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Mood induced cognitive and emotional reactivity, life stress, and the prediction of depressive relapse

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ABSTRACT

This study examined a group of participants who were fully remitted from a previous episode of major depressive disorder, and evaluated the role of cognitive and emotional reactivity to a mood challenge, and life stress in the prediction of relapse. Fifty-two participants were evaluated during remission, and their reactivity (i.e., change in dysfunctional attitudes and emotional state) to a depressed mood induction was evaluated. The cohort was followed up 12 months after the initial assessment. Thirty-five percent of the sample experienced a relapse during the follow-up period. Relapse was predicted by higher rates of life stress, and lower levels of emotional reactivity (specifically less reduction in happiness) to the mood induction during the initial assessment. Cognitive reactivity to the mood induction did not predict relapse, nor did the interaction between cognitive reactivity and life stress. These findings are discussed in terms of recent literature suggesting that depression is associated with insensitivity to emotion context, such that depressed individuals display blunted emotional responses to affective stimuli, including sadness-inducing stimuli. These findings suggest that insensitivity to emotional context may also be a characteristic of euthymic individuals at risk of relapse.

Introduction

A major contributor to depression prevalence rates across the world is the return of new episodes of depression in people who have already experienced an episode (Belsher & Costello, 1988; Lewinsohn, Zeiss, & Duncan, 1989). It has been suggested that at least 50% of patients who recover from an initial episode of depression will have one or more subsequent depressive episodes over their lifetime (Paykel et al., 1995). Further, in many individuals, depressive episodes show a worsening pattern over the course of repeated episodes, characterized by increased severity, frequency and autonomy (i.e., episodes are less clearly precipitated by psychosocial stress), and lack of responsiveness to initially effective treatments (Kendler, Thornton, & Gardner, 2000; Post et al., 1996). These characteristics highlight the urgent need to identify vulnerability or risk factors tied to the relapse and recurrence of depression.

Cognitive models of depression identify information-processing biases as a critical diathesis for depression (e.g., Abramson, Metalsky, & Alloy, 1989; Clark, Beck, & Alford, 1999; Scher, Ingram & Segal, 2005). Beck's (1967, 1987) cognitive model predicts that formerly depressed individuals remain vulnerable to future depression due to the existence of latent negative schemas. Current research suggests that dysfunctional thinking patterns embedded in such schemas can be activated by negative mood states (Hedlund & Rude, 1995; Miranda, Persons, & Byers, 1990; Scher et al., 2005; Teasdale & Dent, 1987). While dysfunctional attitudes were initially thought to be a vulnerability factor for recurrent depression, there is no clear evidence to suggest that the stable aspects of depression-related cognitive style constitutes a vulnerability factor for depression (Ingram, Miranda, & Segal, 1998). Indeed it appears that the cognitive features of depression tend to remit when symptoms remit (e.g., Hollon, Kendall & Lumry, 1986; Silverman, Silverman, & Eardley, 1984). However, a key postulate of Beck's (1967) model is that the depressogenic schemas are latent, with phenomena such as dysfunctional attitudes only becoming activated and accessible in response to certain stressors (Beck, Rush, Shaw, & Emery, 1979).

The mood-state hypothesis of cognitive dysfunction suggests that in some people, a lowering of mood might reactivate thinking styles associated with previous depressive episodes, bringing about a significant change in thought patterns (also referred to as the 'differential activation hypothesis'; see Teasdale, 1983, 1988). In effect, the sad mood state is thought to activate schemas, making dysfunctional attitudes more salient (Beck et al., 1979). Therefore, the risk of relapse in individuals who have recovered from

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depression may depend on the ease with which such depressogenic, ruminative processing schemas can be reinstated by factors such as evaluative self-related information or dysphoric mood (Teasdale, 1999).

While previous studies have established mood-linked cognitive reactivity as a correlate of vulnerability to depressive relapse (see review by Lau, Segal, & Williams, 2004), there is less evidence to validate the causal aspects of the relationship (Scher et al., 2005). In other words, is differential cognitive reactivity to sad mood a marker of past depression or a separate indicator of risk for future depression? Whether cognitive reactivity is an independent predictor of return episodes of MDD depends on the degree to which mood-activated reinstatement of negative thinking patterns *predict* relapse and recurrence of depression. It seems likely that in a person with a history of major depression, reactivated negative thinking might act to maintain and intensify mild sadness, leading to a more persistent state of dysphoria and increasing the risk of future onset of major depressive episodes. Thus, in a person who has previously suffered from depression the degree of cognitive reactivity following mood change may have implications for future depression.

