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Mechanisms of Anxiety-Depression Co-Occurrence

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Abstract of the Dissertation

Mechanisms of Anxiety-Depression Co-Occurrence

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Anxiety and depression are highly comorbid, and mechanisms of their co-occurrence remain largely unclear. Several longitudinal studies suggest that anxiety disorders tend to temporally precede depression, but few comorbidity theories integrate this information. Furthermore, it is unclear whether this temporal pattern replicates when examining symptoms on a daily basis (potentially the time frame over which comorbidity mechanisms unfold). In addition, little research has attempted to identify mechanisms through which anxiety leads to later depressive symptoms. For example, anxiety may prompt rumination about one's anxiety symptoms, or may lead individuals to feel hopeless, in turn prompting depressive symptoms. The current study uses diary methods to examine several questions: First,

does anxious mood precede depressed mood on a daily basis (replicating patterns over longer time frames)? Second, do anxiety-focused rumination and hopelessness mediate this association? Finally, moderation models (where the association between anxious and depressed mood differed according to levels of rumination and hopelessness) were also tested. Fifty-five adults meeting full criteria for generalized anxiety disorder with a history of major depression symptoms were recruited from community sources. Participants completed a 21-day daily survey assessing anxious mood, depressed mood, anxiety-focused rumination, and hopelessness. Results showed that anxious mood predicted later depressed mood much more robustly than the reverse effect, and over multiple time lags. Results were similar for other symptoms of anxiety and depression. Hopelessness did not emerge as a significant mediator or moderator over the time lags tested. A moderational model was supported for anxiety-focused rumination, where anxious and depressed mood were more strongly associated on days when rumination was high. Results provide new, compelling data on the daily temporal patterns of anxiety and depressive symptoms, and offer preliminary suggestion that anxiety-focused rumination may play a role in generating this symptom co-occurrence.

To Jonathan

thanks for humoring me

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I. INTRODUCTION

Research has consistently documented extensive co-occurrence between anxiety and depression, both at the symptom and syndrome level (Maser & Cloninger, 1990). For example, according to the National Comorbidity Study-Replication, 57.5% of all individuals with major depressive disorder (MDD) also meet criteria for an additional anxiety disorder within the same 12-month period (compared to much lower comorbidity rates for impulse-control disorders or substance use disorders; Kessler, et al., 2003; Kessler, Merikangas, & Wang, 2007). This comorbidity does not appear to be limited to a particular form of anxiety, as each individual anxiety disorder shows a tetrachoric correlation with MDD in the range of .42 to .62 (Kessler, Chiu, Demler, & Walters, 2005). Taken collectively with the broad range of studies documenting co-occurrence of anxiety and depression throughout the life span (e.g., Brady & Kendall, 1992; T. A. Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Hiller, Zaudig, & von Bose, 1989; Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997; Mineka, Watson, & Clark, 1998; Sanderson, Beck, & Beck, 1990), the evidence suggests that anxiety-depression comorbidity is, in many ways, the rule rather than the exception.

Moreover, the presence of co-occurring anxiety and depression seems to have negative implications that go beyond the impact of each individual disorder. For example, comorbidity is associated with poorer prognosis, academic difficulties, suicide risk, worse overall quality of life, and worse treatment

outcomes than individually occurring disorders (Kessler, Stang, Wittchen, Stein, & Walters, 1999; Ledley, et al., 2005; Lewinsohn, Rohde, & Seeley, 1995; Rush, et al., 2005; Young, Mufson, & Davies, 2006). Further, some evidence suggests that comorbid anxiety may underlie gender differences in depression (Breslau, Schultz, & Peterson, 1995; Parker & Hadzi-Pavlovic, 2001, 2004), suggesting that better understanding comorbidity may help identify distal causes of women's higher vulnerability to depression. Clearly, a stronger comprehension of the origins of comorbidity has important theoretical and practical implications, and yet, insufficient research has identified mechanisms accounting for the relation between anxiety and depression.

What is Anxiety-Depression Comorbidity?

The term *comorbidity* has been used somewhat inconsistently in the literature (Lilienfeld, Waldman, & Israel, 1994). First, the time frame to which it refers varies across studies. At times comorbidity refers to two disorders occurring within the same lifetime (e.g., Lewinsohn, et al., 1997); other times it is defined as two disorders occurring simultaneously or within a designated time period (Rush, et al., 2005). Studying lifetime comorbidity can be informative, as it allows for the examination of long-term changes and patterns. On the other hand, examining processes of symptom co-occurrence in shorter, discrete time periods may be more effective in identifying day-to-day processes as they unfold, and this latter approach has rarely been used.

Second, comorbidity sometimes refers to symptom co-occurrence and other times to disorder co-occurrence (although some have argued that the term "comorbidity" should be limited to disorder co-occurrence; Mineka, et al., 1998). Although the majority of comorbidity research has focused on disorder co-occurrence, it may also be important, for several reasons, to examine if, how, and why the *components* of anxiety and depressive disorders (i.e., symptoms such as anxious and depressed mood) co-occur within short time frames during episodes. First, as noted by Mineka et al. (1998), the study of disorder comorbidity starts with observing how the symptoms that define the disorders co-occur. In other words, although symptom co-occurrence is not equivalent to disorder comorbidity, it may have implications for disorder comorbidity. Symptoms often develop into disorders (Judd, et al., 1998). In addition, disorders are, after all, made up of symptoms, and thus symptom co-occurrence and disorder comorbidity may operate under similar mechanisms. Next, understanding symptom co-occurrence may be useful in its own right, as it would enhance our understanding of the phenomenological experience of depressive and anxious symptoms within episodes. Finally, examining co-occurrence of symptoms rather than diagnostic categories eliminates the confounding effect of errors in the underlying nosological system (T. A. Brown & Barlow, 1992; Mennin, Heimberg, Fresco, & Ritter, 2008). For example, generalized anxiety disorder and major depression share several similar diagnostic criteria (e.g., difficulty concentrating, restlessness, psychomotor agitation, fatigue), and this overlap has

the obvious potential to inflate comorbidity rates. Examining relationships between symptoms rather than disorders helps correct for this problem. Thus, an important step in understanding depressive-anxiety disorder comorbidity is to clarify the relationship between depressive and anxiety symptoms.

Following the recommendations of Mineka et al. (1998), in this manuscript I reserve the term "comorbidity" for co-occurring disorders, and use the term "co-occurrence" to refer to symptoms. However, because the literature on disorder comorbidity is both more developed than the literature of symptom co-occurrence and relevant to making predictions about symptom co-occurrence, I also review research pertaining to comorbidity of major depression and anxiety disorders.

Pitfalls of Existing Models

Existing comorbidity models fall short of explaining anxiety-depression co-occurrence in several ways. The most widely cited theories of depression-anxiety co-occurrence, such as the tripartite theory (Clark & Watson, 1991), largely suggest that depression and anxiety are two facets of the same underlying phenomenon, and that depression-anxiety co-occurrence is a relic of overlapping features, shared core psychopathological processes, or inappropriately drawn diagnostic boundaries. For example, the tripartite theory (Clark & Watson, 1991) proposes that anxiety and depression share a common factor (negative affectivity), but are each distinguished by unique components, including low positive affectivity or anhedonia (depression) and physiological hyperactivity (anxiety). Other researchers have presented similar structural

models, in which shared underlying factors account for depression-anxiety co-occurrence (e.g., Barlow, 1991; T. A. Brown & Barlow, 1992; Tellegen, Tuma, & Maser, 1985). These structural models fit under the umbrella of the "lumper" perspective that anxiety and depression and their components cannot be adequately distinguished, standing in contrast to the "splitter" standpoint that anxiety and depression are fundamentally separate phenomena, distinguished by disparate risk factors, courses, and phenomenological experiences (see Wittchen, Kessler, Pfister, & Lieb, 2000).

Structural theories such as the tripartite model have greatly enhanced our knowledge of anxiety-depression co-occurrence by providing descriptive insight into which aspects of anxiety and depressive symptoms are most likely to co-occur. However, the tripartite model and other structural models do not sufficiently explain depression-anxiety co-occurrence, for several reasons. First, although a number of studies have supported the tripartite model (e.g., Joiner, 1996; Watson, Clark, et al., 1995), others have found that models with two correlated factors representing depression and anxiety provide a better fit with data (for a review, see Anderson & Hope, 2008; Burns & Eidelson, 1998; Ollendick, Seligman, Goza, Byrd, & Singh, 2003). Second, studies have shown both that physiological hyperactivity is correlated with depression and that anhedonia is correlated with anxiety (Chorpita & Daleiden, 2002; Jacques & Mash, 2004), contradicting the specificity hypothesis of the tripartite model and suggesting that comorbidity is not entirely accounted for by the hypothesized

shared substrates. Third, depression and anxiety have distinct courses, predictors, and correlates (Finlay-Jones & Brown, 1981; Moffitt, Caspi, et al., 2007; Starr & Davila, 2008b; Wittchen, Beesdo, Bittner, & Goodwin, 2003) supporting the "splitter" perspective that anxiety and depression are separate (although perhaps overlapping) phenomena, rather than the "lumper" notion espoused by structural theories. Fourth, structural models of comorbidity are largely descriptive, detailing which aspects of symptoms are most likely to co-occur rather than specifying mechanisms to explain why symptoms co-occur. Although descriptive theories (detailing *what* ways depression and anxiety overlap) are important for understanding the limits of categorical nosology, it is important to also develop theories positing *how* and *why* anxious and depressed mood co-occur.

Finally, structural models fail to incorporate an important and consistent finding in psychopathology research that may be critical to understanding comorbidity: the temporal ordering of anxiety disorders and depression.

Temporal Antecedence of Anxiety over Depression

Numerous studies have shown that anxiety disorders tend to temporally precede depression, using both retrospective (de Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003; Essau, 2003) and longitudinal (Burke, Loeber, Lahey, & Rathouz, 2005; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Kovacs,

Paulauskas, Gatsonis, & Richards, 1988; Lewinsohn, et al., 1997; Orvaschel, Lewinsohn, & Seeley, 1995; Wittchen, et al., 2000) designs (but see also Moffitt, Harrington, et al., 2007 for a notable exception). In addition, some observational evidence suggests that anxiety symptoms precede depressive symptoms within episodes (Alloy, Kelly, Mineka, & Clements, 1990). Also, although anxiety disorders often occur without depression, "pure" depression (i.e., without comorbid anxiety) is relatively rare (Dobson, Cheung, Maser, & Cloninger, 1990).

Several researchers have argued that the temporal precedence of anxiety may have important implications for models of comorbidity (Lewinsohn, et al., 1997; Wittchen, et al., 2003). Few existing comorbidity theories, however, explain this sequence. In one exception, Alloy et al. (1990) proposed that stressful life events first elicit helplessness (characteristic of both depression and anxiety), which later proceeds to hopelessness (characteristic of only depression) for those with particular cognitive vulnerabilities. Although this theory is novel in that it incorporates the temporal sequence of anxiety disorders and major depression, it is again largely descriptive, with little specification of mechanisms of comorbidity. The empirical support for this theory has also been somewhat inconsistent (Swendsen, 1997). Thus, the existing comorbidity models fail to incorporate anxiety's temporal antecedence over depression.

Furthermore, although strong evidence suggests that anxiety disorders tend to precede major depression over the course of many years or a lifetime, the temporal relationship of day-to-day anxious and depressed mood needs

clarification, a question best examined using daily diary methods. Most longitudinal studies examining temporal associations have examined comorbid diagnosable disorders using follow-up periods of six months or more (Burke, et al., 2005; Cole, et al., 1998; Orvaschel, et al., 1995; Wittchen, et al., 2000). Although this is informative, clarifying whether anxious mood also tends to precede depressed mood may be important for the development of comorbidity models. Depressive and anxious symptoms co-occur at almost twice the rates of diagnosable depressive and anxiety disorders (Hiller, et al., 1989), possibly suggesting that mechanisms of co-occurrence act at the symptom level. If so, a more thorough understanding of the temporal relationship between symptoms (particularly cardinal symptoms such as depressed and anxious mood) may be critical to understanding comorbidity. Some evidence (drawing from sources as diverse as experimental research on response to uncontrollable negative events, non-human primate research, and attachment research; Alloy, et al., 1990) suggests that anxiety symptoms tend to precede depressive symptoms within episodes, but this evidence remains limited and needs to be supplemented with further data. As anxious and depressed mood varies considerably from day-to-day (de Vries, Dijkman-Caes, & Delespaul, 1990), investigating how symptoms predict each other on a daily basis may shed light on mechanisms of symptom co-occurrence. One study using a daily experiences design found that daily fluctuations in anxiety predicted later depressed symptoms (and not the reverse; Swendsen, 1997). Another study, using a clinical sample, found the reverse

trend (but nonsignificant and with only 15 participants; de Vries, et al., 1990). Given the paucity of studies and the inconsistency of results, these findings clearly need replication.

The examination of the temporal relation between daily depressed and anxious moods is an important supplement to existing research, for several reasons. Many previous studies on temporal sequencing of anxiety disorders and major depression may have been confounded by the fact that different disorders tend to have differential ages of onset. For example, anxiety disorders tend to emerge in childhood (Kessler, Berglund, Demler, Jin, & Walters, 2005), whereas depression tends to emerge in adolescence or later (Lewinsohn, Hops, Roberts, & Seeley, 1993). The temporal precedence of anxiety disorders over depression may simply reflect this disparity in course. Examining daily changes in mood eliminates this potential confound, and may be a more powerful test of the idea that aspects of anxiety act as a risk factor for depressive symptoms.

Further, examining change at the daily level may uncover patterns that are not discernable over long follow-up periods. For example, one recent study showed that depression and generalized anxiety disorder (GAD) often develop simultaneously (Moffitt, Harrington, et al., 2007). Even in this case, anxiety may precede depressed mood within simultaneous episodes, a finding that would be obscured by looking only at disorders over long follow-up periods.

In sum, although depression and anxiety co-occur at overwhelmingly high rates, the mechanisms driving their co-occurrence remain largely a mystery.

Most existing models are largely descriptive rather than being mechanism-driven, and many fail to incorporate the typical temporal sequence of anxiety and depression. The purpose of this dissertation is to delineate a new model of depression-anxiety co-occurrence that both 1) helps explain why anxiety tends to temporally precede depression and 2) specifies mechanisms by which anxiety leads to later depressive symptoms.

Does Anxiety Act as a Risk Factor for Later Depression? And If So, Why?

A parsimonious explanation of the temporal antecedence of anxiety is that anxiety acts as a risk factor for later depression. Several researchers (e.g., Kessler, Nelson, McGonagle, & Liu, 1996; Lewinsohn, et al., 1997; Wittchen, et al., 2003) have proposed this idea, but little research has expanded upon it. An important step in determining whether anxiety serves as a risk factor for depression is to identify mediators in the anxiety-depression relationship. Although identifying mediators will not determine causality, it may offer preliminary insight into possible mechanisms.

