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The Temporal Course of Anxiety Sensitivity in Outpatients with Anxiety and Mood Disorders: Relationships with Behavioral Inhibition and Depression

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Abstract

The present study evaluated the temporal course of three dimensions of anxiety sensitivity (AS; concerns over physical symptoms, mental incapacitation, and social embarrassment) and their relationships with behavioral inhibition (BI) and depression (DEP) in 606 outpatients with anxiety and mood disorders. A semi-structured interview and self-report questionnaires were administered on three occasions over a two-year period. All three constructs decreased over the study period and AS temporally functioned more similar to DEP than BI. Cross-sectional and temporal correlations supported the discriminant validity of AS from BI. As expected, initial levels of BI predicted less improvement in all AS dimensions. In contrast, higher initial levels of mental incapacitation AS were associated with *greater* improvement in DEP. Our results are discussed in regard to the measurement of AS in clinical samples, conceptualizations of AS as a lower-order vulnerability, and prognostic implications of directional paths between BI and AS and AS and DEP.

Keywords

anxiety sensitivity; personality; depression; longitudinal study; outpatients

1. Introduction

Anxiety sensitivity (AS) is defined as a fear of anxiety and anxiety-related sensations. AS is a heritable vulnerability (Stein, Jang, & Livesley, 1999) toward beliefs that anxiety symptoms may have harmful physical (e.g., heart attack or stroke), social (e.g., embarrassment), and psychological (e.g., indication that one is “going crazy”) consequences (Reiss, 1987). The Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1987) is the most widely used measure of AS in research and clinical practice. Although the ASI was originally hypothesized to have a unidimensional structure (Reiss, Peterson, Gursky, & McNally, 1986), subsequent psychometric evaluations have supported a hierarchical factor structure (Zinbarg, Barlow, & Brown, 1997; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004). According to the hierarchical model, the ASI consists of three lower-order factors

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representing fear of the physical (e.g., heart palpitations; ASI-P), social (e.g., appearing nervous; ASI-S), and mental (e.g., being unable to keep focused, feeling “nervous”; ASI-M) consequences of anxiety, as well as a single higher-order factor reflecting general AS. Although AS and its dimensions have often been studied in relation to other personality/temperament vulnerabilities (e.g., trait anxiety) and the anxiety disorders (e.g., panic disorder, social phobia), research has not extensively evaluated the relationships between AS and behavioral inhibition (BI) or depression (DEP).

1.1. Anxiety Sensitivity and Behavioral Inhibition

There has been considerable debate as to placement of AS within a broader nomological network of temperament/personality vulnerabilities believed to influence psychopathology (e.g., trait anxiety, neuroticism). Specifically, although AS was originally conceptualized to be distinct from trait anxiety (i.e., AS is a fearful response to anxiety-related symptoms, trait anxiety is a fearful response to general stressors; McNally, 1989), others argued that AS lacked discriminant validity from trait anxiety (Lilienfeld, Jacob, & Turner, 1989; Jacob & Lilienfeld, 1991). Subsequent theory and research focused on AS and trait anxiety have reconciled much of this debate. For instance, conceptualizations of AS as a lower-order personality construct (Lilienfeld, Turner, & Jacob, 1993; Clark, Watson, & Mineka, 1994) have been supported by hierarchical structural models of AS and trait anxiety (i.e., significant path from trait anxiety to AS; Taylor, 1995). Such findings have encouraged research examining the relationship between AS and constructs closely related to trait anxiety, including neuroticism (Norton, Sexton, Walker, & Norton, 2005), negative affect (Zinbarg & Barlow, 1996), and negative emotionality (Lilienfeld, 1997).

