Five-year prospective neuroticism-stress effects on major depressive episodes: Primarily additive effects of the general neuroticism factor and stress

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Abstract:

The past decades of research on predictors of depression have frequently emphasized interactive diathesis–stress questions: What kinds of vulnerabilities under stressful circumstances increase risk of developing depression? This study addresses 3 theoretically important gaps in our knowledge regarding diathesis–stress models of depression: the role of temperament (neuroticism), interactive versus additive effects of neuroticism–stress relationships, and effects of stressor characteristics (acute vs. chronic, major vs. minor events, interpersonal vs. noninterpersonal content). We addressed these gaps in the prediction of major depressive episodes (MDEs) in a sample of high schoolers (n = 559) oversampled for high neuroticism and assessed for presence of MDEs annually for 5 years. Survival analyses provided relatively consistent support for the main effects of the broad vulnerability factor of the general neuroticism factor, acute stressors, and chronic stressors in the prediction of MDEs. In contrast, the majority of our analyses failed to support interactive neuroticism–stress accounts of MDE risk. Integrating our results with the extant literature reinforces the notion that both the general neuroticism factor and stress prospectively predict depressive disorders and highlight that their main effects are significantly larger than their interaction.

Keywords: episodic stress | chronic stress | depression | diathesis-stress | neuroticism

Article:

Large epidemiological samples emphasize that depression is widespread among older adolescents and emerging adults with a sharp rise in prevalence in this developmental period (e.g., Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Given the prevalence of major depressive episodes (MDEs), a critical task for researchers who study their development is the identification of risk factors. Investigators have often relied on diathesis– stress models to guide such research efforts. These models posit that individual vulnerabilities and exposure to stressors are both involved in the initiation of psychopathology. Monroe and Simons (1991) explained that the effects of diatheses (or vulnerability factors) and stress can be interactive or additive. Additive diathesis–stress models

postulate that diatheses and stress each have a main effect on psychopathology onset with no multiplicative effect between them. That is, the combined effect of diathetic loading and stress is equivalent to the additive sum of their individual effects. In contrast, interactive diathesis–stress models posit a multiplicative effect between diathesis and stress. That is, their combined effect is greater than the additive sum of their individual effects. Of course, Monroe and Simons noted that it is possible for diatheses and stress to have main effects in addition to a multiplicative effect. Although research has supported interactive diathesis–stress accounts of major depressive onset, comparisons of the effect sizes of main effects for diatheses and stress and their interaction in the generation of MDEs have not been the focus of empirical scrutiny and merit further study. The primary aim of the current study is to conduct such a comparison.

Main Effects of Stress and Depression

Both chronic and episodic stress are implicated in the etiology of MDEs (e.g., Brown & Harris, 1986; Hammen, 2005; McGonagle & Kessler, 1990; Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015). Several characteristics of stressful life events (SLEs) influence the likelihood of a consequent MDE. Severity is the foremost among these. Accordingly, major SLEs— defined on the basis of their objectively threatening psychological impact (i.e., long-term contextual threat)—are predictive of MDEs to a greater degree than minor events (for a review, see Vrshek-Schallhorn, Ditcheva, & Corneau, 2019). The distinction between interpersonal and noninterpersonal stress has also been valuable, with some evidence suggesting that interpersonal stressful events (i.e., those events impacting the condition of one's interpersonal sphere) are particularly depressogenic for adolescents and adults (Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015). Beyond severity and content (interpersonal vs. noninterpersonal), acuteness is another important characteristic of stress. Studies have shown that chronic stress is associated with MDEs, independent of the effects of SLEs (e.g., Monroe, Slavich, Torres, & Gotlib, 2007; Sheets & Craighead, 2014; Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015).

Though highly stressful circumstances are important predictors of depressive episodes, most people who experience major SLEs do not go on to become depressed (Hammen, 2003). It is commonly hypothesized that one pathway from stress exposure to depression runs through preexisting vulnerabilities. Research has shown that individual-differences variables—such as cognitive styles (Alloy et al., 2006; Lewinsohn, Joiner, & Rohde, 2001), particular genotypes (e.g., Caspi et al., 2003; Vrshek-Schallhorn, Stroud, Mineka, Zinbarg, et al., 2015; but see Border et al., 2019), or personality traits (e.g., Zinbarg et al., 2016)—play a role in the etiology of MDEs either considered in isolation from stress or in interaction with stress. The present report focuses primarily on the personality trait of neuroticism as represented by the general neuroticism factor (GNF) of the hierarchical CFA model described in Zinbarg et al. (2016).

Neuroticism as a Vulnerability for Depression

Neuroticism, conceptualized as the trait that reflects individual differences in the experience and expression of negative emotion, has often been hypothesized to be a vulnerability for depression and other internalizing disorders. Importantly, there are competing views of the effects of neuroticism in the literature. Shackman et al.'s (2016) able review of this literature reflected this diversity in noting that neuroticism could raise risk through one or more of three pathways.

One common view, which we will label the stably elevated negative affect hypothesis, asserts that neuroticism is primarily affective and associated with negative affect that is stably elevated under nearly all circumstances (e.g., Bolger & Schilling, 1991; for reviews of the large body of evidence supporting the stably elevated negative affect hypothesis see Shackman et al., 2016 and Watson & Clark, 1984). In other words, the stably elevated negative affect hypothesis is an additive neuroticism– stress model. On the other hand, neuroticism has been defined by others in terms of its capacity to amplify (i.e., interact with) the negatively valenced emotional effects of stressors—what we will label the stress amplification hypothesis (e.g., Clark, Watson, & Mineka, 1994). Thus, Clark, Watson, and Mineka defined the core of the trait as a "sensitivity to negative stimuli" (p. 104). Lahey (2009) captured this idea when he wrote that "neuroticism would have little meaning if persons high in neuroticism did not respond with negative emotions more frequently and intensely when they experience stressful life events." (p. 249). Similarly, in an etiological model of anxiety disorders, we have hypothesized that neuroticism potentiates conditioning of anxiety responses (Mineka & Zinbarg, 2006). Thus, the stress amplification hypothesis is a multiplicative neuroticism–stress model. Finally, neuroticism may contribute to the generation of stressors which, in turn, trigger MDEs—the stress generation hypothesis (e.g., Uliaszek et al., 2012).

