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Cognitive Reactivity to a Depressive Mood Induction Procedure Across Diagnostic Categories

Martin C. Scherrer and Keith S. Dobson*

University of Calgary, Calgary, Alberta, Canada

Abstract

The concepts of cognitive vulnerability and cognitive reactivity are central to cognitive models of depression. These concepts have been extensively examined using mood priming methodology. Research has largely examined cognitive reactivity in individuals theoretically vulnerable to depression and based on self-reported dysfunctional attitudes as a primary index of cognitive vulnerability. The current research was designed to expand the examination of cognitive reactivity through mood priming methodology with regard to assessment of cognitive reactivity and clinical populations examined. This study examined the specificity of cognitive reactivity in the form of self-reported automatic thoughts, dysfunctional attitudes, and rumination to individuals with a history of depression compared to a clinical control group of currently anxious participants and a control sample. The primary results indicated specificity in cognitive reactivity, in the form of increased dysfunctional attitudes and rumination for only the previously depressed participants. These results extend previous research by suggesting specificity of cognitive reactivity in depression compared not only to a nonclinical control sample, which is typically employed in mood priming research, but also compared to a clinical control population. These results are discussed in the context of cognitive models of depression, as are their implications for future theory and research.

Keywords: Cognitive reactivity; Vulnerability; Mood induction; Remitted depression

Introduction

Since its earliest formulation, the cognitive model proposed that reactivity to stressful situations, through the activation of underlying cognitive vulnerability, is a causal factor for depression [1]. Depressogenic beliefs and attitudes (also referred to as schemas) constitute the cognitive vulnerability to depression [2,3]. Cognitive reactivity refers to the proposition that this vulnerability remains latent until activated, but once activated it results in the information processing biases and cognitive products that typify depression [3]. A related tenet of the cognitive model of depression argues that there is a distinct cognitive profile for the content and orientation of schema, information processing biases, and cognitive products for different disorders [2]. In depression, schemas are theoretically characterized by negative attitudes and beliefs, and the themes of loss and failure. Information processing is negatively biased in depression, at the expense of positive information processing, and cognitive products (thoughts) in depression center on these same themes.

The concept of cognitive vulnerability is reflected in the "cognitive stability hypothesis" which states that depressive cognition is not simply a concomitant of depression, but reflects activation of a stable, underlying cognitive vulnerability to depression [2]. Early research, largely based on simple self-report studies, did not appear to support this proposition. For example, Gotlib and Cane [4] concluded that "considered collectively, the results of these studies suggest that these cognitive processes are more parsimoniously considered as state-dependent concomitants of depression rather than as trait-like variables that are implicated in the etiology of the disorder" (p. 203). More recent studies, however, have demonstrated cognitive vulnerability in individuals at increased risk for depression, such as recovered depressed patients, through the use of laboratory mood induction procedures [2,3]. MIPs have played a significant role in the examination of the concepts of cognitive vulnerability and cognitive reactivity to depressed mood states [3,5].

While studies of remitted patients provide some evidence to support the stability of depressive cognitions, adequate study requires activation of the hypothesized cognitive vulnerability [3,6]. A considerable body of research has incorporated the procedure of priming to activate cognitive vulnerability in depression and as such, provide a stronger test of the diathesis-stress model of depression [2,3,6]. A "typical" priming study compares remitted depressed to control participants on a range of indices of cognitive content and information processing prior to and following a negative mood priming procedure. Given equivalent negative mood shift across groups following the mood induction procedure [3], any observed difference in cognitive content or information processing between remitted depressed and control participants is viewed as evidence for cognitive vulnerability.

Persons et al., examined the "mood state hypothesis" [7], which emphasizes that cognitive vulnerability remains latent until activated by some stressor, but also suggests that this stressor may take the form of negative mood state. Research has provided empirical support for cognitive reactivity in the form of increased reporting of dysfunctional attitudes, with a range of priming procedures, including naturally occurring as well as laboratory induced depressive mood states [8-10]. These results were observed in individuals with a history of depression, but not among individuals without this history [11,12].