Few studies to date have attempted to identify mediators in the anxiety-depression relationship (see also Wittchen, et al., 2000). Grant, Beck, Farrow, and Davila (2007) found that avoidance of expressing emotion mediated the relationship between baseline social anxiety and depressive symptoms one year later, suggesting that interpersonal behavior may play a role in the causal relationship between anxiety and depression. Moitra, Herbert, and Forman (2008) found that, in a sample of patients in treatment for social phobia,

behavioral avoidance mediated the cross-sectional relationship between social anxiety and depression, and that reduction in behavioral avoidance over the course of treatment predicted reductions in post-treatment depression. These studies are an important start to identifying mediators in the anxiety-depression relationships. However, each only examined one form of anxiety (social anxiety), and tested few potential mediators. Further, Moitra et al.'s study (2008) included several methodological weaknesses (e.g., cross-sectional design) that limited its conclusions. Also, examining mediators at the daily level has yet to be attempted and could be informative. Some mechanisms of co-occurrence, such as those outlined below, may occur over the course of hours and days rather than over months and years. As such, diaries may be more effective in capturing experiences and events in their natural contexts. Diary methods also avoid biases from retrospection, as some evidence suggests that retrospective recall overestimates symptom occurrence (de Vries, et al., 1990).

Persistent anxiety is a difficult experience, and as such may be construed as a chronic stressor. Thus, anxious mood may lead to depressed mood in much the same way that stress often leads to depressed mood, by activating depressogenic processes (i.e., processes that have been empirically shown to predict the onset of depressive symptoms). For example, anxious mood may, like stress, lead to feelings of hopelessness. Abramson et al. (1989) define hopelessness as negative expectancies for important outcomes that one feels helpless to change. If anxiety is perceived to have a negative impact and to be

uncontrollable, it may have the potential to lead to hopelessness. In turn, hopelessness is a robust predictor of depression. Several theorists have identified hopelessness as an etiological factor in depression (e.g., Abramson, et al., 1989; G. W. Brown & Harris, 1978). Abramson et al. (1989) argued that hopelessness is a *sufficient* cause of (i.e., always leading to) depression, particularly a subtype of depression termed "hopelessness depression." Empirical support has strongly supported the notion that hopelessness predicts and temporally precedes depression (Joiner, Wingate, & Otamendi, 2005; Metalsky, Joiner, Hardin, & Abramson, 1993; Rholes, Riskind, & Neville, 1985). Thus, as anxious mood has the potential to spur hopelessness and, in turn, hopelessness provokes depressed mood, hopelessness may mediate the change from anxiety to depressed mood. Interestingly, cognitive vulnerabilities associated with hopelessness strongly predict comorbid depression-anxiety, but not anxiety by itself (Alloy, et al., 2006), fitting with the idea that hopelessness is involved in the generation of depression-anxiety co-occurrence.

Rumination may also link anxious and depressed mood. Rumination has been shown to predict depressive onset following a naturalistic stressor (Nolen-Hoeksema & Morrow, 1991), and to prolong and exacerbate existing depressive symptoms (Nolen-Hoeksema, 1991). Although rumination has been previously defined as a style of responding to depressive symptoms (Nolen-Hoeksema, 1991) or stressors (Robinson & Alloy, 2003) rumination may also focus on anxiety symptoms. For example, an anxious person may think "Why am I so

anxious?" and "I'll lose my job if I don't get control over my anxiety." These processes may disrupt adaptive problem-solving and instill more pessimistic thinking (Lyubomirsky, Tucker, Caldwell, & Berg, 1999; Nolen-Hoeksema, Parker, & Larson, 1994), leading to depressive symptoms. Commenting on his recent study showing that ruminative response moderated the relation between anxiety and later depressive symptoms, Hankin (2008) made a similar conjecture, suggesting that rumination may act as a cognitive vulnerability for depression and anxiety as an emotional stressor.

As hopelessness and rumination are theoretically likely to be spurred by anxious mood and to lead to depressed mood, they each may serve as mediators in the anxious mood-depressed mood sequence. I will evaluate this hypothesis using diary methods, exploring whether symptom-focused hopelessness and rumination mediate the relationship between anxious mood and later depressed mood. These potential mediators may not operate independently. In fact, evidence suggests their relationship is far more complex and intertwined with other factors such as interpersonal distress, with hopelessness mediating the rumination-depression relation, interpersonal distress partially mediating the hopelessness-depression relation, and rumination leading to loss of social support (Joiner, et al., 2005; Nolen-Hoeksema & Davis, 1999; Sarin, Abela, & Auerbach, 2005). The role of these variables in depressed/anxious mood co-occurrence also may be complex, but this study represents one important step toward beginning to clarify their role.

Importantly, these two mechanisms may occur over different time lags. Hopelessness may not be provoked by the experience of anxious mood in itself, but rather the stability of the mood. A person who is rarely anxious may not feel hopeless after a brief period of anxiety. In contrast, a person who experiences a prolonged period of anxious mood lasting several days or more may start to lose hope that the anxiety will ever remit. In comparison, worry (a close cognitive cousin of rumination) has been shown to increase with the induction of anxious mood (Eysenck, 1984). Thus, the time lag between anxious mood and the onset of rumination may be much shorter or even non-existent. To ensure that effects are not masked by these issues, I tested multiple time lags.

In addition, it is possible that these cognitive factors may be better conceptualized as moderators of the relationship between anxious and depressed mood rather than mediators. This would imply, for example, that anxious mood would be more likely to lead to depressed mood on days in which the person is ruminating. This idea is distinct from the mediation hypothesis in that it does not suppose that the anxious mood, in itself, causes the rumination, which in turn causes the depressed mood. Instead, it suggests that anxious mood is particularly depressogenic when it co-occurs with rumination, consistent with diathesis-stress models. Supporting this idea, in a recent study, Hankin (2008) found that rumination interacted with anxiety to predict increases in depressive symptoms in an adolescent sample. This study, however, focused on subclinical symptoms in an adolescent community sample over a multi-year

period; no studies to my knowledge have examined rumination as a mediator of anxiety-depression co-occurrence over shorter periods in adult or clinical samples. Because of the lack of research in this area, it is unclear whether the mediation or moderation approach is more appropriate; thus, I tested both.

Both anxiety and depression are multifaceted constructs. Their specific factor structure varies somewhat across studies, but there is consensus that they are multidimensional disorders (Nitschke, Heller, Imig, McDonald, & Miller, 2001). The DSM-IV conceptualization of depression, for example, consists of two cardinal components: sad mood (or negative affect) and anhedonia (or lack of positive affect; American Psychiatric Association, 1994). Anxiety consists of a physiological component (i.e., anxious arousal), a cognitive component (e.g., worry), and a behavioral component (avoidance; American Psychiatric Association, 1994; Nitschke, et al., 2001). Some existing studies examining daily depressed and anxious mood have assessed multiple aspects of depression and anxiety (Hankin, Fraley, & Abela, 2005); others broadly assess subjective feelings of depressed and anxious mood (e.g., visual analog scales; Swendsen, 1998). Because of the minimal research in this area, it is unclear which is the most appropriate approach. It may be that particular aspects of anxious mood predict particular aspects of depressed mood (as some components of depressed and anxious mood have greater overlap; Clark & Watson, 1991). Because there is no clear empirical or theoretical rationale for one particular

approach over the other, I examined the temporal relation between several different aspects of depressed and anxious mood.

Pilot Study

I conducted a preliminary pilot study to test some of these basic ideas (see Starr & Davila, 2008a). First, I constructed a measure, the Response to Anxiety Questionnaire (or RAQ), to assess the degree to which individuals responded to their anxiety symptoms with rumination and hopelessness. The RAQ was heavily based on pre-existing validated scales of hopelessness and rumination, specifically the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974)) and the Ruminative Response Scale (RRS; Nolen-Hoeksema, Larson, & Grayson, 1999). The pilot study tested the validity of this measure in an undergraduate sample. Although my original intention was to divide the RAQ into separate rumination and hopelessness subscales, an exploratory factor analysis suggested that the RAQ better fit with a single factor comprising all items.

This pilot study revealed several findings consistent with the model presented here. First, given that the model predicts that anxiety-focused rumination and hopelessness would be predicted by anxiety and lead to depressive symptoms, we would expect the RAQ to be positively correlated with both anxiety and depressive symptoms. Indeed, that was the case—the RAQ was associated with numerous anxiety and depression scales that are known for having high discriminant validity, including the subscales of the Depression

Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995), the Mood and Anxiety Disorder Questionnaire (MASQ; Watson, Weber, et al., 1995), and the Inventory of Depression and Anxiety Symptoms (IDAS; Watson, et al., 2007). However, as the RAQ was also associated with the RRS and the BHS (i.e., the scales on which it was based), and as the RRS and BHS were both also associated to all anxiety and depression scales, the precise specificity of these associations may be obscured.

Thus, I next examined partial correlations between the RAQ and depression and anxiety measures, controlling (separately) for the RRS and BHS. I found that the RAQ maintained its association to all anxiety and depression scales when controlling for either the RRS or the BHS. However, when I conducted the reverse analysis (looking separately at the RRS's and BHS's partial correlations to anxiety and depression scales, controlling for the RAQ), I found that they were primarily associated with the depression scales, losing their associations with anxiety scales. This provides some preliminary evidence that the construct of anxiety-focused rumination and hopelessness captures something distinct from conventional hopelessness scales and measures of depressive rumination. Next, I tested whether controlling for the RAQ would reduce the association between anxiety and depression, and confirming predictions, I found significant reductions in the associations between every anxiety scale and its corresponding depression scale.

This pilot study had results that were in line with the predictions of my model. However, the pilot study was also limited by several methodological features. For example, the study used an undergraduate sample of convenience, and although such samples can be important first steps, it is important to ultimately test predictions about clinical phenomena in clinical samples. Even more critically, this pilot study was cross-sectional, despite testing the unfolding of processes over time. The current study overcomes these limitations to test the same underlying model.

The Current Study

I examined several specific hypotheses using daily diary methods. Prior to testing my hypotheses, I confirmed whether anxious mood was associated with concurrent depressed mood. I next tested the following hypotheses: First, to determine whether the temporal sequencing of anxiety disorders and depression extends to the temporal sequencing of anxious and depressed mood, I examined both whether broadly defined daily anxious mood predicts later daily depressed mood and whether individual daily anxiety symptoms (e.g., worry) predict individual daily depression symptoms (e.g., anhedonia). I expected that daily anxious mood would predict increases in daily depressed mood, although the analyses of specific mood components were exploratory; I made no specific predictions about which aspects of anxiety predict which aspects of depression. Also, as it was unclear what time period these processes might occur over, I tested multiple time lags.

Second, I hypothesized that daily anxious mood would predict daily symptom-focused rumination and hopelessness. Third, I anticipated that daily symptom-focused rumination and hopelessness would, in turn, predict increases in daily depressed mood.

Finally, I predicted that the daily relation between anxious mood and depressed mood would be mediated by symptom-focused rumination and symptom-focused hopelessness. I also examined, as an alternative hypothesis, interaction models, in which anxious mood most strongly predicts depressed mood on days in which rumination (or hopelessness) occurs.

I used diary methods to examine daily temporal patterns of depressed mood, anxious mood, hopelessness and rumination. The diary method offers several benefits over traditional designs. First, within-subjects designs dramatically increase power. Second, diaries allow for the examination of phenomena in their natural, unstructured context (Bolger, Davis, & Rafaeli, 2003). Next, diaries significantly reduce memory biases introduced by retrospection (Bolger, et al., 2003). These advantages make diary methods an important compliment to traditional cross-sectional and longitudinal designs, and as such diary methods have yielded important insight into a broad range of aspects of psychopathology (e.g., De Vries, 1992; Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001).

The limited previous research on daily mood co-occurrence has primarily used samples of convenience, such as undergraduates, with primarily sub-

syndromal symptoms (Swendsen, 1997, 1998). Some (Coyne, 1994) argue that the transient psychological distress found among samples of convenience is a distinct construct from clinical disorders, with different patterns and predictors. The sub-syndromal anxiety may also not be clinically significant enough to spur the hypothesized depressogenic processes. In this study, I instead use a sample of individuals with a current anxiety disorder and at least some propensity toward the development of depression (as evidenced by a history of depressive symptoms). This both provides a more powerful test of study hypotheses and helps maximize the generalizability of results to relevant clinical populations.

The model described above may be applicable to multiple anxiety disorders. All anxiety disorders show significant comorbidity with depression (Kessler, Chiu, et al., 2005). All anxiety disorders share analogous features, including anxious thoughts, physiological hyperactivity, and behavioral avoidance, which may activate symptom-focused hopelessness and rumination in a common manner. On the other hand, different anxiety disorders may relate to depression via different pathways as a function of their unique characteristics. As a starting point, the current study focuses on a single anxiety disorder, generalized anxiety disorder (GAD). Comorbidity between GAD and major depression is particularly high. The National Comorbidity Study Replication found a one-year tetrachoric correlation between GAD and major depression of .62, higher than depression's correlation with any other anxiety disorder (Kessler, Chiu, et al., 2005). Similarly, Hunt, Slade, and Andrews (2004) found that 39.3%

of individuals with GAD also met criteria for major depression within the same one-month period. In fact, GAD co-occurs with depression with such regularity that some have argued that GAD should be classified with depression as a "general distress" disorder (Watson, 2005), although Mennin et al. (2008) raise important counterarguments to this model. Thus, although GAD may not be the only anxiety disorder relating to depression via the proposed mechanisms, examining mechanisms of GAD-MDD comorbidity may be a good launching point for the development of a broad model of anxiety-depression comorbidity.

II. METHOD

Participants

All participants were required to meet the following inclusion criteria: 1) meet full DSM-IV criteria for current GAD (ignoring the major depression exclusion criterion); 2) report some history of at least 1-2 cardinal symptoms of major depression or dysthymia (to ensure that participants have some risk of depressive symptoms); 3) have no present psychotic or bipolar disorders; 4) fall in age range of 18-65 years; 5) report no difficulties or disabilities with reading English that may impair questionnaire comprehension. No other exclusion criteria were imposed, including regarding diagnostic category. Indeed, as

detailed in Table 1, participants showed a great deal of intra-anxiety disorder comorbidity.

