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"Cold" Cognitive Control and Attentional Symptoms in Anxiety: Perceptions Versus Performance

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Clinically significant anxiety is associated with an array of attentional symptoms (e.g., difficulty concentrating; unwanted thought) that are subjectively experienced as severe. However, neuropsychological findings are mixed with respect to the presence of cognitive deficits that can account for these symptoms. Contextualizing predictions from established clinical theories (e.g., Attentional Control Theory) within contemporary, neurobiologically derived models of cognitive control (Dual Mechanisms of Control Theory), the present study investigated the relationship between "cold" proactive and reactive cognitive control, task effort, and subjective attentional symptoms (difficulty concentrating; unwanted thought) in a mixed clinical sample of individuals with generalized anxiety disorder (GAD) and/or obsessive-compulsive disorder (OCD) and a comparison sample of healthy controls. Clinical status moderated the relationship between attentional symptoms (attentional focusing and trait worry) and proactive cognitive control response time. Clinical status also moderated the relationship between trait worry and task effort. Higher trait worry was associated with slower proactive control and lower effort in healthy participants, but faster proactive control in clinical participants. Self-reported attentional focusing showed differential validity vis-à-vis proactive control response time in clinical versus healthy participants. Post-hoc conditional effects analysis suggested more accurate self-appraisals in healthy controls, but was not significant after correction for multiple comparisons. Preliminary evidence suggested that differences in task effort

in anxious versus healthy adults may relate to subjective attentional symptoms in GAD and OCD.

CLINICALLY SIGNIFICANT ANXIETY is often associated with subjective impairments in attentional control. Negatively valenced attentional symptoms such as uncontrollable worry and unwanted, intrusive thoughts (i.e., obsessions) are the cardinal features of generalized anxiety disorder (GAD) and obsessive-compulsive disorder (OCD), respectively (American Psychiatric Association [APA], 2013). Individuals with these disorders also endorse difficulty concentrating, even in neutral (i.e., non-affective or "cold") contexts (Armstrong, Zald, & Olatunji, 2011). These symptoms may reflect underlying deficits in cognitive control—an umbrella term that encapsulates an array of cognitive mechanisms that govern top-down control of attention (Braver, 2012; Paulus, 2015). However, the neuropsychological literature is inconclusive with respect to the presence of deficits that relate mechanistically to these symptoms (Paulus, 2015).

One reason for the lack of clarity is a lack of research, particularly with respect to subjective difficulty concentrating (sometimes framed positively as "attentional control"). Difficulty concentrating is one of the most pervasive symptoms associated with anxiety and related forms of psychopathology (APA, 2013; Hallion, Steinman, & Kusmierski, 2018). GAD and OCD in particular have been linked to significant impairments in self-reported attentional control (e.g., Armstrong et al., 2011). However, little research has systematically considered potential mechanistic links between subjective attentional control and objective neuropsychological or "cold" cognitive performance (i.e., performance at "baseline" or in the absence of specific affective stimuli or symptom inductions). Many studies of attentional control in anxiety-related psychopathology rely on self-report as an index of ability (e.g., Armstrong et al., 2011; Vasey, Chriki, & Toh, 2017). This limitation is significant, especially because GAD and OCD are each associated with elevated performance standards (Frost & Steketee, 1997; Gentes & Ruscio, 2014), which may bias subjective self-reports. Additionally, research suggests underestimation (or simply inaccurate estimation) of cognitive abilities such as attention and memory in a range of psychological disorders (Moritz, Ferahli, & Naber, 2004; Mowla et al., 2008) including OCD (Tolin et al., 2001). As

such, a clearer picture of the relationship between subjective attentional control and objective cognitive control ability is needed.

A somewhat larger body of research has explored the role of "cold" cognitive control in the experience and regulation of worry and obsessions. Recent meta-analyses on neuropsychological functioning in OCD (e.g., Bragdon, Gibb, & Coles, 2018; Snyder, Kaiser, Warren, & Heller, 2015) report mild to moderate deficits of debatable clinical significance in one or more facets of executive functioning (i.e., a term that has significant conceptual overlap with "cognitive control" but arguably refers to a more circumscribed set of processes responsible for the regulation and control of cognitive activity; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). However, data on the relationship between executive functioning and obsession severity specifically is lacking, because empirical studies tend to report correlations for total symptom scores only and nonsignificant relationship often are not reported (Abramovitch, McCormack, Brunner, Johnson, & Wofford, 2018). Of the few studies that dissociate obsessions versus compulsions, conclusions are limited by inconsistent reporting practices and findings (Harsányi et al., 2014; Yazdi-Ravandi et al., 2018). The neuropsychological literature on "cold" cognitive functioning in GAD is even more sparse. Prospective studies have found a relationship between executive functioning deficits and future worry or GAD severity, but data on concurrent relationships are mixed (Bredemeier & Berenbaum, 2013; Zainal & Newman, 2018). Two studies did not find evidence of inhibition impairment in GAD (Leonard & Abramovitch, 2019; Price & Mohlman, 2007), while one found modest impairments that were significant relative to healthy controls, but not relative to normative data (Hallion, Tolin, Assaf, Goethe, & Diefenbach, 2017). Of note, none of these studies has reported the expected inverse relationship between task performance and trait worry that would be predicted by a strict deficit account of pathological worry. Price and Mohlman (2007) in fact found a *positive* relationship between inhibition performance and trait worry in older adults with GAD, which has been interpreted as suggestive of a role for cognitive control in voluntarily sustaining pathological worry (e.g., for emotion regulation purposes; Llera & Newman, 2014, 2017).