Priming designs also provide evidence for cognitive vulnerability and reactivity in depression through examination of the association between mood state and information processing [3,6]. For example,

***Corresponding author:** Paulo Correia Ferrajão, European University, Laureate International Universities, Estrada da Correia, nº53, 1500-210 Lisbon, Portugal, Tel: 351-218-811-700; Fax: +351 218 860 954; E-mail: paulo.ferrajao@europeia.pt

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negatively biased recall following negative mood induction in formerly depressed has been demonstrated, relative to never depressed individuals [3]. Attention biases have also been demonstrated among formerly depressed individuals, included increased tracking errors for negative stimuli using a dichotic listening paradigm [13] and increased attention for negative stimuli (Ingram and Ritter) following negative mood induction. Scher et al. [3] concluded that "the bulk of the data clearly and consistently point to activation processes that characterize vulnerability, and do so in ways that are generally consistent with cognitive diathesis-stress models of depression" [6].

Cognitive reactivity to mood induction has predicted depressive relapse in vulnerable populations [14,15]. In the first study to examine cognitive reactivity to negative mood induction as a predictor of depressive relapse, Segal et al., [14] assessed dysfunctional attitudes prior to and following an autobiographical recall with depressive music mood induction procedure in a sample of formerly depressed participants who had been treated to remission through either cognitive-behavioral therapy (CBT) or pharmacotherapy. Results indicated that the two participant groups did not differ on self-reported dysfunctional attitudes prior to mood induction. However, those participants treated with pharmacotherapy exhibited greater cognitive reactivity to negative mood induction than those treated with CBT. Further, degree of cognitive reactivity predicted relapse 13-48 months later. These findings were replicated in a similar but more methodologically rigorous study conducted by Segal et al. [15]. These studies suggest possible effects of CBT on cognitive reactivity to depressive mood states, though it remains uncertain if underlying cognitive vulnerability itself is differentially impacted by CBT [16].

In summary, priming designs have played a central role in the evaluation of the fundamental tenets of the cognitive model of depression of cognitive vulnerability and reactivity [2,3.6]. Scher et al. [6] asserted that "the general principle of schema activation can be considered now to be well-established" (p. 34). A number of significant issues have yet to be addressed in the cognitive reactivity literature, however. For example, the majority of the available research has employed the Dysfunctional Attitudes Scale (DAS) as the primary or sole measure of cognitive vulnerability [9,10,14,15,17]. In addition, research has compared remitted depressed to never depressed samples, which has not allowed for examination of the specificity of cognitive reactivity to depression, in relation to other clinical disorders, such as the commonly comorbid anxiety disorders. While the predominant cognitive themes in depression are related to personal loss or deprivation, anxiety related cognitions center on themes of physical or psychological threat or danger. There is considerable empirical support for the cognitive content specificity hypothesis in depression [2], with regard to both content and information processing. Further, while depression and anxiety share significant overlap in symptoms and cognitive content, depression and anxiety appear to have unique cognitions, when general negative affect is controlled [2].

The present study addressed some of the limitations seen in past research. First, a wider range of indicators of cognitive reactivity, including dysfunctional attitudes as an index of schema content, as well as positive and negative and depression- and anxiety-specific automatic thoughts, and the cognitive process of rumination, were examined. Further, participants included individuals with a past Major Depressive Disorder (MDD), a clinical control group comprised of participants with a current anxiety disorder, and a community control sample which included individuals with no history of depression or current anxiety disorder diagnosis.

The three participant groups were selected for several reasons. First, and consistent with mood priming research, a formerly rather than currently depressed sample allowed examination of cognitive reactivity in depression. Second, including a clinical control group of currently anxious participants allowed comparison of cognitive reactivity in those theoretically vulnerable to depression (remitted depressed) to an additional clinical population, including examination of specificity of certain aspects of cognitive reactivity to depression, which extends previous research. Finally, these groups enabled further examination and comparison of cognitive reactivity across clinical populations, as well as between clinical and nonclinical populations.

It was hypothesized that an increase in negative automatic thoughts and a decrease in positive automatic thoughts would be observed across participant groups following a mood induction procedure. It was further hypothesized that formerly depressed participants would exhibit greater increase in reported depression-related attitudes, cognitions, and rumination than both currently anxious and control participants. Finally, it was hypothesized that currently anxious participants would exhibit greater increase in reported anxiety-related cognition than formerly depressed and control participants.

