



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

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### 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,

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