How can we reconcile the apparent discrepancies in the literature between severe subjective attentional symptoms associated with GAD and OCD on the one hand, and the failure to find robust associations between these symptoms and objective cognitive impairments on the other hand? Some

researchers have suggested that, because worry and obsessions are inherently affective, they may be best characterized in terms of cognitive control specifically over—or in interaction with—emotional stimuli (e.g., Hirsch & Mathews, 2012; Theiss, McHugo, Zhao, Zald, & Olatunji, 2019; see Joormann, 2018, for a consideration of these issues in rumination). However, a strictly affective model of cognitive control impairments cannot account for subjective difficulty concentrating, which is endorsed even with reference to entirely neutral material and contexts. For example, the Attentional Control Scale (ACS; Derryberry & Reed, 2002), a leading self-report measure of concentration ability, uses exclusively neutral language (e.g., Focusing subscale, "It's very hard for me to concentrate on a difficult task when there are noises around"; Shifting subscale, "After being interrupted or distracted, I can easily shift my attention back to what I was doing before"), with no reference to affective contexts, anxiety, or distress. If difficulty concentrating is at all related to objective cognitive impairments, as opposed to distorted self-appraisal, those impairments should in theory be detectable given a sufficiently sensitive "cold" cognitive task.[1]

Attentional control theory (ACT; Eysenck & Derakshan, 2011; Eysenck, Derakshan, Santos, & Calvo, 2007) offers one potentially useful framework for understanding the relationship between cognitive control and attentional symptoms in anxiety. ACT proposes that chronic anxiety is associated with reductions in "cold" cognitive efficiency (particularly in the inhibition and shifting functions), such that increased effort or resources are required to achieve comparable performance (described in terms of effectiveness). A logical extension of the model is that attentional symptoms such as subjective difficulty concentrating might reflect a reality in which anxious individuals must work harder to achieve comparable performance relative to healthy controls (i.e., reduced efficiency, but intact effectiveness).

Narrative reviews of the ACT literature typically conclude that there is support for a relationship between chronic anxiety and reduced efficiency, for some if not all executive functions (Berggren & Derakshan, 2013). However, several interpretive challenges arise for applying findings and their theoretical context (i.e., ACT; Eysenck et al., 2007; Eysenck & Derakshan, 2011) to understanding the relationship of "cold" cognitive control to atten-

tional symptoms in the context of clinically significant anxiety. One challenge is that previous studies on the relationship between "cold" cognitive control and anxiety, particularly those conducted within the ACT framework, have tended to focus on global anxiety as the construct of interest. With the exception of worry, which was a major focus of an earlier iteration of ACT (i.e., processing efficiency theory; Eysenck & Calvo, 1992), potential relationships between reduced cognitive efficiency and specific attentional symptoms have rarely been explored. A second challenge is that the overwhelming majority of published studies were conducted in healthy and analogue samples, including the relatively few studies that have explicitly dissociated worry from anxiety when considering the potential for differential relationships to cognitive efficiency (e.g., Forster, Elizalde, Castle, & Bishop, 2015). Third, there is variability in how efficiency and effectiveness are operationalized in the literature. Across studies, accuracy (or error rate) is reliably interpreted as an index of performance effectiveness. Response time (RT) is typically interpreted as an index of efficiency, such that slower RTs would reflect increased cognitive resources or effort needed to respond correctly (Berggren & Derakshan, 2013; Eysenck & Derakshan, 2011); however, RT is sometimes interpreted at least implicitly as an index of effectiveness (e.g., when biological measures show differences in the absence of group differences in RT). For example, studies of conflict adaptation in GAD (Larson, Clawson, Clayson, & Baldwin, 2013) and GAD and OCD (Xiao et al., 2011) found evidence for altered event-related potentials (ERPs) relative to healthy controls (i.e., lower N2 ERPs and enhanced error-related negativity on error trials, respectively), but no differences in accuracy or RT. These lower ERP amplitudes were explicitly interpreted as indicative of compensatory activity in one study (Larson et al., 2013). Consistent with predictions from ACT, ERP amplitude was correlated with anxiety severity in both studies; however, attentional symptoms were not measured.

A final obstacle to a clearly articulated model of the role of "cold" cognitive control in attentional symptoms is that previous research has been grounded in descriptive models of executive functioning (e.g., the tripartite model of working memory, inhibition, and shifting; e.g., Miyake et al., 2000). Although these models have considerable descriptive and organizational value, neurobiologically derived models of cognitive control have begun to gain favor on the grounds that they are more biologically plausible, mechanistic, and flexible with respect to accounting for variability in goal-directed attentional control

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[1] One might reasonably argue that endogenous threat cues (e.g., intrusive thoughts) might help to account for difficulty concentrating in apparently neutral situations. We agree, but also suggest that these cues would not be systematically absent from the laboratory context (which is not traditionally known for being a relaxing experience for participants).

within and between individuals (Braver, 2012; Hampshire & Sharp, 2015). We therefore consider the possibility that certain cognitive mechanisms (i.e., underlying attentional symptoms) that were not observable using earlier models of executive functioning could well emerge when operationalized and measured according to newer (and ostensibly more refined and accurate) models of cognitive control (see Grahek, Everaert, Krebs, & Koster, 2018, for a similar perspective in depression).

Dual mechanisms of control theory (DMC; Braver, 2012; Braver, Gray, & Burgess, 2007) is one such account that is especially promising in terms of both its clinical relevance and neurobiological plausibility. DMC proposes the existence of two dissociable facets of cognitive control: *proactive control* and *reactive control*. Proactive control is a form of early selection (i.e., filtering out irrelevant information at earlier stages of processing) and involves the effortful and active maintenance of task goals over time. By contrast, reactive control is a late-stage correction mechanism that is mobilized only after a conflict or other triggering stimulus is detected. An interesting feature of DMC theory is that proactive and reactive control describe not only abilities, but also strategies that an individual might implement when confronted with a task that involves some form of conflict. In most scenarios, proactive control is the more effective strategy, assuming the individual is able