Method

Participants

The sample consisted of three groups: 1) a formerly depressed group; 2) a currently anxious group; and 3) a control group. Participants were recruited from the community and a pool of volunteers who had previously participated in research at the Depression Research Laboratory at the University of Calgary. Inclusion criteria included an age between 18 and 65 years of age, and the criteria for one of the three participant groups. Exclusion criteria for all groups included a current or past diagnosis of Bipolar Disorder or Psychotic Disorder, current Substance Abuse Disorder, or current diagnosis of anxiety disorder(s) other than Generalized Anxiety Disorder (GAD), Social Anxiety Disorder (SAD), Panic Disorder (PD), or Specific Phobia (e.g. Obsessive-Compulsive Disorder, Post Traumatic Stress Disorder).

Participants completed a telephone screen to determine eligibility for participation in the study, which included assessment of inclusion and exclusion criteria and potential diagnostic status. Each of the participant groups was defined in terms of current diagnostic criteria as outlined in the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* (*DSM-IV)* [18]. All diagnostic interviews were completed, and diagnoses determined, by the primary investigator (M.C.S.), and inter-rater reliability was examined with a blind expert rater (K.S.D.), who rated a randomly selected 21 (7 from each group) interviews. Analysis of the concordance between raters, using group status as the criterion variable, was conducted. There was an initial 92.5% absolute agreement for diagnoses, and the single disagreement was related to perceived degree of symptom-related impairment, rather than the presence or absence of assessed symptomatology *per se*.

From an initial pool of 155 contacts, 99 individuals were invited to participate in the study, and 81 participants finally completed the study. All 33 participants in the formerly depressed group met criteria for past Major Depressive Disorder (MDD) for a minimum of 2 weeks, but not for a current Major Depressive Episode (MDE). Five participants in this sample met the Frank et al. [19] criteria for remission, having not met criteria for an MDE for 2-8 weeks, and 28 met criteria for recovery, having not has their past episode for greater than 8 weeks. The currently anxious group was comprised of 22 individuals

who met criteria for Generalized Anxiety Disorder (GAD), Social Anxiety Disorder (SAD) and/or Panic Disorder (PD), with or without Agoraphobia. Four participants were diagnosed with PD (2 with and 2 without Agoraphobia), 4 with SAD, 9 with GAD, and 5 with multiple anxiety disorders (3 for comorbid GAD and SAD and 2 for SAD and PD without Agoraphobia). Participants in the anxious group were also required to have no current or past depressive disorder, including MDD or Dysthymic Disorder. GAD, SAD, and PD were included as they are among the anxiety disorders that are most commonly comorbid with depression [20]. Finally, the control sample included 26 individuals who met the global study inclusion and exclusion criteria, but did not meet criteria for a current or past depressive disorder or a current diagnosis of GAD, SAD, or PD.

Measures

Diagnostic assessment: Participants' diagnostic information was gathered using the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I) [21]. The SCID-I is a semi-structured interview that determines current and lifetime Axis I disorders following the diagnostic criteria in the *DSM-IV* [18]. Reliability estimates for the SCID meet or exceed those for earlier versions of the interview [22] including, for example, acceptable reported interrater and test-retest reliabilities [23]. While it is difficult to establish validity, given the current lack of a diagnostic "gold standard" [24] among available semistructured diagnostic interviews, the SCID-I is often identified as the preferred choice [24,25], and is commonly used in research for diagnostic purposes [23].

Extent of mood induction. Extent of mood induction was assessed through both self-report and psychomotor indices of mood state. The Visual Analogue Mood Scale (VAMS) consists of a single 10-cm line with an anchor of "0" and "Very Sad" on the left end and "100" and "Very Happy" on the right, and participants are instructed to place a slash through the line to indicate the valence of their current mood. The score is obtained by measuring the distance between the left end of the line and the dash in millimetres. The VAMS demonstrates strong psychometric properties, particularly for depression [26], and various forms of the VAMS have been used in previous mood induction research [6,14,27,28].