Participants were recruited from a variety of sources. First, flyers with study contact information were posted around the campus of Stony Brook University and the surrounding area, including on the bulletin board of the waiting room of the Stony Brook University Psychological Center/ Anxiety Center (see Appendix A for a copy of the flyer). Second, advertisements were posted on the internet (e.g., newyork.craigslist.org), with text similar to the paper flyers. Third, research staff contacted therapists at the Stony Brook University Psychological Center and the Stony Brook University Anxiety Center (both graduate training clinics that treat patients with anxiety disorders). Therapists were given a list of study criteria and asked to give a letter with a description of the study and contact information (see Appendix B) to patients whom they believed may be eligible and interested in participating. All potential participants recruited through the above methods were screened using major depressive episode, GAD, and psychotic disorder screening modules of the Mini-International Neuropsychiatric Interview (MINI; Sheehan, 1998) to determine eligibility. 45 individuals who appeared to fit research criteria were interviewed with the mood and anxiety disorder modules of the Structural Interview for the DSM-IV (SCID; Spitzer, Williams, Gibbon, & First, 1995) to verify project eligibility. Based on SCID results, 7 were determined to be ineligible and excused from the study, leaving 38 participants who completed full procedures. Participants were paid $25 for

completing their interview and $125 for participating in the remainder of the study and were eligible to participate in raffles for an iPod and a GPS navigation system.

Additional participants were recruited from recent studies that administered the SCID within the Department of Psychology at Stony Brook University. First, I recruited participants from the Project on Adolescent Relationships, a community study of adolescent girls and their mothers (PAR; see Davila, et al., 2009). Adult participants (mothers) who met study criteria were contacted following their participation in PAR and recruited for the current study. Two PAR participants were contacted; both agreed to participate in the current study. In addition, I recruited two participants from the Psychophysiology of Major Depressive Disorder (PMDD), a project investigating error-related negativity in subjects with GAD and current and remitted depression. To reduce burden, for these participants eligibility was determined using SCID data collected in the previous studies (all of which were collected within six months) rather than re-interviewing participants. Because they did not participate in the interview portion of the study, these participants were paid $125 and were also eligible to win raffle prizes. One of these participants did not complete the SCID for the PMDD study and thus completed the SCID for the current study according to normal study procedures).

Finally, I recruited additional participants from undergraduate psychology courses at Stony Brook University. These participants completed self-report

screening measures (including a self-report version of the MINI constructed for this study and the Penn State Worry Questionnaire; Meyer, Miller, Metzger, & Borkovec, 1990) as a component of a mass testing day in the psychology classes; potentially eligible students were contacted and scheduled for participation. In total, 24 students participated, with 10 determined to be ineligible following the SCID, leaving 14 eligible participants (the increased ineligibility rate is likely a result of the decreased specificity of self-report screening measures). Students were compensated with course credit comparable to the payment amounts and were also eligible to win raffle prizes.

In total, 55 eligible participants completed all study procedures. Demographic characteristics of the sample and recruitment information are listed in Table 2. The sample included 49 women and 6 men. The low percentage of men is likely a result of the female preponderance in anxiety disorders (Armstrong & Khawaja, 2002; Robichaud, Dugas, & Conway, 2003) and, given that participants were recruited from both treatment clinics and psychology classes, the greater treatment-seeking tendencies of women (Aalto-Setälä, Marttunen, Tuulio-Henriksson, & Lönnqvist, 2002) and the overrepresentation of female students in collegiate psychology courses (Metzner, Rajecki, & Lauer, 1994). The mean age was 28.76 (*SD*= 12.43, age range 18 to 59). Participants endorsed a broad range of racial and ethnic backgrounds, with 71% describing themselves as non-Hispanic white, 4% as Latino, 18% as Asian or Asian-

American, 2% as Native-American, and 5% as representing other or multiple racial/ethnic backgrounds (no eligible African-Americans were identified).

Furthermore, participants reported a broad range of annual household income, with 20% earning less than $30,000, 26% earning between $30,000 and $50,000, 11% earning between $50,000 and $70,000, 16% making between $70,000 and $90,000, and the remaining 27% earning more than $90,000. Forty-four percent of participants were currently in some form of treatment for their psychiatric disorders, with 26% of the total sample taking psychiatric medications and 36% of the total sample receiving some form of psychosocial intervention.

Although participants were recruited from a wide range of sources, it is important to remember that they were subjected to the same strict inclusion criteria. To ensure that the diversity of recruitment sources did not introduce unwanted heterogeneity into the sample, I examined whether recruitment source was associated with demographic variables, baseline symptom measures, and daily anxious and depressed mood. I compared groups of subjects recruited from 1) advertisements (*n*= 31), 2) treatment clinics (*n*= 6), 3) psychology classes (*n*= 14), and 4) research studies (*n*= 4) using one-way ANOVAs (or chi-square goodness-of-fit tests as necessary), although note that this is likely somewhat underpowered given the 4-level independent variable and the unequal groups. I found no differences between recruitment sources on gender, number of baseline diagnoses, nor depression or anxiety symptom measures at baseline,

including the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), Beck Anxiety Inventory (Beck, Epstein, Brown, & Steer, 1988), all depression and anxiety scales of the Depression Anxiety Stress Scale (Antony, Bieling, Cox, Enns, & Swinson, 1998) and the Mood and Anxiety Symptom Questionnaire (Watson, Weber, et al., 1995), nor on baseline measures of rumination and hopelessness (including the RRS and the BHS). There were significant differences between recruitment groups on mean age (F=5.39, p= .003), and post-hoc tests suggested that participants recruited from undergraduate psychology courses were unsurprisingly younger (mean age= 18.64, SD= 1.15) than were participants recruited from all other sources (community advertisement M= 31.52, SD= 13.59; treatment clinics M= 33.17, SD= 11.53, research studies M= 36.25, SD= 6.34). Also note that the different forms of compensation did not relate to diary compliance; participants who were reimbursed with course credit did not differ in their number of missed diaries from participants reimbursed with cash payment.

The Stony Brook University Committee on Research Involving Human Subjects approved this research. Appendix C displays the approved consent form for this project.

Measures

Screening measures. As described above, participants who responded to advertisements on the internet, around campus, and through their therapy clinics were screened for project eligibility using the GAD, depression, and

psychotic disorder screening modules of MINI (Sheehan, 1998), a brief structured diagnostic interview. The MINI has been shown to generate similar results to longer structured and semi-structured interviews in substantially less time (Sheehan, 1998). MINI interviews were conducted by an advanced graduate student or by trained advanced undergraduates.

For logistical reasons, participants recruited through psychology classes were screened with self-report measures rather than structured interviews. These included the Anxiety and Depression Detector (ADD; Means-Christensen, Sherbourne, Roy-Byrne, Craske, & Stein, 2006), the Penn State Worry Questionnaire (PSWQ; Meyer, et al., 1990), and a self-report version of key MINI items. The PSWQ shows adequate specificity and sensitivity as a screener for GAD in community and clinical samples (Behar, Alcaine, Zuellig, & Borkovec, 2003; Fresco, Mennin, Heimberg, & Turk, 2003). The ADD has demonstrated strong sensitivity but less than ideal specificity (GAD specificity .59; Means-Christensen, et al., 2006). The self-report version of the MINI was constructed for use in this study and included face valid questions assessing the presence of current GAD and current or past MDD symptoms. It is important to remember that all participants were interviewed with the SCID to confirm project eligibility, and the final sample included only participants meeting eligibility criteria according to the SCID results. Furthermore, screening measures were not used in any of the data analyses presented in this manuscript. Thus, these measures'

specificity shortcomings should neither have substantially diluted the quality of the sample nor the validity of the analyses.

Diagnostic interview. The SCID is a widely-used semi-structured interview designed to generate DSM-IV diagnoses (Spitzer, et al., 1995). The SCID has excellent psychometric properties, including test-retest and inter-rater reliability (Zanarini, et al., 2000). As with previous studies (e.g., Starr & Davila, 2008b), to capture both categorical diagnoses and dimensional symptoms I used a 4-point scoring system, where 0= no symptoms present, 1= mild symptoms (1-2 symptoms present), 2= moderate symptoms (3-4 symptoms present), and 3= DSM-IV diagnosable disorder. To reduce participant burden, I only administered the anxiety disorder and mood disorder modules, including generalized anxiety disorder, panic disorder, agoraphobia, social phobia, post-traumatic stress disorder, specific phobia, obsessive-compulsive disorder, separation anxiety disorder, major depressive episode, manic episode, and dysthymic disorder. Because participants recruited through undergraduate psychology courses did not complete a psychotic disorder screen as part of their preliminary screening questionnaires, they also completed the SCID's psychotic disorder screen. Both current and past psychopathology were assessed. In order to meet eligibility criteria, participants had to score a three on current GAD, a one or more on either past or present major depressive episode or dysthymia, and a zero on bipolar disorder and psychotic disorders. Interviews were taped using digital audio recorders, and 12 interview audio recordings (22% of eligible participants)

were re-coded by a second rater; for all disorders, reliability was adequate to excellent. Table 3 presents SCID reliability data. Table 1 presents SCID results for all eligible participants. As Table 1 illustrates, there was substantial diagnostic diversity among the sample.

Self-report baseline and follow-up measures. Demographic variables including age, race/ ethnicity, gender, and other variables were collected at baseline.

Diary items. As excessive diary length can result in poor compliance (Morren, Dulmen, Ouwerkerk, & Bensing, 2009), diary items were designed to assess constructs of interest quickly and efficiently. Daily depressed mood, anxious mood, and hopelessness were assessed using face-valid questions about mood at the moment of diary completion ("How anxious do you feel right now?" "How depressed do you feel right now?" "How hopeless do you feel right now?") with a 10-point Likert-type scale. Asking about mood at the moment of diary completion minimizes retrospective recall (Parkinson, Briner, Reynolds, & Totterdell, 1995), a principal goal of diary research (Stone, Litcher-Kelly, Eid, & Diener, 2006). On the other hand, asking about mood at the moment of diary completion captures only a thin slice of daily mood, and also may be inflated by the regular diurnal mood variation that some depressed individuals experience (as research suggests that negative affect peaks toward the end of the day, when participants were asked to complete their diaries; Robbins & Tanck, 1987). Thus, I also assessed mood and hopelessness experienced over the course of

the day (e.g., “How anxious did you feel, on average, over the course of the day today?”). Throughout this manuscript, these items are distinguished as “momentary” versus “course of day.” The diary also assessed other components of daily depressed and anxious mood, including anhedonia (“Felt little or no enjoyment in activities you usually enjoy”) and worry (“worried”), and to reduce length those items were only assessed over the course of the day.

Daily rumination was assessed with the item “how much did you think or ruminate about feeling anxious today?” with a Likert-type scale ranging from “not at all” to “a lot.” For these items, it was more logical to ask about their experiences over the course of the day, because it is less likely that they will be ruminating at the moment of filling out the diary.

Procedure

Study procedures following the recruitment stage were divided into three phases: baseline, diary, and follow-up.

Phase 1: Baseline. The baseline phase consisted of three components: consent procedures, SCID interview, and baseline questionnaires. The baseline questionnaires were completed on the same day as the SCID. In order to recruit more participants (including those living outside of the Stony Brook area and those who may prefer not to come to the lab for anxiety-related reasons) and reduce burden, participants had the option of completing this phase in several different manners. First, they were given the option of signing their consent forms in the lab or returning it through the mail. Second, they were given the

option of completing their baseline questionnaires in the lab or at home through a secure online website (if they did not have internet access, paper questionnaires were provided through the mail as a back-up). Studies suggest that completing questionnaires online generates results comparable to paper-and-pencil questionnaires (Coles, Cook, & Blake, 2007; Fouladi, McCarthy, & Moller, 2002), and in the current study there were no significant differences on baseline measures completed online versus on paper. Third, participants had the choice of completing their SCID interview in person in the laboratory or over the phone. Phone interviews and in-person interviews also produce similar results (Rohde, Lewinsohn, & Seeley, 1997), and in this study SCID results were not significantly higher or lower according to interview format. Participants were given thorough instructions on how to complete their daily diary after their interviews.

Phase 2: Diary. Participants were instructed to begin their first diary entry on the day of their interview, and complete their diary once every day for the next 21 days. They were told to complete their diaries toward the end of the day (for example, just before going to bed), or as late in the day as was convenient for them.

Participants were given the option of completing their diaries online or on paper, and all participants were given copies of the paper diaries as a back-up option. The vast majority of diaries (92%) were completed online. The diary website (administered through www.psychdata.com) electronically stamped each survey with the date and time at which it was completed, allowing me to verify

participant compliance. In cases where a participant completed multiple surveys in one day, all of that participant's diary data for that day were excluded. Paper diaries (which were intended for use by participants uncomfortable with computers or without internet access, see Appendix D) contained the same content as the online diaries. Participants were asked to return the daily surveys by mail over the course of their participation, and postmarks were examined for compliance.

I took several lengths to maximize participant compliance with the diary. First, to help participants remember to complete the diary each day, a computer program developed specifically for this project automatically emailed participants every day at a participant-designated time. The email contained the person's ID number and a link to the survey. Second, to provide additional incentive to complete the diary as often as possible, participants were awarded one raffle for each diary entry they completed, and participants who completed all 21 diary entries received a bonus of 10 raffle entries. Raffle prizes included an MP3 player and a GPS navigation device. Eligible participants completed an average of 18.82 diary entries, or 90% compliance rate.

Phase 3: Follow-up. One week after their last daily survey, participants were asked to complete follow-up questionnaires. These were again completed online, although participants had the option to complete paper follow-up questionnaires through the mail (but nobody chose to do so). Follow-up questionnaires were identical to baseline questionnaires, but excluded

demographic data and other variables not expected to change over time). Follow-up data are not relevant to the analyses presented here. After completing their follow-up questionnaires, participants were sent payment through the mail or awarded course credit.

IV. DATA ANALYSIS STRATEGY

Unless otherwise noted, all analyses were conducted using each of the following steps.

All data analyses were conducted using IBM SPSS Statistics 18.0.1 Mixed Methods. Most of the study hypotheses were tested using mixed effects modeling, specifically multi-level modeling (MLM), with daily reports of symptoms, rumination and hopelessness nested within-subjects. MLM offers several benefits over traditional data analysis approaches. Within-subjects designs substantially increase power, and MLM controls for the non-independence of nested effects and copes well with missing data.

Most analyses presented here are lagged, with the time-varying predictor variable temporally preceding the time-varying outcome variable. All predictor variables were centered with the grand mean. Time was included in initial models as a fixed effect, and was subsequently dropped when highly non-significant ($p > .2$) but was otherwise included. An unstructured convariance type

was specified for random effects, and a first-order auto-regressive covariance (AR[1]) type was specified to control for auto-correlation of residuals. All main effects of interest (not interactions) were initially included as fixed effects and as random effects, and random effects that were highly non-significant ($p > .2$) were dropped (see Nezlek, 2001) but kept as fixed effects.

In some cases, analyses failed to converge using this strategy. In that case, I took the following steps to increase the likelihood of convergence. First, I took the following measures, as recommended by Garson (2009): a) remove any variables with correlations near 1.0, b) increase maximum iterations, b) increase step-halvings, c) increase singularity tolerance value, d) increase scoring steps, and e) increase parameter convergence value. Second, if the model still did not converge, I changed the repeated covariance type from AR(1) to diagonal. Third, if the model still did not converge, it likely indicated that the model was attempting to estimate random effects that were very small (Garson, 2009; Nezlek, 2001). In that case, I undid the changes from steps #1 and #2 and then removed the smallest random effects (keeping the variable only as a fixed effect) until the model converged.

Time Frame of Symptom Assessment

Where possible, I tested hypotheses using both symptoms assessed at the moment of daily diary completion (momentary items; i.e., with the instructions "using the scale below, check the square that best describes how you are feeling *right now*") and symptoms assessed over the course of the day of diary

completion ("course of day" items; i.e., with the instructions "using the scale below, check the square that best describes how you have felt *over the course of the day today*"). Time frames of predictor variables always corresponded with outcome variables; in other words, when the predictor variable was momentary, the outcome was momentary.

Time Lags

Because little research has been conducted in this area, the time lags under which the hypothesized processes may unfold is unknown. In other words, it may be that anxious mood today predicts depressed mood tomorrow, or it may be that anxious mood experienced over the course of several days predicts depressed mood. Thus, I tested several time lags to determine the most appropriate. To do so, I began with a time lag of one day (depressed mood on day_t predicted by anxious mood on $day_{t-1)}$. I next tested a two-day lag, and then three day, until significance peaked and dropped. I chose the time lag with the peak significance rate as the "optimal" time lag. Once the optimal time lag was determined for a particular predictor and outcome, I used that time lag for the remaining analyses. Multiday lags were aggregated, so a two-day lagged predictor predicting an outcome on day_t would include a summation of variables on day_{t-1} and day_{t-2}. Time lags for mediators were taken from intermediate days (i.e., days occurring in between the predictor and outcome days). Where possible, mediators were assigned a time lag that followed the predictor but preceded the outcome.

Mood Components

Because mood states are heterogeneous constructs, in addition to broad constructs of anxious mood and depressed mood, I also examined worry, an additional component of anxiety central to GAD, as well as anhedonia, an additional component of depression.

Mediation

Hypothesized mediation effects were each subjected to Baron & Kenny's (1986) guidelines for determining mediation. In addition, the reduction in effect size was tested for significance using Sobel's (1982) test.

Comparing Lagged and Concurrent Effect Models

In addition to lagged analyses, as a preliminary analysis, concurrent effect models (i.e., with all predictor variables, the outcome, and any covariates all assessed on day *t*) were typically tested. This analysis examines whether changes in the outcome variable can be predicted from changes in the predictor(s), but does not offer information on temporal sequencing. This can be summarized in the following equation:

$$Y_{(t)} = a + b*X_{(t)} + c*\text{time}$$

where a is the intercept, b is the unstandardized coefficient, Y is the outcome variable, X is the predictor, and *c* is the unstandardized coefficient for time.

For most lagged analyses, where the outcome was measured on day *t*, I included only the main predictor variable(s) (lagged at *t-k*, where *k* is the

appropriate time lag determined as outlined above), time (if significant at $p < .20$), and any appropriate covariates also lagged at t-k. A simple version of this analysis (one predictor variable, no covariates) can be represented as the following function, which examines whether changes in the outcome variable predicted by previously occurring changes in the predictor variable.

$$Y_{(t)} = a + b*X_{(t-k)} + c*\text{time}$$

However, for a more conservative test, I also examined whether the lagged predictor (at t-k) would remain significant when controlling for concurrent model (i.e., controlling for the predictor on day t).

$$Y_{(t)} = a + b*X_{(t)} + c*X_{(t-k)} + d*\text{time}$$

Power Analyses

Power analyses focus on Hypothesis 1, because there is no accepted method of calculating power for indirect effects, as hypothesized in Hypothesis 2. Power for Hypothesis 1 was calculated using a Monte Carlo simulation with the Mplus program (Muthén & Muthén, 1998). Because there is limited research examining the daily relationship between depressed and anxious mood, the simulation used multiple estimated parameters. Depressed mood at t-1 predicting depressed mood at t was estimated at .50 and .80; anxious mood at t-1 predicting depressed mood at t was estimated at .20; the correlation between anxious and depressed mood at t-1 was estimated at .80 and .50; and the residual variance for depressed mood at t was estimated at .70 and .40. Each permutation of these estimated parameter values were used to generate two

hundred datasets. After generating the data, analyses were conducted examining the probability of predicting depression at *t* from anxiety at *t*-1, controlling for depression at *t*-1. In the current sample, with the originally planned 40 participants and 21 observations (840 "cases"), power was estimated at 1.00 even using the most conservative parameter estimates. Moreover, to further bolster power, I opted to further increase the sample size to 55, thus increasing power past this preliminary estimate.

III. RESULTS

Missing Data

Multilevel modeling is able to handle data that are missing at random (Fitzmaurice, Laird, & Ware, 2004). In the current dataset, missing a daily survey was not predicted by key study variables such as anxious mood, depressed mood, anxious rumination, or hopelessness on the prior day. This provides reasonable evidence that data were missing completely at random and therefore the missing data are ignorable (Fitzmaurice, et al., 2004; Howell, 2009).

Descriptive Data

Descriptive data for all variables are presented in Table 4.

Concurrent Association Between Anxious Mood and Depressed Mood

First, I examined whether participants tended to feel anxious on the same days that they felt depressed by running a multilevel model with depressed mood$_t$ as the outcome variable and anxious mood$_t$ as the predictor, also including time as a fixed effect and entering the intercept and anxious mood as random effects. As shown in Table 5, depressed and anxious moods assessed at the moment were highly concurrently associated (unstandardized effect size= .52, standard error= .04, t(46.07)= 12.53, p< .001). In addition, depressed and anxious moods assessed over the course of the day were also highly concurrently associated (unstandardized coefficient= .54, standard error= .04, t(44.67)= 13.13, p< .001, further results in Table 5).

Note that, as one would expect, these "momentary" and "course of day" variables were concurrently associated with each other (for depressed mood unstandardized estimate= .65, standard error= .04, t(50.81)= 15.97, p< .001; for anxious mood unstandardized estimate= .63, standard error= .04, t(50.63)= 16.21, p< .001; for hopelessness unstandardized estimate= .62, standard error= .04, t(46.83)= 15.42, p< .001).

Hypothesis 1: Anxious Mood Will Predict Later Depressed Mood

Time lag determination. My first step was to determine the optimal time lag for testing the association between anxious mood and later depressed mood. Once again, I tested a model using depressed mood today as the outcome variable and various lags of anxious mood as the predictor. I also included time as a fixed effect. For the purposes of these exploratory time lag analyses, I

entered only the intercept as a random effect. Results are listed in Table 6. Using the steps outlined in the Data Analyses Strategy section, I found that 1) anxious mood yesterday predicted depressed mood today (one-day lag; unstandardized coefficient= .09, standard error= .03, $t(884.77)= 2.53$, $p= .012$); 2) the sum of anxious mood over days $t-1$ and $t-2$ predicted depressed mood on day t (two-day lag) at apparently higher magnitude and significance than for the one day lag (unstandardized coefficient= .25, standard error= .05, $t(397.66)= 5.57$, $p< .001$), although the differences in effect sizes were not subjected to significance testing; 3) using a three-day lag (i.e., Σ [anxious $mood_{t-3,}$ anxious $mood_{t-2}$, anxious $mood_{t-1}$] predicting depressed $mood_t$) yielded still significant and apparently slightly smaller effect sizes (unstandardized coefficient= .25, standard error= .05, $t(277.73)= 4.48$, $p< .001$; again, these differences in effect size were not tested for significance, and also note that the unstandardized effect sizes were identical for the three-day lag as for the two-day lag and that the t and p statistic values may simply reflect greater sample size for the two-day lag); 4) using a four-day time lag generated still significant but again apparent smaller effect sizes (unstandardized coefficient= .10, standard error= .03, $t(350.48)= 3.81$, $p< .001$). Given the downward trend of effect sizes, the two-day time lag was determined to be optimal (although again note that the three-day lag may be equally appropriate).

As an additional test of time lag appropriateness, I simultaneously entered anxious mood (non-aggregated) on days $t-1$, $t-2$, $t-3$, and $t-4$ (along with time)

as fixed effect predictors, with depressed mood on day *t* as the outcome variable. As shown in Table 7, in this analysis, only the two-day time lag was a significant predictor of depressed mood, again suggesting that the optimal time lag to examine anxious mood predicting depressed mood in this dataset is two days. A two-day time lag was therefore chosen for remaining analyses.

Thus, supporting Hypothesis 1, fluctuations in anxious mood do predict fluctuations in later depressed mood, with a two-day time lag yielding results of highest magnitude. Anxious mood was not significant as a random effect in this analysis, and the final analysis, including anxious mood and time as fixed predictors and only the intercept as a random predictor, yielded an unstandardized coefficient of .25 and a standard error= .05 ($t(397.66)= 5.57$, $p< .001$).

Given the high concurrent association between anxious mood and depressed mood, it is reasonable to question whether the association between anxious mood and later depressed mood is better accounted for by the association between anxious mood and concurrent depressed mood. To test this hypothesis (and thereby provide a more conservative test of my original hypothesis), I next re-ran the prior analysis (with a two-day lag) controlling for concurrent anxious mood. Specifically, depressed mood today was the outcome, and a) anxious mood two days ago, b) anxious mood today, and c) time were all entered as fixed effect predictors (both anxious mood today and previous anxious mood were initially entered as a random effects; anxious mood today

was significant as a random effect and was retained, but previous anxious mood was highly non-significant and was subsequently dropped as a random effect). Full results are displayed in Table 8. Results showed that anxious mood was a significant predictor of depressed mood both as a lagged predictor and as a concurrent predictor. This provides much more powerful evidence that fluctuations in anxious mood predict fluctuations in later depressed mood. Note that although the results presented in Table 8 reflect anxious mood as a non-aggregated predictor, results were similar when using the two-day aggregated variables instead (lagged aggregated predictor: unstandardized coefficient= .08, standard error= .04, $t(507.85)= 2.06$, $p= .040$; concurrent predictor: unstandardized coefficient= .52, standard error= .03, $t(708.65)= 18.31$, $p< .001$).

I next conducted the same analyses using the "course of the day" mood variables rather than the "right now" variables. The results were relatively similar. Anxious mood no longer predicted depressed mood using a one day lag (unstandardized coefficient= .06, standard error= .03, $t(875.85)= 1.60$, $p= .11$), but anxious mood significantly predicted depressed mood over all subsequent time lags, including using a two-day lag (unstandardized coefficient= .22, standard error= .05, $t(379.62)= 4.38$, $p< .001$), a three-day lag (unstandardized coefficient= .27, standard error= 0.06, $t(256.86)= 4.42$, $p< .001$), and a four-day time lag (unstandardized coefficient= .10, standard error= .03, $t(332.47)= 3.35$, $p= .001$). Because the effect size pattern with the increased time lags pointed less clearly to a single time lag, I again simultaneously entered anxious mood

(non-aggregated) on days *t*−1, *t*−2, *t*−3, *t*−4, and *t*-5 (along with time) as fixed effect predictors, with depressed mood on day *t* as the outcome, as a further test of optimal time lag. Once again, the two-day lag yielded the strongest effect.

When controlling for random effects and time, the two-day lag for the "course of day" variables yielded an unstandardized effect size of .23, a standard error of .06, $t(37.26) = 3.65$, $p = .001$.

Test of reverse hypothesis: Depressed mood predicting later anxious mood. To provide a still more powerful test of Hypothesis 1, I tested the reverse effect, examining depressed mood as a predictor of later anxious mood. Again, I tested this effect using multiple time lags. First, using a one day time lag (i.e., regressing anxious mood today on depressed mood yesterday and time), depressed mood yesterday was not a significant predictor of anxious mood today (unstandardized coefficient= -.02, standard error= .04, $t(934.84) = -0.65$, *ns*). Next, using a two day aggregated time lag, fluctuations in depressed mood *did* predict fluctuations in later anxious mood (understandardized coefficient= .12, standard error= .06, $t(427.07) = 2.14$, $p = .033$). However, a few caveats should be noted: 1) this analysis would not converge when including depressed mood as a random predictor, and thus depressed mood was included only as a fixed predictor, potentially inflating effects; 2) when using the "course of day" variables rather than the momentary variables, depressed mood was not a predictor of later anxious mood at any time lag; 3) when controlling for the concurrent effect model, as described above (i.e., controlling for depressed mood concurrent with

the outcome variable), previous depression was no longer a significant predictor of anxious mood (understandardized coefficient= -.02, standard error= .05, *t*(501.78)= -0.33, *p*= .74), while concurrent depressed mood remained a strong predictor of anxious mood (understandardized coefficient= .65, standard error= .03, *t*(686.23)= 19.26, *p*< .001); 4) longer (three- and four-day) time lags were not significant for depressed mood predicting anxious mood. Taken collectively, this evidence suggests that fluctuations in depressed mood may weakly predict fluctuations in anxious mood, but this effect is not as robust as found with anxious mood predicting later depressed mood, and may be better accounted for by the strong concurrent association between anxious mood and depressed mood.

Different components of anxious and depressed mood. To examine whether varying components of depressed and anxious mood yield differing temporal patterns, I examined worry as an additional component of GAD and anhedonia as an additional symptom of depression. Please note that anhedonia and worry were only assessed over the course of the day, so the corresponding course-of-day anxious and depressed mood variables were also used for these analyses. Also note that there were strong concurrent associations between both anxious mood and worry (unstandardized coefficient= .55, standard error= .04, *t*(57.94)= 13.42) and depressed mood and anhedonia (unstandardized coefficient= .55, standard error= .03, *t*(47.83)= 16.08) both *p*s < .001, so the results presented above may better account for the following results.

Table 9 lists concurrent associations between worry, anhedonia, and the broad indicators of depressed and anxious mood. First, worry was concurrently associated with both depressed mood (unstandardized coefficient= .44, standard error= .04, $t(50.57)= 10.93$, $p< .001$) and anhedonia (unstandardized coefficient= .43, standard error= .05, $t(53.82)= 9.44$, $p < .001$), and both remained significant when entered as simultaneous fixed (and random) effects (depressed mood: unstandardized coefficient= .48, standard error= .05, $t(47.22)= 9.66$, $p < .001$; anhedonia: unstandardized coefficient= .14, standard error= .05, $t(38.37)= 3.50$, $p < .001$), suggesting they each independently predict anxious mood. In turn, anhedonia was associated with both worry (as stated above) and anxious mood (unstandardized coefficient= .43, standard error= .04, $t(45.52)= 10.91$, $p < .001$), and worry and anxious mood both predicted anhedonia when entered as simultaneous predictors (worry: unstandardized coefficient= .30, standard error= .05, $t(47.45)= 6.49$, $p < .001$; anxious mood: unstandardized coefficient= .25, standard error= .04, $t(41.74)= 6.12$, $p < .001$).

