



Temporal patterns of anxious and depressed mood in generalized anxiety disorder: A daily diary study

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ARTICLE INFO

Article history:

Received 29 July 2011

Received in revised form

28 October 2011

Accepted 25 November 2011

Keywords:

Anxiety

Depression

Comorbidity

Symptom co-occurrence

Generalized anxiety disorder

ABSTRACT

Research suggests that anxiety disorders tend to temporally precede depressive disorders, a finding potentially relevant to understanding comorbidity. The current study used diary methods to determine whether daily anxious mood also temporally precedes daily depressed mood. 55 participants with generalized anxiety disorder (GAD) and history of depressive symptoms completed a 21-day daily diary tracking anxious and depressed mood. Daily anxious and depressed moods were concurrently associated. Daily anxious mood predicted later depressed mood at a variety of time lags, with significance peaking at a two-day lag. Depressed mood generally did not predict later anxious mood. Results suggest that the temporal antecedence of anxiety over depression extends to daily symptoms in GAD. Implications for the refinement of comorbidity models, including causal theories, are discussed.

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Research has consistently documented extensive comorbidity between anxiety and depression (Maser & Cloninger, 1990). Major depressive disorder (MDD) co-occurs substantially with each individual anxiety disorder, at much higher rates than with other diagnostic categories, such as impulse-control or substance use disorders (Kessler et al., 2003; Kessler, Chiu, Demler, & Walters, 2005; Kessler, Merikangas, & Wang, 2007). Moreover, co-occurring anxiety and depression have negative implications beyond the impact of each individual disorder, including poorer prognosis, academic difficulties, suicide risk, lower quality of life, and worse treatment outcomes (Kessler, Stang, Wittchen, Stein, & Walters, 1999; Ledley et al., 2005; Lewinsohn, Rohde, & Seeley, 1995; Rush et al., 2005; Young, Mufson, & Davies, 2006). Clearly, a full understanding of the origins of comorbidity has important theoretical and practical implications, and yet many aspects of comorbidity are poorly understood.

Existing comorbidity models have traditionally fallen into two categories: the “lumper” perspective that anxiety and depression and their components cannot be meaningfully distinguished, and 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). Some models, such as the widely-

cited tripartite theory (Clark & Watson, 1991; Watson, Clark et al., 1995; Watson, Weber et al., 1995), both split and lump by identifying overlapping factors (negative affectivity) as well as specific components distinct to anxiety (physiological hyperarousal) and depression (anhedonia), but ultimately take the lumper approach of attributing comorbidity to shared substrates.

The field appears to be, in many ways, gravitating toward lumping. Most prominently, the DSM-5 task force recently introduced in its proposed revisions criteria for a “mixed anxiety/depression” diagnosis, which includes symptoms of both major depression and anxious distress (APA, 2010). If adopted, this diagnosis could fundamentally change the way researchers view anxiety–depression comorbidity by relabeling its manifestation as a distinct disorder. As a consequence, we may ultimately see a decrease in research on the separate predictors, course, and correlates of anxiety versus depression. Before taking such an important step, research should ensure that alternate theories of anxiety–depression co-occurrence, including splitter models that account for the association between anxiety and depression without abandoning their nosological distinctions, are adequately considered. In doing so, a critical piece of evidence may be anxiety and depression’s temporal pattern.

Temporal antecedence of anxiety over depression

Numerous studies have shown that anxiety disorders tend to temporally precede depression, using both retrospective (Essau,

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2003; de Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003) and longitudinal designs (Burke, Loeber, Lahey, & Rathouz, 2005; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997; Orvaschel, Lewinsohn, & Seeley, 1995; Wittchen et al., 2000; but see also Moffitt et al., 2007). Also, although anxiety disorders often occur without depression, “pure” depression (i.e., without comorbid anxiety) is relatively rare (Dobson, Cheung, Maser, & Cloninger, 1990, pp. 611–632).

Several researchers have argued that the temporal precedence of anxiety may have important implications for models of comorbidity (Lewinsohn et al., 1997; Wittchen, Beesdo, Bittner, & Goodwin, 2003), although few existing comorbidity theories incorporate it. A parsimonious explanation for this temporal pattern is that anxiety acts as a risk factor for later depression. Several researchers have proposed this idea (e.g., Kessler, Nelson, McGonagle, & Liu, 1996; Lewinsohn et al., 1997; Wittchen et al., 2003), but research has only recently begun to expand upon it, identifying such possible mediators as interpersonal dysfunction, behavioral avoidance, and anxiety response styles (Grant, Beck, Farrow, & Davila, 2007; Moitra, Herbert, & Forman, 2008; Starr & Davila, in press).

Although the temporal sequencing of anxiety and depression may have important conceptual implications, before we can translate this observation into testable comorbidity models, important gaps in the literature need to be addressed. First, most longitudinal studies examining temporal associations have used follow-up periods of months or years (Burke et al., 2005; Cole et al., 1998; Orvaschel et al., 1995; Wittchen et al., 2000). In contrast, most proposed mediators of the association between anxiety and later depression (e.g., anxious rumination and hopelessness, interpersonal dysfunction, behavioral avoidance; Grant et al., 2007; Moitra et al., 2008; Starr & Davila, in press) would be more likely to occur over much shorter intervals, such as days and weeks. Clarification of day-to-day patterns of co-occurrence is needed, as understanding microprocesses of the phenomenological experience can help us understand development of symptoms that may then lead to macro-level changes.

Similarly, previous studies on temporal associations, like the majority of comorbidity research, have focused on diagnosable anxiety disorders and major depression. Although this is informative, it may also be important to examine how the *components* of anxiety and depressive disorders (i.e., symptoms such as anxious and depressed mood) co-occur within short time frames during episodes of diagnosable disorders. As noted by Mineka, Watson, and Clark (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. Subthreshold symptoms often develop into disorders (Judd et al., 1998). Furthermore, disorders themselves are, after all, made up of symptoms. Thus, symptom co-occurrence and disorder comorbidity may operate under similar mechanisms. Moreover, depressive and anxious symptoms co-occur at almost twice the rates of diagnosable depressive and anxiety disorders (Hiller, Zaudig, & von Bose, 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 crucial to understanding comorbidity.

In addition, examining temporal associations between daily symptoms offers several methodological benefits over traditional designs. Exploring symptom co-occurrence within disorders rather than diagnostic comorbidity eliminates the confounding effect of errors in the underlying nosological system (Brown & Barlow, 1992; Mennin, Heimberg, Fresco, & Ritter, 2008). For example, generalized

anxiety disorder (GAD) and MDD share several similar diagnostic criteria (e.g., difficulty concentrating, restlessness, psychomotor agitation, fatigue, sleep impairment), and this overlap has the obvious potential to inflate comorbidity rates. Examining relationships between symptoms rather than disorders helps correct for this problem.

Furthermore, many previous studies on temporal sequencing of anxiety disorders and major depression may have been confounded by the fact that different disorders have varying ages of onset. For example, anxiety disorders often emerge in childhood (Kessler, Berglund, Demler, Jin, & Walters, 2005), whereas depression tends to emerge in adolescence or later (Lewinsohn, Hops, Roberts, & Seeley, 1993). The apparent temporal primacy of anxiety over depression may simply reflect developmental differences 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 risk factors for depressive symptoms. Further, scrutinizing symptoms 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 GAD often develop simultaneously (Moffitt 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.