BI is another construct similar to trait anxiety that is thought to be related to AS (Lilienfeld et al., 1993). Gray (1987) defined BI as a temperament that influences an individual's experience of negative affect (e.g., anxiety) and directs behavior in response to threat, punishment, and novelty (e.g., avoidance). Although AS has been studied in relation to higher-order vulnerabilities like BI, nearly all of the extant literature has been cross-sectional in nature (e.g., incremental validity of AS in predicting current symptoms; Norton et al., 2005). Conversely, longitudinal studies have separately documented reductions in BI (e.g., Brown, 2007) and AS (e.g., Smits, Berry, Tart, & Powers, 2008) over time and with treatment. However, there has yet to be an examination of individual differences in the temporal course of AS dimensions or their interrelationships (e.g., discriminant validity, directional paths) with BI. Although AS is conceptualized as a vulnerability (Reiss, 1987), AS dimensions also resemble features of psychopathology (e.g., fear of physical symptoms and panic disorder; fear of social consequences and social phobia). Thus, AS may temporally function like a higher-order vulnerability (i.e., stability increasing as a function of initial severity) or like a *DSM* disorder construct (i.e., stability decreasing as a function of initial severity; Brown, 2007; Curran, Stice, & Chassin, 1997) in clinical samples receiving treatment. In addition to enhancing our understanding of the hierarchical structure of emotional disorders (see Watson, 2005), longitudinal directional relationships between AS and BI (i.e., BI predicting extent of change in AS) could have important implications regarding treatment response (e.g., psychopathological outcomes) and focus (e.g., specifically targeting BI in treatment).

1.2. Anxiety Sensitivity and Depression

The confluence of AS and symptoms of anxiety, occurring in the context of a generalized disposition to experience heightened levels of negative affect (e.g., BI), is presumed to lead to manifestation and maintenance of psychopathology (Barlow, 2002). Although AS was originally conceptualized as related to anxiety and panic (Reiss et al., 1986), studies have also found elevated levels of AS among individuals with DEP (e.g., Otto, Pollack, Fava,

Uccello, & Rosenbaum, 1995). Others extended these findings by clarifying that the relationship between AS and DEP may be specific to mental incapacitation AS (Taylor, Koch, Woody, & McLean, 1996), leading some to hypothesize that mental incapacitation concerns may amplify symptoms of DEP during its course (e.g., interpreting concentration difficulties as signs of an impairing depressive state; Cox, Enns, Freeman, & Walker, 2001; Cox, Taylor, & Enns, 1999). Although subsequent research has predominantly offered support for the unique relationship between the mental incapacitation dimension of AS and DEP (e.g., Cox et al., 1999, 2001; Rector, Szacun-Shimizu, & Leybman, 2007; Schmidt, Lerew, & Joiner, 1998; Zinbarg, Brown, Barlow, & Rapee, 2001), it is noteworthy that some studies have also found significant associations between the other AS dimensions and DEP (ASI-S, Rodriguez et al., 2004; ASI-P, Grant, Beck, & Davila, 2007; ASI-S and ASI-P, McWilliams, Becker, Margrat, Clara, & Vriendt, 2007).

Although the aforementioned literature has been useful in establishing the relationship between AS and DEP, it has been limited by examining their relations almost exclusively in a cross-sectional fashion. Moreover, much of the extant literature on AS and DEP has relied on categorical measurement of DEP (e.g., McWilliams et al., 2007; Rodriguez et al., 2004), ignoring other important clinical information such as individual differences in symptom severity (cf. Brown & Barlow, 2005, 2009). Although the longitudinal relationship between AS and DEP severity has rarely been discussed, researchers have more generally hypothesized that higher levels of temperament/personality traits should be associated with a poorer prognosis of psychopathology (e.g., less decreases in Axis I symptoms over time and with treatment; Clark et al., 1994; Brown, 2007).

Studies of the longitudinal relationship between dimensions of AS and DEP have been sparse and produced mixed findings. For example, whereas Schmidt et al. (1998) found that fears of mental incapacitation predicted more severe levels of DEP over a five-week period, others have shown that fears of physical symptoms are associated with increased levels of DEP over a one-year period (Grant et al., 2007). More recently, Olatunji et al. (2008) found that none of the AS dimensions predicted the rate of reduction in DEP symptoms among 38 patients with generalized anxiety disorder receiving pharmacotherapy. Unfortunately, generalizability of these studies is limited due to use of non-clinical samples (Grant et al., 2007) and small clinical samples with limited diagnostic coverage (Olatunji et al., 2008; Schmidt et al., 1998). Additional longitudinal research is needed to further clarify how AS, particularly the fear of mental incapacitation, may influence the course of DEP in clinical samples.

