The Direct and Interactive Effects of Neuroticism and Life Stress on the Severity and Longitudinal Course of Depressive Symptoms

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The direct and interactive effects of neuroticism and stressful life events (chronic and episodic stressors) on the severity and temporal course of depression symptoms were examined in 826 outpatients with mood and anxiety disorders, assessed on 3 occasions over a 1-year period (intake and 6- and 12-month follow-ups). Neuroticism, chronic stress, and episodic stress were uniquely associated with intake depression symptom severity. A significant interaction effect indicated that the strength of the effect of neuroticism on initial depression severity increased as chronic stress increased. Although neuroticism did not have a significant direct effect on the temporal course of depression symptoms, chronic stress significantly moderated this relationship such that neuroticism had an increasingly deleterious effect on depression symptom improvement as the level of chronic stress over follow-up increased. In addition, chronic stress (but not episodic stress) over follow-up was uniquely predictive of less depression symptom improvement. Consistent with a stress generation framework, however, initial depression symptom severity was positively associated with chronic stress during follow-up. The results are discussed in regard to diathesis–stress conceptual models of emotional disorders and the various roles of stressful life events in the onset, severity, and maintenance of depressive psychopathology.

Keywords: temperament and psychopathology of depression, predictors of longitudinal course of mood disorders, role of life stress in severity and temporal course of depression, diathesis–stress models of emotional disorders

Over the past several decades, sizeable literatures have accrued on the constructs that act as putative risk factors for unipolar depression. Although a number of vulnerability dimensions have been identified, a considerable portion of this research has focused on two constructs: neuroticism and stressful life events. Indeed, there are two largely separate literatures that attest to the empirical and conceptual significance of neuroticism and stressful life events in predicting the onset, severity, and course of depression (for reviews, see Hammen, 2005; Kessler, 1997; Mineka, Watson, & Clark, 1998). However, fewer studies have examined simultaneously the roles of neuroticism and stressful life events on depression, and thus the nature of the relationships between these variables is not well understood. Moreover, most studies of life stress and depression have examined episodic stressors, despite recent indications of the relevance of ongoing strains in predicting the severity and course of depression symptoms. Thus, the goal of the present study was to examine the nature of the effects of neuroticism, chronic stress, and episodic stress on the severity and temporal course of depression symptoms in a large sample of outpatients with mood and anxiety disorders.

Neuroticism

Most prominent theories underscore neuroticism as a genetically based core dimension of temperament that is instrumental to the etiology and course of the mood disorders (see e.g., Barlow, 2002; Mineka et al., 1998). Indeed, extensive evidence indicates that neuroticism is strongly heritable (see e.g., Fanous, Gardner, Prescott, Cancro, & Kendler, 2002; Hettema, Prescott, & Kendler, 2004; Viken, Rose, Kaprio, & Koskenvuo, 1994), temporally stable (see e.g., Costa & McCrae, 1988), and plays a key role in accounting for the severity, overlap, and maintenance of depression and anxiety (see e.g., Bienvenu et al., 2001, 2004; Brown, 2007; Gershuny & Sher, 1998). Despite the considerable empirical efforts expended on this issue, the specific nature of the relationship between neuroticism and depression has remained elusive. For instance, although leading conceptualizations emphasize the possibility that neuroticism is a vulnerability to emotional disorders (a *predispositional* explanation; see e.g., Barlow, 2002; Clark, 2005), most studies have not been methodologically equipped to address this hypothesis in a compelling fashion (e.g., inability to evaluate directional effects in cross-sectional designs; lack of premorbid assessment of neuroticism in studies using clinical samples). Given that conceptual models also posit that neuroticism predicts a poor prognosis for the treated and untreated course of emotional disorders (see e.g., Mineka et al., 1998), longitudinal studies of clinical samples have addressed the nature of the relationship between temperament and psychopathology within a *pathoplastic* framework (i.e., temperament influences the course and expression of disorders). Several studies of this

nature have emanated from treatment outcome research for major depression and dysthymia. These studies are based on the premise that because constructs such as neuroticism are construed as risk factors for depression, these dimensions should predict depression treatment outcome (i.e., high neuroticism is associated with poorer treatment response). However, whereas some studies have found that higher pretreatment levels of neuroticism predict poorer outcomes for depression (see e.g., Bock, Bukh, Vinberg, Gether, & Kessing, 2010; Hayden & Klein, 2001; Quilty et al., 2008), others have failed to support this relationship (see e.g., Clark, Vittengl, Kraft, & Jarrett, 2003; Petersen et al., 2002).

Brown (2007) examined the longitudinal course and temporal structural relationships of dimensions of temperament (i.e., a latent variable defined by indicators of neuroticism and behavioral inhibition [N/BI] and a latent variable defined by indicators of behavioral activation and positive affect [BA/P]) and selected disorder constructs (depression, social phobia, generalized anxiety disorder) according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) in outpatients with anxiety and unipolar mood disorders ($N = 606$) who were assessed at intake and at 1-year and 2-year follow-ups. Most patients (76%) received treatment after intake, and thus the overall rate of anxiety and mood disorders declined over the follow-up period (e.g., 100% to 58% for intake and 2-year follow-up, respectively). A series of parallel-process latent growth models indicated that, holding intake levels of the *DSM–IV* construct constant, initial levels of N/BI were associated with less improvement in social phobia and generalized anxiety disorder. However, neither N/BI nor BA/P was predictive of the temporal course of depression. In view of theory and limited evidence that the effects of vulnerability on psychopathology are augmented by stressful life events, Brown (2007) raised the possibility that the influence of N/BI on the course of the *DSM–IV* disorder constructs was underestimated in this study due to the failure to include measures of life stress.

Indeed, in addition to the aforementioned methodological issues (e.g., overreliance on cross-sectional designs), most studies to date have focused exclusively on the direct effects of neuroticism on depression. This is despite the fact that current conceptual models assert that dimensions of temperament do not act alone in determining the etiology, course, and complications of emotional disorders (see e.g., Barlow, 2002). Specifically, such models espouse a "diathesis–stress" conceptualization such that vulnerable individuals (e.g., those high in neuroticism) are posited to be at higher risk for experiencing psychopathology in the context of triggering stressful events. Moreover, the construct of neuroticism itself has been conceptualized in part as the tendency to experience negative emotions and react adversely to stressors (see e.g., Costa & McCrae, 1992). Along these lines, studies using nonclinical samples have found an interaction between neuroticism and daily stress/hassles in predicting negative affect over short-term periods (see e.g., Hutchinson & Williams, 2007; Mroczek & Almeida, 2004). In a population-based sample of twins, Kendler, Kuhn, and Prescott (2004) found that individuals with high neuroticism were significantly more likely than individuals with low neuroticism to experience a major depressive episode when exposed to stressful life events.

