Testing a hierarchical model of neuroticism and its cognitive facets: Latent structure and prospective prediction of first onsets of anxiety and unipolar mood disorders over three years in late adolescence

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Abstract:

Neuroticism and several other traits have been proposed to confer vulnerability for unipolar mood disorders (UMDs) and anxiety disorders (ADs). However, it is unclear whether the associations of these vulnerabilities with these disorders are attributable to a latent variable common to all vulnerabilities, more narrow latent variables, or both. In addition, some researchers have suggested that neuroticism predicts UMDs, ADs, and substance use disorders (SUDs) with comparable strength, whereas other researchers have hypothesized that neuroticism is more strongly related to UMDs and ADs. We tested hypotheses about the factor structure of several vulnerabilities and the prospective associations of these latent variables with initial onsets of UMDs, ADs, and SUDs during a 3-year period in 547 participants recruited as high school juniors. Although a general neuroticism factor predicted SUDs, it predicted UMDs and ADs more strongly and especially predicted comorbid UMDs and ADs. There was also mixed support for specific associations involving more narrow latent vulnerabilities.

Keywords: neuroticism | cognitive vulnerability | anxiety disorders | mood disorders | substance use disorders

Article:

Neuroticism (N) has been proposed as a common vulnerability for anxiety disorders (ADs) and unipolar mood disorders (UMDs; e.g., Eysenck, 1967; Gray & McNaughton, 2000). Other personality traits and cognitive-style variables have been hypothesized to be more narrow

vulnerabilities for either ADs or UMDs. For example, several cognitive-style variables have been proposed to be vulnerability factors for UMDs (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1983; Blatt & Zuroff, 1992; Clark & Beck, 1999). These include dysfunctional attitudes (including the need for approval and the need for achievement), negative inferential style (the tendency to interpret negative life events as having stable and global causes that lead to negative consequences), sociotropy (having heightened needs for support and acceptance), and autonomy (being excessively concerned with achievement issues and being highly self-critical). In contrast to dysfunctional attitudes, negative inferential style, sociotropy, and autonomy, anxiety sensitivity (AS)—the belief that anxiety and physical sensations of anxiety are harmful—has been hypothesized to be a risk factor for ADs in general and panic disorder (PD) in particular (Reiss & McNally, 1985).

It is already known that most of these hypothesized vulnerability factors do prospectively predict depressed mood and initial onsets of major depression (e.g., Alloy et al., 2006; Kendler, Kuhn, & Prescott, 2004; Klein, Durbin, & Shankman, 2009; Lewinsohn, Joiner, & Rohde, 2001). We also know that one or more facets of AS prospectively predict the onset of panic attacks (e.g., Hayward, Killen, Kraemer, & Taylor, 2000; Schmidt, Lerew, & Jackson, 1997, 1999), worry about panic (Schmidt, 1999), and ADs considered as a group (Schmidt, Zvolensky, & Maner, 2006). In addition, it is already established that AS has unique associations with anxiety symptoms above and beyond measures of broader constructs, such as N (e.g., Eke & McNally, 1996; Rapee & Medoro, 1994; Schmidt et al., 1999).

There are, however, many unanswered questions about N, dysfunctional attitudes, negative inferential style, sociotropy, autonomy, and AS and their prospective associations with psychopathology. For example, does the cognitive vulnerability conferred by elevations on dysfunctional attitudes, negative inferential style, sociotropy, or autonomy predict UMDs significantly more strongly than ADs? Similarly, does AS predict ADs significantly more strongly than UMDs? And does N prospectively predict initial onsets of ADs other than posttraumatic stress disorder (e.g., Breslau & Schultz, 2013)?1

There is also theoretical disagreement regarding the nature of N and its associations with psychopathology. Within Gray's reinforcement-sensitivity theory (RST; e.g., Gray & McNaughton, 2000), N is hypothesized to be specifically associated with sensitivity to cues for punishment, frustrative nonreward, and conflict (but not to cues for reward and relieving nonpunishment). For this reason, RST predicts that N should be more strongly associated with internalizing than with externalizing psychopathology, with the latter involving a stronger contribution from reward circuits (e.g., Gray & McNaughton, 2000; Zinbarg & Yoon, 2008). By contrast, after reviewing evidence of cross-sectional associations with many forms of psychopathology, including substance use disorders (SUDs), Claridge and Davis (2001) concluded that N "is such a universal accompaniment of abnormal functioning (both psychological and biological) that by itself it has little descriptive or explanatory value" (p. 383). However, whereas it is clear that N predicts SUDs (e.g., Sher, Grekin, & Williams, 2005), an unanswered question is whether N predicts UMDs or ADs more strongly than SUDs (or other forms of externalizing psychopathology).

Cognitive-vulnerability theorists have rarely considered the possibility that cognitive-risk variables might be facets of N. Thus, a hypothesis that appears to be implicitly incorporated into many of their theories is that cognitive-risk variables are either unrelated to N or have prospective effects beyond those of N. By contrast, at least some N theorists have explicitly incorporated cognitive constructs into their definitions of N. For example, Lilienfeld, Turner, and Jacob (1993)

proposed that AS is a facet of N. Similarly, Costa and McCrae (1992) considered irrational ideas to be a facet of N. In addition, Eysenck and Eysenck (1985) considered low self-esteem to be a facet of N, and Scheier, Carver, and Bridges (1994) noted that pessimism often has been hypothesized to be a facet of N.

Consistent with the hypotheses that cognitive constructs similar to dysfunctional attitudes, sociotropy, autonomy, and negative inferential style are facets of N, research has demonstrated that these cognitive constructs often show strong associations with N (e.g., Bagby et al., 2001; Dunkley, Blankstein, & Flett, 1997). Furthermore, negative inferential style, dysfunctional attitudes, sociotropy, or autonomy are associated with at least certain ADs (e.g., Mineka, Pury, & Luten, 1995), and some aspects of AS are associated with depression (e.g., Zinbarg, Brown, Barlow, & Rapee, 2001). The nonspecificity of these associations and correlations of these vulnerabilities with N suggests that the general N factor (GNF), at least in part, accounts for the associations between these cognitive-risk variables and psychopathology. Indeed, some cross-sectional evidence failed to show unique associations between ADs and UMDs with negative inferential style, dysfunctional attitudes, sociotropy, or autonomy above and beyond N (Zinbarg et al., 2010). Thus, whether dysfunctional attitudes, sociotropy, autonomy, and negative inferential style have unique and specific predictive effects beyond the GNF is also an open question.

Other gaps in theoretical understanding in this area stem from the fact that the hierarchical structure of the vulnerability factors that we are focused on is likely quite complex and difficult to fully account for or comprehend. For example, a number of factor analytic studies have suggested that the structure of AS is hierarchical with three group factors (i.e., factors common to some but not all items) and a general factor (e.g., Stewart, Taylor, & Baker, 1997; Zinbarg, Barlow, & Brown, 1997). Moreover, AS is thought to be embedded within a larger hierarchical structure along with N (Lilienfeld et al., 1993). Thus, it is unclear whether the unique effects of AS that have been demonstrated should be attributed to one (or more) of the AS group factors, the general AS factor, or to factors at both levels of the AS hierarchy. It is also unclear whether the general factor common to all AS items might, in fact, be the GNF. Similarly, whether dysfunctional attitudes and negative inferential style share an additional common factor that is not the GNF is an open question. The practice of defining cognitive risk for depression on the basis of elevations on both dysfunctional attitudes and negative inferential style (e.g., Alloy et al., 2000, 2006) is equivalent to defining risk on the basis of a composite of dysfunctional attitudes and negative inferential style. This practice assumes that dysfunctional attitudes and negative inferential style share a factor, and interpretation of the results in terms of cognitive risk for depression (rather than N) implies that this common factor is distinguishable from the GNF. Unfortunately, this assumption has not been previously tested.

Another unresolved theoretical question regarding overlap among the personality and cognitive-risk factors included in this study stems from the substantial overlap of Sociotropy and Autonomy scales with the Needing Approval and Needing Achievement subscales of the Dysfunctional Attitudes Scale–Form A (DAS-A; e.g., Dunkley, Sanislow, Grilo, & McGlashan, 2004; Zuroff, 1994). Indeed, it has been suggested that Sociotropy and Needing Approval were likely indicators of one construct, whereas Autonomy and Needing Achievement were likely indicators of a second construct (e.g., Dunkley et al., 1997; Ouimette, Klein, Anderson, Riso, & Lizardi, 1994). However, to our knowledge, this hypothesis has not yet been tested.

Gender is another variable related to risk for internalizing disorders with females at greater risk for UMDs (e.g., Nolen-Hoeksema & Hilt, 2009) and many ADs (e.g., Craske, 2003). Females also score higher than males on N (e.g., Costa, Terracciano, & McCrae, 2001), including in the

present sample (Zinbarg et al., 2010), as well as on the cognitive vulnerabilities (e.g., Hankin & Abramson, 2001). There is also some evidence that suggests that gender moderates the associations between N and the emotional disorders. For example, N has been shown to be significantly more strongly related cross-sectionally to major depression in males than in females (Fanous, Gardner, Prescott, Cancro, & Kendler, 2002). Similarly, in the present sample, N was shown to be significantly more strongly related to past diagnoses of UMDs and major depressive disorder (MDD) in males (Zinbarg et al., 2010). Unfortunately, it is unclear whether gender moderates the prospective association between N and MDD (Kendler et al., 2004). Notably, the interpretability of gender differences on N has been questioned on the grounds that N scales may not be invariant across men and women (e.g., Reise, Smith, & Furr, 2001). Indeed, if the factor structure of our risk measures is not highly similar for men and women, the measures would be tapping different constructs for men and women and, therefore, tests of gender moderation of vulnerability associations with emotional disorders could not be interpreted in a straightforward manner.

Our first aim in the present study was to test several hypotheses regarding the prospective associations between the latent variables tapped by our hypothesized risk measures and initial onsets of ADs, UMDs, and SUDs. On the basis of RFT (e.g., Gray & McNaughton, 2000) and cross-sectional evidence (e.g., Claridge & Davis, 2001), we hypothesized that N is a common risk factor for ADs and UMDs. For this reason, N should predict ADs in addition to UMDs and should be an especially strong predictor of comorbid ADs and UMDs. We also pitted against each other two contrasting hypotheses regarding the associations between N and psychopathology. According to RST (e.g., Gray & McNaughton, 2000), N should prospectively predict UMDs and ADs more strongly than SUDs. In contrast to this RST hypothesis, the Claridge and Davis (2001) perspective hypothesizes that N should predict SUDs as strongly as UMDs and ADs. On the basis of theory (e.g., Abramson et al., 1989; Beck, 1967) and existing prospective evidence (e.g., Alloy et al., 2006), we hypothesized that dysfunctional attitudes and negative inferential style predict UMDs and do so more strongly than ADs. On the basis of AS theory (e.g., Reiss & McNally, 1985) and earlier prospective evidence (e.g., Hayward et al., 2000), we hypothesized that one or more AS factors predict ADs and do so more strongly than UMDs. Finally, on the basis of past cross-sectional (Fanous et al., 2002) and retrospective evidence (Zinbarg et al., 2010), we hypothesized that N is a stronger predictor of UMDs in males than in females.

A second aim was to test hypotheses regarding the overlap among the risk factors included here. Thus, following the practice of defining cognitive risk on the basis of elevations on both dysfunctional attitudes and negative inferential style (e.g., Alloy et al., 2006), we hypothesized that dysfunctional attitudes and negative inferential style share a common factor beyond the GNF. On the basis of existing theory (e.g., Lilienfeld et al., 1993) and past factor analyses (e.g., Zinbarg et al., 1997), we also hypothesized that the hierarchical structure of N includes an intermediate-breadth AS factor in addition to the general (broad) N factor and three AS group (narrow) factors. Considering past correlational evidence (e.g., Dunkley et al., 1997), we hypothesized that Sociotropy and Needing Approval are indicators of one construct, whereas Autonomy and Needing Achievement are indicators of a second construct. Finally, we tested the hypothesis suggested by Reise et al. (2001) that the factor structure of measures of N and its cognitive facets differs meaningfully between males and females.

Testing these predictions has important implications not only for theory but also for preventive interventions. Different preventive interventions may be called for depending on which hypotheses are supported by the data. For example, if only the GNF has unique predictive power for both UMDs and ADs, then those individuals at risk might benefit most from broad-based

preventive interventions for general emotional regulation. By contrast, if only specific risk factors for different disorders have unique predictive power, then more narrowly targeted preventive intervention strategies for specific risk factors might be most valuable. One example of more narrowly targeted prevention programs is those that target AS to reduce risk for PD (e.g., Gardenswartz & Craske, 2001).

Method

Participants and screening procedures

Participants (n = 547) were recruited into the Northwestern–UCLA Youth Emotion Project study from the 11th grade of two ethnically and socioeconomically highly diverse high schools: one in suburban Chicago and the other in suburban Los Angeles. Given that many UMDs, ADs, and SUDs have their first onset during late adolescence (e.g., Kessler, Bergland, Demler, Jin, & Walters, 2005) and that this age range involves changing life roles, this is a useful age range in which to study the onset and course of UMDs, ADs, and SUDs (Prenoveau et al., 2011). Eleventh-grade students who provided assent and parental consent completed a screening questionnaire—a 22-item version of the N scale of the Eysenck Personality Questionnaire–Revised (EPQ-R N; Eysenck & Eysenck, 1975). Students were categorized as low, medium, and high scorers on the EPQ-R N, and when we invited participants into the longitudinal study, we oversampled those individuals classified as high scorers and maintained equal proportions of females to males across the three EPQ-R N categories. There were 627 students who completed the baseline assessment, which included an assessment of lifetime Axis I psychopathology using the nonpatient edition of the Structured Clinical Interview for DSM–IV–TR Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2002).

Participants with low, medium, and high scores on the EPQ-R N represented 18.4%, 23.0%, and 58.6% of the sample, respectively. The sample was 68.7% female and 31.3% male. Participants identified themselves as 48.6% Caucasian, 15.3% Latino, 12.4% African American, 5.2% “other,” 4.5% Asian, 0.7% Pacific Islander, and 13.2% as having more than one race or ethnicity. Participants had a mean age of 16.9 years (SD = 0.4) at the time of their first interview.2 These participants, or subsets of them, have been used in a number of previous publications that tested different hypotheses than those tested here (i.e., Adam et al., 2010; Adam et al., 2014; Craske et al., 2009; Craske et al., 2012; DeSantis et al., 2007; Griffith et al., 2009; Griffith et al., 2010; Hauner et al., 2008; Lewis et al., 2010; Mor et al., 2010; Prenoveau et al., 2009; Prenoveau et al., 2010; Prenoveau et al., 2011; Sumner et al., 2011; Sumner, Mineka, Adam, et al., 2014; Sumner, Mineka, Zinbarg, et al., 2014; Sumner, Vrshek-Schallhorn, et al., 2014; Sutton et al., 2011; Uliaszek et al., 2009; Uliaszek et al., 2010; Uliaszek et al., 2012; Vrshek-Schallhorn, Czarlinski, Mineka, Zinbarg, & Craske, 2011; Vrshek-Schallhorn et al., 2013; Vrshek-Schallhorn et al., 2014; Waters et al., 2014; Wolitzky-Taylor, Bobova, Zinbarg, Mineka, & Craske, 2012; Wolitzky-Taylor et al., 2014; Zinbarg et al., 2010).

Measures

Diagnostic measure The SCID (First et al., 2002) was used to assess for Diagnostic and Statistical Manual of Mental Disorders (DSM–IV, 4th ed.; American Psychiatric Association, 1994) psychiatric diagnoses. Interviews were conducted at the baseline assessment and then every 10 to

18 months during the subsequent 3 years. All interviewers had at least a bachelor's degree and underwent extensive training and supervision, and interviewers presented each completed SCID at a diagnostic-consensus meeting led by a doctoral-level supervisor.

Reliability for diagnoses at baseline was assessed by having trained interviewers observe live SCIDs for 69 cases. Reliability for diagnoses at follow-up (FU) assessments was assessed by having trained interviewers listen to a random selection of audio-recorded SCIDs from both sites, including at least 10% of SCIDs for each time point at each site.

Table 1. Number of New Onsets of Each Diagnosis at Each Assessment Point

		Follow-up		
Diagnosis	**Baseline[a]**	**1 Year**	**2 Year**	**3 Year**
UMD	182	21	19	20
MDD	129	24	16	21
DYS	9	2	2	2
DDNOS	54	5	9	4
AD	138	19	18	7
PD	6	4	3	1
GAD	17	0	4	0
SAD	58	12	7	4
Spec	41	12	3	3
PTSD	5	0	3	1
ASD	3	0	0	1
OCD	17	2	5	0
ADNOS	31	10	6	5
Comorbid	78	15	15	10
SUD	12	4	9	11

Note: Any case with a particular diagnosis at a given time point, including at baseline, was censored from the subsequent time points of the survival analyses of that diagnosis to ensure that we were predicting initial onsets of each diagnosis. There are more cases of MDD than UMD at the 1-year and 3-year follow-ups because individuals with a diagnosis of a UMD other than MDD at an earlier assessment had their subsequent person-years excluded from the analyses of UMD but not MDD. Thus, for example, a case with a diagnosis of DDNOS at baseline and an initial diagnosis of MDD at the 1-year follow-up would have been included as a new onset of MDD but not of UMD at the 1-year follow-up. UMD = unipolar mood disorder; MDD = major depressive disorder; DYS = dysthymia; DDNOS = depressive disorder not otherwise specified; AD = anxiety disorder; PD = panic disorder; GAD = generalized anxiety disorder; SAD = social anxiety disorder; Spec = specific phobia; PTSD = posttraumatic stress disorder; ASD = acute stress disorder; OCD = obsessive-compulsive disorder; ADNOS = anxiety disorder not otherwise specified; Comorbid = comorbidity of at least one UMD and at least one AD; SUD = substance use disorder.

a. Baseline diagnoses were lifetime diagnoses and were not included in the survival analyses to ensure prospective prediction of initial onsets of each diagnosis.

Given the small number of participants meeting criteria for initial onsets of many of the individual diagnoses, we conducted our primary tests at the level of diagnostic spectra. By diagnostic spectra, we mean groups of disorders classified together in the DSM–IV: UMDs included MDD, dysthymia, and depressive disorder not otherwise specified; ADs included PD, generalized anxiety disorder, social anxiety disorder, obsessive-compulsive disorder, specific phobias, posttraumatic stress disorder, acute stress disorder, and AD not otherwise specified; and SUDs included alcohol abuse, alcohol dependence, nonalcohol substance abuse, and nonalcohol substance dependence. In addition, we conducted separate analyses of MDD, given the sufficiently

large subsample with initial onsets, and we conducted separate analyses of initial onsets of PD, given its strong theoretical link with AS. Table 1 shows the new onsets of each disorder during the course of the 3-year FU period.

When kappa values are interpreted, it is important to keep in mind that kappa is attenuated when the simple probabilities of the categories of a coding system deviate markedly from equiprobable (e.g., Bakeman, Quera, McArthur, & Robinson, 1997). Given marked deviation from equiprobable categories in the current study due to low base rates of many disorders, we followed the recommendations of Byrt, Bishop, and Carlin (1993) and Sim and Wright (2005) and report adjusted kappa that adjusts for the low base rates.3 Adjusted kappa for MDD equaled .91 at baseline, .94 at first FU, .92 at second FU, and .86 at third FU; for UMDs equaled .82 at baseline, .94 at first FU, .88 at second FU, and .90 at third FU; for ADs equaled .76 at baseline, .85 at first FU, .80 at second FU, and .76 at third FU; and for SUDs equaled .97 at baseline, 1.00 at first FU, .88 at second FU, and .83 at third FU. We did not have a sufficient number of cases of PD in the reliability subsamples at any time point to calculate kappa. Thus, overall, in the context of the low base rates, there was acceptable to very good interrater reliability.

Measures of N and facets of N At baseline, participants completed the following eight vulnerability questionnaires: (a) the EPQ-R N (Eysenck & Eysenck, 1975), (b) the N scale from the International Personality Item Pool NEO Personality Inventory–Revised (IPIP-NEO-PI-R N; Goldberg, 1999), (c) the Behavioral Inhibition Scale (BIS; Carver & White, 1994), (d) the N scale from the Big Five Mini-Markers Scale (Big5 N; Saucier, 1994), (e) the Cognitive Style Questionnaire (CSQ; Alloy et al., 2000; Hankin, Abramson, Miller, & Haeffel, 2004), (f) the DAS-A (Weissman & Beck, 1978), (g) the Personal Style Inventory (PSI; Robins et al., 1994), and (h) the Anxiety Sensitivity Index Expanded Form (ASI-X; Li & Zinbarg, 2007; Reiss, Peterson, Gursky, & McNally, 1986).

Assessment procedure

Participants were contacted by phone or e-mail 10 months after each SCID to schedule the subsequent SCID, and the interval between successive SCIDs was 10 to 18 months. Participants who could not be reached or were unable to complete a particular FU assessment in that time frame were contacted for the subsequent FU assessment; in all cases, the FU SCIDs covered the entire period since the last completed SCID. Participants were mailed a check after completion of each assessment. All study procedures were approved by institutional review boards at Northwestern University and the University of California–Los Angeles. Of the 627 participants who completed the baseline assessment, 496 (79.1%) completed the first FU, 420 (67.0%) completed the second FU, and 422 (67.3%) completed the third FU. Of the 627 participants who completed the baseline assessment, 547 (87.2%) completed at least one of the three FU assessments and were included in the present analyses, 474 (75.6%) completed at least two, and 319 (50.9%) completed all three.

Data analysis

All analyses were conducted using Mplus (Muthén & Muthén, 1998–2012). Missing data were accommodated using full-information maximum likelihood. The level of statistical significance (p) in all inferential analyses was less than or equal to .05 unless otherwise specified. There are different approaches one could take to teasing apart the unique effects of the cognitive-vulnerability factors and the GNF, including conventional multiple regression. However, because

we also tested predictions regarding the latent structure of the set of risk measures included in this study, we chose to use structural equation modeling. More specifically, we used the hierarchical factor model for its known strengths in separating common and unique variance sources (e.g., Chen, West, & Sousa, 2006; Reise, Morizot, & Hays, 2007; Zinbarg, Revelle, Yovel, & Li, 2005). Thus, we began by specifying a hierarchical confirmatory factor analysis (CFA) model of N in which dysfunctional attitudes, sociotropy, autonomy, negative inferential style, and AS are conceptualized as N facets.4 We then used this hierarchical CFA model to test hypotheses regarding the (1) overlap among the risk factors and (2) unique prospective associations of the risk factors with the initial onset of UMDs, ADs, and SUDs during a 3-year FU period. In addition, we tested the invariance of the CFA model of N across males and females and the role of gender in moderating the prospective associations of the GNF with initial onsets of UMDs, ADs, and SUDs.

Vulnerability measurement model We randomly selected one half of the sample with which to conduct preliminary analyses, including item-level exploratory factor analyses (EFAs) and initial subscale-level CFAs. To minimize capitalizing on sampling error that can arise from the use of EFA and modification indices in the model-specification process, we conducted the EFAs, used modification indices, and made other adjustments to models that had inadequate fit only in the first half of the sample. We then conducted confirmatory model testing in the second half of the sample (i.e., testing the models that were specified, in part, on the basis of the results in the first half of the sample). We also conducted analyses of metric and configural invariance between the two subsamples in our final model as a further test of the extent to which we capitalized on sampling error in the model-specification process.

The following fit indices were used to evaluate model fit in the CFAs: comparative fit index (CFI), root-mean-square error of approximation (RMSEA), and standardized root-mean residual (SRMR). Hu and Bentler (1998, 1999) recommended that good fit is indicated by CFI at or above .95, RMSEA at or below .06, and SRMR at or below .08. However, we were not rigid in our use of these cutoffs for two reasons. First, Hu and Bentler cautioned against interpreting their results as universal golden rules (see also Marsh, Hau, & Wen, 2004). Second, we used three indices (which is quite common) despite the absence of data on the performance of cutoffs if more than a pair of indices are used. In addition, although there are no cutoffs for the Bayesian information criterion (BIC), it has been shown to be useful for model comparisons (e.g., Markon & Krueger, 2006); thus, we also report the BIC values for our models (with lower BIC values indicating better fit).

Latent-variable survival analyses Proportional-hazard survival analyses were conducted using a person-year database with the diagnostic variables (i.e., UMDs, ADs, comorbid UMDs and ADs, MDD, and PD) as dependent variables. Individuals with a lifetime history of a particular disorder at baseline were excluded from analyses of that diagnostic outcome (for comorbid UMDs and ADs, an individual was excluded from analyses only if he or she had a history of a comorbid UMD and AD at baseline). Similarly, for individuals who developed an initial onset of a particular disorder at a given FU assessment, their subsequent person-years were excluded from the analyses of that disorder (for comorbid UMDs and ADs, an individual's subsequent person-years were excluded from analyses only after they had comorbid UMD and AD).

We first report associations of each disorder with each of the observed measures of N and its facets. Next, we report the associations of each disorder with each of the latent variables in our vulnerability measurement model. Given that the latent variables in our vulnerability measurement

model are constrained to be orthogonal, these results should be interpreted as unique effects (S. G. West, personal communication, January 25, 2013). We also conducted analyses of pure UMDs and pure MDD in which the outcomes being predicted were the development of new onsets of these disorders in the absence of a history of an AD. Similarly, we conducted analyses of pure ADs in which the outcome being predicted was the development of new onsets of ADs in the absence of a history of a UMD. We did not analyze pure PD, given the small number of PD cases and even smaller number of pure PDs.

Gender was a covariate in all analyses, given that UMDs and most ADs are more common in women than in men (e.g., Craske, 2003; Nolen-Hoeksema & Hilt, 2009) and that women score higher than do men on the GNF (e.g., Costa et al., 2001), as well as on several of our N facets (e.g., Hankin & Abramson, 2001). (Results obtained without the use of gender as a covariate produced virtually identical results and are available upon request from the corresponding author.) To test whether gender moderated the associations of the GNF with initial onsets of any of the diagnostic outcomes, we conducted multiple-group survival analyses with the GNF as the predictor. These analyses used a likelihood-ratio test to compare a model that constrained the hazard ratio for the GNF to be equal across the sexes with a model that allowed that hazard ratio to differ across the sexes.

For each latent variable in the vulnerability measurement model that had a significant hazard ratio with UMDs or MDD, we conducted specificity comparisons by testing whether that hazard ratio was significantly stronger than the hazard ratio for that latent variable's prediction of ADs. Similarly, for each latent variable that had a significant hazard ratio with pure UMDs or pure MDD, we conducted specificity comparisons by testing whether that ratio was significantly stronger than the ratio for that latent variable's prediction of pure ADs. Likewise, for each latent variable that had a significant hazard ratio with ADs, pure ADs, or PD, we tested whether that ratio was significantly stronger than that latent variable's ratio with UMDs. For each latent variable that had a significant hazard ratio with comorbid ADs and UMDs, we tested whether that ratio was significantly stronger than that latent variable's ratio with pure cases (pure UMDs and pure ADs). Finally, for each latent variable that had a significant hazard ratio with SUDs, we tested whether that hazard ratio was significantly different from the hazard ratio for that latent variable's prediction of UMDs and of ADs. We conducted all of these specificity comparisons by using inferential confidence intervals (e.g., Tryon, 2001).

Results

Overlap among the risk factors

Preliminary analysesPreliminary analyses were directed toward specification of a base, hierarchical CFA model that could provide the foundation for testing our three hypotheses regarding the latent structure of the observed vulnerability measures. This model was specified on the basis of three considerations. The first consideration was prior theoretical and empirical research on the structure of one or more of the vulnerability measures (e.g., Lewis et al., 2010; Prenoveau et al., 2009; Whisman & Friedman, 1998). The second consideration was the results of item-level EFAs conducted in the first random subsample of some of the measures.