Two psychomotor indices were also employed to assess the degree of negative mood induction. The Velten depressive MIP has been found to be related to psychomotor slowing as measured by both count time and writing speed [29], and both psychomotor tasks have been used extensively in past research as a measure of the effects of the Velten MIP [30,31]. In the current study, count time (the time to count aloud the numbers 1 through 10) and writing speed (the number of number written in descending order from 100 over 60 seconds) were used.

Cognition: Cognition was assessed with regard to both cognitive content (i.e. negative beliefs, automatic thoughts,) and cognitive process (i.e., rumination). The Dysfunctional Attitudes Scale (DAS-A) (Weissman) [32] was employed to assess dysfunctional beliefs associated with depression. The DAS is a widely used measure of maladaptive thinking and dysfunctional cognitions, and was designed to measure the presence of Beck's postulated "negative cognitive triad". The DAS-A has been widely used in previous research [11,28]. Items are rated on a 7-point scale, with higher scores reflective of greater level of dysfunctional beliefs. The DAS-A demonstrates strong psychometric with clinical and nonclinical populations [32-34].

The Automatic Thoughts Questionnaire-Revised (ATQ-R) [35] was employed to assess the frequency of depressive automatic thoughts. The ATQ-R is a 40-item self-report measure on which participants rated self-statements on a 5-point scale of frequency of occurrence over the past week. The ATQ-R includes the 30 negative self-statements from the original Automatic Thoughts Questionnaire (ATQ) [36] most associated with depression, as well as 10 positive self-statements thought to be inversely associated with depression. Greater scores on the negative and positive subscales reflect increased negative and positive self-referent cognition, respectively. The ATQ-R exhibits strong psychometric properties in both analogue and clinical samples [35]. The Cognition Checklist (CCL) [37] was utilized as a measure of frequency of automatic thoughts specific to either depressive or anxiety disorders. The CCL is a 26-item self-report measure that includes a 14-item depression subscale (CCL-D) and a 12-item anxiety subscale (CCL-A). Items are rated on a 5-point scale, with higher scores on the depression and anxiety subscales indicating increased frequency of depression- and anxiety-related automatic thoughts, respectively. The CCL has been used widely in research examining the relationship between anxiety and depression [38], and has demonstrated strong psychometric properties in both analogue and clinical samples [37,39].

The Ruminative Responses Scale (RRS) [40] was employed to assess ruminative cognitive style. The 22 items of the RRS assess the degree to which participants' response to depressed mood involves a focus on the self, symptoms, and the possible consequences and causes of their mood [40,41],. Items are rated on a 4-point scale, with higher total RRS scores indicating increased rumination in response to depressed mood. The RRS is widely used in examinations of rumination in depression [41,42], and has demonstrated strong psychometric properties in community and clinical samples [40,41].

Mood induction

As noted above, participants were administered a negative mood induction procedure (MIP). Thirty statements from each of Velten's negative or "depressive," and positive or "elation," mood induction statement sets [43] were used. A spiral booklet comprised of Velten's MIP instructions, and the 30 negative and 30 positive mood induction statements, printed individually on 14 cm by 21.5 cm pages, was employed. Participants first read the instructions to read each statement and try to feel the mood that each suggests. Participants read each statement for 20 s, before being prompted to flip the page to the next statement by an audio recording. Each mood induction required 10 minutes to complete. The Velten MIP generally followed that originally outlined by Velten [43] and has been employed in more recent mood induction research [44,45].

Although the effects of the Velten negative MIP are understood to be relatively brief (i.e., 6-12 minutes among a student sample) in duration [46], an elation MIP was administered to all participants after they had completed the experimental task as an ethical consideration. The elation Velten MIP has been shown to effectively ameliorate the depressive effects of the negative Velten MIP [47]. Except for the content of the statements themselves, the elation MIP was conducted identically to the depressive induction.

Procedure

Participants attended one experimental session. Informed consent was obtained from all participants, and involved a brief review of the study's objectives and explanation of the procedure. Participants were informed that they might or might not experience a negative mood shift as a result of taking part in the study. