Daily Depression and Cognitions About Stress: Evidence for a Traitlike Depressogenic Cognitive Style and the Prediction of Depressive Symptoms in a Prospective Daily Diary Study

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The authors examined the stability and dynamic structure of negative cognitions made to naturalistic stressors and the prediction of depressive symptoms in a daily diary study. Young adults reported on dispositional depression vulnerabilities at baseline, including a depressogenic cognitive style, dysfunctional attitudes, rumination, neuroticism, and initial depression, and then completed short diaries recording the inferences they made to the most negative event of the day along with their experience of depressive symptoms every day for 35 consecutive days. Daily cognitions about stressors exhibited moderate stability across time. A traitlike model, rather than a contextual one, explained this pattern of stability best. Hierarchical linear modeling analyses showed that individuals’ dispositional depressogenic cognitive style, neuroticism, and their daily negative cognitions about stressors predicted fluctuations in daily depressive symptoms. Dispositional neuroticism and negative cognitive style interacted with daily negative cognitions in different ways to predict daily depressive symptoms.

Keywords: depression, cognitions, vulnerability, daily diary, trait

Social–cognitive theories of depression (e.g., Abramson et al., 2002; Hankin & Abramson, 2001) offer a promising framework for understanding individual differences in the way people respond, both cognitively and affectively, to negative life events. Generally, these theories posit that some people explain stressful and negative life events in a more optimistic way and, consequently, may be relatively unscathed by them. In contrast, other people interpret such stressors in a more pessimistic manner and as a result may respond to these events with strong and enduring negative emotions. In particular, the hopelessness theory of depression (HT; Abramson, Metalsky, & Alloy, 1989), a prominent social–cognitive vulnerability–stress model of depression, posits that individuals have characteristic ways of understanding negative life events. According to HT, people are more likely to become depressed when a negative event is (a) attributed to stable (i.e., persisting over time) and global (i.e., affecting multiple areas in life) causes, (b) perceived as leading to other negative consequences in the future, and (c) viewed as implying something negative about the self (e.g., worthlessness). When made in response to stressors in the flow of daily life, these cognitions are defined in HT as event-specific inferences. HT postulates that there are individual differences in the way people make event-specific inferences, such that those who exhibit a dispositional depressogenic cognitive style are more likely than others to make negative inferences about events and thus are at greater risk for becoming depressed (Abramson et al., 1989). In other words, not only is it postulated that event-specific inferences lead to depression but that some people are more likely to make these kinds of negative inferences than others.

Trait and Contextual Models: The Structure and Stability of Event-Specific Inferences

The majority of research that has examined cognitive vulnerability to depression has focused on a depressogenic cognitive style as if it were a traitlike or dispositional variable. Although a depressogenic cognitive style has not been defined explicitly as a trait per se in HT, it has been conceptualized and treated as such. For example, Abramson and her colleagues have referred to this cognitive style as a “general tendency” to make negative inferences about events and thus are at greater risk for becoming depressed (Abramson et al., 1989). In other words, not only is it posited that event-specific inferences lead to depression but that some people are more likely to make these kinds of negative inferences than others.

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the causes of and inferences made about positive and negative events.

Although the traitlike nature of cognitive styles appears to be a fundamental assumption of HT, there is no research that has explicitly sought to determine whether the attributions and inferences that people make about negative life events are structured in a traitlike manner. Several researchers have studied the test–retest stability of various measures of depressogenic cognitive style and found a high degree of stability (e.g., Alloy et al., 2000; Burns & Seligman, 1989), but recent advances in the study of personality development suggest that this empirical strategy (i.e., testing the test–retest correlation over two time points) cannot provide an accurate test of trait-based models. Specifically, Fraley and Roberts (2005) demonstrated that both trait-based models and contextual models (i.e., models that do not assume the existence of an underlying enduring trait) are capable of predicting a high degree of stability across any two time points. Because both kinds of models can accommodate the same test–retest data, the magnitude of simple test–retest correlations cannot be used to corroborate or refute these models. To distinguish traitlike and contextual models, it is necessary to study the pattern of test–retest correlations across multiple time points (see Fraley & Brumbaugh, 2004; Fraley & Roberts, 2005).

Trait models predict that the empirical test–retest correlations will be invariant (ignoring statistical fluctuations in measurement precision) as the length of test–retest intervals increase, because a stable psychological variable is hypothesized to organize manifestations of the construct across time (Fraley & Roberts, 2005). In contrast, contextual models predict that the magnitude of the test–retest correlations for a construct will decrease monotonically as the size of the interval increases (i.e., the correlations will adhere to a simplex pattern; see Kenny & Zautra, 2001). Contextual models make this prediction because they do not assume the existence of an enduring, organizing psychological variable contributing to stability (Fraley & Roberts, 2005). Without such an organizing construct, the kinds of life events that influence people’s thoughts, feelings, and behaviors have only transitory, rather than enduring, effects on people.

There are at least two reasons why it is necessary to determine which of these structures best characterizes the way that attributions and inferences about negative life events are made across time. First, if the nature of the inferences that people make about life events is not an enduring facet of their personalities but only a temporary response to current circumstances, then contemporary theories of cognitive vulnerability to depression need to be reevaluated. HT, for example, assumes that there is a stable quality underlying event-specific inferences, and if that assumption were found to be unwarranted, it would be necessary to construct alternative models to explain the associations between negative life events, inferences, and depression. Second, if these inferences do not conform to a traitlike model, then future research may profit by focusing more on the situational contexts that confer vulnerability to depression and less on the dispositional cognitive styles that are hypothesized to serve as a stable vulnerability for depression. This would require a fundamental shift in the study of vulnerability to depression. One of our objectives in this research is to evaluate the traitlike assumption of HT by studying the patterns of continuity in negative event-specific inferences.

