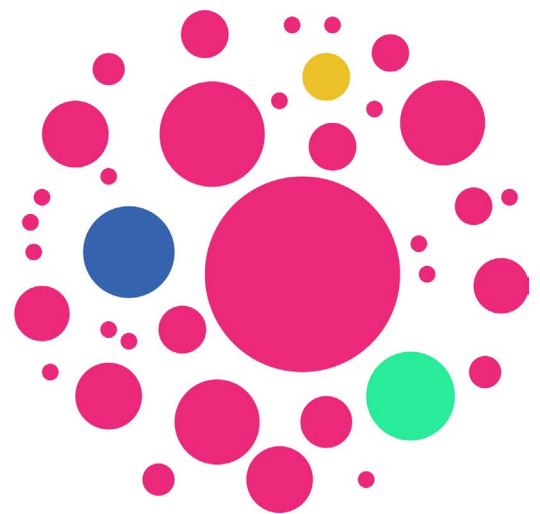


CADRe Lab

Cognition &
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CLINICAL PSYCHOLOGY & NEUROPSYCHOLOGY | RESEARCH ARTICLE

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CLINICAL PSYCHOLOGY & NEUROPSYCHOLOGY | RESEARCH ARTICLE

Fear of anxiety or fear of emotions? Anxiety sensitivity is indirectly related to anxiety and depressive symptoms via emotion regulation

Allison J. Ouimet^{1*}, Leanne Kane¹ and Jessica S. Tutino¹

Abstract: *Background and objectives:* Both anxiety sensitivity (AS) and maladaptive emotion regulation (ER) may contribute to anxious and depressive symptoms. Given the overlap between ER and AS—They both pertain to maladaptive beliefs about emotions (BE)—We tested whether AS would demonstrate an indirect relationship with anxiety and depressive symptoms via BE and ER. *Design:* Participants were 150 undergraduate students who completed an online survey. *Methodology:* Participants completed the Anxiety Sensitivity Index-3, difficulties with emotion regulation scale, Beliefs about Emotions Questionnaire, and Depression Anxiety Stress scales. *Results:* Bootstrapped serial mediation analyses demonstrated that the relationship between AS and anxiety symptoms was partially attributable to BE and ER, but not to BE alone. Similarly, the relationship between AS and depressive symptoms was completely attributable to BE and ER, but not to BE alone. Supplemental analyses suggested that beliefs about the controllability of emotions/anxiety were particularly important in the indirect nature of the relationship between AS and anxiety and depressive symptoms. *Conclusions:* AS and ER play an important role in the maintenance of anxiety and depressive symptoms. These results highlight the uncontrollability of emotions as a potentially important construct in cognitive-behavioural

ABOUT THE AUTHORS

The Cognition and Anxiety Disorders Research (CADRe) Lab directed by Dr Allison J. Ouimet, in the School of Psychology at the University of Ottawa is devoted to understanding the development, maintenance, and treatment of anxiety disorders. We are particularly interested in how cognition contributes to the experience of anxiety. Moreover, we endeavour to use this knowledge to continue to refine cognitive behavioural therapy (CBT) for these problems. The findings from this paper contribute to our understanding of how people think about and regulate their emotions, and how these tendencies contribute to anxiety symptoms. We also have several ongoing projects related to: Understanding the differential impact of implicit (e.g. urges) and explicit (e.g. reappraisal) cognition on the regulation of anxiety and other emotions; Using psychophysiological measures to assess information processing in anxiety; Integrating findings with CBT models to develop and refine current treatments.

PUBLIC INTEREST STATEMENT

People who experience anxiety and depressive symptoms tend to have difficulty regulating their emotions. We tested whether this tendency was related to catastrophic beliefs about fear and anxiety specifically, or emotions generally. 150 participants completed an online survey assessing depressive and anxiety symptoms, fear of anxiety symptoms, beliefs about emotions, and emotion regulation abilities. Although fear of anxiety symptoms was related to both anxiety and depressive symptoms, these relationships were accounted for by maladaptive beliefs about emotions and emotion regulation strategies. Additionally, believing you have little control over your emotions seems to be particularly important in these relationships. In other words, the more people fear anxiety symptoms, the less control they believe they have over their emotions, and the more symptoms of anxiety and depression they report. As such, targeting beliefs about the controllability of emotions may be particularly important for treating mood and anxiety problems.

models of anxiety and emotion regulation. The cross-sectional design and non-clinical sample limit the generalizability of our findings; replication and extension in other samples and via experimental designs is warranted.

Subjects: Emotion; Cognition & Emotion; Mood Disorders in Adults - Depression, Mania, Bi-polar; Anxiety in Adults

Keywords: anxiety sensitivity; emotion regulation; anxiety; depression; beliefs about emotions

Anxiety sensitivity (AS)—the fear of anxiety-related symptoms because of their potential harmful consequences (Reiss & McNally, 1985)—plays an important role in the development and maintenance of anxiety symptoms (Olatunji & Wolitzky-Taylor, 2009). According to cognitive-behavioural therapy (CBT) models, maladaptive beliefs about the meaning of such symptoms (e.g. “I am having a heart attack”) and/or ability to cope with symptoms (e.g. “I can’t tolerate feeling out of breath”) contribute to fear of physiological sensations and behaviours designed to decrease anxiety (e.g. avoidance, entering feared situations only with a “safe person”) (see Clark & Beck, 2010; for a detailed review). Research findings suggest that AS is associated with symptoms of anxiety and depression (Naragon-Gainey, 2010; Taylor, Koch, Woody, & McLean, 1996). Indeed, beliefs about the meaning of physiological symptoms feature prominently in cognitive models of these disorders.

Recent reviews suggest that anxiety sensitivity does not predict psychological symptoms directly, but rather that other factors such as experiential avoidance and deficits in emotion regulation (ER) may moderate the relationships between AS and specific emotional problems (Cisler, Olatunji, Feldner, & Forsyth, 2010; Naragon-Gainey, 2010). Emotion regulation refers to a series of strategies employed in response to emotional experiencing (e.g. Cisler et al., 2010). Models of emotional dysregulation suggest that ER encompasses the awareness, understanding, and acceptance of emotions (Gratz & Roemer, 2004), which have demonstrated relationships with beliefs about emotions (BE) (Manser, Cooper, & Trefusis, 2012). For example, people who have maladaptive BE (e.g. as intolerable) may hold similar maladaptive beliefs about anxiety symptoms. One of our goals for the current study was to test whether maladaptive beliefs about anxiety (i.e. anxiety sensitivity) were distinct from maladaptive beliefs about emotions generally. Moreover, we investigated whether beliefs about emotions may predict ER strategies—at least statistically—particularly in relation to anxiety and depressive symptoms.

