Moving Beyond Main Effects: A Data Analytic Strategy for Testing Complex Theories of Clinical Phenomena

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The goal of this study was to advance a data analytic strategy for testing sophisticated models of clinical phenomena. We illustrate this method by testing a prominent and highly specific cognitive model of depression (Abramson et al., 1989). Specifically, we used multilevel modeling (MLM) to test the entire sequence featured in the hopelessness theory. The study used a daily diary design with a sample of undergraduates (n = 210). To our knowledge, this is the first study to use multilevel modeling with multiple waves of data to test a model with two mediators and a moderator. Results of analyses provided strong support for the MLM strategy and offer a concrete example for how to test complex theories of clinical phenomena.

Key words: **clinical phenomena, cognitive vulnerability, data analysis, hopelessness theory, multilevel modeling. [Clin Psychol Sci Prac 21:385–397, 2014]**

Most research studies in clinical psychology aim to detect a "main effect" of some condition. For example, studies focused on the etiology of particular disorders aim to detect a main effect of "group" (i.e., a difference between a disordered group and a control group). Similarly, studies focused on the effectiveness of psychotherapies aim to detect a main effect of "intervention" (i.e., a difference between the therapy of interest

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and a control condition). Such studies are necessary for identifying markers of psychopathology and for determining which interventions are efficacious. However, it is time to move beyond the relatively simple question of whether or not two groups are different. It is critical that the field begin to test the complex sequences of mediators and moderators specified by many theories of psychopathology and psychotherapy (Baker, McFall, & Shoham, 2008; Kazdin, 2007; Kazdin & Blasé, 2011; Meehl, 1978).

The ability to test and corroborate highly specific theories of clinical phenomena has important implications for understanding the etiology of psychopathology and how best to prevent it. For example, highly specific theories of etiology can delineate the mechanisms (i.e., mediators) by which various risk factors culminate in clinical symptoms as well as the individual and contextual factors (i.e., moderators) that affect their potency. This type of theoretical specificity can then be used to identify who is most at risk for developing a particular disorder and provide information about what specific factors to target for promoting resilience.

It is equally important to identify mediators and moderators of intervention outcomes. Understanding the mechanisms (i.e., mediators) that drive symptom change not only advances etiological theories of mental illness, but also fuels novel and more effective interventions. Once scientists identify mechanisms of change, they can eliminate intervention components that are ineffective or inefficient and focus on advancing techniques that actually do work. This should lead to more focused,

time-efficient, and diagnostically tailored therapies (Baker et al., 2008). Further, the identification of moderators of intervention efficacy can shed light on for whom a particular intervention might work best (e.g., men versus women) as well as the contexts that lead to optimal outcomes (e.g., outpatient versus inpatient; Baker et al., 2008; Kazdin, 2007).

Despite its clear importance, the testing of complex theories (i.e., those with multiple mediators and moderators) of clinical phenomena has progressed slowly. We suspect that the slow progress is due, in part, to a lack of appropriate data analytical strategies for testing sophisticated theoretical sequences. To date, there are few, if any, examples of statistical models from any area of psychology that deal with multiple mediators and moderators. Our objective in the current study was to illustrate an advanced multilevel modeling (MLM) procedure for testing such models.

Multilevel modeling (Bryk & Raudenbush, 1987) is ideally suited for testing complex theories because it can handle multiple observations across multiple time points. This is critically important for testing theories of mediation, which hypothesize processes that unfold over time. Further, MLM can effectively handle the statistical dependencies and missing data that are common in repeated measure data[1] (Kenny, Korchmaros, & Bolger, 2003). MLM is also the preferred strategy for dealing with hierarchical data (e.g., daily within-person data for each individual nested in within-person-level data between individuals). In such hierarchical data structures, traditional methods of evaluating mediation are inappropriate. In hierarchical data, the independence assumption of traditional methods of data analysis (e.g., regression analysis) is compromised, and thus, traditional methods will yield biased estimates of the effects of the model (Bauer, Preacher, & Gil, 2006; Kenny et al., 2003). An appropriate alternative method to model these data is

through the use of MLM (Bauer et al., 2006; Kenny et al., 2003). This results not only in the statistical benefits just described, but also permits the testing of predictors at different levels of data (within-person and between-person) to be analyzed simultaneously (Nezlek & Allen, 2006).