Table 2. Correlations Among Subscale Indicators

Subscale indicator	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29
1. IPIP Anx	—																												
2. IPIP Ang	.56	—																											
3. IPIP Dep	.57	.53	—																										
4. IPIP SC	.52	.32	.48	—																									
5. IPIP Imp	.30	.25	.29	.17	—																								
6. IPIP Vul	.59	.42	.51	.55	.37	—																							
7. Big5 Anx	.58	.42	.45	.32	.16	.37	—																						
8. Big5 Ang	.43	.39	.39	.35	.27	.35	.36	—																					
9. Big5 Dep	.48	.68	.40	.24	.29	.37	.46	.46	—																				
10. BIS SC	.42	.29	.37	.39	.11	.33	.35	.31	.32	—																			
11. BIS Anx	.55	.35	.36	.39	.17	.38	.48	.27	.42	.58	—																		
12. EPQ Dep	.26	.40	.37	.15	.15	.23	.33	.17	.35	.19	.24	—																	
13. EPQ Soc	.44	.29	.39	.29	.19	.33	.33	.28	.30	.42	.42	.32	—																
14. EPQ Anx	.48	.29	.32	.27	.11[a]	.30	.43	.17	.24	.24	.39	.30	.38	—															
15. EPQ Ang	.21	.40	.18	.08[b]	.09[a]	.16	.22	.17	.33	.03[b]	.12	.39	.14	.17	—														
16. DAS-A Ach	.43	.37	.54	.39	.20	.38	.39	.33	.29	.30	.33	.23	.35	.22	.17	—													
17. DAS-A App	.40	.33	.51	.37	.21	.35	.35	.35	.32	.47	.43	.19	.38	.17	.11[a]	.56	—												
18. Sociotropy	.46	.34	.46	.38	.25	.40	.36	.35	.39	.61	.55	.26	.47	.29	.03[b]	.37	.60	—											
19. Autonomy	.26	.42	.39	.29	.15	.27	.18	.23	.31	.19	.18	.30	.22	.08[a]	.19	.41	.22	.36	—										
20. Global	.37	.29	.42	.29	.22	.34	.29	.31	.30	.30	.32	.27	.29	.17	.10[a]	.36	.37	.44	.32	—									
21. Stable	.23	.19	.26	.16	.11[a]	.16	.19	.18	.23	.20	.20	.19	.22	.08[b]	.11	.19	.31	.25	.23	.59	—								
22. Cons	.38	.29	.44	.31	.22	.33	.30	.33	.31	.32	.34	.24	.35	.11	.09[a]	.39	.40	.45	.34	.83	.61	—							
23. Flaw	.41	.27	.47	.35	.21	.39	.34	.39	.29	.36	.39	.20	.41	.15	.10[a]	.46	.46	.49	.28	.68	.46	.76	—						
24. Physical 1	.31	.25	.20	.19	.15	.24	.19	.17	.24	.27	.29	.14	.22	.15	.01[b]	.20	.25	.38	.22	.27	.11[a]	.25	.27	—					
25. Physical 2	.33	.21	.22	.18	.10[a]	.21	.23	.12	.20	.21	.32	.18	.21	.19	.04[b]	.19	.19	.32	.23	.25	.13	.24	.24	.67	—				
26. Mental 1	.41	.31	.39	.27	.18	.32	.32	.19	.30	.18	.27	.25	.22	.26	.13	.36	.25	.31	.36	.34	.16	.33	.33	.46	.50	—			
27. Mental 2	.41	.25	.30	.31	.17	.34	.29	.15	.22	.21	.30	.14	.21	.28	.02[b]	.35	.26	.32	.29	.28	.10[a]	.24	.27	.57	.57	.73	—		
28. Social 1	.26	.29	.25	.20	.12	.17	.24	.18	.25	.33	.35	.13	.28	.17	.09[a]	.30	.31	.38	.34	.28	.17	.31	.28	.39	.36	.36	.41	—	
29. Social 2	.36	.34	.41	.35	.09[a]	.26	.33	.25	.30	.35	.38	.26	.30	.26	.12	.41	.36	.40	.39	.38	.22	.38	.35	.42	.41	.53	.49	.57	—

Note: All unmarked coefficients are significant (α = .01). IPIP = International Personality Item Pool NEO Personality Inventory–Revised Neuroticism scale; Anx = anxiety; Ang = anger; Dep = depression; SC = self-consciousness; Imp = impulsivity; Vul = vulnerability; Big5 = Big Five Mini-Markers Neuroticism scale; BIS = Behavioral Inhibition Scale; EPQ = Eysenck Personality Questionnaire–Revised Neuroticism scale; Soc = social concerns; DAS-A = Dysfunctional Attitudes Scale–Form A; Ach = needing achievement; App = needing approval; Cons = consequences to self; Physical 1 and 2 = physical concerns; Mental 1 and 2 = mental concerns; Social 1 and 2 = social concerns.

a. Correlations are significant (α = .05). b. Correlations are not significant.

The third consideration was the results of initial subscale-level CFAs that we conducted. Details of the model-specification process are presented in the Item-Level Analyses of Measures Consensually Considered to Tap One or More N Facets and the Subscale-Level Analysis of the Base, Hierarchical Factor Model of N and Its Cognitive Facets sections in the Supplemental Material available online.

The preliminary analyses resulted in the identification of 15 subscales derived from the EPQ-R N, the IPIP-NEO-PI-R N, the Big5 N, and the BIS scales (Table S1 in the Supplemental Material presents the items assigned to each subscale and subscale reliabilities). Table 2 displays the correlations among those 15 subscales, as well as the correlations with the DAS-A, PSI, CSQ, and AS subscales. As shown in Table 2, all of these correlations are positive, which is consistent with the presence of a GNF that runs through all of the subscales.

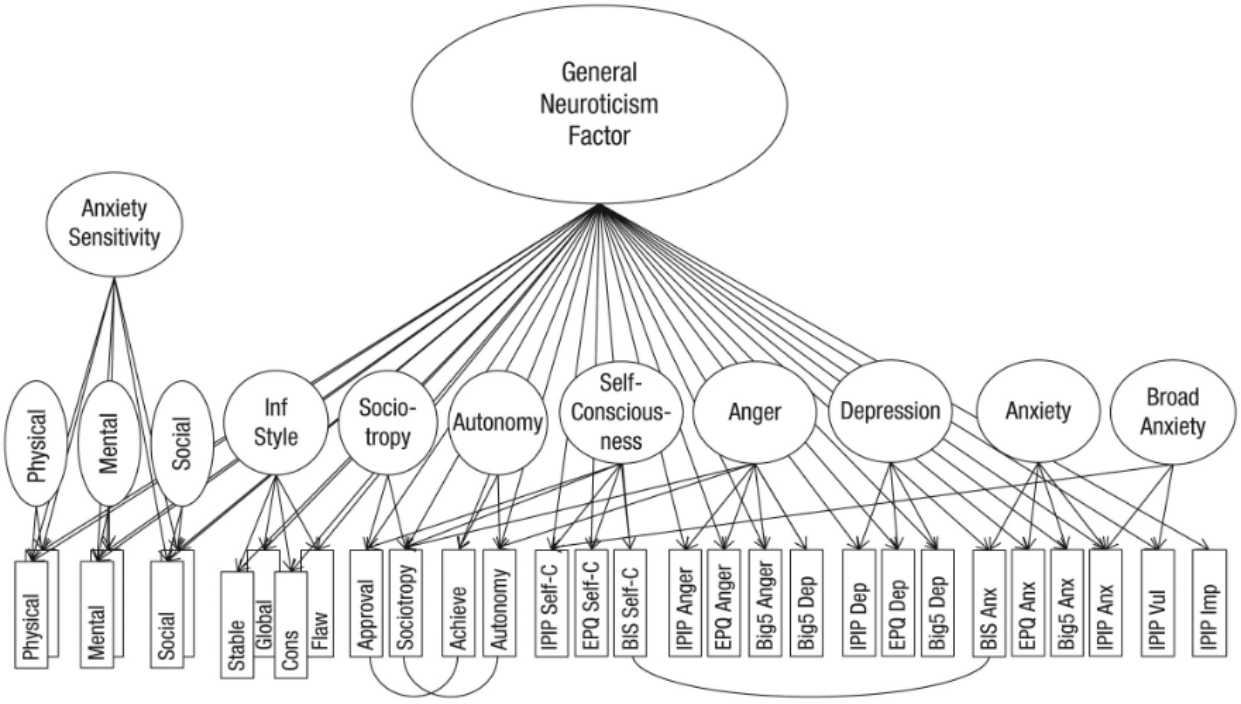

Fig. 1. Base, hierarchical confirmatory factor analytic model of neuroticism, including its cognitive facets. Inf Style = negative inferential style; Stable, Global, Cons (Consequences), and Flaw = subscales of the Cognitive Styles Questionnaire; Approval and Achieve = Dysfunctional Attitudes Scales Needing Approval and Needing Achievement scales; Sociotropy and Autonomy (in boxes) = Personal Style Inventory scales; IPIP = International Personality Item Pool NEO Personality Inventory–Revised N scale; EPQ = Eysenck Personality Questionnaire–Revised N scale; BIS = Behavioral Inhibition Scale; Self-C = self-consciousness; Big5 = Big Five Mini-Markers N scale; Dep = depression; Anx = anxiety; Vul = vulnerability; Imp = impulsivity. Observed indicators for the specific Anxiety Sensitivity factors (i.e., concerns about physiological, mental, and social consequences) were created by randomly assigning half of the items to the first and the other half to the second subscale for each type of concerns. The model also contained an EPQ method factor (not shown).