Anxiety and depression are highly comorbid in primary care service users (e.g. Hirschfeld, 2011)—this co-occurrence may reflect an overlap in cognitive styles (e.g. catastrophizing, rumination; Martin & Dahlen, 2005). Furthermore, individuals with anxiety and depression share a tendency to react maladaptively to aversive emotions. These maladaptive BE (e.g. “Feeling anxious is always bad”) may lead people to use unhelpful strategies to neutralize, negate, or avoid these emotions (e.g. suppression of anxiety, avoidance of stressful situations; Manser et al., 2012). However, because anxiety sensitivity refers to beliefs about the dangerousness of anxiety symptoms, we propose that maladaptive BE are specifically related to AS. For example, one of the most well-supported CBT models of panic disorder contends that panic disorder develops because people misinterpret anxiety symptoms as catastrophic, and then engage in maladaptive ER strategies such as avoidance or escape (Clark, 1986). Researchers have further demonstrated that reductions in these catastrophic misinterpretations precede symptom reductions during CBT for panic disorder (Teachman, Marker, & Clerkin, 2010). Similarly, catastrophic misinterpretations of physical symptoms have been implicated in CBT models of social anxiety disorder, highlighting fears of social consequences of such symptoms, and again leading to engagement in maladaptive regulation strategies (e.g. Clark & Wells, 1995). Researchers recently demonstrated that, following group CBT for social anxiety disorder, reductions in socially- and physically-relevant catastrophic beliefs about anxiety (as measured by the ASI; Reiss, Peterson, Gursky, & McNally, 1986) predicted lower symptoms of social anxiety, after controlling for pre-treatment social anxiety and changes in depressive symptoms (Nowakowski, Rowa, Antony, & McCabe, 2016).

The fear of anxiety-related symptoms—*anxiety sensitivity*—stems from the belief that experiencing fear or anxiety will lead to harmful outcomes (Reiss & McNally, 1985). Individuals with high AS tend to perceive anxiety as harmful in more than one way (e.g. “anxiety causes embarrassment and it causes me to feel ill”; Reiss et al., 1986). Anxiety sensitivity is closely related to anxiety disorders (Taylor et al., 1996), and may play a causal role in the experience of panic symptoms (Dixon, Sy, Kemp, & Deacon, 2013). As such, AS is likely a broad vulnerability factor for increased anxiety symptomatology, and is particularly important in the development of specific anxiety disorders.

Whereas existing literature has focused largely on the role of AS in anxiety, research findings demonstrate that depressive symptoms are also related to high AS (Naragon-Gainey, 2010). Individuals with depression scored higher on the Anxiety Sensitivity Index (ASI) than did individuals without a clinical diagnosis (Taylor et al., 1996). Taylor et al. also suggest that whereas fear of publicly observable symptoms and fear of bodily symptoms relate solely to symptoms of anxiety, fear of loss of cognitive control may be specific to depressive symptoms (Taylor et al., 1996). These research findings indicate that AS may be involved in both anxiety and depressive symptoms, but the core fear-related cognitions likely differentiate AS' influence on the different emotional states.

People with clinical levels of anxiety and depression tend to be more aware and less accepting of their emotions (Campbell-Sills, Barlow, Brown, & Hofmann, 2006). Consequently, these individuals are more susceptible to emotion over-regulation, and may use ER strategies to avoid aversive emotions. Indeed, in a meta-analytic review of ER strategies across various disorders, Aldao, Nolen-Hoeksema, and Schweizer (2010) found that, in both clinical and community samples, anxiety and depression were positively associated with avoidance, suppression and rumination. By contrast, they were negatively associated with adaptive ER strategies, such as reappraisal and problem solving.

These findings suggest that people with symptoms of anxiety and depression may use specific ER strategies (e.g. avoidance, suppression) at different points during emotional processing. According to Gross' (1998) process model of emotion regulation, people can regulate emotions at five stages: situation selection, situation modification, attentional deployment, cognitive change and response modulation. Regulating emotions at any of these stages serves to enhance, lessen, or neutralize an emotion (Gross, 1998). For example, researchers have demonstrated that two of the most common ER strategies, cognitive reappraisal and expressive suppression, occur at different stages in the emotional process (cognitive change and response modulation, respectively; Gross & John, 2003). This pattern suggests that these strategies serve different functions: cognitive reappraisal involves cognitively manipulating an emotion in order to change its intensity, whereas expressive suppression involves inhibiting emotions once they have already begun to occur. Moreover, situation selection may be particularly important in anxiety and depressive states, as it involves avoiding situations that elicit certain emotional responses.

Beliefs about emotions may lead to maladaptive ER strategies in anxiety and depression. For the current study, we used the Beliefs about Emotions Questionnaire (BAEQ; Manser et al., 2012) and the Difficulties in Emotion Regulation scale (DERS; Gratz & Roemer, 2004), because they appear to highlight distinct but related emotion dysregulation constructs. Whereas the BAEQ measures specific beliefs about emotions (e.g. emotions as overwhelming and uncontrollable), the DERS assesses emotion regulation in a broader scope, measuring both cognitions and behavioural strategies. The BAEQ and the DERS have never been used together in a single study. As such, our findings will also provide information about how separable the measurement of different components of emotion regulation may be.

Research findings have shown that emotion regulation deficits as measured by the DERS are associated with increased anxiety symptomatology, such as worry and symptoms of GAD (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). Lynch, Robins, Morse, and Krause (2001) and found that suppression of emotions mediates the relationship between emotional distress and

negative affect in both heterogeneous clinical and non-clinical samples. Additionally, the “overwhelming and uncontrollable” subscale of the BAEQ accounted for a substantial proportion (10.3%) of the variance in the anxiety scores of individuals in a non-clinical sample (Manser et al., 2012). By comparison, the “emotions as damaging” subscale was the greatest predictor of depression scores, accounting for 5.3% of the variance.

Joormann and Gotlib (2010) found that people with clinical depression used maladaptive ER strategies more so than did control or remitted participants. Longitudinal studies revealed that maladaptive ER strategies predicted future depressive episode severity (e.g. O’Neill, Cohen, Tolpin, & Gunthert, 2004), suggesting a causal role of ER on depression. Rumination appears particularly important in the development and maintenance of depression (e.g. Aldao et al., 2010), and has predicted depression severity over a one-year period (Nolen-Hoeksema, Larson, & Grayson, 1999). Furthermore, recovered depressed individuals demonstrated lower emotional acceptance and clarity than never depressed individuals as measured by the DERS scores (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008). These results suggest that similar to anxiety, certain facets of ER appear to be more strongly related to depression, and that consequently, targeting ER strategies directly may improve treatment outcomes for depression.

Recent research findings indicate that there may be interactive effects between AS and ER in predicting anxious and depressive pathological behaviour (Vujanovic, Zvolensky, & Bernstein, 2008). Specifically, in a correlational study within a community-based sample, the interaction between anxiety sensitivity (as measured by the ASI-3) and emotion regulation (as measured by the DERS) predicted anxiety symptomatology (e.g. worry, arousal), though this effect was marginal. Eifert and Forsyth (2005) suggest that maladaptive ER may be a pivotal contributor in transitioning from prodromal AS to clinically relevant levels of anxiety. Assessing these risk factors in an integrated model may provide a more holistic view of anxiety and mood disorders. By exploring the extent to which individuals who report varying levels of anxiety and depressive symptoms perceive, tolerate, and regulate their emotions, we can better understand the factors that contribute to the development and maintenance of these disorders.

Tull (2006) demonstrated that ER deficits as measured by the DERS better predicted panic *symptom severity* than did AS, whereas AS better predicted panic *attack frequency*, suggesting that anxiety sensitivity and emotion regulation may play distinct roles in the context of panic (and perhaps other anxiety problems). Similar to Vujanovic et al. (2008), Kashdan, Zvolensky, and McLeish (2008) demonstrated that although there was no relationship between AS and anxiety symptoms, an interaction emerged such that participants with poorer ER evidenced correlations between AS and worry, anxious arousal, and agoraphobic cognitions. Relatedly, people with high anxiety sensitivity and low emotion regulation have demonstrated high levels of anxiety and related constructs, whereas only deficits in ER predicted depressive symptoms (Vujanovic et al., 2008).