Life Stress

A large literature has been amassed on the role of life stress in the development and maintenance of depression. The consistent finding that stressful life events precede major depressive episodes has led many researchers to underscore the probable existence of a causal effect of stress on the onset of depression in vulnerable individuals (for reviews, see Hammen, 2005; Kessler, 1997; Mazure, 1998; Tennant, 2002). Evidence for a bidirectional relationship between stress and depression has also been obtained; specifically, that in addition to the precipitating effects of life stress on depression, depressed individuals are more likely to experience subsequent stressful events (i.e., *stress generation*; Hammen, 1991). Furthermore, concurrent stress (i.e., stressors during the study follow-up period) has been associated with poorer depression outcome over various follow-up periods in treatment outcome (see e.g., Monroe, Kupfer, & Frank, 1992; Monroe, Roberts, Kupfer, & Frank, 1996; Zlotnick, Shea, Pilkonis, Elkin, & Ryan, 1996) and naturalistic studies (see e.g., Lehmann, Fenton, Deutsch, Feldman, & Engelsmann, 1988; Spijker, Bijl, de Graaf, & Nolen, 2001), although some null findings have been reported (Monroe, Bellack, Hersen, & Himmelhoch, 1983; Wildes, Harkness, & Simons, 2002). Less consistent results have been obtained for the influence of antecedent stressors on the course and treatment outcome of depression. For instance, whereas some studies have found that pretreatment stressful events predict poorer response to depression treatment (see e.g., Monroe et al., 1992, 1996), others have not (see e.g., Monroe et al., 1983; Reno & Halaris, 1990).

Notably, most research on the relationship of life stress and depression has focused on episodic stressors (i.e., discrete life events such as the death of a loved one or an automobile accident). In response to concerns that the field had erroneously neglected the potential effects of ongoing stressors (e.g., social isolation, family conflict, work dissatisfaction, financial hardship; Hammen, 2005), a smaller body of research has emerged on the role of chronic stress in predicting the onset and symptom severity of depression (see e.g., Hammen, Kim, Eberhart, & Brennan, 2009; Hammen, Shih, & Brennan, 2004; Monroe, Slavich, Torres, & Gotlib, 2007; Muscatell, Slavich, Monroe, & Gotlib, 2009). Some evidence has suggested that chronic stressors are more strongly predictive of depression symptom severity than are episodic stressors (see e.g., McGonagle & Kessler, 1990). Researchers have also raised the possibility that episodic and chronic stressors are differentially related to depression. For instance, many studies have found that episodic stress is more strongly associated with first episodes of depression than with depression recurrences (see e.g., Hammen, 2005). Chronic stress, however, may be more germane to the maintenance of depressive episodes (Hayden & Klein, 2001; Moerk & Klein, 2000; Riso, Miyatake, & Thase, 2002). Yet, few studies have examined the influence of chronic stress on the temporal course of depression. In a study of 97 patients with dysthymia, Hayden and Klein (2001) found that chronic stress during the follow-up period was a strong predictor of disorder nonrecovery and depression symptom severity at 5-year follow-up. This relationship was replicated in another investigation of this sample using a longer follow-up period (7.5 years; Dougherty, Klein, & Davila, 2004). However, these studies did not include measures of episodic stress, thereby precluding evaluation of the

differential relevance of chronic and episodic stress in the prediction of the course of depression.

Studies Examining Both Neuroticism and Life Stress

By and large, two separate literatures have developed on the influence of neuroticism and life stress on the emergence and course of depression. However, a smaller set of studies have conducted simultaneous evaluations of these constructs. These studies have often examined, in addition to direct effects, conceptually driven indirect and interactive effects of neuroticism and life stress on depression. Mediational models are based on the notion that neuroticism plays a role in stress generation (see e.g., Bolger & Zuckerman, 1995; Fergusson & Horwood, 1987; Kendler, Gardner, & Prescott, 2003) and that these stressors subsequently lead to emotional distress (i.e., depression). In a general population sample, for instance, Ormel and Wohlfarth (1991) found that neuroticism exerted an indirect effect on general psychological distress though chronic stress (i.e., long-term difficulties) over an 8-year period. Similar findings have been obtained in briefer prospective studies that examined the relationships between neuroticism (or negative emotionality), episodic stressors (or daily life events), and symptoms of depression in adolescents and undergraduates (Hankin, 2010; Kercher, Rapee, & Schniering, 2009; Lakdawalla & Hankin, 2008; Wetter & Hankin, 2009). However, a study of elderly individuals failed to find that chronic or episodic stressors mediated the effect of neuroticism on depressive episode onset (Ormel, Oldehinkel, & Brilman, 2001).

In accord with diathesis–stress models of psychopathology (i.e., life stress potentiates the effects of neuroticism), some studies have also evaluated the multiplicative effects of neuroticism, chronic stress, and episodic stress on depression. Evidence for moderating effects of episodic stress on the relationship between neuroticism and depression has been mixed. For example, significant Neuroticism × Episodic Stress interactions have been obtained in the prediction of depression onset (see e.g., Kendler et al., 2004; Ormel et al., 2001) but not the severity of depression symptoms (see e.g., Hankin, 2010; Wetter & Hankin, 2009). Few studies have considered the moderating effects of chronic stress on the relationship between neuroticism and depression. In an elderly sample, Ormel et al. (2001) found that individuals with high levels of neuroticism were at increased risk of depression onset if they were also experiencing long-term difficulties. Although Hayden and Klein (2001) found that higher levels of both neuroticism and chronic stress were associated with less improvement in depression among individuals with dysthymia, significant interaction effects were not reported. To our knowledge, no studies to date have evaluated simultaneously the roles of neuroticism, chronic and episodic stress, and their interaction in the prediction of the temporal course of depression in adult clinical samples.

Present Study

Accordingly, the present study examined the direct and interactive effects of neuroticism, chronic stress, and episodic stress on the severity and temporal course of depression symptoms in a large and diagnostically diverse sample of treatment-seeking outpatients who were followed over a 1-year period. On the basis of prevailing research findings, it was predicted that chronic and episodic stress

would be positively associated with initial depression severity and that less improvement in depression would be seen in patients who experienced higher levels of life stress during the follow-up period (perhaps with stronger effects for chronic stress than episodic stress given the potentially greater relevance of chronic stress in depression maintenance; cf. Hayden & Klein, 2001; Moerk & Klein, 2000; Riso et al., 2002; Tennant, 2002). Although neuroticism was expected to have a significant unique association with initial depression severity, any effects of this dimension on the temporal course of depression (cf. Brown, 2007) would be significantly potentiated by the level of life stress experienced during the follow-up period (again, with the possibility that chronic stress would have a stronger moderating effect). Specifically, it was hypothesized that the deleterious effects of neuroticism on the course of depression would significantly increase as the level of exposure to life stress during follow-up increased. In addition to initial depression severity, these relationships were examined controlling for extraversion. Along with neuroticism, the construct of extraversion has been identified in conceptual models and research as a core dimension of temperament with specific relevance to mood disorders, as well as social phobia (see e.g., Brown & Barlow, 2009; Mineka et al., 1998). Given the potential role of extraversion in accounting for individual differences in the severity and course of depression symptoms, a measure of this construct was included as an exogenous variable in the cross-sectional and longitudinal analyses (e.g., are there direct and interactive effects of neuroticism and life stress on depression holding extraversion constant?).