Second, I looked at lagged worry and anhedonia. For simplicity, I used the two-day time lag suggested by previous analyses. Results are presented in Table 10. First, worry predicted later anhedonia (final results of two day lag: unstandardized coefficient= .18, standard error= .05, $t(434.62)= 3.57$, $p < .001$). Worry also predicted later depressed mood (final results of two day lag for predicting depressed mood: coefficient= .22, standard error= .05, $t(406.17)= 4.78$, $p < .001$).

Third, I looked at the opposite direction of effect. Anhedonia was not a significant predictor of later anxious mood (unstandardized coefficient= .06, standard error= .06, *t*(469.91)= 1.07, *ns*), nor of later worry (unstandardized coefficient= .11, standard error= .06, *t*(444.29)= 1.83, *ns*).

Hypothesis 2: Fluctuations in Anxious Mood Will Predict Later Daily a) Anxiety-Focused Rumination and b) Hopelessness

Because the two-day time lag was found to be optimal for anxious mood predicting depressed mood, I chose an intermediate time lag (i.e., following the predictor variable but preceding the outcome variable; here, hopelessness and rumination were lagged one day after anxious mood and one day before depressed mood) for these and all following mediation analyses.

Anxiety-Focused Rumination. Elevated anxious mood on day *t*-1 (momentarily assessed) predicted elevated ruminating about anxious mood on day *t* (unstandardized coefficient= .09, standard error= .04, *t*(52.15)= 2.02, *p*= .049). Elevated anxious mood over the course of the day was not a significant predictor of elevated rumination.

Hopelessness. Anxious mood on day *t*-1 was not a significant predictor of hopelessness on day *t* (unstandardized coefficient= .07, standard error= .04, *t*(62.32)= 1.73, *p*= .089). Anxious mood over the course of the day also did not predict increases in hopelessness (unstandardized coefficient= .08, standard error= .05, *t*(50.29)= 1.63, *p*= .110).

Hypothesis 3: Fluctuations in a) Daily Symptom-Focused Rumination and b) Daily Hopelessness Will Predict Later Fluctuations in Depressed Mood

Anxiety-focused rumination. Prior fluctuations in anxious rumination on day *t*-1 predicted recent fluctuations in depressed mood on day *t* although only when depressed mood was assessed momentarily (unstandardized coefficient= .12, standard error= .04, $t(41.19)= 2.86$, $p= .007$), and not over the course of the day (unstandardized coefficient= .04, standard error= .03, $t(887.48)= 1.19$, *ns*).

Hopelessness. Elevated hopelessness levels on day *t*-1 predicted depressed mood over the course of day *t* (unstandardized coefficient= .14, standard error= .05, $t(52.60)= 3.05$, $p=. 004$, but not momentarily-assessed depressed mood on day *t* (unstandardized coefficient= .06, standard error= .04, $t(50.62)= 1.36$, *ns*).

Hypothesis 4: The Daily Association Between Anxious Mood and Depressed Mood Will Be Mediated by a) Daily Symptom-Focused Rumination and b) Daily Hopelessness

As described in the data analysis strategy section, I used Baron & Kenny's (1986) steps for testing mediation. The first step is to demonstrate that the predictor (anxious mood) predicts the outcome (depressed mood), although note that several analysts have argued that this step is not always necessary (MacKinnon, Fairchild, & Fritz, 2007). As previously noted, anxious mood predicts later depressed mood, most strongly over a two-day lag, meeting this first step toward confirming mediation. The second step is to demonstrate that

the predictor variable predicts the mediator. The third step is to test whether the mediator affects the outcome variable, controlling for the predictor variable. Finally, the last step is to show that controlling for the mediator eliminates (complete mediation) or significantly reduces (partial mediation) the association between the predictor and the outcome variables. Significance testing of the reduction in the predictor-outcome association was performed with a Sobel's test (Sobel, 1982). Steps 2-4 and Sobel's test results are outlined for each mediator below.

Anxiety-focused rumination. Results for these mediation analyses are outlined in Table 11. As noted above, symptom-focused rumination a) was predicted by anxious mood, meeting Step 2, and b) predicted depressed mood, supporting Step 3. Furthermore, anxious rumination predicted depressed mood when controlling for anxious mood (unstandardized coefficient= .10, standard error= .04, $t(27.88)= 2.35$, $p= .026$), fully meeting Step 3. Step 4 was tested by entering anxious mood on day $t-2$ and anxious rumination on day $t-1$ as simultaneous fixed predictors of depressed mood on day t (analyses used only momentarily-assessed mood, as mood assessed over the course of each day was not a significant predictor or outcome of rumination). As shown in Table 11, results showed that both anxious mood and rumination were significant predictors in this analysis, but there was no apparent reduction in the coefficient for anxious mood, and mediation was not significant according to a Sobel's test (Sobel's test statistic= .20, $p= 0.838$).

Hopelessness. As outlined above, hopelessness was not predicted by anxious mood nor did it predict depressed mood, so Baron and Kenny's second and third steps were not satisfied. Thus, hopelessness does not meet criteria as a mediator.

Alternate Hypothesis: The Daily Association Between Anxious Mood and Depressed Mood Will Be Moderated by a) Daily Symptom-Focused Rumination and b) Daily Hopelessness

Anxiety-focused rumination. First, anxious mood and rumination, each aggregated over a concurrent two-day lag, were entered as fixed effects, along with their interaction and time. All main effects and time were also entered as random effects initially. This analysis led to problems with convergence, which were not solved using the initial steps outlined in the planned data strategy. By removing the smallest random effects, the analysis did converge, but because main effects are typically preserved as random effects in interaction analyses, these results should be interpreted with caution. The analysis showed that anxious mood and rumination about anxious mood did not significantly interact.

It might make more sense that anxious rumination would moderate the concurrent association between anxious mood and depressed mood. In other words, anxious mood may be more strongly associated with depressed mood on days when people ruminate about their anxious mood, perhaps because the lag between anxious mood and depressed may be short enough to be lost in analyses using a one-day lag. To examine this possibility, depressed mood on

day *t* was included as the outcome variable, with time and concurrent anxious mood, anxious rumination, and the interaction of anxious mood and anxious rumination (all also on day *t*) were all entered as fixed predictors, with time and the main effects of anxious mood and anxious rumination included as random effects. The interaction term was significant (unstandardized coefficient= .03, standard error= .01, $t(305.39)= 3.39$, $p = .001$), as were the main effects of anxious mood (unstandardized coefficient= .43, standard error= .04, $t(54.92)= 10.62$, $p < .001$), and anxious rumination (unstandardized coefficient= .12, standard error= .03, $t(42.03)= 3.39$, $p = .001$). Full results are presented in Table 12. To interpret this significant interaction, simple slope tests were conducted according to the procedures of Aiken and West (1991). On days when anxious rumination was high (one standard deviation above the mean), elevated anxious mood was more strongly associated with elevated concurrent depressed mood (unstandardized coefficient=.52, standard error= .04, $t(64.14)= 11.90$, effect size $r= .83$, $p< .001$) compared to low levels of anxious rumination (defined as one standard deviation below the mean; unstandardized coefficient= .35, standard error= .03, $t(114.72)= 6.81$, effect size $r= .54$, $p< .001$). Of course, removing the temporal lag between anxious mood and depressed mood raises questions about direction of causality, making this a weaker test of my model.

Hopelessness. First, hopelessness and anxious mood, each aggregated over a two-day lag, were entered as fixed predictors along with their interaction (and time), with depressed mood as an outcome. Both main effects were also

initially entered as random effects; however, this led to problems with convergence. Altering convergence criteria and using different covariance structures did not help with convergence. The smaller random effect (hopelessness) was removed; again, these results should be interpreted with caution. Hopelessness was not a significant moderator of the association between anxious mood and later depressed mood in this analysis.

Next, as with the above analyses, hopelessness was tested as a moderator of the concurrent association between anxious mood and depressed mood. Anxious mood $_t$, hopelessness$_t$, and their interaction (along with time) were entered as fixed effect predictors of depressed mood, with both main effects also entered as random effects. The interaction term was not significant.

V. DISCUSSION

This study used daily diary methods to examine the daily temporal patterns between anxious mood and depressed mood, and tested several potential mechanisms of symptom co-occurrence as mediators and moderators. Several important findings were revealed. First, anxious mood showed a significant concurrent association with depressed mood; in other words, participants tended to be anxious on the same days they were depressed. On the one hand, this finding seems somewhat predictable, given that anxiety and

depression measures are so highly and consistently correlated. On the other hand, it provides some basic but important temporal information about the experience of mood and anxiety symptoms that was previously missing from the literature: that anxiety and depression symptoms seem to ebb and flow in concert.

This underscores a general note about comorbidity research: comorbidity and co-occurrence have generally been conceptualized as between-subjects phenomenon. In other words, research has largely focused on determining whether individuals with one disorder are at elevated risk for another. This is an important question, but another, largely unexplored consideration is whether experiencing a disorder or symptom at a particular time is associated with higher risk of another type of symptom at that particular time—or, stated otherwise, within-subjects co-occurrence or comorbidity. These results support the existence of within-subjects symptom co-occurrence in the case of anxious and depressed mood among adults with GAD. Of course, these results may also be inflated by systematic error such as shared method variance.

Next, a major aim of this study was to clarify the temporal associations between anxious and depressed mood. Specifically, I hypothesized that the sequence typically found in studies looking at disorders over the course of months and years (wherein anxiety tends to precede depression) would replicate when looking at symptoms assessed over the course of days. This was indeed the case. As predicted, fluctuations in daily anxious mood predicted later

fluctuations in depressed mood. This sequence was found in every time lag tested and was maintained when subjected to the more conservative test of controlling for the concurrent effect model. Adding to this evidence, the pattern was also replicated when using variables reflecting anxious mood over the course of the day, rather than anxious mood assessed at the moment of diary completion.

I also tested the reverse direction of effect, with depressed mood predicting later anxious mood, to rule it out as an alternative. Notably, over a two-day lag and using the momentarily assessed symptom variables, fluctuations in depressed mood did significantly predict fluctuations in later anxious mood. On the one hand, this weakens my conclusions, because it opens the possibility that anxious and depressed mood are simply difficult to differentiate on a daily basis, rather than having specific predictive power over each other. However, the support for depressed mood as a predictor of later anxious mood was substantially weaker than the support for the opposite direction of effect, for several reasons. First, depressed mood did not predict later anxious mood over any time lag tested other than two days. Second, when using the more conservative effect of controlling for concurrent depressed mood, prior depressed mood was no longer a significant predictor of later anxious mood. This implies that the association between fluctuations in depressed mood and later fluctuations in anxious mood may be better explained by the high association between anxious mood and concurrent depressed mood. Third, only

momentarily-assessed depressed mood predicted later anxious mood; when mood was assessed over the course of the day, depressed mood was not a significant predictor of later anxious mood over any time lag tested. In comparison, as described previously, anxious mood predicted later depressed mood on a much more consistent basis. Because of these issues, the data suggest that anxious mood more robustly predicts depressed mood than vice versa, although conclusions should be interpreted with the requisite caution. Further replication is needed to more clearly delineate the temporal sequencing of daily anxious and depressed mood, but this evidence makes at least a preliminary case that anxious mood tends to come first.

A component of these analyses attempted to determine the optimal time lag over which anxious mood leads to depressed mood, something that to my knowledge has not been explored before. A two-day lag emerged as the strongest time lag in these analyses. There is no real theoretical basis to support one time lag over another, and any conjecture about why the two-day lag was most supported would purely be post-hoc speculation. That said, it seems likely that there is some recency effect at play, in which too long of a lag would lead to less significant effect sizes. On the other hand, anxiety experienced over too short of a time span might not be chronic enough to spur the mechanisms that lead to depressed mood. It is possible that the two-day lag optimally balanced these opposing processes. Replication of these findings in other datasets will be an important next step. It is especially intriguing that even the one-day effect

was rendered non-significant when controlling for the two-day effect, suggesting a possible “sleeper effect,” where the consequences of anxious mood are not immediately apparent, but develop over the course of time.

Depressed mood and anxious mood seem like reasonable starting points for exploring the temporal associations of symptoms of depression and anxiety. They are each a cardinal symptom of their disorders, and may be easier for laypeople to recognize compared to more specific symptoms. However, there are also drawbacks to defining mood states so broadly. The degree to which these broad indicators tap specific aspects of depressed and anxious mood versus shared components is somewhat unclear (Burns & Eidelson, 1998; Clark & Watson, 1991; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995), and to the extent to which they do tap non-specific elements, questions are raised about their discriminant validity. In order to get some idea of the generalizability of this temporal pattern to other types of symptoms, I examined other components of depression and anxiety. Specifically, I tested anhedonia (a cardinal symptom of major depression thought to be specific to depression; Clark & Watson, 1991; Joiner, 1996; Joiner, Brown, & Metalsky, 2003; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) and worry (a central component of generalized anxiety disorder; American Psychiatric Association, 1994; to the extent that proposed revisions of the DSM have suggested renaming GAD "Generalized Anxiety and Worry Disorder;" see American Psychiatric Association, 2010). I found that these symptoms yielded similar temporal

patterns to broadly defined anxious and depressed moods. This adds to evidence that anxious mood generally precedes depressed mood, even when anxious and depressed mood are defined in different and more specific ways.

Although it is only one component of this study, the finding that anxious mood tends to precede depressed mood on a daily basis makes an extremely important contribution to comorbidity research. Previous studies have rather consistently shown that anxiety disorders, over the course of months and years, tend to precede depression (Burke, et al., 2005; Cole, et al., 1998; de Graaf, et al., 2003; Essau, 2003; Kovacs, et al., 1988; Lewinsohn, et al., 1997; Orvaschel, et al., 1995; Wittchen, et al., 2000), and this piece of evidence has prompted several researchers to suggest that anxiety acts as a causal risk factor for depression (e.g., Lewinsohn, et al., 1997; Wittchen, et al., 2003). Some, however, have raised the alternative explanation that anxiety disorders may simply be markers of more fundamental risk factors for depression, and that their earlier emergence may simply represent differences in course (Kessler, et al., 2007). This study's results suggest that the temporal precedence of anxiety symptoms over depression can be replicated in a time frame too short to be explained by differences in course. These results also replicate and extend the findings of Swendsen (1998), who showed that anxious mood tends to precede depressed mood in an experience sampling study, but explored these associations in much less detail. For example, Swendsen's (1998) study did not

explore different time lags, examine different components of mood, nor test mediators or moderators.

Furthermore, these findings may also suggest that the mechanisms that link depressed mood and anxious mood—and perhaps their disorder counterparts—occur over a briefer period of time than longer term longitudinal studies would be able to capture. This implies that shorter time periods may be more appropriate in comorbidity and co-occurrence research. It is even possible that the day-long minimum lags in this study were too long in some cases, and it may be useful to use experience sampling methods to examine mood changes over even shorter intervals (Swendsen, 1998).