Finally, in addition to the methodological benefits and implications for comorbidity models, understanding daily symptom co-occurrence may be useful in its own right, as it would enhance our understanding of the phenomenological experience of the naturalistic course of symptoms within episodes. As anxious and depressed moods vary considerably from day-to-day (de Vries, Dijkman-Caes, & Delespaul, 1990), investigating how symptoms within disorders unfold on a daily basis may provide a more nuanced view of the experience of comorbidity. For example, comorbidity typically implies that two disorders are experienced contemporaneously, but among people with comorbid disorders, it is unclear if symptoms within each disorder emerge and desist in relative synchronicity (i.e., with people feeling depressed on the same days they feel anxious), or if symptoms of one disorder trigger symptoms of the other, or if symptoms of each disorder operate relatively independently. Furthermore, syndromes are made up of different kinds of symptoms, and these may show differing temporal patterns. For example, anxious mood could potentially predict fluctuations one symptom of depression (e.g., depressed mood) but not another (e.g., anhedonic mood). Ultimately, a better understanding of the descriptive nature of symptom co-occurrence could potentially generate hypotheses about the maintenance of symptoms and disorders.

Despite its conceptual and methodological importance, research on daily temporal sequencing of symptoms within disorders is virtually nonexistent. Some evidence (drawing from sources as diverse as experimental research on response to uncontrollable negative events, non-human primate research, and attachment research; Alloy, Kelly, Mineka, & Clements, 1990) suggests that anxiety symptoms may precede depressive symptoms within episodes, but this research remains very limited. One study found that daily fluctuations in anxiety predicted later depressive symptoms (and not the reverse; Swendsen, 1997), but given the paucity of studies, more research is clearly needed.

The current study

We explored temporal associations between anxious and depressed moods over the course of a three-week daily diary study. Diary methods offer 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 conventional designs, and accordingly these methods have yielded important insights into psychopathology (e.g., Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001; de Vries, 1992).

We conducted this study using a sample of individuals with a current anxiety disorder and a history of depressive symptoms. The limited research on daily mood co-occurrence has generally used undergraduate samples of convenience with primarily sub-syndromal symptoms (Swendsen, 1997, 1998). Using a clinical sample both ensures that participants will experience significant depressed and anxious mood over the course of the diary and helps maximize the generalizability of results to relevant clinical populations. Further, by recruiting a sample with both an anxiety disorder and a history of depressive symptoms, we ensured that participants would experience a sufficient degree of anxious and depressed moods over the course of the diary period.

All anxiety disorders share analogous features and show significant comorbidity with depression (Kessler, Chiu et al., 2005). 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, GAD. MDD shows higher comorbidity with GAD than with any other anxiety disorder (Kessler, Chiu et al., 2005); Hunt, Slade, and Andrews (2004) found that 39.3% of individuals with GAD also met criteria for MDD within the same one-month period. In fact, GAD co-occurs with depression with such regularity that some argue that it should be classified with MDD as a “general distress” disorder (Watson, 2005; although others have raised important counterarguments, Mennin et al., 2008). Thus, understanding comorbidity between depression and GAD may have especially important conceptual implications. Further, the high co-occurrence between GAD and depression may imply that individuals with GAD experience a particularly high degree of symptom co-occurrence (although this needs to be verified empirically). As a result, examining mood sequencing within GAD may be a good launching point for the development of a broad model of anxiety-depression comorbidity.

Time lags

A complicating issue in diary research is the question of time lag determination. Researchers often have insufficient information to predict the precise time intervals over which hypothesized processes unfold, and analyzing data over inappropriate time lags can conceal significant findings. Because almost no research has been conducted in this area, it is difficult to anticipate the time lags over which anxious and depressed moods might predict each other. 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, we tested several time lags to determine the most appropriate. In addition to facilitating our own analyses, determining the optimal time lag over which symptoms predict each other may help generate hypotheses about the timing of mediators.

Study hypotheses

We examined several predictions related to daily associations between anxious and depressed moods. First, we expected that daily anxious mood would be associated with concurrent depressed mood. Next, to determine whether the temporal

sequencing of anxiety disorders and depression extends to daily symptoms, we examined whether anxious mood fluctuations predict later daily depressed mood fluctuations. In addition, to ensure that results are not unique to depressed and anxious mood, we also examined whether specific daily anxiety symptoms (worry, a hallmark of GAD) predict specific daily depression symptoms (anhedonia, a cardinal symptom of MDD). Based on one previous study on temporal sequencing of symptoms (Swendsen, 1997) and numerous studies on temporal sequencing of disorders (e.g., Wittchen et al., 2000), we expected that elevations in daily anxious mood would predict later elevated depressed mood. We tested multiple time lags, but made no a priori hypotheses about them.

Method

Participants

All participants met the following inclusion criteria: a) full *Diagnostic and Statistical Manual, Fourth Edition* (DSM-IV; APA, 1994) criteria for current GAD (ignoring the MDD exclusion criterion); b) history of at least 1–2 cardinal symptoms of MDD or dysthymia (i.e., significant depressed mood or anhedonia) to ensure elevated risk of depressive symptoms (all otherwise eligible participants met this criterion); c) no present psychotic or bipolar disorders; d) age range of 18–65 years; e) no impairments in reading English. No other exclusion criteria were imposed. Participants showed substantial intra-anxiety comorbidity, with 42% meeting current criteria and 60% meeting lifetime criteria for one or more additional anxiety disorder.

Participants were recruited from a variety of sources. Advertisements with study contact information were posted on campus, in clinic waiting rooms, around the community, and on the Internet. Therapists at graduate training clinics were given with a list of study criteria and asked to give a letter with a description of the study and contact information 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 et al., 1998) to determine eligibility. Forty-five 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-IV; Spitzer, Williams, Gibbon, & First, 1995) to verify eligibility. Based on SCID-IV results, seven were ineligible, leaving 38 participants who completed full procedures. Participants were paid \$25 for their interviews and \$125 for the remainder of the study and were entered in raffles.

Additional participants ($n = 3$) were recruited from recent studies that administered the SCID-IV within the Department of Psychology at Stony Brook University (these samples were recruited from the community). To reduce burden, when possible, participants' eligibility was determined using SCID-IV data collected in the previous studies (all collected within six months) rather than re-interviewing participants. These participants were paid \$125 for the non-interview study portion and entered in raffles.

Finally, we recruited additional participants from undergraduate psychology courses. Students completed self-report screening measures; potentially eligible students were contacted and scheduled for participation. In total, 24 students were recruited, with 10 determined to be ineligible following the SCID-IV, leaving 14 eligible participants. Students were compensated with course credit comparable to payment amounts and were entered in raffles.

In total, 55 eligible participants completed all study procedures. The sample included 49 women and 6 men. The unbalanced gender ratio was likely in part 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, consistent with 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). Mean age was 28.76 ($SD = 12.43$, range = 18–59). Seventy-one percent of participants described 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. Participants reported a broad range of annual household income (20% earned less than \$30,000). Forty-four percent were currently receiving treatment for psychiatric disorders (with 26% taking psychiatric medications and 36% receiving psychosocial intervention).

Although participants were recruited from a wide range of sources, they were subjected to the same inclusion criteria. Accordingly, we found no differences between recruitment sources on gender, number of baseline diagnoses, or symptom measures at baseline (including the Beck Depression Inventory, Beck Anxiety Inventory, and other commonly-used anxiety and depression measures; Beck, Epstein, Brown, & Steer, 1988; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Participants recruited from undergraduate psychology courses were significantly younger than participants recruited from all other sources, $F(51, 3) = 5.39$, $p = .003$. Form of compensation (cash payment versus course credit) was not related to diary compliance.

The Stony Brook University Committee on Research Involving Human Subjects and UCLA Institutional Review Board approved this research.

Measures

Screening

As described above, participants recruited through advertisements and therapy clinics were screened for project eligibility using portions of the MINI, a brief structured diagnostic interview that generates similar results to longer interviews in substantially less time (Sheehan et al., 1998). For logistical reasons, participants recruited through psychology classes were screened with self-report depression and anxiety measures. Importantly, all participants were interviewed with the SCID-IV to confirm project eligibility, so the differing screening procedures should not have diluted sample quality.

Baseline

The SCID-IV (Spitzer et al., 1995) is a widely-used semi-structured interview designed to generate DSM-IV diagnoses, with excellent psychometric properties including test-retest and inter-rater reliability (Zanarini et al., 2000). Anxiety and mood disorder modules (current and past) were administered by an advanced graduate student. To capture both diagnoses and severity, we used a 4-point scoring system, where 0 = no symptoms, 1 = mild symptoms, 2 = moderate symptoms, and 3 = DSM-IV disorder. To meet eligibility criteria, scores of three on current GAD, one or more on past or current major depressive episode or dysthymia, and zero on bipolar and psychotic disorders were required. Interviews were audiotaped and 22% were re-coded by a second rater; intraclass correlation coefficients for current GAD, lifetime MDD, and lifetime dysthymia were 1.00, .90, and .77 respectively. Demographic data including age, race/ethnicity, gender, and other variables were also collected at baseline.

Diary

As excessive diary length can reduce compliance (Morren, Dulmen, Ouwerkerk, & Bensing, 2009), diary items were designed

to assess constructs of interest quickly and efficiently. Daily depressed and anxious mood were assessed using face-valid questions about mood at the moment of diary completion (“How [anxious/depressed] do you feel right now?”) with a 10-point Likert-type scale (1 = *not at all*, 10 = *extremely*). Asking about mood at the moment of diary completion minimizes retrospection (Parkinson, Briner, Reynolds, & Totterdell, 1995), a principal goal of diary research (Stone, Litcher-Kelly, Eid, & Diener, 2006), but also only captures a thin slice of daily mood and may be inflated by diurnal mood variation (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, we also assessed mood over the course of the day (“How anxious did you feel, on average, over the course of the day today?”). Throughout this manuscript, these items are distinguished as “moment-of-diary” versus “course-of-day,” although note that both items were completed at bedtime and thus may have been influenced by diurnal variation in mood. Time frames of predictor and outcome variables always corresponded. Participants also rated daily anhedonia (“Felt little or no enjoyment in activities you usually enjoy”) and worry (“worried”) on 10-point scales; these were only assessed over the course of the day. As a basic test of item validity, we examined baseline self-report anxiety and depression measures as predictors of anxious and depressed moods. The Beck Anxiety Inventory (BAI; Beck et al., 1988) and Beck Depression Inventory-II (BDI; Beck, Steer, & Brown, 1996, pp. 1–82) were entered simultaneously into separate multilevel models with a) daily anxious mood and b) daily depressed mood as outcomes. For both moment-of-diary and course-of-day variables, the BAI but not BDI predicted anxious mood, and the BDI but not BAI predicted depressed mood.

Procedure

Phase 1: baseline

For practical reasons and to reduce participant burden, individuals were given choices of completing baseline procedures in person, by phone, or via internet/mail, depending on the component. For example, SCID interviews were conducted in person or via phone. There were no significant differences on online versus paper measures nor on phone versus in-person interview results, consistent with prior studies supporting the equivalency of these modalities (Coles, Cook, & Blake, 2007; Fouladi, McCarthy, & Moller, 2002; Rohde, Lewinsohn, & Seeley, 1997).

Phase 2: diary

After interviews, participants were given thorough diary instructions. They were asked to begin their diary that night and complete it once daily for the next 21 days. Participants were instructed to complete diaries as close to bedtime as was convenient.

Participants were given the option of completing diaries online or on paper, and all participants were given copies of paper diaries (with content identical to the online version) as back-ups. The vast majority of diaries (92%) were completed online. The diary website (administered through www.psychdata.com) electronically date- and time-stamped each survey, allowing for compliance verification. In cases where a participant completed multiple surveys in one day, all of that participant's data for that day were excluded. Participants were asked to return the paper surveys by mail within 1–2 days of completion, and postmarks were inspected for compliance.

We took several lengths to maximize diary compliance. First, a computer program automatically sent participants a reminder email, containing their ID number and a survey link, every day at a participant-designated time. Second, to boost compliance

incentive, participants were awarded raffle tickets based on the number of diary entries completed. Raffle prizes included an MP3 player and GPS navigation device. Participants completed an average of 18.82 diary entries (90% compliance rate).

Data analysis strategy

All analyses were conducted using SPSS 18.0.1 Mixed Methods. Hypotheses were tested using mixed effects modeling, specifically multilevel modeling (MLM), with daily reports of symptoms 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 were lagged, with the time-varying predictor variable temporally preceding the time-varying outcome variable. All predictor variables were grand mean centered. Time was included in initial models as a fixed effect, but was dropped when highly non-significant ($p > .2$). We specified an unstructured covariance type for random effects, and a first-order auto-regressive covariance [AR(1)] type to control for auto-correlation of residuals. All effects were initially included as both fixed and random effects; highly non-significant random effects ($p > .2$) were dropped but retained as fixed effects (see Nezelek, 2001).

In some cases, analyses failed to converge using this strategy, and we took the following steps. Taking the measures recommended by Garson (2009), we a) inspected variables for correlations near 1.0 (none were found in analyses reported here), b) increased maximum iterations, c) increased step-halvings, d) increased singularity tolerance value, e) increased scoring steps, and f) increased parameter convergence value. If the model still did not converge, we changed the repeated covariance type from AR(1) to diagonal. 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; Nezelek, 2001). In that case, we reset changes from prior steps and then removed the smallest random effects (keeping the variable only as a fixed effect) until the model converged.

Time lags

To determine appropriate time lags, we began with a lag of one day (depressed mood on day_t predicted by anxious mood on day_{t-1}). We then increased the lag in one-day intervals until significance peaked and dropped. We chose the time lag with the peak significance rate as “optimal.” Multiday lags were aggregated, so a two-day lagged variable predicting an outcome on day_t included a summation of variables on day_{t-1} and day_{t-2}. Non-aggregated lagged analyses produced similar results. For the purposes of these analyses, we entered only the intercept as a random effect.

