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Examining experiential avoidance as a mediator of the relation between anxiety sensitivity and depressive symptoms

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Examining experiential avoidance as a mediator of the relation between anxiety sensitivity and depressive symptoms

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Abstract

Examining experiential avoidance as a mediator of the relation between anxiety sensitivity and depressive symptoms

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Initial evidence suggests that experiential avoidance (EA) mediates the relation between anxiety sensitivity (AS) and depression. We examined the AS-EA-depression pathway, examining both concurrent, and prospective (cross-lag), mediation models. Utilizing data from a study that examined the effects of exercise on AS (N = 60), we modeled depressive symptoms, EA, and AS over four time points. Time-varying predictors were disaggregated into between- subjects (each person's mean level of the predictor) and within- subjects change (each person's deviations, at each time point, from their mean level on the predictor) components. Tests of the concurrent relations were partially consistent with predictions, with mean EA levels, but not within-subjects changes in EA, partially mediating the relation between AS and depression symptom severity. However, the prospective, cross-lag mediation model, in which AS predicted future EA controlling for previous EA, and EA predicted future depression, controlling for previous depression, yielded no significant effects. These results suggest that observed between-subjects mediation findings, found here and in previous studies, may not replicate using more

stringent, quasi-causal, cross-lag mediation analyses. These results highlight the importance of estimating causal pathways in mediation analyses. Clinical implications and directions for future research are discussed.

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Chapter 1: Introduction[1]

Anxiety sensitivity (AS) is a tendency to fear bodily sensations associated with anxious arousal due to the belief that these will cause physical, social, or cognitive harm (Reiss, 1991). Although initially conceptualized as relating specifically to panic disorder (Cox, Borger, & Enns, 1999), AS has emerged as a transdiagnostic risk factor for a range of psychopathology (Otto et al., 2016; Schmidt, Zvolensky, & Maner, 2006). In addition to panic disorder, elevated AS confers vulnerability to the development of other anxiety disorders (Cox et al., 1999; Maller & Reiss, 1992), substance use disorders (Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006; Leventhal & Zvolensky, 2015) and borderline personality disorder (Gratz, Tull, & Gunderson, 2008; Lilienfeld & Penna, 2001). There has also been growing evidence suggesting that AS, particularly the cognitive concerns dimension, is related to depression (Allan, Capron, Raines, & Schmidt, 2014; Naragon-Gainey, 2010). Furthermore, reductions in AS have been associated with reduced symptoms of panic (Hazen, Walker, & Eldridge, 1996; Schmidt, Raines, Allan, & Zvolensky, 2016) as well as symptoms of depression, anxiety, and suicidal ideation (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Schmidt, Capron, Raines, & Allan, 2014; Schmidt, Norr, Allan, Raines, & Capron, 2017). Reductions in anxiety sensitivity have also been associated with positive treatment outcomes for health behaviors, such as smoking, problematic alcohol use, and insomnia (for review, see Otto

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et al., 2016). These findings suggest that, although AS has been conceptualized as a dispositional variable (Reiss & Havercamp, 1996), it is malleable with treatments such as interoceptive exposure, cognitive restructuring, and aerobic exercise (for review, see Smits, Berry, Tart, & Powers, 2008). Hence, AS is an important target for interventions that aim to prevent or ameliorate psychological disorders (Smits, Otto, Powers, & Baird, 2018).

Otto and colleagues (Otto et al., 2016; Otto, Smits, Fitzgerald, Powers, & Baird, 2018; Otto & Smits, 2018) have discussed the mechanism by which AS confers risk for psychological and related disorders. Specifically, they describe AS an "amplification" factor, such that elevated levels of AS potentiate the aversiveness of negative affective or somatic states, which promotes the use of maladaptive emotional regulation strategies in an effort to prevent, avoid, or escape from such experiences. In the context of panic disorder, for example, anxiety sensitivity signals threat following changes in somatic arousal that occur because of stress or physical activity, which results in fear and promotes actions that help the person manage these false alarms (e.g., rescue medications, escape). In the context of depression, AS may also amplify emotions (e.g., sadness, guilt, worthlessness, or hopelessness). The motivation to escape from these intense internal experiences (i.e., experiential avoidance), is thus likely to increase in persons with elevated AS, which in turn can reinforce the cycle (i.e., depressed state – withdrawal/avoidance – loss of reward – depressed state) that is characteristic of depression (Zvolensky & Forsyth, 2002).