Associations Between Daily Negative Event-Specific Inferences and Depression

HT posits a specific etiological chain leading from dispositional negative cognitive style to depression. Specifically, HT holds that individuals exhibiting this dispositional depressogenic cognitive style are more likely to make negative inferences in response to negative life events and as a result are more susceptible to depression. Despite an abundance of research demonstrating that a dispositional depressogenic cognitive style increases risk for depression (for reviews, see Abramson et al., 2002; Ingram, Miranda, & Segal, 1998), little research has actually investigated the inferences that people make to specific events. There are a number of studies that use cross-sectional designs to correlate global measures of dispositional depressogenic cognitive style with broad measures of negative affect and depression (e.g., Sweeney, Anderson, & Bailey, 1986). There are also prospective studies that have found that dispositional depressogenic cognitive style predicts either increases in depressive symptoms or the onset of depressive episodes at another point in time (e.g., Alloy et al., 2000; Haefel et al., 2003; Hankin, Abramson, Miller, & Haefel, 2004; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993). Nonetheless, a key component of HT is that depressogenic inferences are made in response to specific events (i.e., they are “event-specific” inferences) and that such inferences may serve as more proximal causes of increases in depressive symptoms. To test this aspect of the theory, it is necessary to investigate the role that event-specific inferences play in response to day-to-day stressors.

A few investigations have focused on event-specific inferences as opposed to a global or dispositional cognitive style. Metalsky and colleagues (Metalsky et al., 1993; Metalsky, Halberstadt, & Abramson, 1987) assessed dispositional attributional style, achievement event-specific inferences about failure for a midterm exam, and depressive symptoms immediately and several days after receiving the exam grade. Failure on the exam predicted an immediate increase in depressive symptoms, whereas the interaction of dispositional attributional style and exam failure predicted endurance of symptoms days later. It is important to note that the association between dispositional attributional style and enduring depressive symptoms was mediated by specific inferences made about the exam failure. Swendsen (1997, 1998) used an idio- graphic, experience-sampling method to assess depressed mood and event-specific causal attributions five times daily over a 1-week period. He found that participants’ event-specific attributions were associated with dispositional attributional style and depressed mood in the flow of daily life across multiple contexts and situations. Unexpectedly, Swendsen also found that dispositional attributional style was not associated with daily depression. This result is particularly surprising because the accumulated corpus of evidence shows that dispositional cognitive vulnerability reliably predicts depression (e.g., Abramson et al., 2002; Ingram et al., 1998). Whereas these two investigators’ studies are largely consistent with HT, it is unclear whether Swendsen’s last finding reflects a true problem for HT or whether it would have been possible to establish an association between dispositional cognitive style and daily depressive symptoms using a different design.

In summary, although there are a number of studies that have established relations between measures of dispositional cognitive style and depressive symptoms, there has been little work on the role that event-specific inferences play in this process. The most
intensive study that has been conducted on this matter (e.g., Swendsen, 1997) was unable to establish these links unambiguously. In this respect, our design is similar to Swendsen’s, but as we explain in the next section, our study addresses a broader range of dispositional depression vulnerabilities in addition to cognitive style. As a result, we can provide a more comprehensive examination of the way in which dispositional vulnerabilities and event-specific inference processes influence depressive symptoms.

Dispositional Predictors of Daily Event-Specific Inferences and Depressive Symptoms

In addition to addressing questions about the role of event-specific inferences in affective experience and stability of those inferences, we also sought to explore the role of other vulnerabilities to depression. Along with the dispositional depressogenic cognitive style featured in HT, there are at least two other cognitive vulnerability factors that have received a lot of attention in depression research: dysfunctional attitudes (Beck, 1987) and a ruminative response style (Nolen-Hoeksema, 1991). Beck’s cognitive theory (BT; Beck, 1987) posits that dysfunctional attitudes—rigid and extreme beliefs about the self and the world—serve as cognitive vulnerabilities to depression following the occurrence of negative events. These dysfunctional attitudes often involve themes such as deriving one’s worth from being perfect or needing approval from others. Nolen-Hoeksema’s (1991) perspective holds that the way in which individuals respond to their depressed mood helps to determine the severity and duration of their depression. People who ruminate tend to respond to their depressed mood by repetitively focusing attention on their symptoms and the implications of their symptoms.

These three vulnerability factors are theoretically and empirically distinct cognitive risk factors for depression (for factor analytic evidence, see Hankin, Carter, & Abela, 2003). HT’s negative cognitive style, but not BT’s dysfunctional attitudes, has been found to predict a lifetime history of clinical depression (Haefel et al., 2003) and prospective elevations of depressive symptoms (Lee et al., 2003). Although these findings suggest that the cognitive vulnerabilities are distinct, it is not known whether they function in distinct ways (i.e., whether the processes leading to depression are the same for each vulnerability).

In addition to these widely studied cognitive vulnerabilities, traditional personality traits, such as neuroticism, have been shown to predict prospective elevations in depression (e.g., Krueger, 1999; Widiger, Verheul, & van den Brink, 1999). Neuroticism has also been theorized to be associated with cognitive vulnerability to depression (e.g., Clark, Watson, & Mineka, 1994; Hankin & Abramson, 2001). Given the theoretical and empirical associations between neuroticism, depression, and cognitive vulnerability, it is also important to examine whether and how the relations among HT’s dispositional cognitive style, event-specific inferences, and depression may be influenced by neuroticism.

Overview of Current Research

The present research examined several fundamental questions emanating from HT, a prominent social–cognitive vulnerability–stress model of depression. First, what is the organization and structure of event-specific inferences made to stressors in diverse environmental contexts and situations over time? Specifically, is the dynamic structure of event-specific inferences more consistent with a trait or contextual model (or some combination of the two)? Second, are daily levels of depressive symptoms predicted by the causal etiological chain posited in HT? In other words, are individual differences in depressogenic cognitive style associated with event-specific inferences and daily depressive symptoms, and, in turn, do event-specific inferences covary with trajectories in depressive symptoms? Third, to what extent do HT’s etiological factors (dispositional cognitive style and negative event-specific inferences) uniquely predict daily depressive symptoms above and beyond alternative vulnerability factors (i.e., dysfunctional attitudes, rumination, and neuroticism)?

These novel questions were addressed in a naturalistic daily diary study in which young adults made event-specific inferences about the most negative event they experienced each day and rated their levels of depressive symptoms. These ratings were made for 35 consecutive days. In addition, participants completed dispositional measures at the start of the study that assessed the three central conceptualizations of cognitive vulnerability to depression, initial depressive symptoms, and neuroticism.