The available evidence is consistent with a main effect of neuroticism on depression as well as an interactive effect when combined with SLEs. Kendler, Kuhn, and Prescott (2004) reported evidence consistent with both effects; they interpreted their interaction as showing that in response to events rated by trained interviewers as high on long-term contextual threat, neurotic individuals showed increased risk of an MDE. Though their interaction was statistically significant, our scrutiny of their results reveals that the main effects of neuroticism and SLEs appear to be larger than their interaction. This was borne out when we used the results reported by Kendler et al. (2004) to create confidence intervals (CIs) around their point estimates for the main effects. The absolute value of their point estimate for the interactive effect of neuroticism by high moderate or severe threat events ($b = -.14$) falls outside the 95% CI [0.44, 0.64] of the absolute values of the neuroticism and high moderate or severe threat events main effects, 95% CI [0.48, 0.92]. That is, their interaction of neuroticism by SLEs was significantly smaller than either their main effect of neuroticism or their main effect of SLEs.1

An improved understanding of the relative strength of the main effects of neuroticism and SLEs compared to their interactive effects could have important implications for diathesis–stress models of depression. If main effects are larger than interactive effects, then this may direct research attention away from stress-amplification and toward further study of the direct paths from neuroticism and stress to depression. Such findings would also necessitate a conceptual overhaul of neuroticism on the part of those (including ourselves) who have defined the construct in stress amplification terms (Lahey, 2009; Mineka & Zinbarg, 2006).

Bridging Cognitive and Temperamental Diatheses

Theories focused on cognitive diatheses such as Beck's (1987) schema theory and hopelessness theory proposed by Abramson, Metalsky, and Alloy (1989) have also been tested empirically (e.g., Iacoviello, Grant, Alloy, & Abramson, 2009; Lewinsohn et al., 2001; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). Though cognitive and temperamental diatheses for depression have traditionally been studied in isolation, there is both conceptual and empirical overlap among them. As noted in Zinbarg et al. (2016), many leading neuroticism theorists have explicitly incorporated

cognitive constructs into their definitions of neuroticism. For example, Lilienfeld, Turner, and Jacob (1993) proposed that anxiety sensitivity is a facet of neuroticism. Similarly, Costa and McCrae (1992) considered irrational ideas to be such a facet, Eysenck and Eysenck (1985) viewed low self-esteem to be one and Scheier, Carver, and Bridges (1994) suggested that pessimism is one. Consistent with the hypotheses that cognitive constructs are facets of neuroticism, empirical evidence has consistently shown strong associations of cognitive constructs with neuroticism (e.g., Bagby & Parker, 2001; Dunkley, Blankstein, & Flett, 1997). Thus, prior research that has studied cognitive and temperamental diatheses for depression in isolation leaves open the question of whether effects are due to the broader construct of neuroticism or to the narrower cognitive constructs.

A recent hierarchical2 confirmatory factor analytic (CFA) model of traditional neuroticism measures and a host of measures of cognitive diatheses (e.g., inferential style, sociotropy) teases apart the variance each of these measures share in common from the variance specific to the facets (Zinbarg et al., 2016). This CFA model includes a general factor which is loaded on by each of the measures, including the cognitive diatheses measures. It is labeled the latent GNF. Given that the GNF is loaded on by cognitive diatheses measures as well as measures more traditionally thought of as neuroticism scales, we believe the existing diathesis–stress literatures could be bridged through analysis of how the GNF combines with stress to set risk for depression.

Refining the Candidate Life Stress Variables in a Neuroticism-Stress Multiplicative Model

Are main effects of neuroticism and stress, which, as noted earlier, have already been demonstrated, incompatible with a neuroticism– stress interaction? If there were ceiling effects of the main effects of life stress variables on depression risk (nearly everyone experiencing those life stress variables experiences an onset of depression), life stress variables showing significant main effects could not be associated with amplifying interactions by diatheses (in such a scenario, even those low on the diatheses are at maximal risk for depression after experiencing these life stress variables). Even after experiencing forms of life stress associated with a significant main effect, however, only a minority of individuals experience a depression onset. Therefore, these forms of life of stress are not associated with ceiling effects in precipitating depression. Thus, life stress variables that have demonstrated significant main effects are capable of being associated with amplifying interactions by diatheses.

Moreover, if a life stress variable interacts with a diathesis without either having a main effect, it is most probably the case that the interaction is a crossover interaction. In the case of neuroticism such a crossover interaction would mean that the life stress variable would be associated with elevated risk among those high in neuroticism but would be protective among those low in neuroticism. Given the absence of theory or empirical evidence suggesting crossover interactions between neuroticism and any life stress variable, they seem implausible. Instead, we should expect life stress variables that have demonstrated significant main effects to also be the ones most likely to reveal diathesis by stress interaction effects (Vrshek-Schallhorn et al., 2014). Thus, based on the results of Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015) showing which types of stress produced significant unique main effects, this reasoning suggests that (a) major interpersonal SLEs but not major noninterpersonal SLEs or minor interpersonal, or noninterpersonal SLEs will be involved in neuroticism by stress interactions, and (b) chronic interpersonal stress but not chronic noninterpersonal stress will be involved in neuroticism by stress interactions.

The Current Study

Although research has supported the interactive diathesis–stress account of major depressive onset, the effect sizes of main effects for diatheses and stress and their interaction in the generation of MDEs merit further study. We investigated these effects using our recently developed hierarchical CFA model of neuroticism and its cognitive facets (Zinbarg et al., 2016). This hierarchical CFA model permits a new investigation of diathesis–stress additive and interactive effects that meaningfully extends our understanding of how vulnerability and stress shape risk for depression. Thus, the goal of the current study was to examine whether stressors of different severity (major and minor), content (interpersonal and noninterpersonal), and duration (SLEs and chronic stress) interact with the GNF in the hierarchical neuroticism (i.e., cognitive and temperamental vulnerability) measurement model to heighten risk for MDEs. We used a series of survival analyses to test additive and interactive effects.