There is some evidence supporting the hypothesis that experiential avoidance (EA) mediates the AS-depression relation. First, reflecting evidence for the effect of the independent variable (AS) on the mediator variable (EA), several studies have documented the concurrent relation between AS and EA at a single time point (e.g., Kämpfe et al., 2012; Kashdan, Barrios, Forsyth, & Steger, 2006; Zvolensky et al., 2015). Second, reflecting evidence for the effect of the mediator (EA) on the dependent variable (depression), EA has been shown to be related to concurrent depression both cross-sectionally (e.g., Leahy, 2002; Tull & Gratz, 2008) and longitudinally (Bohlmeijer, Fledderus, Rokx, & Pieterse, 2011; Spinhoven, Drost, de Rooij, van Hemert, & Penninx, 2014). For example, decreases in experiential avoidance have been shown to mediate the effect of an Acceptance and Commitment Therapy (ACT) on subsequent depressive symptoms in a community sample of people with depressive symptoms (Bohlmeijer et al., 2011). Similarly, EA has been shown to prospectively predict future depression among individuals with current emotional disorders or with prior histories of emotional disorders (Spinhoven et al., 2014), although this finding was not replicated in a more recent prospective longitudinal study (Schut & Boelen, 2017). Third, Tull and Gratz (2008) showed cross-sectionally that EA and difficulties engaging in goal-directed behavior mediated the relationship between aspects of AS and depressive symptoms. Zvolensky and colleagues (2015) replicated this finding and extended it by demonstrating a reciprocal relation between EA and AS with respect to depressive symptoms (i.e., AS mediated the relation between EA and depressive symptoms, and depressive symptoms mediated the relation between EA and AS). Unfortunately, neither study (i.e., Tull &

Gratz, 2008; Zvolensky et al., 2015) could provide evidence for causality as all measures were assessed at just one time point.

Aiming to build upon the aforementioned research, this study sought to examine the AS-EA-depression pathway longitudinally and prospectively, using a cross-lag mediation model. Such models can provide quasi-causal estimates of the mediating pathways (Hamaker, Kuiper, & Grasman, 2015). Utilizing data from a study that examined the effects of exercise on AS (Smits, Berry, Rosenfield, et al., 2008), we modeled the repeated measures of depressive symptoms, EA and AS to test the hypothesis that the relation between reductions in AS and depression symptoms over time would be mediated by reductions in EA.

Chapter 2: Method[2]

The present study represents a secondary analysis of published data from a study that evaluated the efficacy of exercise for reducing AS in adults with clinically elevated AS (Smits, Berry, Rosenfield, et al., 2008).

PARTICIPANTS

Participants were (N=60; 45 Female; M_{Age} = 20.68, SD=5.80) individuals with elevated AS (ASI score > 25; Peterson & Reiss, 1993). Participants were introductory psychology students recruited from a classroom screening survey administered in introductory psychology courses (n=50) at a university in the Southwest United States of America (USA). The ASI was administered to students in class and potentially eligible individuals were invited to participate in an additional phone screen with additional screening measures including the Physical Activity Readiness Questionnaire (PAR-Q; Thomas, Reading, & Shephard, 1992) and assessment of exclusion criteria. Community volunteers (n=10) were recruited from advertisements near a large university in the Northeast USA. Participants who responded to these ads were also invited to participate in a phone screen, which included the ASI and assessment of readiness for physical activity and of inclusion/exclusion criteria. Eligible participants based on the phone screens were then invited into the lab to participate.

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PROCEDURES

Procedures for this study have been detailed elsewhere (Medina et al., 2014; Smits, Berry, Rosenfield, et al., 2008). Briefly, participants were randomized to one of the three conditions: prescriptive exercise alone, exercise plus cognitive restructuring, or a wait-list control. The active interventions consisted of multiple brief sessions over a 2-week period. The wait-list control condition consisted of completing assessments at the same time intervals as the other conditions. All measures were administered pre-treatment, mid-treatment (after week 1), post-treatment (after week 2), and at three-week follow up (after week 5).

MEASURES

Anxiety sensitivity. The 16-item Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992) is a self-report questionnaire assessing AS. Using a Likert-type scale, (0=very little to 4=very much), participants are asked to rate the degree to which they find bodily sensations associated with anxious arousal distressing. The ASI has strong psychometric properties, with high internal consistency (alpha = 0.80-0.90) (Peterson & Reiss, 1993; Taylor, Koch, & McNally, 1992; Telch, Shermis, & Lucas, 1989), test-retest reliability (Maller & Reiss, 1992; Peterson & Reiss, 1993), and construct validity (McNally & Lorenz, 1987; Taylor, Koch, & Crockett, 1991). In the current sample, internal consistency was mostly good across time points ($\alpha_{Pre} = 0.61$; $\alpha_{Mid} = 0.87$ $\alpha_{Post} = 0.89$; $\alpha_{FU} = 0.92$).

Experiential avoidance. The 9-item Acceptance and Action Questionnaire-short form (AAQ; Hayes, Strosahl, Wilson, & Bissett et al., 2004) was initially designed to measure changes in EA and psychological flexibility during Acceptance and Commitment Therapy (ACT). The self-report questionnaire assesses (1) unwillingness to experience distress and (2) the extent to which unwillingness to experience distress interferes with one's pursuit of values and goals. Higher scores on the measure reflect greater EA tendencies (i.e., lower experiential acceptance and flexibility). Although there is now a revised AAQ (AAQ-II; Bond et al., 2011), the 9-item version of the AAQ has previously been used to examine experiential avoidance (not only psychological flexibility) and has demonstrated satisfactory internal consistency and reliability (Boelen & Reijntjes, 2008). In the current sample, internal consistency was variable across time points ($\alpha_{Pre} = 0.56$; $\alpha_{Mid} = 0.57$ $\alpha_{Post} = 0.70$; $\alpha_{FU} = 0.67$).

Depressive symptoms. The Beck Depression Inventory-II (BDI-II; (Beck, Steer, & Carbin, 1988; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report measure designed to measure the severity of depressive symptoms over the previous week. Higher scores indicate greater symptom severity. The scale has been validated for use in college students (Whisman, Perez, & Ramel, 2000) and in a variety of clinical and nonclinical samples (Beck et al., 1988). In the current sample, internal consistency was good across timepoints ($\alpha_{Pre} = 0.84$; $\alpha_{Mid} = 0.89$ $\alpha_{Post} = 0.89$; $\alpha_{FU} = 0.92$).

ANALYTIC APPROACH

We employed a multilevel modeling (MLM) approach using SPSS® Version 23 to test the hypothesis that EA would mediate the longitudinal relation between AS and depression symptom severity. MLM has several advantages over other methods of analyzing data involving repeated assessments. As an intent-to-treat (ITT) approach, MLM includes all subjects regardless of missing data on the repeated measures; this enhances statistical power and overall generalizability. MLM also allows for modeling the covariance matrix for the errors at repeated assessments, optimizing model accuracy.

We proposed an MLM path model (Figure 1) to test the hypothesis that weekly levels of self-reported EA (indexed via AAQ) would mediate the longitudinal relationship between AS (indexed via ASI) and depression symptoms (indexed via BDI-II) at the subsequent assessment. We examined the effect of both the between-subjects and within-subjects component of each time-varying predictor (TVP) on outcome. This required us to disaggregate the original TVP variables for ASI and AAQ into a mean and deviation variable and enter separate variables for each in predicting outcome (Hedeker & Gibbons, 2006; Hoffman, 2015; Wang & Maxwell, 2015). The "mean" statistic for each TVP is a level 2 predictor (i.e., between-subjects), reflecting the individual's mean on that predictor over all four assessments. The deviation variable is a level 1 variable (i.e., within-subjects), reflecting the individual's deviation from their mean at each assessment. The primary reasons that we chose cross lag panel autoregressive models using disaggregated time varying predictors in MLM are that these models (1) calculate within-person relations between the variables across time and that (2) MLM models have

been shown to make accurate, unbiased estimates of regression coefficient in small samples like ours (Maas & Hox, 2005).