Method

Participants

The sample consisted of 826 outpatients who presented for assessment or treatment at the Center for Anxiety and Related Disorders.[1] Women constituted the larger portion of the sample (60.4%); average age was 33.62 years ($SD = 12.52$, range = 18–79). The sample was predominantly Caucasian (86.3%; African American = 4%, Asian = 4.8%, Latino/Hispanic = 4.5%, Other/Mixed = 0.4%). Intake diagnoses (Time 1 [T1]) were established with the Anxiety Disorders Interview Schedule for *DSM–IV*–Lifetime version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994), a semistructured interview designed to ascertain reliable diagnosis of the *DSM–IV* anxiety, mood, somatoform, and substance use disorders and to screen for the presence of other conditions (e.g., psychotic disorders). Patients were reevaluated at 6 months (Time 2 [T2]) and 12 months (Time 3 [T3]) using the follow-up version of the ADIS-IV, which is identical to the ADIS-IV-L except that (a) sections for past diagnoses are omitted and (b) a section is included to assess treatment follow-up (e.g., nature and extent of treatments received since intake). Both ADIS-IV versions provide dimensional assessment of the key and associated features of disorders (0–8 ratings); such features are dimensionally rated regardless of whether a formal *DSM–IV* diagnosis is under consideration. A reliability study entailing two independent adminis-

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[1] There was no overlap between the current sample and the sample reported in Brown (2007).

trations of the ADIS-IV-L indicated good-to-excellent interrater agreement for current disorders (range of κs = .67 to .86) except dysthymia (κ = .31; Brown, Di Nardo, Lehman, & Campbell, 2001). The rates of current clinical disorders occurring frequently in the sample at intake were as follows: social phobia (47.6%), mood disorders (i.e., major depression, dysthymic disorder, depressive disorder not otherwise specified [NOS]; 39.8%), generalized anxiety disorder (29.4%), panic disorder with or without agoraphobia (24.5%), obsessive–compulsive disorder (16.7%), and specific phobia (15.4%). Of the 329 cases with a current mood disorder, 251 had major depression (160 recurrent, 91 single episode), 64 had dysthymia (45 early onset), and 17 had depressive disorder NOS (total exceeds 329 because three cases were diagnosed with double depression).

Measures

Neuroticism and extraversion. Neuroticism and extraversion were assessed at intake with the NEO Five-Factor Inventory (NFFI; Costa & McCrae, 1992). The NFFI is a 60-item self-report measure of the five-factor model of personality. Items are composed of self-descriptive statements rated on a 5-point Likert scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). The five domain scores (e.g., neuroticism [NFFI-N], extraversion [NFFI-E]) are calculated by summing their respective 12-item responses. The latent structure of the NFFI has been supported in clinical samples (Rosellini & Brown, 2011), and each domain has been found to possess adequate internal consistency (α = .68 to .86; Costa & McRae, 1992) and temporal stability (e.g., in normal samples, rs = .86 to .90; Robins, Fraley, Roberts, & Trzesniewski, 2001).

Chronic and episodic life stress. Chronic and episodic life stress were assessed at intake and at both follow-up evaluations using the UCLA Life Stress Interview (UCLA-LSI; Hammen et al., 1987). The UCLA-LSI is a semistructured interview that assesses stress occurring over the prior 6 months in eight domains: social life, romantic relationships, family, work, school, finances, health of self, and health of others. Assessment of chronic stress in multiple domains has been emphasized by depression researchers (see e.g., Mazure, 1998). As in prior longitudinal studies examining stress and depression outcomes (see e.g., Hayden & Klein, 2001), the UCLA-LSI defines chronic stress as a strain lasting at least 6 months. Interviewers made a chronic stress rating for each domain on a 5-point scale ranging from 1 (*exceptionally positive circumstances*) to 5 (*extremely adverse circumstances*) in increments of 0.5 using descriptive behavioral anchors. Within the social life domain, for instance, a rating of 2 reflects having a number of friends, weekly socializing (e.g., in person or via telephone), diversity in activities, and good conflict resolution, whereas a rating of 4 would be used to describe a limited number of friends (e.g., one or two), infrequent social contact, engagement in limited social activities every few months, and poor conflict resolution. Chronic stress at intake (CS_I) was a sum composite of the eight stress domain ratings assessed at T1; chronic stress during follow-up (CS_F) was a composite of the stress domains collapsing the T2 and T3 assessments.

Episodic stress referred to any acute events that had occurred over the prior 6 months. Specifically, patients were asked whether there had been "any particular event that had occurred" in a specific domain (e.g., social life, romantic relationships, family).

When inquiring about episodic stressors, interviewers provided patients with examples of what would constitute a "particular event." For example, interviewers asked patients whether any major arguments or separations had been present when assessing for episodic stress within social, romantic, and family domains. Within health domains (self and others), patients were asked whether any acute illnesses, accidents, or injuries had occurred. In addition to assessment of episodic stress within the eight domains mentioned earlier, acute events regarding migration, bereavement, legal difficulties, crime, and auto accidents were assessed (i.e., 13 episodic stress domains were assessed in total). Information was gathered about the nature of the episodic events (i.e., surrounding circumstances), coping resources, and consequences. Interviewers then made ratings of the impact of the particular event on a 5-point scale ranging from 1 (*no impact*) to 5 (*severe impact*) in increments of 0.5. Up to three episodic stressors were rated within each domain. A rating of 0 was assigned if the episodic stress was absent. As with chronic stress, episodic stress at intake (ES_I) was a sum composite of the T1 episodic stress ratings, whereas episodic stress over follow-up (ES_F) was a composite of the ratings summing across T2 and T3.

Depression. A latent variable of unipolar depression (DEP) was formed using the following two questionnaire indicators and ADIS-IV clinical rating composite (collected at each assessment): (a) Beck Depression Inventory (BDI; Beck & Steer, 1987); (b) Depression scale of the 21-item version of the Depression Anxiety Stress Scales (DASS-D; Lovibond & Lovibond, 1995; cf. Antony, Bieling, Cox, Enns, & Swinson, 1998; Brown, Chorpita, Korotitsch, & Barlow, 1997); and (c) the ADIS-IV dimensional ratings of the nine-symptom criteria of *DSM–IV* major depression, which ranged from 0 (*none*) to 8 (*very severe*; interrater r = .74, Brown et al., 2001). In accord with prior studies (see e.g., Brown, 2007; Brown, Chorpita, & Barlow, 1998), the BDI was scored using the 10 items that load on a Cognitive/Affective factor (Items 1–9, 13) because they are more specific to the unipolar mood disorders. As in Brown (2007), the DASS-D was used as the marker indicator for the DEP latent variable. Observed DASS-D scores were multiplied by two to foster comparability to the unstandardized DEP metric reported in Brown (2007), who used the 42-item version of the DASS (thus, the possible range of DASS-D scores was 0 to 42, with higher scores reflecting more severe depressive symptoms).

Data analysis. The raw data were analyzed using a latent variable software program and maximum-likelihood minimization functions (Mplus 6.0; Muthén & Muthén, 1998–2010). Missing data due to attrition (25% at T2, 40% at T3) were accommodated in all analyses using direct maximum likelihood (cf. Allison, 2003; Raykov, 2005). Goodness of fit of the models was evaluated using the root-mean-square error of approximation (RMSEA) and its 90% confidence interval (CI) and test of close fit (CFit), the Tucker–Lewis index (TLI), the comparative fit index (CFI), and the standardized root-mean-square residual (SRMR). Acceptable model fit was defined in part by the criteria forwarded by Hu and Bentler (1999): RMSEA values close to .06 or below (90% CI upper limit close to .06 or below, nonsignificant CFit), CFI and TLI values close to .95 or above, and SRMR values close to .08 or below. In the case of nested models (e.g., evaluation of longitudinal measurement invariance), comparative fit was evaluated with chi-square difference tests (χ^2_{diff}). The acceptability of the models

was further evaluated by the presence/absence of salient localized areas of strains in the solutions (e.g., modification indices) and the strength and interpretability of the parameter estimates.