Comparing lagged and concurrent effect models

In addition to lagged analyses, concurrent effect models (i.e., with 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, as summarized in the following equation:

$$Y_{(t)} = a + b * X_{(t)} + c * \text{time} \quad (1)$$

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*, we included only the predictor variable (lagged at $t - k$, where *k* is the appropriate time lag determined as outlined above) and time (if significant at $p < .2$). This analysis can be represented as the following function, which examines whether changes in the outcome variable are predicted by previously occurring changes in the predictor variable:

$$Y_{(t)} = a + b * X_{(t-k)} + c * \text{time} \quad (2)$$

However, for a more conservative test, we 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*), as represented here:

$$Y_{(t)} = a + b * X_{(t)} + c * X_{(t-k)} + d * \text{time} \quad (3)$$

Power analyses

Power for lagged analyses 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. Anxious mood at $t - 1$ predicting depressed mood at *t* was estimated at .20; 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 $t - 1$. Using a sample size of 55 and 21 observations (1155 “cases”), power was estimated at 1.00 even using the more conservative parameter estimates.

Missing data

MLM can handle data that are missing at random (Fitzmaurice, Laird, & Ware, 2004). In the current dataset, missing a daily survey was not predicted by key variables such as prior-day anxious or depressed mood, providing reasonable evidence that data were missing at random and therefore missing data are ignorable (Fitzmaurice et al., 2004; Howell, 2009).

Results

Descriptive data for all variables are presented in Table 1.

Concurrent associations between anxious and depressed moods

Moment-of-diary depressed and anxious moods were concurrently associated ($B = .52$, $SE = .04$, $t(46.07) = 12.53$, $p < .001$), as were course-of-day depressed and anxious moods ($B = .54$, $SE = .04$, $t(44.67) = 13.13$, $p < .001$). Note that, as one would expect,

Table 1
Descriptive statistics for study variables.

Variable	M	SD	Minimum	Maximum
Depressed (moment-of-diary)	3.55	1.88	1.14	7.90
Anxious now (moment-of-diary)	4.20	2.08	1.19	9.35
Depressed (course-of-day)	3.93	1.78	1.27	7.47
Anxious (course-of-day)	4.80	1.86	1.52	8.71
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 1–10. Descriptive statistics were computed by first taking within-person means across all time points, and then computing descriptive statistics across participants.

moment-of-diary mood was concurrently associated with course-of-day mood (depressed mood, $B = .65$, $SE = .04$, $t(50.81) = 15.97$, $p < .001$; anxious mood, $B = .63$, $SE = .04$, $t(50.63) = 16.21$, $p < .001$).

Does anxious mood predict later depressed mood?

Table 2 displays results for anxious mood predicting later depressed mood, at different time lags using moment-of-diary variables. First, anxious mood predicted depressed mood at a one-day lag. Second, the sum of anxious mood over days $t - 1$ and $t - 2$ predicted depressed mood on day t (two-day lag) at higher magnitude and significance than for the one-day lag, although differences in effect sizes were not subjected to significance testing. Next, 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 but slightly smaller effect sizes; 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 the t and p statistic values may simply reflect greater sample size for the two-day lag. Finally, using a four-day time lag generated still significant but again apparently smaller effect sizes. Given the downward trend of effect sizes, the two-day time lag was determined as optimal (although the three-day lag may be equally appropriate).

As an additional test of time lag appropriateness, we 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 3, in this analysis, only the two-day time lag significantly predicted depressed mood, again supporting this lag as optimal for examining anxious mood predicting depressed mood in this dataset. Thus, supporting predictions, fluctuations in anxious mood do predict fluctuations in later depressed mood, with a two-day time lag yielding results of highest magnitude.

To provide a more conservative test, we next re-ran the prior analysis (with a two-day lag) controlling for the association between concurrent anxious and depressed moods. Specifically, depressed mood at t was the outcome, and a) anxious mood at $t - 2$, b) anxious mood at t , and c) time were entered as fixed effect predictors (anxious mood at both t and $t - 2$ were initially entered as a random effects; anxious mood at t was significant as a random effect and was retained, but previous anxious mood was highly

Table 2
Lagged associations between anxious and depressed moods at one-, two-, three-, and four-day time lags Analyzed separately.

Dependent variable = depressed mood, day t	Unstandardized coefficient	Standard error	df	t	p
<i>One-day lag</i>					
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
<i>Two-day lag</i>					
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
<i>Three-day lag</i>					
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
<i>Four-day lag</i>					
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. $N = 55$. Random effects (other than intercept) were not included in models. Time lags were aggregated.

Table 3

Results of multilevel modeling analysis simultaneously predicting depressed mood from anxious mood at four time lags.

Dependent variable = depressed mood, day t	Unstandardized coefficient	Standard error	df	t	p
Intercept	3.27	.26	73.65	12.50	<.001
Anxious mood day $t - 1$.02	.04	594.18	.47	.638
Anxious mood day $t - 2$.20	.04	595.09	5.11	<.001
Anxious mood day $t - 3$.03	.04	597.12	.72	.470
Anxious mood day $t - 4$.04	.04	595.63	.98	.330
Time	-.04	.02	113.02	-1.99	.049

Note. $N = 55$.

non-significant and was dropped as a random effect). Anxious mood was a significant predictor of depressed mood both as a lagged ($B = .07$, $SE = .03$, $t(771.30) = 2.84$, $p = .005$) and concurrent ($B = .51$, $SE = .05$, $t(43.42) = 11.13$, $p < .001$) predictor, providing more powerful evidence that fluctuations in anxious mood predict fluctuations in later depressed mood.

We next conducted the same analyses using the course-of-day mood variables rather than the moment-of-diary variables. Results were very similar, and once again, the two-day lag yielded the strongest effects.

Does depressed mood predict later anxious mood?

Again, we tested this effect using multiple time lags. First, using a one-day time lag, depressed mood at $t - 1$ was not a significant predictor of anxious mood at t ($B = -.02$, $SE = .04$, $t(934.84) = -.65$, ns). Next, using a two-day aggregated time lag, fluctuations in depressed mood *did* predict fluctuations in later anxious mood ($B = .12$, $SE = .06$, $t(427.07) = 2.14$, $p = .033$). However, a few caveats should be noted: a) 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; b) when using course-of-day variables rather than moment-of-diary variables, depressed mood was not a predictor of later anxious mood at any time lag; c) when controlling for the concurrent effect model, as described above, previous depressed mood was no longer a significant predictor of anxious mood ($B = -.02$, $SE = .05$, $t(501.78) = -.33$, $p = .74$), while concurrent depressed mood remained a strong predictor of anxious mood ($B = .65$, $SE = .03$, $t(686.23) = 19.26$, $p < .001$); 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 in some circumstances weakly predict fluctuations in anxious mood, but this effect is not as robust as with anxious mood predicting later depressed mood and may be better accounted for by the concurrent association between anxious and depressed mood.

Temporal associations between anhedonic mood and worry

We next examined whether other symptoms of anxiety and depression (worry and anhedonia) would yield differing temporal patterns, to ensure that results were not specific to depressed and anxious mood. Note the concurrent associations between both anxious mood and worry ($B = .55$, $SE = .04$, $t(57.94) = 13.42$) and depressed mood and anhedonia ($B = .55$, $SE = .03$, $t(47.83) = 16.08$), both $ps < .001$.