Support for the idea that the degree of mood-activated reinstatement of negative thinking patterns can independently predict relapse is found in an elegant study by Segal, Gemar, and Williams (1999). Segal and his colleagues used a mood induction procedure to induce a transient sad mood in patients who had recovered from depression, either through cognitive therapy or through pharmacotherapy. They found that those participants who exhibited the greatest cognitive reactivity were more likely to experience a depressive relapse or recurrence over the 30 month follow-up period. The Segal et al. (1999) study suggests that patients' reactions to a dysphoric mood stimulus, in the form of a mood induction procedure, are independently predictive of depressive relapse. Segal and his colleagues (2006) have more recently replicated this study over an 18-month follow-up period, and again found that the magnitude of mood-linked cognitive reactivity was a significant predictor of relapse. Taken together with the findings of Lewinsohn, Allen, Seeley, and Gotlib (1999), these results establish an important link between mood-related changes in measures of negative cognition and risk for relapse. These findings all provide empirical support for Teasdale's differential activation hypothesis, and highlight the possibility of identifying those patients more at risk at relapse after recovery from depression.

Within a 'diathesis–stress' model of depression (e.g., Monroe & Simons, 1991; Zuckerman, 1999), life stress is also an important component in the etiology of depression. The relationship between heightened life stress and the onset of depression is well documented, with "occurrences that are defined as undesirable, major life events likely to be associated with depressive onset" (Mazure, 1998, p. 294). Across 10 general population studies of women with depression, 83% of women were found to have experienced a severe event or major difficulty prior to the onset of their depression (Brown, Bifulco, & Harris, 1987). Furthermore, although some studies have suggested that life stress might be more relevant to the etiology of first episodes rather than relapse and recurrence (e.g., Lewinsohn et al., 1999), recent reviews have identified the need for further research in order to provide a more nuanced understanding of the role of life stress in different phases of depressive disorder (Monroe & Harkness, 2005).

The primary aim of this study was to further examine whether the degree of cognitive reactivity following a mood induction procedure predicts subsequent depressive relapse. The current study also aimed to extend the studies by Segal and colleagues (1999, 2006) by examining, in addition to cognitive reactivity, both the role of emotional reactivity to a mood challenge and life stress, in the return of an episode of depression. Recent studies have found that emotional reactivity may also play a role in prospective outcomes. For example, Rottenberg, Kasch, Gross, and Gotlib (2002) found that depressed patients who showed inhibited emotional reactivity to sad and amusing films had poorer concurrent functioning, and that those who were *under*-reactive to the amusing film showed poorer outcomes over a follow-up period. Indeed, following a series of studies that have demonstrated that depressed individuals are less emotionally reactive to sadness-inducing stimuli, despite their persistently depressed mood, Rottenberg (2005) has recently proposed that clinical depression is characterized by emotion context insensitivity (ECI). This model makes opposite prediction to the more conventional view, which holds that mood states and mood disorder should potentiate congruent emotional responses (i.e., depressed mood should render people more likely to have a strong sadness response to sadness-inducing stimuli). It is unclear, however, whether ECI will also characterize depression-vulnerable people when fully recovered. In other words, does ECI represent a trait-like characteristic of vulnerable individuals, or is ECI only evident during acutely depressed states? Given the recent evidence in support of the ECI theory, and the demonstrated prospective role of blunted emotional response in poor outcomes (Rottenberg et al., 2002), we predicted that blunted affective response to the mood induction during remission would be associated with greater likelihood of relapse.

It was therefore hypothesized that a sad mood induction (comprising listening to sad music combined with autobiographical recall of a sad event) would activate both dysphoric mood and dysfunctional thinking in individuals who had recovered from depression. Moreover, it was expected that the degree of emotional and cognitive reactivity (i.e., the degree to which changes in mood activate changes in emotional state and dysfunctional thinking) following the sad mood induction would significantly and independently predict subsequent depressive relapse. Specifically it was predicted that greater cognitive reactivity and blunted affective reactivity would be associated with greater likelihood of relapse.

The second hypothesis was that stressful life events would also predict subsequent relapse. Life stress was assessed both by the number and subjective intensity of stressful life events experienced by participants in the 12-month period prior to relapse, or in the case of no relapse, the 12-month period prior to the follow-up interview. Investigation of both cognitive and affective reactivity, and life stress, as predictors of relapse within the one study will allow comparison of the influence of these predictors. This is of importance as some previous studies have suggested that cognitive reactivity may be a more potent predictor of relapse than is life stress (e.g., Lewinsohn et al., 1999), although these measures have not been directly compared within a prospective, longitudinal design. Finally, given that the cognitive model of depression is a diathesis–stress model (Beck, 1967, 1976; Lewinsohn, Joiner & Rohde, 2001), we also examined whether there was any interaction between cognitive or emotional reactivity and life stress in predicting relapse or recurrence.

Method

Participants

A community sample of individuals in remission or recovery from clinical depression participated in the research. They were recruited from the University of Melbourne and from readers of the Herald Sun, the largest circulating Melbourne newspaper. Participants were recruited via research advertisements placed around the University of Melbourne campus and in the post-graduate email newsletter and from the general community via an editorial in the 'Health Watch' section of the newspaper; 20 male and 32 female participants between the ages of 17 and 59 years who met