DAILY COGNITIVE REACTIVITY AS A PREDICTOR OF LATER DEPRESSIVE SYMPTOMS

by

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Using a daily diary methodology, we assessed whether daily cognitive reactivity—activation of dysfunctional attitudes by negative affect—predicts later depressive symptoms and, conversely, whether depressive symptoms predict later cognitive reactivity. At two time points, 10 weeks apart, 161 undergraduates completed a measure of depressive symptoms and daily surveys for seven days that assessed daily negative affect, daily dysfunctional attitudes, and daily negative events. Using multilevel modeling, we computed participants' daily negative affect-dysfunctional attitudes reactivity slopes, which were used as predictors of later depressive symptoms. Our results showed that Time 1 daily cognitive reactivity was a significant, negative predictor of Time 2 depressive symptoms, controlling for Time 1 depressive symptoms, and that Time 1 depressive symptoms were not a significant predictor of Time 2 daily cognitive reactivity. The negative relationship between Time 1 cognitive reactivity and Time 2 depressive symptoms is in contrast to the positive relationship reported in most previous studies. However, our study was unique in that we assessed daily cognitive reactivity occurring naturally in participants' everyday experiences. Further examination of our data revealed that the Time 1 positive relationship between negative affect and dysfunctional attitudes was moderated by Time 1 depressive symptoms, such that those higher in depressive

symptoms demonstrated weaker daily cognitive reactivity. Based on the emotional context insensitivity literature, we then assessed the possibility that daily stressors did not produce a strong negative emotional reaction for participants with higher depressive symptoms. Specifically, we examined participants' daily affective reactivity—increases in negative affect in response to negative events—and found that initial depressive symptoms moderated the positive relationship between negative events and negative affect, with those higher in depressive symptoms demonstrating weaker daily affective reactivity. Overall, we suggest that participants with higher depressive symptoms experienced smaller increases in naturally-occurring negative affect in response to daily stressors, producing flatter slopes for both affective and cognitive reactivity. Our findings highlight the need to integrate the research literatures on daily cognitive reactivity and daily affective reactivity.

Chapter 1

INTRODUCTION

Beck (1967) proposed that cognitive processes underlie the development, maintenance, and recurrence of depression. Advocates of cognitive models of depression contend that cognitive factors are the primary causal agents of depression (Ingram, Miranda, & Segal, 1998). Specifically, depression-vulnerable individuals are said to differ from non-vulnerable individuals in that the former possess maladaptive cognitive schemas characterized by rigid, pessimistic, and dysfunctional cognitions, focused on negative perspectives about oneself, the world, and the future (Scher, Ingram, & Segal, 2005).

Researchers generally regard cognitive vulnerability as a stable but latent individual difference (Ingram et al., 1998). Beck's original (1967) theory proposed that negative, dysfunctional cognitions remain dormant until activated by stress. Thus, maladaptive cognitive schemas are necessary but not sufficient for the development of depression (Scher et al., 2005). Cognitive theorists suggest that, although vulnerable individuals may be indistinguishable from non-vulnerable individuals under neutral conditions, stressful events activate dormant negative schemas in vulnerable individuals, resulting in depression (Hammen, Bistricky, & Ingram, 2010).

The idea that cognitive vulnerability must be activated to be observable has had important implications for research in this area. In order to substantiate the causal

role of cognitive variables articulated in the cognitive model, such as dysfunctional attitudes, researchers must demonstrate that cognitive vulnerability persists even after depression remits (Scher et al., 2005). Initially, many researchers were unable to detect cognitive differences between formerly depressed and never depressed individuals, which led to the conclusion that such cognitive factors might represent correlates or consequences of depression rather than causes (Scher et al., 2005). It was not until researchers began to directly activate latent depressive schemas that such differences could be more readily observed.

The notion that internal states, such as an individual's mood, might influence cognition led to the mood-state dependent hypothesis (Miranda & Persons, 1988). This hypothesis holds that "cognitive vulnerability factors for depression are indeed present in individuals who are at risk for depression, but are simply inaccessible until they have been activated by negative mood" (Miranda & Gross, 1997, p. 589). The mood-state hypothesis may therefore explain why early findings did not show that vulnerable and non-vulnerable individuals differed on dysfunctional attitudes. This hypothesis suggests that vulnerable individuals do indeed have more dysfunctional attitudes than non-vulnerable individuals, but such differences are only observable when participants are in a negative mood state (Miranda, Persons, & Byers, 1990).

1.1 Prior Research on Cognitive Reactivity

Activation of maladaptive, depressive cognitions by negative mood is commonly referred to as cognitive reactivity. Researchers typically operationalize cognitive reactivity as self-reported change in dysfunctional attitudes, typically

assessed with the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), in response to self-reported increases in negative affect, either manipulated in a lab or occurring naturally. Research that followed Miranda and Persons' (1988) mood-state dependent hypothesis, and Teasdale's (1983; 1988) similar concept of differential activation, provided clearer evidence that those vulnerable to depression exhibit cognitive reactivity. For example, Beevers and Miller (2004) followed depressed participants from psychiatric hospitalization, to outpatient treatment, to a one-year follow-up, assessing dysfunctional attitudes and cognitive bias. Their findings suggested that depressed individuals have stable negative cognitions, but their accessibility to such cognitions fluctuates as a function of mood state. In another study, Wenze, Gunthert, and Forand (2007) found that individuals higher in depressive symptoms exhibited a stronger association between daily negative mood and thinking than individuals low in depressive symptoms.

In a review of the literature, Scher et al. (2005) noted that two methodologies characterize the majority of studies on cognitive reactivity. First, when comparing depression-vulnerable and non-depression-vulnerable individuals, researchers often compare those with a history of depression (currently in remission) to those who have no history of depression. The rationale for this approach is that formerly depressed individuals are at high risk for experiencing a future depressive episode and therefore likely possess latent cognitive vulnerability factors. The use of depression history as a proxy for vulnerability assumes that those who have already had depression have a cognitive vulnerability factor that, unless treated with cognitive therapy, is likely to

still be present (e.g., Miranda, Gross, Persons, & Hahn, 1998). Correlational studies provided initial support for cognitive reactivity as a vulnerability factor for depression by demonstrating a link between greater negative affect and greater dysfunctional attitudes among formerly depressed participants but not among never-depressed participants (e.g., Miranda & Persons, 1988; Miranda et al., 1990). However, a disadvantage of this type of research is that it does not address those unidentified individuals who are vulnerable to, and may go on to develop, depression. Some researchers have opted to use an older sample considering that older individuals have had more time to express their vulnerability to a depressive episode, if one exists for them, thereby presumably decreasing the possibility that vulnerable individuals would be misclassified as non-vulnerable (e.g., Miranda et al., 1998).