We established the value of using MLM by testing the entire etiological chain featured in a prominent cognitive theory of depression, the hopelessness theory (HT; Abramson, Metalsky, & Alloy, 1989). The HT specifies one of the most detailed etiological chains in all of clinical psychology. Whereas many "theories" of depression posit an association between a single risk factor (e.g., serotonin, poor social support) and depression, HT specifies an entire sequence of factors that culminates with increases in depressive symptoms. As shown in Figure 1, stressful life events are hypothesized to interact with cognitive vulnerability to produce specific negative inferences about the cause, consequences, and self-worth implications of these events. Such event-specific negative inferences are then hypothesized to contribute to the development of hopelessness, which, in turn, is posited to lead to depressive symptoms. Thus, HT elucidates the mechanisms by which stress leads to depressive symptoms as well as identifies which individuals (those with a cognitive vulnerability) are most likely to follow this depressive pathway.

Each component of the theory has garnered at least some empirical support. Most studies have focused on the cognitive vulnerability by stress interaction hypothesis. Using prospective longitudinal designs, these studies have consistently found that cognitive vulnerability interacts with measures of stressful life events to predict the development of depressive symptoms and depressive disorders (Alloy, Abramson, Walshaw, & Neeren, 2006; Haeffel et al., 2008; Hankin, Abramson, Miller, & Haeffel, 2004). Far fewer studies have attempted to test the mediating
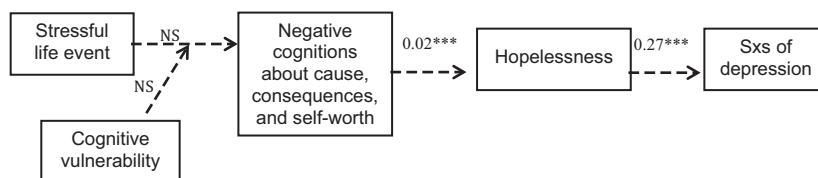

Figure 1. The hopelessness theory of depression as applied to the lagged model. Each outcome represents the score on that variable on the day following the predictor (e.g., "Sxs of depression" refers to depression levels on the day following the hopelessness symptoms). *Note*: *** represents $p < .001$.

components of hopelessness theory's etiological chain. That said, preliminary work has largely been positive. For example, stressful life events and cognitive vulnerability do combine to predict event-specific negative inferences (e.g., Haeffel, 2011; Hong, Gwee, & Karia, 2006; Panzarella, Alloy, & Whitehouse, 2006) and hopelessness (e.g., Gibb, Alloy, Abramson, & Marx, 2003; Gibb et al., 2001; Haeffel, Abramson, Brazy, & Shah, 2008), which has been shown to mediate the relationship between the cognitive vulnerability and depression (e.g., Gibb et al., 2001; Hong et al., 2006; Metalsky & Joiner, 1992). However, despite nearly two decades of research, all components of the theory (i.e., the entire etiological chain) have yet to be tested simultaneously. One likely reason for this is the complexity of the statistical model needed to test it.

We chose to illustrate our data analytic approach using HT for a number of reasons. First, this theory specifies a highly detailed etiological chain that includes two mediators (e.g., event-specific negative inferences and hopelessness) and a moderator (cognitive vulnerability). Second, a study exists in which all of the relevant HT model variables were measured over multiple time points. Finally, the HT maps closely onto existing cognitive interventions (e.g., cognitive behavioral therapy) and, thus, also can provide an example for testing complex models of psychotherapy outcomes. To our knowledge, this is the first study to use multilevel modeling with multiple waves of data to test a model with two mediators and a moderator.

METHOD

Overview

Undergraduates completed a baseline measure of cognitive vulnerability and then a daily diary form for 35 consecutive days (see Hankin, 2010; Hankin, Fraley, & Abela, 2005; for additional information about the sample and study design). The daily diary form assessed stressful life events, event-specific negative inferences, hopelessness, and depressive symptoms.

Participants

Participants were 217 undergraduates (mean age = 18.7 [range 18–23]; 62 males, 155 females; self-reported race/ ethnicity = 84% Caucasian, 1% African American, 4% Hispanic, 5% Asian American, 6% Other) who were recruited from an introductory psychology course. Seven participants were excluded from the sample because they did not complete the daily diary portion of the study. Thus, a total of 210 were included in the analyses. Participants received extra credit for their participation.

Measures

Cognitive Vulnerability. The Cognitive Style Questionnaire (CSQ; Haeffel et al., 2008) was used to assess the cognitive vulnerability factor featured in HT (negative inferences for cause, consequence, and self-worth). The CSQ assesses participants' causal attributions for the 12 hypothetical negative events (six achievement and six interpersonal) on dimensions of stability and globality; in addition, participants rate the probable consequences of each event and the self-worth implications of each event. Mean-item scores can range from 1 to 7, with higher scores reflecting more negative cognitive styles. The CSQ has good internal consistency, reliability, and validity (see Haeffel et al., 2008, for review). The CSQ was administered at the baseline assessment ($\alpha = .92$).

Daily Depressive Symptoms. Each day, participants responded to a depression scale consisting of the nine depressive symptoms featured in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994). Participants rated how much they had experienced each symptom on that day (1–5 scale). The daily depressive symptoms were scored by summing the participants' responses; therefore, scores could range from 9 to 45 each day. This short depression scale has demonstrated good reliability (both internal consistency and test–retest) and validity (face validity, convergent validity, and construct validity) in prior research (e.g., Hankin, 2010; Hankin et al., 2005). Internal consistency (coefficient alpha) in the current study for a composite depressive symptom score averaged across one week was .92.

Hopelessness. Each day, participants responded to an abbreviated version of the Expanded Hopelessness Scale (Metalsky & Joiner, 1992). The scale consists of four items that are rated on a 1–5 scale. The scale was created from the Expanded Hopelessness Scale using factor

analysis to determine the four items that exhibited the highest factor loadings on hopelessness (n = ~650 undergraduate participants). Dr. Lyn Abramson, creator of the hopelessness theory, was also consulted with regard to the four items chosen. The abbreviated scale demonstrated good reliability (e.g., internal consistency and test–retest) and convergent validity (e.g., scale is highly correlated with depressive symptoms) in the current study. Internal consistency (coefficient alpha) in the current study for composite hopelessness scale score averaged across one week was .95.

Daily Stressful Life Events. Each day, participants could list up to five stressful events that occurred. Coders were trained to rate the objective threat of the stressors using an objective, investigator-based coding system (Brown & Harris, 1978). All of the negative events that participants recorded in the diary booklets for the 35 consecutive days were coded twice by independent coders. The coders were reliable in their ratings of the objectiveness coding of each event (>85% agreement; kappa = .68) for the objective stressors. Prior research indicates that the contextual threat method of coding daily events is valid (Hankin, Mermelstein, & Roesch, 2007). Analyses used the sum of the objective stressors encountered on a particular day as the coded variable.

Event–Specific Negative Inferences. The Particular Inferences Questionnaire (PIQ; Metalsky, Halberstadt, & Abramson, 1987) was used to assess event-specific negative inferences. The PIQ is a four-item questionnaire that assesses students' inferences for a specific life stressor (e.g., a poor midterm grade). Using the same exact format as the CSQ, the PIQ assesses participants' inferences about the cause, consequences, and self-worth implications of the life stressor (on scales ranging from 1 to 7). In this study, participants were instructed to "Think about the most stressful event that day" and then write down the one major cause of that event. Then they made ratings on dimensions of stability and globality; in addition, participants rated the probable consequences of each event and the self-worth implications of each event. The PIQ has demonstrated good reliability (both internal consistency and test–retest) and excellent predictive validity in

prior studies (e.g., Haeffel, 2011; Metalsky et al., 1987). We scored the daily event-specific inferences by summing the participants' responses; thus, scores could range from 5 to 35 each day, with higher scores reflecting a greater degree of event-specific negative inferences. Alpha was .82 for event-specific inferences composite.

Procedure

At the baseline assessment, participants completed a packet of questionnaires, including the CSQ. After completing the baseline packet, participants individually came to the laboratory, where they were instructed how to complete the daily diaries for the next 35 days. Participants used the daily diary record form to rate their level of depressive symptoms and hopelessness, to write down the occurrence of daily stressors, and to rate the event-specific inferences they made for the most stressful life event experienced every day. Participants were instructed to complete the diary every day at the end of the day and to turn in their daily diaries to the laboratory on the day of their Introduction to Psychology class. 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. Overall, it appeared that participants 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). Attrition analyses revealed no significant differences on dispositional measures for those who completed all diaries from those who did not.

DATA ANALYTIC STRATEGY

The etiological chain of the hopelessness theory was assessed, including all theorized mediations. In particular, the daily chain from stress to depression is tested, *within* individuals. This relationship is tested by a lagged analysis. In other words, we were assessing whether stress on one day predicts daily cognitions on the next day, whether daily cognitions on one day influence hopelessness on the following day, and whether hopelessness on one day influences depression on the following day.

The entire lower-level (within–person) chain can be described by the following three equations:[2]

$$DailyNegCog_{(i+1)j} = \beta_{NC0_j} + \beta_{NC1_j}(Stress)_{ij} + \varepsilon_{NC_{ij}}$$

$$Hopelessness_{(i+1)j} = \beta_{H0_j} + \beta_{H1_j}(dailyNegCog)_{ij} \\ + \beta_{H2_j}(stress)_{ij} + \varepsilon_{H_{ij}}$$

$$Depression_{(i+1)j} = \beta_{D0_j} + \beta_{D1_j}(Hopelessness)_{ij} \\ + \beta_{D2_j}(dailyNegCog)_{ij} + \beta_{D3_j}(stress)_{ij} \\ + \varepsilon_{D_{ij}}$$

In the first equation, $dailyNegCog_{(i+1)j}$ refers to the negative cognition score on day $i + 1$ (i.e., on the subsequent day as the predictor variables) for person j. β_{NC0_j} refers to the intercept of daily negative cognitions, β_{NC1_j} is the parameter testing the within–person daily relationship between stressful events on one day and negative cognitions the following day, and $\varepsilon_{NC_{ij}}$ is the within–person variability (i.e., it is the deviation of each person's negative cognition from the predicted negative cognition based on the Level 1 model).

In the second equation, $Hopelessness_{(i+1)_j}$ refers to the hopelessness score on day $i + 1$ for person j. β_{H0_j} is the intercept of hopelessness, and β_{H1_j} is the parameter testing the within–person relationship between negative cognitions on one day and hopelessness on the subsequent day. Controlled for is the influence of stress (measured the same day as negative cognitions) on hopelessness the next day (this relationship between stress and hopelessness is represented by β_{H2_j} in the above equation). Finally, $\varepsilon_{H_{ij}}$ represents the within–person variability in hopelessness within each measurement occasion.

In the third equation, $Depression_{(i+1)j}$ refers to the depression score on day $i + 1$ for person j. β_{D0_j} is the intercept in depression, and β_{D1_j} is the parameter testing the influence of hopelessness on one day on depression the following day. Controlled for is the relationship between negative cognitions (measured on the same day as hopelessness) and depression (represented by the parameter β_{D2_j}) and the relationship between stressful events (measured on the same day as hopelessness) and depression (represented by parameter β_{D3_j}). Finally, $\varepsilon_{D_{ij}}$ represents the within–person variability in depression within each measurement occasion.

Additionally, through the use of MLM, the between-person moderating influence of cognitive vulnerability on the first piece of this within-person process was investigated, as part of the entire chain tested simultaneously. Specifically, the moderation analysis assessed whether individuals higher in general cognitive vulnerability exhibit a stronger daily relationship between negative cognition and exposure to stressful events. This can be seen in the following Level 2 equation:

$$\beta_{NC1_j} = \gamma_0 + \gamma_1(NegCog)_j + U_j$$

In the above equation, β_{NC1_j} represents individuals' average daily relationship between stressful events and negative cognitions on the following day. Specifically of interest was parameter γ_1, representing the influence of general cognitive style on this average daily relationship. Also included in the model are the intercept of the average daily relationship (γ_0) and the deviation from the predicted average relationship based on the Level 2 model (U_j).

The described analyses were performed using SAS PROC MIXED version 9.3 (Bauer et al., 2006; see Appendix for model syntax). Normality was checked using QQ plots of the residuals, and homogeneity was assessed via residual plots; results of these checks indicated that residuals were marginally normal and showed no evidence of heterogeneity. Although the QQ plots suggested that the residuals may be slightly skewed, inferences regarding fixed effects are generally robust to moderate violations of normality (Maas & Hox, 2004). Using the approach outlined in detail by Bauer and colleagues (2006) and modeling allows for simultaneous modeling of variables, and in the present study, the full etiological chain was tested. Restricted maximum likelihood was the estimation method used, and convergence criteria were met. A heterogeneous compound symmetry covariance structure was used.

Predictor variables were all centered. Level 1 daily predictors were centered on individual means (Bryk & Raudenbush, 1987); for example, a positive value for stressful life events after centering for a given participant on a particular day indicated an above-average number of events for him or her on that day relative to his or her mean across time. In this way, individuals are

used as their own control; we test the theorized chain for an individual by studying intraindividual variability. The Level 2 global predictor, cognitive vulnerability, was centered on group means (Bryk & Raudenbush, 1987). In this way, positive scores on cognitive vulnerability indicated individuals who reported above-average levels of cognitive vulnerability.

As described, the data used in the present analyses occurred over consecutive days. In such data, responses made on days closer in time to each other may be more highly correlated than days that are farther apart, and this should be accounted for (Bauer et al., 2006). As a result, a continuous-time autoregressive structure was used, which accounts for this potential serial autocorrelation (Schwartz & Stone, 1998).

In the present analyses, the within-person chain between stressful life events and depression (described in the equations above) was tested simultaneously, with all variables shown in Figure 1 included in one model. Joint significance testing (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002) was used to assess mediation processes in the present study. In joint significance testing, the presence of mediation (i.e., an indirect effect) in mediation analysis is based on the significance of component paths in the model (Fairchild & MacKinnon, 2009). By definition, mediation occurs if the indirect effect is nonzero; this requires that the mediator be significantly predicted by the independent variable and the outcome be significantly predicted by the mediator. If both of these coefficients are statistically significant, then mediation is said to have occurred. For example, if the effect of stress on negative cognitions is significant (notated as β_{NC1} in the above equations) and if the effect of negative cognitions on hopelessness is significant (notated as β_{H1} in the above equations), these two tests jointly imply the existence of a mediated effect. In other words, this shows that negative cognitions at least partly mediate the effect of stress on hopelessness. As compared to other tests of mediation, the joint significance test has been shown to produce the greatest power and the most accurate Type I error rates and has been recommended as a preferred test of mediation in simple models (Fritz & MacKinnon, 2007; MacKinnon et al., 2002) as well as in more complex models like the one presented here (Fairchild & MacKinnon, 2009).

RESULTS

Descriptive statistics are reported in Table 1; mean within-person simultaneous correlations and mean within-person lagged correlations are in Table 2. The full model results are reported in Table 3. Results showed that daily stressful events were not a significant predictor of negative cognitions on the following day ($\beta_{NC1} = -0.05$, $p = .75$). Results also showed that this relationship between daily stressful events and daily negative cognitions was not significantly moderated by cognitive vulnerability ($\gamma_1 = -.17$, $p = .44$).

Results also showed that after controlling for stressful events, daily negative cognitions significantly predicted hopelessness ($\beta_{H1} = .02$, $p < .001$). According to the joint significance test, there is a significant influence of negative cognitions on hopelessness, but there is not a significant mediated effect of stress on hopelessness.

Additionally, results indicated a significant relationship between hopelessness and depression on the following day ($\beta_{D1} = .27$, $p < .001$) after controlling for the effect of stressful events and negative cognitions. According to the joint significance test, then, this result, taken together with the significant effect of negative cognitions on hopelessness (β_{H1}), indicates a significant mediated effect of negative cognitions on depression.

DISCUSSION

The purpose of this study was to illustrate a data analytic strategy for testing sophisticated theories of clinical

Table 1. Means, standard deviations, and between-person correlations for variables at study onset

	1	2	3	4	5
Variable					
1. CSQ	—				
2. Depressive Sxs	**.25**	—			
3. Hopelessness	**.25**	**.54**	—		
4. Stressful Events	.11	.07	.10	—	
5. Event Inferences	**.46**	**.39**	**.35**	.09	—
M	3.91	17.62	10.36	.52	17.73
SD	.71	4.75	3.07	.65	6.90

Note. CSQ = Cognitive Style Questionnaire; Depressive Sxs = Daily Depressive Symptoms; Hopelessness = Hopelessness; Stressful Events = Daily Stressful Life Events; Event Inferences = Particular Inference Questionnaire (PIQ). For all measures, scores indicate greater levels of the construct being measured. Correlations in bold are significant at the .05 level.

Table 2. Mean within-person simultaneous correlations and mean within-person lagged correlations for daily variables

Variable	1	2	3	4
1. Hopelessness	1 (.24)	.16 (.05)	.56 (.16)	.12 (.02)
2. Event Inferences	.16 (.06)	1 (.10)	.21 (.08)	.06 (.00)
3. Depressive Sxs	.56 (.15)	.21 (.07)	1 (.21)	.14 (.03)
4. Stressful Events	.12	.06	.14	1

Note. Depressive Sxs = Daily Depressive Symptoms; Hopelessness = Hopelessness; Stressful Events = Daily Stressful Life Events; Event Inferences = Particular Inference Questionnaire (PIQ). The first number in each cell represents the mean unlagged within-person correlation between the variables. The number in the parentheses is the mean within-person lagged correlation, and the row indicates the lagged variable. For example, in row 2, column 1, the number in the parentheses represents the average within-person correlation of lagged Event Inferences with unlagged Hopelessness. Of note is this does not match the number in parentheses in row 1, column 2, as would be typical in a correlation table, because row 1, column 2 displays the correlation between lagged Hopelessness and unlagged Event Inferences. There are no lagged correlations for Stressful Events because this variable was not lagged in the analyses.