A potential limitation of this study is the use of simple, face-valid questions to assess daily mood. On the one hand, this helped keep the diary short (which likely helped keep compliance high; Morren, et al., 2009). On the other hand, some may question the validity of these items in comparison to longer, systematically validated measures. Note that numerous previous diary studies have assessed mood using either single-word items, visual analog scales based on single word descriptors, or scales based on single-adjective descriptors (e.g., Brinker & Dozois, 2009; Mor, et al., 2010; Swendsen, 1998), and that no mood scales currently exist that have been explicitly validated for diary use (Ebner-Priemer & Trull, 2009). It remains unclear, however, if these simple assessments of anxious and depressed mood would show the same differentiation or factor structure as more complex measures. Future research should explore whether

daily assessment of depression and anxiety using measures with demonstrated discriminant validity would yield similar results, and the findings from the current study should be considered somewhat preliminary. Despite this limitation, the novelty of this study makes it an important contribution to the literature.

I proposed that anxiety-focused rumination would act as a mediator or moderator of the association between anxious mood and later depressed mood. Anxious rumination was not significant as a mediator. It was, however, supported as a moderator of the association between anxious and depressed mood, where concurrent anxious and depressed mood were more strongly linked on days when people ruminated about their anxiety. This suggests that anxious rumination may be better conceptualized as a diathesis for the development of comorbidity that interacts with the "stress" of anxiety. Converging with these findings, in a recent five-month multi-wave study of adolescents, Hankin (2008) showed that baseline rumination levels interacted with prospective fluctuations in anxious arousal to predict increases in depression.

This finding could also be interpreted as anxious mood moderating the association between anxious rumination and depressed mood; in other words, anxious rumination was a stronger predictor of depressed mood on days when anxious mood was elevated. Rumination may only have an impact on mood when the person effectively has something about which to ruminate. In the case of anxious rumination, this may be the experience of anxious mood; for depressive rumination, it may be dysphoric mood. Along these lines,

experimental research show that depressive rumination tends to generate negative autobiographical memories, elicit negative and pessimistic thinking, and increase depressive symptoms among dysphoric but not non-dysphoric individuals (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Lyubomirsky & Nolen-Hoeksema, 1995; Morrow & Nolen-Hoeksema, 1990). Thus, rumination seems to have its most potent effects among those who effectively have material about which to ruminate, such as dysphoric mood in the case of depressive rumination and anxious mood in the case of anxious rumination. On the other hand, because the significant moderation finding was generated using concurrently assessed anxious mood, depressed mood, and rumination, the directions of these effects cannot be clearly differentiated. In addition, because these analyses did not also examine depressive rumination, it is unclear at this point if anxious rumination has specific associations to mood, or if is better subsumed by the pre-existing construct of depressive rumination.

Unlike anxious rumination, hopelessness was neither supported as a mediator nor a moderator of the association between anxious mood and later (or concurrent) depressed mood. However, a potential explanation for this would be that hopelessness operates as a symptom co-occurrence mechanism over a different time frame than what was tested here. For example, perhaps anxious mood leads to hopelessness over a longer time frame, as feeling anxious for just one day may not be sufficient to spur hopelessness. I purposefully made a priori decisions about the specific analyses that were conducted, because the high

number of analyses produces the potential for Type I errors. A one-day lag was tested for both hopelessness and rumination because the two-day lag was shown to be optimal for the association between anxious mood and later depressed mood, and a one-day lag for the potential mediators (so that they occurred after the predictor variable but before the outcome variable) provided the strongest test of mediation. However, future research should examine this issue of temporal lags and mediators more closely. It may also be that anxiety only leads to hopelessness under certain circumstances (e.g., cognitive appraisal of anxiety symptoms, anxiety sensitivity, meta-worry, etc.), and future studies should consider examining possible moderated mediators. Also, one difference between rumination and hopelessness in this study is that rumination was assessed specifically as a reaction to anxiety, whereas hopelessness was assessed more broadly. It is unclear if hopelessness as a specific reaction to anxiety symptoms taps a slightly different construct from general hopelessness mood.

In addition to exploring the vulnerability factors and mechanisms that elevate risk for symptom co-occurrence, it may be worthwhile to explore protective factors that help to prevent the progression of anxiety into depression. For example, in the RAQ pilot study (Starr & Davila, 2008a), I examined distraction as a moderator of the association between anxiety and depressive symptoms. In her original papers, Nolen-Hoeksema and colleagues identified distraction as an alternative to rumination with the reverse effect (e.g.,

Lyubomirsky & Nolen-Hoeksema, 1993; but also see more recently Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), although note that distraction is not conceptualized as necessarily being adaptive, as it can also be accomplished through non-adaptive means such as substance abuse, self-harm, or behavioral avoidance. In the RAQ pilot study, participants who reported distracting themselves when feeling anxious showed lower associations between anxiety and depressive symptoms, compared to participants who did not distract. This is just one of many potential protective factors. For example, perhaps anxious individuals with greater social support are less likely to develop secondary depressive symptoms.

In this study, I limited my exploration to cognitive mechanisms of the co-occurrence of anxiety and depression. Further research should explore other potential mechanisms as well, including behavioral aspects of anxiety and depression. Interpersonal dysfunction can be spurred by anxiety and is predictive of depressive symptoms (e.g., Alden & Taylor, 2004; Borkovec, Ray, & Staber, 1998; Darcy, Davila, & Beck, 2005; Davila & Beck, 2002; Davila, Karney, Hall, & Bradbury, 2003; Joiner & Metalsky, 2001; Meleshko & Alden, 1993; Segrin & Dillard, 1992; although note that much of the research linking anxiety to interpersonal dysfunction has specifically focused on social anxiety). Furthermore, interpersonal dysfunction (specifically the social anxiety-related interpersonal style of avoiding emotional expression) has been previously supported as a mediator of the association between baseline social anxiety and

depressive symptoms one year later (Grant, et al., 2007). Interpersonal dysfunction should be further examined as a potential mediator of co-occurrence between anxiety and depression. In addition, it may be worthwhile to examine behavioral and experiential avoidance as further mediators. Avoidance is a trademark of all anxiety disorders, and it can deprive anxious individuals from gratifying life experiences and hamper self-efficacy. Thus, it seems logical that greater engagement in avoidance may put anxious individuals at risk of developing co-occurring depressive symptoms, although ironically, as described previously, distraction, which could be arguably labeled as a proxy for avoidance, was actually associated with reduced depressive symptoms among anxious individuals). A gain, this may be an important inquiry for future work.

This study is likely the first to look at the temporal sequencing of daily anxious and depressed mood with such precision. Still, it may be useful to look further at daily symptoms of anxiety and depression in expanded detail. For example, numerous researchers have examined the factor structure of anxiety and depression on a between-subjects basis (e.g., Marshall, Sherbourne, Meredith, Camp, & Hays, 2003; Mineka, et al., 1998; Watson, Clark, et al., 1995). It is unclear, however, whether this structure will replicate on a within-subjects basis. For example, the DSM-IV (American Psychiatric Association, 1994) is a famous attempt to classify symptoms into separate categories based on the degree to which these symptoms are presumed to co-occur in nature. Would the symptoms of individual DSM-IV disorders fall into similar categories when

assessed on a daily basis? For example, amongst individuals with multiple comorbid disorders, would one symptom of (for example) post-traumatic stress disorder (PTSD) be more likely to occur on the same day as other symptoms of PTSD? Future work using diary methods may be able to answer this and similar questions.

The current study used a sample of participants with GAD. This particular disorder shows perhaps the highest associations with depression of any anxiety disorder (Kessler, Chiu, et al., 2005), to the extent that some have argued that it would be better classified as a mood disorder (e.g., Watson, 2005), although others have protested this idea (see Mennin, et al., 2008). This study takes the novel approach of examining how symptoms interact within GAD and co-occurring depressive symptoms, rather than focusing on the overall disorders' likenesses and differences. However, looking specifically at GAD may also limit conclusions. First, one recent study challenged the notion that all anxiety disorders typically temporally precede depression, showing that in the case of GAD, patterns of MDD preceding GAD and of GAD and MDD emerging simultaneously were equally common (Moffitt, Harrington, et al., 2007). Second, in an important caveat, the defining feature of GAD is worry, and several researchers have noted substantial construct overlap between worry and rumination. Worry is typically considered future focused and has been more strongly linked to anxiety than depression, whereas rumination is typically considered present- or past-focused and has been strongly linked to depressive

symptoms; overall, research suggests that these are related but distinct constructs (Fresco, Frankel, Mennin, Turk, & Heimberg, 2002; McLaughlin, Borkovec, & Sibrava, 2007; Muris, Roelofs, Meesters, & Boomsma, 2004; Nolen-Hoeksema, et al., 2008; Watkins, Moulds, & Mackintosh, 2005; although note that most previous studies have focused on depressive rumination). However, given their high overlap, it may be important to more closely differentiate worry and rumination as mechanisms versus symptoms of GAD and depression.

This also begs the question of how results may differ if other anxiety disorders had been explored instead of GAD. All anxiety disorders are associated with depression (Kessler, Chiu, et al., 2005), but it may be that mechanisms of mood co-occurrence differ according to the type of anxiety experienced. For example, social anxiety may lead to more interpersonally-focused mechanisms of comorbidity, such as negative interpersonal styles, interpersonal conflict, relationship dependency, poor interpersonal competence, inadequate communication skills, and difficulty building and maintaining high-quality relationships (Alden & Taylor, 2004; Darcy, et al., 2005; Davila & Beck, 2002; Grant, et al., 2007; Starr & Davila, 2008b; Wenzel, Graff-Dolezal, Macho, & Brendle, 2005). In contrast, other anxiety disorders may lead to depression through different mechanisms. The extent to which symptom co-occurrence mechanisms are common across anxiety disorders versus specific to each individual disorder needs further investigation.

From a broader standpoint, it might be important to explore the extent to which this model applies to comorbidity between other psychological disorders. For example, substance use disorders are strongly associated with a number of DSM-IV disorders (Kessler, Chiu, et al., 2005). It is possible that substance use disorders can be substituted for depression as the "outcome" in my model. For example, perhaps to the extent that individuals respond to their distress with rumination and/or hopelessness, they may use substances as an attempt to "check out" (or distance themselves) from these feelings and thoughts. Similarly, other symptoms and behaviors that have been conceptualized as maladaptive emotion regulation devices, such as binge eating or self-harm behaviors (Gratz & Roemer, 2008; Whiteside, et al., 2007), may also follow on similar pathways from other forms of co-occurring distress (note, for example, that both self-harm and binge eating have been associated with rumination; Nolen-Hoeksema, et al., 2008), while perhaps also operating via mechanisms unique to their specific disorders. In addition to different "outcome" disorders, this model may also apply to different "predictor" disorders, including a range of potential options such as externalizing disorders, eating disorders, and so on. Some disorders, however, may show bidirectional and reciprocal associations, so it is important to not view comorbidity models in an overly simplistic manner.

Furthermore, it may be important to replicate these findings in non-clinical samples. Including only highly anxious participants in this sample could have led to issues of restricted range. Testing these ideas in normative samples, or

perhaps in samples at elevated risk for the development of anxiety and depression problems (e.g., children of depressed parents, high-stress samples, individuals with high cognitive vulnerabilities) may lead to differing results.

The results of this study may have important clinical implications. Comorbidity is associated with worse treatment outcomes (Ledley, et al., 2005; Young, et al., 2006), perhaps stemming from a lack of understanding of its etiological underpinnings. Although some treatments have been specifically developed for comorbid depression-anxiety (Kush, 2004), most treat the disorders as independently co-occurring symptoms, without considering how disorders may be etiologically related. If anxious mood causes depressed mood, perhaps focusing treatment efforts on anxiety symptoms would help reduce both existing anxious *and* depressed symptoms. Similarly, if anxiety disorders act as causal risk factors for later depression, it may imply that treating anxiety disorders early prevents their progression to depression. Along these lines, Kessler, Merikangas, and Wang (2007) noted that among individuals with prior panic disorder in the National Comorbidity Study-Replication, those who received treatment for panic were at lower risk for subsequent major depression (Goodwin & Olfson, 2001). Kessler et al. (2007) point out that this pattern is counter to what would be expected if there was a non-causal relationship between anxiety and MDD, as in that case you might expect treatment-seeking to act as a marker for greater symptom severity. More research should focus on treatment of

anxiety disorders when they first emerge (usually in childhood, Kessler, Berglund, et al., 2005) as an effort toward long-term prevention of depression.

Similarly, if anxiety activates depressogenic processes, treatment efforts with anxious clients should target these mechanisms (such as rumination and tendency to make hopeless inferences). This may both ameliorate existing comorbid depression and, among non-depressed clients, prevent the development of depressive symptoms. Also, several newer, empirically-supported treatments (such as acceptance and commitment therapy and mindfulness-based approaches; Eifert & Forsyth, 2005; Hayes, Luoma, Bond, Masuda, & Lillis, 2006) have emphasized acceptance rather than control of psychological symptoms. In line with this philosophy, to the extent that depressed mood results from hopelessness and rumination about anxiety symptoms, accepting anxious symptoms rather than viewing them as stressful and uncontrollable may prevent the development of comorbid depression.

It may also be interesting to look naturalistically at changes in and associations between depressed and anxious mood over time during treatment. For example, do depressed mood and anxious mood decrease over similar trajectories following treatment? Do decreases in anxious mood prompt subsequent decreases in depressed mood, or does anxious mood remain stable even when depressed mood decreases? Would different types of treatments (e.g., cognitive-behavioral therapy, acceptance-based therapy, psychopharmological treatments, etc.) produce differing trajectories of change?

Exploring how specific symptoms change when they are targeted through intervention would both extend current findings and create a more finite understanding of treatment response.

Ultimately, this study furthers our understanding of the co-occurrence of anxious and depressed mood, both by describing its phenomenological and dynamic nature and by examining several potential mechanisms. Further research attention is needed to better understand the nature of co-occurrence and comorbidity.

Table 1.