The base, hierarchical CFA model that was specified, in part, on the basis of the preliminary analyses in the first random subsample is displayed in Figure 1. All subscales were specified as indicators of a GNF. The four widely recognized N facets of depression, anxiety, self-consciousness, and anger were each indicated by at least two subscales. A broad anxiety factor identified in the IPIP-NEO-PI-R by Uliaszek et al. (2009) was indicated by the IPIP-NEO-PI-R Anxiety, Self-Consciousness, and Vulnerability subscales. BIS Anxiety and Self-Consciousness

subscales were allowed to correlate (thus accounting for the method variance due to these two subscales' coming from the same measure, which differed from the measure the other three subscales came from). A sociotropy facet was indicated by the PSI-Sociotropy and the DAS-A-Need for Approval subscales; an autonomy facet was indicated by the PSI-Autonomy and the DAS-A-Need for Achievement subscales. A negative inferential style facet was indicated by the four CSQ subscales. AS-physical concerns, AS-mental incapacitation concerns, and AS-social concerns facets were each indicated by two ASI-X subscales, and all six ASI-X subscales were specified as indicators of a general AS factor.

The fit of the base, hierarchical CFA model in the second random subsample was acceptable, $\chi 2(339, N = 308) = 766.41$, $p < .001$, RMSEA = .064, CFI = .90, SRMR = .056, BIC = 16,666.38. All loadings except for that of PSI-Sociotropy on the anger facet were significant. We also tested invariance across the two random subsamples by conducting a multiple-group analysis of our base, hierarchical CFA model. A configural-invariant model provided an adequate fit to the data, $\chi 2(693, N = 607) = 1{,}406.64$, $p < .001$, RMSEA = .058, CFI = .91, SRMR = .054, BIC = 33,208.56. In the metric-invariant model, we applied across-samples equality constraints to factor loadings and intercepts for all factors and all items. The fit of the metric-invariant model was adequate, $\chi 2(757, N = 607) = 1{,}467.16$, $p < .001$, RMSEA = .056, CFI = .91, SRMR = .061, BIC = 32,858.92, and was not significantly worse than the configural-invariant model, $\chi 2\text{diff}(64, N = 607) = 60.52$, $p > .10$. In addition, all loadings were in the expected direction and significant. Because the metric-invariant model did not significantly degrade model fit and provided adequate fit, these results indicated that the model built in the first subsample was cross-validated in the second subsample. Table 3 displays the standardized loadings from the base, hierarchical CFA model estimated in the full sample (fit in the full sample was adequate)—$\chi 2(339, N = 607) = 973.99$, $p < .001$, RMSEA = .056, CFI = .92, SRMR = .046, BIC = 32,628.70.

Are dysfunctional attitudes, sociotropy, and autonomy best represented by two distinguishable group factors in addition to the GNF? We began our testing of the hypothesis that dysfunctional attitudes, sociotropy, and autonomy are best represented by two group factors in addition to the GNF by allowing the Sociotropy and Autonomy factors to correlate. If adding this correlation provided a significant increment in model fit, it would indicate that there is additional shared variance between the Sociotropy subscales and the Autonomy subscales beyond what can be accounted for in the base, hierarchical CFA model. The fit of this model was not significantly better than the base, hierarchical CFA model, $\chi 2\text{diff}(1, N = 308) = 3.28$, n.s. Thus, the results failed to provide evidence that an additional factor beyond the GNF and the Sociotropy and Autonomy group factors is needed to model the covariances among the four DAS-A and PSI subscales.

We next tested whether the GNF accounts for a significant portion of the covariances among the Sociotropy and Autonomy subscales by removing the loadings of the four DAS-A and PSI subscales on the GNF. The fit of this model was significantly worse than the base, hierarchical CFA model, $\chi 2\text{diff}(4, N = 308) = 262.86$, $p < .001$. Allowing the Sociotropy and Autonomy factors to correlate in the version of the model in which the DAS-A and PSI subscales were not specified as indicators of the GNF led to a significant improvement in model fit, $\chi 2\text{diff}(1, N = 308) = 36.37$, $p < .001$. This pair of results reveals that the subscales loading on the Sociotropy factor share significant variance with the subscales loading on the Autonomy factor and that the GNF accounts for a signification proportion of this shared variance. The model with correlated Sociotropy and Autonomy factors in which the four DAS-A and PSI subscales were not specified as indicators of

the GNF stands in a nested relationship with a useful comparison model. In this comparison model, the four DAS-A and PSI subscales were again not specified as indicators of the GNF, but these four subscales were specified as loading on a single group factor rather than on two group factors. Comparison of these two models tests whether two group factors are necessary to account for the covariances among the DAS-A and PSI subscales. Reducing the number of DAS-A and PSI group factors from two to one resulted in a significant decrement in fit, χ2diff(1, N = 308) = 8.62, $p < .001$. This result indicates that the Sociotropy and Autonomy factors, although correlated, are distinguishable.

Finally, we tested whether Sociotropy and Autonomy are distinguishable from the GNF by removing the Sociotropy and Autonomy factors from the base, hierarchical CFA model. Doing so produced a significant decrement in fit, χ2diff(2, N = 308) = 22.46, $p < .001$, χ2(341, N = 308) = 788.87, $p < .001$, RMSEA = .065, CFI = .90, SRMR = .056, BIC = 16,677.37.5 These results suggest that Sociotropy and Autonomy are distinguishable from the GNF. Thus, the pattern of results presented in this section show that the four DAS-A and PSI subscales are best modeled by two distinguishable group factors in addition to the GNF.

Do dysfunctional attitudes and negative inferential style tap a common cognitive-vulnerability factor that is distinguishable from the GNF?We attempted to test whether a factor common to all of the cognitive- and personality/cognitive-style vulnerability measures should be added to the model. Thus, we added one more common latent factor and allowed the subscales of the DAS-A, PSI, and CSQ to have loadings on this additional latent factor in addition to their other loadings described earlier. This model did not converge, even as we increased the number of computational iterations to 10,000. We also tried to model a factor common to the cognitive- and personality/cognitive-style measures after removing the Sociotropy and Autonomy factors, but this model also did not converge. The lack of convergence of these models suggests that the variance that the DAS-A, PSI, and CSQ subscales all share is due to the GNF. That is, these results fail to support the existence of a distinct vulnerability factor shared by the DAS-A, PSI, and CSQ subscales independent of the GNF.6

Does the hierarchical structure of AS include a common AS factor in addition to the GNF?We tested whether the GNF accounts for a significant portion of the covariances among the AS subscales by removing the loadings of the six ASI-X subscales on the GNF. The fit of this model was significantly worse than the base, hierarchical CFA model, χ2diff(6, N = 308) = 121.32, $p < .001$. Thus, the ASI-X subscales are indicators of the GNF. We then tested whether all six ASI-X subscales have an AS factor in common that is distinguishable from the GNF by removing the latent AS factor from the base, hierarchical CFA model. Doing so led to a significant decrement in fit, χ2diff(6, N = 308) =157.89, $p < .001$. This result shows that the common AS factor is distinguishable from the GNF. Thus, the results show that the ASI-X subscales are best modeled by a common AS factor in addition to the GNF.7

Table 3. Standardized Loadings From the Base, Hierarchical Factor Model of Neuroticism Including Its Cognitive Facets (Full Sample)

					Latent Factor									
Subscale indicator	GNF	Anx	Dep	SC	Ang	Broad Anx	Sociotropy	Autonomy	Inf style	AS physical	AS mental	AS social	AS	EPQ
IPIP Anx	.74	.35				.24								
IPIP Ang	.64				.59									
IPIP Dep	.75		.13											
IPIP SC	.57			.09		.39								
IPIP Imp	.37													
IPIP Vul	.63					.49								
Big5 Anx	.61	.38												
Big5 Ang	.51				.32									
Big5 Dep	.62		.07		.37									
BIS SC	.56			.53										
BIS Anx	.62	.27												
EPQ Dep	.42		.48											.60
EPQ Soc	.56			.22										.20
EPQ Anx	.42	.46												.22
EPQ Ang	.22				.40									.40
DAS-A Ach	.63							.43						
DAS-A App	.60			.25			.35							
Sociotropy	.68			.37	-.09		.33							
Autonomy	.46				.19			.36						
Global	.56								.66					
Stable	.34								.55					
Cons	.59								.75					
Flaw	.63								.51					
Physical 1	.38									.41			.58	
Physical 2	.37									.47			.60	
Mental 1	.53										.40		.49	
Mental 2	.47										.39		.65	
Social 1	.44											.45	.32	
Social 2	.57											.45	.35	

Note: GNF = general neuroticism factor; Anx = anxiety; Dep = depression; SC = self-consciousness; Ang = anger; Inf style = negative inferential style; AS = anxiety sensitivity (this latent factor was indicated by six subscales measuring three lower-order factors—Physical 1 and 2 = physical concerns, Mental 1 and 2 = mental concerns, and Social 1 and 2 = social concerns); EPQ = Eysenck Personality Questionnaire–Revised Neuroticism scale; IPIP = International Personality Item Pool NEO Personality Inventory–Revised Neuroticism scale; Imp = impulsivity; Vul = vulnerability; Big5 = Big Five Mini-Markers Neuroticism scale; BIS = Behavioral Inhibition Scale; Soc = social concerns; DAS-A = Dysfunctional Attitudes Scale–Form A; Ach = needing achievement; App = needing approval; Cons = consequences to self; EPQ = an EPQ method factor. All loadings are significant ($\alpha = .05$).

Are there sex differences in the latent, hierarchical structure of N and its cognitive facets? We performed multiple-group CFAs to test the invariance across men and women of the base, hierarchical CFA model of N and its cognitive facets. We began by testing for metric invariance. In the metric-invariant model, we constrained each of the nonzero loadings on the various factors and each item intercept to be equal across men and women. The metric-invariant model provided an adequate fit, $\chi 2(757, N = 607) = 1{,}436.208$, $p < .001$, RMSEA = .054, CFI = .91, SRMR = .059. We then tested whether allowing any of the factor loadings to be free to vary between men and women provided a significant increment in fit compared with the metric-invariant model. None of these tests provided support for differences in loadings between men and women.8 Together with the adequate fit of the metric-invariant model, these results suggest that the base, hierarchical CFA model of N and its cognitive facets was highly similar in men and women.

Prospective prediction of diagnoses

Prospective associations of observed vulnerability measures and gender with diagnoses As shown in Table 4, the observed measures of N and its facets showed consistently positive associations with the development of UMDs, ADs, MDD, and comorbid UMDs and ADs. All 48 of these hazard ratios were greater than 1, and 41 (85.4%) of them were significant. In contrast, there were less consistent associations of the observed measures of N and its facets with the development of pure UMDs, pure ADs, PD, and SUDs. Only 8 (16.7%) of these 48 hazard ratios were significantly greater than 1, and another 9 (18.8%) had point estimates less than 1 (though none were significantly less than 1). The pattern for pure MDD was intermediate between these first two patterns; all 12 of the hazard ratios were greater than 1, and 6 (50%) of them were significant. The CSQ and DAS-A-Need for Achievement subscale, which have been hypothesized to be specific predictors of UMDs, had significant hazard ratios with ADs. Similarly, the ASI-X Physical Concerns and ASI-X Social Concerns subscales, which have been hypothesized to be specific predictors of ADs, had significant hazard ratios with UMDs. Thus, none of these measures may be as specific as some have thought but, rather, all of them are saturated to a substantial degree with variance due to the GNF.

Table 4 also shows that UMDs were significantly more common in women and SUDs were significantly more common in men. None of the other hazard ratios for gender were significant. The hazard ratios for comorbid UMDs and ADs, pure UMDs, MDD, and pure MDD were, however, in the direction of being (nonsignificantly) more common in women.

Prospective associations of latent variables with UMDs and ADs As shown in Table 5, the GNF predicted significantly greater rates of developing each of the diagnostic outcomes except for pure UMDs, pure ADs, and PD. The depression facet predicted significantly greater rates of developing UMDs, MDD, and comorbid ADs and UMDs. The anxiety facet predicted significantly lower rates of developing pure UMDs and pure MDD. The inferential style facet predicted a significantly greater rate of developing pure UMDs (but not pure MDD). Finally, the ASI-X mental incapacitation concerns facet predicted a significantly lower rate of developing pure MDD and a significantly greater rate of developing PD.

Table 4. Hazard Ratios of Gender and the Observed Measures of Neuroticism and Its Facets With Initial Onsets of Diagnoses

Measure	UMD	AD	Com	PureU	PureA	MDD	PureM	PD	SUD
Gender	0.50* (–0.69, 0.33)	1.02 (0.02, 0.33)	0.61 (−0.50, 0.38)	0.50 (−0.68, 0.46)	1.26 (0.23, 0.44)	0.61 (−0.50, 0.31)	0.79 (−0.24, 0.39)	1.38 (0.32, 0.73)	2.65* (0.97, 0.38)
EPQ	23.52* (3.16, 0.71)	9.40* (2.24, 0.85)	117.21* (4.76, 0.89)	1.43 (0.36, 0.85)	1.18 (0.16, 1.08)	36.20* (3.59, 0.74)	2.38 (0.87, 0.88)	3.10 (1.13, 2.00)	0.67 (−0.40, 0.96)
IPIP	4.91* (1.59, 0.26)	2.27* (0.82, 0.29)	6.57* (1.88, 0.31)	1.77* (0.57, 0.29)	1.19 (0.18, 0.40)	4.57* (1.52, 0.25)	1.86* (0.62, 0.30)	2.66 (0.98, 0.66)	2.35* (0.85, 0.33)
Big5	1.73* (0.55, 0.104)	1.34* (0.29, 0.12)	2.13* (0.76, 0.13)	1.17 (0.16, 0.13)	1.22 (0.20, 0.16)	1.63* (0.49, 0.10)	1.18 (0.17, 0.13)	0.70 (−0.36, 0.32)	1.42* (0.35, 0.15)
BIS	2.88* (1.06, 0.25)	2.33* (0.84, 0.30)	7.06* (1.96, 0.37)	1.12 (0.11, 0.30)	1.36 (0.31, 0.37)	2.97* (1.09, 0.26)	1.10 (0.09, 0.30)	0.58 (−0.55, 0.64)	1.16 (0.15, 0.34)
DAS-A Ach	1.61* (0.48, 0.16)	1.71* (0.54, 0.20)	2.03* (0.71, 0.20)	1.17 (0.16, 0.22)	1.20 (0.18, 0.27)	1.64* (0.49, 0.17)	1.23 (0.21, 0.22)	0.97 (−0.03, 0.50)	1.23 (0.21, 0.24)
DAS-A App	1.77* (0.57, 0.15)	1.37 (0.31, 0.18)	1.92* (0.65, 0.17)	1.38 (0.32, 0.20)	1.00 (0.00, 0.25)	1.45* (0.38, 0.15)	1.06 (0.06, 0.20)	0.81 (−0.21, 0.46)	1.40 (0.34, 0.21)
CSQ	2.17* (0.78, 0.15)	1.45* (0.38, 0.18)	2.29* (0.83, 0.19)	1.65* (0.50, 0.18)	0.98 (−0.02, 0.23)	2.12* (0.75, 0.15)	2.09* (0.74, 0.15)	1.47 (0.39, 0.41)	1.54* (0.43, 0.21)
Sociotropy	2.00* (0.69, 0.16)	1.37 (0.31, 0.18)	2.48* (0.91, 0.20)	1.33 (0.29, 0.19)	1.10 (0.10, 0.22)	2.05* (0.72, 0.16)	2.13* (0.76, 0.16)	1.06 (0.06, 0.42)	1.43 (0.36, 0.22)
Autonomy	1.70* (0.53, 0.14)	1.11 (0.11, 0.17)	1.53* (0.43, 0.17)	1.54* (0.43, 0.17)	0.86 (−0.15, 0.21)	1.62* (0.48, 0.14)	1.59* (0.47, 0.15)	0.89 (−0.11, 0.42)	1.60* (0.47, 0.20)
ASI-X-P	1.46* (0.38, 0.19)	1.24 (0.22, 0.24)	1.14 (0.43, 0.17)	1.25 (0.22, 0.25)	1.13 (0.12, 0.31)	1.19 (0.17, 0.20)	1.24 (0.22, 0.19)	1.00 (0.00, 0.56)	1.17 (0.15, 0.28)
ASI-X-M	1.76* (0.56, 0.18)	1.44 (0.37, 0.23)	1.83* (0.61, 0.18)	0.94 (−0.06, 0.28)	1.16 (0.15, 0.33)	1.59* (0.46, 0.17)	1.60* (0.47, 0.17)	2.45* (0.90, 0.37)	1.27 (0.24, 0.26)
ASI-X-S	2.25* (0.81, 0.20)	1.65* (0.50, 0.23)	2.11* (0.75, 0.22)	1.39 (0.33, 0.23)	1.31 (0.27, 0.31)	1.60* (0.47, 0.19)	1.59* (0.47, 0.19)	1.39 (0.33, 0.52)	1.41 (0.34, 0.26)

Note: Standardized coefficients and standard errors are shown in parentheses. UMD = unipolar mood disorder; AD = anxiety disorder; Com = comorbidity of at least one UMD and at least one AD; PureU = pure UMD; PureA = pure AD; MDD = major depressive disorder; PureM = pure MMD; PD = panic disorder; SUD = substance use disorder; EPQ = Eysenck Personality Questionnaire–Revised Neuroticism scale; IPIP = International Personality Item Pool NEO Personality Inventory–Revised Neuroticism scale; Big5 = Big Five Mini-Markers Neuroticism scale; BIS = Behavioral Inhibition Scale; DAS-A Ach = Dysfunctional Attitudes Scale–Form A Need for Achievement scale; DAS-A App = Dysfunctional Attitudes Scale–Form A Need for Approval scale; CSQ = Cognitive Style Questionnaire; Sociotropy = Personal Style Inventory Sociotropy scale; Autonomy = Personal Style Inventory Autonomy scale; ASI-X-P = Anxiety Sensitivity Index Expanded Form–Physical Concerns subscale; ASI-X-M = Anxiety Sensitivity Index Expanded Form–Mental Incapacitation Concerns subscale; ASI-X-S = Anxiety Sensitivity Index Expanded Form–Social Concerns subscale.

* $p \leq .05$.

Table 5. Hazard Ratios of the General Neuroticism Factor (GNF) and the Facets of Neuroticism With Initial Onsets of Diagnoses With Gender as a Covariate

Measure	UMD	AD	Com	PureU	PureA	MDD	PureM	PD	SUD
GNF	3.19* (1.16, 0.19)	1.92* (0.65, 0.19)	4.64* (1.53, 0.25)	1.35 (0.30, 0.19)	1.27 (0.24, 0.25)	3.03* (1.11, 0.18)	1.48* (0.40, 0.19)	1.41 (0.34, 0.41)	1.65* (0.50, 0.21)
Dep	2.39* (0.87, 0.32)	1.19 (0.17, 0.28)	2.07* (0.73, 0.34)	1.96 (0.67, 0.35)	0.96 (−0.04, 0.38)	2.02* (0.70, 0.30)	1.73 (0.55, 0.38)	1.82 (0.60, 0.73)	1.48 (0.39, 0.36)
Anx	0.83 (−0.19, 0.19)	1.09 (0.09, 0.25)	1.31 (0.27, 0.24)	0.46* (−0.77, 0.29)	1.30 (0.26, 0.33)	0.90 (−0.10, 0.20)	0.56* (−0.59, 0.29)	0.89 (−0.11, 0.60)	0.66 (−0.42, 0.32)
Broad	0.95 (−0.06, 0.20)	1.42 (0.35, 0.24)	1.15 (0.14, 0.24)	0.81 (−0.21, 0.27)	1.35 (0.30, 0.32)	1.00 (0.00, 0.20)	0.86 (−0.15, 0.28)	1.88 (0.63, 0.59)	0.78 (−0.25, 0.30)
Ang	1.22 (0.20, 0.17)	1.04 (0.04, 0.21)	1.11 (0.11, 0.20)	1.19 (0.18, 0.22)	1.07 (0.06, 0.29)	1.26 (0.23, 0.17)	1.27 (0.24, 0.23)	0.71 (−0.34, 0.56)	1.86* (0.62, 0.25)
Self-C	0.86 (−0.15, 0.19)	0.99 (−0.01, 0.24)	1.22 (0.20, 0.24)	0.75 (−0.28, 0.24)	0.84 (−0.18, 0.32)	1.03 (0.03, 0.19)	0.82 (−0.20, 0.25)	0.48 (−0.74, 0.55)	1.03 (0.03, 0.30)
Inf	1.30 (0.27, 0.15)	1.06 (0.06, 0.18)	1.11 (0.11, 0.19)	1.59* (0.46, 0.20)	0.97 (−0.03, 0.24)	1.30 (0.23, 0.17)	1.41 (0.35, 0.20)	1.23 (0.20, 0.45)	1.46 (0.38, 0.23)
Socio	1.19 (0.18, 0.22)	0.90 (−0.10, 0.28)	0.99 (−0.01, 0.29)	1.32 (0.28, 0.31)	1.03 (0.03, 0.37)	0.96 (−0.04, 0.23)	0.90 (−0.10, 0.33)	0.81 (−0.21, 0.69)	1.27 (0.24, 0.37)
Aut	0.94 (−0.06, 0.22)	1.19 (0.17, 0.27)	0.92 (−0.09, 0.27)	1.11 (0.10, 0.29)	0.96 (−0.05, 0.36)	1.02 (0.02, 0.23)	1.22 (0.20, 0.31)	0.71 (−0.34, 0.68)	0.88 (−0.13, 0.34)
AS	0.90 (−0.10, 0.18)	0.99 (−0.01, 0.22)	0.77 (−0.26, 0.23)	0.88 (−0.12, 0.24)	1.05 (0.05, 0.28)	0.77 (−0.27, 0.19)	0.71 (−0.34, 0.25)	1.72 (0.54, 0.38)	0.92 (−0.09, 0.25)
ASM	0.99 (−0.01, 0.24)	0.97 (−0.03, 0.31)	1.36 (0.31, 0.26)	0.57 (−0.56, 0.36)	0.91 (−0.10, 0.42)	1.10 (0.09, 0.23)	0.42* (−0.87, 0.44)	3.32* (1.20, 0.44)	0.95 (−0.05, 0.34)
ASP	0.80 (−0.23, 0.22)	0.88 (−0.13, 0.27)	0.44 (−0.82, 0.32)	1.17 (0.16, 0.27)	1.07 (0.06, 0.36)	0.72 (−0.33, 0.22)	1.13 (0.12, 0.28)	0.28 (−1.26, 0.71)	0.90 (−0.10, 0.32)
ASS	1.26 (0.23, 0.21)	1.23 (0.21, 0.26)	1.13 (0.12, 0.25)	1.25 (0.23, 0.25)	1.23 (0.21, 0.35)	0.87 (−0.15, 0.21)	1.02 (0.02, 0.27)	0.85 (−0.17, 0.61)	1.00 (0.00, 0.30)

Note: Standardized coefficients and standard errors are shown in parentheses. UMD = unipolar mood disorder; AD = anxiety disorder; Com = comorbidity of at least one UMD and at least one AD; PureU = pure UMD; PureA = pure AD; MDD = major depressive disorder; PureM = pure MMD; PD = panic disorder; SUD = substance use disorder; Dep = depression facet; Anx = anxiety facet; Broad = broad anxiety facet; Ang = anger facet; Self-C = self-consciousness facet; Inf = negative inferential style facet; Socio = sociotropy facet; Aut = autonomy facet; AS = anxiety sensitivity facet; ASM = anxiety sensitivity mental incapacitation concerns facet; ASP = anxiety sensitivity physical concerns facet; ASS = anxiety sensitivity social concerns facet.

* $p \leq .05$.

Table 6. Hazard Ratios of the General Neuroticism Factor With Initial Onsets of Diagnoses by Gender

Gender	UMD	AD	Com	PureU	PureA	MDD	PureM	PD	SUD
Female	3.61* (1.29, 0.22)	2.28* (0.82, 0.24)	5.38* (1.68, 0.30)	1.44 (0.36, 0.21)	1.44 (0.37, 0.30)	3.23* (1.17, 0.21)	1.57* (0.45, 0.22)	3.45a (1.24, 0.60)	1.55 (0.44, 0.31)
Male	1.97 (0.68, 0.39)	1.34 (0.29, 0.31)	3.07* (1.12, 0.47)	1.06 (0.05, 0.40)	0.88 (−0.13, 0.47)	2.42* (0.88, 0.36)	1.28 (0.24, 0.35)	0.54b (−0.61, 0.63)	1.77* (0.57, 0.28)

Note: Standardized coefficients and standard errors are shown in parentheses. Within the PD column, values with different subscripts differ significantly with an alpha level of .05. Italics indicate $.05 < p < .10$. UMD = unipolar mood disorder; AD = anxiety disorder; Com = comorbidity of at least one UMD and at least one AD; PureU = pure UMD; PureA = pure AD; MDD = major depressive disorder; PureM = pure MMD; PD = panic disorder; SUD = substance use disorder.

* $p \leq .05$.

Differences in prospective associations with latent variables for UMDs versus ADs Only four of the comparisons that tested for differences in a latent variable's associations with UMDs versus its associations with ADs were significant. The hazard ratio for the anxiety facet was significantly smaller for pure UMDs than for pure ADs or even any ADs. In addition, the hazard ratio for the ASI-X mental incapacitation concerns facet was significantly larger for PD than for UMDs or MDD. None of the remaining latent variables had significantly different hazard ratios with ADs than with UMDs, including that the inferential style facet with pure UMDs was not significantly larger than for any other outcome (including pure ADs or ADs). Thus, there was very little evidence of the latent variables' being significantly stronger predictors of UMDs than ADs or vice versa.

Differences in prospective associations with latent variables for comorbid UMDs and ADs versus the other UMD and AD outcomes The hazard ratio for the GNF was significantly stronger for comorbid UMDs and ADs than for ADs, pure UMDs, pure ADs, pure MDD, and PD. The hazard ratios for the anxiety facet were significantly smaller for both pure UMDs and pure MDD than for comorbid ADs and UMDs. None of the remaining latent variables had significantly different hazard ratios with comorbid ADs and UMDs than with the other UMD and AD outcomes.

Prospective *associations of latent variables with SUDs* As shown in Table 5, the GNF and the anger facet predicted significantly greater rates of developing SUDs, whereas none of the other latent variables did.

Differences in prospective associations for SUDs versus UMDs and ADs For each latent variable that had a significant hazard ratio with UMDs and/or ADs, we also conducted specificity comparisons by testing whether that ratio was significantly stronger than that latent variable's hazard ratio with SUDs. Consistent with RST and disconfirming the nonspecificity conceptualization of N, results showed that the hazard ratios of the GNF with UMDs, comorbid UMDs and ADs, and MDD were significantly stronger than its hazard ratio with SUDs. None of the remaining associations of a latent variable with UMDs or ADs were significantly stronger than for SUDs.

Did gender moderate the prospective associations of the GNF with initial onsets of UMDs, ADs, and SUDs? As shown in Table 5, only one of the prospective associations of the GNF, the one with PD, was significantly moderated by gender. Thus, the GNF was associated with a significant increase in risk of initial onsets of PD in females but was associated with a nonsignificant decrease in risk among males. The hazard ratios were also consistent: UMDs, ADs, comorbid UMDs and ADs, pure UMDs, pure ADs, MDD, and pure MDD were more strongly predicted by the GNF in women than in men, although these ratios did not significantly differ across the genders.

Discussion

Our results produced five major sets of findings. First are the findings regarding N. The GNF was a significant prospective predictor of new onsets of UMDs, including MDD, ADs, comorbid UMDs and ADs, pure MDD, and SUDs. It is important to note, however, that the GNF predicted UMDs and ADs even more strongly than SUDs and predicted comorbid UMDs and ADs even more strongly. Second are the findings regarding inferential style and dysfunctional attitudes. The Inferential Style group factor was a significant predictor of pure UMDs and did not significantly

predict ADs or pure ADs. However, the Inferential Style group factor did not predict pure UMDs significantly more strongly than ADs or even pure ADs. In addition, our CFA results suggest that the variance shared by the DAS-A and CSQ subscales is attributable to the GNF. Third are the findings regarding AS. The AS-mental incapacitation concerns facet was a significantly stronger predictor of PD than of UMDs or of MDD. Our CFA results demonstrated that the hierarchical structure of N includes an intermediate-breadth AS factor in addition to the (broad) GNF and three (narrow) AS group factors. Fourth, there was little evidence of gender moderation of the ability of the GNF to predict disorders. Finally, our CFA revealed the base, hierarchical factor model of N and its cognitive facets to be quite similar in men and women.