Table 3. Parameter estimates: Testing the one-day-lagged full within-person chain from stressful events to depressive symptoms

	Fixed Effect Estimate	Estimated Variance of Random Effect
β_{NC0} (intercept of negative cognitions)	17.91*** (0.22)	15.83***
β_{NC1} (stressful events → negative cognitions)	−0.05 (0.15)	0.00
γ_1 (moderation of general cognitive styles)	−0.17 (0.22)	–
β_{H0} (intercept of hopelessness)	9.98*** (0.17)	4.86***
β_{H1} (negative cognitions → hopelessness)	0.02*** (0.01)	0.001**
β_{H2} (stressful events → hopelessness)	−0.02 (0.05)	0.00
β_{D0} (intercept of depression)	17.24*** (0.25)	10.41***
β_{D1} (hopelessness → depression)	0.27*** (0.03)	0.03*
β_{D2} (negative cognitions → depression)	0.04*** (0.01)	0.00
β_{D3} (stressful events → depression)	−0.05 (0.09)	0.00

Note. Standard errors are shown in parentheses beneath their corresponding fixed effect. Detailed descriptions of the above subscripts can be found in the Data Analytic Strategy section. ***refers to $p < .001$, **refers to $p < .01$, and *refers to $p < .05$.

phenomena. To this end, we tested a prominent etiological theory of depression (HT; Abramson et al., 1989), which also maps closely onto the theories underlying cognitive interventions. Results of analyses provide strong support for the proposed MLM strategy

for testing theories that contain multiple mediating and moderating factors.

In our demonstration, we were able to test the entire sequence posited by the HT. Much of the model behaved as predicted. As hypothesized, increases in negative cognitions lead to increases in future feelings of hopelessness and, in turn, increases in future depressive symptoms. Importantly, results supported the mediating role of hopelessness in the causal chain. These results are the first to provide evidence for this specific causal path that provides at least one explanation for how negative cognitions eventually culminate in depressive symptoms.

Finding support for this specific pathway (using our MLM strategy) highlights the usefulness of a strong theory. It is not good enough to simply identify risk factors for psychopathology. For example, one of the most established findings in clinical research is that stressful life events confer risk for future depression. A naïve reinterpretation of this finding might state that if stressful life events constitute the risk, then eliminating these events should foster resilience. Unfortunately, it is not feasible to create an intervention that can eliminate all of life's stressors. And, even if stressful life events could be eliminated, it is unclear who should receive this intervention (e.g., not everyone who experiences a stressful life event develops psychopathology, and such stressors might even lead to long-term positive changes for some individuals; Park, Cohen, & Murch, 1996; Southwick, Vythilingam, & Charney, 2005). The point of this example is that risk factors cannot stand alone as "main" causes of psychopathology. Without more information about the relationship between stressful life events and psychopathology, it is difficult to know how to intervene. However, in light of the current findings, it is possible to identify multiple time points at which it might be possible to buffer an individual experiencing stressful life events from developing depression. To break the chain and prevent an individual from developing depression, one could target (a) negative inferences before hopelessness develops, (b) hopelessness, or (c) both negative inferences and hopelessness. Intervening at any of these positions in the chain should help to create resilience to depression.

Along these same lines, our MLM model has the potential to advance how clinical scientists think about

therapy outcome research. For example, according to the cognitive model of depression (Abramson et al., 1989), treatment intervention studies need to consider the moderating effects of stress when evaluating long-term efficacy (Haeffel, 2010). If this cognitive theory is correct, then the effectiveness of any intervention that reduces participants' cognitive vulnerability should only emerge in the presence of stressful events. In the absence of stress, those with (and without) a cognitive vulnerability should be resilient to depression. Thus, it is critical to test the statistical interaction of intervention and stress. Unfortunately, studies to date have only focused on the main effect of intervention (Murphy, Cooper, Hollon, & Fairburn, 2009), which may lead to diminished effect sizes and misleading findings. The current study provides a data analytic strategy for conducting a more theoretically accurate test of the many interventions that contain a vulnerability–stress hypothesis.

The present results also have implications for data analytic strategies more generally. To our knowledge, this is the first study to statistically model a theoretical sequence with two mediators and a moderator. We used MLM, which allowed us to nest daily within-person data for each individual (Level 1) in within-person-level data across individuals (Level 2). The use of MLM allowed for both levels of data to be analyzed simultaneously, allowing for investigation of both intra-individual and interindividual influences. By testing the chain from stressful events to depression at the within-person level, each individual is used, in essence, as his or her own control. In a traditional between-person analysis, we would, for example, analyze whether individuals higher in negative cognitions also tend to be higher in depressive symptoms. In the present analyses, however, we investigated whether on a day in which an individual has elevated negative cognitions for him or her (compared to his or her own average level of negative cognitions), "How might that lead to changes in future depressive symptoms within the *same* individual?" This is particularly advantageous because the chain is in fact theoretically expected to occur within individuals. It also allows for the exploration of whether daily within-person fluctuations (not just between-person differences) in negative cognitions are associated with a path to depressive symptoms (see

Abela & Hankin, 2008, for greater discussion). Moreover, using MLM, we were able to investigate whether a between-person factor (cognitive vulnerability) influenced that within-person relationship between negative events and depressive symptoms.

With regard to the hopelessness theory specifically, it is impressive that much of the chain behaved as theorized. However, there was one notable exception. Specifically, cognitive vulnerability and stressful events did not combine to predict future negative inferences. This finding belies a growing number of prospective longitudinal studies that support the vulnerability by stressful life event interaction (see Haeffel et al., 2008, for review). We suspect that this interaction was not supported in the current study for at least two reasons. First, the data set we used was not ideally suited for testing the entire hopelessness theory chain because of how life stress and event-specific inferences were measured. Each day, participants were instructed to choose the event that was most stressful to them. They then generated event-specific inferences for those events. This strategy was advantageous because it allowed us to easily link the occurrence of a stressful life event with participants' actual inferences about that event on a particular day. However, it is problematic for testing the effect of stressful life events on future event-specific inferences (i.e., conducting time-lagged analyses). This is because the inferences for a future date are likely in reference to an entirely different stressor. Thus, our lagged analyses did not represent a very accurate test of the effect of the cognitive vulnerability–stress interaction. However, we chose to conduct the time-lagged analyses in order to demonstrate the power of our MLM approach and also because it provided a valid test of the rest of the theoretical chain. Second, it is important to consider that the present analyses tested how *between-person* differences in cognitive vulnerability influence the *within-person* relationship between stressful events and negative inferences. Although this within-person approach likely provides a more accurate test of the hopelessness theory, it strays from the typical statistical approach used in prior studies, which is to only test between-person differences. Thus, it will be important for future research to determine whether or not this change in statistical approach affects researchers' ability to detect the vulnerability by stress interaction.

Although the current study provided strong evidence of the feasibility of the MLM technique for testing complex theories, it is important to note limitations. First, our goal was to advance data analytic strategies for testing theories of etiology and psychotherapy, but we only illustrated our technique using data from an etiological study. We chose this data set because we were unaware of any existing intervention studies (or even other etiological tests) that have measured all of the relevant mediators and moderators from a complex model. Thus, this data set offered the closest approximation to an ideal intervention study that would assess multiple mediating and moderating factors over multiple time points. A second limitation was the use of abbreviated measures of depressive symptoms and hopelessness. These modified scales were necessary because of the daily diary format (e.g., they were needed to reduce burden on participants who were filling out measures every day). However, future work using more established measures is needed to corroborate the current findings. Finally, our study used a relatively homogenous college sample, and thus, the results for the hopelessness theory may not generalize to more diverse samples.

In conclusion, many elegant and theoretically sophisticated models in clinical science have not received careful empirical scrutiny due to a lack of appropriate data analytic strategies. This study provides one way for psychological scientists to properly test their full conceptual models, instead of merely slices in isolation of each other. The current study also highlights the need for more specific theories of psychopathology and psychotherapy. Creating testable and well-defined etiological chains can be a cornerstone for prevention and treatment of psychopathology. Strong theories of psychopathology can specify how to identify at-risk populations, the time points for interventions, and strategies for promoting resilience.

NOTES

1. MLM only includes people who have data on all variables on a given day. In the present study, approximately 12% of the sample did not complete their diaries on a given day. This means that, on any given day, an average of 12% of participants were not included in the full analyses. The strength of this is approach is that it maximizes the data; for example, participants are not automatically excluded if they happened to neglect one day's surveys. In addition, because the current model focused on processes within a person within a day (i.e., how does the chain operate within a person within a day), even if a person only had one day of complete data, it makes sense to include that one day because it still provides additional information about the process within a day within that person. An additional advantage of MLM is that while it allows such a person's data to be included in the analysis, the data for such a person are effectively given less weight than the data for someone else with multiple days of data.