Scher et al. (2005) also noted that researchers studying cognitive reactivity usually employ negative mood priming procedures. Specifically, researchers employ in-lab techniques to manipulate participants' mood, measuring cognitions before and after the mood change. The rationale for this approach is to create an affective state that might be elicited by stressful life events in the real world and that might therefore lead to the occurrence of a depressive episode. In one study, Miranda et al. (1998) primed individuals with and without a history of depression with a negative mood induction and then examined their dysfunctional attitudes. Participants in each group did not differ on dysfunctional attitudes before the mood induction, but following the induction, those participants with a history of depression tended to score higher on dysfunctional attitudes than those without such a history. Similarly, Gemar, Segal,

Sagrati, and Kennedy (2001) found that formerly depressed individuals showed an increase in dysfunctional attitudes and a more negative self-evaluative bias after a sad mood induction, relative to controls. Although mood primes provide the opportunity to directly activate and measure dysfunctional cognitions, a major disadvantage of this methodology is that it does not examine individuals' reactions to everyday events in their lives or their experience of and reaction to naturally-occurring mood.

The research reviewed thus far has been largely cross-sectional. However, prospective designs are required to support the role of cognitive reactivity as a causal factor in depression (Scher et al., 2005). There has been some research in this area linking cognitive reactivity to later episodes of depression, particularly depression recurrence. In a review of mood-induced cognitive reactivity, Lau, Segal, and Williams (2004) indicated that research has provided ample evidence for the causal role of cognitive reactivity in risk for depressive recurrence. For example, in an often cited study, Segal, Gemar, and Williams (1999) examined cognitive reactivity as a predictor of depression recurrence among a sample of patients remitted through either cognitive therapy or pharmacotherapy. Segal et al. measured reports of dysfunctional attitudes before and after a sad mood induction and found that greater cognitive reactivity was predictive of relapse approximately 2 ½ years later. This relationship held across both treatment groups and after accounting for dysfunctional attitudes under normal mood. In a similar study, Segal et al. (2006) assessed the endorsement of dysfunctional attitudes following a sad mood induction among patients they had treated to remission with either cognitive behavior therapy or antidepressant

medication. They found that, regardless of treatment type, cognitive reactivity was a significant predictor of relapse over the following 18 months, even after controlling for patients' number of past depressive episodes, a robust predictor of recurrence. Further, greater cognitive reactivity was also associated with shorter time to relapse.

The benefit of recurrence studies is that they can provide evidence for a causal role of cognitive reactivity, at least for depression recurrence. However, Just, Abramson, and Alloy (2000) suggest that recurrence studies are systematically flawed. For instance, they utilize a backward participant selection strategy in which researchers select participants on the basis of the presumed dependent variable (depression) and compare them on the presumed independent variable (cognitive vulnerability). Further, Just et al. argue that such designs fail to disambiguate whether maladaptive cognitive styles are a cause or a consequence of depression. Thus, although recurrence studies provide evidence for cognitive reactivity as a causal factor in depression recurrence, they do not provide information about cognitive reactivity's role in the onset of depression. Because most studies to date assessed relapse rather than first onset, conclusions about causality in general are limited, particularly given some speculation that there are differences in the mechanisms that lead to an initial onset versus a recurrence of depression (Hammen et al., 2010).

Taken together, research in this area suggests that depression-vulnerable individuals exhibit cognitive reactivity, and that such cognitive reactivity may play a causal role in the occurrence of depressive symptoms. However, a tendency to rely on

mood manipulations, cross-sectional designs, or recurrence studies limits the generalizability of these findings.

1.2 The Present Study

In the present study, we chose a different approach from the most common strategies in this area. We used a prospective daily diary methodology to assess the relationship between daily cognitive reactivity and later depressive symptoms. For two one-week assessment periods, separated by 10 weeks, participants completed a measure of depressive symptoms and seven days of daily surveys that assessed their daily negative affect, daily dysfunctional attitudes, and daily negative events. We assessed whether Time 1 daily cognitive reactivity—the relationship between negative affect and dysfunctional attitudes—predicted depressive symptoms at Time 2, 10 weeks later.

The present study differs from the majority of studies in this area in at least two major ways. First, we examined an unidentified sample of individuals and used their initial cognitive reactivity scores to prospectively predict depressive symptoms 10 weeks later. We felt that identifying individuals at risk for depressive symptoms on the basis of cognitive reactivity alone would be an important addition to the literature. We also used a daily diary methodology to assess participants' cognitive reactivity in response to naturally-occurring changes in negative affect. Daily examination of cognitive reactivity affords some advantage over traditional laboratory studies. Daily diaries permit study of individuals' regular, day-to-day experiences (Bolger, Davis, & Rafaeli, 2003), which have been largely overlooked in the cognitive reactivity

literature thus far. To our knowledge, only one other study to date has examined the relationship between daily cognitive reactivity and later depressive symptoms. Wenze, Gunthert, and Forand (2010) found that greater daily cognitive reactivity was associated with greater depressive symptoms six months later. Wenze et al. (2010) examined the relation between daily occurrences of negative affect and endorsement of daily automatic thoughts. In our study, however, we assessed cognitive reactivity using the more traditional measure of dysfunctional attitudes.

Based on previous research on cognitive reactivity (e.g., Gemar et al., 2001; Miranda et al., 1998; Segal et al., 2006), it seemed appropriate to expect that daily cognitive reactivity would be predictive of later depressive symptoms in our study. However, because previous work has relied primarily on mood manipulations, we considered the possibility that daily examination of these processes might reveal a different pattern of results. In particular, we considered whether daily experiences would elicit the same affective responses in participants that lab-based procedures have successfully done. Detection of cognitive reactivity in everyday life relies on increases in negative affect presumed to be necessary to activate dysfunctional attitudes. Our study therefore relies on the assumption that daily experiences will exert detectable changes in vulnerable individuals' negative affective states. This set of assumptions highlights a necessary and important marriage between the cognitive reactivity literature and the affective reactivity literature.

1.2.1 Affective Reactivity

Affective reactivity refers to changes in mood in response to stressful experiences. There have been mixed findings on affective reactivity among individuals vulnerable to depression. Some daily studies have demonstrated a positive association between negative affective reactivity and later depressive symptoms (e.g., Cohen, Gunthert, Butler, O'Neill, & Tolpin, 2004; Parrish, Cohen, & Laurenceau, 2011). In contrast, other researchers have provided evidence for diminished affective reactivity among depression vulnerable individuals (e.g., Rottenberg, Gross, & Gotlib, 2005).

Based on this second line of research demonstrating diminished affective reactivity, investigators have advanced the emotion context insensitivity hypothesis (Rottenberg, 2005). Emotion context insensitivity refers to reduced affective reactivity to both positive and negative emotional cues among depressed individuals. Researchers have provided evidence in support of valence-independent deficits in emotional reactivity in depression. Bylsma, Morris, and Rottenberg (2008) conducted a meta-analysis of studies comparing affective reactivity of depressed and healthy participants and found support for the emotional context insensitivity hypothesis, such that depressed individuals generally exhibited reduced affective reactivity to cues, regardless of valence. Emotion context insensitivity is consistent with the notion of positive attenuation—attenuated emotional reactivity to positive emotional stimuli—but inconsistent with the notion of negative potentiation—increased emotional reactivity to negative emotional stimuli (Rottenberg et al., 2005). Positive attenuation

is in many ways consistent with the defining features of depression, such as anhedonia (Rottenberg et al., 2005). Disconfirmation of negative potentiation may seem less intuitive. Although it might seem likely that depressed individuals would demonstrate greater negative affective reactivity, a large body of evidence suggests just the opposite—reduced negative affective reactivity among depressed individuals (Bylsma et al., 2008).