Method

Participants and Procedures

Undergraduate students, who volunteered for extra credit in an introductory psychology class, served as participants. Participants completed a packet of questionnaires for the initial assessment (T1). The T1 packet included dispositional measures of depressogenic cognitive style, dysfunctional attitudes, rumination, neuroticism, and initial depressive symptoms. A total of 217 (62 male) participants completed the initial assessment. Participants’ ages ranged from 18 to 23 years (M = 18.7, SD = 0.96); over 85% of the sample was Caucasian.

After completing the T1 packet, participants individually came to the lab where they were instructed how to complete the daily diaries for the next 35 days. From the 217 participants who completed T1 measures, 210 came to the laboratory to receive instructions for completing the diaries; these 210 participants made up the sample for the present 35-day diary study. Participants were instructed to complete the diary every day at the end of the day (approximately between 9 p.m. and midnight). We emphasized the importance of completing the diary every day and told the participants not to fill out several diaries at the same time. Participants were told to turn in their daily diaries to the lab on the day of their Introduction to Psychology class (every Monday, Wednesday, and Friday). A research assistant was present to receive the participants’ daily diaries for a particular day and to check that each daily diary was completed. The participants were then given another packet of diaries to complete over the next few days. Participants were not given more than four diaries at any one time to ensure that they would complete the diaries on a regular, daily basis. Overall, it appeared that participants generally completed the diaries on a regular basis. On an average day, an average of 27 people (12%) did not complete their diaries (SD = 11.5, Mdn = 25, range = 15–76). There were more missing data near the end of the study (i.e., 57–76 people did not complete the diaries during the last 3 days) than at the beginning. The 12% average attrition is consistent with that found in other prospective research (e.g., Hankin et al., 2004; Metalsky & Joiner, 1992). Attrition analyses revealed no significant differences on dispositional measures for those who completed all diaries from those who did not.

Measures

Cognitive Style Questionnaire (CSQ; Alloy et al., 2000). The CSQ is designed to assess a person’s overall dispositional depressogenic cognitive style, including negative inferences for cause, consequence, and self,
featured in HT. The CSQ consists of 12 hypothetical scenarios (6 interpersonal and 6 achievement) relevant to young adults, each of which presents the participant with a hypothetical negative event and allows the participant to write down one cause for the event. For example, one scenario is, “You do not have a boyfriend/girlfriend but you want one.” Respondents then rate the degree to which the cause of the hypothetical negative event is stable and global (negative inferences for causal attributions; 24 items). In addition, they rate the likelihood that further negative consequences will result from the occurrence of the negative event (negative inferences for consequences; 12 items) and the degree to which the occurrence of the event signifies that the person’s self is flawed (negative inference for self; 12 items). The CSQ was scored by summing participants’ responses for the negative inferences for cause (stable and global attributions), consequence, and self and then dividing by the total items. This results in average item scores on the CSQ ranging from 1 to 7, with higher scores indicating a more negative cognitive style. Evidence for combining these three inferential styles together into a composite cognitive style measure comes from previous factor analytic work showing these three styles load onto the same latent factor (Hankin et al., 2003). Coefficient alpha for the composite cognitive style measure in the present study was .92, and the average intercorrelation among the three inferential styles was .62 (range = .51–.71). Validity for the CSQ is supported by research showing that the CSQ, alone or in combination with negative events, predicts depressive symptoms and episodes (Alloy et al., 2000; Hankin et al., 2004; Metalsky & Joiner, 1992). The CSQ was given at the initial assessment.

Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978). The DAS is a 40-item questionnaire designed to measure dysfunctional attitudes, the cognitive vulnerability featured in BT. An example item is, “If I fail at my work, then I am a failure as a person.” Higher scores reflect more dysfunctional attitudes. The DAS’s validity has been supported by studies finding that the DAS, alone or in combination with negative events, predicts depression (e.g., Hankin et al., 2004; Ilardi & Craighead, 1999). Coefficient alpha was .89 in this study. The DAS was administered at the initial assessment.

Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991). We used the Rumination Response Subscale (RRS), which is a 22-item subscale of the RSQ. Participants are asked how often they engage in various behaviors or have certain thoughts in response to depressed mood. Items are rated on a Likert scale from 1 to 4. An example is, “Think about how alone you feel.” The RRS has demonstrated good internal consistency (Nolen-Hoeksema & Morrow, 1991) and validity for predicting depression (Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1991). The coefficient alpha for RRS in this study was .92.

Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). The MPQ is a self-report instrument that contains 177 items representing 10 different personality factors. The 14-item Stress-Reaction subscale from the Negative Emotionality factor of the MPQ (NEM-SR) was used to assess the participants’ cognitive style. The NEM-SR scale has been shown to predict concurrent and prospective elevations in depression (e.g., Krueger, 1999; Krueger, Caspi, Moffitt, Silva, & McGee, 1996). The coefficient alpha for the scale in this sample was .82.

Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI assesses levels of depressive symptoms with 21 items that are rated on a scale from 0 to 3, with total scores ranging from 0 to 63. Higher scores reflect more depressive symptom severity. The BDI is a reliable and well-validated measure of depressive symptomatology (see Beck, Steer, & Garbin, 1988). The BDI was given at the initial assessment. Consistent with other prospective studies (e.g., Metalsky & Joiner, 1992), participants were instructed to answer the BDI items for the past 5 weeks prior to the start of the study. The coefficient alpha for the BDI was .88 in the present sample.

Daily diary form. A daily diary form was created for use in the present 35-day diary study. The diary form consisted of two parts. On the front page there was a list of the nine depressive symptoms as defined by the Diagnostic and Statistical Manual of Mental Disorders (4th ed; American Psychiatric Association, 1994). Example items include “felt sad,” “felt slowed down,” and “had problems concentrating.” Participants were instructed to rate how much they experienced each of the nine depressive symptoms on that particular day using a 1–5 scale. The daily depressive symptoms were scored by summing the participants’ responses; therefore, scores could range from 9 to 45 each day. Coefficient alpha for the depressive symptoms composite on an average day was .47 (rs ranged from .11 between weight change and fatigue to .50 between fatigue and psychomotor retardation). Coefficient alpha for the composite depressive symptoms averaged across 1 week was .92. On the back page there was space for participants to list up to five negative events that occurred on that particular day. They were instructed to circle the most negative event. Then, the participants completed event-specific inference ratings for the most negative event that they had experienced that day. The negative event-specific inferences for the most negative life event of that day were modeled after the CSQ, in that participants rated, with 7-point scales, their negative inferences for the cause of the specific event (i.e., stability, globality attributions), negative inferences for future consequences that may occur given the event, and negative inferences for their self given the specific event. The daily event-specific inferences were scored by summing the participants’ responses; thus, scores could range from 5 to 35 each day. Coefficient alpha for the event-specific inferences composite was .82 (rs ranged from .35 between stable attributions and self-inferences to .74 between global attributions and consequence inferences). In sum, the participants used the daily diary record form to rate their level of depressive symptoms; to write down the occurrence of daily stressors; and to rate the event-specific inferences they made for the cause, consequence, and self-meaning for the most negative life event experienced every day for 35 days.

Results

Descriptive statistics and correlations for the main variables from the initial assessment are presented in Table 1. All measures were moderately intercorrelated with each other.

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Note. N = 217 for all variables. All correlations are significant at p < .01. CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; RUM = Rumination subscale; BDI = Beck Depression Inventory; NEM-SR = Stress-Reaction subscale from Negative Emotionality factor of the Multidimensional Personality Questionnaire.
What Is the Dynamic Structure and Organization of Daily Negative Event-Specific Inferences?

To assess the structure and organization of daily ratings of negative event-specific inferences, we compared the pattern of empirical test–retest correlations against those predicted by both trait and contextual models of personality structure (Fraley & Roberts, 2005). Overall, the test–retest correlations were relatively moderate for event-specific inferences, because the correlations tended to range consistently from .30 to .45 regardless of the interval between assessments (mean test–retest $r = .38$, $SD = .08$, range = .56–.15). This suggests that the degree of stability across 3 days, for example, is just as high as the degree of stability over 30 days. Moreover, the average correlation between baseline negative cognitive style (i.e., CSQ) and a negative event-specific inference on any particular day was .33 ($SD = .13$, range = .46–.13). This suggests that the association between the CSQ and daily inferences also was relatively invariant across time. Taken together, these patterns are highly consistent with predictions made by a trait model.

To more formally evaluate alternative structural models of event-specific inferences, we compared the fit of each model to the empirical data. A trait model assumes that the pattern of correlations among event-specific inferences can be explained by a latent variable (i.e., a common factor) that contributes to variation in those inferences across time and circumstance (see the upper panel of Figure 1). The trait model predicts that the correlation between the negative event-specific inferences should be relatively invariant across time. For example, the correlation between the inferences made over a 2-day lag should be identical to the correlation observed over a 20-day lag. In contrast, a contextual model assumes that various statistical factors operate to influence or reorganize the nature of the inferences that people make (see the central panel of Figure 1). As such, to the extent to which there is stability in the inferences that people make from, say, Day 1 to Day 3, it is not because a latent trait is giving rise to those inferences but because the kinds of inferences made on Day 1 indirectly influence the kinds of inferences made on Day 3. The contextual model predicts that the stability of inferences between any 2 days will become smaller as the temporal interval between those days becomes larger.

For the purposes of evaluating each model, we developed a higher order system of structural equations that captured the causal mechanisms implied by both trait and contextual models. The structure of this superordinate model is illustrated in the lower portion of Figure 1. Notice that negative event-specific inferences are modeled as a function of a latent trait—a trait that is assumed to be stable across time and circumstance. In addition, we assume that the latent trait can be (imperfectly) indexed by the CSQ—a commonly used measure of dispositional depressogenic cognitive style. This model also allows for the form of event-specific inferences to carry over from 1 day to the next (i.e., the kinds of inferences made on Day $k$ have direct effects on the kinds of inferences made on Day $k + 1$). These autoregressive paths capture the dynamics assumed by the contextual model. In the analyses below, we constrain different parameters of this superordinate model to test separately the implications of trait and contextual models. After examining each model separately, we evaluate the ability of the combined or superordinate model to account for the data.

Figure 1. Alternative models of stability and change. The upper panel illustrates the dynamic relations among variables according to a trait model. The middle panel illustrates the dynamic relations among variables according to a contextual model. The lower panel illustrates a superordinate model that combines both trait and contextual processes. The upper row of etas ($\eta$) represents variation in event-specific inferences across repeated measurements (moving from left to right); the lowermost eta represents the latent trait of a dispositional cognitive style. Zeta ($\zeta$) represents residual or random variance.

In order to identify each parameter and to simplify the model as much as possible, we imposed the following constraints. (a) Each latent variable was scaled to have a variance of 1.00. The variance of each exogenous variable was constrained by fixing the variance of its residual term to be equal to 1.00 minus the weighted variance–covariance matrix of the predictor variables. (b) The paths leading from the latent trait to the daily inferences were freely estimated but constrained to be identical across the 35 days.

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1 Contact Benjamin L. Hankin for the test–retest correlation matrix across all 35 days.
Although it is unlikely that the latent trait would have identical effects from 1 day to the next, there is no way in which to derive from HT which days will have larger effects than others. Although we could allow for such variation by freely estimating the effect for each day (i.e., not constraining the paths to be identical), it seemed more conservative to us to constrain these paths to be equal. The added fit that would accrue from relaxing this constraint would not provide a test of the model per se (see Roberts & Pashler, 2000). We also constrained the autoregressive paths to be equal across time. (c) We assumed that the daily inferences were measured with perfect precision. As such, we set the causal path leading from those inferences to the measured variable to 1.00 and set the corresponding residual terms to 0. There is no reason to assume, of course, that these inferences were measured without measurement error, but imposing this constraint facilitates our ability to evaluate the theoretical structure of the model rather than the vicissitudes of measurement error. Relaxing this assumption would improve the fit of each model but would not improve our ability to compare the models, because this assumption and lack of measurement precision applies equally to each model. (d) The structure of the superordinate model implies that the covariation between Day 1 and all other days will necessarily be low because the “natural” or “implied” covariation among variables governed by the model’s structure requires time to accumulate. (There are no variables feeding into Day 1 inferences beyond the latent trait, whereas once the process gets started, each day’s inferences are influenced by the trait and the kind of inferences made the day before.) This state of affairs is an artifact because, technically, the model must begin somewhere, but there is no reason to assume that Day 1 of the empirical daily diary study captures the true beginning of the processes under empirical investigation. To adjust for this, the estimation procedure was based on generating the predicted correlation matrix across 50 days for a given set of parameter values and then extracting the lower \(35 \times 35\) correlation matrix from that larger matrix. The parameters of the models—as well as their final fit—were estimated and based on this submatrix. This allowed us to calibrate the models as if the 35 days under investigation were a “slice of life” (which they are) rather than the beginning of the processes under consideration.\(^2\)

We examined first the trait model by constraining the autoregressive parameters to equal zero. This model fit the data well (root-mean-square error [RMSE] = .081). Values between .05 and .08 suggest a good fit to data (Browne & Cudeck, 1993). The standardized coefficients for effect of the latent trait on event-specific inferences were estimated as .56, and the standardized effect of the latent trait on CSQ scores was estimated as .50. For the contextual model, we constrained the paths leading from the latent trait to the daily inferences to zero. The one exception, of course, was the path leading from the latent variable assessed by the CSQ to Day 1 inferences. This model did not provide as good of a fit to the data (RMSEA = .233). The standardized coefficients for the autoregressive paths were estimated as .85.

Finally, we examined the ability of the superordinate model—one that models both trait and contextual dynamics—to capture the data. This model fit the data as well as the trait model (RMSEA = .081). The standardized paths from the trait to daily inferences were estimated as .36, the path from the trait to the CSQ was estimated as .50, and the standardized autoregressive paths were estimated as .35. In summary, not only was a contextual model unable to explain the observed data well, but a model that combined autoregressive and trait structures was unable to explain the data any better than a model that assumed a trait structure exclusively.

To better illustrate the relative ability of each model to capture the empirical data, in Figure 2 we have plotted the stability functions for Days 1, 8, 15, 21, 28, and 35 (for in-depth discussion of stability functions, see Fraley, 2002; Fraley & Roberts, 2005). In short, a Day \(k\) stability function captures the correlation between inferences at Day \(k\) and all other days (i.e., Days 1–35). The empirical stability coefficients are illustrated as points in the figure. For example, the left-hand panel on the top row illustrates the correlation between inferences made at Day 1 with inferences made at Day 1 through Day 35; the middle panel on the top row illustrates the correlations between inferences made at Day 8 and all days. The best-fitting stability functions predicted by each model are illustrated by the curves in each panel. As can be seen, the empirical coefficients do not exhibit a simplex-like pattern. In fact, the curves generated by the best-fitting contextual model tend to underpredict the empirical data to a sizable degree as the test–retest interval increases. The fitted curves for the trait and combined models are virtually the same and explain the data as well as would be expected given the fact that the models were constrained to have equivalent paths from 1 day to the next—a highly restrictive constraint. In summary, the dynamic structure of event-specific inferences was most consistent with a model that assumes that a latent trait (i.e., an enduring vulnerability factor) influences the inference that people make across time and circumstance. This is consistent with the assumptions of HT.

**Overview of Individual-Level Analyses Over Time**

Hierarchical linear modeling (HLM; Bryk & Raudenbush, 1992; Raudenbush, 2000) was used to address the remaining questions in the study: (a) Are daily levels of depressive symptoms predicted by the causal etiological chain posited in HT, and (b) how much do HT’s etiological factors uniquely predict daily depressive symptoms above and beyond alternative vulnerabilities? HLM is a useful approach for understanding variation in depressive symptoms, because it can represent change within a person over multiple time points while also ascertaining how individuals may differ from one another in these trajectories over time (Bolger, Davis, & Rafaeli, 2003; Curran & Willoughby, 2003). The analysis of multiple levels of data is accomplished in HLM by constructing Level 1 and 2 equations. At Level 1, regression equations are constructed that model separately the variation in the repeated measure (i.e., depressive symptoms, event-specific inferences) as a function of time (i.e., the 35 days). Each equation includes parameters to capture features of an individual’s daily experience of depression and inferences over time, such as an intercept that describes an individual’s average level on the variable across time and a time-varying covariate that describes the strength of association between depressive symptoms and event-specific inferences. At Level 2, equations are specified that model individual differences in the Level 1 parameters as a function of between-

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\(^2\) Because of this last consideration, we were unable to estimate the parameters of the models in commonly used structural equation modeling packages. Instead, we estimated the parameters using programs written by John R. Z. Abela for the S-Plus programming environment (MathSoft, 1999). These programs are available on request.
subjects variables (e.g., HT's cognitive style, BT's dysfunctional attitudes, neuroticism). Thus, the Level 1 equations capture a single individual's trajectories for depressive symptoms and event-specific inferences over time, and the Level 2 model organizes and explains the between-subjects differences among these trajectories as a function of between-persons factors.

A significant advantage of HLM is that it can flexibly handle cases with missing data (i.e., depressive symptoms or event-specific inferences). Random-effects models, such as HLM, do not require that every participant provide complete, nonmissing data over the 35 days. The program used for the present analyses, HLM 5.04 (Raudenbush, Bryk, Cheong, & Congdon, 2001), assumes that completed ratings of depressive symptoms and event-specific inferences are representative of the particular participant's modal trajectory (Bryk & Raudenbush, 1992; Hedeker & Gibbons, 1997). As such, participants with missing data are not eliminated from the data set because their available data points are used to estimate the individual-level parameters at Level 1 (e.g., intercepts and time-varying covariates).

Are Dispositional Depression Vulnerabilities Associated With Negative Event-Specific Inferences?

To start, HLM was used to model individuals' negative event-specific inferences made each day for negative events occurring in different situational contexts. At Level 1, participants’ negative event-specific inferences were modeled as a function of two parameters (i.e., intercept and linear slope) plus random error. Table 2 summarizes the results of this analysis. Participants were char-

![Figure 2](image-url)
acterized by an average event-specific inference level of 18 and a slope of approximately zero. There were no sex differences for either the intercepts or slopes. Thus, on average, individuals did not change systematically over time in the trajectory or slope of their event-specific inferences made to various stressors.

Next, analyses were conducted to examine whether participants’ overall dispositional depression vulnerabilities, especially depressogenic cognitive style as hypothesized by HT and as assessed at the start of the study with the CSQ, relate meaningfully to the event-specific inferences they made every day for the most negative life events experienced daily over the 35 days of the prospective study. To address this question, participants’ T1 dispositional CSQ scores, DAS scores, RUM scores, T1 NEM-SR scores, and T1 BDI scores were entered together as Level 2 variables to explain variability in the Level 1 intercept parameters for the event-specific inferences (i.e., the mean of these scores). Table 2 summarizes the results of this model. Participants’ T1 CSQ and T1 neuroticism scores at Level 2 were associated positively with the mean event-specific inference level. T1 dysfunctional attitudes, rumination, and initial depression were not significantly associated with the intercept parameters. With all of the depression vulnerabilities entered at Level 2, a moderate amount of variability in participants’ negative event-specific inference intercepts (48%) was accounted for (for discussion of percentage of variance accounted for by Level 2 variables in HLM, see Bryk & Raudenbush, 1992, pp. 74, 141–142). CSQ scores accounted for a moderate 41% and neuroticism explained a relatively small 7% of variability in average event-specific inferences. These results show that participants’ mean levels of negative event-specific inferences were associated particularly with dispositional levels of HT’s depressogenic cognitive style scores and mildly with neuroticism.

Predicting Daily Depressive Symptoms

Are negative event-specific inferences related to daily depressive symptoms? To examine whether daily negative cognitions were associated with daily depressive symptoms, an idiographic Level 1 model was fit in which daily depressive symptoms were modeled as a function of HT’s negative event-specific inferences. This model allows us to examine whether individuals’ fluctuations in depressive symptoms over the 35 days are associated with fluctuations that deviate from individuals’ mean negative event-specific inference score. The results for this analysis show that fluctuations in depressive symptoms over time were associated with the within-person deviations from individuals’ mean level of negative-event specific inferences (coefficient = .13, p < .001), accounting for 41.76% of the variance in daily depressive symptoms.

Are dispositional depression vulnerabilities and negative event-specific inferences associated with daily depressive symptoms? We next examined a more complete, within-person model to investigate variation in daily rated depressive symptoms. In this model, idiographic deviations from an individual’s mean level of negative event-specific inferences were included in Level 1 as a time-varying covariate, and at Level 2 all depression vulnerabilities (T1 CSQ, T1 DAS, T1 RUM, T1 BDI, and T1 NEM-SR) were included to account for variation in depressive symptoms. Results for this more comprehensive model are presented in the top of Table 3. Dispositional T1 CSQ, T1 DAS, and T1 NEM-SR were associated significantly with daily rated depressive symptoms, whereas T1 BDI and T1 RUM were not. The idiographic time-varying covariate of negative event-specific inferences at Level 1 remained a significant predictor of depressive symptom variability (coefficient = .13, p < .001), even when these other factors were considered. The inclusion of the full set of variables accounted for a large amount of variation in daily rated depressive symptoms (53.65%). The addition of the Level 2 dispositional variables accounted for an additional 12.00% of variance in explaining depression variability beyond the variance explained by the previous model that included only the idiographic negative event-specific inferences as a time-varying covariate.

It is worth noting that this model, combined with information obtained in the previous analyses, provides the necessary information to test HT’s etiological chain according to the mediation criteria outlined by Baron and Kenny (1986). Specifically, these analyses show that negative event-specific inferences mediated the association between negative cognitive style and daily depressive symptoms because (a) dispositional cognitive style predicted daily negative event-specific inferences, (b) dispositional cognitive style predicted daily depressive symptoms, (c) daily negative event-specific inferences covaried with daily depressive symptoms, and

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3 We did not include a linear slope term in the depressive symptoms models, like we did with the negative event-specific inference models, because there was no strong a priori theoretical justification for assuming linear change over this “slice of life.” Moreover, visual inspection of the depressive symptoms data over time suggested there was not a linear trend. Because our primary goal with these depression analyses was to examine dispositional and time-varying predictors of daily depression, we chose to examine individuals’ fluctuations in daily depressive symptoms (i.e., intercepts) over the course of the study. We also conducted all of the HLM analyses reported next using only the symptom of sad, depressed mood as the outcome, because it was conceivable that we may have been examining secondary features of depression (e.g., loss of appetite, change in sleeping) more than the primary, obligatory affective symptoms of depression by using the depression symptom composite reported in the main text. All of the findings reported in the main text, using the composite depressive symptoms variable as outcome, were similar to the sad, depressed mood symptom as the outcome. The only difference was that rumination predicted sad/depressed mood but not composite depressive symptoms and that dysfunctional attitudes predicted composite depressive symptoms but not sad/depressed mood. Contact Benjamin L. Hankin for these results.

4 It is conceivable that individuals with higher levels of dispositional vulnerabilities may experience more objectively negative life events that could influence negative event-specific inferences and, in turn, daily depressive symptoms. To evaluate this hypothesis, we coded all of the negative events that participants recorded for seriousness/threat level (e.g., Hammen, 1991; Monroe & Simons, 1991). Negative cognitive style, dysfunctional attitudes, rumination, and initial depression were not significantly associated with objectively coded serious level of events. Only neuroticism, τ(210) = 2.04, p < .05, was associated with the severity of coded event variables: Higher levels of neuroticism were related to encountering more serious events. Given this finding, we reconducted the primary analyses shown in Table 3 while including the number of objectively coded events as a Level 1 covariate along with the daily event-specific inferences at Level 1, all dispositional variables at Level 2, and the cross-interactions. Number of objective events, as a time-varying covariate, was significantly associated with daily depression, but this did not alter the pattern or magnitude of findings presented in Table 3.
Table 3
Hierarchical Linear Model Results for Depressive Symptoms Across 35 Days

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSQ</td>
<td>0.57*</td>
</tr>
<tr>
<td>DAS</td>
<td>0.02*</td>
</tr>
<tr>
<td>RUM</td>
<td>0.03</td>
</tr>
<tr>
<td>NEM-SR</td>
<td>0.19**</td>
</tr>
<tr>
<td>BDI</td>
<td>0.04</td>
</tr>
<tr>
<td>HT’s negative event-specific inferences idiographic time-varying covariate</td>
<td>0.13***</td>
</tr>
</tbody>
</table>