to sustain the control for as long as is needed (Braver, 2012). However, proactive control is cognitively costly and requires more effort to sustain (Braver, 2012; Westbrook & Braver, 2015). By contrast, a reactive control strategy requires less sustained effort, but leaves more opportunity for attentional capture by irrelevant stimuli and a corresponding failure of goal reactivation (i.e., leading to poor task performance). Broadly speaking, healthy adults tend to favor a proactive control strategy for demanding cognitive control tasks (Paxton, Barch, Racine, & Braver, 2007). However, older age and several forms of psychopathology have been linked to a reduced bias toward proactivity and an increased tendency to favor a less-effortful reactive control strategy (Mann, Footer, Chung, Driscoll, & Barch, 2013; West, Choi, & Travers, 2010). To our knowledge, no studies have applied the DMC framework to study "cold" cognitive control in the context of clinically significant anxiety. However, studies in healthy and analogue samples provide preliminary support for a similar pattern associated with higher state or trait anxiety (Schmid, Kleiman, & Amodio, 2015; Yang, Miskovich, & Larson, 2018).

DMC offers a unique opportunity to reconsider questions about the role of cognitive control in attentional symptoms of anxiety within a contemporary, neurobiologically derived model. Synthesizing across DMC (Braver, 2012), ACT (Eysenck

Table 1
Clinical Characteristics and Standardized Task Performance

	Healthy M (SD)	Clinical M (SD)	p	d	V
Age	40.51 (16.80)	39.95 (16.68)	.684	0.03	
Gender (% female, % transgender)	64%, 3%	52%, 0%	.262		0.18
Education			.641		0.15
High school or less	31%	32%			
Some college	31%	32%			
Bachelor's degree or greater	39%	36%			
Race					
American Indian or Alaska Native	3%	2%	.926		0.01
Asian or Asian-American	6%	2%	.481		0.08
Black or African-American	28%	0%	< .001		0.41
White	67%	91%	< .001		0.41
Ethnicity (% Hispanic or Latinx)	0%	11%	.042		0.23
ACS	59.83 (9.22)	47.69 (10.36)	< .001	1.24	
PSWQ	36.67 (12.20)	64.57 (9.24)	< .001	-2.58	
Y-BOCS-SR Obsessions	2.22 (1.99)	10.18 (3.45)	< .001	-2.83	
DASS-Depression	—	9.47 (6.62)			
Overall accuracy	0.96 (0.05)	0.96 (0.04)	.865	0.00	
AX trial accuracy	0.96 (0.04)	0.97 (0.04)	.852	-0.25	
BX trial accuracy	0.91 (0.16)	0.92 (0.18)	.516	-0.06	
AY trial accuracy	0.93 (0.11)	0.94 (0.09)	.852	-0.10	

Note. d = Cohen's d; V = Cramer's V. ACS = Attentional Control Scale; PSWQ = Penn State Worry Questionnaire; Y-BOCS-SR = Yale-Brown Obsessive Compulsive Scale – Self-Report Version; DASS = Depression, Anxiety, and Stress Scales.

et al., 2007), and other literatures, several non-mutually-exclusive predictions emerge. For example, the subjective experience of difficulty concentrating or other attentional symptoms may relate to impairments in the ability to effectively recruit proactive or reactive control resources to regulate attention. Within the DMC framework, proactive and reactive control impairments might express as subjectively different symptoms; for example, proactive control impairments might relate to difficulty staying focused on a task without becoming distracted (e.g., as reflected in the Focusing subscale of the ACS; Derryberry & Reed, 2002), while reactive control impairments could relate to a reduced ability to shift attention between tasks or mental sets or reinstate attention on goal-relevant activities following distraction (e.g., as reflected in the Shifting subscale of the ACS; Derryberry & Reed, 2002). Extending predictions from ACT, individuals might alternatively (or also) engage in compensatory strategies such as relying on a more effective but also more cognitively demanding proactive control strategy, reflecting a reduction in cognitive efficiency. A final possibility is that anxious individuals may underestimate their attentional control abilities, both relative to healthy controls and relative to their generally intact performance on behavioral tasks.

The present study leveraged recent developments in the basic cognitive control literature (DMC framework; Braver, 2012) to characterize the relationship between attentional symptoms, including difficulty concentrating (focusing and shifting), worry, obsessions, and "cold" cognitive control (proactive and reactive) in a transdiagnostic sample of treatment-seeking adults with GAD and/or OCD and a comparison sample of healthy controls. Extending predictions of ACT (Eysenck et al., 2007), we further considered the relationship between attentional symptoms and cognitive efficiency using a behavioral index of task effort (i.e., the proactive behavioral index [PBI], which provides a ratio of the extent to which participants used a more effortful proactive control strategy versus a less effortful reactive control strategy; Chiew & Braver, 2013; Locke & Braver, 2008). Clinical status was considered as a potential moderator of these relationships, in line with predictions from ACT that efficiency impairments are specific to chronically anxious individuals.

Although a number of specific hypotheses were of interest, three major considerations led us to adopt a comprehensive approach for the analytic plan (with corrections for multiple comparisons), rather than strictly limiting our analyses to only those contrasts that were hypothesis-driven. First,

ACT would predict the null with respect to many comparisons that are commonly made as a matter of course. We did not wish to omit those comparisons, but neither were we in a position to consider them through the lens of traditional null hypothesis testing. Second, competing theoretical accounts and previous studies offer plausible competing hypotheses for most, if not all, of the major relationships of interest. Finally, little is known about the DMC framework in the context of clinically significant anxiety, especially with reference to attentional symptoms. As such, unexpected relationships between the variables could prove informative for future confirmatory research.