Extant literature on the combined influences of anxiety sensitivity and emotion regulation in depression is substantially more limited. Tull and Gratz (2008) have demonstrated, however, that in a university sample, the relationship between AS and depression is mediated by experiential avoidance. Moreover, Cox, Enns, and Taylor (2001) found that rumination mediated the relationship between AS (specifically, fears of cognitive dyscontrol) and depression in a clinical outpatient sample. These findings suggest that facets of maladaptive ER may underlie the link between AS and depression. Research clarifying the differential impact of ER and AS may elucidate how different beliefs about emotions—and anxiety specifically—contribute to the development and maintenance of anxiety and depressive symptoms and disorders.

2. Current study

We designed the current study as a first step in investigating whether anxiety sensitivity demonstrates an indirect relationship with anxiety and depressive symptoms via maladaptive beliefs about emotions and emotion regulation strategies, as measured by self-report questionnaires in an

unselected sample. Furthermore, we were interested in examining whether AS is relevant to affective symptoms in general or to anxiety more specifically. As such, we hypothesized the following: (1) maladaptive BE would predict maladaptive ER (both potential statistical mediators) in this model, and (2) AS, although related to both anxiety and depressive symptoms, would be a stronger independent predictor (i.e. demonstrate a stronger direct effect) in the anxiety model than in the depressive symptoms model. In other words, we predicted that beliefs about emotions and emotion regulation would account for the relationship between anxiety sensitivity and depressive symptoms to a greater degree than between AS and anxiety symptoms. Although we are not able to assess the actual chronology of these relationships because of our cross-sectional design, our first hypothesis is consistent with cognitive-behavioural models, which contend that thoughts drive emotions and related behaviours (see Clark & Beck, 2010). Finally, we were also interested in investigating which subscales of the BAEQ and DERS contributed most to the indirect relationship between AS and anxiety and depressive symptoms, to clarify whether certain components of ER are more relevant to anxiety and depression symptoms.

3. Methods

We followed procedures in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 1983. The Research Ethics Board at the University of Ottawa approved all of our methods and procedures in advance of data collection.

3.1. Participants

We recruited 150 people (84% female, $M_{\text{age}} = 18.83$ years; $SD = 1.51$) from the undergraduate psychology participant pool at the University of Ottawa (Canada), who were compensated for their participation with course credit. In order to participate in the study, participants were required to understand written English. We obtained informed consent online via an active click from all individual participants included in the study. The majority of participants were single (81.3%). They described their ethnicity as White (62%), Asian (14%), other (8%), multiple ethnicities (6%), Black (4.7%), European (3.3%), Hispanic (0.7%) and Native Canadian (0.7%); 0.7% preferred not to answer. Additionally, 12% of participants reported that they had previously been diagnosed with a psychological disorder.

3.2. Measures

3.2.1. Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007)

The ASI-3 is a multifactorial measure of AS composed of 18 items. People rate their level of agreement (0 = *Very little*, 4 = *Very much*) to items across three 6-item subscales. The Cognitive Concerns subscale contains items such as: “When I cannot keep my mind on a task, I worry that I might be going crazy”. The Social Concerns subscale contains items such as: “It is important for me not to appear nervous”. The Physical Concerns subscale contains items such as: “When my throat feels tight, I worry that I could choke to death”. Scores are generated by computing the sum of items for each subscale and the total score. The Cognitive, Social and Physical Concerns subscales and the overall AS score demonstrate good to excellent internal consistency ($\alpha = .90, .80, .88$ and $.93$, respectively; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012). See Table 1 for internal consistency for all scales in the current sample.

3.2.2. Beliefs about Emotions Questionnaire (BAEQ; Manser et al., 2012)

The BAEQ is a 43-item scale that assesses beliefs about emotions across six subscales: (1) Overwhelming and uncontrollable (e.g. “Once I start feeling upset, there’s nothing I can do to stop it”; nine items), (2) Shameful and irrational (e.g. “I should feel ashamed of feeling upset”; 10 items), (3) Invalid and meaningless (e.g. “When I feel upset I should take notice of it [reverse-scored]”; seven items), (4) Useless (e.g. “I’d prefer it if I never felt upset”; eight items), (5) Damaging (e.g. “Feeling upset will harm others”; five items) and (6) Contagious (e.g. “If I feel upset, other people will become upset”; four items). People rate their level of agreement (1 = *Strongly disagree*, 5 = *Strongly agree*).

agree) with each item. The six subscales have demonstrated adequate to good internal consistency ($.69 \leq \alpha \leq .88$) and adequate test-retest reliability (Manser et al., 2012). In the current sample, however, the Beliefs about Emotions as Invalid and Meaningless and Beliefs about Emotions as Contagious subscales demonstrated inadequate internal consistency ($\alpha = .35$ and $\alpha = .46$, respectively); the overall scale demonstrated good internal consistency. As such, we excluded those two subscales from any analyses that required the use of individual subscales, but retained all items when conducting analyses that required the use of the total scale score.

3.2.3. Depression Anxiety Stress scales—Depression and Anxiety subscales (DASS; Lovibond & Lovibond, 1995)

The DASS is a 42-item measure of depressive, anxiety and stress symptoms. We used the Depression and the Anxiety subscales for the current study, because they measured constructs that best mapped onto previous research. Respondents rate how much each statement applied to them over the past week (0 = *Did not apply to me at all*, 3 = *Applied to me very much, or most of the time*). The 14-item Depression subscale contains items such as “I couldn’t seem to experience any positive feeling at all”. The 14-item Anxiety subscale contains items such as “I felt I was close to panic”. The Depression and Anxiety subscales demonstrate excellent internal consistency ($\alpha = .97$ and $.92$, respectively; Antony, Bieling, Cox, Enns, & Swinson, 1998). The DASS differentiates between clinical and non-clinical groups, as well as between individuals with depression and panic disorder (Antony et al., 1998).

3.2.4. Difficulties in Emotion Regulation scale (DERS; Gratz & Roemer, 2004)

The DERS measures emotion dysregulation and consists of 36 items across six subscales: (1) Non-acceptance of emotional responses (e.g. “When I’m upset, I become angry with myself for feeling that way”; six items), (2) Difficulties Engaging in Goal-Directed Behaviour (e.g. “When I’m upset, I have difficulty getting work done”; five items), (3) Impulse Control Difficulties (e.g. “When I’m upset, I become out of control”; six items), (4) Lack of Emotional Awareness (e.g. “I pay attention to how I feel” [reverse-scored]; six items), (5) Limited access to ER strategies (e.g. “When I’m upset, I believe there is nothing I can do to make myself feel better”; eight items) and (6) Lack of Emotional Clarity (e.g. “I am confused about how I feel”; 5 items). Participants rate how often items apply to them on a five-point scale (1 = *Almost never*, 0–10%; 5 = *Almost always*, 91–100%). The six subscales and total scale demonstrate good internal consistency ($0.80 \leq \alpha \leq 0.93$); the scale demonstrates good test-retest reliability (Gratz & Roemer, 2004).