Results

Cross-Sectional Analyses

The first set of analyses entailed structural regression models to determine whether chronic stress (CS_I) and episodic stress (ES_I) over the 6 months preceding the intake evaluation had significant main effects on T1 depression severity and whether CS_I and ES_I moderated the influence of neuroticism on depression severity. As noted previously, depression severity was a latent variable (DEP) defined by three observed measures. Because this measurement model was just-identified ($df = 0$; cf. Brown, 2006), goodness-of-fit evaluation was not germane to this set of analyses (completely standardized factor loadings were .90, .89, and .77 for DASS-D, BDI, and ADIS-IV major depression disorder dimensional ratings, respectively; all $ps < .001$).

Chronic stress. To examine the main effects of neuroticism and chronic stress on depression severity at intake, the DEP latent variable was regressed onto the T1 measures of CS_I and neuroticism (N). As seen in Table 1, both main effects were statistically significant ($ps < .001$) and collectively accounted for 57.6% of the variance in DEP. Next, the $CS_I \times N$ product term was included as a predictor, which resulted in a significant increase in the model R^2 ($p < .001$, $f^2 = .02$, a small effect per the standards set forth by Cohen, 1988). The regression coefficient for the product term was positive, indicating that the strength of the effect of neuroticism on depression increased as chronic stress increased. The nature of this interaction effect is depicted in Figure 1.

Figure 1. Nature of the interaction of neuroticism and chronic stress on depression symptoms at intake. CS_I = chronic stress in the 6 months preceding the intake evaluation. The possible range of scores for the unstandardized depression latent variable is 0 to 42.

These analyses were rerun controlling for extraversion (NFFI-E). As expected, extraversion had a significant direct effect on DEP (unstandardized and completely standardized γs = -0.09 and $-.07$, respectively; $ps = .02$). The inclusion of extraversion did not affect the size or statistical significance of the N and CS_I main effects, or the N $\times$ CS_I interaction (e.g., unstandardized γ for product term continued to be 0.03; $p < .001$).

Episodic stress. As shown in Table 1, ES_I had a significant main effect on DEP ($p = .003$), holding N constant. This direct effect remained statistically significant controlling for extraversion (unstandardized and completely standardized γs for ES_I = -0.16 and $-.10$, respectively; $ps < .001$). In addition, a model in which ES_I and CS_I were simultaneously included as predictors (along with the neuroticism and extraversion covariates) indicated that both episodic stress and chronic stress explained significant unique variance in initial depression severity ($ps < .03$ and $.001$ for ES_I and CS_I, respectively). However, the N $\times$ ES_I product term did not significantly contribute to the prediction of DEP ($f^2 = 0$), indicating that the effect of neuroticism on depression severity did not vary as a function of the level of episodic stress.

Longitudinal Analyses

Diagnostic outcome over follow-up. Nearly three quarters (74.1%) of the sample received treatment at the Center for Anxiety and Related Disorders after the intake assessment. As expected, the overall rate of emotional disorders in the sample declined by 6-month follow-up (i.e., from 100% to 74.8%; McNemar test $p < .001$), with relatively less additional change at the 12-month assessment (69.4%, $p = .04$). A similar pattern was found specifically for the *DSM–IV* mood disorders (i.e., 39.6%, 25.5%, and 20.4% for T1 through T3, respectively); although the T1 to T2 reduction in mood disorders was significant ($p < .001$), the additional decline at T3 was not ($p = .15$).

Unconditional longitudinal models. To establish the suitability of the DEP latent variable as an outcome in the subsequent latent growth models (LGMs; i.e., to ensure that temporal change in the DEP latent construct was not confounded by change in its measurement over time), a longitudinal confirmatory factor analytic model was evaluated. As shown in Figure 2, indicators of the same variable assessed at different times (e.g., DASS-D_1, DASS-

Table 1
Cross-Sectional Structural Regression Models of the Effects of Chronic Stress, Episodic Stress, and Neuroticism on Depression

Variable	γ	SE_γ	γ^*	SE_{γ^*}
Chronic stress				
Main effects (model R^2 = .58)				
CS_I	0.60***	0.08	.23***	.03
N	0.69***	0.03	.65***	.02
Interaction effect (model R^2 = .59, f^2 = .02)				
N $\times$ CS_I	0.03***	0.01	.69***	.18
Episodic stress				
Main effects (model R^2 = .54)				
ES_I	0.14**	0.05	.08**	.03
N	0.76***	0.03	.72***	.02
Interaction effect (model R^2 = .54, f^2 = .00)				
N $\times$ ES_I	0.00	0.01	.01	.11

Note. γ = unstandardized path coefficient, γ^* = completely standardized path coefficient; N = neuroticism; CS_I = chronic stress in the 6 months preceding the intake evaluation; ES_I = episodic stress in the 6 months preceding the intake evaluation.
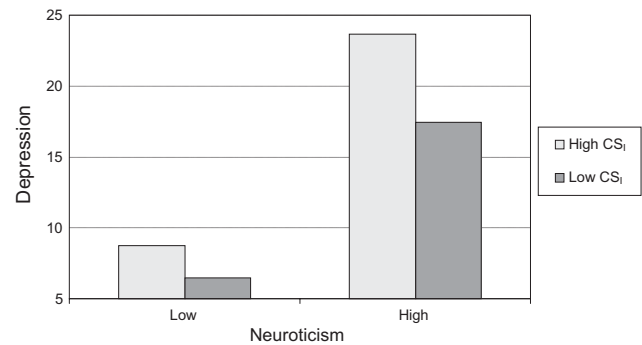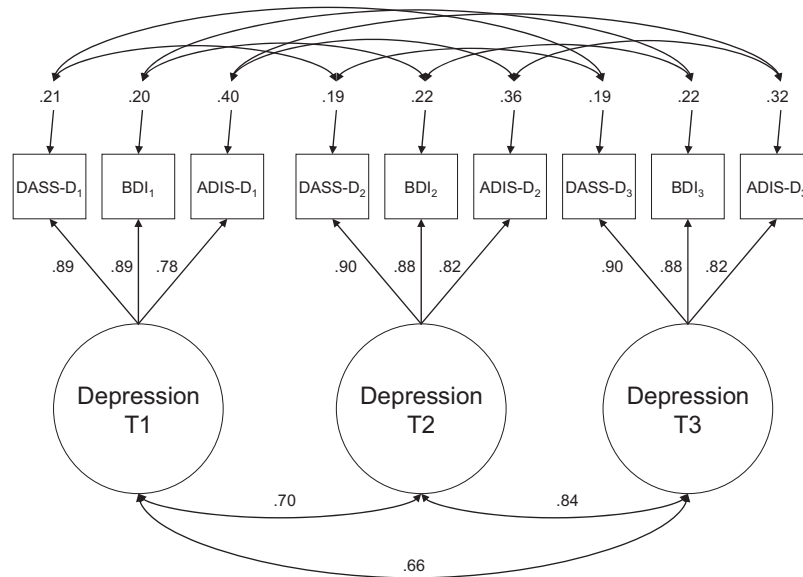** $p < .01$. *** $p < .001$.

Figure 2. Longitudinal measurement model of depressive symptoms. Completely standardized parameter estimates are provided (all *p*s < .001). For presentational clarity, correlated error estimates are not presented (range = .19 to .42). DASS-D = Depression scale of the Depression Anxiety Stress Scales; BDI = Beck Depression Inventory; ADIS-D = dimensional ratings of major depression on the Anxiety Disorders Interview Schedule for *DSM–IV*; T1 = Time 1 (intake); T2 = Time 2 (6-month follow-up); T3 = Time 3 (12-month follow-up).

