

Attentional Control Mediates Fearful Responding to an Ecologically Valid Stressor

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RUNNING HEAD: ATTENTIONAL CONTROL

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For Peer Review Only

Abstract

Background and Objectives: Attentional control (AC) is defined as the ability to voluntarily shift and disengage attention, and is thought to moderate the relationship between pre-existing risk factors for fear and the actual experience of fear.

Design: This longitudinal study elaborates on current models of attentional control by examining whether AC moderates or mediates effects of an ecologically valid stressor (a college exam), and also whether AC is predictive of state-like fear over longer timescales than previously reported.

Methods: Based on previous findings we hypothesized that AC would moderate the relationship between trait anxiety and affective distress in response to the exam stressor. We also tested a competing mediational model based on attentional control theory (Eysenck et al., 2007). These models were tested in two separate samples (Sample 1 N=219; Sample 2 N=129; Total N= 348) at two time points, at the beginning of a college semester in a large undergraduate class, and five minutes prior to a college exam.

Results: Mediation but not moderation of anxiety by AC was supported in both samples using multiple dependent measures.

Conclusion: We conclude that AC may be useful in predicting affective distress in naturalistic settings, particularly in cases where anxiety is anticipatory.

Keywords: Anxiety, Attention, Mediation, Moderation, Stress.

Attentional Control Mediates Fearful Responding to an Ecologically Valid Stressor

Attentional control (AC) is defined as the ability to voluntarily shift and disengage attention, and subsumes distinct abilities that involve disengagement of attention and maintenance of ongoing attentional focus (Derryberry & Reed, 2002; Fox, Russo, & Dutton, 2002; Friedman & Miyake, 2004). There is increasing evidence that individual variation in AC not only exists (Olafsson et al., 2011; Peers, Simons, & Lawrence, 2013), but also that variation in this trait is linked to normative variation in emotional development (Morasch & Bell, 2012; Tottenham, Hare, & Casey, 2011) as well as pathological affective conditions such as social anxiety (Morrison & Heimberg, 2013; Wieser, Pauli, & Muhlberger, 2009), trait anxiety (Bishop, 2009) excessive worry (Hirsch & Mathews, 2012) and depression (Korgaonkar, Grieve, Etkin, Koslow, & Williams, 2012). Current influential theories propose that individuals high in AC are able to use attention to constrain their emotions by either orienting away from threat-stimuli, including environmental threats as well as internal sensations vis a vis attentional inhibition (Ferri, Schmidt, Hajcak, & Canli, 2013), or orienting toward “safe” stimuli, possibly including direct coping strategies such as cognitive reappraisal (Derryberry & Reed, 2002; Gross, 2002; Ochsner & Gross, 2008).

A growing body of literature has clarified the behavioral consequences of intra-individual variation in AC. For example, it is increasingly appreciated that individual differences in AC may play an important role in both normative (Perez-Edgar & Fox, 2007) and pathological outcomes (Kanske & Kotz, 2012). In the latter case, it has been speculated that low attentional control might be linked to psychopathology because pre-existing vulnerabilities are effectively uncensored due to a broad failure to deploy regulatory resources (Armstrong, Zald, & Olatunji, 2011; Derryberry & Reed, 2002; Levens, Muhtadie, & Gotlib, 2009). In the case of anxiety, diminished attentional control may mechanistically confer risk for pathological fear by reducing one’s ability to deploy coping or reappraisal strategies (Lonigan & Phillips, 2001). Consistent with this prediction, Jones,

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3 Fazio, and Vasey (2012) demonstrated that among individuals with a fear of public speaking, only
4 those low in attentional control experienced a subsequent negative impact on speaking performance.
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6 Indeed, if the selection and execution of regulatory strategies require a focusing of attentional
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8 resources, and if such attentional resources are degraded, then the degree of self-reported anxiety
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10 could be expressed as a joint effect incorporating pre-existing risk factors for anxiety and the
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12 “filtering” effect provided by AC.
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18 The ability to deploy complex and effortful regulatory processes such as cognitive
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20 restructuring should therefore be impacted by individual differences in AC, because a stronger or
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22 weaker capacity to disengage attention from threat should be linked to greater or lessened ability to
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24 respond to a stressor with an effortful regulatory or coping strategy (respectively). A related
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26 observation was reported in a recent study by Robinson, Ode and Hilmert (2014) in which cortisol
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28 reactivity during the Trier Social Stress Test (Kirschbaum, Pirke & Hellhammer, 1993) was linked to
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30 reductions in attentional control, and reductions in AC were further linked to increases in rumination
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32 and worry. Along the same lines, Putman and colleagues (2013) report resting-state
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34 electroencephalography (EEG) data from subjects who either completed a stress-induction task or
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36 control procedure (difficult or easy mental arithmetic under observation or no observation
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38 conditions, respectively). Results of this study indicated that a potential EEG biomarker of
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40 prefrontally situated attentional and inhibitory functions, the theta to beta ratio, moderated the
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42 relationship between stress and self-reported attentional control. Richey Keough and Schmidt
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44 (2012) also reported similar results from a lab-based study in which individual variability in AC was
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46 found to moderate the relationship between trait anxiety and the amount of fear one reports during a
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48 highly standardized stressor (a single, vital capacity inhalation of CO₂ enriched gas mixture). Results
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50 indicated that individual differences in AC moderated the relationship between trait anxiety, as
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52 measured by the State-Trait Anxiety Inventory (STAI: Spielberger, 1970, 1983, 1989) and self-
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3 reported fear (subjective units of distress; SUDS) in response to inhaling the CO₂ enriched gas
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5 mixture, such that individuals high in trait anxiety but also high in AC reported comparatively less
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7 fear in response to the stressor than individuals high in trait anxiety but low in AC. Conversely,
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9 individuals high in trait anxiety but low in AC reported significantly greater fear in response than
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11 high AC counterparts. Although these findings indicate that attentional control (AC) plays an
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13 important role in modulating the immediate experience of fear, it is still unknown whether AC
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15 predicts the experience of acute fear across longer timescales, and whether these effects apply
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17 equally to naturalistically occurring (rather than experimentally controlled) stressors.
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22 To further investigate the role of AC in the relationship between trait anxiety and stress, we
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24 also considered an alternative prediction based on attentional control theory (Eysenck, Derakshan,
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26 Santos, & Calvo, 2007), specifically, that AC *mediates* the effect of trait anxiety on acute stress
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28 responses. Attentional control theory proposes that anxiety interferes with the inhibition, shifting and
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30 updating processes of working memory, resulting in a discrete reduction in cognitive performance
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32 because anxiety-relevant stimuli receive prioritized access to limited attentional resources
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34 (Derakshan, Smyth, & Eysenck, 2009; Vuilleumier, 2005). Evidence for attentional control theory
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36 currently comes from studies that are exclusively lab-based, which indicate only that this
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38 relationship occurs in the context of carefully manufactured and highly standardized stressors. While
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40 lab-based experiments such as these have established crucial features related to hypothesized
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42 mechanisms of action in the attention-anxiety interface, an important next step in progress toward
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44 new treatments is establishing whether the same principles apply equally to stressors that occur in
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46 the course of everyday life.
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53 The notion that individual variation in AC should longitudinally predict pathological fear is
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55 consistent with a central prediction of attentional control theory: that degraded attentional control
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57 processes should be a primary cognitive vulnerability factor for the development and maintenance of
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pathological forms of anxiety, because chronic reductions in a limited-capacity resource such as the executive control of attention may subsequently and consistently impair one's ability to deploy coping resources in response to a stressor (Eysenck et al., 2007; Coombes et al., 2009). While no studies have directly tested this hypothesis, a small number of previous reports have evaluated similar questions, by assessing the longitudinal impact of AC on general affective functioning (Belsky, Pasco Fearon, & Bell, 2007; Busch & Hofer, 2012; Morasch & Bell, 2012; Morrison & Heimberg, 2013; Posner & Rothbart, 2009). For example, Belsky, Pasco Fearon and Bell (2007) reported data from a large-scale longitudinal study of childcare and youth development, in which AC was found to partially mediate the effects of parenting on externalizing problems in youth. Similarly, Morrison and Heimberg (2013) report data from a two-part study, which included longitudinal data from 50 individuals with high levels of social anxiety. Participants were measured at three time points, each separated by roughly three to four months, and attentional control was found to mediate the effects of social anxiety on self-reported positive affect, even after controlling for depression. Interestingly, these longitudinal studies consistently report a mediational, rather than moderational effect for AC in predicting affective functioning, which may have to do with the prediction of trait-like, rather than state-like aspects of affective function. Thus, it still unknown whether the longitudinal relationship between AC and affective function holds true for transient or state-like aspects of affective functioning in a manner consistent with attentional control theory.