Dispositional predictors and cross-level interactions between dispositional variables and negative event-specific inferences predicting daily rated depressive symptoms across 35 days:

**Predictor Coefficient**

Dispositional predictor

| CSQ       | 1.00**      |
| DAS       | 0.02*       |
| RUM       | 0.00        |
| NEM-SR    | 0.05        |
| BDI       | 0.04        |
| Cross-level interaction

| CSQ × Event Inferences | −0.03*     |
| DAS × Event Inferences | 0.00       |
| RUM × Event Inferences | 0.00       |
| NEM-SR × Event Inferences | 0.01** |
| BDI × Event Inferences | 0.00       |
| HT’s negative event-specific inferences idiographic time-varying covariate | 0.11*** |

Note. N = 210. Asterisks indicate that the coefficient differs significantly from 0. CSQ = Cognitive Style Questionnaire; DAS = Dysfunctional Attitudes Scale; RUM = Rumination subscale; BDI = Beck Depression Inventory; NEM-SR = Stress-Reaction subscale from Negative Emotionality factor of the Multidimensional Personality Questionnaire; HT = hopelessness theory of depression.


(d) the association between dispositional cognitive style and daily depression was reduced by including the time-varying covariate of negative event-specific inferences. Sobel’s (1982) test shows that negative event-specific inferences significantly mediated the association (z = 5.64, p < .001), and this was a medium-sized mediated effect (MacKinnon et al., 2002). Daily negative inferences explained 8.5% of the neuroticism and daily depression association.

Is there an interactive effect between dispositional depression vulnerabilities and daily negative cognitions as predictors of daily depressive symptoms? In the final model, we examined the interactive effects of dispositional vulnerabilities and idiographic negative event-specific inferences in predicting depressive symptoms. Specifically, depressive symptoms were modeled as a function of deviations from individuals’ mean level of negative event-specific inferences over the 35 days at Level 1. At Level 2, both the Level 1 intercepts and the coefficients for event-specific inferences were modeled as a function of all the depression vulnerabilities (i.e., T1 CSQ, T1 DAS, T1 RUM, T1 BDI, and T1 NEM-SR). By modeling the coefficients for the event-specific inferences as a function of these vulnerabilities, we were able to study the cross-level interactions between idiographic daily negative event-specific inferences and each of the depression vulnerabilities. Results for this comprehensive model are presented in the bottom of Table 3. The idiographic time-varying covariate of negative event-specific inferences at Level 1 predicted depressive symptom variability. Dispositional T1 CSQ and T1 DAS remained as significant predictors of daily rated depressive symptoms, whereas T1 NEM-SR, T1 BDI, and T1 RUM were not associated with depressive symptoms. Finally, the cross-level interaction of idiographic negative event-specific inferences with T1 CSQ and with T1 NEM-SR significantly predicted daily depressive symptoms. The inclusion of all of the main effect dispositional vulnerabilities and the cross-level interactions of idiographic daily negative event-specific inferences with dispositional CSQ and NEM-SR accounted for a large degree of variation in daily rated depressive symptoms (55.90%). The cross-level interactions accounted for an incremental 2.25% of variance in explaining depression variability.

The interactions are illustrated in Figure 3. As seen in the top panel, dispositional negative cognitive style differentiated individuals’ experience of daily depressive symptoms at low, but not high, levels of idiographic negative event-specific inferences. Specifically, individuals with high levels of dispositional negative cognitive style (1 standard deviation above the mean on CSQ) exhibited more depressive symptoms than those with low levels of dispositional negative cognitive style (1 standard deviation below mean on CSQ) when making relatively positive inferences (i.e., low negative event-specific inferences) about the most negative event of the day. People high and low in dispositional cognitive style reported equivalent (and high) levels of depressive symptoms when more daily negative event-specific inferences were made. The lower panel of Figure 3 illustrates the cross-level interaction between neuroticism and idiographic daily negative cognitions. Individuals with high levels of neuroticism (+1 standard deviation on NEM-SR) exhibited the highest elevations in depressive symptoms, especially when they made more negative inferences about a stressor, compared with those low on neuroticism (−1 standard deviation on NEM-SR).

Discussion

The present investigation sought to test predictions derived from social–cognitive theories of depression, HT in particular, using an intensive, naturalistic daily diary study. Young adults completed
baseline assessments of dispositional depression vulnerabilities and made event-specific inferences concerning the most negative event they experienced for 35 days. Across that time span, these young adults also rated their daily depressive symptoms. In the sections that follow, we review the main findings from the present study and discuss the ways in which this research advances current knowledge on etiological factors for the development of depression.
The Organization and Structure of Event-Specific Inferences for Daily Stressors

Although HT assumes that the inferences people make to daily stressors have a traitlike structure, this fundamental assumption has never been tested. To evaluate this hypothesis, we compared the ability of a trait model, a contextual model, and a combined model to explain the patterning of event-specific inferences. Results indicate that the event-specific inferences made to daily stressors are organized in a traitlike manner and not in a contextual fashion. Indeed, the trait model fit as well as a superordinate model that combined both trait and contextual features.

Although we found support for the assumption that there is an enduring latent trait that contributes to the stability of negative inferences over time, it is important to point out that the magnitude of the cross-time correlations was modest. The moderate strength of these test–retest correlations across days (.38 across all lags) suggests that other factors, in addition to a dispositional negative cognitive style, contribute to the negative event-specific inferences that people make to stressors on any given day. However, it does not appear that these factors fundamentally impact the way in which the person functions across time. Although depressogenic cognitive style modestly impacted the inferences that were made on any one day, the influence of that style remained constant across the 35-day period.

Prediction of Daily Depressive Symptoms by HT’s Causal Etiological Chain and Other Dispositional Depression Vulnerabilities

HT posits that individual differences in dispositional depressogenic cognitive style will be associated with negative event-specific inferences and daily depressive symptoms and that event-specific inferences will be associated with daily experiences of depressive symptoms. We tested these hypotheses by examining (a) the relationship between individuals’ cognitive styles as assessed at the start of the study and depressive symptoms assessed daily over time and (b) the association between fluctuations in daily rated event-specific inferences and depressive symptoms. Consistent with HT, our results show that individuals’ dispositional depressogenic cognitive style predicted their average levels of negative event-specific inferences and daily depressive symptoms. Further, individuals’ deviations from their average level of daily negative event-specific inferences were associated with and explained considerable variance in individuals’ daily depressive symptoms. Both negative cognitive style and idiographic negative event-specific inferences predicted fluctuations in daily depressive symptoms when included in the same model.

These findings are consistent with and extend previous research on HT’s etiological factors. Prior research has found that a dispositional negative cognitive style is associated with depressive symptoms cross-sectionally (e.g., Sweeney et al., 1986), retrospectively over the lifetime (e.g., Alloy et al., 2000; Haefeli et al., 2003), and prospectively (e.g., Alloy, Just, & Panzarella, 1997; Hankin et al., 2004; Metalsky et al., 1993; Metalsky & Joiner, 1992). In his intensive multiwave research, Swendsen (1997, 1998) found that a dispositional cognitive style predicted negative event-specific inferences, and event-specific inferences were associated with depressive symptoms. He used an experiencesampling design in which participants reported on attributions and depressive symptoms five times daily over 1 week. We extended Swendsen’s research by focusing less on the multiple hassles that might take place on a given day and more on the most salient and negative event of the day. Despite this methodological difference, it is important to note that the findings were remarkably similar across studies.

In addition to testing HT’s specific causal etiological chain, we also addressed the unique predictive power of HT’s etiological components compared with other known vulnerabilities to depression. Specifically, we examined dispositional cognitive style, rumination, dysfunctional attitudes, initial depressive symptom levels, and neuroticism as individual-difference predictors of daily negative event-specific inferences and depressive symptoms. Neuroticism and cognitive style, but not dysfunctional attitudes, rumination, or initial depressive symptoms, predicted individuals’ average levels of daily negative cognitions. Dispositional negative cognitive style, dysfunctional attitudes, and neuroticism as well as idiographic daily negative cognitions, but not rumination or initial depressive symptoms, significantly predicted daily depressive symptoms. It is interesting to note that some, but not all, of these dispositional vulnerabilities predicted daily levels of depressive symptoms. We speculate that rumination and initial depression may not have remained significant predictors of depression after including the other depression vulnerabilities in the same model because rumination and initial depression are more strongly associated with neuroticism than are a dispositional negative cognitive style and dysfunctional attitudes. Thus, neuroticism, but not rumination or initial depression, may have emerged as the significant predictor of daily depression after controlling for the overlapping variance among these moderately intercorrelated vulnerabilities. However, because this study is one of the first prospective, intensive daily diary studies to examine multiple dispositional vulnerabilities as predictors of daily depression, we await future research before offering a more detailed account of why certain dispositional vulnerabilities predict elevations in daily depressive symptoms more strongly than others.

These findings provide important evidence that the dispositional negative cognitive style proposed in HT can be meaningfully differentiated from other cognitive vulnerabilities to depression. Other research, aimed at unpacking generic cognitive vulnerability to depression, has also supported the discriminant validity position. Factor analytic work, for example, has shown that HT’s negative cognitive style and BT’s dysfunctional attitudes load meaningfully onto different latent factors (Hankin et al., 2003; Joiner & Rudd, 1996). This is the first study, to our knowledge, to find evidence for the unique predictive power of HT’s etiological causal chain when compared with other prominent cognitive risk factors for depression in an intensive, multiwave prospective study. This evidence also suggests that HT’s constructs of dispositional negative cognitive style and negative event-specific inferences are not merely generic social–cognitive factors that are interchangeable with other depression vulnerabilities. Nonetheless, it is important to note that our study did not provide a rigorous, microanalytic examination of the other theories’ etiological chains. For example, BT posits that dysfunctional attitudes express themselves more specifically in relation to stressors encountered in particular domains, such as when sociotropic people become depressed in the face of social stressors.

Finally, we explored whether any of the dispositional depression vulnerabilities moderate the daily association over time between
depressive symptoms and individuals’ changes in their average level of daily negative event-specific inferences beyond the main-effect influence of each of these factors. We found that the cross-level interactive combination of idiographic daily negative cognitions and dispositional negative cognitive style and neuroticism, respectively, predicted significant incremental variability in daily depressive symptoms. Of particular interest, these novel findings reveal that on days when individuals made more negative inferences about daily stressors than they did on average, they exhibited elevated depression regardless of their dispositional negative cognitive style. When they made more benign, nonnegative inferences for a daily stressor than they did on average, people with a more negative cognitive style, compared with those with a more optimistic cognitive style, reported relatively greater depressive symptoms. In contrast, highly neurotic individuals who made more daily negative event-specific inferences than their average reported the greatest elevation in daily depression.

The cross-level interaction between negative cognitive style and daily negative cognitions is most consistent with a titration model, in which lower levels of vulnerability differentiate one’s likelihood of exhibiting depression, whereas the Neuroticism × Daily Negative Cognitions interaction conforms more to a traditional vulnerability model (see Abramson, Alloy, & Hogan, 1997). These novel findings also suggest that neuroticism and dispositional negative cognitive style are not interchangeable vulnerabilities (cf. Clark et al., 1994; Zuroff, Mongrain, & Santor, 2004) because they interact with daily negative cognitions differently to predict daily depressive symptoms. This suggests that potentially different processes may explain how neurotic and pessimistic people become depressed over time. For example, highly neurotic people may exhibit elevated depression levels because they are more likely to generate additional stressors over time (see, e.g., Footnote 4; Lakdawalla & Hankin, 2004; Van Os & Jones, 1999), whereas pessimistic people were not found to generate more stressors (see Footnote 4). As neurotic individuals experience these additional stressors, they may in turn experience more depression, especially when they then explain these stressors in more negative ways (i.e., make negative event-specific inferences). Because this differential pattern of interactions represents a novel finding, it will be important for future research to explore how potentially different processes contribute to depression. Further elucidating these processes would help advance knowledge about the development of depression from social–cognitive and personality perspectives.

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