First, worry was concurrently associated with both depressed mood ($B = .44$, $SE = .04$, $t(50.57) = 10.93$, $p < .001$) and anhedonia ($B = .43$, $SE = .05$, $t(53.82) = 9.44$, $p < .001$), and both remained significant when entered as simultaneous predictors (depressed mood: $B = .48$, $SE = .05$, $t(47.22) = 9.66$; anhedonia: $B = .14$,

$SE = .05$, $t(38.37) = 3.50$, both $ps < .001$), suggesting independent associations with worry. In turn, anhedonia was associated with both worry (as stated above) and anxious mood ($B = .43$, $SE = .04$, $t(45.52) = 10.91$, $p < .001$), and worry and anxious mood both predicted anhedonia when entered simultaneously (worry: $B = .30$, $SE = .05$, $t(47.45) = 6.49$; anxious mood: $B = .25$, $SE = .04$, $t(41.74) = 6.12$, both $ps < .001$).

Second, we examined lagged worry and anhedonia. For simplicity, we used the two-day lag suggested by previous analyses. Results are presented in Table 4. Converging with other results, worry predicted later anhedonia and later depressed mood, but anhedonia was not a significant predictor of later anxious mood nor later worry.

Discussion

This study examined daily temporal patterns between anxious mood and depressed mood in a 21-day diary study conducted in a GAD sample. 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 (although findings may be inflated by systematic error such as shared method variance). Although expected, the result provides some basic but important 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 finding underscores a general point about comorbidity research: comorbidity and co-occurrence have generally been conceptualized as between-subjects phenomena (individuals with one disorder are at elevated risk for another). A less explored but similarly important 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 (i.e., within-subjects co-occurrence or comorbidity). Our results support the existence of within-subjects symptom co-occurrence in the case of anxious and depressed mood among adults with GAD and a history of depressive symptoms.

Exploring within-subjects symptom co-occurrence in greater detail may be important. For example, research has extensively documented the between-subjects factor structure of anxiety and depression (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 (APA, 1994) represents 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 within-subjects basis? For example, amongst individuals with multiple comorbid disorders, would the individual daily symptoms of each disorder temporally coincide? Future diary studies should pose this and similar questions.

Second, we hypothesized that the sequence typically found in disorders over the course of years (where anxiety tends to precede depression) would replicate when looking at symptoms assessed over the course of days. This was indeed the case; 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. Further, the pattern was replicated using other symptoms of GAD and MDD, specifically anhedonia and worry. We also tested the reverse direction of effect, with depressed mood predicting later anxious mood. Generally, depressed mood did not precede anxious mood; however, over a two-day lag and using moment-of-diary mood variables, fluctuations in depressed mood did significantly predict fluctuations in later anxious mood. On one hand, this tempers our 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, support for depressed mood as a predictor of later anxious mood was substantially weaker, failing to replicate under repeated and more stringent tests. Anxious mood much more robustly predicted depressed later mood than vice versa. Further replication is needed to more clearly delineate temporal patterns, but this evidence makes a reasonable case that anxious mood tends to come first.

The finding that anxious mood tends to precede depressed mood on a daily basis makes an 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; Essau, 2003; de Graaf et al., 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 questioned whether anxiety disorders may simply be markers of more fundamental risk factors for depression, with their earlier

Table 4
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)	Unstandardized coefficient	Standard error	df	t	p
Worry	→ Depressed mood					
	Intercept	.44	.26	80.11	1.70	.092
	Worry	.22	.05	406.17	4.78	< .001
Worry	→ Anhedonia					
	Intercept	-.02	.02	219.46	-1.62	.106
	Time					
Anhedonia	→ Anxious mood					
	Intercept	3.72	.28	90.99	13.49	< .001
	Worry	.18	.05	434.62	3.57	< .001
Anhedonia	→ Worry					
	Intercept	-.03	.02	208.88	-2.06	.040
	Time					
Anhedonia	→ Depressed mood					
	Intercept	3.77	.26	50.98	14.36	< .001
	Anhedonia	.06	.06	469.91	1.07	.286
Anhedonia	→ Anxious mood					
	Intercept					
	Time	Dropped for non-significance ($p > .2$)				
Anhedonia	→ Worry					
	Intercept	4.90	.28	47.22	17.30	< .001
	Anhedonia	.11	.06	444.29	1.83	.068
Anhedonia	→ Depressed mood					
	Intercept					
	Time	Dropped for non-significance ($p > .2$)				

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

emergence simply representing differences in course (Kessler et al., 2007). Our results suggest that the temporal precedence of anxiety symptoms over depression emerges in a time frame too short to be explained by differences in course. Thus, although we cannot conclude that anxious mood causes depressed mood (as other explanations are possible; Alloy et al., 1990), the study offers some evidence supporting this hypothesis.

Of course, this study examined symptoms and not disorders, and the two are not equivalent and should not be confused. However, patterns of symptom co-occurrence may have implications for disorder comorbidity. For example, daily fluctuations in anxiety symptoms experienced as part of an anxiety disorder may repeatedly trigger depressed mood, perhaps ultimately culminating in a major depressive episode. This intriguing idea merits further exploration. Minimally, our results provide an interesting glimpse into naturalistic patterns of dysregulation within GAD.

We also attempted to determine the optimal time lag over which anxious mood leads to depressed mood, something that to our knowledge has not been explored before. Among the time lags tested, a two-day lag emerged as strongest. 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 time. With no real theoretical basis to support one time lag over another, conjecture about why the two-day lag was most supported is purely post hoc speculation. That said, recency effects, in which too long a lag leads to weaker effect sizes, may contribute. On the other hand, anxiety experienced over too short a time span may not be chronic enough to spur the processes that lead to depressed mood. The two-day lag may optimally balance these opposing factors. Replication of these findings in other datasets will be an important next step. Of course, time lags that are shorter than one day, which were not examined in the current study, could potentially produce even stronger effects; future research should explore this question.

Results must be considered in the context of limitations of the current study. One is the use of simple, face-valid questions to assess daily mood. Although this method helped keep the diary short (maximizing compliance; Morren et al., 2009), longer, well-validated measures might be preferable. Note that numerous previous diary studies have assessed mood using items or visual analog scales based on single-adjective descriptors (e.g., Brinker & Dozois, 2009; Mor et al., 2010; Swendsen, 1998), and that to our knowledge no mood scales currently exist that have been explicitly validated for diary use (Ebner-Priemer & Trull, 2009). Future research should explore whether daily assessment of depression and anxiety using measures with demonstrated discriminant validity yields similar results, and the current study should be considered a novel but somewhat preliminary expansion of the literature. In addition, the ratio of women to men in our sample was high even when considering the female preponderance in anxiety and overrepresentation of women in the recruitment sources (i.e., psychology courses and treatment centers; Aalto-Setälä et al., 2002; Metzner et al., 1994). The low number of men in the sample precluded the examination of potentially important gender differences. Furthermore, some research suggests that the inherent demands of diary study often result in samples self-selected for agreeableness and conscientiousness (Scollon, Prieto, & Diener, 2009); to the extent that this was the case in the current study, it may affect the generalizability of results.