Accordingly, the purposes of the current study are threefold: 1) establish the prospective relationship between AC and state-like forms of anxiety over longer timescale than previously reported, 2) determine the extent to which predictions apply to ecologically valid, albeit less standardized stressors, 3) test in the same samples two alternative hypotheses regarding the role of AC, as either moderator or mediator in the relationship between trait anxiety and acute responses to stress. In addition, we also conducted an exploratory analysis in which we evaluated the specificity

of AC in predicting debilitating versus facilitative forms of stress (i.e. stress that increases performance versus stress that impairs performance, respectively). To accomplish these inter-related objectives, the current report presents data from two longitudinal studies, a validation sample and a replication sample, in which AC was hypothesized to predict the degree of self-reported fear during an ecologically valid stressor (a college exam). We measured both samples at two time points separated by approximately three weeks. Consistent with our previous lab-based study, we predicted a moderational effect for AC, in which individuals high in trait anxiety and low in AC would be at particularly elevated risk for anxious responding on the day of the test, whereas those high in trait anxiety but also high in AC would be at comparatively less risk for fearful responding due to the putative potentiating or filtering effect of AC on these groups, respectively. We also tested a mediational model, based both on attentional control theory (Eysenck et al., 2007) as well as previous support for a possible mediational role for AC in predicting stable or trait-like aspects of temperament and personality (e.g., Belsky et al., 2007; Morrison & Heimberg, 2013).

Study 1: Method

Participants and Overview of Procedure

Sample 1 consisted of 448 nonclinical participants. Only participants who completed both Time 1 (T1; baseline) and Time 2 (T2; stressor) time points (N=219) comprised the final sample. The 219 participants who completed both time points were included in the analysis (mean age 18.42 years, SD .82; 63.3% female, 36.7% male; 78.9% Caucasian, 6.0% African-American, 7.8% Hispanic/Latino, 3.2% Asian, 2.3% Native American, 1.8% Other/Did not wish to disclose). No participants were excluded based on demographic or any other characteristics. All participants were students at a public university in the southeastern United States. At Time 1 (T1; baseline) collected within the first week of the semester, a series of self-report questionnaires were administered in a single classroom session lasting approximately 20 minutes. Approximately three weeks later at Time

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3 2 (T2: Stressor), participants completed a self-report measure of subjective test anxiety immediately
4 before a college exam.
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8 *Measurement Point 1: Baseline*
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10 *Attentional Control Scale (ACS)*. The ACS is a 20-item self-report measure of attentional
11 control (Derryberry & Reed, 2002; Derryberry & Rothbart, 1988). The total score indexes both
12 attentional focus, the ability to maintain sustained attention (e.g., “When I am working hard on
13 something, I get distracted by events around me”) and attentional shifting, the ability to switch
14 attention from one focal point to another (e.g., “I have trouble carrying on two conversations at
15 once”). The psychometric properties of the ACS appear favorable in both adult (Derryberry & Reed,
16 2002; Morrison & Heimberg, 2013; Olafsson et al., 2011) and youth samples (Muris, de Jong, &
17 Engelen, 2004; Muris, Mayer, van Lint, & Hofman, 2008). The ACS total scale score used in the
18 present study demonstrated adequate internal consistency ($\alpha=.87$).
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31 *Spielberger State-Trait Anxiety Inventory (STAI) – Trait version*. The STAI is a widely used
32 and well-validated measure of anxiety (Spielberger, 1970, 1983, 1989). Items are rated using a four-
33 point scale from 1 (“not at all”) to 4 (“very much so”), to indicate how well a given statement
34 describes the participant at that particular moment. The current study utilized the trait version, which
35 is identical in nearly all respects to the state version, except that the participant is instructed to rate
36 each statement in terms of how he or she *generally feels*. A wide body of research has supported the
37 construct validity, test-retest reliability, and reliability of the STAI (Sharma, 1977; Guthrie &
38 Lonner, 1986; Spielberger, 1989). The STAI-T total scale demonstrated good internal consistency in
39 the current sample ($\alpha=.90$).
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52 *Measurement Point 2: Stressor*
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55 *Test Anxiety Measure: Worry-Emotionality Scale –Revised (WES-R)*. At time 2,
56 participants completed the WES-R, a 47-item measure of test anxiety focused on state-like fears
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3 associated with the test stressor. Items such as “I am concerned about the possibility of making a
4 bad grade” are rated on a 5-point scale (1= very much true; 5= not at all true). The original WES
5 was developed by Liebert and Morris (1969) as a brief (10-item) measure of test anxiety. The
6 revised version (Morris, Davis & Hutchings, 1980) contains 47 items also rated on a 1- to 5-point
7 scale, and has adequate psychometric properties including internal consistency estimates of
8 approximately .83 (Morris et al., 1981). In the current study, WES-R demonstrated excellent
9 internal consistency ($\alpha=.96$).

10 11 *Overview of Analytic Strategy*

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13 Zero-order correlations between theoretically relevant variables were examined and tests of
14 moderation and mediation were conducted using Hayes’s (2012) PROCESS macro for SPSS.
15 PROCESS uses a regression-based path analytical framework based on a traditional OLS approach as
16 well as bootstrapped estimation of indirect effects for testing mediation. For both samples, we tested
17 two primary models: simple mediation (Hayes’ 2012 PROCESS model 4), in which ACS was
18 evaluated as a potential mediator in the relationship between trait anxiety at T1 and reactivity to the
19 stressor at T2, and simple moderation (PROCESS model 1), in which ACS was evaluated as a
20 potential moderator in the relationship between trait anxiety at T1 and reactivity to the stressor at T2.
21 For tests of mediation, regression analyses of total effect (path c), direct effect (path c'), and
22 bootstrapped bias-corrected 95% confidence intervals of the indirect effect (product of a path and b
23 path) were computed using the PROCESS in SPSS (Hayes, 2012) with 5000 bootstrapped samples
24 following the procedure outlined in Preacher and Hayes (2008). Confidence intervals that do not
25 contain zero indicate a significant indirect effect (mediation). Effect sizes were calculated for all
26 significant indirect effects using the recommended Preacher and Kelly (2011) kappa-squared (κ^2)
27 measurement. κ^2 ranges from 0 to 1 and indicates “the proportion of maximum possible indirect effect
28 that could have occurred” (Preacher & Kelley, 2011). This is a standardized measure of effect size not
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3 influenced by the variable scales and independent of sample size. Its value can be interpreted using
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5 guidelines similar to those for Cohen's (1988) squared correlation coefficients, where .01, .09, and .25
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7 κ^2 values equate to Cohen's "small," "medium," and "large" effect sizes, respectively (Preacher &
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9 Kelley, 2011).
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12 Study 1: Results

13 *Descriptive Data and Zero-Order Relations among Theoretically-Relevant Variables*

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15 Means, standard deviations and zero-order correlations for all variables are presented in
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17 Table 1. Age and gender were correlated indicating males were significantly older than females in
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19 the sample ($r = -.45, p < .001 = .0006$); otherwise, demographic factors (age, race, and gender) did
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21 not correlate with other study variables. Consistent with the conceptual model, the ACS was
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23 significantly (negatively) correlated with anxiety-relevant measures such as STAI ($r = -.49, p <$
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25 $.001 = .0005$) and WES-R ($r = -.45, p < .001 = .006$).
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32 *Power Analyses*

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34 *Moderational Model.* In order to identify the minimum required sample size to detect the
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36 involved interaction term and therefore provide an adequate test of moderation, a power analysis was
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38 conducted using GPower (v.3.1). We followed the recommendations of Faul, Erdfelder, Buchner, and
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40 Lang (2009) for evaluating the deviation of a subset of linear regression coefficients (example 4.2), to
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42 specifically determine the minimum sample size required to detect an r^2 increase for the interaction
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44 term across a range of effect sizes that included traditional cutoffs for small ($f^2=0.02$), medium
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46 ($f^2=0.15$) and large ($f^2=0.35$) effect sizes (with cutoff values for f^2 drawn from Cohen, 1977; 1988). In
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48 accordance with the procedure of Faul and colleagues (2009), we specified the total number of
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50 available predictors as 3: (1) main effect of predictor A, (2) main effect of predictor B, and (3) the
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52 interaction of A*B, and the number of predictors to be tested as 1. Results indicated that with $\alpha=0.05$,
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54 and a desired power of 0.80, a minimum sample of 95 subjects would be required to detect a 'medium'
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3 effect size ($f^2=0.15$). We also include a visual depiction of the joint distributions of predictors as
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6 Figure 1, in order to provide an overview of the variance structures encountered in our data.
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8 *Mediational Model.* We computed the required sample size to test for mediation, using the
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10 Vittinghoff, Sen, and McCulloch (2009) method as implemented in the publicly available R package
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12 ‘powerMediation.’ Using the same parameters outlined above ($1-\beta = 0.8$, $\alpha=0.05$), and the observed
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14 correlation between the predictor x and mediator m (STAI and ACS, respectively), we identified a
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16 minimum required sample of 115 cases to detect a medium effect size, which is also generally
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18 consistent with previous published guidelines for detecting a partially mediated effect of a medium
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20 effect size (N=118; Fritz & MacKinnon, 2007).
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24 *Moderational Analysis*

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27 *Primary Model: Prediction of Test Anxiety (WES-R).* We used the PROCESS macro (model 1)
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29 to test the hypothesis that ACS moderates the relationship between trait anxiety and the actual
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31 experience of affective distress in response to an ecologically valid stressor. To test the conditional
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33 effect, we evaluated a simple moderation model with the effect of time 1 STAI (X) on time 2 WES-R
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35 (Y) moderated by time 1 ACS. Results of this analysis revealed that higher levels of Time 1 STAI
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37 predicted greater Time 2 WES-R ($b = 1.577, p < .001 = .0002$), as did lower levels of Time 1 ACS ($b =$
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39 $-.863, p < .001 = .0004$); however, there was no interaction between trait anxiety and attentional control
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41 ($b = .007, p = .721$), suggesting that the influence of ACS on the relationship between STAI and WES-
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43 R was not moderational in nature.
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48 *Mediational Analysis*

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50 In light of emergent literature suggesting a possible mediational role for ACS in predicting fear
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52 (e.g. Morrison & Heimberg, 2013), we also tested the possibility that the relationship between ACS,
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54 STAI and fearful responding might be mediational rather than moderational in nature.
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Primary Model: Prediction of Test Anxiety (WES-R). Using Hayes' 2012 PROCESS model 4, a test of simple mediation (model 4) was used to evaluate the effect of state anxiety (STAI) at time 1 (X), via attentional control (ACS) on state-like test anxiety (WES-R) at time 2 (Y). As illustrated in Figure 2, Time 1 STAI predicted Time 2 WES-R, and this relationship was partially mediated by Time 1 ACS ($\kappa^2 = .120$, Boot SE: .035, Boot CI_{95%}: .057 to .194), providing initial support that the relationship between STAI and WES-R is mediated by attentional control.

Study 1: Discussion

The collective interpretation of results from study 1 is that although no evidence was found for a moderational role of ACS, we found that attentional control mediated the effect of trait anxiety on fearful responding to the test stressor, and that this indirect effect was medium in size by conventional standards (Cohen, 1977; 1988). To attempt to replicate this finding, we next applied a similar design to a new and independent sample and dependent measures in Study 2.

Study 2: Method

Sample 2 consisted of 217 nonclinical participants. Only participants who completed both Time 1 (T1; baseline) and Time 2 (T2; stressor) time points (N=129) were included in the analysis (mean age 18.96 years SD .98; 53.5% female; 46.5% male, and 3.7 % did not wish to disclose; 77.5% Caucasian, 3.1% African-American, 10.1% Hispanic/Latino, 2.3% Asian, 3.1% self-identified as belonging to more than 1 race, 3.9% Other/Did not wish to disclose). All participants were students at a large university in the southeastern United States. Participants in Study 2 completed a protocol that was nearly identical to study 1. Specifically, participants completed a series of self-report measures at two measurement points (baseline, and stressor), separated by approximately three weeks. As was the case in study 1, baseline was collected at the beginning of a college semester. The analytic approach was nearly identical to Study 1, with the exception that gender was associated with both the predictor and outcome and was therefore controlled in the regression

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3 analyses. Because the pattern of results was unchanged by inclusion of gender as a covariate, we
4 report effects without gender, which permitted estimation of κ^2 effect sizes in PROCESS. In
5 contrast, in our exploratory analysis we were interested in potential unique effects of AC on
6 debilitating versus facilitative forms of stress, which required inclusion of the non-focal form of
7 stress as a covariate in the regression models, precluding κ^2 estimation in PROCESS.
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17 *Attentional Control Scale (ACS).* We administered the ACS at baseline. In sample 2, the
18 ACS total scale score demonstrated adequate internal consistency in our data ($\alpha = .84$)
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21 *Spielberger State-Trait Anxiety Inventory (STAI) – Trait version.* We administered the STAI
22 to the sample at baseline (total scale $\alpha = .90$).
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25 26 27 *Measurement Point 2: Stressor*

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29 *Test Anxiety Scale (TAS).* The TAS consists of 37-items rated as True/False (Total scale
30 $\alpha = .84$). The TAS was developed as a measure of excessive worry, apprehension and tension in
31 response to test situations (Spielberger et al., 1976; Sarason, 1978; Spielberger & Vagg, 1995). The
32 TAS has established psychometric properties in undergraduate samples (Richardson et al., 1977) and
33 cross-cultural samples (Raju, Mesfin & Alia, 2010).
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37 *Achievement Anxiety Test (AAT).* The AAT is a 19-item questionnaire consisting of two scales,
38 indexing the degree to which test anxiety facilitates or debilitates performance. This measure has a
39 high test-retest reliability ($r = .75$) and the two sub-scales are negatively correlated (r range = $-.37$ to $-.48$;
40 Albert & Haber, 1960). Internal consistency for this scale was in the acceptable (total scale $\alpha = .85$,
41 facilitating $\alpha = .64$, debilitating $\alpha = .89$).
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44 45 46 47 48 49 50 51 *Overview of Analytic Approach*

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54 Similar to Study 1, moderational and mediational analyses were carried out using the
55 PROCESS macro for SPSS (Hayes, 2012). ACS was again evaluated as a potential intermediary
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variable, influencing the relationship between trait anxiety at T1 and reactivity to the stressor at T2. Separate analyses were conducted for each dependent variable of interest (TAS; AAT total scale score, AAT debilitating anxiety subscale, and AAT facilitating anxiety subscale).

Study 2: Results

Descriptive Data and Zero-Order Relations among Theoretically-Relevant Variables

Means, standard deviations and zero-order correlations for all variables are presented in Table 3. In contrast to Study 1, age and gender were not interrelated, but gender did correlate with other study variables, indicating males reported higher ACS scores ($r = .30, p < .01 = .003$), and lower TAS ($r = -.23, p < .01 = .005$), AAT facilitating ($r = -.23, p < .01 = .005$), AAT debilitating ($r = -.34, p < .01 = .002$), and AAT Total ($r = -.34, p < .01 = .002$) scores. Consistent with the conceptual model, the ACS was significantly (negatively) correlated with anxiety-relevant measures such as STAI ($r = -.34, p < .01 = .002$), TAS ($r = -.29, p < .01 = .004$), AAT facilitating ($r = -.33, p < .01 = .002$), AAT debilitating ($r = -.47, p < .01 = .0003$), and AAT total ($r = -.48, p < .01 = .0001$).

Moderational Analysis

Primary Model: Prediction of Test Anxiety (TAS). In Study 2, we tested (using PROCESS model 1; Hayes, 2012) the hypothesis that ACS (M) moderates the relationship between Time 1 STAI (X) and TAS (Y) just prior to taking a college exam. Controlling for gender, higher levels of Time 1 STAI predicted greater Time 2 TAS ($b = .205, p = .001$), whereas the effect of Time 1 ACS on TAS fell below statistical significance ($b = -.139, p = .093$). We observed no interaction between trait anxiety and attentional control on Time 2 TAS total scale score ($b = .003, p = .716$).

Comparison Model: Prediction of Achievement Anxiety (AAT and Subscales). Next, the dependent variable was changed to AAT total scale score to determine whether this pattern generalized to another index of test anxiety. There was a significant relationship between Time 1 STAI

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3 on Time 2 AAT total ($b = .174, p = .02$), and a significant inverse effect of Time 1 ACS on Time 2
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5 AAT total ($b = -.512, p < .001 = .0009$). As was the case for TAS, there was no interaction between
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7 Time 1 STAI and ACS in predicting Time 2 AAT total scale score ($b = .009, p = .407$). Next,
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9 predictions for AAT subscale scores were tested. To test the hypothesis that the relationship between
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11 Time 1 STAI and Time 2 AAT debilitating (versus facilitating) anxiety would be moderated by ACS,
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13 we repeated this analysis controlling for gender and Time 2 AAT facilitating anxiety. The effect of
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15 Time 1 STAI on Time 2 AAT debilitating anxiety was marginal albeit nonsignificant ($b = .100, p$
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17 $= .079$). However, Time 1 ACS directly predicted Time 2 AAT debilitating anxiety ($b = -.257, p =$
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19 $.001$). Again, there was no interaction between Time 1 STAI and ACS ($b = .004, p = .550$). To test for
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21 unique effects on facilitating anxiety, the analysis was repeated with Time 2 AAT facilitating anxiety
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23 scores as the dependent variable, controlling for gender and Time 2 AAT debilitating anxiety. There
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25 was no unique effect of Time 1 STAI ($b = .014, p = .739$) or Time 1 ACS on Time 2 AAT facilitating
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27 anxiety ($b = -.074, p = .219$), and again the interaction between Time 1 STAI and ACS was not
28
29 significant ($b = .002, p = .755$).

36 *Mediational Analysis*

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39 *Primary Model: Prediction of Test Anxiety (TAS).* Using Hayes' (2012) PROCESS model 4,
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41 a test of simple mediation was used to evaluate ACS as potential mediator of the relationship
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43 between Time 1 STAI (X) and Time 2 TAS (Y). Results are detailed in Figure 3. In short, Time 1
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45 STAI predicted Time 2 TAS and this relationship was partially mediated by Time 1 ACS.

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48 *Comparison Model: Prediction of AAT Total, Debilitating and Facilitating Anxiety Subscales.*
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50 To further validate the primary model and parallel the moderational analyses outlined above, we next
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52 substituted in the AAT (total and subscale) scores separately, in a series of comparison models. As
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54 illustrated in detail in Figure 3, Time 1 STAI predicted Time 2 AAT total, and this relationship was
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56 partially mediated by Time 1 ACS. To investigate the prediction that Time 2 AAT debilitating
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3 achievement anxiety (subscale 1) would uniquely be predicted by Time 1 STAI (and mediated by
4 ACS), analyses were repeated first with Time 2 AAT debilitating anxiety scores as the dependent
5 variable, controlling for gender and Time 2 AAT facilitating anxiety. As shown in Figure 3, Time 1
6 STAI predicted Time 2 AAT debilitating achievement anxiety, and this relationship was mediated by
7 Time 1 ACS. To test for unique effects on facilitating anxiety, the analysis was repeated with Time 2
8 AAT facilitating anxiety (subscale 2) scores as the dependent variable, controlling for gender and
9 Time 2 AAT debilitating anxiety. As illustrated in Figure 3, there was no total effect of Time 1 STAI
10 on Time 2 AAT facilitating anxiety. Time 1 STAI was inversely associated with Time 1 ACS.
11 However attentional control did not predict Time 2 AAT facilitation anxiety scores or function as a
12 mediator between STAI and AAT facilitation anxiety.
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27 Study 2: Discussion

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29 Results from Study 2 were highly consistent with observations reported in Study 1,
30 ultimately supporting a mediational rather than moderational role for attentional control in the
31 relationship between baseline indices of trait-like anxiety and the actual experience of distress in
32 response to the stressor. Results further indicated a medium effect size for the ACS mediated indirect
33 effect of STAI on AAT. Analysis of subscales on the AAT provided further nuance to the STAI,
34 ACS, and AAT relations, indicating that debilitating, rather than facilitating facets of anxiety were
35 predictable on the basis of a mediational model that includes ACS. Collectively, these results
36 replicate the structural effects observed in Study 1, and extend this finding by providing further
37 descriptive detail about the discriminant properties of this relationship.
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51 General Discussion

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53 The main objectives of this study were to (1) establish the properties of the longitudinal
54 relationship between AC and state-like forms of anxiety, (2) determine the extent to which results
55 from lab-based studies apply equally to ecologically valid, albeit less standardized stressors, (3)
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3 examine competing hypotheses regarding the mediational versus moderational relationship between
4 AC and acute stress, and to explore specificity of effects as they relate to facilitative versus
5 debilitating forms of anxiety. In terms of our first objective, overall results across both samples were
6 consistent with the initial prediction that attentional control would predict subjective anxiety in
7 response to the exam, thus supporting the existence of a measurable relationship between baseline
8 AC and state-like anxiety across at least three weeks.
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17 For objective two, our results supported the notion that indices of AC can be used to predict
18 responses to everyday stressors, which is an incremental extension of lab-based work that has
19 demonstrated primarily that AC-anxiety relationships occur in the context of exquisitely controlled
20 experimental setups. In terms of objective three, we found less support for a putative moderational
21 role for ACS, and comparatively greater support for an alternative model, in which ACS mediated
22 the relationship between trait-anxiety and fearful responding to the test stressor was supported in
23 both samples. Thus, a central finding in our study was support for a mediational rather than
24 moderational relationship between pre-existing risk factors for distress and the actual experience of
25 distress in response to a stressor.
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39 Overall, we found a medium effect size for the mediational role of ACS between STAI and
40 state-related anxiety (WES-R), which suggests a modest role for attentional control in modulating
41 the relationship between trait- and state-anxiety. Exploratory results further demonstrated that this
42 relationship may be specific to debilitating aspects of anxiety, which may account for the smaller
43 effect size seen with general measures of test anxiety (TAS) relative to measures that differentiate
44 debilitating from facilitating forms of anxiety (ATT). In clinical contexts, individuals with high trait
45 anxiety and low attentional control, a potential target for treatment could focus on improvements in
46 attentional control rather than decreasing baseline anxiety levels.
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58 The pattern of findings reported here runs somewhat counter to existing research on the
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3 impact of attentional control on the experience of emotion (Richey, Keough, & Schmidt, 2012; Susa,
4 Pitica, Benga, & Miclea, 2012; Vasey, Harbaugh, Mikolich, Firestone, & Bijttebier, 2013). However
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6 there are several factors in our study that may have contributed to this result. First, the anticipatory
7
8 nature of the distress that was indexed in our study may in part be related to the unexpected
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10 difference in the structural nature of the observed relationship. For instance, study participants were
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12 aware of their exam well in advance, and presumably had experience with previous testing situations
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14 either in other undergraduate courses or secondary education, and were therefore familiar with both
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16 the timing and nature of the stressor itself. The increase in familiarity with the exam stressor may
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18 have had the effect of eliciting anticipatory cognitions, which would be relatively less common, or
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20 perhaps absent in stressors that were novel in nature. In addition, we note that our primary dependent
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22 measures of distress (WES-R, TAS, AAT) were all administered 5 minutes *prior to* the exam
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24 stressor. These factors may have conspired to create a scenario in which anticipatory, versus reactive
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26 aspects of distress were indexed, thus revealing a “filtering” effect for attentional control on
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28 anticipatory distress, rather than a direct interactive effect on the experience of distress itself. This
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30 may also explain the discrepancy between the current results and our previous study using a lab-
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32 based stressor. Previously studies have used highly standardized lab-based stressors such as a CO₂
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34 challenge (Richey et al., 2012) in which subjects were not aware of the phenomenological quality of
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36 the novel stressor, and distress was measured immediately *after* the stressor. ~~As such, measuring
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38 distress after a novel stressor may have exposed a relationship that indexed more automatic aspects
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40 of processing, rather than anticipatory cognition, which may be more voluntary or effortful in nature.~~