D_2, DASS-D_3) were specified to have correlated uniquenesses (cf. Brown, 2006). A baseline model, which contained no restrictions on the factor loadings and indicator intercepts fit the data well, $\chi^2(15) = 42.25$, $p < .001$, SRMR = .02, RMSEA = 0.05 (90% CI [0.03, 0.06], CFit = .59), TLI = 0.98, CFI = .99. The next model, which constrained the factor loadings to equality, produced a statistically nonsignificant increase in model chi-square, indicating that the loadings were time-invariant, $\chi^2_{\text{diff}}(4) = 8.54$, *ns*. A final model established the temporal equivalence of the indicator intercepts, $\chi^2_{\text{diff}}(4) = 9.01$, *ns*. The completely standardized parameter estimates of this solution are presented in Figure 2 (all factor loading and factor correlation *p*s < .001).

The longitudinal measurement model was respecified as an unconditional LGM. Because more depression symptom change occurred during the first 6 months of follow-up (when most patients received treatment), linear growth was untenable. Thus, the Slope factor loadings were specified as follows for T1, T2, and T3, respectively: 0, *, and 1 (* = freely estimated parameter). This specification centers the Intercept factor on T1 (i.e., mean and variance of DEP when Time = 0), and the mean (and variance) of the Slope factor reflects the extent of change (and individual differences in change) in the DEP latent construct over the 1-year period. Identification and parsimony of the LGMs was further fostered by fixing the intercepts of the DEP latent variables to zero and holding their residual variances to equality.

The unconditional LGM fit the data well, $\chi^2(25) = 59.84$, $p < .001$, SRMR = .03, RMSEA = 0.04 (90% CI [0.03, 0.06], CFit = .86), TLI = 0.99, CFI = .99. As expected, the majority of symptom reduction in the DEP construct occurred in the first 6 months of follow-up, as reflected by the freely estimated T2 Slope factor loading ($\lambda_{\text{T2}} = .87$). Estimates for the growth factors are presented in Table 2. The random effects for both the Intercept and

Slope were statistically significant (ps < .001), indicating the presence of considerable individual differences in the initial levels (T1) and change in DEP over time. The sign (i.e., negative) and statistical significance ($p < .001$) of the Slope mean indicated that, on average, patients experienced a significant reduction in DEP over the follow-up period (Cohen's $d = 0.60$). The Intercept and Slope were significantly ($p < .001$) and inversely (e.g., correlation = –.40) related; thus, as in Brown (2007), temporal reductions in depression were more pronounced in patients with higher initial levels of depressive symptoms.

Chronic stress. Next, a conditional LGM was specified to examine whether T1 neuroticism (N) and the level of chronic stress experienced during the follow-up interval (CS_F) predicted individual differences in the extent of change in DEP (holding initial levels of DEP constant). This model provided a good fit to the data, $\chi^2(39) = 102.44$, $p < .001$, SRMR = .03, RMSEA = 0.04 (90% CI [0.03, 0.06], CFit = .80), TLI = 0.98, CFI = .99. Collectively, N and CS_F accounted for 32.3% of the variance in the DEP Slope ($p < .001$). However, consistent with the findings of

Table 2
Structural Parameter Estimates From the Unconditional Latent Growth Model of Depression

Variable	Intercept	Intercept–Slope	Slope
Mean	14.57 (0.38)		−5.47 (0.36)
Variance	83.57 (5.95)		36.34 (5.76)
Covariance		−22.19 (4.30)	
Correlation		−0.40 (0.05)	

Note. Standard errors are in parentheses. All parameter estimates are significant at $p < .001$.

Brown (2007), initial levels of N did not account for significant unique variance in the DEP Slope (unstandardized and completely standardized γs for N = -0.04 and $-.06$, respectively; ps = .61). The effect of $CS_F \rightarrow$ DEP Slope was statistically significant ($p <$ .001) and positively signed (unstandardized and completely standardized γs for CS_F = 0.47 and .47, respectively). This path reflects a two-way interaction effect; specifically, that the effect of time on DEP differs across levels of CS_F. The positive sign of this path (in tandem with a Slope mean showing an overall reduction in depression over follow-up) indicates that, holding initial levels of depression and neuroticism constant, patients who experienced higher levels of chronic stress during the follow-up period evidenced less symptom improvement in depression.

The N $\times$ CS_F interaction term was then included in the conditional LGM. Prior to forming the product term, N and CS_F were mean-centered to foster interpretability of the parameter estimates and to eliminate multicollinearity in the predictor set. The parameterization and goodness of fit of this model are shown in Figure 3. The N $\times$ $CS_F \rightarrow$ DEP Slope path was statistically significant ($p <$ .001) and resulted in an R^2 change of .03 (f^2 = .09, between a small and medium effect per Cohen, 1988). The completely standardized parameter estimates (and p levels) of this solution are presented in Figure 3. The significant N $\times$ $CS_F \rightarrow$ DEP Slope path indicates that the strength of the relationship between neuroticism and the trajectory of depression varies as a function of the level of chronic stress experienced during the follow-up period. However, in view of the manner in which time is parameterized in the LGM, the two-way interaction of these predictors must be probed as a three-way interaction with time. Thus, the model-implied trajec-

tories of DEP were computed and plotted at low, medium, and high levels of N ($-SD$, M, $+SD$, respectively) within high and low levels of CS_F ($\pm SD$; cf. Curran, Bauer, & Willoughby, 2004).

The nature of this three-way interaction is depicted in Figure 4. Each of the six conditional trajectories shown in Figure 4 was statistically significant (largest p = .01). Within low CS_F, although the three conditional trajectories are decreasing over time, the magnitude of the reduction in DEP is significantly larger as the initial level of N increases. The opposite pattern occurs when the level of chronic stress is high. Within high CS_F, high N is associated with the least amount of DEP symptom reduction. In other words, when chronic stress during follow-up is high, the degree of improvement in DEP is significantly smaller as N increases.

The previous two conditional LGMs were reconducted including extraversion (NFFI-E) and CS_I. Importantly, the direct effects of CS_F and the N $\times$ CS_F interaction remained statistically significant when controlling for these covariates (e.g., chronic stress reported during the follow-up period continued to predict change in DEP when holding initial levels of chronic stress constant). Interestingly, the direct effect of extraversion on the DEP Slope was statistically significant (unstandardized and completely standardized γs = -0.11 and $-.15$, respectively; ps < .01). Thus, controlling for other predictors (i.e., chronic stress and initial levels of DEP and neuroticism), higher levels of T1 extraversion were associated with greater decreases in DEP.

Finally, it should be noted that although the direction of the $CS_F \rightarrow$ DEP Intercept path shown in Figure 3 is at odds with conceptual reasoning (i.e., chronic stress during follow-up should not have a directional influence on depression severity at intake; see dotted path line in Figure 3), the growth factors in these models were nonetheless regressed onto all background variables because the substantive focus was on the paths relating these predictors to the DEP Slope (i.e., this parameterization allowed the exogenous variables to be freely intercorrelated and accounted for any significant covariance that existed between these variables and the DEP Intercept). In fact, as seen in Figure 3, a significant relationship existed between the DEP Intercept and CS_F. Thus, in accord with a stress generation framework, the last conditional LGM was respecified such that CS_F was regressed onto N, extraversion, DEP Intercept, and CS_I. The DEP Intercept $\rightarrow CS_F$ path was statistically significant (unstandardized and completely standardized γs = 0.27 and .43, respectively; ps < .001). Therefore, even when these other covariates were held constant, higher levels of depression at intake were associated with higher levels of chronic stress during follow-up.