The current study used daily diary assessments, which is an important first step, given the lack of existing research on daily mood co-occurrence. However, future studies should examine mood co-occurrence over shorter intervals, using such methods as experience sampling methods or ecological momentary

assessment (ESM/EMA). These techniques offer several benefits over daily diaries (Scollon et al., 2009; Wenze & Miller, 2010). First, it is possible that daily assessments are too infrequent to capture some co-occurrence processes (Swendsen, 1998). Assessing mood several times per day could yield differing temporal patterns. Second, participants in the current study were instructed to complete diaries at bedtime, and results may have been influenced by diurnal variation in mood. Anxious and depressed mood states tend to widely over the course of the day, and typically peak at the end of the day (Robbins & Tanck, 1987). On one hand, collecting mood at the same time each day allowed us to capture patterns that hold beyond this diurnal variation; on the other hand, examining mood at another time of day (e.g., morning) may have produced different results. Future ESM/EMA studies, in which participants complete assessments at random intervals throughout the day, would add important supplementary data to the current results. Furthermore, the feasibility of applying such approaches in clinical populations has been strongly supported (Johnson et al., 2009).

The current study used a sample of adults with GAD. GAD shows the highest associations with depression of any anxiety disorder (Kessler, Chiu et al., 2005); however, looking specifically at GAD may limit conclusions. For example, 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 et al., 2007). Perhaps results would have differed if other anxiety disorders had been explored. All anxiety disorders co-occur with depression (Kessler, Chiu et al., 2005), but mechanisms of mood co-occurrence may differ according to the type of anxiety experienced, and these differences may be reflected in temporal sequencing of symptoms. Further, participants were also required to report a history of depressive symptoms (although no otherwise eligible participants were actually excluded based on this criterion, reflecting the high co-occurrence between GAD and depression). Participants with pure (i.e., non-comorbid) anxiety disorders or depression likely still experience subthreshold symptoms of other disorders; future research should examine whether symptom co-occurrence follows similar patterns in these groups.

Similarly, little is known about mood sequencing in normative samples. It is possible that subthreshold anxiety symptoms in non-clinical samples (which are likely minor and transitory) are not severe enough to spur depressive symptoms. Likewise, individuals suffering from chronic anxiety disorders may respond to symptoms differently than healthy individuals experiencing normative fluctuations in anxious mood; the former group may feel more discouraged, overwhelmed, and hopeless in response to anxiety symptoms, and may be more apt to develop depressed mood as a result, whereas healthy people may dismiss occasional feelings of anxiety as inconsequential. Future research should utilize normative comparison groups. One study showed, using a non-clinical sample, that anxious mood preceded depressed mood on a daily basis (Swendsen, 1997), but this requires replication.

The current study does not explain why anxious mood precedes depressed mood, but one conceptual possibility is that anxious mood functions as a chronic stressor, which (like other forms of stress) eventuates in depressed mood. An important next step will be to identify and test mechanisms by which anxiety symptoms lead to increased depressed mood, including both mediators that serve as causal links and moderators that make anxiety symptoms more depressogenic. For example, we (Starr & Davila, in press) recently proposed and outlined preliminary support for a model where the manner in which people respond to their anxiety symptoms (e.g., with ruminative or hopeless thoughts) plays a role in the development of comorbid symptoms. Grant et al. (2007)

showed that negative interpersonal styles (specifically avoidance of expressing emotions) mediated the association between social anxiety and later depression. Behavioral and experiential avoidance may also act as symptom co-occurrence mechanisms (Moitra et al., 2008), as anxious mood could lead to avoidance, which in turn exacerbates life stressors, prompting depressed mood. Conversely, acceptance or distress tolerance may buffer against the effects of anxiety symptoms on depressed mood. Each of these processes is likely to unfold over the course of days and weeks, and should therefore be studied using diary methods.

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; c.f., Brown, Antony, & Barlow, 1995), 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 independent syndromes, without considering how symptoms interact. If anxious mood causes depressed mood, perhaps focusing treatment efforts on anxiety symptoms would help reduce both existing anxious and depressive symptoms (Flannery-Schroeder, 2006; Tsao, Mystkowski, Zucker, & Craske, 2002). 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 et al. (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 MDD (Goodwin & Olsson, 2001), counter to the pattern expected if there were a non-causal relationship between anxiety and MDD (as treatment-seeking would likely act as a marker for greater symptom severity). More research should focus on treatment of anxiety when it first emerges (often in childhood, Kessler, Berglund et al., 2005) as an effort toward long-term prevention of depression.

It may also be interesting to look naturalistically at changes in and associations between daily depressed and anxious mood over time during treatment. For example, do depressed and anxious moods decrease over similar trajectories during treatment? Do decreases in anxious mood prompt subsequent drops in depressed mood, or does anxious mood remain stable even when depressed mood decreases? Would treatments approaches produce differing trajectories of change? Exploring how specific symptoms change when 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. As noted previously, planned revisions of the DSM may radically alter the way researchers view comorbidity, as co-occurring anxiety and depression may be reconceptualized as a single disorder, rather than coexisting syndromes (APA, 2010). Although our findings do not necessarily contraindicate these revisions, they do suggest that anxiety–depression co-occurrence may not solely attributable to poorly drawn nosological categories. Indeed, relationships between symptoms within disorders may denote a more complex picture of co-occurrence and comorbidity than previously envisioned.

Acknowledgments

This research was made possible through a grant from the National Institute of Mental Health (F31MH082545) and an American Psychological Association Dissertation Award, both awarded to the first author. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of

the NIMH or the APA. The authors would like to thank Joseph Schwartz, Ph.D., Thomas Olin, Ph.D., and Niall Bolger, Ph.D. for their statistical consultation and Daniel Klein, Ph.D., Greg Hajcak, Ph.D., Bonita London-Thompson, Ph.D., Joseph Schwartz, Ph.D., and Constance Hammen, Ph.D. for their manuscript feedback, and would also like to acknowledge the following people for their project assistance: Rachel Hershenberg, M.A., Athena Yoneda, Ph.D., Jonathan Powers, M.S., Kaitlyn Gorman, Kayla Whearty and Kevin Lee.

These results were presented at the 2010 Annual Meeting of the Association for Behavioral and Cognitive Therapies in San Francisco, CA.

References

- Aalto-Setälä, T., Marttunen, M., Tuulio-Henriksson, A., & Lönnqvist, J. (2002). Psychiatric treatment seeking and psychosocial impairment among young adults with depression. *Journal of Affective Disorders*, 70(1), 35–47. doi:10.1016/S0165-0327(01)00316-0.
- Alloy, L. B., Kelly, K. A., Mineka, S., & Clements, C. M. (1990). Comorbidity of anxiety and depressive disorders: a helplessness-hopelessness perspective. In J. D. Maser, & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders*. Washington, D.C.: American Psychiatric Press.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, D.C.: Author.
- American Psychiatric Association. (2010). *Proposed draft revisions to DSM disorders and criteria*. DSM-5 Development Retrieved November 23, 2010, from www.dsm5.org.
- Armstrong, K. A., & Khawaja, N. G. (2002). Gender differences in anxiety: an investigation of the symptoms, cognitions and sensitivity towards anxiety in a nonclinical population. *Behavioural and Cognitive Psychotherapy*, 30(2), 227–231.
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *Journal of Consulting and Clinical Psychology*, 56, 893–897.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck depression inventory-II*. San Antonio, TX: Psychological Corporation.
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.
- Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: capturing life as it is lived. *Annual Review of Psychology*, 54, 579–616.
- Brinker, J. K., & Dozois, D. J. A. (2009). Ruminative thought style and depressed mood. *Journal of Clinical Psychology*, 65(1), 1–19. doi:10.1002/jclp.20542.
- Brown, T. A., Antony, M. M., & Barlow, D. H. (1995). Diagnostic comorbidity in panic disorder: effect on treatment outcome and course of comorbid diagnoses following treatment. *Journal of Consulting and Clinical Psychology*, 63(3), 408–418.
- Brown, T. A., & Barlow, D. H. (1992). Comorbidity among anxiety disorders: implications for treatment and DSM-IV. *Journal of Consulting and Clinical Psychology*, 60(6), 835–844.
- Burke, J. D., Loeber, R., Lahey, B. B., & Rathouz, P. J. (2005). Developmental transitions among affective and behavioral disorders in adolescent boys. *Journal of Child Psychology and Psychiatry*, 46(11), 1200–1210.
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, 100, 316–336.
- Cole, D. A., Peeke, L. G., Martin, J. M., Truglio, R., & Seroczynski, A. D. (1998). A longitudinal look at the relation between depression and anxiety in children and adolescents. *Journal of Consulting and Clinical Psychology*, 66(3), 451–460.
- Coles, M. E., Cook, L. M., & Blake, T. R. (2007). Assessing obsessive compulsive symptoms and cognitions on the internet: evidence for the comparability of paper and Internet administration. *Behaviour Research and Therapy*, 45(9), 2232–2240.
- Dobson, K. S., Cheung, E., Maser, J. D., & Cloninger, C. R. (1990). *Relationship between anxiety and depression: Conceptual and methodological issues comorbidity of mood and anxiety disorders*. American Psychiatric Association.
- Ebner-Priemer, U. W., & Trull, T. J. (2009). Ecological momentary assessment of mood disorders and mood dysregulation. *Psychological Assessment*, 21(4), 463–475. doi:10.1037/a0017075.
- Essau, C. A. (2003). Comorbidity of anxiety disorders in adolescents. *Depression and Anxiety*, 18(1), 1–6.
- Fitzmaurice, G. M., Laird, N. M., & Ware, J. H. (2004). *Applied longitudinal analysis*. Hoboken, NJ: Wiley Interseries.
- Flannery-Schroeder, E. C. (2006). Reducing anxiety to prevent depression. *American Journal of Preventive Medicine*, 31(6, Suppl. 1), 136–142. doi:10.1016/j.amepre.2006.07.006.
- Fouladi, R. T., McCarthy, C. J., & Moller, N. P. (2002). Paper-and-pencil or online? Evaluating mode effects on measures of emotional functioning and attachment. *Assessment*, 9(2), 204–215.
- Garson, G. D. (2009, December 16). *Linear mixed models: Hierarchical linear, random effects, multilevel, random coefficients, and repeated measures models*. Retrieved