We tested six hypotheses. Consistent with past life stress research (e.g., Sheets & Craighead, 2014; Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015), we hypothesized depressogenic main effects of major interpersonal SLEs but not major noninterpersonal SLEs or minor interpersonal and noninterpersonal SLEs (Hypothesis 1). We also hypothesized depressogenic main effects of interpersonal but not noninterpersonal chronic stress (Hypothesis 2). Based on the results of Zinbarg et al. (2016), we expected that the GNF, too, would have a main effect in predicting MDEs (Hypothesis 3). In testing Hypotheses 1–3, we extend our prior demonstrations of prospective prediction of MDEs by the GNF (Zinbarg et al., 2016) and by stress (Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015) by testing whether the prospective prediction by the GNF remains when stress is included in the statistical model and vice versa.

With regard to the potential interactive effects of the GNF and different forms of life stress, we tested the hypothesis derived from the literature on main effects of life stress and the multiplicative stress amplification model of neuroticism that we would find evidence for interactive effects in the prediction of MDEs of the GNF with major interpersonal SLEs but not with major noninterpersonal SLEs, minor interpersonal SLEs, minor noninterpersonal SLEs (Hypothesis 4) and chronic interpersonal stress but not chronic noninterpersonal stress (Hypothesis 5). Based on extant results (e.g., Kendler et al., 2004) and the stably elevated negative affect hypothesis of neuroticism, a final hypothesis we tested is that any such interactive effect would be smaller than the respective main effects of the GNF and the life stress variable (Hypothesis 6). Given correlations among the various forms of life stress, it is possible for one form of life stress to act as a proxy for another in analyses that only included one particular form of life stress (for the correlations among the stress variables, see Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015, Table 2). Thus, we also tested whether significant main effects or interactions in an analysis with a particular form of life stress remained significant in a model simultaneously accounting for all the forms of life stress that had significant effects.

Method

Participants and Screening Procedures

Participants (n = 627) were recruited into the baseline assessment of the Northwestern–University of California at Los Angeles (UCLA) Youth Emotion Project study from the 11th grade of two ethnically and socioeconomically highly diverse high schools: one in suburban Chicago, Illinois,

and the other in suburban Los Angeles, California. Given that many MDEs have their first onset during late adolescence and that this age range involves changing life roles, this is a useful age range in which to study the onset and course of MDEs (Prenoveau et al., 2011). Eleventh-grade students (n = 1,976) who provided assent and parental consent completed a screening questionnaire—a 22-item version of the Neuroticism (N) scale of the revised Eysenck Personality Questionnaire (EPQR-N; Eysenck & Eysenck, 1975). Students completing the screener were categorized as low, medium, and high EPQ-R-N scorers. We invited 1,269 of them into the longitudinal study and oversampled those classified as high-scorers to increase the number of participants likely to develop unipolar mood and anxiety disorders in the follow-up period. We also maintained equal proportions of females to males across the three EPQ-R-N categories. Among the 668 who agreed to participate in the longitudinal study and had parental consent to do so, 627 students completed the baseline assessment, which included an assessment of lifetime Axis I psychopathology using the Structured Clinical Interview for DSM–IV, nonpatient edition (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 2002).

Low, medium, and high EPQ-R-N scoring participants represented 18.4%, 23.0%, and 58.6% of the sample, respectively. The sample was 68.7% female. 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.

Measures

Neuroticism and cognitive diatheses. At baseline, participants completed the following eight vulnerability questionnaires: (a) EPQ-R-N (Eysenck & Eysenck, 1975), (b) the N scale from the International Personality Item Pool representation of the Revised NEO Personality Inventory (Goldberg, 1999), (c) the Behavioral Inhibition Scale (Carver & White, 1994), (d) the N scale from the Big Five Mini-Markers Scale (Saucier, 1994), (e) the Cognitive Style Questionnaire (Alloy et al., 2000; Hankin, Abramson, Miller, & Haeffel, 2004), (f) the Dysfunctional Attitudes Scale (Weissman & Beck, 1978), (g) the Personal Style Inventory (Robins et al., 1994), and (h) the Anxiety Sensitivity Index–Expanded Form (Li & Zinbarg, 2007; Reiss, Peterson, Gursky, & McNally, 1986). The means and standard deviations for these measures are reported in Table 2 of Zinbarg et al. (2010), their reliabilities are reported in Zinbarg et al. (2010), and the correlations among them are reported in Table 2 of Zinbarg et al. (2016). These measures were chosen based on evidence supporting their psychometric properties and their associations with symptoms of anxiety and depression.

Diagnostic measure. The SCID-I/NP (First et al., 2002) was used to assess for Diagnostic and Statistical Manual of Mental Disorders (fourth edition; American Psychiatric Association, 2000) psychiatric diagnoses. Interviews were conducted at the baseline assessment and then every 10–18 months over the subsequent 5 years. All interviewers had at least a bachelor's degree and underwent extensive training and supervision, and interviewers presented each completed SCID-I/NP at a diagnostic consensus meeting led by a doctoral-level supervisor.

Reliability for diagnoses at baseline was assessed by having trained interviewers observe live SCID-I/NPs for 69 cases. Reliability for diagnoses at follow-up assessments was assessed by having trained interviewers listen to a random selection of audio recorded SCID-I/NPs from both sites, including at least 10% of SCID-I/NPs for each time point at each site. When interpreting kappa values, it is important to keep in mind that □ 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 that adjusts for the low base rates. Adjusted k for MDD ranged from .82 to .94. Thus overall, in the context of the low base rates, there was acceptable to very good interrater reliability.

Life stress assessment. Chronic and episodic stress over the past year were assessed at the baseline interview using the UCLA Life Stress Interview (LSI), which elicits specific contextual information about stressors and chronic ongoing conditions (Hammen, 1991; Hammen et al., 1987). The LSI administered at each follow-up interview assessed chronic stress and SLEs occurring in the interim since the previous interview, unless an interview had been missed, in which case only the previous 12 months were assessed. Person-months with missing LSI information were excluded from the present analyses. Chronic stress was measured in 10 life domains: best friend relationship, social circle, romantic relationship, family relationships, academics, work, finances, neighborhood conditions, physical health, and family's health. Ratings for family stress were assigned by the interviewer for each domain on a scale from 1 (best circumstances) to 5 (worst circumstances) in half-point increments using behaviorally specific anchors for each interval of the scale based on objective information. To the extent possible, episodic stressors were excluded from consideration in the rating of chronic stress so as to keep episodic stress ratings and chronic stress ratings operationally distinct. Interrater reliability (interclass correlations) ranged from 0.72 to 0.83 within site and 0.62 to 0.80 cross-site using approximately 10% of interviews.