The results regarding N clearly disconfirm the nonspecificity model advanced by Claridge and Davis (2001) of the predictive power of the GNF (according to which, the GNF is incapable of discriminating risk for UMDs and ADs from risk for SUDs). Rather, the results are consistent with the RST hypothesis that the GNF is more specifically related to elevated negative affectivity, sensitivity to aversive cues, and behavioral inhibition than to elevated positive affectivity, cues for reward, and behavioral disinhibition (e.g., Gray & McNaughton, 2000; Zinbarg & Yoon, 2008). Thus, the GNF was a stronger predictor of comorbid UMDs and ADs than of SUDs, ADs, pure UMDs, pure ADs, pure MDD, and PD. This is consistent with the notion that the GNF is especially strongly associated with comorbid UMDs and ADs and, thus, a core vulnerability factor common to both UMDs and ADs.

The results are partially consistent with cognitive models of vulnerability for depression. The evidence reported here for the specificity of the Inferential Style group factor as a predictor of pure UMDs is suggestive but not conclusive, given that the Inferential Style group factor did not predict pure UMDs significantly more strongly than ADs. In addition, the results that suggest that the variance shared by the DAS-A and CSQ subscales is attributable to the GNF call into question whether the results in past research defining cognitive risk on the basis of elevations on both the DAS-A and the CSQ are attributable to the GNF or to the Inferential Style group factor.

The evidence reported here for AS theory is also best characterized as suggestive, given that the finding that the AS-mental incapacitation concerns facet was a specific predictor of PD is based on only eight new onsets of PD. In addition, our CFA results showed that a common AS factor can be reliably distinguished from the GNF, but our results failed to support the validity of the common AS factor or the AS-Physical Concerns or AS-Social Concerns group factors as predictors of ADs. Our results also bear on the incremental validity of negative inferential style versus dysfunctional attitudes. Our results provide support for the validity of the negative inferential style facet as a predictor of pure UMDs. However, our results failed to support the validity of the dysfunctional attitudes facets as predictors of UMDs. This pattern of results suggests that researchers or clinicians interested in cognitive vulnerability for depression who choose to administer just one of these measures would be better off measuring negative inferential style than dysfunctional attitudes.

The current work has a number of limitations. First, selecting participants on the basis of total scores on a screening measure for N, the EPQ-R N, might have increased statistical power to detect unique effects of the GNF relative to the N facets. However, simulations suggest that this is not the case (Hauner, Zinbarg, & Revelle, 2014). Another limitation is that our sample has not yet entered the peak age of PD onset (e.g., Kessler et al., 2006) and, as noted earlier, included only eight new onsets of PD. In addition, we did not include measures of life stress in the analyses reported here, and several of the vulnerabilities tested here have been explicitly proposed to be diatheses that are activated by stressors (and in some cases to be activated only by congruent

stressors, such as sociotropy's being activated by interpersonal rejection and autonomy's being activated by achievement-related stressors). Finally, we did not specifically assess for hopelessness depression (Abramson et al., 1989). It is possible that if we had assessed for it, then the cognitive facets—and not the GNF—would have uniquely predicted hopelessness depression.

In terms of future research studies to follow up on our results, one very important FU study would involve the design and testing of broad-based preventive interventions for high-N youth. Just as transdiagnostic treatment programs that have emerged in recent years have great potential to treat both UMDs and ADs (e.g., Barlow, Allen, & Choate, 2004; Craske, 2012), our results suggest that an effective, broad-based prevention program with high-N youth should hold great promise to reduce risk for both UMDs and many ADs (and especially comorbid UMDs and ADs). Our results suggest that such a preventive intervention could possibly even reduce risk for SUDs, although to a lesser extent than UMDs and ADs.

Computer programs and smartphone apps would seem to have the potential for reaching the largest number of youth. Such programs or apps certainly could reach many of those individuals without access to a local mental-health worker. It might even be that such interventions would be acceptable to the large numbers of individuals who might benefit from mental-health services and otherwise could be seen by a mental-health worker but who want to solve their problems on their own (Mojtabai et al., 2011). In addition, this automated approach could be implemented in a very consistent manner without the need for training therapists. What is unclear is whether it would be more effective to attempt to directly reduce general sensitivity to threat or to enhance general emotional regulation to buffer the effects of elevated threat sensitivity. A promising example of the former strategy would be a cognitive-bias modification (CBM) program to reduce attentional bias toward threat at a relatively automatic level (e.g., MacLeod, Rutherford, Campbell, Ebsworth, & Holker, 2002; Schmidt, Richey, Buckner, & Timpano, 2009). And an example of the latter strategy that has promise would be a CBM program to increase downregulation of cognitive reappraisal of aversive events or negative cognitions at a strategic level of processing (e.g., Denny & Ochsner, 2014; Mashal, Paller, & Zinbarg, 2015). Thus, the FU study we most want to see conducted would randomize high-N youth to one of three conditions: CBM to reduce attentional bias toward threat, CBM to strengthen downregulation of cognitive reappraisal tendencies, and a control condition, such as watchful waiting (e.g., Meredith, Cheng, Hickey, & Dwight-Johnson, 2007).

A second important future FU study would be one designed to more directly elucidate the mechanisms through which N confers risk for emotional disorders. Given that the GNF was a stronger predictor of comorbid UMDs and ADs than of SUDs, we inferred that the GNF is more specifically related to elevated negative affectivity, sensitivity to aversive cues, and behavioral inhibition than to elevated positive affectivity, cues for reward, and behavioral disinhibition. It would be important, however, for future research to test this notion more directly by measuring sensitivity to aversive cues and sensitivity to appetitive cues. Multiple methods should be used to assess these sensitivities, including both behavioral measures and patterns of activation in threat- and reward-related neural circuitries. Such a study should yield insights that would inform the development of broad-based prevention programs. Indeed, these two research directions should ultimately converge. Full understanding of a broad-based preventive intervention requires identification of the intervention's mechanisms, just as is the case for any intervention (e.g., Kazdin, 2007), and strong causal inference about risk requires studies that manipulate the hypothesized mechanisms of risk.

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The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at
http://cpx.sagepub.com/content/by/supplemental-data

Notes

1. There are also several prospective studies of behavioral inhibition—a vulnerability closely related to N—predicting onsets of ADs (e.g., Biederman et al., 1993; Hayward, Killen, Kraemer, & Taylor, 1998).
2. For details regarding the reasons for the gender imbalance and other methodological details, see Zinbarg et al. (2010).
3. Unadjusted kappa for MDD equaled .83 at baseline, .65 at first FU, .73 at second FU, and .68 at third FU; for UMDs equaled .72 at baseline, .65 at first FU, .64 at second FU, and .79 at third FU; for ADs equaled .66 at baseline, .51 at first FU, .39 at second FU, and .40 at third FU; and for SUDs equaled .66 at baseline, 1.00 at first FU, .55 at second FU, and .24 at third FU.
4. One previous study, reported by Hankin, Lakdawalla, Carter, Abela, and Adams (2007), had the potential to examine a hierarchical structure similar to the one tested here. However, although the researchers in that study extracted multiple factors, they did not tease apart the different levels of the underlying hierarchical structure. That is, Hankin et al. compared only models representing different levels of the hierarchical structure (e.g., a multifactor oblique first-order factor model vs. a single factor model) without ever comparing these models with an integrative model that included both broad and narrow factors. In addition, the current study extends the Hankin et al. results by including measures of sociotropy, autonomy, and AS.
5. Indeed, removing either the Sociotropy, $\chi 2\text{diff}(1, N = 308) = 10.21$, $p < .01$, or the Autonomy, $\chi 2\text{diff}(1, N = 308) = 12.29$, $p < .001$, factors produced a significant decrement in model fit.

6. To further test the possibility of a cognitive- or personality/cognitive-style vulnerability factor in addition to the GNF, we examined the estimates of the correlated residuals among the DAS-A, PSI, and CSQ indicators in the base, hierarchical CFA model (that does not include a factor common to the subscales of the DAS-A, PSI, and CSQ other than the GNF). This examination revealed very little shared variance among the 8 subscales of the DAS-A, PSI, and CSQ remaining after we accounted for the GNF and the Sociotropy, Autonomy, and Negative Cognitive Style factors. In particular, the residual correlations among these 8 subscales ranged from –.001 to .154 with a root-mean-square residual correlation of .068 and a root-median-square residual correlation of .038. Moreover, the shared remaining variance among the 8 subscales of the DAS-A, PSI, and CSQ was quite comparable in magnitude with the remaining variance that the 8 subscales of the DAS-A, PSI, and CSQ shared with the 15 IPIP-NEO-PI-R N, EPQ-R N, Big5 N, and BIS subscales (these residual correlations ranged from –.237 to .132 with a root-mean-square residual correlation of .059 and a root-median-square residual correlation of .027). This pattern is inconsistent with the notion that the DAS-A, PSI, and CSQ subscales all share variance in common with each other that is not also shared with the other indicators of N.
7. That the narrowest AS factors of AS-Physical Concerns, AS-Mental Incapacitation Concerns, and AS-Social Concerns are also distinguishable from more general variance sources and are needed to model the ASI-X in this sample was already demonstrated by Lewis et al. (2010).
8. Additional details regarding tests of configural invariance between men and women can be found in the Testing Configural Invariance of Hierarchical N Model in Men and Women section of the Supplemental Material.

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