In a daily examination of this theory, Peeters, Berkhof, Rottenberg, and Nicolson (2010) examined whether affective reactivity to daily events predicted outcomes for individuals receiving treatment for major depressive disorder. Their findings were consistent with the emotional context insensitivity model, such that less affective reactivity to daily events was associated with higher depression severity, and those with less negative affective reactivity were less likely to recover from depression. The authors concluded that unresponsiveness to the environment may contribute to chronic depression by hindering positive engagement and coping with life events. This finding is particularly relevant to the present study, as it suggests that affective reactivity in response to daily events may be attenuated among participants vulnerable to depression.

Although researchers have primarily examined emotional context insensitivity among individuals with clinical depression, attenuated emotional reactivity may apply more generally to individuals high in depressive symptoms. Ellis, Beevers, and Wells (2009) provided evidence for this extension by examining affective reactivity to positive and negative feedback among a sample of college students assessed as either

relatively low or high in depressive symptoms. In accordance with the emotional context insensitivity literature, they found that those with greater depressive symptoms displayed attenuated reactivity compared to controls. For those high in depressive symptoms, emotional responses did not differ in response to positive and negative feedback, whereas the control group exhibited differentiated responses across contexts.

1.2.2 Hypotheses

Based on previous research on cognitive reactivity, we hypothesized that cognitive reactivity would serve as a vulnerability factor for later depressive symptoms. We predicted that Time 1 daily cognitive reactivity would be positively associated with Time 2 depressive symptoms, controlling for Time 1 depressive symptoms. For exploratory purposes, we also examined whether this relationship was bi-directional, such that Time 1 depressive symptoms predicted Time 2 cognitive reactivity, controlling for Time 1 cognitive reactivity. However, because our study is one of the first to examine cognitive reactivity as a function of naturally- occurring negative affect from daily stressors, and based on the emotion context insensitivity literature, we considered the possibility that daily stressors might not be successful at eliciting negative affect, and therefore dysfunctional attitudes, among those high in depressive symptoms. Thus, our study may provide further clarification about the nature of daily cognitive and affective reactivity.

Chapter 2

METHODS

2.1 Participants

We recruited college students from an introductory psychology course to participate in an "electronic diary study of college students' daily events, mood, and attitudes." Students received extra credit in their course for completing either the study or an alternative course-related assignment of comparable time commitment. One hundred ninety-four students initially consented to participate. The 161 participants who completed the cross-sectional surveys and at least four evening surveys at both Time 1 and Time 2 were included in subsequent analyses. Time 1 depressive symptom scores were not significantly different for those participants who began but did not complete the study compared to those who completed the study. Participants in the final sample were 62.1% female. Ninety-nine percent of the participants were between the ages of 18 and 22 years of age (M = 19.43 years, SD = 1.26 years). The racial background of participants was 47.8% White, 44.1% Asian, 4.3% Black or African American, 0.6% American Indian or Alaskan native, 1.2% more than one race, and 1.9% other, and their ethnic background was 2.5% Hispanic.

2.2 Procedure

The study was approved by the Institutional Review Board at the authors' university. Interested students attended a group information session at which they

provided informed consent and received instruction on how to complete the study measures. Following this meeting, participants completed the Time 1 initial online survey, followed by a series of online surveys to be completed in the morning and evening for seven consecutive days. Participants were instructed to complete the morning surveys when they first awoke and no later than 11 a.m., and to complete the evening surveys between 8 p.m. and 2 a.m. This process of completing an initial survey followed by seven days of daily surveys was repeated 10 weeks later at Time 2. Each morning and evening, participants received a reminder email containing a link to the online survey. Participants who failed to complete a survey were sent a personal message encouraging them to continue to participate in the study. Participants were debriefed at the end of the study via email.

2.3 Initial Measure of Depressive Symptoms

At the beginning of both Time 1 and Time 2, participants completed online cross-sectional surveys that included the Center for Epidemiological Studies—Depression Scale (CES-D; Radloff, 1977). The CES-D is a 20-item scale used to assess depressive symptoms in community samples. Participants were asked to indicate the frequency with which they experienced each item during the past week, for example, "I felt that I could not shake off the blues even with help from my family or friends," "I felt that everything I did was an effort," and "I felt hopeful about the future" (reverse-scored). Participants rated each item using a 5-point scale ranging from *rarely or none of the time (less than 1 day)* to *most or all of the time (5-7 days).* The CES-D has demonstrated excellent reliability and validity (e.g., Turk & Okifuji,

1994). In the present study, Cronbach's alphas for the CES-D at Time 1 and Time 2 were .91 and .90, respectively.

2.4 Daily Measures

After completing the initial survey, participants completed an online daily survey each morning and evening for seven consecutive days. In the morning, participants reported on their current negative affect and dysfunctional attitudes. In the evening, they again reported on their negative affect and dysfunctional attitudes, as well as negative events that occurred that day.

2.4.1 Negative Affect

We assessed state negative affect each morning and evening with the Positive and Negative Affect Schedule—Expanded Form (PANAS-X; Watson & Clark, 1994). We combined the 10-item Negative Affect scale (e.g., "scared," "upset," "ashamed") and the 5-item Sadness scale (e.g., "sad," "blue," "downhearted") to compute an aggregate negative affect score (using participants' mean item rating). Participants rated how much they felt each emotion "right now, at this moment," using a 5-point scale where 1 indicated *very slightly/not at all* and 5 indicated *extremely.* Research has supported the reliability and validity of the PANAS-X scales (Watson & Clark, 1994). For each of the daily measures, we estimated within-person reliability using methods outlined by Cranford et al. (2006). For our 15-item aggregate negative affect scale, morning and evening within-person reliability was .90 and .88, respectively, at Time 1. At Time 2, within-person reliability was .87 for both morning and evening.

2.4.2 Dysfunctional Attitudes

Each evening, participants completed the Dysfunctional Attitudes Scale (DAS, Form A; Weissman & Beck, 1978). The DAS is a 40-item scale that measures maladaptive (e.g., rigid, perfectionistic, evaluation-focused) beliefs. Participants were asked to indicate the degree to which they agreed or disagreed with each statement, for example, "If I fail partly, it is as bad as being a complete failure," and "My value as a person depends greatly on what others think of me." Each item was rated using a 7-point scale ranging from *totally agree* to *totally disagree*. The DAS has demonstrated good reliability and validity (Weissman & Beck, 1978; Weissman, 1980). Each morning, participants completed an abbreviated version of this scale, the Dysfunctional Attitudes Scale—Short Form 2 (DAS-SF 2; Beevers, Strong, Meyer, Pilkonis, & Miller, 2007). The short-form includes nine of the same items from the 40-item version. The short form has demonstrated good reliability and validity (Beevers et al., 2007). In the present study, within-person reliability for the DAS was .79 at Time 1 and .75 at Time 2. Within-person reliability for the DAS-SF was .64 at Time 1 and .55 at Time 2.

2.4.3 Daily Negative Events

The evening survey included a checklist of 19 common daily stressors adapted from a college student diary study by Dasch, Cohen, Sahl, and Gunthert (2008; e.g., "Had an argument or got along poorly with a friend, family member, or romantic partner," and "Took an exam today and I think I did poorly"). Participants indicated

(*yes/no*) whether each event occurred that day. We calculated the total number of negative events reported each day.

Chapter 3

RESULTS

3.1 Descriptive Analyses

The means and standard deviations of the major study variables are listed in Table 3.1.

Table 3.1: Means and Standard Deviations for Study Variables

Measure	*M*	*SD*
Time 1		
Depressive Symptoms[a]	1.78	0.53
Number of Daily Negative Events	1.92	1.55
Morning Negative Affect[a]	1.51	0.66
Evening Negative Affect[a]	1.60	0.69
Morning Dysfunctional Attitudes[a]	2.21	0.55
Evening Dysfunctional Attitudes[a]	3.54	0.83
Evening Negative Affect/Evening Dysfunctional Attitudes Slope	0.11	0.20
Negative Event/ Evening Negative Affect Slope	0.08	0.05
Time 2		
Depressive Symptoms[a]	1.78	0.49
Number of Daily Negative Events	1.68	1.44
Morning Negative Affect[a]	1.59	0.69
Evening Negative Affect[a]	1.66	0.71
Morning Dysfunctional Attitudes[a]	2.31	0.51
Evening Dysfunctional Attitudes[a]	3.67	0.75
Evening Negative Affect/Evening Dysfunctional Attitudes Slope	0.12	0.17
Negative Event/Evening Negative Affect Slope	0.09	0.04

[a]These variables reflect mean item scores

The mean number of negative events reported each day was consistent with descriptive data reported in previous daily diary studies of college students (e.g., Dasch et al., 2008; LoSavio et al., 2011). Our mean depressive symptom scores were also consistent with similar studies of college students that used the CES-D (e.g., Parrish et al., 2011; Wenze et al., 2010). Negative and sad affect scores were similar to those reported by Watson and Clark (1994) and by researchers who conducted daily surveys with college students (e.g., LoSavio et al., 2011; Parrish et al., 2011). To our knowledge, no study has previously examined dysfunctional attitudes on a daily level; however, our overall means on the DAS were consistent with scores obtained from single-administrations of this scale (e.g., Wenze et al., 2010).

At Time 1, the average number of completed morning and evening surveys was 6.40 (*SD* = 0.99) and 6.49 (*SD* = 0.88), respectively. At Time 1, the average number of days with *both* morning and evening surveys completed was 6.05 (*SD* = 1.28). At Time 2, the average number of completed morning and evening surveys was 6.37 *(SD* = 1.00) and 6.44 (SD = 0.86), respectively. At Time 2, the average number of days with both morning and evening surveys completed was 5.94 (*SD* = 1.38). For the morning surveys, the average time of completion was 9:27 a.m. (*SD* = 78 minutes). For the evening surveys, the average time of completion was 9:54 p.m. (*SD* = 105 minutes).

3.2 Correlations

The Pearson's correlations among the major study variables are presented in Table 3.2.

Table 3.2: Pearson's Correlations for Major Study Variables

	1	2	3	4	5	6
1. Time 1 Depressive Symptoms	--	--	--	--	--	--
2. Time 1 Mean Evening Negative Affect	.461***	--	--	--	--	--
3. Time 1 Mean Evening Dysfunctional Attitudes	.414***	.424***	--	--	--	--
4. Time 2 Depressive Symptoms	.544***	.571***	.516***	--	--	--
5. Time 2 Mean Evening Negative Affect	.301***	.754***	.435***	.575***	--	--
6. Time 2 Mean Evening Dysfunctional Attitudes	.295***	.371***	.846***	.439***	.431***	--

***$p < .001$.

Our correlations showed that depressive symptoms, mean evening negative affect, and mean evening dysfunctional attitudes were each significantly correlated with the others in the expected, positive direction within and across time points. We also calculated the within-person correlations among the daily variables using procedures outlined by Snijders and Boskers (1999). These correlations represent the relationships between the within-person deviations of each variable (see Table 3.3).

Table 3.3: Within-Person Correlations for Major Study Variables

Time 1	1	2	3
1. Evening Negative Affect	--	--	--
2. Evening Dysfunctional Attitudes	.06	--	--
3. Number of Daily Negative Events	.18	.04	--

Table 3.3 continued

Time 2	1	2	3
1. Evening Negative Affect	--	--	--
2. Evening Dysfunctional Attitudes	.10	--	--
3. Number of Daily Negative Events	.22	.11	--

Note. These correlations represent the relationships between the within-person deviations of each variable. Significance levels are not applicable and thus are not reported.

3.3 Cognitive (DAS) Reactivity

We estimated each person's unique reactivity slope using Hierarchical Linear Modeling (HLM; Raudenbush & Bryk, 2002), which employs maximum likelihood estimation. HLM is accommodating of missing data and particularly useful for analyzing data with a nested structure (i.e., daily assessments within individuals). We first examined daily cognitive reactivity. Because research has suggested that cognitive vulnerability is mood-state dependent (e.g., Miranda & Persons, 1988), we assessed concurrent affect and attitudes. To implicate negative affect as the predictor of attitudes (and not vice versa), we assessed cognitive reactivity by measuring change in dysfunctional attitudes from morning to evening as a result of negative affect. To do so, we examined evening negative affect as a predictor of evening dysfunctional attitudes, controlling for morning dysfunctional attitudes. We estimated the following model in HLM:

$$\textit{Evening Dysfunctional Attitudes}_{ij} = \gamma_{00} + \gamma_{10}(\textit{Evening Negative Affect}_{ij}) + \gamma_{20}(\textit{Morning Dysfunctional Attitudes}_{ij}) + u_{0j} + u_{1j}(\textit{Evening Negative Affect}_{ij}) + e_{ij}$$

In this equation, *Evening Dysfunctional Attitudes*$_{ij}$ represents Participant *j*'s dysfunctional attitudes on Day *i*. We group-mean centered the predictor variables, so the intercept, γ_{00}, represents Participant *j*'s level of evening dysfunctional attitudes at his or her average levels of evening negative affect and morning dysfunctional attitudes. The slope of evening negative affect, γ_{10}, represents the change in Participant *j*'s evening dysfunctional attitudes from morning to evening for every one unit increase in his or her evening negative affect. Thus, each participant has his or her own slope coefficient that represents the relationship between negative affect and change in dysfunctional attitudes. The model intercept, as well as the slope for evening negative affect, were allowed to randomly vary. The term u_{0j} represents the between-person deviations in the model intercept. The term u_{1j} represents the between-person deviations in the slope for evening negative affect.

