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Behaviour Research and Therapy 42 (2004) 1001–1017
BEHAVIOUR RESEARCH AND THERAPY
www.elsevier.com/locate/brat

Teasdale's differential activation hypothesis: implications for mechanisms of depressive relapse and suicidal behaviour

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Received 14 November 2003; received in revised form 26 March 2004; accepted 26 March 2004

Abstract

Teasdale's differential activation hypothesis (DAH) has been proposed as one account of cognitive vulnerability to depression. This view holds that important factors determining whether one's initial depression becomes more severe or persistent are the degree of activation, and content, of negative thinking patterns that become accessible in the depressed state. This phenomenon has been referred to as cognitive reactivity. Empirical support for the predictions of this model derives from a combination of cross-sectional and prospective studies. In this article, we evaluate this evidence with the goal of determining whether mood-induced cognitive reactivity can be considered a risk factor for depressive relapse/recurrence. Our review demonstrates sufficient evidence to consider cognitive reactivity as a potential causal risk factor for depressive relapse/recurrence. Furthermore, we extend the application of this model to the problem of suicidal relapse/recurrence including a review of preliminary support for this approach.

Keywords: Major depression; Relapse/recurrence; Differential activation hypothesis; Cognitive reactivity; Risk factor; Suicide

1. Prescript

This paper forms part of a special issue organized to mark John Teasdale's intellectual contributions to research on emotion, cognition and mood disorders. His singular ability to bridge

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0005-7967/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2004.03.003

the worlds of basic research and clinical application as well as to excite those around him by the prospect of participating in this synthesis has made an enormous contribution to our field. He has promoted in his theories and their application, a scientific rigour and sense of deep intellectual curiosity that has affected many, and made profound contributions to the personal research development of the authors of this paper, in particular.

This paper touches on one aspect of his work: cognitive models of depression vulnerability. These models were once assailed for their weak empirical footing. Yet, they have become one of the best examples of cumulative science in our field largely due to John Teasdale's theoretical and empirical work. All recent studies in this domain have benefited hugely by paying close attention to the nature of psychological predisposition described in his model. When applied clinically, this model has informed the design of treatments that provide hope for recovered depressed patients searching ways to reverse the cognitive imprints left by their previous bouts of depression.

We are delighted to be part of this celebration of the work of a mentor, colleague and friend.

2. Introduction

Major depressive disorder (MDD) remains a daunting mental health challenge, with lifetime prevalence rates estimated between 2.9 and 12.6 per 100 and lifetime risk estimated at 17–19% (Kessler et al., 1994). MDD is now viewed as a chronic, lifelong illness with a high risk for relapse and/or recurrence (Berti Ceroni, Neri, & Pezzoli, 1984; Keller, Lavori, Lewis, & Klerman, 1983; for review see Judd, 1997). Those who suffer from one depressive episode will "experience an average of four lifetime major depressive episodes of 20 weeks duration each" (Judd, 1997: p. 990). According to the World Health Organization, unipolar major depression imposed the fourth greatest burden of ill health of all diseases worldwide (Murray & Lopez, 1998). Alarmingly, this burden is projected to increase both absolutely and relatively such that by the year 2020 depression will impose the second greatest burden of ill health, very close behind the top cause, ischaemic heart disease. Together, these statistics serve to highlight the urgent need to identify risk factors tied to episode relapse/recurrence.

The risk factors for depressive relapse/recurrence can be understood as multifaceted involving a complex and dynamic interaction of biological, social and psychological factors (Segal & Dobson, 1992; Teasdale, 1988). In the past 30 years, however, there has been a growing interest in the role of psychological factors. In particular, more recent theories of depression suggest that the risk factors underlying relapse/recurrences may indeed be different from those risk factors that underlie first onsets of this disorder (Lewinsohn, Allen, Seeley, & Gotlib, 1999). Researchers have identified two factors predisposing an individual to depressive relapse, stressful life events (Hammen, 1991; Kendler, Thornton, & Gardner, 2000, 2001; Simons, Angell, Monroe, & Thase, 1993) and a diathesis referred to as "cognitive reactivity", the activation of negative information processing biases when an individual experiences dysphoric mood (Teasdale, 1983, 1988). While there is a growing body of evidence that stressful life events contribute to the onset of depressive episodes (Hammen, 1991; Kendler et al., 2000, 2001; Simons et al., 1993), many people faced with similar environmental stressors do not become depressed

(Brown & Harris, 1978). This suggests that psychological factors specific to the individual also play a role in depressive relapse.