3.3. Procedure

Participants completed the study online through the participant pool system. Following informed consent, they completed a demographic questionnaire, followed by the four self-report questionnaires in a completely randomized order.

3.4. Data analysis

To test for our hypothesized indirect effects, we conducted two separate bootstrapped multiple mediation models (5,000 samples, 95% confidence interval, bias corrected) using PROCESS Macro for SPSS (Hayes, 2013).¹ We determined mediational pathway significance by examining its confidence interval: A confidence interval including zero was statistically non-significant (Preacher & Hayes, 2004). For all models, the independent variable was ASI-3 score. We selected a serial mediation analysis over a parallel mediation analysis for two main reasons. First, we contend that beliefs about emotions precede actual selection of emotion regulation strategies, consistent with cognitive-behavioural models. Second, BAEQ and DERS scores remained significantly correlated when accounting for ASI-3 scores, $r_{\text{partial}} = .67$, $p < .001$, indicating a possible directional relationship between the two mediators. For the first model, we conducted a serial mediation analysis with DASS Anxiety scores entered as the outcome variable and BAEQ and DERS scores entered as mediating variables in this order (Model 1).² We conducted the second serial mediation analysis with DASS Depression scores as the outcome variable

(Model 2). We defined the order of the mediators to test the hypothesis that BE underlies ER. We used standardized Z-scores for all measures in the analyses to enable a better comparison across models.

Additionally, we conducted supplemental mediation models to identify which facets of ER accounted for any observed meditation between AS and anxiety severity and AS and depression severity. First, we entered the four BAEQ subscales (those with adequate reliability) as parallel statistical mediators between AS and anxiety severity (Model 3) and depressive severity (Model 5), respectively. Second, we entered all six DERS subscales as parallel statistical mediators between AS and anxiety severity (Model 4) and depressive severity (Model 5), respectively.

4. Results

4.1. Sample characteristics

See Table 1 for descriptive data. Analyses of variance (ANOVAs)³ indicated that there were no significant differences on the measures of interest depending on ethnicity or household annual income. A significant gender difference emerged for the DASS Anxiety subscale such that people who identified as female obtained higher scores than those who identified as male, *Welch's* $F(1,44) = 9.14, p = .004$.⁴ In addition, people who reported being previously diagnosed with a psychological disorder by a professional scored significantly higher on various measures, ASI-3, $F(1,148) = 7.66$; BAEQ, $F(1,148) = 4.55$; DASS Anxiety, $F(1,148) = 4.02$; DERS, $F(1,148) = 6.39$, all p 's < .05, than did participants who reported no previous diagnoses.

Table 1. Descriptive data

Variable	Mean	SD	Min	Max	α
ASI-3	21.17	13.83	0	66	.91
BAEQ	2.79	.42	1.84	4.01	.89
Overwhelming and uncontrollable	2.88	.81	1.11	5.00	.90
Shameful and irrational	2.13	.75	1.00	4.80	.88
Invalid and meaningless	3.01	.45	1.57	4.14	.35
Useless	3.28	.62	1.38	4.63	.69
Damaging	2.48	.76	1.00	4.40	.67
Contagious	2.97	.60	1.25	4.25	.46
DERS	85.63	23.24	36	173	.95
Impulse control	11.88	4.90	6	30	.87
Awareness	15.07	4.78	6	27	.86
Strategies	18.14	7.26	8	40	.91
Clarity	11.79	4.21	5	24	.87
Nonacceptance	13.09	5.40	6	30	.89
Goals	15.67	4.95	5	25	.90
DASS Anxiety	8.79	7.69	0	38	.90
DASS Depression	9.97	9.94	0	40	.96

Notes: $N = 150$. ASI-3 = Anxiety Sensitivity Index-3; BAEQ = Beliefs about Emotions Questionnaire; DERS = Difficulties in Emotion Regulation scale; Impulse Control = DERS Impulse Control Difficulties subscale; Awareness = DERS Lack of Emotional Awareness subscale; Strategies = DERS Limited Access to Emotion Regulation Strategies subscale; Clarity = DERS Lack of Emotional Clarity subscale; Nonacceptance = DERS Nonacceptance of Emotional Responses subscale; Goals = DERS Difficulties Engaging in Goal-Directed Behaviour subscale; DASS Anxiety = Depression Anxiety Stress scales, Anxiety subscale; DASS Depression = Depression Anxiety Stress scales, Depression subscale.

Table 2. Correlations between anxiety sensitivity, emotion regulation full-scales, and anxiety and depressive symptoms

Variable	1	2	3	4	5
1. ASI-3	–	.55*	.60*	.68*	.49*
2. BAEQ	–	–	.78*	.55*	.56*
3. DERS	–	–	–	.61*	.69*
4. DASS Anxiety	–	–	–	–	.69*
5. DASS Depression	–	–	–	–	–

Notes: ASI-3 = Anxiety Sensitivity Index-3; BAEQ = Beliefs about Emotions Questionnaire; DERS = Difficulties in Emotion Regulation scale; DASS Anxiety = Depression Anxiety Stress scales, Anxiety subscale; DASS Depression = Depression Anxiety Stress scales, Depression subscale.

* $p < .01$.

4.2. Correlations

All measures demonstrated large and significant correlations with one another (all r 's $> .54$, all p 's $< .01$; see Table 2). We used *Steiger's Z*-tests to test whether AS was more related to anxiety than to depressive symptoms (FZT Computator, available for download at <http://psych.unl.edu/psycrs/statpage/regression.html>). We found that the ASI-3 was significantly more correlated with the DASS Anxiety subscale than with the DASS Depression subscale, *Steiger's Z* = 3.75, $p < .01$. We also calculated correlations amongst the six DERS subscales and four BAEQ subscales (see Table 3). Significant correlations emerged between many—but not all—subscales across the two questionnaires.

4.3. Mediation analyses

4.3.1. Model 1

Results indicated that AS demonstrated an indirect relationship with anxiety severity through its relationships with BE and ER via two pathways (see Figure 1 for all coefficients). First, higher anxiety

Table 3. Correlations between emotion regulation subscales

Variable	1	2	3	4	5	6	7	8	9	10
<i>BAEQ subscales</i>										
1. Overwhelming & uncontrollable	–	.55**	.14	.54**	.68*	.15	.77**	.40**	.57**	.57**
2. Shameful & irrational	–	–	.33**	.53**	.55**	.39**	.64**	.49**	.76**	.33**
3. Useless	–	–	–	.10	.10	.45**	.32**	.30*	.22**	.14
4. Damaging	–	–	–	–	.45**	.25**	.46**	.28**	.50**	.31**
<i>DERS subscales</i>										
5. Impulse control	–	–	–	–	–	.18*	.73**	.42**	.63**	.47**
6. Awareness	–	–	–	–	–	–	.34**	.60**	.22**	.12
7. Strategies	–	–	–	–	–	–	–	.51**	.66**	.49**
8. Clarity	–	–	–	–	–	–	–	–	.35**	.22**
9. Nonacceptance	–	–	–	–	–	–	–	–	–	.51**
10. Goals	–	–	–	–	–	–	–	–	–	–