SLEs were assessed throughout the LSI in each of the 10 life domains. Any additional SLEs not reported in one of the 10 LSI life domains were queried at the interview's conclusion. Interviewers gathered information regarding the context, impact, and date of each SLE, then later presented this information to a team of two or more raters who were blind to the participant's diagnoses. Context based SLE severity ratings were assigned by the consensus of the independent rating team, ranging from 1 (a nonevent, no significant threat or negative implications) to 5 (a very severe event, maximal negative impact or threat) in half-point increments. Each SLE was assigned a code from a modified list of 77 numeric codes (Paykel & Mangen, 1980), describing the nature of each event (e.g., traffic accident, end of a friendship). Cross-site interrater reliability for SLE severity (using 10% of interviews annually) ranged from .69 to .76 (M = .72, SD = .03); due to team rating of SLE severity, no within-site reliabilities are available.

Based on an a priori, contextually based decision applied to all previous published LSI analyses in the present sample, events were classified as major SLEs if assigned a severity rating of 2.5 or greater, reflecting events with moderate to severe levels of contextual impact or threat (e.g., Uliaszek et al., 2012; Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015). Events with a severity rating of 1.5–2.0 were classified as minor SLEs. To classify SLEs as interpersonal or noninterpersonal, two coauthors with LSI experience (S. V. S. and K. W. T.) assigned a category to each of the 77 Paykel codes. Interpersonal SLEs were defined as those events that, in the majority of instances, primarily affect the quality or quantity of the participant's relationships. Agreement was 96% (k = .92); three discrepant ratings were resolved by consensus.

To address temporal precedence for SLEs and MDEs (i.e., whether the SLE preceded and potentially triggered the MDE, or vice versa), in all instances when an MDE and an SLE were dated to the same person-month, trained staff examined records to determine the order of occurrence. When the MDE preceded the SLE, or when the order was indeterminate, the SLE (but

not the MDE or the participant) was excluded from analyses. There were 128 events that occurred in the same study month as MDE onsets. On closer review of the SCID-I/NP and LSI by research assistants, it was clear that in 65 of these instances (51%) that the event occurred prior to the MDE onset. In 19 instances (15%) it was clear that the MDE onset preceded the event occurrence, and thus could not have been caused by the event. Finally, in 44 instances (34%), research assistants classified the order as indeterminant. Interrater reliability for these classifications examined in a subset of 20 of the 128 events was very good (k for determinate = .88), and ratings agreed in 19 of 20 cases. In a prior article, we conducted follow-up sensitivity tests with indeterminate events included in analyses, and the pattern of results was unaltered as compared to excluding them (Vrshek-Schallhorn et al., 2014). As described in Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015), events were dated to the month of occurrence and lagged to a second month to permit each event to be included in statistical prediction of MDEs for two person months.

Assessment Procedure

Participants were contacted by phone or e-mail 10 months after each SCID-I/NP and LSI to schedule the subsequent SCID-I/NP and LSI, and the interval between successive SCID-I/NPs and LSIs was 10 to 18 months. Participants who could not be reached or were unable to complete a particular follow-up assessment in that time frame were contacted for the subsequent follow-up assessment; in all cases the follow-up SCID-I/NPs covered the entire period since the last completed SCID-I/NP. As mentioned previously, LSIs focused on an interval lasting no more than the previous 12 months. All study procedures were approved by institutional review boards at Northwestern University (Protocol Number 00007246) and UCLA (Protocol Number 10–001607). Of the 627 participants who completed the baseline assessment, 559 (89.2%) completed at least one of the five follow-up assessments used here and were included in the present analyses. Those who were included did not differ from those who were excluded on demographic variables or factor scores for the GNF (see Table 1 for details). (As also shown in Table 1, these two groups did not differ on factor scores from the hierarchical neuroticism measurement model corresponding to the unique variance in the diathetic measures derived from Beck's, 1987, schema theory and Abramson et al.'s, 1989, hopelessness theory.)

Data Analysis

All vulnerability measurement model analyses were conducted using Mplus version 7.4 (Mac; Muthén & Muthén, 1998–2017) and proportional hazard survival analyses were conducted using SPSS version 25. Missing data were accommodated using full information maximum likelihood. To test whether the survival analyses data were missing at random, we created a variable corresponding to the number of months of missing data each participant had. The number of months of missing data was not significantly correlated with either GNF factor score estimates (r = -.04) nor with the number of MDEs (r = .01). Nor did the number of months of missing data for men (M = 20.43, SD = 12.07) versus women (M = 19.71, SD = 12.58) differ significantly, t(557) = -0.63. In addition, the number of months of missing data did not correlate reliably with the presence of SLEs (r = .00 averaged across months with only six significant associations across all four types of SLEs and all 59 months of followup) nor with chronic interpersonal stress (r = .04 averaged across months with only six associations significant across 59 months of follow-up). On the other hand, and perhaps unsurprisingly, the number of months of missing data did show a small

positive correlation that was consistently reliable with chronic noninterpersonal stress (r = .09 averaged across months with 30 associations significant across 59 months of follow-up).

Table 1 Baseline Characteristics of Included and Excluded Participants

Variable	Included (*N* = 559)	Excluded (*N* = 68)
Sex		
Female	69.4%	64.7%
Race and ethnicity		
Asian	3.9%	7.4%
Black	13.4%	10.3%
Caucasian	47.8%	51.5%
Hispanic/Latino	15.9%	10.3%
Pacific Islander	0.7%	0.0%
Other	5.2%	7.4%
Multiple	13.1%	13.2%
GNF, *M* (*SD*)	0.00 (0.92)	0.03 (0.88)
Hollingshead SES Score, *M* (*SD*)	47.93 (12.97)	49.32 (12.75)

Note. GNF = general neuroticism factor scores; SES = socioeconomic status. Individuals from the original Youth Emotion Project sample of 627 who were included in the present analyses (N = 559) did not differ from those who were excluded (N = 68) in sex, x2(1) = .63, p = .43; minority racial or ethnic status, x2(6) = 4.54, p = .61; SES, t(611) = .82, p = .41 (Hollingshead, 1975); general neuroticism factor scores, t(625) = .29, p = .77; sociotropy factor scores, t(625) = .32, p = .75; autonomy factor scores, t(625) = .28, p = .78; or inferential style factor scores, t(625) = -1.33, p = .18.