Cognitive models of depression, in particular, have identified information processing biases as a diathesis for depressive reactions (Robins & Hayes, 1993). For example, Beck's cognitive model of depression (Beck, 1967; Beck, Rush, Shaw, & Emery, 1979) posits that one's emotional and behavioral reactions to a given situation are mediated through one's thoughts, interpretations or meanings attributed to the experience. In depression, the content of these thoughts is predominantly negative regarding the self, the world, and the future. These thoughts ultimately derive from negative or depressogenic self-schemas, formed as a result of negative childhood experiences, which serve to negatively bias processing of self-relevant information (Williams, Watts, MacLeod, & Mathews, 1997).

Research on depressive cognition has provided considerable evidence that mood state can powerfully affect the information processing system. For example, there is a large body of evidence demonstrating the effects of mood on memory, either mood-congruous (Blaney, 1986; Teasdale, 1983) or mood-dependent (Eich, 1995). Mood-congruent memory refers to the tendency for individuals in a depressed mood to demonstrate a bias for the encoding or retrieval of negatively versus positively valenced information. This occurs when the individual encodes or retrieves information in a mood state that matches the emotional tone of the information. Mood-dependent memory refers to the improved retrieval of information encoded in a similar mood state. Eich (1995) has defined the conditions under which mood-dependent memory can be reliably demonstrated. Thus, a depressed individual would be more likely to remember material that was originally learned in previous depressed mood. Finally, there is evidence that depressed mood negatively biases a range of cognitive processes in addition to memory including, for example, explicit recall (Derry & Kuiper, 1981), memory load (Bargh & Tota, 1988), and semantic priming (Segal, Gemar, Truchon, Guirguis, & Horowitz, 1995).

A core feature of the cognitive model is that depressogenic self-schemas represent a diathesis that can increase risk for major depression. According to this model, these schemas are typically latent until activated by a life event that is severe and schema congruent (Beck et al., 1979; Monroe & Simons, 1991). This view suggests the existence of identifiable differences at the level of cognitive schemas between those not currently depressed but vulnerable to becoming clinically depressed versus those not vulnerable to depression. One of the major challenges of testing this view has been the measurement of dysfunctional schemas (Dozois & Dobson, 2001a). One questionnaire measure of dysfunctional basic assumptions that researchers have accepted is the Dysfunctional Attitudes Scale (DAS) (Weissman & Beck, 1978). Thus, the demonstration that individuals who are at increased risk for depression endorse higher scores on the DAS than those not at risk, would validate the existence of depressogenic schemas. However, the majority of studies using this measure failed to demonstrate differences in DAS scores between recovered depressed subjects (i.e., at increased risk for depression) and controls (e.g., Hamilton & Abramson, 1983; Silverman, Silverman, & Eardley, 1984; Simons, Garfield, & Murphy, 1984) and therefore failed to provide support for this aspect of the cognitive model of depression.

3. A differential activation model of vulnerability to depressive relapse

Teasdale's (1988) consideration of a distinct but related model of cognitive vulnerability to depressive relapse, the differential activation hypothesis (DAH), however, can account for the results of these studies. Teasdale built on Beck's cognitive model by proposing that "in addition to any differences in cognitive organization that may be apparent in the non-depressed state, and in addition to any idiosyncratic cognitive schemas that may be activated by a limited class of environmental situations, vulnerability to severe and persistent depression is powerfully related to differences in patterns of thinking that are activated *in the depressed state*." (Teasdale, 1988: p. 251). In other words, Teasdale (1985) assumes that the normal course of depression is of remission and recovery. However, the interpretation of one's experience in particular negative ways contributes to the maintenance or exacerbation of depressive symptoms. These interpretations depend on the nature of the representations in memory activated by depressed mood. The DAH assumes that during early episodes of depression, certain patterns of processing are established (Segal, Williams, Teasdale, & Gemar, 1996; Teasdale & Barnard, 1993) thereby establishing an association between depressed mood and negative thinking patterns such that subsequent depressed mood, however caused, will re-activate the negative thinking patterns. In addition, depressed mood is hypothesized to activate negative biases in information processing (Gotlib & MacLeod, 1997; Williams et al., 1997) that further contribute to negative interpretations of experience by influencing the information attended to, either external or from memory, and the interpretive categories used to interpret experience. In sum, if the activated negative constructs are sufficiently mild they will not support the persistence of depressed mood. However, if one's experience is interpreted as highly aversive and uncontrollable and/or, the negative self-referent constructs made accessible are global evaluations of oneself (e.g., failure, worthless), consistent with Beck's model, these interpretations are hypothesized to further exacerbate depressed mood. Thus, depressed mood and cognitive processing incrementally interact in a way that potentially results in a positive feedback loop.

The dynamic emphasis of the DAH means that it can account for the failure of previous studies (e.g., Hamilton & Abramson, 1983; Silverman et al., 1984; Simons et al., 1984) to find differences on the DAS between recovered depressed subjects and never depressed controls when these individuals were assessed during the experience of euthymic mood. According to the DAH, while the thinking of vulnerable individuals may be quite normal when not dysphoric, cognitive processing differences between recovered depressed subjects and never depressed controls will more likely be obtained if these individuals are assessed when experiencing a mild depressed mood. Specifically, this model predicts that DAS differences between these groups will only be revealed when individuals are experiencing a mildly depressed mood.

In sum, the DAH makes three general assumptions. First, it is hypothesized that depressed mood negatively biases information processing thereby increasing the accessibility of depressogenic interpretations of experience. Second, as a result of these mood effects on cognitive processing, increased negative interpretations of events would produce further depression. Third, this theory assumes individual differences regarding the nature of thinking patterns activated by depressed mood related to differences in the experiences, and their interpretations, which have previously produced depressed mood. In other words, links between dysphoric mood and negative thinking patterns will be stronger among individuals with a history of depression than those

who have never experienced depression. Therefore, if a depressive state activates significant negatively biased interpretations of experience, this can precipitate further negatively biased, self-referent information processing which promulgates a downward spiral of depression. In addition, the probability of this occurring is enhanced for those individuals who experience more global negative evaluations of oneself. On the other hand, in the absence of the activation of negative thinking patterns, an individual might experience an appropriate level of distress to an event, but would not spiral into depression.

While there is significant empirical support for the DAH, of particular interest is whether dysphoric mood induced increases in negative information processing, known as "cognitive reactivity", in fact represent a risk factor in recurrent depression. Kraemer et al. (1997) have proposed four specific criteria for defining risk factor status in order to increase the consistency and precision with which this term is used: (1) Is the factor associated with the outcome—i.e., is it a *correlate*? (2) Does the factor precede the outcome—i.e., is it a *risk factor*? (3) Can the factor change or be changed—i.e., is it a *variable* risk factor? and, (4) Does manipulation of the factor change the outcome—i.e., is it a *causal* risk factor? In the discussion that follows these criteria will be used to evaluate the status of cognitive reactivity as a risk factor for depressive relapse.

3.1. Is cognitive reactivity associated with increased risk for depressive relapse?

The first criterion to be met is whether cognitive reactivity is in fact associated with increased risk for depressive relapse. The key to testing this assumption is to assess the cognitive content and processes of individuals who have recovered from depression under conditions that are likely to activate negative self-referent cognitive structures, that is, sad or depressed mood. A small but significant number of cross-sectional studies have examined the relationship between cognitive reactivity and increased relapse risk. Most of these studies involved comparing individuals who had recovered from depression versus never-depressed controls on various cognitive measures before and during the induction of a sad mood.

While initial mood priming studies were unsuccessful in inducing sad mood (Blackburn & Smyth, 1985; Gotlib & Cane, 1987) rendering their results inconclusive with respect to assessing the validity of the DAH, subsequent manipulations were more successful. Teasdale and Dent (1987) first reported an adequate mood induction in formerly depressed (FD) and never-depressed (ND) groups of women who were assessed on an incidental recall paradigm under conditions of normal versus induced sad mood. While there were no performance differences on this task between the two groups under normal mood conditions, the FD group demonstrated better recall of negative adjectives that had been previously endorsed as self-descriptive than ND controls at similar levels of induced depressed mood. These results are consistent with the DAH; that negative constructs and processes are associated with increased risk for depression but are active and detectable only under conditions of depressed mood.

Similarly, while initial studies comparing FD and ND individuals when euthymic did not reveal groups differences on the DAS, more recent studies relying on a mood priming methodology as described above have generally provided analogous results (Miranda, Gross, Persons, & Hahn, 1998; Miranda & Persons, 1988; Segal, Gemar, & Williams, 1999; Segal et al., 2003 but see Dykman, 1997 and Dozois & Dobson, 2001b for exceptions). Miranda and Persons

(1988) demonstrated a significant correlation between the reporting of dysfunctional attitudes and dysphoric mood in FD participants but not in ND controls experiencing similar levels of sadness during a negative mood induction; FD individuals increasingly endorsed dysfunctional attitudes as negative mood increased whereas for ND participants, no relationship between level of endorsement of dysfunctional attitudes and intensity of negative mood was found. Miranda et al. (1998) reported a similar relationship between dysphoric mood and dysfunctional attitudes for formerly depressed women using a film-based negative mood induction.

