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Cognitive reactivity to sad mood: structure and validity of a new measure

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Abstract

Cognitive reactivity to the experimental induction of sad mood has been found to predict relapse in recovered depressed patients. The present report describes the development and test of a questionnaire that aims to measure cognitive reactivity independently from a mood induction procedure. The Leiden Index of Depression Sensitivity (LEIDS) was filled out by 198 participants. After Principal Components Analysis, 26 items were retained, which comprised four factors with good psychometric properties: Negative Self-Evaluation; Acceptance/Coping; Indifference; and Harm Avoidance. In a sample of 48 college students, LEIDS scores — particularly Negative Self-Evaluation and Harm Avoidance — were rather strong predictors of cognitive reactivity in a mood induction procedure. In contrast, baseline depression and baseline cognitive dysfunction did not predict cognitive reactivity. It is concluded that the LEIDS is a promising measure of cognitive reactivity, and that clinical studies need to be carried out to test its ability to predict relapse of depression. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Depression; Vulnerability; Cognition; Mood Induction; Reactivity; Relapse

1. Introduction

Dysfunctional cognitions are thought to be important causative and maintaining factors of depression (Beck, 1967). Individuals who endorse statements like “It is important that everyone likes me” are thought to be more prone to develop depression than people who do not. Research has shown, however, that the dysfunctional cognitions of depressed patients disappear after treatment, regardless of whether treatment was biological or psychological (Simons, Garfield, & Murphy, 1984). At first, this finding may seem to indicate that cognitive dysfunctions are symptoms

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or by-products, not causes or vulnerability markers of depression. Modern cognitive theories have accommodated this finding by stating that dysfunctional cognitions do not disappear during remissions of depressive episodes, but become inactive ('latently present') (Teasdale, 1988; Miranda & Persons, 1988; Segal, Williams, Teasdale, & Gemar, 1996). When these cognitions have not been targeted by treatment, they may be easily activated (e.g., during a state of mild dysphoria), increasing the risk of a recurrence of depression (Hollon, DeRubeis, & Evans, 1987). Some support for this contention comes from studies that demonstrated an association between sad mood and dysfunctional cognitions in recovered depressed patients, but not in never-depressed individuals (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990). Other studies have found information processing differences between formerly-depressed and never-depressed participants after experimental induction of a dysphoric mood (Teasdale & Dent, 1987; Ingram, Bernet, & McLaughlin, 1994).

These studies suggest that there may be a residual cognitive deficit that is only measurable during (naturally occurring or induced) sad mood. They do not prove, however, that these deficits are causally involved in recurrences of depression. Much stronger support for this idea has recently become available in a study by Segal, Gemar, and Williams (1999). These authors 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. Dysfunctional cognitions were measured immediately before and after the induction of sad mood. The change in dysfunctional cognitions ('cognitive reactivity') reflects the ease with which latent cognitions may be activated. It appeared that the mean cognitive reactivity score of 29 patients treated with antidepressants was indeed greater than that of 25 patients treated with cognitive therapy. Furthermore, cognitive reactivity contributed significantly to the prediction of depressive relapses at 1 year follow up, regardless of prior treatment modality. If these findings prove to be reliable (replicable), they provide strong support for cognitive theory and treatment of depression. Considering the relatively small sample sizes of this study, a replication seems warranted. An obstacle to replication studies, however, is the fact that the measurement of cognitive reactivity is not very easy. In the procedure as used by Segal et al. (1999), patients were asked to listen to a piece of music and to focus on a sad memory for ca 7 min. Although the mood effects of this procedure seem to be reliable, about 25% of participants may fail to respond with a sad mood (Martin, 1990). A second problem is that the dysfunctional cognitions are typically measured by administration of the 40-item Dysfunctional Attitude Scale (DAS) (Weissman, 1979) both before and after the music. To preclude artifactual effects of repeated administration of the same questionnaire within ten minutes, two parallel forms of the DAS are often used. However, since there are some doubts about the interchangeability of the two DAS forms, some authors have preferred to use the same form twice (e.g., Brosse, Craighead, & Craighead, 1999). Segal et al. (1999) found it necessary to use complicated statistical procedures to control for possible systematic differences between the two DAS forms. Either way, clinical applications are limited, whether one uses the same or different forms of the DAS, especially when one considers the fact that mean cognitive change scores are rather small (4–8 points on a scale with a possible range of 0–280, and a typical mean score of 110). In summary, research could benefit if a questionnaire were available that measures reactivity directly, that is, without mood induction procedure and without the need to statistically control for possible artifacts.

Despite the large number of cognitive measures that already exist in depression research, the

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