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Running head: Autobiographical memory specificity relates to current mood state

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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.

1. 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. 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 it is as yet unclear 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 (Macallum et al., 2000;

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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 the case that the experimental protocol involving the AMT, with its emphasis on emotive autobiographical material, induced a relatively more negative mood in the recovered

depressed participants (compared to the never-depressed controls), thus leading to reduced AMS in the depression-vulnerable group. This final account would need to propose that the null findings of McBride and Cappeliez (2004) using a mood induction procedure were anomalous, perhaps due to a low level of induced mood, or insufficient statistical power.

These different theoretical accounts are all rendered possible for several reasons. First, none of the existing 3 studies of mood induction and AMS (Maccallum et al., 2000; McBride & Cappeliez, 2004; Svaldi & Mackinger, 2003) endeavoured to control for history of depression in their samples. Furthermore, none of the studies examined the relationship between current depression levels and AMS in their participants, although in all three studies participants were group-matched on baseline levels of depression symptoms. Finally, none of these studies examined whether levels of AMS following the mood inductions were related to the participants' actual current emotional state. It therefore remains possible that some other aspect of the induction procedure, such as semantic priming of memory categories, is driving the key effects, independent of mood (the third explanation alone). There are other methodological problems with these studies also. For example, the induction effects on AMS in the Maccallum et al. (2000) study were only present in highly hypnotizable individuals and the Svaldi and Mackinger (2003) study protocol did not screen out participants who were currently depressed.

The primary aim of the present study was therefore to address the question of whether a negative mood-induction could bring about a relative reduction in AMS in healthy participants, even when levels of current *and past* depression were controlled for. A second aim of the study was to examine whether levels of AMS are significantly related to *current* emotional state, irrespective of their relationship to past-week levels of depression symptomatology. If this was the case, this would be clear evidence that reduced AMS could be a function of mood/emotion state *per se*.

Resolving such issues concerning the potential mood-state dependency of AMS is particularly important because, as well as its proclaimed status as a stable marker of clinical depression, reduced AMS seems to have an independent causal relationship in the maintenance of the disorder. For example, Brittlebank et al., (1993) found that reduced AMS predicted clinical recovery in Major Depressive Disorder (MDD) *over and above* initial levels of depressive symptoms. This finding has been replicated, for instance in Seasonal Affective Disorder (Dalgleish et al., 2001). Reduced AMS is therefore not simply a cognitive curiosity associated with clinical depression but a signature of some more fundamental underlying process implicated in maintenance and possibly onset.

The present study involved testing 3 groups of healthy participants on the AMT both before and after a mood manipulation. One group received a sad mood manipulation, one a happy manipulation, and the control group a neutral mood manipulation. We included the happy mood manipulation to investigate whether any effects of induced mood were valence-specific. Based on the possibility that reduced AMS is a function of acute negative mood states (Maccallum et al., 2000; Svaldi & Mackinger, 2003), our first experimental hypothesis was that the sad mood induction group would show reduced AMS (as indexed by a reduction in the number of specific memories following the mood manipulation, compared to the neutral comparison group (even after covarying for any differential effects of current depression levels [baseline BDI scores]. In contrast, we predicted that there would be no effects of the happy mood manipulation on AMS.

In order to verify that the relationship between mood induction and AMS was due to change in mood, rather than some other aspect of the induction procedure (e.g. semantic priming), our second hypothesis was that *change* in AMS from pre-post mood induction (across the sample) would correlate with *change* in mood state, again even after covarying baseline BDI scores.

Finally, to examine the possibility that reduced AMS may be a correlate of current negative mood/emotion state, irrespective of any relationship to depression symptoms over the previous week, our third experimental hypothesis was that levels of AMS, following the mood induction, would correlate with current mood state across the whole sample (again, even after covarying for baseline BDI scores), but may or may not show a significant simple association with baseline BDI scores themselves (cf. Wessel et al., 2001).