Segal and colleagues (1999, 2003) replicated these findings in two studies designed to evaluate whether different treatment modalities produced differences in cognitive reactivity following a negative mood induction and whether cognitive reactivity predicted depressive relapse. In these studies, remitted depressed patients who recovered either through pharmacotherapy (PT) or cognitive behaviour therapy (CBT) were assessed on the DAS before and after a negative mood induction. Participants who recovered through PT demonstrated increased DAS scores in response to increased sad mood. This is similar to the results reported in other studies using the DAS where presumably a good number of individuals recovered from depression via pharmacotherapy. Conversely, participants who recovered through CBT demonstrated no change in DAS scores in response to increased sad mood. The differential finding for the CBT treated group has important implications with respect to whether cognitive reactivity is a variable risk factor and is discussed further below.

In contrast, Dykman (1997) failed to replicate a relationship between dysphoric mood and endorsement of dysfunctional attitudes using a Velten (1968) Mood Induction Procedure in FD and ND participants. This failure to replicate must be understood in the context of the studies demonstrating mood activated dysfunctional information processing and may simply be due to methodological flaws related to the mood induction procedure (Ingram, Miranda, & Segal, 1998).

Diverging from the priming methodology altogether, Miranda, Persons, and Byers (1990) assessed FD and ND participants on the DAS during the course of diurnal mood variations. They found that DAS scores increased with negative mood only for the FD participants. Lewinsohn et al. (1999) provided convergent results by analysing the correlation between naturally occurring dysphoric mood and DAS scores in groups of adolescents with or without a history of MDD. This analysis revealed that the correlation between dysphoric mood and DAS scores was significantly larger for those adolescents with a previous history of MDD suggesting that at similar levels of dysphoric mood the FD adolescents endorsed higher DAS scores. Finally, Dozois & Dobson (2001b) assessed a group of clinically depressed individuals on two information processing and two negative schema indices when depressed and 6 months later following remission subsequent to pharmocotherapy. The results showed that information processing improved with remission as expected; however, the negative schema indices remained stable. Thus, it may be that while cognitive processes and products are activated by depressed mood, negative cognitive structures are in fact present in FD individuals when euthymic.

In sum, across a majority of studies using different methods to prime activation of dysfunctional cognitive processes, formerly depressed individuals do demonstrate distinctly depressotypic attitudes but only when assessed under conditions of depressed mood. Furthermore, these results may represent just one of a variety of cognitive processes that are influenced by mood. One way of categorizing the variety of cognitive processes draws on work in cognitive

psychology (e.g., Hasher & Zacks, 1979; Shiffrin & Schneider, 1977), which distinguishes between effortful and automatic processes. Whereas effortful processes require attention to operate and are deliberative in nature (e.g., incidental recall, DAS), automatic processes do not use attentional resources and are not controlled by the individual. This distinction has proved valuable in efforts to understand the cognitive processes of acute depression (e.g., Hartlage, Alloy, Vasquez, & Dykman, 1993). Thus, it might prove useful to include measures of more automatic processing to determine the extent of mood related changes in cognitive processing.

Studies using measures of automatic rather than effortful processes have generally provided comparable results (Gemar, Segal, Sagrati, & Kennedy, 2001; Ingram, Bernet, & McLaughlin, 1994; Ingram & Ritter, 2000, but see Rude, Covich, Jarrold, Hedlund, & Zentner, 2001 for an exception). Gemar et al. (2001) used the implicit association test (IAT) (Greenwald, McGhee, & Schwartz, 1998), which is well suited to examine automatic biases in the processing of self and non-self information, to assess FD versus ND groups before and after a negative mood induction. Formerly depressed individuals showed a greater negative evaluative bias for self-referent information when in a sad mood as compared to controls. Moreover, the magnitude of the shift in evaluative bias was similar to that observed for a group of currently depressed individuals suggesting that even a mild sad mood may reinstantiate some of the cognitive features of clinical depression.

Ingram and colleagues (1994, 2000) used a dichotic listening task to assess attentional allocation in formerly and never-depressed subjects under conditions of normal and sad mood. In this paradigm, subjects heard a story in one ear while distractor words, either positive or negative adjectives, were presented in the other ear. While no differences in attentional allocation were observed between groups in the normal mood condition, an attentional bias for negative adjectives was observed for the FD subjects in the negative mood condition.

Rude et al. (2001) provided an exception to these results when they failed to demonstrate any mood related changes in attentional bias using the Stroop color-naming task for FD participants. Given positive findings with other measures of automatic processes, it may be that the Stroop does not capture this effect. Thus, in three of four studies, FD individuals demonstrated mood-linked changes in automatic processing for general and self-referent negative information.