1.3. Present Study

The present investigation aims to advance the extant literature on AS, BI, and DEP by examining their temporal course and interrelationships in a diverse sample of outpatients with anxiety and mood disorders. Test-retest correlations, effect size calculations, and latent growth curve modeling (LGM) were used to examine dimensions of AS, BI, and DEP over a two-year period. Because the majority of participants received treatment after the first assessment, all constructs were expected to decrease over the study period. However, unconditional LGMs were conducted to determine if the temporal course of AS was more similar to BI or DEP. In support of their discriminant validity, AS dimensions and BI were expected to be distinguishable at intake and the extent of change in all three AS dimensions was anticipated to be distinct from change in BI. Given conceptualizations of BI as a higher-order vulnerability, it was hypothesized that higher initial levels of BI would predict less reduction in all three AS dimensions, but that none of the AS dimensions would predict change in BI. Based on literature in support of a relationship between concerns of mental incapacitation and DEP, this AS dimension was expected to evidence stronger correlations with DEP at intake than the physical symptom or social dimensions of AS. Mental

incapacitation AS was also expected to predict the temporal course of DEP, such that greater concerns of incapacitation at intake would be associated with less decrease in DEP.

2. Method

2.1. Participants

The sample consisted of 606 outpatients who presented for assessment or treatment at the Center for Anxiety and Related Disorders. The sample was predominantly female (63%) and White (89%), with smaller percentages identifying as African American (4%), Asian (3%), and Latino/Hispanic (3%). The average age of the sample was 34.72 years ($SD = 11.89$) with a range from 18 to 74. Intake diagnoses (Time 1, T1) were established with the Anxiety Disorders Interview Schedule for *DSM-IV*: Lifetime Version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994), a semistructured interview designed to assess the fourth edition *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000) anxiety, mood, somatoform, and substance use disorders. Patients were reevaluated at 12 months (Time 2, T2) and 24 months (Time 3, T3) with the follow-up version of the ADIS-IV-L (ADIS-IV-FU), which is identical except that (a) past diagnoses are not assessed, and (b) a section is included to assess treatment follow-up (e.g., nature of treatments received since intake). When administering either version of the ADIS, clinicians assign diagnoses a clinical severity rating (CSR), ranging from 0 (not at all disturbing/disabling) to 8 (very disturbing/disabling), to represent the degree of distress or impairment in functioning associated with a specific diagnosis. Diagnoses with a CSR of 4 (definitely disturbing/disabling) or higher are considered to be at a clinical level (i.e., meeting the *DSM-IV* diagnostic threshold). Study exclusionary criteria were current suicidal/homicidal intent and/or plan, psychotic symptoms, or significant cognitive impairment (e.g., diagnosis of dementia, mental retardation). Rates of the most common current clinical disorders at intake in the current sample as assessed by the ADIS-IV-L were as follows: social phobia (42%), panic disorder with or without agoraphobia (38%), mood disorders (i.e., major depression, dysthymic disorder, depressive disorder not otherwise specified; 36%), generalized anxiety disorder (22%), specific phobia (20%), and obsessive-compulsive disorder (12%).

2.2. Growth Model Indicators

In addition to the ADIS-IV, patients completed a set of questionnaires that included measures of AS, DEP, and BI at each assessment point.

AS—The Anxiety Sensitivity Index (ASI; Reiss et al., 1986) was used as the indicator of AS. The ASI is a 16-item questionnaire designed to assess fears of physical symptoms, mental incapacitation, and social consequences associated with anxiety (Zinbarg et al., 1997). The ASI has a high degree of internal consistency (Reiss et al., 1986) and stable test-retest reliability over a three-year period (Maller & Reiss, 1992). At intake in our sample, ASI-Total Score $\alpha = .88$, ASI-P $\alpha = .88$, ASI-M $\alpha = .83$, and ASI-S $\alpha = .58$.

DEP—The Beck Depression Inventory (BDI; Beck & Steer, 1987) total score was used as the indicator of unipolar depression. Research has supported the BDI as a reliable ($\alpha = .98$ at intake in the present sample) and valid assessment of DEP symptoms (for a review, see Beck, Steer, & Garbin, 1988).

BI—The Behavioral Inhibition Scale (BIS) of the BIS/BAS (Carver & White, 1994) was used as the indicator of BI. The BIS/BAS is a 20-item self-report measure designed to assess individual differences in sensitivity to reward or behavioral activation (BAS; 13 items) and punishment or behavioral inhibition (BIS; 7 items). The BIS scale has strong internal consistency ($\alpha = .74$ at intake in the present sample) and has demonstrated strong

convergent, discriminant, and predictive validity, including significant correlations with other higher-order temperament/personality constructs (e.g., neuroticism, trait anxiety; Carver & White, 1994; Campbell-Sills, Liverant, & Brown, 2004).

2.3. Data analysis

Raw data were analyzed using a latent variable software program (Mplus 5.2; Muthen & Muthen, 1998–2008). Attrition was low (7%) at T2 but increased to 50% at T3. Missing data were accommodated in all models by using direct maximum likelihood estimation. Model fit was evaluated using the root mean square error of approximation (RMSEA) and its 90% confidence interval (CI) and test of close fit (CFit), the Tucker–Lewis index (TLI), the comparative fit index (CFI), and the standardized root mean square residual (SRMR). Acceptable model fit values were defined as: RMSEA values close to .06 or below (90% CI upper limit close to $\leq .06$, non-significant CFit), CFI and TLI values close to .95 or above, and SRMR values close to .08 or below (Hu & Bentler, 1999). The acceptability of the models was further evaluated by the presence or absence of salient localized areas of strains in the solutions (e.g., modification indices), and the strength and interpretability of the parameter estimates.

3. Results

3.1. Rates of Depression at Intake and Follow-Up

The majority of the sample received treatment (92% in total, 76% at CARD, 16% elsewhere) within the first year following the intake assessment. As reported by Brown (2007), the overall rate of anxiety and mood disorders in the sample declined markedly at the 12-month follow-up (i.e., from 100% to 64%; McNemar test $p < .001$), with negligible additional change at the 24-month assessment (58%, $p = .80$). Rates of depressive disorders (major depression, dysthymia, depression not otherwise specified) at intake, 12 months, and 24 months were 36%, 20%, and 24%, respectively.

3.2. Temporal Course of AS, BI, and DEP

As expected, all constructs demonstrated change over the two-year study period (see Table 1). Test-retest correlations did not differ appreciably from one another (e.g., T1-T3 BDI, BIS, ASI-P, ASI-M, ASI-S r s = .54, .58, .54, .49, .53, respectively) and effect size estimates (i.e., magnitude of change over the two year study period) were all in the medium range (e.g., T1-T3 BDI, BIS, ASI-P, ASI-M, ASI-S d s = .55, .49, .73, .73, .63, respectively). Effect sizes for T1-T2 (BDI, BIS, ASI-P, ASI-M, ASI-S d s = .47, .42, .69, .64, .65, respectively) were substantially larger than those for T2-T3 (BDI, BIS, ASI-P, ASI-M, ASI-S d s = .06, .09, .05, .08, .00, respectively), consistent with expectation as 92% of patients received treatment during the first half of the study period.

3.3. Unconditional Latent Growth Models

In order to evaluate individual differences in the temporal course of AS, DEP, and BI, separate unconditional LGMs were specified for each ASI dimension, BDI, and BIS. As the majority of change in each of the constructs occurred during the first year (i.e., the interval where most patients underwent treatment), linear change across the two-year period was not viable. Thus, the slope factor loadings for T1 and T3 were fixed to 0 and 1 respectively, and the T2 loading was freely estimated to allow for a nonlinear trajectory for each construct (cf. Brown, 2007). As shown in Table 1, all unconditional models fit the data well and significant individual differences in trajectories were evident for each construct (e.g., all slope variances were significant). Consistent with prediction, slope means were all negative and significant, indicating that on average, patients evidenced significant decreases in each

construct over the two-year follow-up period. Inspection of the T2 factor loadings, also presented in Table 1, confirms that the greatest proportion of change occurred over the first year of follow-up (e.g., 96% of the total reduction in ASI-P occurred between T1 and T2).

The longitudinal course of AS, BI, and DEP was further evaluated by examining the intercept-slope correlations estimated by the unconditional LGMs, which indicate how individual differences in initial levels of the construct relate to changes in the construct over time. Higher initial levels of BI were associated with less decreases in BI over time (BIS $r = .35$). In contrast, higher initial levels of each of the AS subscales and DEP were associated with greater reductions over time (ASI-P, ASI-M, ASI-S, and DEP r s = $-.48$, $-.63$, $-.25$, and $-.35$, respectively).

3.4. Parallel Process Growth Models of AS, BI, and DEP

To evaluate hypotheses about the discriminant validity of AS from BI, separate unconditional parallel-process growth models (i.e., AS and BI intercept and slope factors were specified to freely covary) were estimated for each ASI dimension (three models in total). Zero-order cross-sectional (i.e., intercept, T1) and longitudinal (i.e., slope) correlations of ASI dimensions with BIS and BDI are presented in Table 2. In support of the discriminant validity of the three AS dimensions from BI, cross-sectional and temporal correlations were generally in the low to moderate range (e.g., r s = $.17$ to $.42$, but ASI-S/BIS slope $r = .58$).

Predictions regarding directional relationships between BI, dimensions of AS, and DEP were evaluated by regressing latent slopes onto latent intercept (i.e., conditional parallel-process LGMs). In each model, the three slope factors were regressed onto the three latent intercepts (i.e., nine regressive paths estimated in total for each model; see Figure 1). All three models provided good fit to the data. As reported by a prior study using the present sample, initial levels of BI did not predict the course of DEP (for discussion of this result see Brown, 2007). Consistent with hypotheses regarding the hierarchical conceptualization of AS as lower-order to BI, individual differences in initial levels of BIS were associated with the extent of change in all ASI dimensions in the anticipated direction (e.g., higher BIS at T1 predicted less decreases in ASI-P, ASI-M, and ASI-S, γ s = $.28$, $.16$, $.35$; p s < $.05$), while initial levels of AS were not significantly associated with change in BI.

Hypotheses for fears of mental incapacitation and DEP were partially supported. As expected, the correlation between BDI and ASI-M intercepts (i.e., T1 correlations) was significantly greater than those between BDI and ASI-P or BDI and ASI-S ($p < .01$ and $p < .05$, respectively) using the z -test procedures for dependent correlations presented in Meng, Rosenthal, and Rubin (1992). Additionally, individual differences in initial levels of ASI-M significantly predicted the rate of change in BDI over the two-year study period. However, the inverse nature of the path between ASI-M intercept and BDI slope was unexpected ($\gamma = -.22$; $p < .05$). Controlling for initial levels of DEP and BI, individuals endorsing stronger fears of mental incapacitation at T1 exhibited *greater* decreases in DEP over time. Initial levels of ASI-P and ASI-S were not associated with changes in BDI. Unexpectedly, individual differences in initial levels of BDI predicted degree of change in ASI-M ($\gamma = .26$, $p < .001$), such that greater DEP severity at T1 was associated with less decreases in mental incapacitation concerns over time.

4. Discussion

As expected, levels of AS, BI, and DEP declined over the two-year study period. These findings are consistent with studies showing decreases in AS and other temperament/personality traits over the course of psychological and pharmacological treatments (e.g.,

Brown, 2007; Clark, Vittengl, Kraft, & Jarrett, 2003; Otto et al., 1995; Smits et al., 2008; Tang, DeRubeis, Hollon, Amsterdam, Shelton, & Schalet, 2009). Similar to its higher-order counterparts, self-report measurement of AS likely consists of variance due to true “trait” vulnerability as well as variance due to “state” distress associated with experiencing clinically significant symptoms (i.e., mood-state distortion). In other words, the self-report measurement of AS is likely augmented by the presence of anxiety and depression symptoms (i.e., increased worry about autonomic arousal or concentration difficulties in the presence of such symptoms). Although the present study was unable to explicate the relative course of “trait” versus “state” AS, both may be malleable through psychosocial interventions (e.g., psychoeducation on the nature of emotional disorder symptoms).

The current study is the first to examine the temporal course of AS using LGM. Whereas stability of BI increased as a function of initial severity (i.e., positive intercept-slope correlation in the unconditional LGM), AS dimensions displayed a longitudinal course more similar to DEP (i.e., negative intercept-slope correlations).¹ In other words, individuals endorsing stronger fears of physical symptoms, mental incapacitation, and social evaluation at intake exhibited greater decreases in these fears over time. Findings for AS are in line with results often obtained in LGM examinations of *DSM* disorder dimensions (e.g., DEP in the current study; Curran et al., 1997; Brown, 2007). Although AS is conceptualized as a vulnerability to psychopathology (e.g., Reiss, 1987), this finding is consistent with the fact that AS dimensions resemble criteria of *DSM* anxiety and depressive disorders (e.g., fear of autonomic arousal and the A2 criterion of panic disorder, fear of mental incapacitation and distress criterion of depression). Collectively this implies that, in treatment-seeking clinical samples, the temporal course of AS more closely resembles *DSM* disorders dimensions than higher-order temperament/ personality vulnerabilities.

As hypothesized, parallel-process LGMs supported the discriminant validity of AS from BI by estimating zero-order cross-sectional and temporal correlations generally in the low to moderate range. These findings are in line with prior research which has found AS to be distinct from other higher-order temperament/personality vulnerabilities (e.g., Taylor, 1995; Zinbarg & Barlow, 1996; Lilienfeld, 1997). The current study further supports the discriminant validity of AS by being the first to show that the cross-sectional and temporal variability in AS dimensions is largely distinct from BI. Hypotheses were also supported when directional paths were added to the LGMs. Consistent with conceptualizations of AS as a lower-order temperament/personality construct relative to BI (e.g., Lilienfeld et al., 1993; Clark et al., 1994; Taylor, 1995), higher initial levels of BI were associated with less change in all AS dimensions over the two-year study period. In other words, holding initial levels of AS and DEP constant, individuals with a greater tendency to experience negative emotions in response to stress (i.e., higher BI) exhibited less improvement in fears of physical symptoms, social embarrassment, and mental incapacitation over time and with treatment.

Although AS dimensions may serve as vulnerability prior to onset of psychopathology, perhaps they function more like a disorder symptom after onset (e.g., temporal course of AS was similar to *DSM* disorder constructs in the unconditional LGMs). Along these lines, directional paths from BI to AS dimensions may have important implications regarding the course and treatment response for features of certain anxiety and depressive disorders. The current study is the first of our knowledge to demonstrate that higher initial levels of BI are associated with more stable fears of anxiety symptoms and concerns over mental

¹The course of general AS was also examined with test-retest correlations and an unconditional LGM using ASI total score. Consistent with analyses of the three AS dimensions, correlations did not appreciably differ from BI or DEP and the temporal stability decreased as a function of initial severity.

incapacitation. In contrast, the finding that higher initial levels of BI predicted less reduction in social concerns (e.g., ASI-S) is in line with Brown (2007), which demonstrated this relationship using measures of social anxiety in the same sample. Interestingly, the social concerns dimension of AS (i.e., intercept and slope) demonstrated stronger correlations with BI (i.e., intercept) than the physical or mental incapacitation dimensions. Although this finding may reflect the robust relationship between BI, shyness, and social anxiety that has been demonstrated elsewhere (e.g., Biederman et al., 2001; Kagan, 1987), drawing firm connections with this literature should be cautioned because of conceptual differences with Gray's (1982) theory (i.e., in defining and measuring BI).

Results of these LGMs indicate that high initial BI may predict poor outcomes among individuals afraid of autonomic arousal (i.e., panic disorder), concerned with embarrassment (i.e., social phobia), and fearful that nervousness and concentration difficulties may be indicative of severe impairment or incapacitation (i.e., depression, Taylor et al., 1996; most anxiety disorders, Zinbarg et al., 1997). The paths between BI and the AS dimensions may also suggest that interventions targeting BI (in addition to AS) could lead to greater reductions in AS. In other words, in addition to directly targeting AS through tailored psychoeducation (about panic/anxiety) and exposures (e.g., interoceptive and situational), general psychoeducation about emotions and emotional responding (e.g., different components of emotional responses, natural course of emotions, etc.) and non-specific emotion exposures (e.g., conducting exposure outside of the primary feared situation/emotion) may improve outcomes for AS dimensions. This interpretation is consistent with recently developed transdiagnostic treatment approaches, such as the Unified Protocol (UP; Barlow, Ellard, Fairholme, Farchione, Boisseau, Allen, & Ehrenreich-May, 2011). The UP is designed to target higher-order vulnerabilities (e.g., BI) as well as disorder-specific features (e.g., AS dimensions) through a broad focus on identifying and correcting maladaptive patterns of emotional responding using traditional cognitive-behavioral techniques (Fairholme, Boisseau, Ellard, Ehrenreich, & Barlow, 2010).