We predicted that changes in our mediator, AAQ, would predict later changes in the outcome, BDI, thus enhancing the causal interpretation of our mediation model (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). However, given the lack of current evidence regarding the expected time frame for the action of the effects to occur between ASI, AAQ, and BDI, we explored both prospective and concurrent mediation. In our prospective analysis, we expected that disaggregated ASI at the previous assessment would predict disaggregated AAQ during the current assessment (i.e., ASI at time "t-1" would predict AAQ at time "t"), and that disaggregated AAQ at week "t" would impact outcome scores on the BDI at the *next* assessment (e.g., BDI at week "t+1"), controlling for outcome scores from the current week (e.g., BDI at week "t"). This longitudinal cross-lagged approach employing the disaggregated TVPs is similar the approach recommended by (Hamaker, Kuiper, & Grasman, 2015) for assessing quasi-causal relations in non-experimental data.

In our concurrent model (Figure 2), we conducted these analyses without the lag. Across all of our analyses, we controlled for the potential effects of time and study condition by including Time (coded 1-4), Condition (coded 0=exercise condition, 1=control condition), and Time x Condition as covariates.

Mediation. We evaluated the significance of AAQ as a disaggregated mediator of the prospective time-varying relation between ASI and BDI using the distribution of products test (Tofighi & MacKinnon, 2011). Mediation is significant if the 95%

confidence interval for the a*b product does not include 0 ("path a" is the effect of the predictor on the proposed mediator, and "path b" is the effect of the mediator on the proposed outcome, controlling for the effect of other independent variables in the model). In our cross-lagged MLM model we calculated significance of the indirect a1*b1 pathway (ASI Deviation$_{t-1}$ to AAQ Deviation to BDI$_{t+1}$), in addition to the indirect a2*b2 pathway (ASI mean to AAQ mean to BDI$_{t+1}$), as depicted in the model in Figure 1. In testing concurrent mediation, we calculated significance of the indirect a1*b1 and a2*b2 pathways depicted in the path model in Figure 2. As a measure of the effect size of each mediated pathway, we calculated the proportion mediated ($P_M = (a*b)/c$; Shrout & Bolger, 2002). P_M represents the proportion of the total effect of the IV on the DV that is mediated by the mediator.

We used the MLM power analysis program PinT 2.12 (Power in Two-Level Models, Snijders & Bosker, 1993) to calculate the power to detect significance for the paths in our mediation models. We used the data in the current sample to calculate the level-1 and level-2 variances and covariances needed by PinT to make these calculations. PinT indicated that we had power=.92 to detect a medium effect size (d=.50) for the "a" and "b" paths in the concurrent mediation analysis (with 4 data points per participant), and power=.86 to detect a medium effect size for the "a" and "b" paths in the cross-lag analysis (with 3 lagged data point per participant).

Chapter 3: Results[3]

SAMPLE CHARACTERISTICS

Descriptive statistics can be found in Tables 1 and 2. The distributions of the variables of interest (ASI, AAQ, BDI) showed acceptable levels of skewness (AAQ = -0.36; ASI = -0.27; BDI = 0.71) and only 1 outlier > 3SD from the mean (no value > 4 SDs from the mean). There was no missing data on the only between subjects variable in the analysis (treatment condition). Missing data for each variable of interest was: 35 out of 240 possible assessments of ASI (14.58%), 35 out of 240 on AAQ (14.58%), and 35 out of 240 on BDI (14.58%). The pattern of missing data was such that if a participant was missing data on a given variable of interest for a given time point (e.g., missing ASI at post-treatment time point 3), he/she was missing data on the remaining variables for that assessment (e.g., missing BDI and AAQ at post-treatment time point 3). Fifteen participants missed at least one assessment survey containing the AAQ, ASI, and BDI[4]. Average ASI at baseline for our sample (inclusion criterion: ASI>25) was 33.48 (*SD*=6.45). Baseline mean AAQ for the sample was 40.72 (*SD*=6.28), consistent with levels of EA found in other studies conducted in patients with anxiety and mood concerns (Berman, Wheaton, McGrath, & Abramowitz, 2010). BDI-II at baseline was 13.30 (*SD*=7.08), which is indicative of minimal-to-mild depressive symptoms. Results from

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[4] Although MLM is robust to missing data, to strengthen the assumption that missing data did not influence our results, we retested our hypothesis filtering out the *n*=15 participants with missing data. Results from MLM of *n*=45 did not differ from those yielded by the entire sample of *N*=60 participants.

the main outcome paper indicated a steeper decline in both ASI (t (57) = -8.29, $p < .001$) and BDI-II (t (57) = -3.94, $p < .001$) in the exercise relative to control condition (Smits, Berry, Rosenfield, et al., 2008). Current analyses revealed that there also was a significantly greater decline in AAQ scores in the exercise relative to control condition (b = -2.22, t (58) = -4.30, $p < .001$).

HYPOTHESIS TESTING

Prospective Model Path Analysis. Results for path "a1" showed that, controlling for Time, Condition, Time x Condition, and AAQ Deviation$_{t-1}$, ASI Deviation$_{t-1}$ did not predict AAQ Deviation$_t$ (b =.110, t(130) = 1.665, p = .098). As expected, AAQ Deviation$_{t-1}$ predicted AAQ Deviation$_t$, b = -.277, t(131) = -3.449, p = .001, indicating that AAQ deviations from their mean in a given week negatively predicted deviations from their mean in the subsequent week (perhaps reflecting regression to the mean). In evaluating path "a2", ASI Mean was related to AAQ Mean, b = 0.361, t(59) = 4.260, $p <$ .001, indicating participants with higher average levels of ASI tended to report higher average levels of EA on the AAQ.

Controlling for Time, Condition, Time x Condition, disaggregated ASI (ASI Deviation$_t$ and ASI Mean), and current levels of BDI$_t$, results for the "b1" and "b2" paths showed that neither AAQ Deviation$_t$ (b = -0.094, t(124) = -.834, p = .406), nor AAQ Mean (b = .111, t(115) = 1.418, p = .159), predicted BDI$_{t+1}$. Current BDI$_t$ predicted subsequent BDI$_{t+1}$, b = 0.699, t(115) = 11.11, $p < .001$. Neither ASI Deviation$_t$ (p = .308), nor ASI Mean (p = .601) (i.e., c' paths) were significant.

Test of Prospective Mediation. The distribution of products test showed that the mediated pathway from deviations in ASI_{t-1} to deviations in AAQ_t to subsequent BDI_{t+1} was not significant, a1*b1 = -0.010 (*SE* = 0.027), 95% CI: [-0.077, 0.037], p>.05. Similarly, the mediated pathway from ASI mean to AAQ mean to BDI_{t+1} was not significant, a2*b2 = 0.040 (*SE* = 0.031), 95% CI: [-0.016, 0.107], p>.05. Thus, AAQ did not mediate the relation between ASI and BDI in the prospective model.

Concurrent Model Path Analysis. Controlling for Time, Condition, and Time x Condition, results for path a1 showed that ASI $Deviation_t$ were related to AAQ $Deviation_t$, b = 0.22, t(169) = 4.379, p < .001, indicating when participants reported higher than their average level of ASI, they also reported higher than their average level of AAQ. Results for path a2 in the concurrent model are the same as those reported above for the prospective model.

Controlling for Time, Condition, Time x Condition, and disaggregated ASI (ASI $Deviation_t$ and ASI Mean), results showed that AAQ $Deviation_t$ (b = 0.187, t(196) = 1.431, p = .154) did not predict BDI_t (path b1). However, AAQ Mean, b = 0.571, t(193) = 7.160, p < .001, did predict BDI_t (path b2). Neither the ASI $Deviation_t$ effect on BDI_t (c1' path), (p = .271), nor the ASI Mean effect on BDI_t (c2' path) (p = .467), was significant.

Test of Concurrent Mediation. The distribution of products test showed that the mediated pathway from ASI_t deviations to AAQ_t deviations to concurrent BDI_t was not significant, a1*b1 = 0.042 (*SE* = .031), 95% CI: [-.014, 0.108], p>.05. However, the mediated pathway from mean ASI to mean levels of AAQ to BDI_t was significant, a2*b2 = 0.205 (*SE* = .059), 95% CI: [0.098, 0.330], p<.05, P_M=63.41%. These latter results

indicate that between-subjects variability in average AAQ levels mediated the concurrent relation between mean level of ASI and BDI.

Chapter 4: Discussion[5]

The current study aimed to test whether EA mediates the relation between AS and depressive symptom severity. While previous research has supported this pathway cross-sectionally, the current study was the first to examine this longitudinally. Consistent with prior research (Tull & Gratz, 2008; Zvolensky et al., 2015), we found that EA mediated the concurrent, between-subjects association between mean levels of AS and depression symptom severity. However, analyses testing EA as a quasi-causal mediator in the AS-depression symptom severity relation using a prospective model – that is, the cross-lagged AS to EA to depression symptom severity pathway – failed to support our hypothesis. The discrepancy between the concurrent, between-subjects finding and the prospective, cross-lag findings underscores the importance of estimating causal pathways in mediation analyses, as solely relying on concurrent relations or between-subjects analyses may yield false positives (Maxwell & Cole, 2007).

There are a number of potential explanations for this discrepancy. First, it is possible that the time between assessments does not accurately reflect the time-course of the effect. Specifically, it is possible that the effects of EA on depressive symptoms are either immediate or shorter than the time between assessments in this study. If the effects are immediate, then it is possible that the effects could be washed out before the following assessment point, which could explain why the lagged model was not

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significant. Future research should determine the time course of the effect of each variable in order to optimize the time between measurements. Second, it is also possible that the lack of mediation findings may be due to variable or suboptimal reliability for the ASI and AAQ. It is possible that updated versions of these measures (e.g., AAQ-II; Bond et al., 2011or ASI-3; Taylor et al., 2007) would have yielded different results. Finally, it is possible that the significant between-subjects, concurrent mediation analysis does not reflect a true mediation effect. Rather, it may represent relations between variables that may be caused by either third variable confounds (e.g., people with more severe problems having higher scores on all the measured variables) or reverse relations. Since the previous investigations of the relations among AS, EA, and depression were primarily between-subjects studies, our findings suggest that it is possible that these previous findings may have been a result of third variable confounds also.

Unfortunately, in this initial study testing the relation between AS and depressive symptom severity, we opted to focus only on EA as a mediator, consistent with the model outlined by Otto and colleagues (2016). Due to power constraints and the desire to minimize the complexity of these models, we focused our analysis on testing the specific AS to EA to depression model proposed by Otto et al. (2016) and therefore did not to control for potential third variable confounds, such as anxiety or self-efficacy.

We should point out that we did find a longitudinal, within-subjects relation between ASI and EA in the concurrent mediation analysis. Had we not failed to obtain a significant relation between ASI and EA in the cross-lag mediation analysis, we perhaps would take this as some evidence supporting at least the first step in the ASI to EA to

depression mediation pathway. However, since this relation was not evident in the cross-lag analysis, it suggests that the obtained relation may be due to some other within-person change variable (e.g., self-efficacy) or to reverse causation. But there also remains the possibility that the ASI to EA link is causal, just that we weren't able to detect it because our lag was not correct, our measures were not optimal, or our power was insufficient.

Encouraging replication and extension, we suggest that researchers consider a number of measurement issues to improve upon this study and its findings. First, we recommend that tests of causality be complemented by tests of specificity, such that several putative mediators are entered into models simultaneously (Smits, Julian, Rosenfield, & Powers, 2012). Second, replication with a larger sample is warranted. It is possible that we did not have sufficient power to detect a small effect in the lagged model (the effects in the lagged model would likely be smaller than the effects in the concurrent model since relations in the lagged model may be attenuated by the passage of time and by controlling for reciprocal causation). Finally, it is also possible that the depression symptom severity of the sample was too mild to detect an effect. Future research in a sample of participants with major depression is warranted.

Despite these limitations, this is the first study to examine the AS-EA-Depression mediated pathway longitudinally. While some of our between-subjects findings are consistent with the hypothesis that part of the relationship between anxiety sensitivity and depression may be explained by experiential avoidance, the causal nature of this relationship and the specificity of this relationship remain unclear. Clinically, these results provide further support for the malleability of dispositional risk factors (i.e., AS

and EA) for the development and maintenance of psychopathology. Although these relationships have not yet been tested in clinical samples, our results support the viability of interventions targeting AS for prevention of a broader range of psychopathology, including depression.

Tables and Figures

Table 1. Descriptive Statistics for Outcome Measures

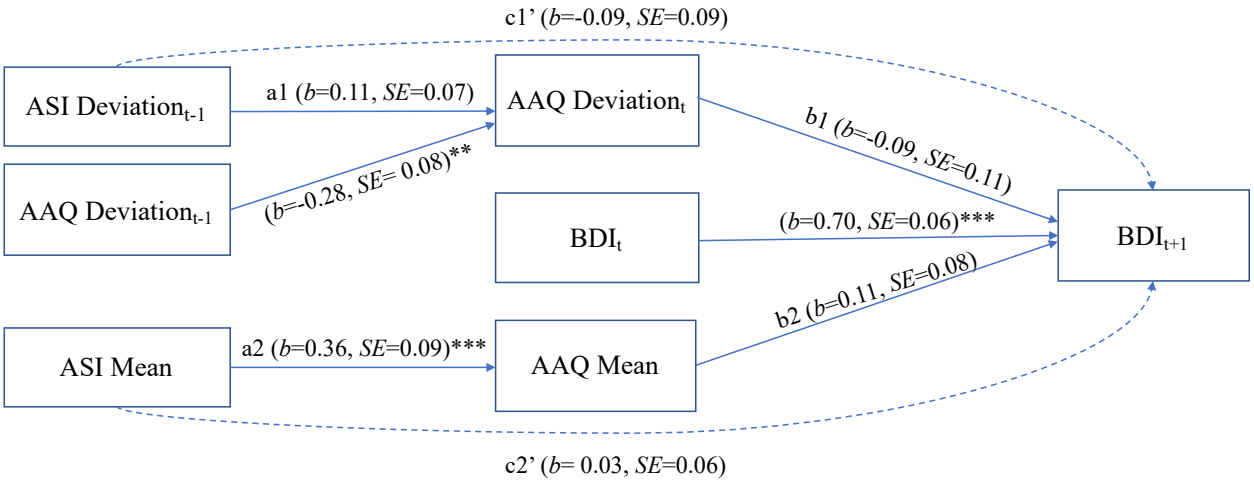
	Waitlist			Exercise			Exercise + CR		
	n	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
ASI									
Pretreatment	20	34.55	6.62	19	34.32	7.02	21	31.71	5.61
Mid-treatment	19	32.58	7.46	17	24.29	8.15	14	21.36	11.32
Posttreatment	19	30.21	6.24	16	14.50	6.52	14	14.35	10.43
Follow-up	16	29.94	7.99	16	12.25	6.59	14	10.57	7.44
BDI									
Pretreatment	20	13.43	6.21	19	13.16	7.98	21	13.31	7.33
Mid-treatment	19	13.76	7.38	17	9.35	6.12	14	8.14	8.75
Posttreatment	19	12.58	7.13	16	5.75	4.19	14	7.07	7.31
Follow-up	16	13.44	8.89	16	5.13	4.56	14	5.86	7.53
AAQ-II									
Pretreatment	20	40.15	6.89	19	39.68	6.57	21	42.29	5.33
Mid-treatment	19	39.53	7.21	17	37.47	5.82	14	38.86	5.488
Posttreatment	19	39.47	8.03	16	34.06	6.71	14	36.07	7.41
Follow-up	16	38.69	7.31	16	33.75	6.92	14	33.68	7.05

Table 2. Descriptive Statistics and Correlations for Outcome Measures

	1	2	3	4	*M*	*SD*
AAQ - Total	---				38.08	7.11
ASI – Total	.48***	---			25.22	11.43
BDI - Total	.56***	.46***	---		10.45	7.64

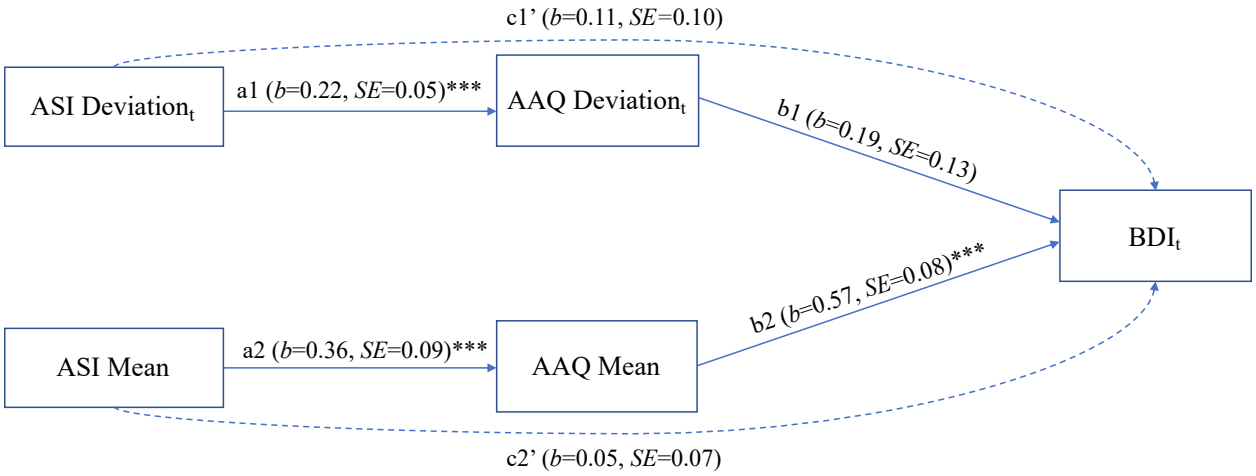
Note: *p <.05, **p <.01, ***p<0.001. AAQ = Acceptance and Action Questionnaire; ASI = Anxiety Sensitivity Index; BDI-II = Beck Depression Inventory-II. Mean, standard deviation, and correlation coefficients represent average across all timepoints.

Figure 1. Results of Cross-Lagged Mediation Model



Note. (Coefficient *b*, std. error); $p < .001$***, $p < .01$**, $p < .05$*. We controlled for the effects of study condition and time by including covariates, Condition and Time, at each step of the analysis. Mediation pathway indirect effect estimate, a1*b1 = -0.010 (*SE* = 0.027), 95% CI: [-0.077, 0.037]; indirect effect estimate, a2*b2 = 0.040 (*SE* = 0.031), 95% CI: [-0.016, 0.107].

Figure 2. Results of Concurrent Mediation Model



Note. (Coefficient *b*, std. error); $p < .001$***, $p < .01$**, $p < .05$*. We controlled for the effects of study condition and time by including covariates, Condition and Time, at each step of the analysis. Mediation pathway indirect effect estimate, a1*b1 = 0.042 (*SE* = .031), 95% CI: [-.014, 0.108]; indirect effect estimate, a2*b2 = 0.205 (*SE* = .059), 95% CI: [0.098, 0.330], P_M=63.41%.

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