Episodic stress. The conditional LGMs were repeated, replacing CS_F with the variable representing the level of episodic stress experienced during the follow-up interval (ES_F). Both the main effects and moderation models fit the data well; for example, goodness of fit for the moderation model, $\chi^2(46)$ = 86.60, $p <$ .001, SRMR = .02, RMSEA = 0.03 (90% CI [0.02, 0.04], CFit = 1.00), TLI = 0.99, CFI = .99. However, neither ES_F nor the N $\times$ ES_F product term accounted for significant unique variance in the DEP Slope (ps = .72 and .52, respectively). Moreover, unlike chronic stress, intake depression severity was not significantly associated with episodic stress during follow-up (zero-order r of DEP Intercept and ES_F = .04; p = .43). As in the chronic stress LGMs, the N $\rightarrow$ DEP Slope path was nonsignificant (p = .25). However, higher T1 extraversion was associated with greater decreases in DEP over follow-up, even when
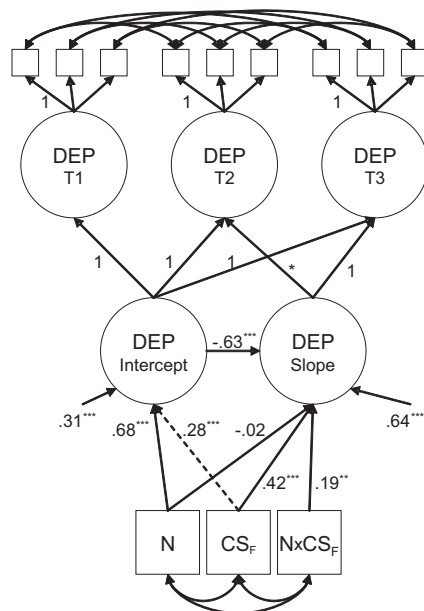

Figure 3. Conditional latent growth model of depressive symptoms. Completely standardized estimates are shown. Overall fit of model: $\chi^2(46)$ = 126.30, $p <$ .001, SRMR = .03, RMSEA = 0.05 (90% CI [0.04, 0.06], CFit = .74), TLI = 0.98, CFI = .98. DEP = depression; T1 = Time 1 (intake); T2 = Time 2 (6-month follow-up); T3 = Time 3 (12-month follow-up); * = freely estimated parameter; N = neuroticism; CS_F = chronic stress during the follow-up period. $^{**} p <$ .01. $^{***} p <$ .001.
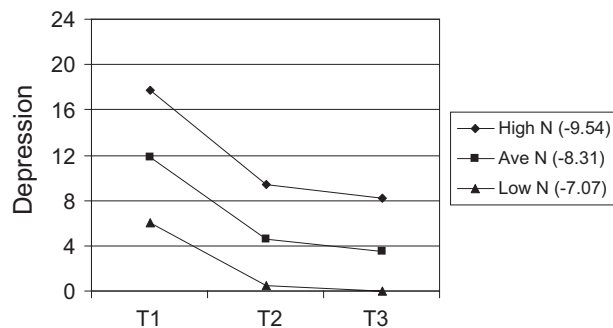
High Chronic Stress:
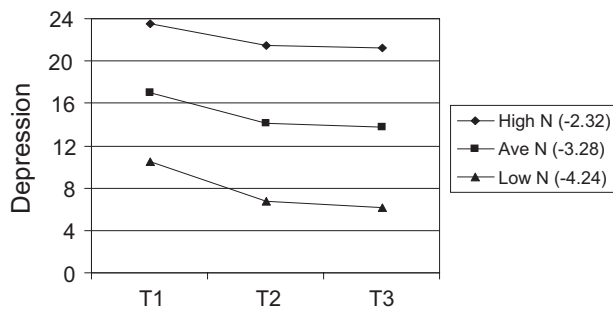
Low Chronic Stress:

Figure 4. Model-implied trajectories of depression as a function of neuroticism within high and low chronic stress. The conditional model-implied trajectories are provided in parentheses within the figure legend. The possible range of scores for the unstandardized depression latent variable is 0 to 42. N = neuroticism; Ave = average; T1 = Time 1 (intake); T2 = Time 2 (6-month follow-up); T3 = Time 3 (12-month follow-up).

controlling for episodic stress, initial levels of DEP, and neuroticism (unstandardized and completely standardized γs = −0.13 and −.18, respectively; ps < .01).

Discussion

As expected (i.e., the majority of the sample received treatment after the intake assessment), results from the unconditional latent growth model indicated that patients experienced a significant reduction in depression symptoms over the follow-up period (Cohen's d = 0.60). Consistent with a common finding in random effects modeling studies of psychopathological processes (see e.g., Brown, 2007), the growth factors were inversely related (Intercept and Slope correlation = −.40). Thus, temporal symptom reductions were more prominent in patients with higher initial levels of depressive symptoms. However, subsequent analyses revealed that several variables moderated this temporal change.

For instance, the collective findings indicate that chronic stress is a salient predictor of the severity and temporal change in depressive symptoms. In the cross-sectional analyses, the level of chronic stress exposure reported during the 6 months prior to the intake evaluation was uniquely and positively associated with depression symptom severity. A CS_F × Time interaction effect was obtained in the longitudinal analyses indicating that patients who reported higher levels of chronic stress during the follow-up

period experienced less depression symptom improvement. These results are in accord with previous assertions and some evidence attesting to the relevance of the relatively neglected role of chronic stress in the severity and maintenance of depression (see e.g., Hammen et al., 2009; Hayden & Klein, 2001; McGonagle & Kessler, 1990; Tennant, 2002). Moreover, these data are consistent with arguments that chronic stress may have stronger relations with the symptom severity and longitudinal course of depression than does episodic stress (Hayden & Klein, 2001; Moerk & Klein, 2000; Tennant, 2002). Indeed, although episodic stress was uniquely predictive of intake symptom severity, nonsignificant effects were obtained in the remaining analyses (i.e., episodic stress did not have a direct effect on depression improvement and was not a significant moderator of the influence of neuroticism on depression symptom severity or temporal course).

These findings may suggest that although exposure to an acute stressor is a stronger precipitant of a depressive episode (as documented by a sizeable literature focused on the role of episodic stress in major depression; cf. Hammen, 2005; Kessler, 1997), ongoing stressors (e.g., poor working conditions, financial difficulties, continuing health problems, conflictual interpersonal relationships) are more germane to the ongoing course of symptoms. Accordingly, chronic strains may work together with a variety of psychological or biological determinants to protract the symptoms of depression (e.g., constant financial or occupational adversities may reinforce feelings of low self-worth; chronic health problems may contribute to a sense of hopelessness). However, it is possible that aspects of the study design fostered the observed differential relationships of chronic and episodic stress. Unlike most previous research, the construct of depression was represented in the analyses as a latent dimension rather than an observed categorical variable (i.e., presence/absence of a *DSM–IV* mood disorder). Although having many advantages (e.g., increased statistical power, reliability, and validity from using dimensional latent variables defined by multiple indicators; cf. Brown, 2006, 2007; Brown & Barlow, 2009), this approach was concerned with accounting for individual differences in depression symptom severity and did not address the specific timing of the onset of stressors and depressive episodes. For instance, as was the case for chronic stress, the episodic stress variable was a composite rating of acute stressors occurring over a 6-month period. Collapsing episodic stressors in this fashion may have weakened the associations that could have been obtained in a finer grained approach (e.g., linking specific acute stressors to spikes in depression symptom severity if stress and depression were assessed more frequently than every 6 months). Moreover, several studies have found that acute stressors play a larger role in the onset of the first depressive episode than in subsequent recurrences (see e.g., Kendler, Thornton, & Gardner, 2000; Monroe et al., 2007; but also see Daley, Hammen, & Rao, 2000). The fact that most study participants with elevated symptoms had recurrent or ongoing depression (e.g., recurrent major depression, early onset dysthymia) may have further attenuated the association seen between episodic stress and depression symptom severity.