Consistent with prior research (e.g., Schmidt et al., 1998; Taylor et al., 1996), parallel-process LGMs produced a significant relationship between the mental incapacitation dimension of AS and DEP (e.g., strongest correlations were between the mental incapacitation dimension of AS and DEP; significant paths from intercept to slope factors). However, the direction of the paths between mental incapacitation AS and DEP were counter to expectation. Whereas the negative regression coefficient from mental incapacitation AS intercept to DEP slope indicated that greater fears of incapacitation at T1 were associated with *more* change in DEP, the positive coefficient from DEP intercept to mental incapacitation slope suggests that more severe DEP at T1 was associated with less change in, or more stable fears of mental incapacitation. Perhaps fears of mental incapacitation motivate patients to be more compliant with treatment interventions (e.g., medication compliance, session attendance, completing homework) in an attempt to prevent incapacitation from occurring. Along these lines, the treatment outcome literature has consistently documented that treatment compliance is a significant predictor of DEP treatment response (e.g., Cowan et al., 2007; Persons, Burns, & Perloff, 1988). It is also possible that individuals whose DEP symptoms are maintained/exacerbated by mental incapacitation AS (e.g., Cox et al., 1999, 2001; Taylor et al., 1996) may show decreases in DEP when they obtain logical understanding of their symptoms over time and with treatment (e.g., nervousness does not necessarily indicate incapacitation). This interpretation is consistent with research demonstrating that individuals distressed about being depressed (e.g., “depression about depression”) respond more rapidly to cognitive treatment because obtaining an understanding of the cognitive model of DEP helped alleviate symptoms being maintained by such distress (Fennell & Teasdale, 1988). Conversely, the significant positive path observed between the DEP intercept and mental incapacitation concerns slope may

imply that individuals with more severe DEP are more likely to experience severe impairment from nervousness and concentration difficulties, thus reinforcing and maintaining concerns about mental incapacitation.

Despite strengths of our sample (e.g., large clinical sample) and methodology (e.g., first examination of AS using LGM), the present study has some limitations. Although our analyses examined the longitudinal course and interrelationships of dimensions of AS, BI, and DEP, our study design precluded more detailed evaluations of the relationships between these constructs. For instance, although pathoplastic relations between BI, AS and DEP (e.g., how AS dimensions influence the course of DEP) were examined using LGM, our study was unable to test for other temperament/personality-psychopathology associations (e.g., predispositional, complication/scar). In order to examine these types of associations, more detailed prospective studies must be conducted (e.g., measuring BI and AS prior to, during, and after the onset of psychopathology).

Nonetheless, the present study adds to the literature by being the first to evaluate the temporal course of AS and its longitudinal relationships with BI and DEP in a large clinical sample. Our findings underscore the importance of BI in predicting AS-related outcomes and highlight interesting longitudinal relationships between mental incapacitation AS and DEP. Future research should replicate and extend our study findings. For instance, although the temporal course of AS was more similar to DEP than BI in our sample of outpatients (e.g., stability decreased as a function of initial severity), perhaps AS functions like BI in non-clinical samples. It would also be interesting to evaluate the relative influence of different treatments on the course of AS (e.g., does CBT or pharmacotherapy lead to greater reductions in AS?). Moreover, although higher initial levels of mental incapacitation concerns predicted greater improvements in DEP, additional research is needed to clarify if mental incapacitation AS promotes decreases in DEP because of increased treatment motivation or because DEP symptoms maintained by such fears are addressed with time and treatment. Finally, future research should evaluate the temporal relationships between AS dimensions and other emotional disorders with which they have been linked, such as post traumatic stress disorder and hypochondriasis (e.g., Lang, Kennedy, & Stein, 2002).

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Figure 1. Parallel process models of dimensions of behavioral inhibition, anxiety sensitivity and depression

Note. Only significant paths and residual correlations and disturbances are shown.

Completely standardized and unstandardized (in parentheses) estimates are presented. A: Anxiety Sensitivity – Social Concerns (ASI-S). B: Anxiety Sensitivity – Mental Concerns (ASI-M). C: Anxiety Sensitivity – Physical Concerns (ASI-P). Behavioral Inhibition Scale (BIS). Beck Depression Inventory (BDI). Overall fit of models: Model A, $\chi^2(18) = 16.74$, $p = .541$, standardized root-mean-square residual (SRMR) = .03, root-mean-square error of approximation (RMSEA) = 0.00 (90% confidence interval [CI] = .00–0.03, test of close fit [CFit] = .99), Tucker-Lewis Index (TLI) = 1.00, comparative fit index (CFI) = 1.00; Model B, $\chi^2(18) = 17.83$, $p = .467$, SRMR = .03, RMSEA = 0.00 (90% CI = .00–0.04, CFit = .99), TLI = 1.00, CFI = 1.00; Model C, $\chi^2(18) = 16.46$, $p = .561$, SRMR = .03, RMSEA = 0.00 (90% CI = .00–0.03, CFit = .99), TLI = 1.00, CFI = 1.00. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1
Temporal Variation and Change in Anxiety Sensitivity, Depression, and Behavioral Inhibition