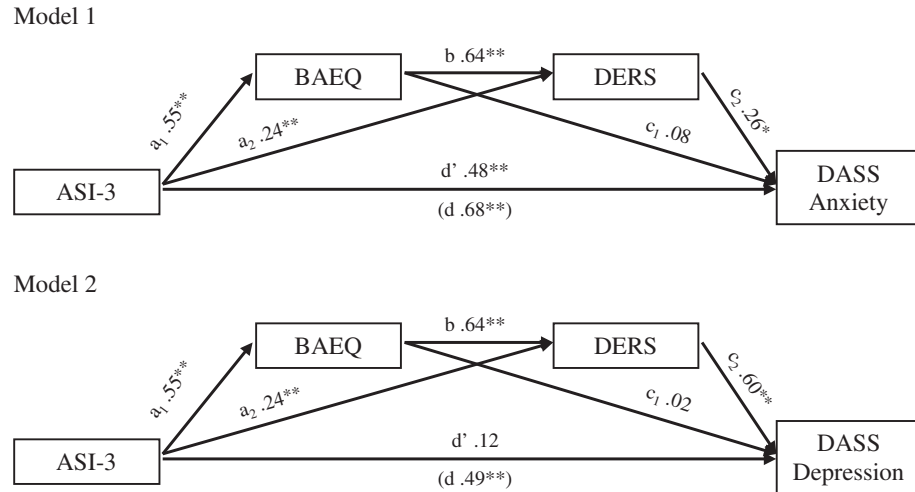
Notes: Overwhelming & uncontrollable = BAEQ Overwhelming & Uncontrollable subscale; Shameful & irrational = BAEQ Shameful & Irrational subscale; Useless = BAEQ Useless subscale; Damaging = BAEQ Damaging subscale; Impulse control = DERS Impulse Control Difficulties subscale; Awareness = DERS Lack of Emotional Awareness subscale; Strategies = DERS Limited Access to Emotion Regulation Strategies subscale; Clarity = DERS Lack of Emotional Clarity subscale; Nonacceptance = DERS Nonacceptance of Emotional Responses subscale; Goals = DERS Difficulties Engaging in Goal-Directed Behaviour subscale.

* $p < .05$.

** $p < .01$.

Figure 1. Mediational Models 1 and 2.

Notes: * $p < .01$, ** $p < .001$; All presented effects are standardized; a_n = effects of IV on mediators; b = effect of mediator 1 on mediator 2; c_n = effects of mediators on DV; d = total effect of IV on DV; d' = direct effect of IV on DV; ASI-3 = Anxiety Sensitivity Index-3; BAEQ = Beliefs about Emotions Questionnaire; DERS = Difficulties in Emotion Regulation scale; DASS Anxiety = Depression Anxiety Stress scales, Anxiety subscale; DASS Depression = Depression Anxiety Stress scales, Depression subscale.



sensitivity scores were related to more maladaptive BE, which was subsequently related to greater emotion regulation difficulties, which was subsequently related to greater anxiety severity. This serial indirect relationship was significant ($a_1bc_2 = .09$, 95% CI = .03–.18).

Second, higher ASI-3 scores were related to greater ER difficulties (without going through the BE pathway), which again were related to higher anxiety severity. This indirect relationship was significant ($a_2c_2 = .06$, 95% CI = .02–.14). Although maladaptive BE was a significant statistical mediator when combined with ER in the serial pathway, there was no indirect relationship through BE alone ($a_1c_1 = .05$, 95% CI = –.06–.15).

Finally, despite AS' significant overall indirect relationship (i.e. through BE and ER; $a_1a_2bc_1c_2 = .20$, 95% CI = .11–.32), it remained significantly related to anxiety symptoms ($d' = .48$, $p < .001$). The overall relationship between AS and anxiety severity was also significant ($d = .68$, $p < .001$). In other words, AS and anxiety severity demonstrated a partially indirect relationship, via BE and ER. This model explained 46.05% of the variability in anxiety severity.

4.3.2. Model 2

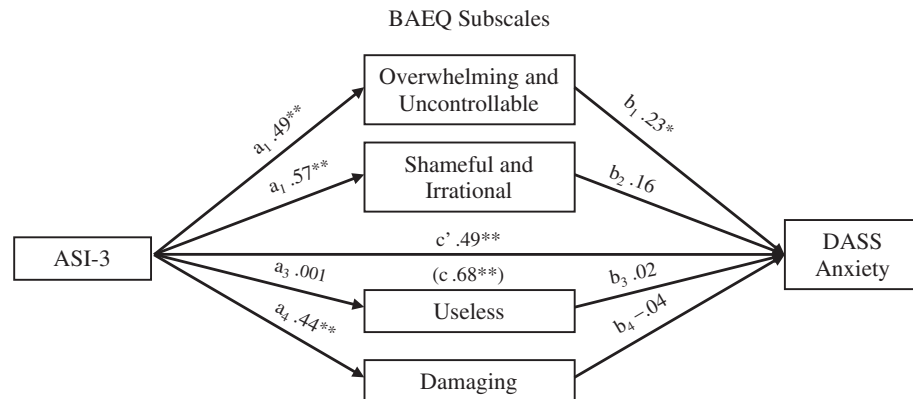
Results of analyses for Model 2 indicated that AS demonstrated an indirect relationship with depressive symptoms through its associations with BE and ER, again via two pathways. First, identical to Model 1, higher anxiety sensitivity scores were related to more maladaptive BE about emotions, which were subsequently related to greater emotion regulation difficulties, which were subsequently related to greater depressive severity. This serial indirect relationship was significant ($a_1bc_2 = .21$, 95% CI = .14–.32).

Second, identical to Model 1, higher ASI-3 scores were related to greater ER difficulties (without going through the BE pathway), which again were related to higher depressive severity. This indirect relationship was significant ($a_2c_2 = .15$, 95% CI = .07–.25). Also similar to Model 1, there was no indirect relationship through BE alone ($a_1c_1 = .01$, 95% CI = –.10–.12).

AS demonstrated significant overall total ($d = .49$, $p < .001$) and indirect relationships (i.e. through BE and ER; $a_1a_2bc_1c_2 = .37$, 95% CI = .25–.51) with depressive symptoms. However, after accounting for the indirect relationships, AS did not remain significantly related to depressive symptoms ($d' = .12$, $p > .10$), indicating that the indirect relationship between AS and depressive symptoms was

Figure 2. Mediation Model 3.

Notes: * $p < .01$, ** $p < .001$; All presented effects are standardized; a_n = effects of IV on mediator; b_n = effects of mediators on DV; c = total effect of IV on DV; c' = direct effect of IV on DV; ASI-3 = Anxiety Sensitivity Index-3; BAEQ = Beliefs about Emotions Questionnaire; DASS Anxiety = Depression Anxiety Stress scales, Anxiety subscale.



completely attributable to BE and ER. This model explained 24.19% of the variability in depressive symptoms.

4.3.3. Supplemental mediational analyses

To better understand what components of ER contributed to the observed indirect relationships between AS and anxiety severity and AS and depression severity, we conducted four parallel bootstrapped mediation analyses using the subscales of the BAEQ and DERS, respectively.