The current cross-sectional findings provide further evidence for the strong relationship between neurotic temperament and depression symptom severity (see e.g., Brown, 2007). Despite the large effect obtained in the cross-sectional analyses, neuroticism did not significantly predict the longitudinal course of depressive symp-

toms. Similarly, a null effect was also reported in Brown (2007), in which the variable of neuroticism/behavioral inhibition was associated with less improvement in generalized anxiety disorder and social phobia but not depression. As noted earlier, studies from the depression treatment outcome literature have produced mixed results with regard to the association between pretreatment levels of neuroticism and depression treatment response. In Brown, this nonsignificant temporal relationship was discussed in terms of the considerable overlap observed between initial levels of neuroticism/behavioral inhibition and depression ($r = .77$, resulting in a relatively smaller amount of unique variance in change that could be potentially accounted for by predictors), as well as the possibility that temperament has stronger effects on psychopathology as a predispositional factor than as a pathoplastic influence (i.e., more robust direct effects of temperament on depression may be obtained in community or at-risk samples than in clinical samples). Indeed, the ability to detect temporal relationships between temperament and psychopathology in clinical samples may be complicated by the effects of *mood-state distortion*; that is, patients' self-reports of temperament are affected (augmented) by their current clinical state (Brown, 2007; Clark et al., 2003; Widiger & Smith, 2008). For instance, if neuroticism is a temporally stable construct that characterizes everyday functioning (i.e., a trait), its measurement in clinical samples is apt to contain a substantial amount of "state" variance due to general clinical distress. Clark et al. (2003) have argued that the variance due to mood-state distortion may make it difficult to detect how stable variance in temperament predicts depression outcomes. As in Brown, the present study used initial depression symptom severity (i.e., the Depression Intercept; cf. Figure 3) as a covariate in the conditional LGMs in an attempt to remove some of the general distress component from neuroticism in the prediction of the temporal course of depression. However, in Brown and the present study, neuroticism did not exert a significant direct effect on depression symptom change.

A different pattern of results arose when chronic stress was included in the analyses as a moderator of the temporal effects of neuroticism on depression symptoms.[2] Consistent with our prediction, neuroticism had an increasingly deleterious effect on depression symptom improvement as the levels of concurrent chronic stress increased. Indeed, the least amount of depression symptom reduction was seen in individuals with the highest intake levels of neuroticism who experienced higher levels of chronic stress during the follow-up period (i.e., a model-implied decrease of only 2.32 points on the DASS-D; cf. Figure 4). Importantly, this interaction effect remained statistically significant after controlling for a number of substantively salient covariates (e.g., initial depression symptom severity, extraversion, chronic stress over the 6 months preceding the follow-up interval). This result is in accord with leading conceptual models of emotional disorders (see e.g., Barlow, 2002), which posit that preexisting biological or psychological vulnerabilities (e.g., genetically based dimensions of temperament, perceptions of low emotional control) must be activated by stressful life events to produce psychopathology. However, in drawing this connection, one must be mindful of the fact that such conceptual models focus primarily on the etiology of disorders (i.e., predisposition) rather than the maintenance of psychopathology (i.e., pathoplasty). Thus, these conceptualizations are more apt to underscore the potentiating role of an acute stressor in the onset

of psychopathological states (although the specific nature of the triggering life stressors is not always explicated in these models; see e.g., Barlow, 2002).

The current findings suggest that this conceptual interplay between temperament and life stress may extend to the maintenance of psychopathology, with chronic stress playing a more important role as a direct and moderating influence than does episodic stress. Specifically, although neuroticism may have a key and perhaps stronger role as a predispositional factor, it continues to exert a negative influence on the course of depressive symptoms if the propensity to react adversely to stress is activated by ongoing life strains. Inspection of the model-implied trajectories in Figure 4, however, also indicates that neuroticism had the opposite effect on the course of depressive symptoms in patients who were exposed to lower levels of chronic stress (i.e., in these participants, higher initial levels of neuroticism were associated with larger depression symptom reductions). Thus, if not triggered by life stressors during follow-up, neuroticism appears to have a similar impact on the temporal course of depressive symptoms as does initial depression symptom severity, where the negative correlation between the latent growth factors indicated that higher levels of T1 depression were associated with greater reductions in depressive symptoms over follow-up (i.e., given the strong positive relationship between T1 neuroticism and depression, participants with high neuroticism also had elevated initial levels of depression). However, this inverse relationship was reversed by the moderating effects of chronic stress; that is, if activated by chronic stress, higher initial levels of neuroticism are associated with less, not more, depression symptom improvement.

In addition, our findings were suggestive of a bidirectional relationship between chronic life stress and depressive symptoms. Specifically, although chronic stress had a direct effect on depression severity in the cross-sectional and time-series analyses, results also indicated that initial depression symptom severity was associated with higher levels of chronic stress during the follow-up period. Although measurement issues cannot be dismissed (see next paragraph), this result is consistent with prior evidence of stress generation (see e.g., Daley et al., 1997; Hammen, 1991); namely, that the presence of depression is positively associated with the incidence of subsequent stressful life events. Whereas stress generation research has primarily focused on episodic stressors (see e.g., Daley et al., 1997), the current results suggest that this effect extends to chronic stressors. Thus, the presence of a persistent depressive disorder may contribute to the emergence or chronicity of ongoing strains (e.g., social withdrawal may contribute to poor interpersonal and occupational relationships; loss of interest/energy may result in underemployment and financial difficulties). Indeed, some research has suggested that depressed individuals are often locked or select themselves into problematic environmental contexts (see e.g., Hammen, 2003; Hammen & Brennan, 2002; Kendler, Karkowski, & Prescott, 1999). Yet, this

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[2] Although mediational models were not pursued (i.e., Neuroticism → Stress → Depression; see e.g., Ormel & Wohlfarth, 1991; Wetter & Hankin, 2009), it is noteworthy that the current results indicated the absence of a direct effect of neuroticism on depression symptom course (no observed relationship between these variables that might be mediated by life stress during the follow-up period).

research has also produced evidence that stress generation is not necessarily due entirely to the direct effects of a depressive disorder. For instance, Kendler and colleagues found that exposure to stressful life events is substantially influenced by genetic factors and that the genetic risk factors for stressful life events are positively correlated with the genetic risk factors for major depression (see e.g., Kendler & Karkowski-Shuman, 1997; Kendler, Neale, Kessler, Heath, & Eaves, 1993). Along these lines, other evidence from this laboratory has indicated that neuroticism is strongly predictive of the occurrence of stressful life events and the quality of interpersonal relationships (Kendler et al., 2003; see also Bolger & Zuckerman, 1995; Fergusson & Horwood, 1987; Gunthert, Cohen, & Armelli, 1999). More research is needed to further explicate the specific role of temperament in the relationship between psychopathology and life stress (e.g., does neuroticism have an indirect effect on life stress, with depression playing an intervening role in this causal pathway?).[3]

However, the fact that the interview measures of chronic and episodic stress were collected concurrently with measures of depression precludes firm conclusions about the directionality of the relationships between these variables. Moreover, it is possible that the magnitudes of these relationships were unduly augmented by aspects of the assessment approach and the use of a clinical sample. For instance, life stress ratings were based on patients' retrospective report and thus, like other variables, were susceptible to the effects of mood-state distortion (e.g., depression severity at the time of the evaluation covaried with a tendency to recall negative life events over the preceding 6 months).