In summary, individuals at risk for depressive relapse do show increased accessibility and activation of negative thinking patterns as demonstrated by measures assessing cognitive processes, both effortful and automatic, with a few exceptions. Specifically, mood-linked cognitive reactivity is evidenced by increased negative cognition (DAS scores; e.g., Miranda & Persons, 1988; Miranda et al., 1990, 1998; Segal et al., 1999, 2003) and by increased encoding and retrieval of negative information (Teasdale & Dent, 1987). With respect to more automatic processes, mood-linked cognitive reactivity has been demonstrated for formerly depressed individuals on the dichotic listening task (Ingram et al., 1994, 2000) and the implicit association task (Gemar et al., 2001) but not the Stroop (Rude et al., 2001). Taken together, there is sufficient evidence to establish mood-linked cognitive reactivity as a *correlate* of vulnerability to depressive relapse.

3.2. Cognitive reactivity as an independent predictor of depressive relapse

The second criterion for evaluating risk status is to determine whether mood-linked cognitive reactivity precedes recurrence. This is the second assumption of the DAH: the information processing patterns activated in depressed mood will determine whether this state will escalate to the level of major depression. Longitudinal studies addressing this assumption of the DAH have relied primarily on measures of effortful cognitive processes under conditions of naturally occurring or induced sad mood.

Mood priming studies have generally supported a link between mood-linked cognitive reactivity and depressive relapse (Segal et al., 1999, 2003; Williams, 1988). As described above, Segal and colleagues conducted two studies to determine whether the degree of cognitive reactivity (increase in DAS scores) following a negative mood induction would predict risk for depressive relapse following successful treatment with PT or CBT. The results of these two studies showed that regardless of which treatment was provided, the degree of cognitive reactivity was an independent predictor of subsequent relapse over the next several years.

Williams (1988) obtained similar results by assessing the recall of positive and negative self-referent adjectives under conditions of neutral and induced sad mood in a student sample. Differential recall of positive versus negative words observed in neutral mood did not predict depression status over the following year. However, differential recall observed under conditions of induced sad mood did predict the occurrence of depression. These results, however, provide only indirect support as the outcome measure was not specifically a recurrence of a depressive episode.

Additional direct support has been provided from studies evaluating the relationship between naturally occurring mild depressed mood and scores on questionnaire measures of negative cognition (typically the DAS) (Evans et al., 1985; Lewinsohn et al., 1999; Simons, Murphy, Levine, & Wetzel, 1986). Evans et al. (1985) and Simons et al. (1986) administered cognitive measures to individuals shortly after they achieved clinical remission but where depression measures indicated mild residual levels of depressed mood. Simons et al. (1986) administered the DAS to patients successfully treated with pharmacotherapy, CT or a combination of the two. DAS scores at termination were an independent predictor of subsequent recurrence in the following year. Similarly, Evans et al. (1985) administered a composite measure of negative cognition (which included the DAS) to remitted depressed patients to show that scores on the composite measure predicted relapse over a two-year period. Lewinsohn et al. (1999) used a longitudinal analysis of an adolescent cohort to demonstrate that elevated dysphoria levels and dysfunctional thinking (as assessed by the DAS) were significantly associated with increased relapse risk in adolescents with a prior history of MDD whereas there was no association between these three variables in a group of adolescents with no prior history of depression. Together, these results establish an important link between mood-related changes in measures of more effortful cognitive processes and relapse risk.

There are no studies, however, that we are aware of directly examining the relationship between cognitive measures of automatic processes and subsequent relapse risk. However, Gemar et al. (2001) failed to demonstrate a relationship between mood-linked changes on the DAS (which were shown to predict depressive relapse) and IAT. If mood related changes in the

IAT are associated with depressive relapse, it must be assumed that this relationship is different from that demonstrated for the DAS.

In sum, these studies provide a convergent set of results supporting the DAH; questionnaire and experimental measures of increased negative cognition manifest during induced sad mood or a naturally occurring mild depressed mood are predictive of depressive recurrence. One limitation of these studies, however, is that they did not ascertain the degree to which cognitive reactivity uniquely predicts depressive relapse. For example, Martin (1985) has proposed neuroticism as a cognitive mechanism for depression, although Teasdale and Dent (1987) suggest the possibility that these two components of cognitive vulnerability to depression may not be mutually exclusive. Despite this caveat, as Kraemer et al. (1997) suggest, mood-linked cognitive reactivity as manifest by effortful cognitive processes fulfills the second criteria and therefore can be considered a *risk factor* for depressive relapse.