Results of this model indicated that, at Time 1, evening negative affect served as a significant, positive predictor of change in dysfunctional attitudes, $\gamma_{10} = .108$, $p = .022$. We repeated this analysis at Time 2 and replicated the Time 1 pattern of results. At Time 2, evening negative affect served as a significant, positive predictor of change in dysfunctional attitudes, $\gamma_{10} = 0.122$, $p = .002$.[1]

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[1] We also ran our HLM model with an additional control for morning negative affect. We found that the Time 1 and Time 2 daily cognitive reactivity results were comparable even when controlling for both morning negative affect and morning dysfunctional attitudes.

We used the empirical Bayes residuals from this model to calculate a cognitive reactivity slope variable for each participant at Time 1 and Time 2, respectively. We then used the Time 1 between-persons cognitive reactivity variable to prospectively predict depressive symptoms 10 weeks later. Specifically, we performed a hierarchical multiple regression to test whether cognitive reactivity at Time 1 predicted depressive symptoms at Time 2, controlling for Time 1 depressive symptoms. Time 1 cognitive reactivity was a significant predictor of Time 2 depressive symptoms, but in the negative direction, $B = -0.537$, $p = .001$ (see Table 3.4).

Table 3.4: Hierarchical Multiple Regression Analyses

Predictor	β	*B*	*SE* for *B*	95% CI for *B*	R^2	*F*	ΔR^2	ΔF
Time 1 Cognitive Reactivity as a Predictor of Time 2 Depressive Symptoms								
Step 1: Control Time 1 Depressive Symptoms	0.54***	0.50***	0.06	[0.38, 0.62]	.30	66.79***		
Step 2: Time 1 Cognitive Reactivity	-0.22**	-.0.54**	0.16	[-0.86, -0.22]	.34	40.92***	.05	10.89**
Time 1 Depressive Symptoms as a Predictor of Time 2 Cognitive Reactivity								
Step 1: Control Time 1 Cognitive Reactivity	0.27***	0.23***	0.06	[0.10, 0.36]	.08	12.84***		
Step 2: Time 1 Depressive Symptoms	-0.04	-0.01	0.03	[-0.06, 0.04]	.08	6.49**	.001	0.20

**$p < .01$. ***$p < .001$.

We also tested whether the relationship between cognitive reactivity and depressive symptoms was bi-directional, with Time 1 depressive symptoms predicting Time 2 cognitive reactivity, controlling for Time 1 cognitive reactivity. Time 1 depressive symptoms did not significantly predict Time 2 cognitive reactivity, B = -0.011, p = .658 (see Table 3.4).[2,3]

3.3.1 Moderating Role of Time 1 Depressive Symptoms

After attaining the unexpected negative association between Time 1 cognitive reactivity and Time 2 depressive symptoms, we re-examined our Time 1 HLM model. Given the literature on diminished reactivity among depressed individuals (Bylsma et al., 2008), we explored our Time 1 daily cognitive reactivity model with Time 1 depressive symptoms as a moderator. We estimated the following model in HLM:

$$\textit{Evening Dysfunctional Attitudes}_{ij} = \gamma_{00} + \gamma_{01}(\textit{Depressive Symptoms}_j) + \gamma_{10}(\textit{Morning Dysfunctional Attitudes}_{ij}) + \gamma_{20}(\textit{Evening Negative Affect}_{ij}) + \gamma_{21}(\textit{Depressive Symptoms}_j \times \textit{Evening Negative Affect}_{ij}) + u_{0j} + u_{2j}(\textit{Evening Negative Affect}_{ij}) + e_{ij}$$

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[2] Because we had a large proportion of Asian participants in the study, we re-ran our prospective models controlling for Asian race and found the same pattern of results. Time 1 cognitive reactivity was still a significant, negative predictor of Time 2 depressive symptoms, and Time 1 depressive symptoms were still non-significant in the prediction of Time 2 cognitive reactivity. Asian race did not moderate either of these relationships.

[3] We assessed the distribution of our between-subject variables and found that they were significantly skewed. We transformed each variable (log, square root, and inverse transformations), re-ran our regression analyses, and found the same pattern of results.

Depressive Symptoms$_j$ represents the between-person value of Time 1 depressive symptoms for Participant *j*. This equation shows the addition of this variable to the previous model as a main effect and as a moderator of the within-person relationship between negative affect and dysfunctional attitudes. *Depressive Symptoms*$_j$ x *Evening Negative Affect*$_{ij}$ represents the interaction of Time 1 depressive symptoms with Time 1 evening negative affect. *Depressive Symptoms*$_j$ was grand-mean centered; therefore γ_{20} represents the relationship between evening negative affect and change in dysfunctional attitudes at average levels of depressive symptoms. Finally, γ_{21} represents the change in this within-person slope for every one unit increase in depressive symptoms. Again, in this model, intercepts and slopes were allowed to randomly vary.

Results of this model indicated that both Time 1 depressive symptoms (γ_{01} = 0.611, $p < .001$) and evening negative affect (γ_{20} = 0.115, p = .016) had significant main effects on change in dysfunctional attitudes. However, these effects were qualified by a significant interaction between depressive symptoms and evening negative affect, γ_{21} = -0.162, p = .042.

To interpret this significant interaction, we calculated simple slopes in accordance with procedures outlined by Preacher, Curran, and Bauer (2006). We assessed the daily relationship between negative affect (*x*) and dysfunctional attitudes (*y*), at low and high levels (1 *SD* below/above the *M*) of the moderator, Time 1 depressive symptoms (*w*). To determine if any slope was significantly different from zero, the standard error of the simple slope was calculated with the variances and

covariances of the variable and interaction coefficients from the asymptotic covariance matrix. The moderation plot shows that the positive relationship between negative affect and dysfunctional attitudes was significant at low levels of depressive symptoms, $p = .004$, but not significant at high levels of depressive symptoms, $p = .661$, indicating that those participants with high depressive symptoms had attenuated cognitive reactivity slopes (see Figure 3.1).