Number of Participants (Percentage of Sample) with Comoribid Unipolar Mood or Anxiety Disorders or Subthreshold Symptoms

	Current				Lifetime			
	No Symptoms	Minor Symptoms	Moderate Symptoms	DSM-IV Criteria	No Symptoms	Minor Symptoms	Moderate Symptoms	DSM-IV Criteria
Unipolar Mood Disorders[a]								
Any	9 (16.4%)	3 (5.5%)	15 (27.3%)	27 (49.1%)	0 (0.0%)	1 (1.8%)	8 (14.5%)	46 (83.6%)
Major Depressive Episode	11 (20.0%)	2 (3.6%)	18 (32.7%)	23 (41.8%)	0 (0.0)%	1 (1.8%)	8 (14.5%)	46 (83.6%)
Dysthymia	26 (47.3%)	5 (8.9%)	9 (16.1%)	14 (25.0%)	26 (47.3%)	3 (5.5%)	9 (16.4%)	16 (29.1%)
Anxiety Disorders (in addition to GAD)[a]								

Any	4 (7.3%)	8 (14.5%)	19 (34.5%)	23 (41.8%)	2 (3.6%)	5 (9.1%)	15 (27.3%)	33 (60.0%)
Panic Disorder	23 (41.8)%	10 (18.2%)	12 (21.8%)	9 (16.4%)	20 (36.4%)	9 (16.4%)	11 (20.0%)	15 (27.3%)
Agoraphobia	39 (70.9%)	3 (5.5%)	4 (7.3%)	8 (14.5%)	37 (67.3%)	3 (5.5%)	4 (7.3%)	10 (18.2%)
Obsessive Compulsive Disorder	30 (54.5%)	6 (10.9%)	10 (18.2%)	8 (14.5%)	27 (49.1%)	6 (10.9%)	12 (21.8%)	9 (16.4%)
Post-Traumatic Stress Disorder	40 (72.7%)	2 (3.6%)	7 (12.7%)	5 (9.1%)	34 (61.8%)	0 (0.0%)	9 (16.4%)	10 (18.2%)
Social Phobia	22 (40.0%)	9 (16.4%)	12 (21.8%)	11 (22.2%)	20 (36.4%)	6 (10.9%)	10 (18.2%)	17 (30.9%)
Specific Phobia	22 (40.0%)	15 (27.3%)	13 (23.6%)	4 (7.3%)	22 (40.0%)	15 (27.3%)	12 (21.8%)	5 (9.1%)
Separation Anxiety Disorder	51 (92.7%)	2 (3.6%)	1 (1.8%)	0 (0.0%)	31 (56.4%)	4 (7.3%)	10 (18.2%)	8 (14.5%)

Notes. N=55. In some cases percentages do not total 100 because of unavailability of complete diagnostic data on one participant.

[a]Inclusion criteria required that participants met full DSM-IV criteria for Generalized Anxiety Disorder and have either current or lifetime history of major depressive episode or dysthymia symptoms.

Table 2.

Demographic Characteristics and Recruitment Sources of Sample

Gender	Male	6 (11%)
	Female	49 (89%)
Race	Non-Hispanic White	39 (71%)
	Latino	2 (4%)
	Asian/Asian-American	10 (18%)
	Native-American	1 (2%)
	Other	3 (5%)
Age	Mean	28.76
	SD	12.43
	Range	18-59
Household Income	< $30,000	11 (20%)
	$30,000-$49,999	14 (26%)
	$50,000-$69,999	6 (11%)
	$70,000-$89,000	9 (16%)
	> $90,000	14 (26%)
Marital Status[a]	Single	40 (74%)
	Married	11 (20%)
	Divorced	3 (6%)

In Psychiatric/ Psychological Treatment	Any	24 (46%)
	Psychotherapy	14 (36%)
	On Psychiatric Meds	20 (25%)
Occupation	Student	33 (60%)
	Other	22 (40%)
Recruitment Source	Flyer	15 (27%)
	Online advertisements	16 (29%)
	Research Studies	4 (7%)
	Stony Brook Psychological/ Anxiety Center	6 (11%)
	Undergraduate Psychology Courses	14 (25%)

Note. N= 55.

[a]One participant left this item blank

Table 3.

Interrater Reliability of SCID Ratings

	Intraclass Correlation Coefficient	
Disorder	Current	Lifetime (Worst)
Major Depressive Disorder	.89	.90
Dysthymia	.77	.77
Bipolar Disorder	1.00	1.00
Generalized Anxiety Disorder	1.00	1.00
Panic Disorder	.81	.90
Agoraphobia	.87	.92
Obsessive-Compulsive Disorder	.85	.85
Post-Traumatic Stress Disorder	.88	.92
Social Phobia	.98	.98
Specific Phobia	.95	.95
Separation Anxiety Disorder	1.00	.82

Note. SCID= Structured Clinical Interview for the DSM-IV (Spitzer, et al., 1995)

Table 4.

Descriptive Statistics for Study Variables

Variable	Mean	Standard Deviation	Minimum	Maximum
Depressed now	3.55	1.88	1.14	7.90
Anxious now	4.20	2.08	1.19	9.35
Hopeless now	3.25	1.99	1.00	8.18
Depressed today	3.93	1.78	1.27	7.47
Anxious today	4.80	1.86	1.52	8.71
Hopeless today	3.60	2.07	1.07	10.00
Anxious rumination	4.39	2.16	1.14	8.94
Anhedonic mood	4.01	1.89	1.05	8.82
Worry	5.77	2.00	2.11	10.00

Notes. *N*= 55. All scales ranged from 1 through 10. Descriptive statistics were computed by first taking the within-person mean for each participant on each variable across all time points, and then computing descriptive statistics across participants. Thus, the minimum and maximum columns reflect the minimum and maximum for individual participants aggregated across time points, not the minimum/ maximum of the actual responses. Depressed/ Anxious /Hopeless now variables= “momentarily” assessed variables, assessed at the time of survey completion. Depressed/

Anxious/ Hopeless today= “course of day” variables, assessed over the course of the day that survey was completed.

Table 5.

Concurrent Associations Between Anxious and Depressed Mood, Assessed Momentarily and Over the Course of the Day

Dependent variable= depressed mood, day *t*	Coefficient[a]	Standard Error	*df*	*t*	*p*
Mood Assessed Momentarily					
Intercept	3.51	.19	85.57	18.49	< .001
Anxious Mood, day *t*	.52	.04	46.07	12.53	< .001
Time	-.02	.01	258.61	-2.26	.025
Mood Assessed Over Course of Day					
Intercept	3.74	.17	49.12	22.45	< .001
Anxious Mood, day *t*	.54	.04	44.67	13.13	< .001
Time	*dropped for non-significance*				

Notes. N= 55.

[a]Unstandardized

Table 6.

Lagged Associations Between Anxious and Depressed Moods at One-, Two-, Three-, and Four-Day Time Lags Analyzed Separately

Dependent variable= depressed mood, day *t*	Coefficient[a]	Standard Error	*df*	*t*	*p*
One-Day Lag					
Intercept	3.51	.19	69.16	13.57	< .001
Anxious Mood	.09	.03	884.77	2.53	.012
Time	-.04	.01	156.35	-3.64	< .001
Two-Day Lag					
Intercept	3.30	.24	77.33	13.71	< .001
Anxious Mood	.25	.05	397.66	5.57	< .001
Time	-.04	.01	253.15	-2.74	.007
Three-Day Lag					

Intercept					<
	3.41	.25	75.08	13.62	.001
Anxious Mood					<
	.25	.05	277.73	4.48	.001
Time	-.05	.02	204.72	-3.24	.001
Four-Day Lag					
Intercept					
	3.18	.26	63.12	12.19	<.001
Anxious Mood	.10	.03	350.48	3.81	<.001
Time	-.03	.02	188.47	-1.96	.052

Note. N= 55. Analyses were exploratory. Random effects (other than intercept) were not included in models for simplicity. Time lags were aggregated (e.g., two-day lagged predictors reflected the means of values on *t*-1 and *t*-2).

[a]Unstandardized

Table 7.

Results of Multilevel Modeling Analysis Simultaneously Predicting Depressed Mood from Anxious Mood at Four Time Lags

Dependent variable= depressed mood, day *t*	Coefficient[a]	Standard Error	*df*	*t*	*p*
Intercept	3.27	.26	73.65	12.50	< .001
Anxious Mood day *t−1*	.02	.04	594.18	0.47	.638
Anxious Mood day *t−2*	.20	.04	595.09	5.11	< .001
Anxious Mood day *t−3*	.03	.04	597.12	0.72	.470
Anxious Mood day *t−4*	.04	.04	595.63	0.98	.330
Time	-.04	.02	113.02	-1.99	.049

Note. N= 55.

[a]Unstandardized

Table 8.

Results of Multilevel Analysis with Simultaneously Entered Lagged and Concurrent Models

Dependent variable= depressed mood, day *t*	Coefficient[a]	Standard Error	*df*	*t*	*p*
Intercept	3.54	.20	78.21	17.80	< .001
Anxious Mood, day *t*	.51	.05	43.42	11.13	< .001
Anxious Mood, day *t*-2	.07	.03	771.30	2.84	.005
Time	-.03	.01	232.15	-3.14	.002

[a]Unstandardized

Notes. N= 55. Anxious mood at *t* retained as a random effect; lagged anxious mood was non-significant as a random effect and was dropped from model.

Table 9.

Concurrent Associations Between Anhedonia, Worry, and Broad Depressed and Anxious Mood

Predictor Variable	Outcome Variable	Coefficient[a]	Standard Error	*df*	*t*	*p*
Worry, day t	→ *Depressed mood, day t*					
Intercept		3.89	.20	88.57	19.09	< .001
Worry		.44	.04	50.57	10.93	< .001
Time		-.01	.01	277.11	-1.32	.189
Worry, day t	→ *Anhedonia, day t*					
Intercept		3.80	.19	54.33	19.84	< .001
Worry		.43	.05	53.82	9.44	< .001
Time		*dropped for non-significance*				
Anxious Mood, *day t*	→ *Anhedonia, day t*					
Intercept		3.85	.19	51.06	20.06	< .001

	Anxious Mood	.43	.04	45.52	10.91	< .001
	Time	*dropped for non-significance*				
Worry, day t → *Anxious mood, day t*						
	Intercept	4.79	.19	91.66	25.59	< .001
	Worry	.55	.04	57.94	13.42	< .001
	Time	-.02	.01	299.51	-1.68	.095
Anhedonia day t → *Depressed mood, day t*						
	Intercept	3.98	.17	92.54	23.54	< .001
	Anhedonia	.55	.03	47.83	16.08	< .001
	Time	-.01	.01	297.77	-1.58	.114

[a]Unstandardized

Notes. N= 55. All analyses conducted separately

Table 10.

Two-Day Lagged Associations Between Anhedonia, Worry, and Depressed and Anxious Mood

Predictor Variable (aggregated days *t-1 and t-2*)	Outcome Variable (day *t*)	Coefficient[a]	Standard Error	*df*	*t*	*p*
Worry	→ *Depressed mood*					
	Intercept	.44	.26	80.11	1.70	.092
	Worry	.22	.05	406.17	4.78	< .001
	Time	-.02	.02	219.46	-1.62	.106
Worry	→ *Anhedonia*					
	Intercept	3.72	.28	90.99	13.49	< .001
	Worry	.18	.05	434.62	3.57	< .001
	Time	-.03	.02	208.88	-2.06	.040
Anhedonia	→ *Anxious mood*					
	Intercept	3.77	.26	50.98	14.36	< .001
	Anhedonia	.06	.06	469.91	1.07	.286
	Time	*dropped for non-significance*				
Anhedonia	→ *Worry*					

Intercept	4.90	.28	47.22	17.30	< .001
Anhedonia	.11	.06	444.29	1.83	.068
Time	*dropped for non-significance*				

[a]Unstandardized

Notes. *N*=55. All analyses conducted separately

Table 11.

Baron and Kenny (1986) Steps for Testing Rumination as Mediator of Anxious Mood Predicting Later Depressed Mood over Two-Day Lags

	Coefficient[a]	Standard Error	*df*	*t*	*p*
Step 1: Predictor variable (anxious mood$_{t-2}$) predicting outcome variable (depressed mood$_t$)					
Intercept	3.22	.25	70.96	12.69	< .001
Anxious Mood	.16	.04	49.20	3.83	< .001
Time	-.04	.01	270.474	-2.79	.006
Step 2: Predictor variable (anxious mood$_{t-1}$) predicting mediator (anxious rumination$_t$)					
Intercept	3.93	.31	72.58	12.69	< .001
Anxious Mood	.09	.04	52.15	2.02	.049
Time	-.02	.02	176.44	-1.60	.111
Step 3: Mediator (anxious rumination$_{t-1}$) predicting outcome (depressed mood$_t$)					
Intercept	3.37	.25	77.65	13.69	< .001
Anxious Rumination	.12	.04	41.19	2.86	.007
Time	-.05	.01	198.61	-3.69	< .001
Step 4: Predictor (anxious mood$_{t-2}$) predicting outcome (depressed mood$_t$), controlling for mediator (anxious rumination$_{t-1}$)					

Intercept	3.18	.24	73.38	13.04	< .001
Anxious Mood	.16	.04	44.29	3.69	.001
Anxious Rumination	.10	.04	27.88	2.35	.026
Time	-.03	.01	211.178	-2.37	.019

Notes. N=55. Mediation was not significant according to a Sobel's (1982) test

Table 12.

Test of Anxious Rumination as Moderator of Concurrent Association Between Anxious Mood and Depressed Mood

Outcome Variable: *Depressed Mood*$_t$	Coefficient[a]	Standard Error	*df*	*t*	*p*
Predictor Variables:					
Intercept	3.42	.19	91.57	17.95	< .001
Anxious Mood$_t$	.43	.04	54.92	10.62	< .001
Anxious Rumination$_t$	.12	.03	42.03	3.39	.001
Anxious Mood$_t$ × Anxious Rumination$_t$	.03	.01	305.39	3.39	.001
Time	-.02	.01	252.27	-2.11	.035

Notes. *N*=55.

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Do you struggle with

Anxiety?

You may be eligible for a research study for payment.

Individuals who are currently experiencing generalized anxiety are needed for research study at Stony Brook University. You do not need to come into Stony Brook to participate. You will be paid **$150** for your participation. You must be 18 or older to participate.

Interested? Call Lisa at 631-632-7837 or email stonybrookrdc@gmail.com **to learn more and determine eligibility.**

STONY BROOK
STATE UNIVERSITY OF NEW YORK

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STONY BROOK
STATE UNIVERSITY OF NEW YORK

May 20, 2010

Dear Client,

You are receiving this letter because you are (or recently were) a client at the Stony Brook Psychological Center or Anxiety Clinic. As you may recall, you were told when you started therapy that you may be contacted to participate in a research study. I am writing you now to do just that..

Participants are currently needed for a study on daily changes in mood, thoughts, and behavior. We are specifically looking for participants who are experiencing certain types of anxiety. The study will entail two parts. First, you would need to come in to Stony Brook for a session in which you will be interviewed about your mood and experiences and given several questionnaires to fill out. This will take around one hour, although the time varies from person to person. Next, you will be asked to fill out brief (approximately 10-15 minute) daily questionnaires from home every day for 21 days, plus a few follow-up questionnaires one week later. You can do that part either online or through the mail. All in all, we expect your participation to take around 4 hours. We know this is a lot of time, but we will pay you --$25 for the Stony Brook part, and $125 for the at home part, for a total of $150!