The level of statistical significance in all inferential analyse was $p < .05$, unless otherwise specified. We used factor scores for the latent GNF saved from the hierarchical CFA model of neuroticism and its facets reported by Zinbarg et al. (2016) to test hypotheses in the survival analyses regarding the contributions of these latent vulnerability variables, along with life stress, to the prospective prediction of initial onset of MDEs over a 5-year follow-up period.

Vulnerability measurement model. Zinbarg et al. (2016) reported the results of a CFA of the measurement of the vulnerability measures administered at Time 1 in the Youth Emotion Project. The CFA was conducted using Mplus (Muthén & Muthén, 1998–2017) with full information maximum likelihood to accommodate missing data. Zinbarg et al. reported that model fit was adequate: comparative fit index .92, root mean square error of approximation .056 (90% CI [0.051, 0.060]), and standardized root mean square residual .046. Given that model fit was adequate, we saved GNF factor score estimates from this model and used them to represent the dimension of the GNF in our analyses relating the GNF and life stress variables to onsets of MDE over the follow-up intervals. The factor score determinacy for the GNF factor score estimates equaled .95.

Survival analyses. Proportional hazard survival analyses were conducted using a person-month database with episodes of MDEs as the dependent variable (for details on creation of the person-month database, see Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al., 2015). A total of 21,845 person-months were included with a total of 147 episodes of MDEs including 62 initial episodes and 85 recurrences. Sex was a covariate in all analyses given that MDEs are more common in women than men (e.g., Nolen-Hoeksema & Hilt, 2009), and women score higher than men on neuroticism (e.g., Costa, Terracciano, & McCrae, 2001), as well as on several of our

neuroticism facets (e.g., Hankin & Abramson, 2001). Prior history of MDE was also covaried for at least two reasons. First, this reduced the likelihood of a prospective association of the GNF with future MDE acting as a proxy for a cross-sectional association of the GNF with a history of MDE combined with a history of MDE predicting future MDE. Second, this reduced the likelihood of a prospective association of stress with future MDE acting as a proxy for a history of MDE predicting future MDE combined with stress generation from a prior episode (Hammen, 1991). To closely replicate the analytic strategy of Kendler et al. (2004), we also included the GNF squared and the sex by life stress product as covariates in all analyses.

First, we conducted a series of six survival analyses, each one testing a neuroticism–stress model consisting of the GNF with one of the six forms of life stress. These analyses were conducted hierarchically with main effects (of prior history of MDE, sex, the GNF, and the particular form of life stress) on a first block and multiplicative terms (of the GNF squared, the sex by life stress product, and the GNF by life stress product) on a second block. Finally, we tested whether significant main effects or interactions in an analysis with a particular form of life stress remained significant in a model simultaneously accounting for all the forms of life stress with significant (main or interaction) effects in the first set of analyses.

Results

Effects of the GNF and Different Forms of Stress Examined in Isolation

Taken together, these models provided evidence for significant main effects of the GNF and of several forms of life stress, but not their interactive effects. Consistent with hypotheses 1 and 2, the main effects of major interpersonal SLEs (hazard ratio [HR] = 2.95) and chronic interpersonal stress (HR = 1.81) were depressogenic whereas minor interpersonal (HR = 1.12) and noninterpersonal SLEs (HR = 0.55) were not depressogenic. (Indeed, the effect of minor noninterpersonal SLEs was significant but protective rather than depressogenic). Contrary to Hypotheses 1 and 2, the main effects of major noninterpersonal SLEs (HR = 3.10) and chronic noninterpersonal stress (HR = 1.71) were depressogenic.

As shown in Table 2, for the model including major interpersonal SLEs, the main effect of the GNF (Hypothesis 3) was significant in predicting MDEs. Indeed, this was the case regardless of which form of life stress was also included in the model with HRs of approximately 1.75 (ranging from 1.62 to 1.77, M = 1.74; Tables S1–S5 in the online supplemental materials present the results for the models containing the GNF and the other forms of life stress). The main effect of a prior history of MDE was also significant regardless of which form of life stress was also included in the model with HRs of approximately 2.00 (ranging from 1.87 to 2.19). The interaction of the GNF with life stress was nonsignificant with each of the six forms of life stress with HRs ranging from 0.81 to 1.47. That is, we did not find evidence for the hypothesized interactions between the GNF and any form of life stress, including the hypothesized interactions between major interpersonal SLEs and chronic interpersonal stress (Hypotheses 4–5). Five of the six HRs were nonsignificantly less than 1.0.3

Main Effects of Diatheses and Stress Compared to Their Interactions

For each set of analyses presented in Table 2 and Tables S1–S5 in the online supplemental materials, we also estimated the 95% CI around the point estimates for the main effects of the

diathesis and the form of life stress included in that analysis. To test whether the magnitude of the main effects of the diathesis and stress were larger than that of their interaction, we examined whether the absolute value of the point estimate for each interaction fell outside of the 95% CIs for its two corresponding main effects. Overall, these results supported the prediction expressed in Hypothesis 6 that interactive effects would be smaller than their corresponding main effects. As shown in Table 2 and Tables S1–S6 in the online supplemental materials, the magnitude of the point estimates for the interactions were significantly smaller than the corresponding main effects of the GNF in five of the six analyses involving the GNF (for all these analyses except the one including minor noninterpersonal events as the form of life stress). Similarly, the point estimates for the interactions were significantly smaller than the corresponding main effects of life stress in four of the six analyses involving the GNF (for all these analyses except the ones including minor interpersonal events and minor noninterpersonal events as the forms of life stress).

Did a Violation of the Homogeneity of Regression Slopes Assumption Regarding the Covariate of a History of MDE Bias the Results?

The literature supports the existence of an interaction between a history of MDE and major SLEs in predicting the occurrences of MDE (e.g., Monroe & Harkness, 2005). Thus, it may be that the above analyses violated the assumption of homogeneity of regression slopes with respect to the covariate of a history of MDE. We therefore conducted several follow-up reanalyses to test whether such a violation biased the results of the above analyses.

First, we tested the interaction of history of MDE with major SLEs and this interaction was not significant for either interpersonal (b = -.37, SE = .45, HR = .69, p = .42) or noninterpersonal major SLE (b = -.47, SE = .51, HR = .62, p = .36). To be even more conservative, we reran our models including this interaction despite it not being significant in our sample for either form of major SLE. Doing so did not appreciably alter the pattern of results compared with our original analyses (the results of these reanalyses are available upon request from R. E. Z.). Third, we reanalyzed the data without history of MDE as a covariate and the same pattern of results emerged for both interpersonal (Table S7 in the online supplemental materials) and noninterpersonal (Table S8 in the online supplemental materials) major SLEs. Finally, we also conducted reanalyses of the models including major SLEs restricted to cases who did not have a history of MDE and were therefore predicting only initial onsets. These reanalyses included a total of 16,268 person-months and 62 initial episodes of MDE. The pattern of results that emerged from these reanalyses are shown in Tables S9 and S10 in the online supplemental materials and was almost identical to those from our original analyses. Thus, the evidence from these reanalyses suggests that we can rule out the possibility that excluding the interaction of a history of MDE and major life stress biased the results of our main analyses.

Multivariate General Neuroticism Factor–Stress Model: Unique Effects of the Life Stress Variables

Table 3 presents the results from the multivariate model that included the GNF in addition to each of the five forms of life stress that had significant main effects in the analyses that tested the effects of the different forms of life stress in isolation from the others. Consistent with Hypotheses 1 and 2, the main effects of major interpersonal SLEs (HR 2.46) and chronic interpersonal stress (HR = 1.61) remained depressogenic and minor noninterpersonal SLEs (HR = 0.49) remained

protective. Also consistent with Hypothesis 2, chronic noninterpersonal stress was no longer depressogenic (HR = 1.09) when accounting for the other life stress variables included in this model (each of the other life stress variables remained significant). Contrary to Hypothesis 1, the main effects of major noninterpersonal SLEs (HR = 2.35) remained depressogenic. The GNF also remained a significant predictor.

Table 2. *General Neuroticism Factor Scores (GNF) and Major Interpersonal Stressful Life Events (MajIP) Predicting Major Depressive Episodes*

Model/variable	x^2 *(df)*	*B*	*SE*	95% CI	*HR*
Main effects step	105.64 (4)***				
History		0.73	.18		2.08***
Sex		-0.23	.20		0.80
MajIP		1.08	.21	[0.67, 1.49]	2.95***
GNF		0.57	.10	[0.37, 0.77]	1.76***
Products step[a]	1.22 (3)				
GNF squared		-0.08	.09		0.92
Sex interaction		-0.23	.58		0.79
GNF interaction		-0.11	.27		0.90

Note. CI = confidence interval; HR = hazard ratio.
a Change in x2 in comparison to the previous model, $\Delta x^2(df)$.
***p < .001.

To test whether the magnitude of the unique main effects of the five forms of life stress included in these models differed significantly from each other, we examined whether the overlap of the 95% CIs was no more than half of the average CI arm length for the two CIs being compared (e.g., Cumming, 2009). As derived from the information provided in Table 3, the overlap of the 95% CIs for the two major SLEs with chronic noninterpersonal stress and minor noninterpersonal stress was less than half of the average CI arm length and therefore statistically significant. In addition, the overlap of the 95% CIs for chronic interpersonal stress and chronic noninterpersonal stress was less than half of one CI arm. Finally, the overlap of the 95% CIs for minor noninterpersonal SLEs differed significantly from the two forms of chronic stress. Thus, in terms of their depressogenic effect, the two major SLEs and chronic interpersonal stress were the strongest, chronic noninterpersonal stress was intermediate and minor noninterpersonal SLEs were the weakest (in fact, as already noted, minor noninterpersonal SLEs were protective).

Discussion

Summary of Major Findings

Our results produced four major sets of findings that shed light on the six hypotheses we tested. First are the findings for the main effect of life stress. As predicted, major interpersonal events (Hypothesis 1) and chronic interpersonal stress (Hypothesis 2) had significant and large main effects in the depressogenic direction (greater life stress associated with higher risk of MDE onset) that remained significant even when the life stress variables were entered simultaneously. Contrary to prediction (Hypothesis 1), major noninterpersonal events were depressogenic even when the life stress variables were entered simultaneously. Thus, Hypothesis 1 received mixed support and

Hypothesis 2 was supported by these results. Second are the findings for main effects of neuroticism as a predictor of MDEs (Hypothesis 3) which was significant and moderate to large regardless of which form of life stress was also included in the model. Third are the findings for the multiplicative interactions of the GNF with various forms of life stress (Hypotheses 4–5). The interaction effects were small and none were significant. Fourth, the interaction effects were significantly smaller than the significant main effects of (a) life stress in four of six analyses and (b) neuroticism in five out of six analyses (Hypothesis 6).

Table 3. Unique Main Effects of General Neuroticism Factor Scores (GNF) and Five Forms of Life Stress Predicting Major Depressive Episodes

Predictor	B	SE	95% CI	HR
History	0.54	.19		1.71*
Sex	-0.21	.20		0.81
MajIP	0.90	.22	[0.47, 1.33]	2.46*
MajNon	0.86	.25	[0.37, 1.35]	2.35*
ChronIP	0.48	.12	[0.24, 0.72]	1.61*
ChronNon	0.09	.15	[–0.20, 0.38]	1.09
MinNon	-0.71	.27	[–1.24, -0.18]	0.49*
GNF	0.51	.10	[0.31, 0.71]	1.66*

Note. CI% confidence interval; HR = hazard ratio; Maj = major; Min = minor; Chron = chronic; IP = interpersonal events; Non = nonintepersonal event.
*$p < .05$.

