to link executive functioning to specific OCD symptoms (Abramovitch et al., 2013; Bragdon et al., 2018; Snyder et al., 2015). However, it is also important to acknowledge that the OCD sample size was relatively small; as such, the present null results should not be overinterpreted. Additional research will be needed to more clearly characterize any potential relationships or the lack thereof with respect to proactive and reactive cognitive control and OCD diagnostic status and cognitive symptom severity.

The present findings should be interpreted in light of several strengths and limitations. The major strengths are use of a treatment-seeking, clinician-diagnosed GAD sample, the inclusion of a clinical comparison group (OCD), and the adaptation of an established, contemporary cognitive control task (i.e., the AX-CPT; Braver, 2012) to measure cognitive control in the presence of negative emotional versus neutral distractors. The use of word (rather than picture) stimuli was also a strength given our specific interest in GAD (Goodwin et al., 2017). Future research would benefit from using idiographic distractor words, which would facilitate an explicit examination of personal relevance versus general negative emotionality in explaining cognitive control. Future studies should also consider using task-relevant (i.e., to-be-attended) negative emotional stimuli. Although avoidance models of GAD conceptualize worry as essentially the “lesser of two evils” from an emotional perspective, worry is nevertheless a negative cognitive and

emotional experience. A more valid test of Borkovec and colleagues' (2004) avoidance-based model of GAD, for example, might instead include idiographic emotional words as the to-be-attended stimuli, with distressing emotional imagery included as distractors. Such studies should also include an explicit assessment of the frequency and severity specifically of voluntary worry in daily life. A final but important limitation is the relatively small sample size for the GAD group. Although the effect sizes for these contrasts were generally moderate-to-large (e.g., $\eta_p^2 = 0.13$ for the interaction between GAD and distractor valence; $\eta_p^2 = .31$ for the main effect of trait worry in the GAD group) and the findings were robust across several sets of sensitivity analyses, replication in a larger sample will be critical.

In addition to the theoretical implications reviewed above, the present findings may also have implications for treatment. Recent efforts to develop cognitive training paradigms for unwanted thought and related symptoms (e.g., Kalanthroff, Steinman, Schmidt, Campeas, & Simpson, 2018; Koster, Hoorelbeke, Onraedt, Owens, & Derakshan, 2017) hinge on the assumption that cognitive deficits are a) present, and b) mechanistically related to symptoms. Although individuals with GAD by definition report that their worry is uncontrollable, the present study does not find evidence of a cognitive control deficit in this population; instead, findings are more suggestive of an enhancement in emotional contexts. Additional research is needed before making strong conclusions about the viability of cognitive training programs for this population; however, an initial impression suggests that more traditional cognitive and meta-cognitive interventions (e.g., aimed at reducing voluntary worry) may be more productive. Such training programs could potentially have promise for individuals with OCD; however, the absence of a relationship between cognitive control and obsession severity suggests that it will be important for basic research to first clarify what, if any, mechanistic role cognitive control might play in OCD, before developing interventions based on presumed relationships.

Taken together, the present findings argue against an impairment-based framework for understanding cognitive control over negative material in GAD. Instead, the present findings are more consistent with suggestions of enhanced cognitive control specifically over emotional material in clinically significant GAD, as well as a tendency for better cognitive control to be associated with more severe worry in adults with the disorder. These findings appear to be relatively specific to GAD and are not observed in healthy controls, nor in treatment-seeking participants with OCD and comparably severe worry, yet who do not meet diagnostic criteria for a diagnosis of GAD. Future studies should examine the extent to which cognitive control enhancements may be a mechanism or consequence of maladaptive voluntary worry in adults with GAD.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.janxdis.2019.02.004>.

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