- February 28, 2010, from <http://faculty.chass.ncsu.edu/garson/PA765/multilevel.htm>.
- Goodwin, R., & Olfson, M. (2001). Treatment of panic attack and risk of major depressive disorder in the community. *The American Journal of Psychiatry*, 158(7), 1146–1148. doi:10.1176/appi.ajp.158.7.1146.
- de Graaf, R., Bijl, R. V., Spijker, J., Beekman, A. T. F., & Vollebergh, W. A. M. (2003). Temporal sequencing of lifetime mood disorders in relation to comorbid anxiety and substance use disorders: findings from the Netherlands mental health survey and incidence study. *Social Psychiatry and Psychiatric Epidemiology*, 38(1), 1–11.
- Grant, D. M., Beck, J. G., Farrow, S. M., & Davila, J. (2007). Do interpersonal features of social anxiety influence the development of depressive symptoms? *Cognition and Emotion*, 21, 646–663.
- Hiller, W., Zaudig, M., & von Bose, M. (1989). The overlap between depression and anxiety on different levels of psychopathology. *Journal of Affective Disorders*, 16(2), 223–231.
- Howell, D. C. (2009). *Treatment of missing data*. Retrieved May 2, 2010, from http://www.uvm.edu/~7edhowell/StatPages/More_Stuff/Missing_Data/Missing.html.
- Hunt, C., Slade, T., & Andrews, G. (2004). Generalized anxiety disorder and major depressive disorder comorbidity in the National survey of mental health and well-being. *Depression and Anxiety*, 20(1), 23–31.
- Johnson, E. I., Grondin, O., Barrault, M., Faytout, M., Helbig, S., Husky, M., et al. (2009). Computerized ambulatory monitoring in psychiatry: a multi-site collaborative study of acceptability, compliance, and reactivity. *International Journal of Methods in Psychiatric Research*, 18(1), 48–57. doi:10.1002/mpr.276.
- Judd, L. L., Akiskal, H. S., Maser, J. D., Zeller, P. J., Endicott, J., Coryell, W., et al. (1998). A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. *Archives of General Psychiatry*, 55(8), 694–700.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., et al. (2003). The epidemiology of major depressive disorder: results from the National comorbidity survey replication (NCS-R). *JAMA: Journal of the American Medical Association*, 289(23), 3095–3105. doi:10.1001/jama.289.23.3095.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National comorbidity survey replication. *Archives of General Psychiatry*, 62(6), 593–602.
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National comorbidity survey replication. *Archives of General Psychiatry*, 62(6), 617–627.
- Kessler, R. C., Merikangas, K. R., & Wang, P. S. (2007). Prevalence, comorbidity, and service utilization for mood disorders in the United States at the beginning of the twenty-first century. *Annual Review of Clinical Psychology*, 3, 137–158. doi:10.1146/annurev.clinpsy.3.022806.091444.
- Kessler, R. C., Nelson, C. B., McGonagle, K. A., & Liu, J. (1996). Comorbidity of DSM-III-R major depressive disorder in the general population: results from the US National comorbidity survey. *British Journal of Psychiatry*, 168(Suppl. 30), 17–30.
- Kessler, R. C., Stang, P., Wittchen, H. U., Stein, M., & Walters, E. E. (1999). Lifetime comorbidities between social phobia and mood disorders in the US National comorbidity survey. *Psychological Medicine*, 29(3), 555–567.
- Kovacs, M., Paulauskas, S. L., Gatsonis, C., & Richards, C. (1988). Depressive disorders in childhood. III. Longitudinal study of comorbidity with and risk for conduct disorders. *Journal of Affective Disorders*, 15, 205–217.
- Kush, F. R. (2004). An Operationalized cognitive therapy approach with mixed anxiety and depression. *Psychotherapy: Theory, Research, Practice, Training*, 41(3), 266–275.
- Ledley, D. R., Huppert, J. D., Foa, E. B., Davidson, J. R. T., Keefe, F. J., & Potts, N. L. S. (2005). Impact of depressive symptoms on the treatment of generalized social anxiety disorder. *Depression and Anxiety*, 22(4), 161–167.
- Lewinsohn, P. M., Hops, H., Roberts, R. E., & Seeley, J. R. (1993). Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III-R disorders in high school students. *Journal of Abnormal Psychology*, 102(1), 133–144.
- Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1995). Adolescent psychopathology: III. The clinical consequences of comorbidity. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(4), 510–519.
- Lewinsohn, P. M., Zinbarg, R., Seeley, J. R., Lewinsohn, M., & Sack, W. H. (1997). Lifetime comorbidity among anxiety disorders and between anxiety disorders and other mental disorders in adolescents. *Journal of Anxiety Disorders*, 11(4), 377–394.
- Marshall, G. N., Sherbourne, C. D., Meredith, L. S., Camp, P., & Hays, R. D. (2003). The tripartite model of anxiety and depression: symptom structure in depressive and hypertensive patient groups. *Journal of Personality Assessment*, 80(2), 139–153.
- Maser, J. D., & Cloninger, C. R. (1990). *Comorbidity of mood and anxiety disorders*. Washington, D.C.: American Psychiatric Association.
- Mennin, D. S., Heimberg, R. G., Fresco, D. M., & Ritter, M. R. (2008). Is generalized anxiety disorder an anxiety or mood disorder? Considering multiple factors as we ponder the fate of GAD. *Depression and Anxiety*, 25(4), 289–299.
- Metzner, B. S., Rajecki, D. W., & Lauer, J. B. (1994). New majors and the feminization of psychology: testing and extending the Rajecki-Metzner model. *Teaching of Psychology*, 21(1), 5–11. doi:10.1207/s15328023top2101.
- Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, 49, 377–412.
- Moffitt, T. E., Harrington, H., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., et al. (2007). Depression and generalized anxiety disorder: cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Archives of General Psychiatry*, 64(6), 651–660. doi:10.1001/archpsyc.64.6.651.
- Moitra, E., Herbert, J. D., & Forman, E. M. (2008). Behavioral avoidance mediates the relationship between anxiety and depressive symptoms among social anxiety disorder patients. *Journal of Anxiety Disorders*, 22(7), 1205–1213.
- Mor, N., Doane, L. D., Adam, E. K., Mineka, S., Zinbarg, R. E., Griffith, J. W., et al. (2010). Within-person variations in self-focused attention and negative affect in depression and anxiety: a diary study. *Cognition and Emotion*, 24(1), 48–62. doi:10.1080/02699930802499715.
- Morren, M., Dulmen, S. V., Ouwerkerk, J., & Bensing, J. (2009). Compliance with momentary pain measurement using electronic diaries: a systematic review. *European Journal of Pain*, 13(4), 354–365.
- Muthén, L. K., & Muthén, B. O. (1998). *Mplus user's guide* (5th ed.). Los Angeles, CA: Muthén & Muthén.
- Myin-Germeys, I., van Os, J., Schwartz, J. E., Stone, A. A., & Delespaul, P. A. (2001). Emotional reactivity to daily life stress in psychosis. *Archives of General Psychiatry*, 58(12), 1137–1144. doi:10.1001/archpsyc.58.12.1137.
- Nezlek, J. B. (2001). Multilevel random coefficient analyses of event- and interval-contingent data in social and personality psychology research. *Personality and Social Psychology Bulletin*, 27(7), 771–785. doi:10.1177/0146167201277001.
- Orvaschel, H., Lewinsohn, P. M., & Seeley, J. R. (1995). Continuity of psychopathology in a community sample of adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(11), 1525–1535.
- Parkinson, B., Briner, R. B., Reynolds, S., & Totterdell, P. (1995). Time frames for mood: relations between monetary and generalized ratings of affect. *Personality and Social Psychology Bulletin*, 21(4), 331–339.
- Robbins, P. R., & Tanck, R. H. (1987). A study of diurnal patterns of depressed mood. *Motivation and Emotion*, 11(1), 37–49. doi:10.1007/bf00992212.
- Robichaud, M., Dugas, M. J., & Conway, M. (2003). Gender differences in worry and associated cognitive-behavioral variables. *Journal of Anxiety Disorders*, 17(5), 501–516.
- Rohde, P., Lewinsohn, P. M., & Seeley, J. R. (1997). Comparability of telephone and face-to-face interviews in assessing axis I and II disorders. *American Journal of Psychiatry*, 154(11), 1593–1598.
- Rush, A. J., Zimmerman, M., Wisniewski, S. R., Fava, M., Hollon, S. D., Warden, D., et al. (2005). Comorbid psychiatric disorders in depressed outpatients: demographic and clinical features. *Journal of Affective Disorders*, 87(1), 43–55.
- Scollon, C. N., Prieto, C.-K., & Diener, E. (2009). Experience sampling: promises and pitfalls, strength and weaknesses. In Diener, E. (Ed.). (2009). *Assessing well-being*, Vol. 39 (pp. 157–180). Netherlands: Springer.
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., et al. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59(Suppl. 20), 22–33, quiz 34–57.
- Spitzer, R. L., Williams, J. B. W., Gibbon, M., & First, M. (1995). *Structured clinical interview for DSM-IV (SCID-IV)*. Washington, D.C.: American Psychiatric Association.
- Starr, L. R., & Davila, J. Responding to anxiety with rumination and hopelessness: mechanism of anxiety–depression symptom co-occurrence? *Cognitive Therapy and Research*, in press. doi:10.1007/s10608-011-9363-1.
- Stone, A. A., Litcher-Kelly, L., Eid, M., & Diener, E. (2006). *Momentary capture of real-world data*. Washington, DC, US: American Psychological Association.
- Swendsen, J. D. (1997). Anxiety, depression, and their comorbidity: an experience sampling test of the helplessness-hopelessness theory. *Cognitive Therapy and Research*, 21(1), 97–114.
- Swendsen, J. D. (1998). The helplessness-hopelessness theory and daily mood experience: an idiographic and cross-situational perspective. *Journal of Personality and Social Psychology*, 74(5), 1398–1408.
- Tsao, J. C. I., Mystkowski, J. L., Zucker, B. G., & Craske, M. G. (2002). Effects of cognitive-behavioral therapy for panic disorder on comorbid conditions: replication and extension. *Behavior Therapy*, 33(4), 493–509. doi:10.1016/s0005-7894(02)80013-2.
- de Vries, M. W. (1992). *The experience of psychopathology: Investigating mental disorders in their natural settings*. Cambridge, U.K.: Cambridge University Press.
- de Vries, M. W., Dijkman-Caes, C. I. M., & Delespaul, P. A. E. G. (1990). The sampling of experience: a method of measuring the co-occurrence of anxiety and depression in daily life. In J. D. Maser, & C. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 707–726). Washington, D.C.: American Psychiatric Press.
- Watson, D. (2005). Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, 114(4), 522–536.
- Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology*, 104(1), 15–25.
- Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology*, 104(1), 3–14.
- Wenze, S., & Miller, I. (2010). Use of ecological momentary assessment in mood disorders research. *Clinical Psychology Review*, .
- Wittchen, H.-U., Beesdo, K., Bittner, A., & Goodwin, R. D. (2003). Depressive episodes – evidence for a causal role of primary anxiety disorders? *European Psychiatry*, 18(8), 384–393.

- Wittchen, H.-U., Kessler, R. C., Pfister, H., & Lieb, M. (2000). Why do people with anxiety disorders become depressed? A prospective-longitudinal community study. *Acta Psychiatrica Scandinavica*, *102*, 14–23.
- Young, J. F., Mufson, L., & Davies, M. (2006). Impact of comorbid anxiety in an effectiveness study of interpersonal psychotherapy for depressed adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*(8), 904–912.
- Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The collaborative longitudinal personality disorders study: reliability of axis I and II diagnoses. *Journal of Personality Disorders*, *14*(4), 291–299.