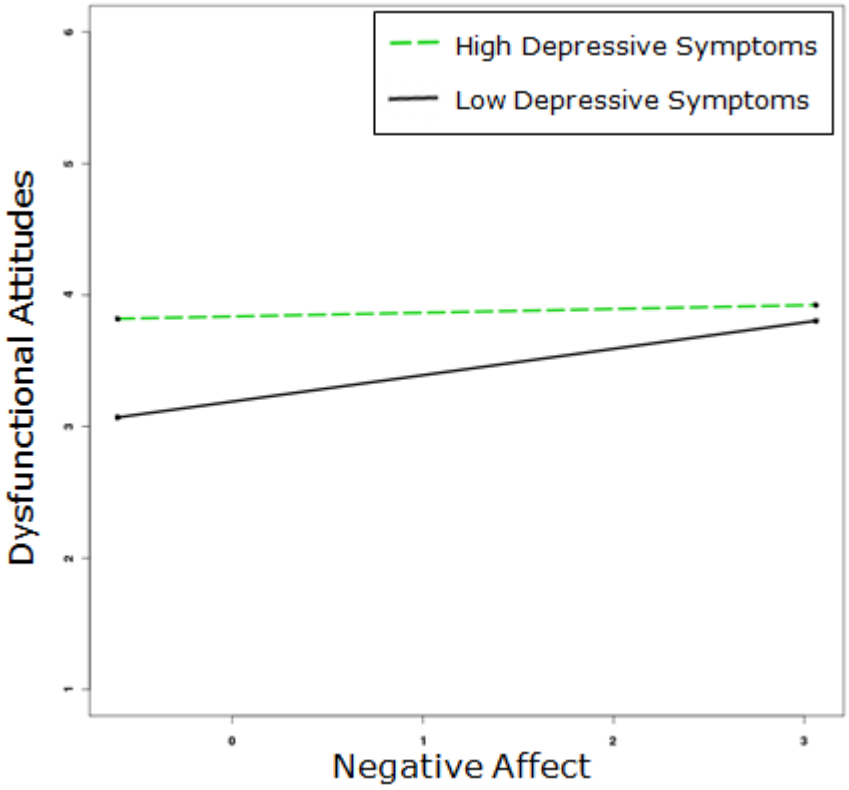

Figure 3.1: Moderation of the Daily Relationship between Evening Negative Affect and Evening Dysfunctional Attitudes by Depressive Symptoms at Time 1.

We repeated this same HLM analysis at Time 2. At Time 2, both Time 2 depressive symptoms (γ_{01} = 0.626, $p < .001$) and evening negative affect (γ_{20} = 0.122, p = .003) had significant main effects on change in dysfunctional attitudes. However, the interaction between depressive symptoms and evening negative affect was not significant, γ_{21} = -0.093, p = .231.

3.4 Affective Reactivity

Our findings thus far indicated that Time 1 cognitive reactivity was negatively associated with Time 2 depressive symptoms, and that Time 1 depressive symptoms moderated Time 1 cognitive reactivity. Therefore, we were interested in whether Time 1 depressive symptoms also moderated affective reactivity. Past research on cognitive (DAS) reactivity has largely relied on lab-based mood manipulations, whereas we relied on naturally-occurring changes in affect. Therefore, we assessed daily affective reactivity in our study to determine if negative affect did in fact increase as a result of daily stressors. To do so, we assessed the relationship between daily negative events and evening affect. To implicate negative events as the predictor of negative affect, we assessed change in negative affect from morning to evening as a function of total number of reported daily stressors. Specifically, we examined number of negative events as a predictor of evening negative affect, controlling for morning negative affect. Further, because we were interested in whether Time 1 depressive symptoms moderated affective reactivity, we included Time 1 depressive symptoms as a main effect and as a moderator. We estimated the following model in HLM:

$$Evening\ Negative\ Affect_{ij} = \gamma_{00} + \gamma_{01}(Depressive\ Symptoms_j) + \gamma_{10}(Morning\ Negative\ Affect_{ij}) + \gamma_{20}(Negative\ Events_{ij}) + \gamma_{21}(Depressive\ Symptoms_j \times Negative\ Events_{ij}) + u_{0j} + u_{2j}(Negative\ Events_{ij}) + e_{ij}$$

In this equation, $Evening\ Negative\ Affect_{ij}$ represents Participant *j*'s level of evening negative affect on Day *i*. $Depressive\ Symptoms_j$ represents the between-person value of Time 1 depressive symptoms for Participant *j*. $Depressive\ Symptoms_j \times Negative\ Events_{ij}$ represents the interaction of Time 1 depressive symptoms with negative events. As before, Level-1 predictor variables were group-mean centered, and the Level-2 variable was grand-mean centered. The slope of negative events, γ_{20}, represents the change in Participant *j*'s evening negative affect for every additional negative event at average levels of depressive symptoms. The value γ_{21} represents the change in this within-person slope for every one unit increase in depressive symptoms. The model intercept, as well as the slope for negative events, were allowed to randomly vary. The term u_{0j} represents the between-person deviations in the model intercept. The term u_{2j} represents the between-person deviations in the slope for negative events.

Results of this model indicated that both Time 1 depressive symptoms (γ_{01} = 0.471, $p < .001$) and number of daily negative events (γ_{20} = 0.084, $p < .001$) had significant main effects on change in evening negative affect. However, these effects were qualified by a significant interaction between depressive symptoms and number of daily negative events, γ_{21} = -0.113, $p = .005$.

To interpret this significant interaction, we again calculated simple slopes, this time assessing the daily relationship between negative events (*x*) and negative affect (*y*), at low and high levels (1 *SD* below/above the *M*) of the moderator, Time 1 depressive symptoms (*w*). The positive relationship between negative events and negative affect was significant at low levels of depressive symptoms, $p < .001$, but not significant at high levels of depressive symptoms, $p = .348$, indicating that those participants with high depressive symptoms had attenuated affective reactivity slopes (see Figure 3.2).

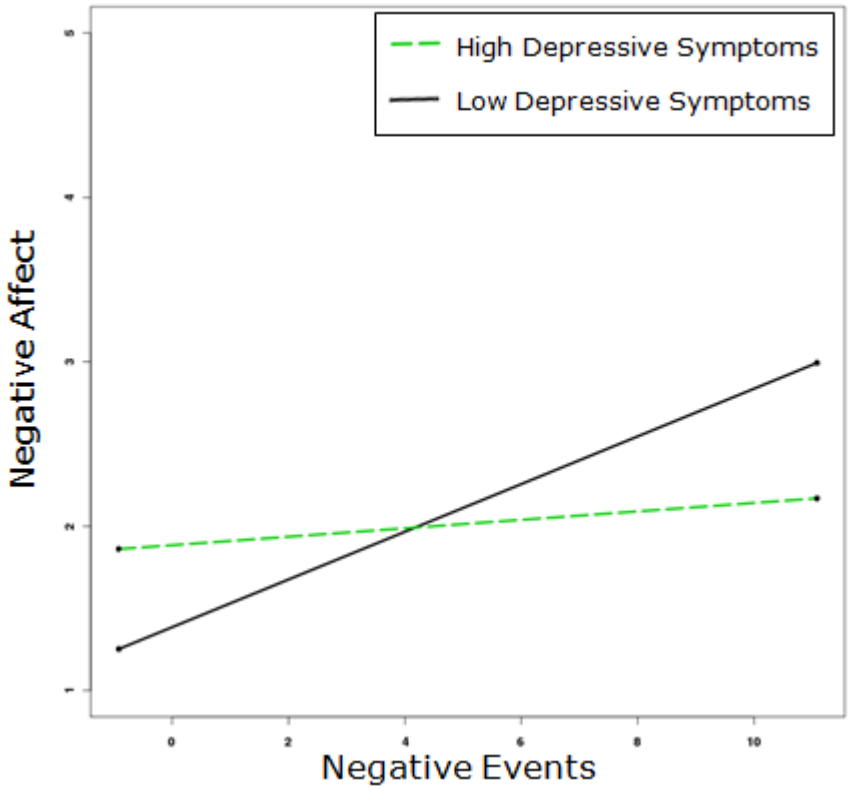

Figure 3.2: Moderation of the Daily Relationship between Number of Negative Events and Evening Negative Affect by Depressive Symptoms at Time 1.