3.3. Modifiability of cognitive reactivity

The third criterion is whether a particular factor can change or be changed thereby characterizing a risk factor as a *variable* risk factor. The limited evidence addressing this question comes from the studies by Segal and colleagues (1999, 2003) described above where FD patients who recovered either through CBT or PT completed the DAS before and after a negative mood induction procedure. Segal et al. (1999) demonstrated that while both PT and CBT groups did not differ on pre-mood induction DAS scores, recovered PT participants demonstrated a significant increase in their DAS scores as compared to recovered CBT patients who showed no change in DAS scores at similar levels of sad mood. Given that the increase in DAS scores for PT treated participants is similar to that observed in a number of previous studies, it appears that CBT, or psychotherapy in general, somehow mitigated the cognitive effects of the mood challenge. This is consistent with the view that CBT reduces relapse risk by teaching individuals specific skills to identify and respond to affective triggers (Barber & DeRubeis, 1989; Jarrett & Kraft, 1997). Important limitations of this study are that patients were not randomly assigned to treatment, and pre-treatment levels of cognitive reactivity were not assessed leaving open the possibility that the two groups already differed with respect to mood-linked cognitive reactivity prior to receiving treatment. Nevertheless, Segal et al. (2003) replicated these results in a study specifically designed to address these limitations by including random assignment to treatment. Thus, cognitive reactivity can be considered a *variable* risk factor as there is evidence that it can be reduced. This contrasts with number of past depressive episodes as a fixed risk factor, about which little can be modified.

3.4. Does changing cognitive reactivity change subsequent risk for depressive relapse?

The fourth criterion is to determine if manipulation of the factor alters the outcome. In other words, does reducing cognitive reactivity result in a reduction in relapse risk? Ideally, answering this question would require assessing baseline cognitive reactivity in a group of individuals at a similar level of risk for depressive relapse, randomly assigning individuals to either a treatment that would change cognitive reactivity (e.g., CBT) or a control group, reassessing cognitive reactivity and then comparing relapse rates between the two groups. An approximation of this study

design comes from the study by Segal and colleagues (2003) described above investigating whether cognitive reactivity changes as a function of treatment and whether it predicts subsequent relapse. Although baseline cognitive reactivity was not assessed in this study, CBT was associated with a reduction in mood-induced cognitive reactivity as compared to PT and reduced cognitive reactivity was associated with reduced risk for relapse. Similar results were demonstrated by Segal et al. (1999) without random assignment to treatment. Both of these studies presume that cognitive reactivity was reduced in the CBT treated group and stayed the same in the PT group. Violation of this assumption would negate the relevance of these studies to this criterion. However, if this assumption is correct, then these results provide evidence supporting cognitive reactivity as a *causal* risk factor for depressive relapse.

Additional indirect support for a link between cognitive reactivity and relapse risk comes from two sources, both of which manipulated the ability to "decenter" or "disidentify" with one's negative thoughts and feelings. While the concept of decentering has been recognized in discussions of CT (e.g., Beck et al., 1979), it typically has been viewed by cognitive therapists as simply a means to the end of thought content change. More recently, however, a "third wave" of cognitive therapy suggests a more central role of changing one's relationship to cognitive content (e.g., Ingram & Hollon, 1986). In particular, Teasdale and colleagues (Teasdale, 1997a,b; Teasdale & Barnard, 1993; Teasdale, Segal, & Williams, 1995) have emphasized the importance of the relationship to one's thoughts and feelings in determining the impact of mood induced activation of negative thinking patterns, that is, cognitive reactivity. Specifically, when one identifies personally with negative thoughts and feelings, these experiences will have a much stronger impact when experienced as "me" or "reality". Conversely, a shift in one's cognitive perspective known as "decentering" or "disidentification", where one relates "to their negative experiences as mental events in a wider context or field of awareness" (Teasdale et al., 2002: p. 276) might mean that negative experiences activated by dysphoric mood will be less likely to lead to depressive relapse.

First, Watkins, Teasdale, and Williams (2003) recently completed a study that provides indirect support for the notion that the ability to decenter may reduce the maintenance of depressed mood. Building on previous research utilizing sad mood inductions, Watkins et al. (2003) examined whether cognitive manipulations could affect the maintenance of induced sad mood in never-depressed participants. In this study, they used scrambled Velten sentences as mood-maintaining primes which participants were required to unscramble. Questions designed to either increase a wider awareness of temporal and personal context pertaining to the individual's current mood (e.g., "How long does any mood last?") versus control questions which made no reference to mood (e.g., "How long does this weather last?") were embedded into the list of scrambled sentences. Contextual questions lead to a reduction in the length of a sad mood induction compared to control questions. While this study does not provide direct evidence that reduced cognitive reactivity leads to a reduction in depressive relapse, the results are consistent with the notion that increased contextual awareness may reduce activation of mood-linked negative representations.

Second, recent studies by Teasdale and colleagues (2002) demonstrated that changes in the ability to decenter from negative thinking, also known as "metacognitive awareness" are associated with reduced relapse risk. Teasdale et al. (2002) conducted two studies to evaluate whether improvements in metacognitive awareness mediated the reduction in relapse risk

resulting from either of two psychological treatments, CBT or mindfulness-based cognitive therapy (MBCT). In these studies, metacognitive awareness was measured using the Measure of Awareness and Coping in Autobiographical Memory (MACAM) (Moore, Hayhurst, & Teasdale, 1996). The MACAM involves analyzing the autobiographical memories stimulated by depression-related cues and measures the ability to see negative thoughts and feelings as passing mental events rather than as an aspect of self.