Method

PARTICIPANTS

Participants were 44 treatment-seeking adults with *DSM-5* GAD and/or OCD (*n* = 15 GAD-only; 22 OCD-only; 7 both disorders) who were recruited from an outpatient specialty anxiety clinic and 38[2] healthy control participants with no history of mental health problems who were recruited via a research registry and paper flyers advertising a nontreatment study on attention and emotion. Clinical and healthy participants did not differ on age, gender, or educational status; however, the healthy control group was more racially representative compared to the clinical group (Table 1). Two healthy control participants were excluded (one was noncompliant and one was found to have a neurological disorder after participation), leaving a final sample of 36 healthy controls. Other inclusion criteria were selected based on the requirements of the cognitive tasks and included normal or corrected-to-normal vision and English language fluency for all participants and a diagnosis of GAD, OCD, or both disorders for clinical participants. The decision to recruit a mixed clinical group consisting of adults with GAD and/or OCD was made *a priori* to allow for a full range of attentional symptom severity that was not confounded with diagnostic status. Consequently, individuals with comorbid GAD-OCD were not explicitly recruited, but nor were they excluded. Other comorbidities were also permitted with the exception of history of psychotic disorder, mania, traumatic brain injury, organic brain illness, acute suicidality, or current (past 12 months) substance use disorder.

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[2] Ten of the 38 healthy controls were recruited in response to a reviewer request (i.e., to address a significant age difference between healthy and clinical participants) for another manuscript that reported on a different task in the same sample. These participants were therefore also included in the present analyses.

Diagnostic interviews were conducted by trained graduate students or postdoctoral fellows as part of the standard clinical intake procedure (clinical participants) or immediately prior to completion of the experimental procedures (healthy controls). All diagnoses and severity ratings were discussed and finalized by a licensed clinical psychologist with expertise in differential diagnosis of anxiety and related disorders. The experimental session typically took place within 2 weeks of the clinical intake. Participants whose intake took place more than 2 weeks prior to the initiation of data collection were eligible to participate only if their treating or supervising clinician confirmed their current diagnostic status before referring them to the study. Semistructured interviews were not readministered in these cases to reduce participant burden and increase feasibility; however, all referring clinicians were licensed, experienced in administering the semistructured interview that was used as a basis for selection and to assign initial diagnoses, and familiar with the selection criteria for the study.

The experimental session took place in a private room. Participants completed all cognitive and self-report measures during a single session, except for the depression severity self-report measure, which was completed by clinical participants only as part of the standard clinic intake procedure. Cognitive measures were administered prior to the self-report measures. Informed consent was obtained for all participants.

MEASURES

Clinical Measures

Diagnostic Interview for Anxiety, Mood, OCD, and Related Neuropsychiatric Disorders (DIA-MOND; Tolin et al., 2018). Diagnostic status was determined by trained diagnosticians via the DIAMOND, a semistructured diagnostic interview for a wide range of psychological disorders. The DIAMOND has strong psychometric properties, including good interrater reliability (IRR) and test-retest reliability (TRR) for GAD (IRR κ = 0.71; TRR κ = 0.68) and OCD (IRR κ = 0.62; TRR κ = 0.83) in an overlapping sample (Tolin et al., 2018).

Attentional Control Scale (ACS; Derryberry & Reed, 2002). Perceived attentional control was assessed using the ACS, a widely used, 20-item self-report measure of perceived ability to control attention (e.g., "My concentration is good even if there is music in the room around me"). Each item is rated on a scale of 1 (*almost never*) to 4 (*always*), with higher scores reflecting better perceived attentional control. Factor analytic studies suggest

two robust factors, which can be interpreted separately as subscales or treated as a total score (Ólafsson et al., 2011). The 10-item Focusing subscale describes the ability to maintain attention on a task without becoming distracted (α = 0.88 in the present sample), while the Shifting subscale describes the ability to voluntarily shift attention between tasks or mental sets (α = 0.85 in the present sample).

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 excessiveness and uncontrollability of worry in daily life. The PSWQ has strong psychometric properties in both clinical and healthy samples (Brown, Marten, & Barlow, 1995; Meyer et al., 1990). Items are rated on a Likert-type scale from 1 (*not at all typical of me*) to 5 (*very typical of me*) and are scored such that higher scores reflect more severe worry. Internal consistency was strong in the present sample (α = 0.97).

Yale-Brown Obsessive-Compulsive Scale – Self-Report (Y-BOCS-SR; Baer, Brown-Beasley, Sorce, & Henriques, 1993). Obsession severity was assessed using the self-report version of the Y-BOCS, which is a 10-item measure assessing obsessions (items 1–5) and compulsions (items 6–10). Items are rated on a scale from 0–4 with descriptive anchors for each response choice. Obsession items (1–5) were summed to create an overall obsession severity score. Possible scores ranged from 0–20, with higher scores reflecting more severe obsessions. The Y-BOCS-SR has generally good psychometric properties in both clinical and healthy samples (Steketee, Frost, & Bogart, 1996). Internal consistency for the obsession severity score was strong in the present sample (α = 0.91).

Depression, Anxiety, and Stress Scales (DASS-21; Lovibond & Lovibond, 1995). Depression severity was assessed using the 7-item depression subscale of the DASS-21 item version, a widely used self-report measure with strong psychometric properties (Antony, Bieling, Cox, Enns, & Swinson, 1998; Henry & Crawford, 2005). DASS-21 items are rated on a scale of 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*) based on past-week functioning. Scores are summed and then doubled (to maintain comparability with the original 42-item version of the DASS), with higher scores reflecting more severe symptoms. The DASS-21 was administered as part of the clinical intake

but was not formally included in the study procedures; therefore, depression data are only available for clinical participants.

Cognitive Control Task

Proactive and reactive cognitive control. The AX-Continuous Performance Task (AX-CPT; Cohen, Barch, Carter, & Servan-Schreiber, 1999; Edwards, Barch, & Braver, 2010) is a well-established, widely used computerized cognitive measure of proactive and reactive cognitive control that provides an index of ability as well as strategy. Task parameters were implemented as in Edwards et al. (2010), in keeping with recommendations for enhancing sensitivity, specificity, and clinical relevance (MacDonald, 2008). In the AX-CPT, participants view sequentially presented individual letter stimuli, which are organized as cue-probe pairs. Each trial consisted 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). Participants were instructed to press the "target" button when the probe is X, but only if the previous letter (i.e., the cue) was A (A-X trial type). In all other cases, participants press the "non-target" button. 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 provide the "target" response to the probe, especially when the probe is X or the cue was A. Performance on B-X trials is interpreted as an index of proactive control because participants must proactively maintain task information (i.e., the cue was B) to make the appropriate "non-target" response to X. By contrast, performance on A-Y trials is interpreted as an index of reactive control because participants must react to the unexpected presentation of Y by overriding the dominant response cued by the presentation of A on the previous trial. Participants completed 20 practice trials and 120 task trials (84 AX; 12 each of AY; BX; BY).

Participants are instructed to respond "as quickly and as accurately as possible." Performance on the AX-CPT is typically reported in terms of accuracy (or error rate) and/or reaction time (RT). Higher accuracy reflects better performance, while higher RT reflects worse (slower) performance or reduced efficiency. In light of this and previous inconsistencies in the literature described above, we remain agnostic with respect to interpreting RT data as reflecting either effectiveness or efficiency. Instead, we center questions of efficiency on the behavioral index of task effort.

Task effort. The tendency to favor a more effortful proactive control strategy versus a less effortful reactive control strategy on the AX-CPT (i.e., the proactive behavioral index, PBI; Braver, Paxton, Locke, & Barch, 2009) was examined as an index of task effort (Chiew & Braver, 2013; Locke & Braver, 2008). Participants who are implementing a proactive control strategy will tend to perform better on B-X trials but worse on A-Y trials. This is because the response bias generated by the cue facilitates performance on B-X trials, but impedes performance on A-Y trials. Conversely, participants who are favoring a less effortful reactive control strategy (i.e., relying primarily on the probe to guide responding) perform better on A-Y trials but worse on B-X trials. PBI is calculated as (AY-BX)/(AY+BX), with higher scores indicating greater proactivity (Braver et al., 2009; Edwards et al., 2010) and by extension task effort (e.g., Chiew & Braver, 2013; Locke & Braver, 2008).

Participants also completed a pilot version of a novel emotional variant of the AX-CPT that included negative emotional and neutral distractor words (see Hallion, Tolin, and Diefenbach, in press). Order administration of the tasks was counterbalanced.

ANALYTIC PLAN

Mean response time (RT) was calculated for each trial type 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. Predictions were null with respect to accuracy (Eysenck & Derakshan, 2011; Eysenck et al., 2007); however, we present descriptive statistics and independent-samples *t*-tests in the interest of thoroughness. Unless otherwise specified, all references to task performance refer to RT specifically. To confirm the appropriateness of considering GAD and OCD together as part of a single clinical group, we compared GAD-only to OCD-only participants on task performance and effort. We also conducted a series of sensitivity analyses to check whether the results varied when controlling for depression status or when GAD-only or OCD-only participants were excluded (i.e., thereby creating all-OCD and all-GAD clinical groups).

For the main analyses, we conducted a series of moderated regression analyses with bootstrapping using the PROCESS macro (Hayes, 2013). To avoid multicollinearity and enable analysis using PROCESS, each attentional symptom (i.e., focusing; shifting; trait worry; obsession severity) was examined in a separate analysis with correction for multiple comparisons. Each analysis therefore included one attentional symptom as the independent variable (X) and one task outcome (proactive

control [BX] RT; reactive control [AY] RT; task effort [PBI]) as the dependent variable (Y). Diagnostic status (healthy versus clinical) was examined as a moderator. AX trial RT was included as a covariate of non-interest for the analyses predicting proactive and reactive control.

Statistical Power and Correction for Multiple Comparisons

A power analysis revealed that, with $N = 80$ and 3 predictors of interest (4 predictors total in the analyses predicting proactive and reactive control), we would have 80% power to detect a small-to-moderate effect ($f^2 \geq 0.10$) for the major regression coefficients of interest. We corrected for multiple comparisons across regression analyses using Benjamini-Hochberg False Discovery Rate (BH-FDR) procedure (Benjamini & Hochberg, 1995). False discovery rate (FDR) refers to expected proportion of incorrect rejections of the null hypothesis (i.e., Type I error). The BH-FDR approach controls FDR by comparing the *p*-value for each family of test statistics to a list of critical values using a step-up (i.e., sequential; smallest-to-largest) approach. Each critical value is corrected for the number of statistical tests and the researcher-set FDR rate using the formula $(i/m)Q$, where i = the rank of the to-be-tested *p*-value (beginning with 1 for the smallest *p*-value and increasing sequentially), m = the number of statistical tests, and Q = the researcher-set FDR (set at 0.05 for the present study). The largest *p* value that is significant for $p < (i/m)Q$ is considered significant, as are all smaller *p*-values, irrespective of their individual critical value score. BH-FDR is more powerful than the Bonferroni familywise error rate correction and is proposed to be more appropriate when the research includes an exploratory component (Benjamini & Hochberg, 1995; Kwong, Holland, & Cheung, 2002).

Results

PRELIMINARY ANALYSES

Missing data represented less than 1% of observations and were determined to be missing completely at random (Little's MCAR χ^2 (15) = 12.52, p = .640). Missing values were handled using pairwise deletion. Clinical and demographic characteristics are presented in Table 1.

Accuracy approached ceiling and did not differ as a function of group (clinical versus healthy) overall or for any of the three main trial types (AX, BX, AY; BY trials were not analyzed), all $t(68) \leq 0.65$, all $p \geq .516$ (see Table 1). To conserve statistical power, we did not consider accuracy further (i.e., in light of the restricted range, all $M = 0.92 - 0.97$, and

because hypotheses were null with respect to accuracy; Eysenck et al., 2007).

To assess the appropriateness of collapsing GAD and OCD participants into a single clinical diagnostic group, response time, and effort (PBI) were compared for GAD-only versus OCD-only participants. GAD-only and OCD-only participants did not differ on any outcome, all $t(33-34) \leq |0.71|$, all $p \geq .436$. The pattern of results was also identical in follow-up sensitivity analyses that excluded GAD-only or OCD-only participants, except for one significant interaction (between clinical status and trait worry) that became marginally significant ($p = .055$) when GAD-only participants were excluded. Results also did not differ when depression status was statistically controlled. The clinical groups were therefore collapsed for all subsequent analyses and results are reported without controlling for depression.

Relationship of Perceived Attentional Control to Cognitive Control and Effort

Results of the bootstrapping moderation analyses are presented in Table 2. There was a significant interaction between ACS Focusing and diagnostic status in predicting proactive control response time. Follow-up conditional effects analyses revealed that, in healthy participants, higher (better) Focusing predicted lower (better) response time on proactive control trials ($B = -5.97$, $p = .025$), although this effect did not survive correction for multiple comparisons. No relationship was observed in clinical participants ($B = 2.50$, $p = .282$). This interaction qualified a main effect of diagnostic status (wherein clinical participants reported lower focusing ability overall) and a main effect of Focusing (better perceived focusing was associated with faster performance) that did not survive correction for multiple comparisons. ACS Shifting was not associated with proactive control response time. There was no relationship between Focusing or Shifting and reactive control response time. There was also no relationship between Focusing or Shifting and task effort.

Relationship of Trait Worry to Cognitive Control and Effort

There was a significant interaction between PSWQ and diagnostic status in predicting proactive control response time (see Table 2), which survived correction for multiple comparisons. In healthy participants, higher trait worry predicted slower performance on proactive control trials ($B = 2.59$, $p = .020$). This relationship was marginal and in the opposite direction in clinical participants ($B = -2.54$, $p = .068$; see Figure 1). This interaction qualified a main effect of trait worry, wherein higher trait worry

Table 2

Hierarchical Regression Predicting Task Effort, Proactive Control Performance (RT), and Reactive Control Performance (RT)

	Proactive Control (RT)				Reactive Control (RT)				Task Effort (PBI)			
	B	Δ R²	p	f²ᵃ	B	Δ R²	p	f²ᵃ	B	Δ R²	p	f²ᵃ
ACS Focusing												
Model Summary		.67	< .001*			.68	< .001*			.06	.237	
AX RT	1.06		< .001*	1.90	1.01		< .001*	2.17	—	—	—	—
ACS-Focusing	-5.97		.025	0.07	-2.83		.237	0.02	.00		.494	0.01
Group	-204.72		.017	0.01	-17.61		.818	0.01	.17		.087	0.00
ACS-Focusing x Group	8.47	.03	.017	0.07	.15	.00	.962	0.00	-.01	.04	.078	0.04
ACS Shifting												
Model Summary		.66	< .001*			.69	< .001			.05	.285	
AX RT	1.07		< .001*	1.85	1.01		< .001	2.20	—	—	—	—
ACS-Shifting	-4.95		.073	0.04	1.39		.558	0.00	.00		.207	0.02
Group	-166.16		.142	0.02	152.14		.121	0.00	.25		.063	0.00
ACS-Shifting x Group	4.91	.01	.167	0.02	-5.41	.01	.080	0.04	-.01	.05	.060	0.05
Trait Worry												
Model Summary		.68	< .001*			.69	< .001*			.14	.011*	
AX RT	1.02		< .001*	1.76	1.00		< .001*	2.16	—	—	—	—
PSWQ	2.59		.020*	0.08	-0.93		.354	0.01	-0.003		.012*	0.09
Group	261.86		.010*	0.01	-142.35		.103	0.00	-0.35		.004*	0.00
PSWQ x Group	-5.13	.04	.005*	0.07	2.56	.01	.103	0.04	0.01	.13	.001*	0.15
Obsession Severity												
Model Summary		.65	< .001*			.68	< .001*			.04	.353	
AX RT	1.07		< .001*		.99		< .001*	2.08	—	—	—	—
Y-BOCS-Obsess	-8.57		.220		8.32		.175	0.02	.01		.078	0.04
Group	-49.30		.268		1.39		.971	0.02	.04		.489	0.02
Y-BOCS-Obsess x Group	11.74	.01	.137		-6.92	.00	.316	0.01	-.01	.03	.127	0.03

Note. * = significant after BH-FDR correction. ᵃEffect size estimates are not available through PROCESS; estimates were therefore calculated as $f^2 = r_{sp}^2 / (1 - R^2)$, where r_{sp}^2 = semi-partial correlation estimates from a standard linear regression. Effect sizes should therefore be interpreted as approximate. Group = clinical or healthy control. RT = Reaction time. PBI = Proactive behavioral index. Higher RT = slower (worse) task performance. ACS = Attentional Control Scale; PSWQ = Penn State Worry Questionnaire; Y-BOCS-Obsess = Yale-Brown Obsessive Compulsive Scale – Self-Report Version Obsession subscale.
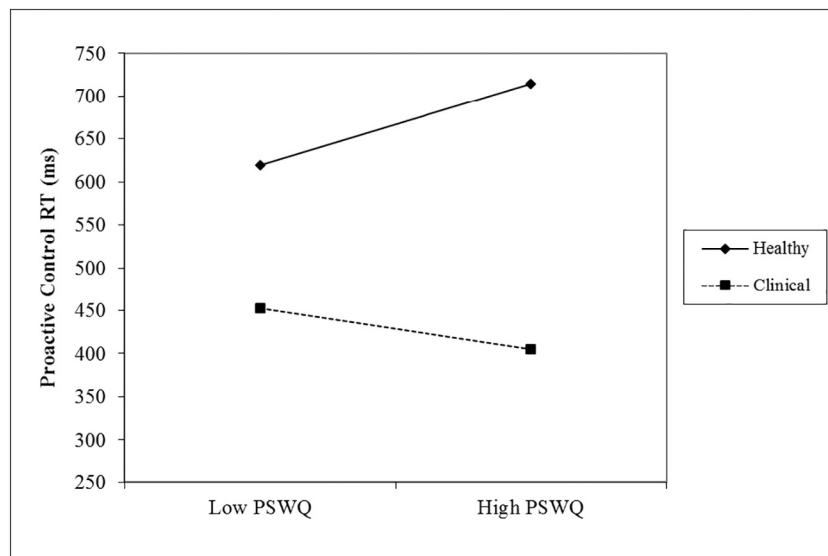

FIGURE 1 Interaction between diagnostic status and trait worry predicting proactive control. Higher values reflect slower (worse) performance. RT = Reaction time. PSWQ = Penn State Worry Questionnaire.
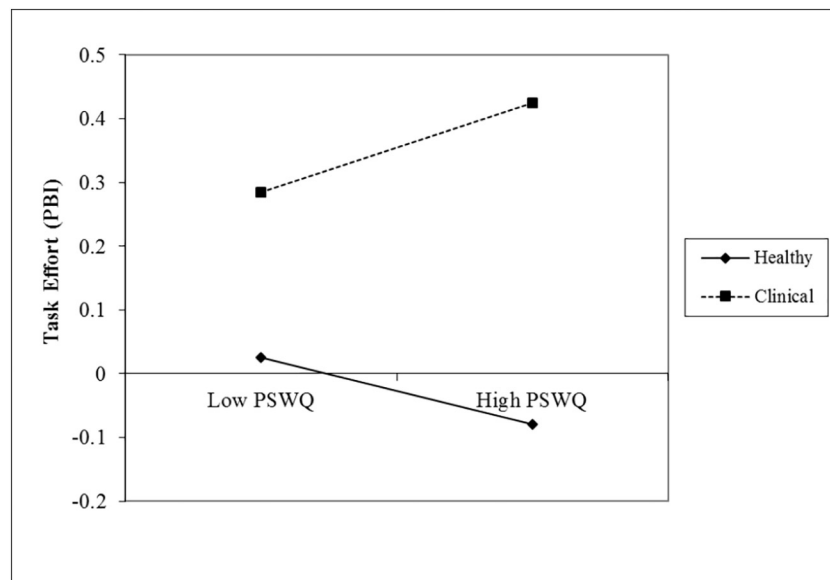
FIGURE 2 Interaction between diagnostic status and trait worry predicting task effort. Higher values reflect greater effort. PBI = Proactive behavioral index. PSWQ = Penn State Worry Questionnaire.

predicted slower performance, and a main effect of group, wherein clinical participants showed faster proactive control response times overall. There was no relationship between trait worry and reactive control response time.

Additionally, there was a significant interaction between trait worry and diagnostic status in predicting task effort, which again survived correction for multiple comparisons. In healthy controls, higher trait worry predicted lower effort ($B = -0.003$, $p = .012$), whereas higher trait worry was associated with greater effort in clinical participants ($B = 0.004$, $p = .027$; see Figure 2). This interaction qualified a main effect of group, wherein clinical participants exerted greater effort, and a main effect of trait worry, wherein higher trait worry was associated with lower effort overall.

Relationship of Obsession Severity to Cognitive Control and Effort

There were no significant main effects or interactions for obsession severity predicting proactive or reactive control trial performance or task effort.

Discussion

The primary aim of the present study was to investigate the extent to which alterations in two facets of "cold" cognitive control, proactive and reactive, might help to account for attentional symptoms (difficulty concentrating; worry; and obsessions) in the context of clinically significant anxiety (specifically, GAD and/or OCD). Clinical participants reported more severe attentional symp-

toms in daily life relative to healthy controls, but performed comparably, and in some cases out-performed healthy controls in terms of response time, on an objective measure of cognitive control (the AX-CPT; Cohen et al., 1999; Edwards et al., 2010). Moreover, the relationship between attentional symptoms and response time varied by clinical status, such that attentional symptoms (specifically, focusing and worry) were associated with slower responding on proactive control trials and lower effort in healthy participants, but faster responding and greater effort in clinical participants (although the pattern in clinical participants was nonsignificant after correction for multiple comparisons).

The positive relationship between perceived focusing ability and proactive control efficiency in healthy participants is intuitively appealing and in line with predictions. Items on the Focusing subscale tap the perceived ability to exert top-down control to sustain task-related attention over time, which maps on cleanly to the definition of proactive control offered by Braver (2012). Thus, the finding of a positive relationship between self-reported Focusing and task-indexed proactive control in healthy participants offers some support for the convergent validity of the self-report measure, although it should be noted that this validity was observed only for healthy participants; no relationship between self-reported attentional control and task performance was found in clinical participants. This pattern is especially notable

because the clinical subsample was larger and therefore better-powered to detect a difference. These findings suggest that discrepancies between subjective attentional impairment but broadly intact objective cognitive task performance may be driven in part by lower metacognitive accuracy (including a tendency to underestimate one's abilities) on the part of clinical participants. Notably, the parallel *a priori* predictions linking Shifting to reactive control were not fully supported. There were some hints of a relationship in the form of a marginally significant interaction (i.e., between diagnostic status and perceived shifting ability in predicting reactive control), but the lack of a significant relationship even in the presence of generally good statistical power suggests that the Shifting subscale may not be a valid index of reactive control.

The findings related to trait worry also aligned with predictions in some respects while diverging in others. Whereas healthy participants showed an inverse relationship between proactive control and trait worry, consistent with a deficit model (e.g., Hirsch & Mathews, 2012), this pattern was not observed in clinical participants. Similarly, effort and its relationship to trait worry also differed by group. Consistent with predictions from ACT (Eysenck & Derakshan, 2011; Eysenck et al., 2007), clinical participants exhibited greater effort overall compared to healthy controls. Increased task effort was associated with lower trait worry in healthy participants, which is broadly consistent with a model of increased effort as generally adaptive in healthy individuals. The pattern was significantly different in clinical participants (i.e., as indicated by a significant interaction that survived correction for multiple comparisons). However, the follow-up contrast showing a positive relationship between task effort and trait worry in the clinical group specifically did not survive correction for multiple comparisons, precluding strong conclusions. Nevertheless, future research should consider the possibility that increased effort may track with symptoms in anxious adults. This possibility aligns with previous findings of elevated performance standards or perfectionism associated with GAD and OCD (e.g., Frost & Steketee, 1997; Gentes & Ruscio, 2014) and may help to resolve some of the discrepancies in the extant literature regarding cognitive control in the context of these disorders.

These findings raise the question of why significant relationships were found for proactive but not reactive control. The pattern cannot be attributed to psychometric issues, since the variance and range were comparable across proactive and reactive control trials. One possibility is that the ability to

sustain goal-directed attention over time is simply more important to the clinical experience of attention dysregulation, compared to the ability to employ a late-stage correction. The failure to find a relationship between attentional symptoms and reactive control (which is most similar to inhibition in the tripartite model; Miyake et al., 2000) adds to the growing but inconsistent literature on the role of inhibition in anxiety-related psychopathology and attentional symptoms. Future research using more face-valid measures of the ability to inhibit dominant cognitive responses (e.g., tasks that do not rely on a motor response, perhaps including EEG or fMRI) and better-powered studies investigating potential moderators (e.g., stimulus valence) are needed to clarify these relationships. By contrast, proactive control does not cleanly map on to any single construct from the tripartite model. For example, better proactive control would most likely be associated with better performance on a working memory task, but its relationship to shifting and inhibition performance would more likely be variable and task-dependent. As such, fewer studies have investigated proactive control as a construct of interest in its own right.

Another question is why trait worry, but not obsessions, showed a relationship with proactive control and task effort. One possible explanation derives from theoretical models that describe pathological worry as a maladaptive emotion regulation strategy that is at least semi-voluntary (e.g., Borkovec, Alcaine, & Behar, 2004; Llera & Newman, 2014; Mennin, Heimberg, Turk, & Fresco, 2005). According to these models, worry may be effortfully and voluntarily initiated or sustained in order to avert some more aversive emotional experience. As such, it logically follows that more severe or persistent worry could be associated with better proactive control in clinical (but not healthy) samples. By contrast, obsessions are by definition involuntary. Although one might expect that obsessions could relate to a failure of proactive control (e.g., in preventing intrusive thoughts from competing for access to awareness) or reactive control (e.g., in disengaging attention from off-task obsessions in order to maintain task focus), it is possible that other mechanisms that are not yet fully-characterized within the DMC framework (e.g., the rebound effect of thought suppression; Abramowitz, Tolin, & Street, 2001) may represent missing pieces of the puzzle that may help to explain successful and failed cognitive regulation of worry and obsessional thoughts.

These findings should be interpreted in light of the study's strengths and limitations. One strength is the use of a transdiagnostic clinical anxiety

sample. This strength is especially important in light of our findings that patterns of results varied as a function of clinical status. Future research should replicate the findings in a larger and more diverse transdiagnostic sample, ideally with an eye toward identifying the specific features or mechanisms that account for this moderation (e.g., global anxiety levels; specific diagnoses or symptom constellations; motivational factors, etc.). A nonanxiety clinical control group would also be valuable to establish the extent to which the findings can be attributed to anxiety *per se*, versus general distress or participation in psychotherapy. A limitation is the absence of a direct experimental manipulation or assessment of state anxiety or subjective attentional symptoms (i.e., subjective difficulty concentrating, worry, and obsessions) during the task. Although "cold" (baseline) cognitive processes were of specific interest in the present study, a complete picture of cognitive control over attentional symptoms in the context of anxiety will necessarily involve a clear articulation of the roles and dynamics of both "cold" and "hot" cognitive processing as they relate to symptoms. Experimental research (e.g., involving manipulation of attentional symptoms, cognitive control, or effort) and prospective studies will also be critical for identifying any causal, mechanistic relationships that may exist between these variables. Finally, although the use of a behavioral index of task effort was a strength, the fact that it was embedded into the task (and therefore not entirely independent) is also a limitation. Future studies would benefit from using complementary methods, such as pupillometry, self-report, and neuroimaging, to provide a richer perspective on the relationships between task effort, task performance, and subjective perceptions. A latent variable approach would also be valuable as a means of reducing task-specific error variance and improving the reliability and ideally replicability of findings.

The present findings have potentially important clinical and empirical implications. Taken together, the present findings highlight proactive control as a promising mechanism for future research in the context of anxiety. However, they also raise important theoretical and methodological issues that must be considered in the conduct of this research, particularly with respect to measurement (e.g., reliance on self-reported attentional control) and participant characteristics (e.g., differential relationships as a function of diagnostic status). First, these findings suggest differential validity of the ACS with respect to objective (specifically, proactive control) task performance in healthy versus clinical participants. Researchers and clinicians should therefore be careful to conceptualize the ACS and other cognitive self-report measures as indicative of *subjective perceptions* only, being careful to avoid any assumptions about actual ability except where converging neuropsychological evidence is available. With respect to treatment, the present findings are more in line with a metacognitive approach focused on challenging distorted self-appraisals and maladaptive beliefs about worry (e.g., Mennin et al., 2005) as compared to a cognitive training approach focused on remediating symptom-specific deficits in cognitive control. Nevertheless, the present findings await replication in larger samples. It will be especially important for future studies to consider the extent to which findings apply in clinically-relevant contexts, including in real-world scenarios and in the presence of emotionally-evocative stimuli.

Conflict of Interest Statement

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