If you are interested in participating, please call us so we can determine whether or not you are eligible for this study.

Your participation will in no way affect your participation at the Psychological Center/ Anxiety Clinic. In fact, we will not even tell your therapist that you are participating. We do not want you to feel pressured to participate in any way—you should only participate if you want to.

If you are interested, please call Lisa at **631-632-7837**, or email stonybrookrdc@gmail.com. If there is no answer, please leave a message and someone will return your call shortly.

Thanks for your time.

Sincerely,

Joanne Davila, Ph.D.

IRB Approved: 9/2/2009
Expiration Date: 9/1/2010
CORIHS Stony Brook University

STONY BROOK
STATE UNIVERSITY OF NEW YORK
COMMITTEES ON RESEARCH INVOLVING HUMAN SUBJECTS
Established 1971

Project Title: Daily Thoughts and Emotions Study
Principal Investigator: Joanne Davila, Ph.D.
Co-Investigators: Greg Hajcak, Ph.D.; Lisa Starr, M.A. (graduate student)

Research Consent Form

You are being asked to be a volunteer in a research study.

Purpose: The purpose of this study is to examine thoughts, behaviors, and experiences that predict changes in mood from day to day. Approximately 80 individuals will participate in this study.

Procedures: If you decide to be in this study, your part will involve the following: First**,** you will fill out a series of questionnaires during a session at Stony Brook. This will take about 15 minutes. Following the session, you will be asked to complete a 21 day "diary" (i.e., a brief survey each day) from home. The diary will consist of questions about your mood and about thoughts and experiences that you may have had that day. This will take about 15 minutes each day. The diary will be completed either on paper (and mailed back to us using postage page envelopes) or online (via a completely secure website), depending on your preference. One week after your final diary entry, we will ask you to complete a final packet of questionnaires (either online or through the mail). This will take approximately 30 minutes.

Risks/Discomforts: There are no major risks associated with participation in this study, except that you may experience some mild negative feelings (e.g., anxiety, sadness) in response to some of the things that you will be asked to think and talk about. However, you may decline to answer any question that you do not want to answer.

Benefits: There is no direct benefit to you for participation in this study. However, some people report that they enjoy the experience of thinking and talking about personal information and that they learn something about themselves in the process. Also, we will provide you with a list of resources/referrals, which may benefit you should you feel the need for counseling services.

Payment to You: You will be paid $125 for your participation in this study. You will receive payment through the mail following study completion. You will also be entered in a raffle for each day that you complete your daily diary surveys, and you will receive a bonus of 10 extra raffle entries if you complete 100% of your daily diary surveys. Winners will be randomly chosen from all entries at the end of the data collection period; prizes may include iPods, iPhones, and gift certificates, among others. If you win a raffle, you will be contacted by study staff to receive your prize.

Confidentiality/Protecting the Privacy of Your Health Information

We will take steps to help make sure that all the information we get about you is kept private. Your name will not be used wherever possible. We will use a code instead. All the study data that we get from you will be kept locked up. The code will be locked up too. If any papers and talks are given about this research, your name will not be used.

We want to make sure that this study is being done correctly and that your rights and welfare are being protected. For this reason, we will share the data we get from you in this study with the study team, the sponsors of this study (the National Institute of Mental Health, and those who work for them), Stony Brook University's Committee on Research Involving Human Subjects,

applicable Institutional officials, and certain federal offices. However, if you tell us you are going to hurt yourself, hurt someone else, or if we believe the safety of a child is at risk, we will have to report this.

In a lawsuit, a judge can make us give him the information we collected about you.

While you are in this study we will get health data from the results of the interviews and questionnaires you will have done in this study. You have a right to privacy but the data we get about your health in this study can be shared with the people referenced above (the study team, the sponsor of this study [the National Institutes of Mental Health], those who work for the sponsor, Stony Brook University's Committee on Research Involving Human Subjects, applicable institutional officials, and certain federal offices).

Your health data are shared to make sure the study is being done correctly, costs are charged correctly, and to make sure your rights and safety are protected. Not all of these people are required by law to protect your health data. They might share it with others without your permission. For example, the sponsor of this study, the National Institute of Mental Health, does not have to make the same promise under the law to protect your health data.

Some of the health information we get from you in this study cannot be shared with you until the end of the study.

You have the right to stop allowing us to use or give out your health data. You can do this at any time by writing to Joanne Davila. If you do this, we will stop collecting any new health data from you, except if we need to keep an eye on a bad side effect you were having in the study. We will use any data we collected before you wrote your letter. When you sign the consent form at the end, it means:

* That you have read this section.
* That you will allow the use and reporting of your health data as described above.

If you are paid $600 or more a year as a research subject, your social security number will be reported to those in charge of taxes. You may have to pay taxes on this money.
Costs to You: Participation in this study does not involve any costs to you.

Alternatives. Your alternative is to not participate in the study.

Consequences of Withdrawing: There are no consequences of withdrawing from this study.

Subject Rights:

- Your participation in this study is voluntary. You do not have to be in this study if you don't want to be.
- You have the right to change your mind and leave the study at any time without giving any reason, and without penalty.
- Any new information that may make you change your mind about being in this study will be given to you.
- You will get a copy of this consent form to keep.
- You do not waive any of your legal rights by signing this consent form.

Questions about the Study or Your Rights as a Research Subject:

- If you have any questions about the study, you may contact Dr. Joanne Davila at (631) 632-7837.
- If you have any questions about your rights as a research subject, you may contact Ms. Judy Matuk, Committee on Research Involving Human Subjects, (631) 632-9036.

If you sign below, it means that you have read (or have had read to you) the information given in this consent form, and you would like to be a volunteer in this study.

_____
Subject Name

_____ _____
Subject Signature Date

_____ _____
Signature of Person Obtaining Consent Date

_____
Printed Name of Person Obtaining Consent

IRB Approved: 9/2/2009
Expiration Date: 9/1/2010
CORIHS Stony Brook University

STONY BROOK
STATE UNIVERSITY OF NEW YORK
COMMITTEES ON RESEARCH INVOLVING HUMAN SUBJECTS
Established 1971

Project Title: Daily Thoughts and Emotions Study
Principal Investigator: Joanne Davila, Ph.D.
Co-Investigators: Greg Hajcak, Ph.D.; Lisa Starr, M.A. (graduate student)

Research Consent Form

You are being asked to be a volunteer in a research study.

Purpose: The purpose of this study is to examine thoughts, behaviors, and experiences that predict changes in mood from day to day. Approximately 80 individuals will participate in this study.

Procedures: If you decide to be in this study, your part will involve the following: First**,** you will fill out a series of questionnaires during a session at Stony Brook. This will take about 15 minutes. Following the session, you will be asked to complete a 21 day "diary" (i.e., a brief survey each day) from home. The diary will consist of questions about your mood and about thoughts and experiences that you may have had that day. This will take about 15 minutes each day. The diary will be completed either on paper (and mailed back to us using postage page envelopes) or online (via a completely secure website), depending on your preference. One week after your final diary entry, we will ask you to complete a final packet of questionnaires (either online or through the mail). This will take approximately 30 minutes.

Risks/Discomforts: There are no major risks associated with participation in this study, except that you may experience some mild negative feelings (e.g., anxiety, sadness) in response to some of the things that you will be asked to think and talk about. However, you may decline to answer any question that you do not want to answer.

Benefits: There is no direct benefit to you for participation in this study. However, some people report that they enjoy the experience of thinking and talking about personal information and that they learn something about themselves in the process. Also, we will provide you with a list of resources/referrals, which may benefit you should you feel the need for counseling services.

Payment to You: You will be paid $125 for your participation in this study. You will receive payment through the mail following study completion. You will also be entered in a raffle for each day that you complete your daily diary surveys, and you will receive a bonus of 10 extra raffle entries if you complete 100% of your daily diary surveys. Winners will be randomly chosen from all entries at the end of the data collection period; prizes may include iPods, iPhones, and gift certificates, among others. If you win a raffle, you will be contacted by study staff to receive your prize.

Confidentiality/Protecting the Privacy of Your Health Information

We will take steps to help make sure that all the information we get about you is kept private. Your name will not be used wherever possible. We will use a code instead. All the study data that we get from you will be kept locked up. The code will be locked up too. If any papers and talks are given about this research, your name will not be used.

We want to make sure that this study is being done correctly and that your rights and welfare are being protected. For this reason, we will share the data we get from you in this study with the study team, the sponsors of this study (the National Institute of Mental Health, and those who work for them), Stony Brook University's Committee on Research Involving Human Subjects,

applicable Institutional officials, and certain federal offices. However, if you tell us you are going to hurt yourself, hurt someone else, or if we believe the safety of a child is at risk, we will have to report this.

In a lawsuit, a judge can make us give him the information we collected about you.

While you are in this study we will get health data from the results of the interviews and questionnaires you will have done in this study. You have a right to privacy but the data we get about your health in this study can be shared with the people referenced above (the study team, the sponsor of this study [the National Institutes of Mental Health], those who work for the sponsor, Stony Brook University's Committee on Research Involving Human Subjects, applicable institutional officials, and certain federal offices).

Your health data are shared to make sure the study is being done correctly, costs are charged correctly, and to make sure your rights and safety are protected. Not all of these people are required by law to protect your health data. They might share it with others without your permission. For example, the sponsor of this study, the National Institute of Mental Health, does not have to make the same promise under the law to protect your health data.

Some of the health information we get from you in this study cannot be shared with you until the end of the study.

You have the right to stop allowing us to use or give out your health data. You can do this at any time by writing to Joanne Davila. If you do this, we will stop collecting any new health data from you, except if we need to keep an eye on a bad side effect you were having in the study. We will use any data we collected before you wrote your letter. When you sign the consent form at the end, it means:

* That you have read this section.
* That you will allow the use and reporting of your health data as described above.

If you are paid $600 or more a year as a research subject, your social security number will be reported to those in charge of taxes. You may have to pay taxes on this money.
Costs to You: Participation in this study does not involve any costs to you.

Alternatives. Your alternative is to not participate in the study.

Consequences of Withdrawing: There are no consequences of withdrawing from this study.

Subject Rights:

- Your participation in this study is voluntary. You do not have to be in this study if you don't want to be.
- You have the right to change your mind and leave the study at any time without giving any reason, and without penalty.
- Any new information that may make you change your mind about being in this study will be given to you.
- You will get a copy of this consent form to keep.
- You do not waive any of your legal rights by signing this consent form.

Questions about the Study or Your Rights as a Research Subject:

- If you have any questions about the study, you may contact Dr. Joanne Davila at (631) 632-7837.
- If you have any questions about your rights as a research subject, you may contact Ms. Judy Matuk, Committee on Research Involving Human Subjects, (631) 632-9036.

If you sign below, it means that you have read (or have had read to you) the information given in this consent form, and you would like to be a volunteer in this study.

_____
Subject Name

_____ _____
Subject Signature Date

_____ _____
Signature of Person Obtaining Consent Date

_____
Printed Name of Person Obtaining Consent

Diary Items

Using the scale below, check the square that best describes how you are feeling *right now*.

		not at all				moderately					extremely
		1	2	3	4	5	6	7	8	9	10
1.	Depressed	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
2.	Hopeless	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
3.	Anxious	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Please answer #4-6 only if you marked "2" or more for #3. You reported feeling anxious right now. To what extent do you feel…

		not at all				moderately					extremely
4.	Like your anxiety is never going to stop	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
5.	Like you can't control your anxiety	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
6.	That your anxiety will negatively impact your life	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**

Using the scale below, check the square that best describes how you have felt *over the course of the day today*.

		not at all					moderately				extremely
		1	2	3	4	5	6	7	8	9	10
A1.	Depressed	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
A2.	Hopeless	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
A3.	Anxious	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Please answer #A4-A6 only if you marked "2" or more for #A3. You reported feeling anxious today. To what extent did you feel…

		not at all					moderately				extremely
A4.	Like your anxiety is never going to stop	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
A5.	Like you can't control your anxiety	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
A6.	That your anxiety will negatively impact your life	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**

Using the scale below, check the square that best describes how you have felt *over the course of the day today.*

		not at all				moderately					extremely
7.	Supported by others	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
8.	Criticized by others	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
9.	Rejected by others	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**

Using the scale below, check the square that best describes how much you have done the following *over the course of the day today.*

		not at all									a whole lot
10.	Think or ruminate about how anxious you feel	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
11.	Think or ruminate about how sad you feel	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**

Did you experience any of the following today (check all that apply), and how much did it negatively impact you?

		How much did it negatively affect you?									
✓		not at all									a whole lot
☐	Academic difficulties	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**
☐	Job-related difficulties	**1** ☐	☐	☐	☐	☐	☐	☐	☐	☐	☐ **10**

☐ Problems in your romantic relationship **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

☐ Problems with your friends/ peers **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

☐ Family-related problems **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

☐ Financial difficulties **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

☐ Other (please list):_____ **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

_____

_____

Please rate the degree to which you have experienced the following **over the course of the day**:

1. Felt little or no enjoyment in activities you usually enjoy **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
2. Had no appetite or didn't eat much **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
3. Had too much appetite or ate too much **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
4. Couldn't sleep **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
5. Slept too much **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
6. Felt agitated, like you couldn't stop **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

moving

7. Felt slowed down, like your limbs were heavy **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
8. Felt tired/ little energy **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
9. Felt worthless or guilty **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
10. Couldn't concentrate or make decisions **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
11. Had thoughts of death **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
12. Had a panic attack **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
13. Feared being in places where you might have trouble escaping or getting help if you had a panic attack **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
14. Avoided being in places where you might have trouble escaping or getting help if you had a panic attack, or had significant distress from being in such places **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**
15. Feared social situations where you'd be around unfamiliar people or where you'd be scrutinized by others. **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

16. Avoided social situations where you'd be around unfamiliar people or where you'd be scrutinized by others, or had significant distress from being in such situations. **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

17. Had intrusive thoughts, images, or impulses that made you anxious and that you tried to get rid of **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

18. Felt compelled to engage in repetitive behaviors or mental acts (including but not limited to counting, checking things, washing hands, etc.) **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

19. Worried **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

20. Felt restless **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

21. Had trouble concentrating **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

22. Felt easily fatigued **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

23. Felt irritable **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

24. Had muscle tension **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

25. Trouble sleeping or unsatisfying sleep. **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

26. Excessively feared something such as **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

flying, heights, certain animals/ insects, blood, injections, or another specific object or experience

27. Avoided a specific object/experience that you are afraid of (e.g., flying, heights, certain animals/ insects, blood, injections, etc.) even though avoiding it negatively impacted your life. **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

28. Experienced great distress while enduring a specific object/experience that you are afraid of (e.g., flying, heights, certain animals/ insects, blood, injections, etc.). **1** ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ **10**

THANK YOU