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50 For our exploratory analysis, findings from subscale analyses support the notion of
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52 specificity for AC in predicting debilitating but not facilitative forms of anxiety. Although this
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54 analysis was largely preliminary in nature, this finding adds a nuance to the AC-anxiety relation that
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56 is worthy of further study. Specifically, our results indicated that baseline scores for AC were
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3 predictive of anxiety that was reported to interfere with rather than improve performance. This
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5 suggests that certain features of attentional control theory could be evaluated in terms of the Yerkes-
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7 Dodson “inverted-U” function to synergistically predict how the cognitive control of emotion might
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9 improve or worsen as stimulation increases or decreases, respectively. For example, there may exist
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11 an optimal level of arousal for the purposes of deploying coping strategies, with poorer results at
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13 lower levels of arousal due to poor concentration or boredom, and interference at higher levels of
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15 arousal due to fatigue or exhaustion. Although our results indicate only that debilitating anxiety
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17 scales monotonically with the joint effect of AC and trait-anxiety (and were not meant to provide a
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19 full test of a quadratic relationship), future research should evaluate quadratic relationships in order
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21 to more fully understand whether this conforms to the traditional U-shaped function.
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27 Results from the current study also have the potential to elaborate on information processing
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29 models of anxiety. In the past three decades, findings from both cognitive and cognitive
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31 neuroscience literatures have largely supported the distinction between automatic and controlled
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33 processing of sensory, cognitive and affective information (Ouimet, Gawronski, & Dozois, 2009;
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35 Posner, 2013; Schneider & Chein, 2003). A wide variety of methodologies including fMRI (Fan,
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37 McCandliss, Fossella, Flombaum, & Posner, 2005), PET (Nobre et al., 1997), and ERP (Hajcak,
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39 MacNamara, & Olvet, 2010) as well as neuropsychological investigations of patients have been
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41 used, with results consistently supporting a serial “dual-process” approach (Birnboim, 2003),
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43 wherein automatic processes occur first in the stream and are primarily responsible for stimulus
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45 detection, and controlled processes occur later and are responsible for interpretation and elaboration.
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47 Within anxiety research, a significant body of work has been premised upon the dual process model,
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49 mainly by investigating individual differences in patterns of attention. Within the automatic
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51 processing domain, there is extensive evidence that anxiety pathology is associated with a low
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53 latency attentional bias toward threat relevant stimuli (Cisler & Koster, 2010; Teachman, Joormann,
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3 Steinman, & Gotlib, 2012). However, despite convincing evidence for associations between anxiety
4 and biased sensory input at the automatic level, it remains unclear whether patterns of selective input
5 also exist at the controlled level. The current study adds to this discussion by including a stressor that
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arguably probes biases at a controlled level of processing. Indeed, the ability to appropriately disengage from the stress of an imminent and prolonged stressor might be best conceptualized as being a function of willful termination of attentional focus, as opposed to automatic aspects of attention. However, additional work will be required in order to more fully understand the role of AC in elaborative or voluntary forms of effortful processing biases.

Although several noteworthy findings were reported in the current study, conclusions must be evaluated in light of several study limitations. In particular, one special consideration that warrants additional discussion is the noted difficulty in detecting the moderated effect in field studies such as ours. The details of this problem were outlined by McClelland and Judd (1993), who demonstrated the variance structures encountered in unselected or naturalistic samples may be suboptimal for detecting interactions because a relatively greater proportion of cases reside in the middle of the bivariate distribution, and a relatively smaller number of cases in the corners of the distribution, the latter of which are vital to testing the interaction. Although the results of our power analyses indicated that both Study 1 and Study 2 were powered to at least detect a medium effect size, as defined by f^2 of >0.15 (Cohen, 1977, 1988), the unique variation in the interaction term w that is not shared with predictors x and z (i.e. residual variance of w) is of special consideration due to the constriction in the ranges of variances observed in the bivariate normal distribution of correlated predictors. This has the net result of decreasing power to detect the moderated (but not mediated) effect, which could explain the pattern of results observed in our studies. Although no specific operational guidelines for power and sample size calculations were presented in McClelland and Judd (1993), we note that the results of subsequent Monte Carlo simulations presented in Shieh

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3 (2009) indicate that moderated multiple regression for moderately correlated predictors such as ours
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5 ($r \sim -0.5$) assuming a desired power of 0.9, require a minimum sample to detect the moderated effect
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7 of $N=137$ under an assumption of bivariate normal distribution of predictors and $N=119$ for bivariate
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9 gamma distribution (c.f. Shieh, 2009, tables 2 and 3). Thus, according to these simulations our
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11 studies were sufficiently powered for at least $1-\beta=0.8$, but nevertheless the intrinsic differences in
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13 statistical power in tests of mediation and moderation must be explicitly considered when
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15 interpreting our results, particularly since perfect agreement has not yet been reached in terms of
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17 power calculation for moderated multiple regression. However, as a reasonable next step, we
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19 highlight the solution outlined in McLelland and Judd (1993), in the form of a “4 quadrant” design,
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21 which oversamples cases in the corners of the distribution, in order to increase the residual variance
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23 of the interaction term and thus increasing the probability that at least a subsample is close to an
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25 optimal design.
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32 Although previous studies have found support for a meditational role for AC in the
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34 relationship between pre-existing risk factors and adverse outcomes (Sportel, Nauta, de Hullu, de
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36 Jong, & Hartman, 2011; Yap et al., 2011), a second limitation of our study is that the mediator (or
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38 moderator) was measured at the same time point as the predictor variable. This design constraint
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40 precludes establishing temporal precedence of either trait-anxiety or AC in our model. However,
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42 existing theory and supportive data have suggested that cognitive processes such as AC may act as a
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44 modifier of the association between temperamental risk-factors and adverse outcomes (Oldehinkel,
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46 Hartman, Ferdinand, Verhulst, & Ormel, 2007; van Oort, Greaves-Lord, Ormel, Verhulst, &
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48 Huizink, 2011; Verstraeten, Vasey, Raes, & Bijttebier, 2009). A third limitation of this study
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50 pertains to its reliance on self-report indices of attentional control. While at least some data suggest
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52 that perceptions of control are correlated with actual control (Judah, Grant, Lechner, & Mills, 2013),
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54 future research should incorporate more proximal measure of AC into adult samples, in order to
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3 compare the performance of self-report to behavioral indices. The present study also would have
4 benefited from a measure of distress that was administered immediately during or after the stressor,
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6 to offer a comparative measure of reactive anxiety. This could have provided insight into the
7
8 differential structural results between trait anxiety, AC, and reactive anxiety reported across past
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10 studies (Richey et al., 2012). Like Eysenck et al. (2007) and others, we focused on anxiety in
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12 (normal) college students and did not screen for mental disorder, thus additional research is required
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14 to determine whether our results generalize to clinical populations. These limitations
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16 notwithstanding, current findings advance theory in the attention-anxiety interface by documenting
17
18 crucial relationships in the AC model and by providing a basis for predictions within effortful
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20 information processing approaches. It is hoped that the current study will stimulate additional work
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22 into higher-latency aspects of the dual process model and therefore provide a more complete
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24 depiction of attentional mechanisms and their role in anxiety maintenance and development.
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Table 1. Descriptive Statistics and Zero-order Correlations among Sample 1 Study Variables

	M	SD	1	2	3	4	5
1. Age	18.42	.82	—				
2. Gender	.39	.54	.17*	—			
3. T1 ACS	53.19	8.85	-.02	.09	—		
4. T1 STAI	37.93	9.38	-.04	-.07	-.49**	—	
5. T2 WES-R	114.29	33.88	.04	-.10	-.45**	.56**	—

Note. Gender was coded female = 0 = female, male. = 1. T1 ACS = Time 1 Attention Control, T1 STAI = Time 1 Trait Anxiety, T2 WES-R = Time 2, * $p < .05$, ** $p < .01$

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Table 2. *Descriptive Statistics and Zero-order Correlations among Sample 2 Study Variables*

	M	SD	1	2	3	4	5	6	7	8
1. Age	18.96	.82	—							
2. Gender	.47	.50	.13	—						
3. T1 ACS	55.9	6.89	.16	.30**	—					
4. T1 STAI	37.24	9.29	.02	-.24	-.31**	—				
5. T2 TAS	15.22	6.47	-.07	-.23**	-.28**	.39**	—			
6. T2 AAT Facil	29.60	4.56	-.07	-.23**	-.33**	.19*	.41**	—		
7. T2 AAT Debil	25.86	6.93	-.05	-.34**	-.44*	.36**	.71**	.46**	—	
8. T2 AAT Total	55.46	9.88	-.07	-.34**	-.46**	.34**	.69**	.78**	.91**	—

Note. Gender was coded female = 0 = female, male. = 1. T1 ACS = Time 1 Attention Control Scale; T1 STAI = Time 1 State-Trait Anxiety Inventory; T2 TAS = Time 2 Test Anxiety Scale (Total Scale Score); T2 AAT Facil = Time 2 Achievement Anxiety Scale - Facilitation Subscale; T2 AAT Debil = Time 2 Achievement Anxiety Scale - Debilitation subscale; T2 AAT total = Time 2 Achievement Anxiety Total, * $p < .05$, ** $p < .01$.

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Figure Captions

Figure 1

Joint distribution, standard deviation and density histograms of STAI and ACS scores in Studies 1 (Upper Panel; A) and 2 (Lower Panel; B).

Figure 2

Study 1: Attentional control as a potential mediator of the prediction by trait anxiety of WES-R.

Note. T1 ACS = Time 1 Attentional Control Scale; T1 STAI = Time 1 Trait Anxiety; T2 WES-R = Time 2 Worry & Emotionality Scale, Revised.

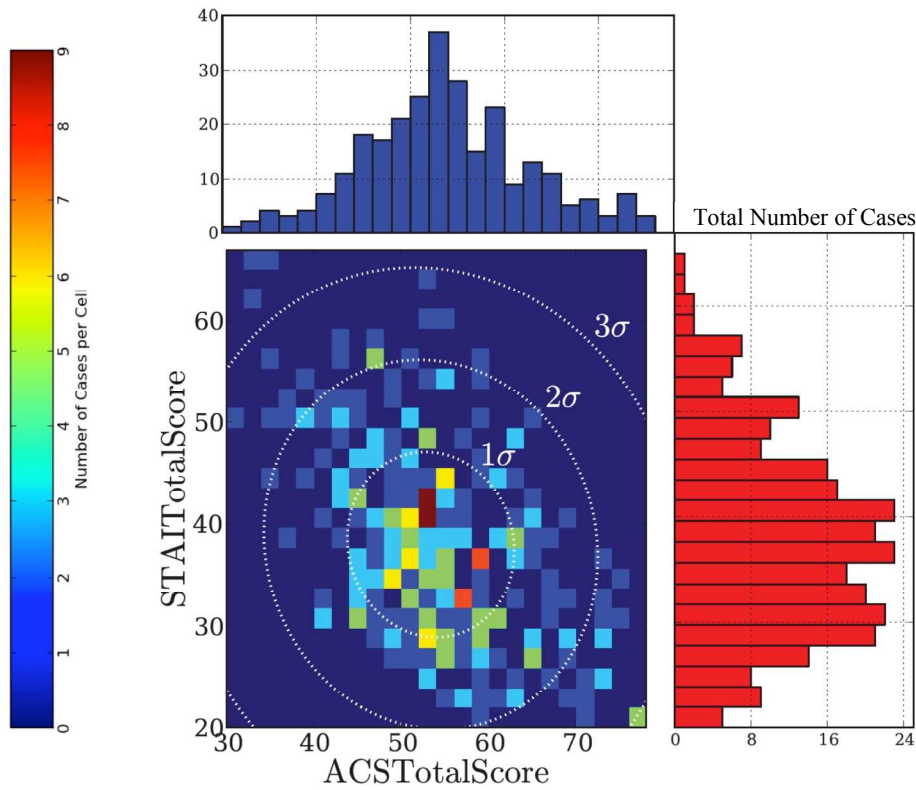
Figure 3

Study 2: Attentional control as a potential mediator of the prediction by trait anxiety of test anxiety and achievement anxiety.

Note. T1 ACS = Time 1 Attentional Control, T1 STAI = Time 1 Trait Anxiety, T2 TAS = Time 2 Test Anxiety, T2 ATT facil = Time 2 Achievement Anxiety Facilitation, T2 ATT debil = Time 2 Achievement Anxiety Debilitation, T2 ATT total = Time 2 Achievement Anxiety Total. All Study 2 analyses included gender as a covariate to control for potential confounding effects. To test for unique achievement anxiety facilitation and debilitation effects, analyses for HA facil also included HA debil as covariate, and vice versa. * $p < .05$, ** $p < .01$.

Figure 1

A)



B)

