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Leading Article

NEW POSSIBILITIES IN COGNITIVE THERAPY FOR DEPRESSION?

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Abstract. This article argues for the benefits of a revision of cognitive therapy for depression around two main points. First, compared to recently developed models for other disorders, our knowledge of *cognitive content* in depression is out of date and attracting little research, as if there is no more to be learned about *what* depressed people think and feel. Recent trends to challenge cognitive processes, without addressing the relevant content, might therefore meet with limited success, depending on how the content and processes are linked. Second, re-reading Beck et al. (1979) suggests the importance of exploring *the meanings attached to precipitating events*, a cognitive strategy that has fallen into the background, is probably used in an ad hoc fashion, and needs to be used more systematically to improve clinical effectiveness.

Keywords: Depression, content specificity, cognitive change, precipitating events.

There is little doubt that Beck's cognitive model of depression and the associated therapy are well-established. In the 20 or so years since Beck, Rush, Shaw and Emery's (1979) seminal work, experimental and observational studies of depressed people have explored the validity of the model, and many of its claims have received broad support (Haaga, Dyck, & Ernst, 1991). Similarly, a range of clinical trials (e.g., Blackburn, Bishop, Glen, Whalley, & Christie, 1981; Elkin et al., 1989; Shapiro, Barkham, Hardy, & Morrison, 1990) have supported the model's efficacy as a focal psychotherapy. Cognitive therapy (CT) for depression is at least as effective as other psychotherapies, and has a key advantage over pharmacological interventions: it moderates to some degree the likelihood of future relapse or recurrence (DeRubeis et al., 1990). It has also provided the foundation for numerous new cognitive models of other emotional disorders (e.g. Clark & Wells, 1995).

With selective attention, we could easily jump to the conclusion that clinical problems involving depression are now well-managed; the glass is at least half full. Of course, this is very far from the truth. In clinical practice, a significant proportion of depressed people either reject cognitive techniques, fail to respond to treatment, relapse

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soon after therapy, or go on to experience recurrent depressive episodes (Paykel & Priest, 1992). From the point of offering cognitive therapy, clinical evidence suggests that only around 50% will complete a course of CT, benefit from the intervention, and then sustain their gains over the following 2 years – from here the glass appears half empty.

Because depression was the pioneering disorder in CT, there is a danger of thinking that further improvements are unnecessary or impossible. In fact, CT for depression seems to have been left behind when compared to more recently developed models of other disorders, in particular concerning their *content specificity*. Identifying the specific content of thoughts that trigger and then maintain these other disorders has allowed greater precision in targeting cognitive change. For instance in panic disorder, specific beliefs about the likelihood of bodily sensations resulting in heart attack are a focus for reappraisal (Clark et al., 1994), and in social phobias, key cognitions are those concerning one's appearance to other people (Clark & Wells, 1995).

Most content specificity research in depression has been broadly supportive of Beck's (1970) hypothesis (that is, the negative cognitive triad of self, world, and future) but has not extended or refined the original model (Beck, Brown, Steer, Eidelson, & Riskind, 1987). As a result, our beliefs about *what* depressed people think are no more advanced than they were 20 years ago (Barton & Morley, in press). Related to this is whether we understand the crucial difference between negative and depressive thinking. It is straightforward to identify negative thoughts about the self, world and future that are clearly not depressive (e.g., "I am not much good at most sport"; "The world is facing difficult social problems"; "Next week is going to be really stressful"), begging the question of how often cognitive therapists inadvertently challenge thoughts unrelated to depression and irrelevant to therapeutic gain.

How this status quo emerged may in part be answered by Beck et al.'s (1979) presentation of their original model having three distinct aspects; schemas (enduring assumptions about self in the world), thinking errors (information processing biases), and negative automatic thoughts (cognitive products). Since then, most depression research has investigated and described these levels separately, underlining the impression that they are distinct elements simultaneously activated during depressive states. As a result, we still lack a useful model of how these elements interact and drive each other. Understanding how they link would simplify the model, not complicate it, hence recent advances towards integration are welcome, most prominently Teasdale and Barnard's (1993) ICS framework. However, their focus on cognitive architecture and sub-systems emphasizes the primacy of changing processes rather than cognitive content.

An alternative view is to modify cognitive content as a means of switching out of depressed, interlocked processes: trying to alter processing without dealing with the content risks rekindling the depression at a later date. From this perspective, the content of depressive cognition is the particular material that creates vulnerability in the first instance, interacts with losses, failures or stressors, and ultimately drives self-defeating processing biases; it's not simply the final product.

Equally important here is the issue of generalization. Because depression is a *spreading disorder*, cognitions concerning specific losses, failures or stressors can easily become obscured in the cloud of resulting negative thoughts. The global, over-generalized thinking that characterizes depression can blur the importance of meanings attached

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to precipitating events. This reflects the clinical distinction between people who present with depression "about X", and people who simply present with depression. As therapists we must not be drawn into the illusion that there is no cause or source, as if depression appears and disappears spontaneously, and all we can offer is a challenge to people's symptomatic beliefs.

Re-reading Beck et al. (1979) reveals a wealth of clinical examples in which *inferences about precipitating events* form the focus of therapeutic change, and this is something we need to rediscover and refine, especially if we are to capitalize on the rich potential of diathesis-stress frameworks (Champion & Power, 1995). Research suggests that much depressive thinking has a maintaining rather than a causal effect (Haaga, et al., 1991), so attempting to modify the majority of negative thoughts is a poor kind of fire-fighting, far from the heat-source. A change in focus which returns to identifying, modelling and challenging the content of hot cognitions that *are* causal to depression would ensure better targeting of cognitive change and therefore better treatment outcomes.

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