2. Method

2.1. Participants

Participants were recruited into the study from the department volunteer panel. Depression history was screened for in two ways: First, participants who, upon registering for the volunteer panel, described themselves as depressed or who reported any history of depression were not invited to participate in the study. Second, those participants invited to take part in the study were asked again if they ever been diagnosed (by their general practitioner or a mental health practitioner) with a past or current diagnosis of clinical depression or had ever been offered or prescribed anti-depressants. If this was the case, those participants were also screened out of the study.

Forty-eight people both agreed to participate and passed the screening criteria for the study. Of these 48, 45 attended the experimental testing session (age range: 17 to 40 years; M = 26.47; SD = 7.92; 33 females), and were randomly allocated to the 3 experimental conditions.

2.2. Materials and measures

2.2.1. Mood manipulation

To ensure that any effects of the mood inductions on AMS were not a function of a particular mood induction medium, half of the participants received a film mood induction and half a music mood induction (cf. Svaldi & Mackinger, 2003). The two types of media had comparable effects in manipulating mood (see below).

2.2.2. Film mood induction

Nineteen film clips (a mixture of positive, negative and neutral) were piloted on 8 participants (3 males; all between 21 and 27 years of age). After each film clip, participants were asked to rate how happy, distressed, sad, fearful, angry and disgusted they felt while watching the film clips, ranging from 1 ("not at all") to 9 ("very much so"). Three film clips were selected on the basis of these ratings. A film clip about the aftermath of a major earthquake with pictures of bodies and a focus on the grief of the survivors had the clearest mean rating of sadness over and above other emotions and was therefore used for the sad mood induction. A film clip about someone winning the lottery with plenty of expressed elation and happiness had the highest and most distinct mean ratings of happiness and was therefore used for the happy mood induction. Finally, an extract from a real estate agency promotion which did not elicit any significant emotions was used for the neutral mood manipulation. The happy and sad film clips were comparable in their degree of emotionality, though they differed in valence (Happy film: sadness rating - $M = 1.63$, $SD = 1.19$; happiness rating - $M = 6.88$, $SD = 2.17$. Sad film: sadness rating - $M = 6.50$, $SD = 1.16$; happiness rating - $M = 1.13$, $SD = 0.35$), and did not generate any other emotions to a marked extent. Film clips were all 2-3 minutes long and presented to participants on a 14 inch television monitor. Participants were encouraged to absorb themselves in the emotions that the films generated as much as they could.

2.2.3. Music clips

Established musical mood inductions were taken from the literature. The music for the sad mood induction was "Russia under the Mongolian Yoke" by Prokofiev, recorded at half

speed (e.g. Clark & Teasdale, 1985). For the elated mood induction we used the "Mazurka", from the ballet "Coppelia" by Delibes (Clark & Teasdale, 1985). For the neutral condition we used the Largo movement from "The New World Symphony" by Dvorak (Mecklenbraeuker & Hager, 1984). All 3 pieces of music were rated on the same emotion scales as for the film clips. The sad and happy mood induction pieces were comparable in their degree of emotionality, though they differed in valence (Happy clip: sadness rating - M = 1.63, SD = 1.19; happiness rating - M = 6.88, SD = 2.17. Sad clip: sadness rating - M = 6.50, SD = 1.16; happiness rating - M = 1.13, SD = 0.35), and did not generate any other emotions to a marked extent. The neutral clip did not generate any emotions to a notable degree. There were no significant differences between emotionality ratings (adjusted for valence) between the film and music clips (Ps > .1).

The music clips were presented via headphones attached to a compact disc player. Music clips were 2-3 minutes long. Participants were encouraged to absorb themselves in the emotions that the music generated as much as they could.

2.2.4. Emotion state rating scale

Current emotion state was assessed before and after the mood inductions using 16 visual analog scales with the range 0 (no emotion) to 100 (very intense emotion). The key variables of interest for the present study were "happiness" and "sadness" as these were the only emotions reliably elicited by the film and music clips during piloting. However, we also included a number of filler variables assessing other emotions (e.g. fear, disgust, anger, guilt, shame).

2.2.5. Proxy measure of lifetime depression

Because we had residual concerns that some of our sample may have experienced significant symptoms of depression in the past, even though they reported no history of clinical depression, we asked all participants questions A1 (Has there ever been a period of

time when you were feeling depressed or down most of the day, nearly every day? What was that like? How long did it last? As long as 2 weeks?) and A2 (Have you ever lost interest or pleasure in things you usually enjoyed? Was it nearly every day? How long did it last? As long as 2 weeks?) from the Major Depressive Episode (MDE) section of the Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbons & Williams, 1997), with respect to the lifetime period. The presence of one or both of these symptoms is necessary, though not sufficient, for a lifetime diagnosis of MDE, and this therefore provides a proxy measure of depression history. This proxy measure was scored present (endorsement of one or both questions) or absent. The intention was to allow the possibility of using these scores as a covariate in all analyses (along with scores of the BDI), if scores differed as a function of mood induction condition.

2.2.6. Autobiographical Memory Test (AMT)

Two parallel versions of the AMT were taken from Watkins, Teasdale and Williams, (2000). Each consisted of 18 cue words (a mix of positive, negative and neutral) presented on cards in a separate random order for each participant. Before the first AMT, participants practiced on three neutral words (e.g. library) with feedback. On the main task, participants were given 60 seconds to retrieve a specific personal memory to each word; that is a memory of a *discrete event located on a single day*. Participants' responses were tape-recorded. Responses that were not categorized as specific memories were coded either as general (comprising 'extended' memories of events that lasted for more than one day and 'categoric' memories representing classes of event that occurred repeatedly), as omissions (failures to recall a memory within the time limit) or as semantic associates (responses that were not memories but that were related to the cue word, e.g. an opinion cued by the word or a simple word associate of the cue word) derived from Williams and Dritschel (1992). If the type of

response initially generated was unclear, participants were prompted once to say more. All coding was blind to condition. Reliability of coding on the AMT is good with previous studies reporting Kappas of 0.70-0.90. AMS on each AMT was indexed by the proportion of specific memories recalled across the 18 words as a fucntion of the total number of actual responses (i.e., 18 minus the number of omissions; Williams, personal communication). All analyses involving the effects of the emotional mood inductions were conducted relative to AMS change following the neutral mood manipulation. The two versions of the AMT were presented in a fixed order as: (a) there was no evidence from previous studies that they generated differential rates of specific memories (Watkins et al., 2000); (b) the study involved a neutral mood induction control condition, relative to which the effects of the emotional mood inductions was always assessed.

2.3. Procedure

Participants were tested individually in a soundproof testing room by the same experimenter (Cecilia Au Yeung). Participants completed the BDI-II (Beck, Steer & Brown, 1996) first to provide a baseline measure of depression levels over the past week and were administered the proxy measure of depression history (questions A1 and A2 from the SCID). The first mood rating scale and the first AMT were then completed, followed by the mood induction. Participants then completed the second mood rating scale and second AMT. At the end of the study, the happy film clip was shown to the participants who had received the sad mood induction to help return their mood to baseline.

3. Results

3.1. Demographic variables

Table 1 shows the demographic data for the participants in the three conditions. The three groups did not significantly differ on any measure (all Ps > .10). As can be seen, a proportion of participants in each group endorsed either or both of the SCID questions and so this proxy measure of lifetime depression symptoms was used as a covariate for all analyses involving AMS.

Table 1 about here

3.2. Mood inductions

The analyses presented below were initially conducted with Media Type (film versus music) as a factor. However, Media Type was not involved as a main effect or as an interaction term in any analyses pertaining to either the mood-inducing effects of the different manipulations or their relationship with the AMS variables (Ps > .1). Analyses are therefore reported without Media Type as a factor.