We repeated this analysis at Time 2. At Time 2, both depressive symptoms (γ_{01} = 0.762, p < .001) and number of daily negative events (γ_{20} = 0.087, p < .001) had significant main effects on change in evening negative affect. However, the interaction between Time 2 depressive symptoms and number of daily negative events was not significant, γ_{21} = -0.024, p = .667.

Chapter 4

DISCUSSION

In the present study, we examined cognitive reactivity—activation of dysfunctional attitudes by negative affect—on a daily level. We also examined daily cognitive reactivity as a predictor of later depressive symptoms. Based on prior research (e.g., Gemar et al., 2001; Miranda et al., 1998; Segal et al., 2006), we hypothesized that cognitive reactivity would be positively associated with later depressive symptoms. However, given that our study is one of the first to examine this process on a daily level, we also considered the possibility of attaining a different pattern of results than has been reported previously. In particular, the detection of cognitive reactivity in participants' everyday environments hinged on the assumption that the changes in negative affect needed to elicit dysfunctional attitudes among depression-vulnerable individuals would in fact occur.

Our findings revealed that, overall, cognitive reactivity did occur on a daily level. This suggested that, at least for some participants, daily events produced negative affective responses, which elicited dysfunctional attitudes. However, when we put cognitive reactivity into our prospective model, examining Time 1 cognitive reactivity as a predictor of Time 2 depressive symptoms, we found a significant, negative relationship. This suggested that participants with weaker cognitive

reactivity subsequently reported more depressive symptoms. Thus, in our study, attenuated cognitive reactivity was a risk factor for later depressive symptoms.

In light of this unexpected finding, we returned to our HLM model to further examine our daily cognitive reactivity model. Given some previous research on weaker affective reactivity among depressed individuals (e.g., Bylsma et al., 2008), we explored our Time 1 daily cognitive reactivity model with Time 1 depressive symptoms as a moderator. We found that Time 1 depressive symptoms moderated the relationship between negative affect and dysfunctional attitudes. Our examination of this interaction further revealed that the positive relationship between daily negative affect and daily dysfunctional attitudes was significant among participants low in depressive symptoms, but not significant among participants high in depressive symptoms. Instead, the slopes of participants with high levels of initial depressive symptoms were flat, indicating attenuated cognitive reactivity. This suggested that, while daily cognitive reactivity occurred overall, it did not occur for those participants with greater depressive symptoms. This was consistent with our finding that weaker Time 1 cognitive reactivity was associated with greater depressive symptoms at Time 2. Further, this prospective analysis controlled for Time 1 depressive symptoms, suggesting that weaker daily cognitive reactivity was a risk factor for later depressive symptoms above and beyond the influence of initial depressive symptoms.

Although we observed that, in general, cognitive reactivity occurred in our sample, the fact that it did not occur among those with higher depressive symptoms brought us back to the initial consideration that daily stressors must exert an influence

on negative affect in order for cognitive reactivity to be observed among depression-vulnerable individuals. Examination of cognitive reactivity as a risk factor for later depressive symptoms required that, among depression-vulnerable individuals, everyday environmental factors produced observable daily changes in negative affect. The affective reactivity literature suggests, however, that this may not typically be the case. Namely, the emotion context insensitivity hypothesis (Rottenberg, 2005) suggests that those vulnerable to depression experience more blunted emotional responses—less change in both positive and negative emotion in response to environmental cues. If correct, then it would be likely that daily stressors would not elicit strong negative affective reactions among depression-vulnerable individuals. We directly tested this in our study by examining affective reactivity—activation of negative affect from negative events. In particular, we assessed whether Time 1 affective reactivity was moderated by Time 1 depressive symptoms. Our results indicated that, as expected given our cognitive reactivity findings, depressive symptoms moderated the daily relationship between negative events and negative affect. Our examination of this interaction further revealed that the Time 1 positive relationship between daily stressors and daily negative affect was significant among participants low in depressive symptoms, but not significant among participants high in depressive symptoms. Instead, the slopes of participants with high levels of depressive symptoms were flat, indicating attenuated affective reactivity.

Taken together, we observed attenuated affective reactivity as well as attenuated cognitive reactivity among participants with relatively high initial

depressive symptoms. This may suggest, in line with the emotion context insensitivity hypothesis (Rottenberg, 2005), that daily stressors did not elicit strong daily changes in negative affect among those participants with high depressive symptoms. Instead, these individuals evidenced affective blunting.

This study highlights an important distinction between activated versus non-activated negative affective states, related, possibly, to a further distinction between naturally-occurring mood changes and lab-based mood manipulations. Whereas much of the literature to date has examined activation of negative cognitive styles under successful mood manipulations (e.g., Miranda et al., 1998; Segal et al., 1999; Segal et al., 2006), activation of negative cognitive styles in the real world may not always occur, particularly if individuals exhibit a low level of affective fluctuations. This is not to say that these phenomena do not unfold in participants' everyday lives, but our work suggests that everyday stressors may not always elicit strong increases in daily negative affect, particularly for those individuals with higher depressive symptoms.

Although it is possible that the daily nature of our study was responsible for our unique outcomes, it is important to consider the results of a similar daily diary study which also assessed daily cognitive reactivity as a prospective predictor of depressive symptoms. Wenze et al. (2010) found a positive association between daily cognitive reactivity and later depressive symptoms. However, there were a number of differences between their study and the present study. First, Wenze et al. used a different schedule of assessments, with participants reporting on cognitive reactivity variables throughout the day, as opposed to twice a day, as was the case in the present

study. We also used different measures. Whereas we used an aggregate measure of negative affect that included negative and sad affect items, Wenze et al. also included items for anxiety, hostility, and guilt. Wenze et al. measured daily automatic thoughts, but not daily dysfunctional attitudes, which we measured to be consistent with the vast majority of studies in this area (e.g., Miranda et al., 1998; Segal et al., 1999; Segal et al., 2006). A final difference is that Wenze et al. did not assess daily affective reactivity. Therefore, it is difficult to directly compare their findings and ours. Namely, if their participants with a high level of initial depressive symptoms demonstrated greater affective reactivity, more in line with research by Cohen et al. (2004) and Parrish et al. (2011) than with the emotion context insensitivity literature, then that might account for our opposite findings on cognitive reactivity.