This first of the two studies was done as part of a clinical trial designed to determine whether CT could reduce relapse in depressed individuals who achieved partial remission with pharmacotherapy (Paykel et al., 1999). Patients who had achieved partial remission of their depression with PT were randomized to receive a 20-week treatment of PT with either clinical management alone or together with CT. The analysis of MACAM scores in CT treated individuals showed that CT reduced relapse through its effects in increasing metacognitive awareness. However, this analysis did not rule out the alternative explanation that the changes in metacognitive awareness were correlates of effective belief change. This alternative explanation was specifically addressed by examining MACAM scores in a clinical trial evaluating the efficacy of a new relapse prevention treatment, Mindfulness-based Cognitive Therapy (MBCT; Teasdale et al., 2000). MBCT was designed to prevent relapse by increasing metacognitive awareness without any explicit attempt to change negative thinking itself. Similar to the findings of the first study, increased metacognitive awareness was associated with MBCT related reduction in relapse risk. A limitation of both studies is that a full mediational analysis to demonstrate the causal status of metacognitive awareness change in the reduction of relapse risk could not be done.

In conclusion, the evidence to date is supportive of cognitive reactivity as a *causal* risk factor for depressive relapse. In addition to this research, more recently the DAH model has been applied to the problem of suicidal relapse/recurrence.

4. A differential activation model of suicidal relapse/recurrence

Suicidal behaviour is one of the most serious outcomes of psychiatric illness. One in seven patients hospitalised for major depression and one in 10 patients with a diagnosis of schizophrenia or alcohol abuse die by suicide (Powell, Geddes, Deeks, Goldacre, & Hawton, 2000). Reducing suicide risk involves improving the outcomes in those groups known to be at most risk for suicidal ideation and behaviour (Appleby, Dennehy, Thomas, Faragher, & Lewis, 1999). There is a great deal of evidence that at-risk individuals have a history of childhood adversity, often involving abuse (Coll, Law, Tobias, & Hawton, 1998; Williams & Pollock, 2001). But research also shows that whether such a history predicts later suicidal behaviour is determined by the occurrence of subsequent psychiatric disorder, most commonly depression (Molnar, Berkman, & Buka, 2001). Consistent with this conclusion, it has been found that the Population Attributable Ratio (PAR) for depression in serious but non-fatal suicidal behaviour (that proportion of suicidal behaviour that would be removed if depression were taken out of the picture) is 80% (Beautrais et al., 1996).

However, within depressed patients, most do not attempt or commit suicide. One needs to look for additional risk factors. However, many such risk factors are sociodemographic (e.g. sex, social class) or historical (e.g. previous psychiatric treatment or previous deliberate self

harm (DSH)) and do not readily lead to any implications for intervention for which we would need to know the risk mechanisms (Sakinofsky, 2000). Only with better knowledge of these underlying mechanisms will we be able to determine which individuals that appear to be at low risk are nevertheless vulnerable, and how best to intervene with both these and those known to be at high risk.

Our current research is testing hypotheses derived from the Teasdale's differential activation hypothesis, examining the extent to which suicidality follows the same pattern that governs relapse and recurrence in depression. As we have seen, the DAH assumes that during early episodes of depression, certain patterns (''configurations'') of processing are established (Segal et al., 1996; Teasdale & Barnard, 1993). We suggest that suicidal ideation arises as part of such patterns of thinking during early episodes and, during these episodes, an association is formed between depressed mood and suicidal ideation such that future depression, however caused, will activate suicidal ideas which are more and more easily re-synthesized each time they are activated. The particular negative cognitions selectively primed in these individuals include, for example, ''I do not deserve to live'', ''I cannot tolerate this pain anymore, it is unbearable'', and ''I have nothing to look forward to''. They result in the establishment of a holistic, implicit sense corresponding to hopelessness themes such as ''myself as a burden to others'', ''my problems as insolvable'' and ''my life as unendurable'' that combine *to produce* a profound sense of defeat with a sense of entrapment (Williams & Pollock, 2001). The fact that it is a whole constellation of thinking that is associated with suicidal ideation is consistent with data that shows that such thinking affects many aspects of information processing, observed both on questionnaire measures (such as measures of hopelessness and dichotomous thinking, Williams & Pollock, 2000), as well as on cognitive tasks assessing attentional bias (Williams & Broadbent, 1986a), or memory bias (Evans, Williams, O'Loughlin, & Howells, 1992; Williams & Broadbent, 1986b). Risk of further suicidal ideation and behaviour, therefore, arises from the ease with which these patterns of processing become re-established, following recovery, in the face of mild negative affect or autobiographical material.