Table 2 shows the mean emotion rating data pre- and post-mood induction, for the three groups. There were no significant differences across groups in levels of baseline mood, $Fs_{(2, 42)} < 2.38$, Ps > .10. Two Condition (sad, happy, neutral) by Time (pre-mood induction, post-mood induction) mixed model ANOVAs were carried out for the happy and sad ratings, as a manipulation check for the mood induction. In both analyses there were the expected significant interactions of Time by Condition, lowest $F_{(2,42)} = 6.91$, highest P = .003. Planned paired t-tests revealed that for the sad mood induction, reported sadness increased and happiness decreased, with the reverse following the happy mood induction, ts > 2.00, Ps < .05. For the neutral manipulation, there was no significant change on either state measure, ts < 1, Ps > .34. The apparent similarity in happiness and sadness ratings post-induction across the positive and neutral conditions is misleading as the means were moderately different across conditions pre-induction.

Table 2 about here

3.3. AMS

Table 2 also shows the AMS data pre- and post-mood induction for the 3 groups. To test the first hypothesis that AMS would decrease following the negative mood manipulation, relative to the neutral, with no change following the positive manipulation, we carried out a Condition (sad, happy, neutral) by Time (pre-mood induction, post-mood induction) mixed model ANOVA with proportions of specific memories (AMS) as the dependent variable, and with baseline BDI scores and our proxy measure of lifetime depression (on the SCID) as covariates. There was no significant main effect of Time, $F < 1$. Interpretation of this null result is difficult because in this study AMT order and practice effects are confounded. There was also a main effect of Condition, $F(1,42) = 4.47$, $P < .02$. This was qualified by a significant Time by Condition interaction, $F(2,42) = 8.39$, $P < .001$. This interaction remained significant even after covarying out baseline BDI scores and our proxy measure of lifetime depression (on the SCID), $F(2, 40) = 8.76$, $P < .001$.

To deconstruct the Time by Condition interaction we performed 2 follow up ANOVAs examining the positive versus neutral and negative versus neutral comparisons. For the positive versus neutral analysis, the critical Time by Condition interaction was not significant, $F(1,29) = 1.22$, ns. However, for the negative versus neutral analysis, there was again a significant Time by Condition interaction, $F(1,28) = 7.35$, $P < .02$. We broke this down further using paired-sample t-tests for the negative and neutral manipulations separately. The negative manipulation led to a significant decrease in the number of specific memories, $t(13) = 2.27$, $P < .05$, with no such effect for the neutral manipulation, $t(15) = 1.46$, ns. This difference across the negative and neutral conditions had a large effect size, Cohen's D = 1.02.

To examine the second hypothesis that change in emotion state following the mood manipulations would be significantly associated with change in AMS we performed correlations between computed variables of mood state change and memory change (post-induction scores minus pre-induction scores), based on the data in Table 2. Change in happiness ratings showed a significant correlation with change in AMS, $r (43) = 0.44$, $P < .01$, with a larger decrease in happiness being associated with a larger decrease in AMS. However, there was no significant association between change in sadness ratings and the AMS measure, $r (43) = -.09$, ns. These results were similar after partialling out BDI scores and the proxy depression measure, $r (41) = .45$, $P < .01$, and $r (41) = -.09$, ns, respectively.

To examine the final hypothesis that levels of AMS after the mood induction would be associated with state emotion levels but may or may not be associated with baseline BDI scores themselves, we performed correlational analyses. There was a significant correlation between post-induction happiness ratings and numbers of specific memories, $r (43) = 0.34$, $P < .03$, with lower happiness associated with lower AMS. There was no such significant correlation between post-induction sadness ratings and the AMS measure, $r (43) = -.10$, ns. These results were similar after partialling out BDI scores and the proxy depression measure, $r (41) = .35$, $P < .03$, and $r (41) = -.13$, ns, respectively. Interestingly, there were no significant zero-order correlation between baseline BDI and either pre- or post- AMS, $rs < \pm .21$, $Ps > 0.18$.

As a final check regarding the influence of depression history, the above sets of analyses were repeated with the 4 participants in the negative mood induction who endorsed questions A1 and/or A2 on the SCID-Lifetime removed. It was not necessary to remove such participants from the other induction conditions, as any elevation in overgenerality in these other groups would have gone against the grain of the present hypotheses. The pattern of results was unaltered. Briefly, the negative induction group showed a reduction in the number

of specific memories recalled pre-post induction relative to the neutral group, t (24) = 2.22, P < .04, Cohen's D = 0.91. Reduction in happiness across the induction procedure correlated with changes in specific memories, r (41) = .53, P < .001. Finally happiness levels post induction correlated with levels of specific memories r (41) = .37, P < .02.

4. Discussion

The present study followed up previous research (Maccallum et al., 2000; McBride & Cappeliezi, Expt.1, 2004; Svaldi & Mackinger, 2003) in investigating whether an induced negative mood/emotion state in healthy participants can lead to a decrease in AMS. It is the first study, as far as we are aware, to examine such effects of a negative mood induction while also controlling for a past history of depression and while covarying out current depression levels. It is the also the first study, to our knowledge, to look directly at the relationship between current mood/emotion state and AMS. The nature of this latter relationship represents an important unresolved issue if a credible claim is to be made that AMS is a stable marker and causal cognitive factor in depression that is relatively independent of current emotion state.

The study examined 3 hypotheses, as outlined in the Introduction. In support of our first hypothesis, the results showed that induced negative mood, compared with neutral mood, led to a relative reduction in AMS (even when controlling for baseline BDI scores and the proxy measure of lifetime depression) with a large effect size, Cohen's D = 1.02. A negative mood manipulation also led to an absolute significant decrease in AMS. In contrast, a positive mood induction had no significant relative effects on AMS. This pattern of findings is consistent with the earlier results of Maccallum et al. (2000) and Svaldi and Mackinger (2003) (though not with those of McBride & Cappeliez, 2004).

In support of our second hypothesis, a greater reduction in happiness ratings (but interestingly not a greater increase in sadness ratings) correlated with a greater reduction in AMS following the mood manipulation, even after controlling for baseline BDI scores and the proxy lifetime depression measure. This suggests that it is the *emotion-changing* effect of the mood induction that is driving the change in AMS, rather than, for example, some form of semantic priming, although priming effects can never be completely ruled out.

With respect to our third hypothesis, following the mood manipulation, lower levels of reported happiness (but again not higher levels of sadness) correlated significantly with reduced AMS, even after controlling for baseline BDI scores and the proxy lifetime depression measure. However, interestingly there was no significant correlation between baseline BDI itself and AMS (consistent with a number of previous studies e.g. Wessel et al., 2001; though see Ramponi, Barnard & Nimmo-Smith, 2004).

The present pattern of data provide the first clear support for the fact that reduced AMS can be a function of *current emotional state*, independent both of levels of depressed mood over the previous week and of depression history.

There remain two explanations (of the four discussed in the Introduction) of these data (allied with those of Maccallum et al., 2000, and Svaldi & Mackinger, 2003) for the literature on AMS and clinical depression. The first is that AMS is a multi-faceted construct with one or more facets relating to a lifetime history of clinical depression and coding stable features of the disorder, and one or more additional facets relating to current emotional state. The second more radical possibility is that AMS is *entirely* a function of current emotional state and that this putative association has gone undetected in earlier studies (e.g. Mackinger et al., 2000), because mood state was not explicitly measured (only depression symptomatology over the previous week).

Further research is clearly warranted to address these issues. The most obvious follow-up study is to repeat the Mackinger et al., (2000) experiment, this time ensuring that groups are matched on *current mood state* throughout the experiment, as well as current levels of depression symptoms.

Resolving such issues regarding AMS is important because, to date, although it seems clear that AMS is a marker for one or more aspects of psychological processing that critically relate to the course of clinical depression (e.g. Brittlebank et al., 1993) (and probably other forms of psychopathology, Harvey et al., 1998), it is not clear what these aspects are. Consequently, the possibility that one such aspect (possibly among several) is current mood state needs to be taken seriously.

An intriguing aspect of the current findings is that reduced AMS was only associated with self-reported decreased feelings of happiness, and not increased feelings of sadness, in the present data. It is unclear why this was the case. However, it may go some way to explaining the lack of any consistent relationship in previous studies between AMS and measures such as the BDI which do not tap happy mood. A bi-dimensional measure such as the Depression-Happiness Scale (Joseph & Lewis, 1998) may therefore be a more sensitive instrument for future research.

In sum, the present study shows that AMS can be manipulated by a state mood induction in healthy volunteers with no reported history of depression and that levels of AMS are significantly associated with current emotional state, though not with current levels of depression symptomatology. These findings, alongside the data of Maccallum et al., (2000) and Svaldi and Mackinger (2003), suggest important questions concerning the nature of the relationship between AMS and a history of clinical depression and the status of AMS as a stable marker for depression. They also reinforce the potentially important contribution of the

mood induction methodology as a tool in elucidating the mechanisms of abnormal

psychology.

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Table 1

Mean and categorical baseline demographic data (SDs where appropriate in parentheses) across the negative-, positive-, and neutral-mood manipulation conditions

	Neutral mood	Positive mood	Negative mood	Test statistic	P value
N	16	15	14		
Age	24.19 (8.85)	25.73 (7.75)	29.86 (6.84)	$F = 2.11$	0.13
Sex (M:F)	4:12	3:12	5:9	Fisher's exact	0.62
Education[a]	0:3:10:1[b]	1:5:6:3	1:1:10:1[c]	Fisher's exact	0.40
BDI	6.25 (10.50)	9.53 (5.66)	7.36 (5.71)	$F < 1$	0.50
Lifetime SCID A1/A2 (N)	7	6	4	Fisher's exact	0.35

Note

M = male, F = female, BDI = Beck Depression Inventory (second edition), SCID A1/A2 = Structured Clinical Interview for the DSM-IV, question A1 and/or A2.

a - Education = left school at 16: left school at 18: college degree: college postgraduate.

b - data from 2 participants are missing.

c - data from one participant are missing.

Table 2

Mean happiness and sadness ratings and autobiographical memory data (SDs in parentheses) pre and post the negative-, positive-, and neutral-mood manipulations

	Neutral mood (N=16)		Positive mood (N=15)		Negative mood (N=14)	
	Pre	Post	Pre	Post	Pre	Post
Happiness	64.88 (12.12)	62.25 (17.52)	55.43 (24.38)	67.00 (18.35)	53.21 (23.38)	37.21 (19.10)
Sadness	12.03 (14.35)	10.53 (14.88)	26.20 (22.28)	9.27 (10.50)	26.58 (25.88)	46.21 (30.03)
Specific memories	13.88 (3.01)	14.75 (2.49)	14.00 (2.62)	15.73 (1.75)	12.79 (3.26)	11.14 (4.26)
Proportion of specific memories	0.77 (0.16)	0.82 (0.14)	0.78 (0.14)	0.88 (0.10)	0.72 (0.17)	0.63 (0.23)
General memories	2.69 (2.06)	1.81 (1.83)	2.80 (2.31)	1.47 (1.36)	3.14 (2.60)	3.36 (1.82)
Semantic associates	1.25 (2.05)	1.25 (1.91)	1.20 (0.86)	0.73 (1.03)	1.86 (2.11)	3.07 (2.70)
Omissions	0.13 (0.46)	0.13 (0.34)	0.00 (0.00)	0.07 (0.26)	0.21 (0.58)	0.43 (0.65)