4.3.3.1. Model 3. Results indicated that AS demonstrated an indirect relationship with anxiety symptoms through its relationships with the Beliefs about Emotions as Overwhelming and Uncontrollable subscale only ($a_1 = .49, p < .001, b_1 = .23, p < .01$; all other b 's $< .16, p > .01$). Specifically, higher ASI-3 scores were related to stronger beliefs that emotions are more overwhelming and uncontrollable, which were subsequently related to greater reported anxiety severity. The indirect relationship with anxiety severity was significant ($a_1 b_1 = .11, 95\% \text{ CI} = .03-.21$), as was the total indirect effect ($a_{1-4} b_{1-4} = .18, 95\% \text{ CI} = .09-.31$). In addition, AS remained significantly and directly associated with anxiety severity ($c' = .49, p < .001$) (Figure 2).

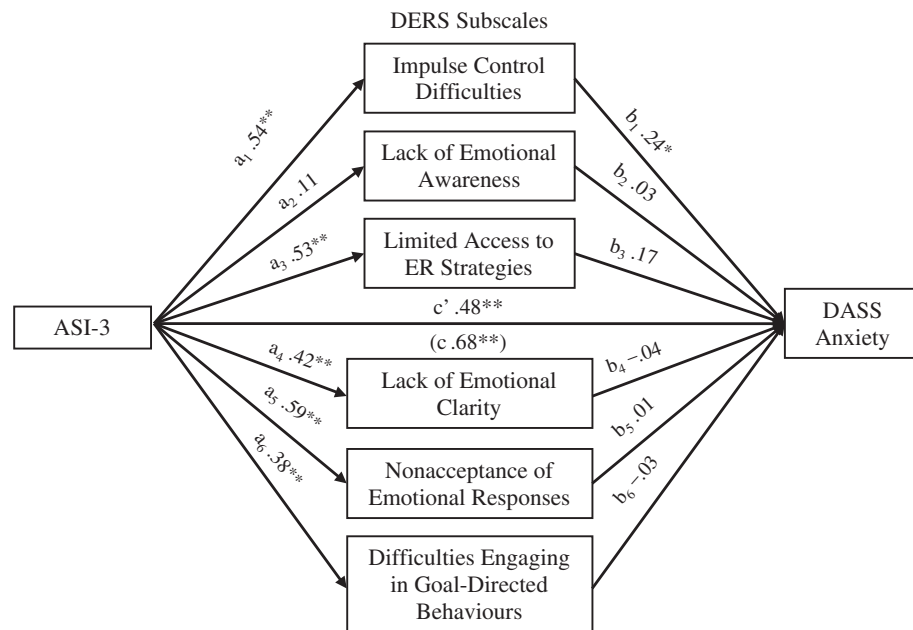
4.3.3.2. Model 4. Results indicated that AS demonstrated an indirect relationship with anxiety severity through its associations with the DERS Impulse Control Difficulties subscale only ($a_1 = .54, p < .001, b_1 = .24, p < .01$; all other b 's $< .17, p > .01$). This subscale pertains to experiencing emotions as out of control and feeling out of control when one is upset. Specifically, higher ASI-3 scores were related to greater perceptions of emotions as being more out of control, which in turn were related to higher reported levels of anxiety severity. The indirect relationship with anxiety symptoms was significant ($a_1 b_1 = .13, 95\% \text{ CI} = .02-.26$), as was the total indirect relationship ($a_{1-6} b_{1-6} = .20, 95\% \text{ CI} = .09-.34$). In addition, AS remained significantly and directly associated with anxiety severity ($c' = .48, p < .001$) (Figure 3).

4.3.3.3. Model 5. Results indicated that AS demonstrated an indirect relationship with depression symptoms through its association with the beliefs about emotions as overwhelming and uncontrollable subscale only ($a_1 = .49, p < .001, b_1 = .45, p < .001$; all other b 's $< .12, p > .01$). Specifically, higher ASI-3 scores were related to stronger beliefs that emotions are more overwhelming and uncontrollable, which were subsequently related to greater depression severity. The indirect relationship with depression severity was significant ($a_1 b_1 = .22, 95\% \text{ CI} = .12-.36$), as was the total indirect relationship ($a_{1-4} b_{1-4} = .27, 95\% \text{ CI} = .14-.41$). In addition, AS remained significantly and directly associated with depression ($c' = .23, p < .01$) (Figure 4).

4.3.3.4. Model 6. Results indicated that AS demonstrated an indirect relationship with depression severity through its association with the DERS Limited Access to ER Strategies subscale only ($a_1 = .53, p < .001, b_1 = .46, p < .01$; all other b 's $< .14, p > .01$). This subscale pertains to the belief that one has little control over the regulation of one's emotions. Specifically, higher ASI-3 scores were related to

Figure 3. Mediational Model 4.

Notes: * $p < .01$, ** $p < .001$;
 All presented effects are standardized; a_n = effects of IV on mediator; b_n = effects of mediators on DV; c = total effect of IV on DV; c' = direct effect of IV on DV; ASI-3 = Anxiety Sensitivity Index-3; DERS = Difficulties in Emotion Regulation scale; DASS Anxiety = Depression Anxiety Stress scales, Anxiety subscale.



greater beliefs that emotion regulation is outside of one's control, which in turn was related to higher levels of depression symptoms. The indirect relationship with depression severity was significant ($a_1b_1 = .24$, 95% CI = $.11-.45$), as was the total indirect relationship ($a_{1-6}b_{1-6} = .37$, 95% CI = $.21-.55$). AS was not significantly and directly related to depression ($c' = .13$, $p = .112$) (Figure 5).

5. Discussion

We tested the hypotheses that anxiety sensitivity (AS) and anxiety severity would demonstrate an indirect relationship via maladaptive beliefs about emotions (BE) and difficulties in emotion regulation (ER), and that maladaptive BE would statistically precede maladaptive ER in this model. Results were partially consistent with our hypotheses. BE and ER did emerge as statistical mediators in the relationship between AS and anxiety severity in a serial manner. However, the indirect relationship was partial and, contrary to our hypothesis, the pathway involving only BE was not significant. Furthermore, we also tested whether AS and depressive symptoms would demonstrate an indirect relationship via BE and ER. Consistent with our hypothesis, results were similar to those in Model 1, although the relationship between AS and depressive symptoms was fully attributable to deficits in BE and ER.

Figure 4. Mediational Model 5.

Notes: * $p < .01$, ** $p < .001$;
 All presented effects are standardized; a_n = effects of IV on mediator; b_n = effects of mediators on DV; c = total effect of IV on DV; c' = direct effect of IV on DV; ASI-3 = Anxiety Sensitivity Index-3; BAEQ = Beliefs about Emotions Questionnaire; DASS Depression = Depression Anxiety Stress scales, Depression subscale.

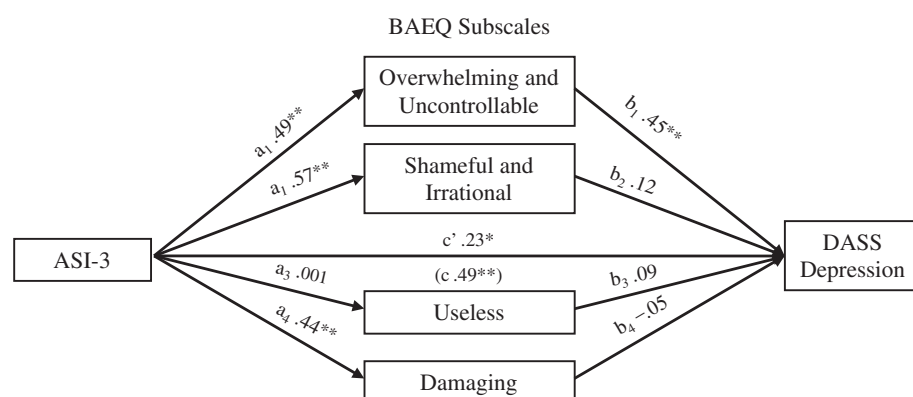
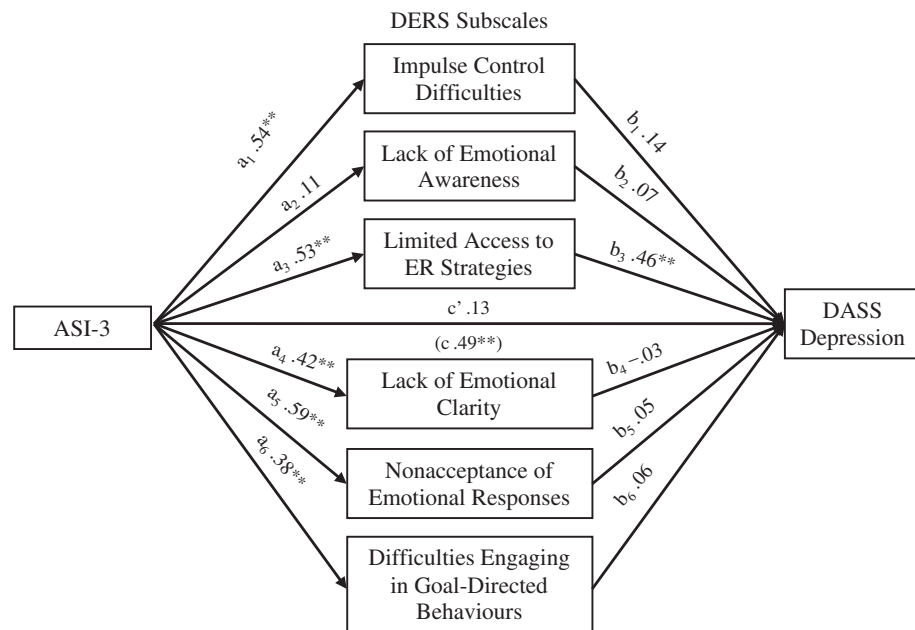


Figure 5. Mediational Model 6.

Notes: * $p < .01$, ** $p < .001$;
 All presented effects are
 standardized; a_n = effects of
 IV on mediator; b_n = effects
 of mediators on DV; c = total
 effect of IV on DV; c' = direct
 effect of IV on DV; ASI-
 3 = Anxiety Sensitivity
 Index-3; DERS = Difficulties
 in Emotion Regulation scale;
 DASS Depression = Depression
 Anxiety Stress scales,
 Depression subscale.



Thus, anxiety sensitivity—although related to both anxiety and depressive symptoms—was a stronger unique construct in the anxiety model than in the depressive symptoms model, when taking ER constructs into account. Moreover, AS was significantly more correlated with anxiety than with depressive symptoms. Taken together, these findings are consistent with previous research suggesting AS is an important and unique construct in anxiety.

Regarding depressive symptoms, our findings suggest that anxiety sensitivity, *per se*, is not particularly important. Rather, the relationship between AS and depressive symptoms may be attributable largely to maladaptive BE and deficits in ER. In other words, people with depressive symptoms may exhibit higher AS *because* of overall ER deficits. Indeed, other models have demonstrated that maladaptive emotion regulation strategies (e.g. rumination, experiential avoidance) mediate the relationship between AS and depression (Cox et al., 2001; Tull & Gratz, 2008). For example, someone who endorsed the item “When I’m upset, I feel out of control” on the DERS may have also endorsed the item “When my breathing becomes irregular, I fear that something bad will happen” on the ASI-3. To the degree that somebody fears emotions generally, they likely fear anxiety symptoms. However, in the context of depressive symptoms, the fear of emotions appears to emerge as most important. Additionally, findings from prior studies have suggested that only one factor of AS—the fear of cognitive dyscontrol—drives the association between AS and depression (Taylor et al., 1996). Fear of cognitive dyscontrol is largely associated with the lack of concentration and the mental fog that individuals with depression often experience. Consequently, Tull and Gratz (2008) highlight the suggestion in the literature that this particular facet of AS might be more indicative of “depression sensitivity”, rather than anxiety sensitivity.

Our findings with respect to anxiety symptoms are partially consistent with a recent review suggesting that emotion dysregulation mediates the relationship between AS and psychopathology (Naragon-Gainey, 2010). Indeed, beliefs about emotions and emotion regulation accounted for some of the variance in this relationship. ER was more important than BE, suggesting that beliefs alone are not sufficient to cause and/or maintain psychopathology. Rather, beliefs may need to influence emotions and behaviour—likely in a maladaptive feedback loop—to impact upon anxious symptoms. In other words, people who hold negative BE, but behave in ways that facilitate ER may not experience mood and anxiety symptoms; whereas people whose negative BE lead them to behave in ways that impair ER likely experience mood and anxiety symptoms. Moreover, those who

engage in maladaptive emotion regulation strategies likely confirm their maladaptive beliefs. For example, somebody who suppresses their anger towards their colleague and then yells at their partner when they get home will confirm their belief that emotions cause problems in relationships. Although the idea that people may hold maladaptive beliefs and yet engage in adaptive behaviours may seem inconsistent with CBT models, there are several instances of maladaptive beliefs occurring in normative samples. For example, in a landmark study, Rachman and de Silva (1978) demonstrated people with OCD and people without a clinical disorder reported obsessions that differed in frequency and distress, but not in content. More recently, researchers demonstrated that cognitive distortions—the hallmark feature of CBT models—occur regularly in non-clinical participants (Covin, Dozois, Ogniewicz, & Seeds, 2011). This possibility, therefore, is consistent with models implicating both cognition and behaviour in the development of anxiety disorders. Cognitive-behavioural models of emotion, however, generally posit that strongly held beliefs facilitate *specific* emotions and types of behaviour (e.g. fear leads to avoidance, sadness leads to isolation, anger leads to attack, etc.; Linehan, 1993). As such, whereas we understand that beliefs alone may not predict psychopathology, we were surprised that they did not play a more important role in the indirect relationship. We expected that BE would account for more variance in its relationship with ER and subsequently anxious and depressive symptoms.

We conducted supplementary analyses to help identify which facets of emotion regulation play a role in explaining anxiety and depressive symptoms. Specifically, we investigated which subscales of the BAEQ and the DERS were most important in the indirect relationships observed in the anxiety and depression symptom models, respectively. For each anxiety model, one subscale emerged as important in the relationship between AS and anxiety severity. Although they purported to measure different constructs, the DERS *Impulse Control Difficulties* and BAEQ *Beliefs about Emotions as Overwhelming and Uncontrollable* subscales may both measure beliefs about the controllability of emotions. For the BAEQ subscale, items assess the degree to which people report a sense of lack of control over their emotions (e.g. “When I’m upset, that feeling takes over completely”). For the DERS subscale, items assess the degree to which people report a sense of lack of control and resulting behaviours (e.g. “When I’m upset, I lose control over my behaviours”). In fact, it is possible that both of these subscales measure a belief related to uncontrollability of emotions—I *cannot control them*—and uncontrollability of relevant behaviours—they *will take over, or I will lose control of the behaviours*. Indeed, these two subscales demonstrated one of the greatest correlations ($r = .68$) among the DERS and BAEQ subscales.

Similarly, only one subscale from each scale emerged as important in each of the models assessing the relationship between AS and depression. The BAEQ *Beliefs about Emotions as Overwhelming and Uncontrollable* and the DERS *Limited Access to ER Strategies* subscales exerted indirect effects on the relationship between AS and depression. Whereas the *Limited Access to ER Strategies* subscale does not directly address control, items such as “When I’m upset, I believe that there is nothing I can do to make myself feel better” allude to a lack of mastery or control over one’s emotions. Notably, the BAEQ *Beliefs about Emotions as Overwhelming and Uncontrollable* had the strongest correlations with the DERS *Limited Access to ER Strategies* ($r = .77$) and *Impulse Control Difficulties* ($r = .68$) subscales, indicating that there is perhaps overlap in these constructs.

These findings are important for two reasons. First, they highlight the similarities between the BAEQ and the DERS. Indeed, these two measures were highly related, providing evidence of the convergent validity of the BAEQ with the DERS. As such, the BAEQ may have emerged as less important simply because of statistical overlap. Future research using structural equation modelling may help clarify whether items on the two scales (and potentially other measures of emotion regulation) actually assess the same latent factors.

Second, our findings suggest that beliefs about the controllability of emotions may form an important aspect of BE and ER—perhaps the most important—in the AS-anxiety relationship. Relatedly, a recent study by Gallagher, Naragon-Gainey, and Brown (2014) indicated that increases in perceived control (as

measured by the revised Anxiety Control Questionnaire [ACQ-R; Brown, White, Forsyth, & Barlow, 2004]) may play a role in the recovery from anxiety disorders. The subscales of the BAEQ and the DERS pertaining to emotion and behaviour control are fairly similar to the items of the ACQ-R, which contains items such as “When I am put under stress, I am likely to lose control [reverse scored]” and “I am able to control my level of anxiety”. Future research examining the role of perceived control in the development and maintenance of anxiety disorders is warranted. Moreover, considering the large impact of ER on depressive symptoms, research examining how beliefs about the controllability of emotions (and consequent behaviours) impact different types of psychopathology may be particularly illuminating.

These findings are also important when considering models of ER generally. Such models are often presented as distinct from cognitive behavioural models of emotional disorders. For example, one recent theoretical model—based on extant research—proposes that maladaptive ER potentiates the effects of anxiogenic situations to facilitate the development and maintenance of anxiety disorders over time (Cisler et al., 2010). Similar to Barlow and colleagues (e.g. Barlow, Allen, & Choate, 2004; Boisseau, Farchione, Fairholme, Ellard, & Barlow, 2010; Farchione et al., 2012), however, we wonder whether ER, rather than a distinct construct, represents a basic cognitive behavioural process wherein “... a common, underlying factor across disorders is the propensity toward increased emotional reactivity, coupled with a heightened tendency to view these experiences as aversive and attempts to alter avoid, or control emotional responding” (Farchione et al., 2012, p. 667). Although Farchione et al. did not use the above quote to define emotion regulation specifically, it clearly applies to various components of emotion regulation. Although the BAEQ was designed to measure only one aspect of ER—beliefs—we found strong correlations between ER and BE. Moreover, subscales of the DERS that were designed to measure behaviour—e.g. Impulse Control Difficulties—appeared to measure *beliefs* about controllability, rather than controllability, *per se*. To this end, we believe that work focused on conceptualizing ER using cognitive-behavioural models may be particularly fruitful. Specifically, parsing ER into emotion-related beliefs, physiological sensations, and behaviours may help clarify its role in emotional disorders, and may also facilitate its integration into existing cognitive-behavioural treatments. An alternative model proposed by Pickett, Lodi, Parkhill, and Orcutt (2012) conceptualizes anxiety sensitivity as a specific vulnerability factor for psychopathology that may result from broader vulnerability factors, such as personality dimensions. Consistent with this theory, the broader risk factor of maladaptive ER may subsequently result in a greater tendency towards AS. Using this top-down model, targeting ER in treatment may consequently reduce AS or even prevent its development.

The current study had certain important limitations. First, we used a sample of unselected undergraduate students; future research testing whether our findings can be replicated in clinical samples is warranted. Second, we used self-report measures and a cross-sectional design. One of the main limitations of such a design is one of method invariance; some constructs—most notably those assessed by the BAEQ and the DERS—may have shown relationships with one another because we assessed each via self-report questionnaires. However, we used the DASS, which has demonstrated excellent diagnostic reliability. Indeed, a comprehensive review provided evidence that the subscales of the DASS are generally specific to the group for whom they are most relevant (Antony et al., 1998). For example, people with depression scored higher on the depression scale than did people with anxiety disorders or non-clinical controls, and people with panic disorder scored higher on the anxiety scale than did people with depression or non-clinical controls. As such, we are confident that our findings with respect to symptom severity and specificity are reliable. However, a limitation of the current study was our inability to use the entire BAEQ scale in our analyses, due to the poor internal consistency of two of the subscales. Importantly, we recognize that although we used a mediational statistical analysis, we cannot infer causality or directionality from our findings. Rather, we view our findings as “laying the groundwork” for research programmes geared towards understanding the specific mechanisms by which anxiety sensitivity uniquely and/or jointly contributes to the development, maintenance, and treatment of mood and anxiety disorders. We look forward to research that investigates the relationships between these variables using prospective and/or experimental designs—to examine whether they are related causally.

Clinically, our findings suggest that treatments designed to reduce anxiety symptoms by targeting AS primarily may be missing an important area of influence. Targeting AS and ER more broadly may help lower anxiety levels more so than targeting AS only. Therapeutic interventions for panic disorder may focus exclusively on reducing catastrophic interpretations of bodily symptoms when in fact catastrophic interpretations of emotions may underlie these more specific bodily fears. Future research would certainly benefit from extending these findings to a clinical sample and testing the utility of these constructs as combined treatment targets, while perhaps concentrating on the sense of lack of control over emotions and ensuing behaviours.

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Competing Interests

The authors declare no competing interest.

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Notes

1. A reviewer suggested we also conduct moderation analyses given that Kashdan et al. (2008) found interactive effects between emotion regulation and anxiety sensitivity on anxiety symptoms. We conducted these analyses for both the anxiety and the depressive symptoms model. However, neither the interactions between anxiety sensitivity and emotion regulation nor the interactions between anxiety sensitivity and beliefs about emotions were significant predictors of psychopathology symptoms. More details regarding these analyses can be obtained from the corresponding author.
2. The mediation analysis was also performed with the Stress subscale—a state of persistent arousal and tension and a low frustration threshold (Lovibond & Lovibond, 1995)—as the outcome variable. Results were very similar to those obtained in Model 1. However, the major focus of this paper is anxiety, which overlaps but is distinct from the Stress subscale (Lovibond & Lovibond, 1995). As such, we did not present the results of the Stress subscale mediation model in the current paper.
3. Welch's adjusted *F* ratio was used when homogeneity of variance assumption was not met.
4. To test the possible confounding effects of gender on results, we performed the analyses for the primary Models 1 and 2 with gender as a covariate, and found virtually identical results. For this reason, analyses presented here are those without gender as a covariate.

Cover image

Source: Allison J. Ouimet.

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