According to this view, because even mild negative affect, or fragments of autobiographical material, can re-trigger these configurations, later episodes of suicidal ideation and behaviour will require less external triggers, analogous to the kindling and sensitisation models in depression (Kendler et al., 2000; Post, 1992; Van Heeringen, Williams, & Hawton, 2000).

There are few studies that have addressed such a hypothesis (see later). However, there are data that are consistent with it. First, such a model would predict that attempted suicide would become more likely, the greater the number of previous episodes of psychiatric illness. Consistent with this suggestion, Malone et al. (2000) and Mann, Waternaux, Haas, and Malone (1999) found that a larger number of previous episodes increased the likelihood that a psychiatric patient had attempted suicide. Second, the differential activation model would predict that suicidal patients in a psychiatric sample should have a longer history of illness than non-suicidal patients. Mann et al. (1999) found that patients who had attempted suicide had a younger age of onset of their psychiatric illness (23 years) than those who had not attempted suicide (27 years). Third, the model would predict that the cognitive-affective spiral will be more likely to be activated if a larger range of disturbances in thought, feeling and behaviour has occurred alongside, and had the opportunity to become associated with, depressed mood in the past. Consistent with this, Brodsky, Malone, Ellis, Dulit, and Mann (1997) found that Cluster B

personality disorder correlated with both the number of lifetime attempts, and the age at first attempt. Kessler, Borges, and Walters (1999) found that both the likelihood and the seriousness of suicide attempts was related to the number of different psychiatric disorders experienced by an individual, (with three or more diagnoses bringing 19.7 times the risk).

Finally, kindling and sensitisation effects may explain the discrepancy between different studies of life events in suicidal behaviour, some of which (e.g. Beautrais et al., 1996) find an association between negative events and suicidal behaviour, and others of which (e.g. Mann et al., 1999) do not. These and other studies of life events in DSH do not take account of how the number of previous episodes of major depression or the number of previous suicidal episodes might affect the association between life events and suicidal behaviour. Van Heeringen et al. (2000) have suggested that early episodes of DSH would be associated with life events, but later episodes would not; the exception being those patients for whom previous life stress has been so severe (for instance those with child sexual abuse) where the evidence suggests that the association of depressive onset with life events, even for the first few episodes is weaker—that they have, in effect, been "pre-kindled" (Kendler et al., 2001).

Although, to our knowledge, there have been no prospective studies of a differential activation model in the field of suicide, there have been two that speak to this issue. The first, by Joiner and Rudd (2000), found that individuals with no more than one previous suicide attempt showed a significant correlation between strength of suicide ideation and the cumulative index of life stress from the previous 12 months. Those patients who had a history of multiple episodes of self-harm showed no such relationship. As a cross-sectional study, however, it is possible that the multiple attempters represent a different sub-population from those with few or no attempts. The second study (Clark, Gibbons, Fawcett, & Scheftner, 1989) examined whether suicidal behaviour (within a two year follow-up period) was best modelled mathematically by a "state-dependent" or a "separate populations" model. Whilst the findings suggested that a "separate populations" model provided the best fit to the data, patients (who included those with unipolar, bipolar and schizoaffective disorders and individuals with substance misuse) were not followed from their first attempts, rates of attempts and repetition of attempts were low and number of previous attempts was not considered as a factor that might influence patterns of repetition. Thus, the findings cannot be considered conclusive. Our current research is examining hypotheses derived from the DAH, a model that is generating novel ways to look at suicidal behaviour and an excellent framework for testing new treatment approaches for this difficult group.

5. Conclusion

The hallmark of a good theory is the extent to which it generates productive avenues of research. Against this benchmark, Teasdale's DAH has already led to important insights not just in explaining the mechanism that underlies patient's risk for relapse and suicidal ideation but has led to the development of prophylactic treatments based on actively addressing this mechanism (e.g., MBCT). Specifically, evidence to date supports cognitive reactivity as a *variable* risk factor for depressive relapse with significant potency.

Future research based on more rigorous experimental designs confirming cognitive reactivity as a causal risk factor would be an important step in moving toward a better understanding and management of depressive relapse. This designation would provide support for future efforts geared towards the design and implementation of relapse prevention programs geared to reducing cognitive reactivity. In particular, the delineation of the mechanism(s) underlying cognitive reactivity as well as the mechanisms of CT or MBCT that contribute to changing cognitive reactivity would be logical next steps. Preliminary evidence from this work is supportive and we await further results of this innovative integration of clinical science with clinical care.

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