These results extend Zinbarg et al. (2016) by demonstrating that the main effect of the GNF remains when life stress variables are in the statistical model. Similarly, these results extend Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015) by demonstrating that the main effect of major interpersonal events and chronic interpersonal stress remain when the GNF is included in the model. Thus, whereas we have previously reported that neuroticism is associated with stress generation (Uliaszek et al., 2012), the present results suggest that the prospective effects of the GNF in predicting MDEs cannot be entirely attributed to stress generation. The present results also suggest that the prospective effects of major interpersonal events and chronic interpersonal stress cannot be entirely attributed to neuroticism acting as a third variable in stress–MDE associations.

Contrary to the results obtained in Sample 1 of Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015), major noninterpersonal events were depressogenic in these analyses even when accounting for the effects of the other life stress variables. Perhaps the primary differences in the methods between the two reports are that the present analyses included the GNF and those applied to Sample 1 of Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015) included early adversity variables. Interestingly, Sample 2 of Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015) also did not include early adversity variables and produced significant unique main effects of major noninterpersonal events. Thus, perhaps major noninterpersonal events are associated with early adversity variables and the variance each shares with the other is associated with depressogenic effects such that neither has unique depressogenic effects above and beyond the other. Certainly, the results reported here and by Vrshek-Schallhorn, Stroud, Mineka, Hammen, et al. (2015) do not support minor events being depressogenic: major interpersonal events consistently appear to be depressogenic, and major noninterpersonal events may be depressogenic (depending on the other variables included in the analysis). Of course, central to the thinking motivating contextual ratings of life stress is the likely

meaning of the event given the circumstances for the individual. Thus, it is possible that putting emphasis on the external qualities of the life event (e.g., interpersonal vs. noninterpersonal) may miss the mark for indexing the personal meaning, and thereby the depressogenic potential, of the life event.

Implications for Neuroticism–Stress Models of Depression: The Stably Elevated Negative Affect Versus Stress Amplification Neuroticism–Stress Models

Given that both neuroticism and several forms of life stress had significant main effects when entered simultaneously in analyses, the results provide strong support for the additive stably elevated negative affect neuroticism–stress model of MDE etiology. In addition to this very broad implication, the present results, especially when considered together with extant results, have several specific and nuanced implications. The first concerns conclusions about the relative size of main effects and interactions in neuroticism–stress models of MDEs. Importantly, our effect size estimates are strikingly similar to Kendler et al.'s (2004) for the main effects of neuroticism (HR = 1.72, $p < .001$) and stress (HR = 2.36, $p < .001$ [low moderate or higher threat events]; HR = 2.01, $p < .001$ [high moderate or severe threat events]), and their interaction (HR = .87, $p < .001$ [neuroticism by high moderate/severe threat events]). In addition, consistent with our interpretation of Kendler et al.'s results presented in the Introduction, our analyses showed that the magnitude of the interaction effect was significantly smaller than the significant main effects of life stress (in four out of six analyses) and the GNF (in five out of six analyses). Taken together, Kendler et al.'s results and ours suggest that the main effects of neuroticism and stress are substantial whereas interactions between them are smaller. We conclude that the neuroticism–stress model of MDE seems to be primarily driven by additive effects. This runs counter to many theoretical accounts that have focused primarily, if not exclusively, on the interactive effects of neuroticism (e.g., Lahey, 2009; Mineka & Zinbarg, 2006).

Given that our results and those of Kendler et al. (2004) suggest the neuroticism–stress model is primarily characterized by additive effects, there appear to be at least two largely independent pathways to MDE—a high neuroticism pathway and a high stress pathway. Critically, however, exposure to stressors does tend to also "tip the balance" into a MDE among those high in neuroticism. That the effects of the GNF and stress are additive means that they do combine together such that those at greatest risk are individuals high on both the GNF and stress. The relatively small magnitude of the interactive effects relative to the main effects of the GNF and stress merely indicates that their combined effects are not substantially different than the level of risk conferred by adding together their main effects.

The second specific implication of the primacy of additive effects in the neuroticism–stress model concerns the mechanism(s) through which neuroticism confers risk. In their review of the literature, Shackman et al. (2016) identified three etiological mechanisms that have been proposed to account for how neuroticism confers risk: stably heightened negative affect, stress amplification, and stress generation. Given that neuroticism has a substantial main effect above and beyond the main effects of stress, it seems clear that stress generation does not fully account for the risk conferred by neuroticism. Further, the evidence presented here and by Kendler et al. suggests that heightened tonic negative affect plays a substantial role whereas heightened stressor reactivity plays a relatively lesser role. Our results call for new research designed to unravel the mechanistic link between elevated tonic negative affect and risk for MDEs. One such mechanism deserving of investigation is perseverative thought including both rumination on past events and worrying about

future events (e.g., Nolan, Roberts, & Gotlib, 1998; Roberts, Gilboa, & Gotlib, 1998; Ruscio, Seitchik, Gentes, Jones, & Hallion, 2011; Watkins, 2008). Highly neurotic individuals' engagement in perseverative thought may effectively represent a maladaptive coping style with the impact of invariant, high daily distress which heightens vulnerability to MDEs. Building on prior methodologies (e.g., Bolger & Schilling, 1991), future ecological momentary assessment studies would be well-suited to test the potential intervening role of stably heightened negative affect and perseverative thought in the neuroticism–MDE relationship.

The third specific implication of the present results is that they are consistent with a recent emphasis by Barlow and his colleagues (e.g., Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014) on the prominent role of neuroticism in the development of depression (and other emotional disorders). A related point emerges when considering that the index of neuroticism we used, the GNF in the Zinbarg et al. (2016) hierarchical model of neuroticism, is loaded on by both cognitive diatheses measures as well as measures more traditionally thought of as neuroticism scales. Thus, the present results and approach have at least some potential to bridge the neuroticism–stress and cognitive diathesis–stress literatures.

Limitations, Future Directions, and Conclusion

The work reported here has several limitations. We will touch on seven here. First, the finding that a history of MDE was a significant predictor of subsequent MDE above and beyond the effects of the GNF and life stress is an interesting and well-known (e.g., Tram & Cole, 2006) finding but beyond the scope of the current article to properly address.