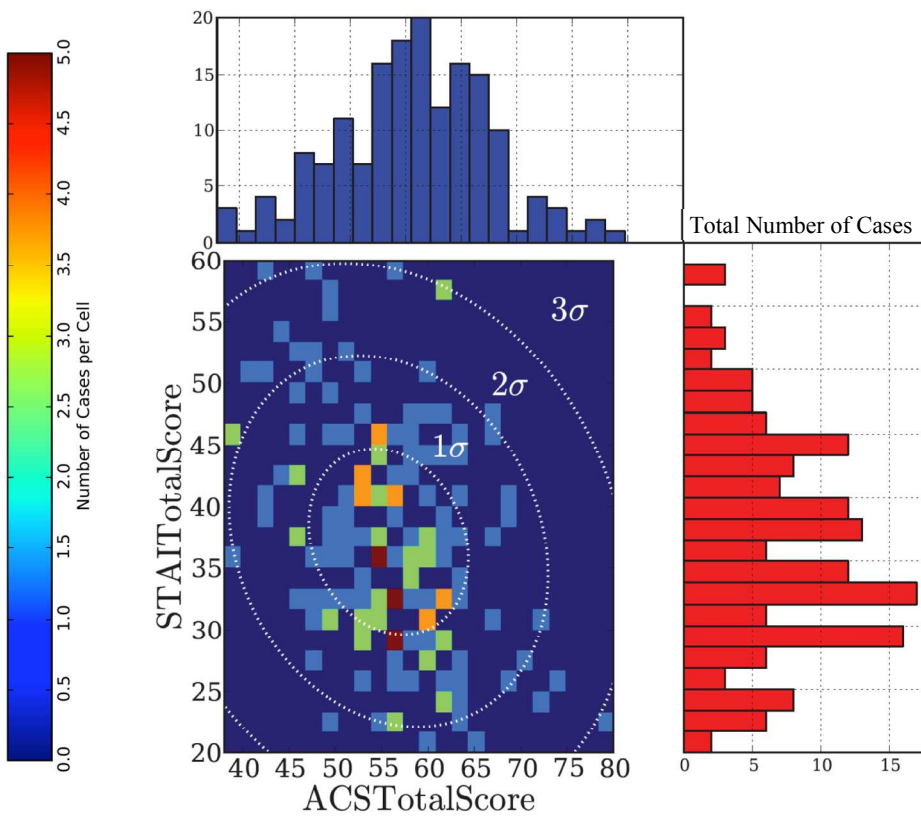


Figure 2

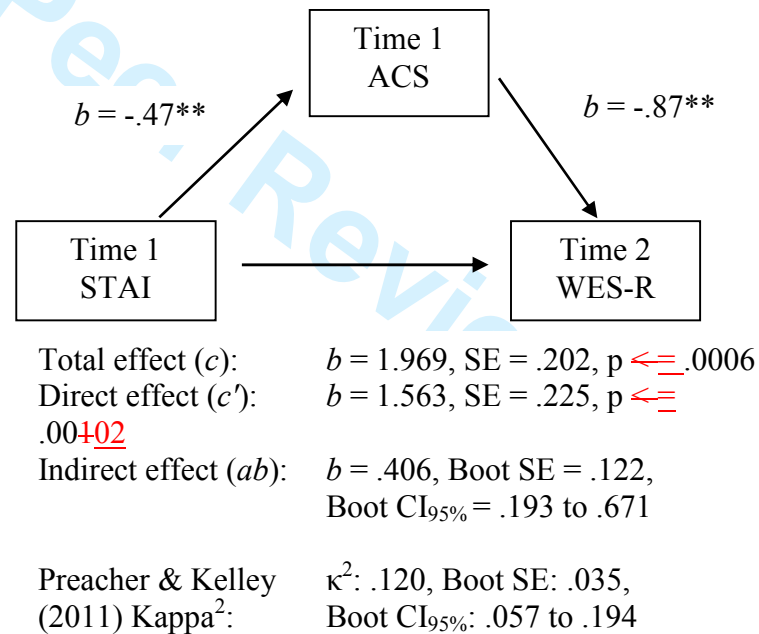
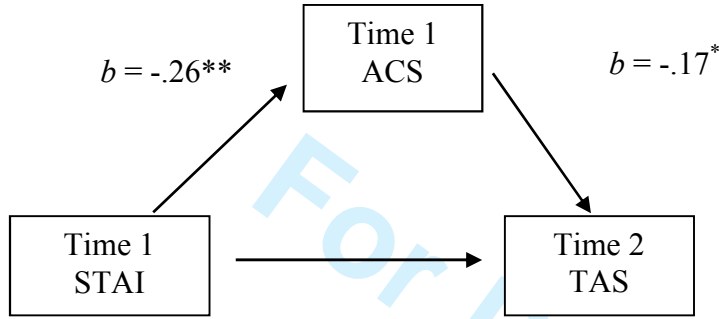
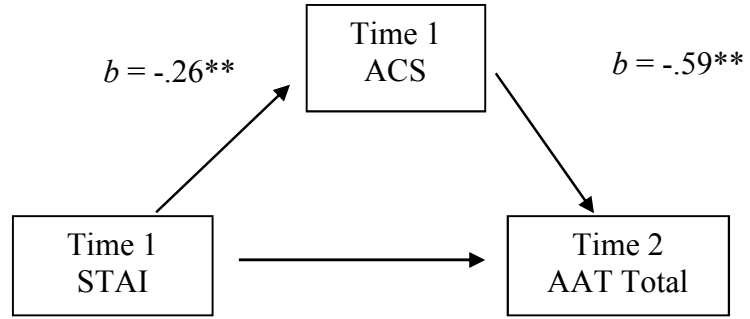


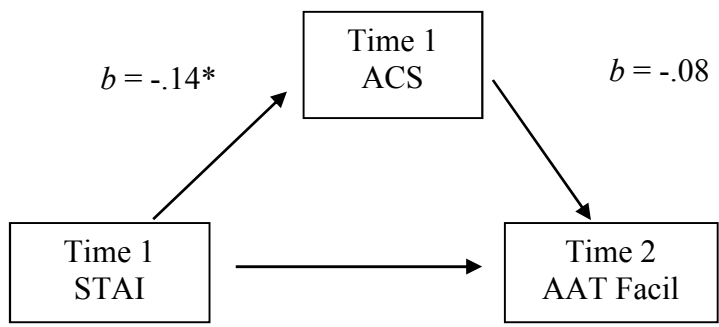
Figure 3



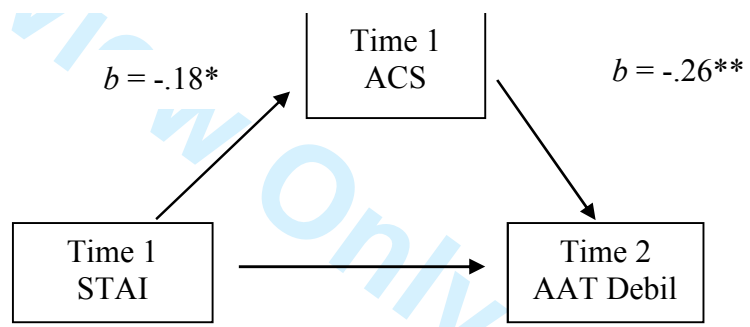
Total effect (c): $b = .259, SE = .058, p \leq .001$
 Direct effect (c'): $b = .216, SE = .061, p = .0005$
 Indirect effect (ab): $b = .043, Boot SE = .023, Boot CI_{95\%} = .006 \text{ to } .096$
 Effect Size^a: $Kappa^2: .062, SE = .030, Boot CI_{95\%} = .010 \text{ to } .128$



Total effect (c): $b = .351, SE = .090, p \leq .001$
 Direct effect (c'): $b = .201, SE = .088, p = .0242$
 Indirect effect (ab): $b = .150, Boot SE = .053, Boot CI_{95\%} = .066 \text{ to } .277$
 Effect Size^a: $Kappa^2: .140, SE = .042, Boot CI_{95\%} = .065 \text{ to } .230$



Total effect (c): $b = .024, SE = .042, p = .570$
 Direct effect (c'): $b = .0132, SE = .042, p = .756$
 Indirect effect (ab): $b = .011, Boot SE = .011, Boot CI_{95\%} = -.002 \text{ to } .044$



Total effect (c): $b = .145, SE = .057, p = .012$
 Direct effect (c'): $b = .098, SE = .056, p = .085$
 Indirect effect (ab): $b = .047, Boot SE = .022, Boot CI_{95\%} = .014 \text{ to } .102$