The negative relationship between Time 1 cognitive reactivity and Time 2 depressive symptoms was surprising. However, the Pearson's correlations between our major study variables, in particular the variables comprising the reactivity slopes, bolster support for the results attained in the present study. Although cognitive reactivity was negatively associated with depressive symptoms, our correlations showed that depressive symptoms were correlated in the expected, positive direction with mean levels of daily dysfunctional attitudes and with mean levels of daily negative affect. Thus, it was not the relationship between depressive symptoms and daily negative affect or between depressive symptoms and daily dysfunctional attitudes that deviated from previous findings in this area. Instead, it was specifically

the relationship of interest between depressive symptoms and daily cognitive reactivity that showed this unexpected association.

We also found that the prospective relationship between cognitive reactivity and depressive symptoms was uni-directional. While cognitive reactivity significantly predicted later depressive symptoms, initial depressive symptoms did not predict later cognitive reactivity. We undertook this analysis for exploratory purposes, as past research has primarily focused on cognitive reactivity as a vulnerability factor, and not a consequence, of depressive symptoms. The finding that this reverse relationship was not significant is consistent with Parrish et al. (2011), who found that, while affective reactivity predicted later depressive symptoms, initial depressive symptoms did not predict later affective reactivity. Taken together, these findings suggest that daily process variables involving daily cognitive reactivity and daily affective reactivity serve as prospective predictors, but not outcomes, in their relationship with depressive symptoms.

While our findings showed that daily cognitive and affective reactivity were moderated by depressive symptoms at Time 1, these moderational patterns were not replicated at Time 2.

It is not clear why these interactions were not significant at both time points. One possibility is the effect of completing daily diaries over time. In our study, participants completed the same measures up to 28 times (morning and evening for 14 total days). Some methodologists have suggested that many repeated measures over time can produce unintended effects. For example, repeated assessments may lead to

reactance (change in participants' experience or behavior), habituation (development of a habitual response style when making diary entries), increased complexity (development of a more complex understanding of the domain), and gradual entrainment (coming to fit one's conceptualization of the domain to what is measured; Bolger et al., 2003). It is possible that one or more of these factors played a role as the study progressed.

It is also important to note that we used a college sample to test our model of cognitive reactivity. Much of the extant research on this topic has been conducted with individuals who currently meet, or have met in the past, criteria for clinical depression. Further, we examined relative levels of depressive symptoms, and not depression per se (i.e., clinical cut-offs). While the use of a college sample was in some ways preferable, for example, to extend knowledge of these processes to an unidentified sample, it also points out the need to understand what it means to study clinical phenomena among a non-clinical sample. In the present study, it is likely that we were examining a restricted range of possible depressive symptoms scores relative to what might exist in the general population. Use of a restricted range could have produced a different pattern of results than what would be observed with the full range of scores. Further, college students represent a sample of presumably high-functioning individuals. It is generally difficult and perhaps even cautionary to measure dysfunctional processes among a generally functional sample. Additionally, our finding that those with lower depressive symptoms (both concurrently and subsequently) exhibited stronger cognitive reactivity is in contrast to the literature,

which suggests that non-vulnerable individuals should not exhibit cognitive reactivity, even in a negative affective state (Miranda & Persons, 1988; Miranda et al., 1990). Overall, it seems important to consider what it means to study cognitive reactivity among a high-functioning, unidentified sample and to measure relative levels of depressive symptoms.

Another potential limitation of our study was the timeline. We chose a timeframe that included assessments 10 weeks apart, as this was the longest feasible timeline during which to assess college students selected from their psychology courses. However, with respect to the constructs assessed, this timeline was arbitrary. Thus, it is possible that if we measured participants for a longer period of time, we may have obtained different prospective findings.

We also chose to assess participants twice daily—once in the morning and once in the evening. This too enhanced the feasibility of our study because it permitted the examination of daily changes in our variables while minimizing participant burden compared to study designs that require many responses throughout the day. Our assessment strategy was also based on some conceptual assumptions. First, we assessed participants at the end of the day so that they could report on all of the stressors that had happened to them that day. Additionally, by assessing participants when they first awoke, we hoped to capture their standing on negative affect and dysfunctional attitudes before the influence of daily stressors, as a sort of baseline to which to compare later negative affect and dysfunctional attitudes. Despite these practical and conceptual decisions, other research designs may permit different

types of questions that we could not address in the present study. For example, Wenze et al. (2010) paged participants several times throughout the day to obtain their negative affect and automatic thoughts ratings. Future work should build upon the present study to establish an assessment schedule that maximizes the interpretability and generalizability of findings.

Overall, the present study provided support for a linking of the cognitive reactivity and affective reactivity literatures, particularly with regard to daily experiences. Additional daily diary research is needed to replicate and further clarify the daily processes involved in reactivity among those vulnerable to depression. We hope that future work in this area will continue to examine cognitive responses to naturally occurring fluctuations in negative affect, as well as negative affective responses to naturally occurring daily experiences.

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APPENDIX – IRB APPROVAL LETTER

RESEARCH OFFICE

210 Hullihen Hall
University of Delaware
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DATE: January 26, 2011

TO: Lawrence Cohen
FROM: University of Delaware IRB

STUDY TITLE: [215302-1] Diary Study of College Students' Daily Events, Mood, and Attitudes

SUBMISSION TYPE: New Project

ACTION: APPROVED (R. Simons)
APPROVAL DATE: January 26, 2011
EXPIRATION DATE: January 25, 2012
REVIEW TYPE: Expedited Review

REVIEW CATEGORY: Expedited review category # 7

Thank you for your submission of New Project materials for this research study. The University of Delaware IRB has APPROVED your submission. This approval is based on an appropriate risk/benefit ratio and a study design wherein the risks have been minimized. All research must be conducted in accordance with this approved submission.

This submission has received Expedited Review based on the applicable federal regulation.

Please remember that informed consent is a process beginning with a description of the study and insurance of participant understanding followed by a signed consent form. Informed consent must continue throughout the study via a dialogue between the researcher and research participant. Federal regulations require each participant receive a copy of the signed consent document.

Please note that any revision to previously approved materials must be approved by this office prior to initiation. Please use the appropriate revision forms for this procedure.

All SERIOUS and UNEXPECTED adverse events must be reported to this office. Please use the appropriate adverse event forms for this procedure. All sponsor reporting requirements should also be followed.

Please report all NON-COMPLIANCE issues or COMPLAINTS regarding this study to this office.

Please note that all research records must be retained for a minimum of three years.

Based on the risks, this project requires Continuing Review by this office on an annual basis. Please use the appropriate renewal forms for this procedure.

If you have any questions, please contact Jody-Lynn Berg at (302) 831-1119 or jlberg@udel.edu. Please include your study title and reference number in all correspondence with this office.