Second, as noted in Zinbarg et al. (2016), selecting participants on the basis of scores on a neuroticism screening measure might have biased our results. However, simulations reported by Hauner, Zinbarg, and Revelle (2014) suggest that oversampling does not bias statistical tests to a degree that compromises their validity.

Third, our sampling strategy included a narrow age range of older adolescents. Different results may be obtained with samples recruited at younger or older ages. Relatedly, as our baseline assessment revealed high rates of lifetime diagnoses, prevention efforts would do well to use samples recruited at younger ages.

Fourth, the number of months of missing data showed a small positive correlation with chronic noninterpersonal stress. Different results may be obtained with samples without this pattern of differential retention. Indeed, future studies would do well to prioritize retention among participants with elevated chronic noninterpersonal stress.

Fifth, our sample size is too small to have adequate power for detecting small effects as the neuroticism–stress interaction is estimated to be on the basis of our results and those reported by Kendler et al. (2004). Thus, we do not conclude from our results that such interactions do not exist. Kendler et al. (2004) found a significant interaction between neuroticism and stress, but with a sample 11 times larger than ours. Compared to the present analyses, Kendler et al. had much greater statistical power to detect a small effect. For this reason, we are inclined to believe that our results are attributable to Type II error because we failed to detect the neuroticism–stress interaction that is likely present, albeit small, as suggested by the results of Kendler et al.

Sixth, the LSI takes a different approach to scoring chronic stress than does the approach taken by Brown and Harris (1986). For example, Brown and Harris dichotomized chronic stress to distinguish major chronic stress from nonmajor chronic stress. Thus, an important direction we

will pursue in future research will be to test whether these different approaches can be reconciled and to identify the optimal approach to scoring chronic stress.

Finally, though an understudied topic relative to the stably elevated negative affect and stress amplification hypotheses, there are a few studies linking neuroticism to elevated mood variability (e.g., Kuppens, Van Mechelen, Nezlek, Dossche, & Timmermans, 2007; Murray, Allen, & Trinder, 2002). Future research clarifying how such variability might produce elevated risk for MDEs and the contribution of such variability relative to that of stably elevated negative affect and stress amplification has the potential to shed light on an additional pathway linking neuroticism to MDE etiology.

Although the results reported here have limitations, they also have notable strengths. Our sample was relatively large for a multiyear prospective study that utilized diagnostic interviews and contextual-threat life stress interviews. We also included measures of both cognitive and temperamental vulnerabilities which enabled the modeling of variance due to a GNF that is shared by cognitive vulnerability measures. In conclusion, the present results, taken together with those of Kendler et al. (2004), provide more support for the stably elevated negative affect model of neuroticism—a neuroticism–stress model of the etiology of MDEs that is primarily characterized by additive effects—than for a stress amplification model of neuroticism characterized by multiplicative effects. Thus, future research on the mechanism(s) underlying the risk for MDEs predicted by neuroticism will likely make the largest impact by focusing on heightened tonic negative affect.

Notes

1 The results of Ormel, Oldehinkel, and Brilman (2001) are also relevant though limited in several important ways. First, this study did not use a completely prospective design (neuroticism was measured prior to the assessment of depression and SLEs but depression and SLEs were each only assessed at one point in time). Second, it is restricted to individuals aged 57 or older which is much older than the typical age of onset of depression. Third, Ormel et al.'s interpretation appears at odds with their statistical results in that they concluded that neuroticism interacted with SLEs but their result for a model that didn't include an interaction term (odds ratio 8.68) was not smaller than the lower limit of the CI for a model that included the interaction (95% CI [6.20, 38.90]), and therefore, the two are not significantly different. Moreover, this was the case even though they adopted a one-tailed p value such that the lower limit of their CI was based on a p value of .05 rather than .025 as would be the case for a two-tailed test. Despite this, Ormel et al. concluded that neuroticism amplifies the effects of life stress.

2 The term currently in vogue for the model that, following McDonald (1999), we refer to as the hierarchical factor model is the bifactor model. We prefer the term hierarchical because it does not imply how many factors most indicators load on, whereas the term bifactor originated in IQ research to describe a model in which all indicators loaded on two factors (a general factor and a specific factor). Thus, the term hierarchical factor model is broader than the term bifactor model and can therefore be used to highlight the conceptual connection between models such as the bifactor IQ model with a model such as the trilevel model of symptoms of anxiety and depression (Prenoveau et al., 2010), in which most indicators load not only on a general factor and a specific factor but also on a factor of intermediate breadth.

3 We also conducted a series of survival analyses testing diathesis–stress models for cognitive diatheses using factor score estimates from the Zinbarg et al. (2016) hierarchical neuroticism model corresponding to the residualized facets of sociotropy, autonomy, and inferential style. This comprised a series of six survival analyses for each of the three residualized cognitive diatheses, for a total of 18 analyses, each one testing a diathesis–stress model consisting of the given diathesis with one of the six forms of life stress. The

main effect of the diathesis was not significant in any of the 18 models that included residualized sociotropy, autonomy, or inferential style as predictors (all main effect HRs were between 0.88 and 1.17, all main effect ps .23). The analysis including residualized sociotropy and chronic interpersonal stress (see Table S6) was also the only one, including the six models that incorporated the GNF as a predictor, in which one of the diatheses significantly interacted with a form of life stress (all other interaction HRs involving residualized sociotropy, autonomy, and inferential style were between 0.55 and 1.74; all other interaction ps .13). However, the significant interaction of residualized sociotropy by chronic interpersonal stress has a negative sign. Simple slope analyses of the influence of chronic interpersonal stress predicting MDE onset revealed regression coefficients of 1.10, 0.67, and 0.24, respectively, for those –1 SD from the mean, at the mean, and 1 SD above the mean on residualized sociotropy. Thus, the interaction was driven by residualized sociotropy becoming a weaker predictor of MDE episodes as chronic interpersonal stress increased. Interestingly, Schweizer, Snyder, and Hankin (2020) reported that the main effect of a residualized dependency factor, which seems conceptually similar to our residualized sociotropy variable, had a negative correlation with depression symptoms. Thus, Schweizer et al.'s suggestion that residualized dependency may tap adaptive social motivation may also apply to residualized sociotropy.

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