2. As described, the full within-person mediational chain between daily negative events and daily depressive symptoms was tested simultaneously. As such, there were two mediations that were investigated at the same time. The first is the mediation of daily negative cognitions on the relationship between daily negative events and daily hopelessness. The second is the mediation of hopelessness on the relationship between daily negative cognitions and daily depressive symptoms.

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APPENDIX

```
/****************  Syntax for Lagged Mediation Analysis  ********************/


/*Variable Names: ID: each individual's identification
                  cNegCog: within-person centered daily negative cognitions
                  cHS: within-person centered daily hopelessness
                  cDep: within-person centered depression symptoms
                  cNegEv: within-person centered daily negative events
                  cCSQN: between-person centered cognitive style
note: Variables that do not begin with 'c' are uncentered versions*/


/*********************** Set up data for Analysis ************************/


/*Lag appropriate variables using 'lag1' SAS function */

/*Create lags for daily negative cognition*/
data lagA; set dailyData;
      IDlag1 = lag1(id);
      cNegCoglag1 = lag1(cNegCog);

      if id=IDlag1 then cNegCoglag1= cNegCoglag1; else cNegCoglag1= .;
      Label cNegCoglag1= "within-Person Neg Cog Lag=1";

      IDlag1 = lag1(id);
      NegCoglag= lag1(NegCog);

      if id=IDlag1 then NegCoglag1= NegCoglag1; else NegCoglag1= .;
      Label NegCoglag1= "uncentered Neg Cog Lag=1";
run;

/*Create lags for daily hopelessness*/
data lagB; set lagA;
      IDlag1 = lag1(id);
      cHSlag1 = lag1(cHS);

      If id=IDlag1 then cHSlag1 = cHSlag1; else cHSlag1 = .;
      Label cHSlag1 = "within-Person HS Lag=1";

      IDlag1 = lag1(id); HSlag1 = lag1(HS);

      if id=IDlag1 then HSlag1 = HSlag1; else HSlag1 = .;
      Label HSlag1 = "uncentered HS Lag=1";
run;

/*Create lags for daily depressive symptoms*/
data lagC; set lagB;
      IDlag1 = lag1(id);
      cDeplag1 = lag1(cDep);

      if id=IDlag1 then cDeplag1 = cDeplag1; else cDeplag1 = .;
```

```
        Label cDeplag1 = "within-Person Depression Lag=1";

        IDlag1 = lag1(id); deplag1 = lag1(Dep);

        if id=IDlag1 then Deplag1 = Deplag1; else Deplag1 = .;
        Label Deplag1 = "uncentered depression lag=1";
run;

/*Create dummy codes to test the full model simultaneously*/
data totlagC; set lagC;
        Z=Deplag1; *this coding allows us to identify depression
                    as the outcome when sd3=1;
        Sd1=0;
        Sd2=0;
        sd3=1;
        dv='DEPlag';
output;
        Z = HSlag1; *this coding allows us to identify
                        hopelessness as the outcome when sd2=1;

        Sd1 = 0;
        Sd2 = 1;
        Sd3=0;
        dv = 'HSlag';
output;
        Z = NegCoglag1; *this coding allows us to identify daily
                            negative cognitions as the outcome when sd1=1;
        Sd1 = 1;
        Sd2 = 0;
        Sd3=0;
        dv = 'Negcoglag';
output;
run;


/************** Analyze Full Model Using Lagged Dataset ****************/

proc mixed data=totlagC asycov covtest noclprint method=reml maxiter=200;
        class dv id;
        model Z = Sd1 Sd1*cNegev sd1*cCSQN sd1* cCSQN *cNegEv sd2 sd2*cNegCog
                sd2*cNegEv sd3 sd3*cHS sd3*cNegCog sd3*cNegEv /noint solution
        ddfm=kr covb;
        random Sd1 Sd2 Sd3 Sd1*cNegEv Sd2*cNegCog sd2*cNegEv sd3*cHS
              sd3*cNegCog sd3*cNegEv /g gcorr type=csh subject=id;
        repeated / group=dv;
        ods output covb=acovfix asycov=acovrand solutionf=estfix
                    covparms=estrand convergencestatus=converge;
run;
```