Available online at www.sciencedirect.com
ScienceDirect
Behaviour Research and Therapy 44 (2006) 1481–1490
BEHAVIOUR RESEARCH AND THERAPY
www.elsevier.com/locate/brat

Reduced specificity of autobiographical memories following a negative mood induction

Cecilia Au Yeung, Tim Dalgleish*, Ann-Marie Golden, Patricia Schartau

Emotion Research Group, Medical Research Council Cognition and Brain Sciences Unit, Cambridge, UK

Received 3 March 2005; received in revised form 7 September 2005; accepted 26 October 2005

Abstract

Reduced autobiographical memory specificity (AMS) to emotional and neutral cue words appears to be a stable cognitive marker of clinical depression. For example, reduced AMS is present in remitted/recovered depressed patients and shows no reliable relationship with current levels of depressed mood in correlational studies. The present study examined whether reduced AMS could be induced in healthy volunteers with no history of depression, using a negative mood manipulation and whether levels of AMS and induced mood were positively correlated. Results showed a reduction in AMS following negative mood induction, compared to a neutral induction, whereas positive mood induction had no effects on AMS. Furthermore, lower happiness following the induction phase correlated positively with reduced AMS, and the extent of happiness reduction from pre- to post-induction correlated positively with reduction in AMS. These results suggest that AMS is, at least in part, a function of current emotion state. The implications for the literature on AMS as a stable marker of clinical depression are discussed.

Keywords: Depression; Autobiographical memory; Mood induction; AMT

Introduction

When prompted to generate detailed and specific (in time and place) autobiographical memories in response to cue words (the autobiographical memory test (AMT); Williams & Broadbent, 1986), people sometimes find it difficult to produce suitably *specific* responses, instead generating overly general summaries of their past. So, for example, the cue "summer" might prompt the generic recollection "I enjoyed every summer when I was a child", instead of the more specific "I remember the summer's day that we went to Disneyland". Williams and Broadbent (1986) discovered that reduced autobiographical memory specificity (AMS) was more common in depressed parasuicide patients than in matched controls. Since this initial finding, reduced AMS has been found to be a characteristic of performance on the AMT in individuals suffering from clinical depression (e.g. Brittlebank, Scott, Williams, & Ferrier, 1993; Dalgleish, Spinks, Yiend, & Kuyken, 2001; Kuyken & Dalgleish, 1995; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001), posttraumatic stress disorder (PTSD; e.g.

*Corresponding author. Tel.: +44 1223 355 294x630; fax: +44 1223 359 062.
E-mail address: tim.dalgleish@mrc-cbu.cam.ac.uk (T. Dalgleish).

0005-7967/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2005.10.011

McNally, Lasko, Macklin, & Pitman, 1995), acute stress disorder (Harvey, Bryant, & Dang, 1998), and eating disorders (Dalgleish et al., 2003), though not, for example, generalized anxiety disorder (Burke & Mathews, 1992).

Mackinger, Pachinger, Leibetseder, and Fartacek (2000) also showed that *recovered* clinically depressed patients exhibited reduced AMS, relative to never-depressed controls, matched on level of depressive symptoms over the previous week. Related to this, a number of studies have shown no reliable relationship between past-week levels of depression symptoms (on self-report questionnaires) and AMS (e.g. Dalgleish et al., 2001; Wessel et al., 2001). These data have been taken as evidence that reduced AMS is a *stable* marker for a vulnerability to clinical depression (and potentially other disorders also), rather than simply a function of current mood state (Mackinger et al., 2000). Stable markers of depression are particularly important as they may potentially reveal ways in which asymptomatic depression-vulnerable individuals represent or process emotional information differently from their never-depressed peers. These differences might therefore provide a window into why depression-vulnerable people relapse into depression (or indeed develop the disorder in the first place). Consequently, targeting such differences therapeutically represents a promising relapse-prevention strategy (e.g. Teasdale et al., 2001).

However, data from the three published studies that have examined the effects of induced mood on the AMS paint a slightly different picture regarding AMS as a strictly stable marker of depression (Maccallum, McConkey, Bryant, & Barnier, 2000; McBride & Cappeliez, 2004, Expt. One; Svaldi & Mackinger, 2003). Maccallum et al. (2000) showed that hypnotically induced negative mood led to reduced AMS relative to induced neutral or positive mood. Svaldi and Mackinger (2003) reported similar findings in response to a musical mood induction allied to remembering and reflecting on a negative autobiographical event. However, McBride and Cappeliez (2004) found no effects on AMS of elated or depressed mood inductions, using a Velten procedure. These studies would therefore seem to indicate that the data are equivocal as to whether AMS can be simply a function of current mood *state*. There seem to be 4 possible explanations of these data and the literature on AMS as a stable marker.

One possible explanation is that the AMS effect is *multifaceted*, with one or more facets that are mood-state dependent (independent of any history of clinical depression), and one or more facets that are a stable function of a history of clinical depression. This would mean that any mood-induced AMS effects could sometimes be detectable but could also be 'washed out' as a function of differential levels of depression history across groups, leading to the mixed findings reviewed above.

A second possibility is that there are no pure effects of induced mood on AMS and that the existing positive results using mood induction procedures (Maccallum et al., 2000; Svaldi & Mackinger, 2003) were a function of depression history. This could have manifested itself in two ways. First, by chance, there could have been more individuals with a history of depression in the negative mood induction groups in these studies. Secondly, and more likely, proportions of recovered-depressed participants could have been broadly comparable across mood-induction conditions, but the induction of negative mood could have differentially elicited reduced AMS in the recovered-depressed individuals in the negative-mood induction, due to a process of differential activation (Lau, Segal, & Williams, 2004).

A third possibility is that the AMS effects in the mood-induction studies are related to *neither* depression history nor to induced mood. Instead, they could be due to differential priming across conditions whereby a negative induction procedure semantically primes generic negative representations thus leading to reduced AMS, independently of mood (e.g. negative mood primes concepts such as failure, helplessness, that are then given as generic responses to negative cue words on the AMT). This is particularly plausible in the Svaldi and Mackinger (2003) study where the negative mood induction led to decreased AMS only in response to negative cue words.

Finally, a more radical possibility is that AMS is *not* a stable marker for clinical depression at all but is *always* a function of mood state. This account would argue that the key studies cited in support of the stable marker hypothesis (e.g. Mackinger et al., 2000) have not detected the state-dependency of the AMS phenomenon for various methodological reasons. For example, although studies such as that by Mackinger et al. (2000) were careful to control for current levels of self-reported depression over the past week (using the Beck Depression Inventory (BDI); Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), they did not actually measure mood state per se at the time of experimental testing. It may therefore be