Parameter Estimate	ASI-P	ASI-M	ASI-S	BDI	BIS
Test-Retest Correlations					
T1-T2	.52	.52	.53	.59	.61
T2-T3	.75	.68	.72	.71	.71
T1-T3	.54	.49	.53	.54	.58
Effect Size Estimates (Cohen's <i>d</i>)					
T1-T2	.69	.64	.65	.47	.42
T2-T3	.05	.08	.00	.06	.09
T1-T3	.73	.73	.63	.55	.49
Growth Intercept					
Mean	15.52***	6.24***	8.56***	14.44***	24.42***
Variance	51.62***	15.31***	7.03***	65.65***	6.60***
Growth Slope					
Mean	-5.72***	-2.88***	-2.16***	-4.97***	-1.89***
Variance	31.77***	8.59***	4.05***	28.86***	2.57*
T2 Slope Loading	.96	.93	.97	.90	.78
Intercept-Slope					
Covariance	-19.31***	-7.19***	-1.34*	-15.16**	1.45*
Correlation	-.48	-.63	-.25	-.35	.35

Note. ASI-P = Anxiety Sensitivity Index – Physical Concerns; ASI-M = Anxiety Sensitivity Index – Mental Concerns; ASI-S = Anxiety Sensitivity Index – Social Concerns; DEP = Beck Depression Inventory; BIS = Behavioral Inhibition Scale. Overall fit of unconditional growth models: ASI-P, $\chi^2(2) = 2.07$, $p = .356$, standardized root-mean-square residual (SRMR) = .03, root-mean-square error of approximation (RMSEA) = 0.01 (90% confidence interval [CI] = 0.00–0.08, test of close fit [CFit] = .74), Tucker–Lewis index (TLI) = 1.00, comparative fit index (CFI) = 1.00; ASI-M, $\chi^2(2) = 4.25$, $p = .120$, SRMR = .05, RMSEA = 0.04 (90% CI = 0.00–0.10, CFit = .48), TLI = 0.99, CFI = 0.99; ASI-S, $\chi^2(2) = 1.25$, $p = .536$, SRMR = .03, RMSEA = 0.00 (90% CI = 0.00–0.07, CFit = .85), TLI = 1.00, CFI = 1.00; DEP, $\chi^2(2) = 2.99$, $p = .224$, SRMR = .04, RMSEA = 0.03 (90% CI = 0.00–0.09, CFit = .63), TLI = 1.00, CFI = 1.00; BI, $\chi^2(2) = 1.32$, $p = .517$, SRMR = .02, RMSEA = 0.00 (90% CI = 0.00–0.07, CFit = .84), TLI = 1.00, CFI = 1.00. Time 1 (T1) = intake; Time 2 (T2) = 12-month follow-up; Time 3 (T3) = 24-month follow-up.

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 2
Zero-Order Temporal Correlations of Anxiety Sensitivity, Depression, and Behavioral Inhibition

Construct	BDI _{INT}	BIS _{INT}	BDI _{SLP}	BIS _{SLP}
ASI-P _{INT}	.36	.23	-.22	.08
ASI-M _{INT}	.52	.34	-.33	.04
ASI-S _{INT}	.42	.42	-.22	-.02
ASI-P _{SLP}	-.05	.16	.51	.17
ASI-M _{SLP}	-.10	-.02	.56	.23
ASI-S _{SLP}	.01	.19	.43	.58

Note. BDI = Beck Depression Inventory; BIS = Behavioral Inhibition Scale; ASI-P = Anxiety Sensitivity Index – Physical Concerns; ASI-M = Anxiety Sensitivity Index – Mental Concerns; ASI-S = Anxiety Sensitivity Index – Social Concerns; INT = intercept; SLP = slope.