Researchers must also consider the possibility that the associations routinely observed between life stress and depression are spuriously inflated by confounded measurement (i.e., the measurement of life stress may overlap with the measurement of depression). This issue may be particularly germane to the measurement of chronic stress. For example, a higher rating for the UCLA-LSI social domain of chronic stress might be assigned to characterize an individual with impoverished friendships and limited social contacts (see the Method section). Because clinical depression is often associated with social withdrawal, the chronic social stress rating could be construed as another indicator of depression. As discussed by Hammen (2005), life stress interviews were developed to address this and other potential confounds and are regarded as the standard of this field (i.e., psychometrically superior to other methods such as life event checklists; cf. Kessler, 1997). Specifically, these interviews entail, in addition to the systematic identification of events, the collection of contextual information (e.g., circumstances surrounding the event) used as a basis for rating the "objective" threat of the stressors, independent of an individual's symptomatology. Indeed, research has indicated that interview methods are less prone to potential measurement confounds and biases (mood-state distortion, cognitive vulnerabilities; see e.g., McQuaid, Monroe, Roberts, Kupfer, & Frank, 2000). However, it has been suggested that the contextual information used to bolster the quality of interview ratings may itself be influenced by the risk factors (e.g., neuroticism) that account for the relationship between life stress and depression (Kessler, 1997; Mazure, 1998). Thus, despite the use of an interview method of life stress measurement (UCLA-LSI), these psychometric issues cannot be dismissed.

Nonetheless, aspects of the current results may indicate that measurement confounds were not chiefly responsible for the observed effects. A multivariate approach was undertaken in part to control for the overlap between the various predictors of the longitudinal course of depression symptoms. In these analyses, for instance, chronic stress had a significant direct and moderating effect on the trajectory of depression, even after holding initial levels of depression and neuroticism constant. Moreover, the direction of the effect that chronic stress had on the course of depression symptoms was opposite to that of other predictors (e.g., although the initial level of depression was positively related to chronic stress during follow-up, it was inversely related to depression symptom improvement), a result pattern that may argue against the redundancy of these variables. A more compelling evaluation of the nature of the relationship between chronic stress and depression would require alternative research designs such as studies focused on the impact of stressors that are presumably unrelated to depression and its risk factors (e.g., studies of parents of children with serious medical disorders) or prospective studies of individuals entering stressful life roles (e.g., military service; Kessler, 1997).

Although mood disorders were well represented (e.g., $n = 329$ at intake), it is important to note that this study used a diagnostically diverse sample along with outcome variables that were dimensional latent variables of depression symptoms rather than *DSM–IV* mood disorder diagnoses. As has been discussed at length elsewhere (see e.g., Brown & Barlow, 2005, 2009), this approach has several methodological advantages over the use of *DSM* diagnoses as units of analysis—advantages such as better reliability (further fostered by the use of latent variables that are theoretically free of measurement error), ability to capture individual differences in symptom severity, and increased statistical power. Whereas some previous studies have used research designs similar to that of the current study (e.g., depression as a continuous variable; Kercher et al., 2009; Wetter & Hankin, 2009), the more common strategy in this literature entails the use of *DSM* diagnoses as outcomes (e.g., the ability of neuroticism or stress to predict the onset of *DSM*-defined major depressive episodes; Hammen et al., 2009; Monroe et al., 2007; Ormel et al., 2001). Because this study focused on the severity and longitudinal course of depressive *symptoms*, it is not clear to what extent these findings would inform the nature of the relationship that neuroticism and life stress have in predicting the onset or recurrence of major depression *diagnoses* or other *DSM–IV* mood disorder categories. For instance, the question of whether episodic stressors are more strongly related to first episodes of major depression than to recurrences (see e.g., Moerk & Klein, 2000) may be better addressed by studies using *DSM* disorders as units of analysis than by treating depression as a continuous variable. The dimensional and

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[3] To evaluate this possibility in the current data set, we respecified the final structural model in the Results section with the following indirect effect: $N \rightarrow DEP_{Intercept} \rightarrow CS_F$. In accord with this conceptualization, a significant mediational relationship was obtained; unstandardized and standardized indirect effects = 0.22 and .34, respectively ($ps < .001$). Thus, holding other covariates constant (e.g., chronic stress during the 6 months preceding intake), neuroticism was positively associated with higher levels of chronic stress during follow-up (as mediated by initial depression severity).

categorical approaches both have their strengths and place in the stress-vulnerability literature, but care should be taken to avoid the assumption that findings from one approach will readily generalize to the other.

Interestingly, as well as its association with depression symptom severity at intake, extraversion was found to be significantly and uniquely predictive of the temporal course of depression (i.e., lower initial levels of extraversion were associated with less depression symptom improvement). Because extraversion was included in this study as a covariate (to examine the unique effects of neuroticism and its interaction with life stress controlling for another salient temperamental predictor of depression), this should be regarded as a rather incidental finding. Indeed, previous studies have produced mixed findings with respect to the ability of extraversion and related constructs (e.g., behavioral activation, positive affect) to predict the temporal course and treatment response of depression (see e.g., Brown, 2007; Hayden & Klein, 2001; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Quilty et al., 2008). Given the extensive empirical and conceptual basis for the significance of neuroticism in stress-depression research (e.g., as a dimension associated with sensitivity to stressful life events; Hammen, 2005), investigators have not considered the possibility that the effects of extraversion on depression vary as a function of life stress. Although the direct effect of extraversion on depression typically reflects an inverse relationship (i.e., lower levels of extraversion are associated with higher levels of depression), interaction effects involving life stress may be more complex (i.e., the direction of effects vary depending on the facet of extraversion and the type of life stress). Whereas extraversion is not a trait reflecting the propensity to react adversely to stressors (cf. neuroticism), it is defined in part by facets such as sociability and activity that might interact with stressful life events to elicit clinical distress. In line with a congruency model of stress and personality (see e.g., Nietzel & Harris, 1990), perhaps depression is apt to arise when the nature of the activating stressor is relevant to the specific facets of extraversion that are characteristic of the individual; for example, persons scoring high on the excitement-seeking or activity facets may be more prone to depression if exposed to a stressor such as a physically debilitating injury that limits the ability to be active and engaged with their environment (an effect that is opposite to the direction of the typical main effect of extraversion on depression).

Finally, in addition to the aforementioned limitations (e.g., measurement issues, inability to address predispositional relationships), this study focused exclusively on neuroticism as the focal independent variable in the prediction of the severity and course of depressive symptoms. However, current conceptual models assert that dimensions of temperament are not solely responsible for the onset and maintenance of psychopathology. For instance, the triple vulnerability model of emotional disorders (Barlow, 2002) posits that in addition to biologically based temperaments (e.g., neuroticism), a general psychological vulnerability (self-perceptions of low emotional control) and disorder-specific vulnerabilities (e.g., cognitive dimensions such as sociotropy/autonomy and dysfunctional attitudes) act in concert to produce clinical depression. It is possible that these dimensions also influence the temporal course of depression, both directly and if activated by life stress. In addition to studies of predispositional effects (e.g., longitudinal studies using community or at-risk samples), future research

should evaluate whether these theoretically relevant dimensions contribute to the maintenance of depressive symptoms, over and beyond the influence of neurotic (and intraverted) temperament.

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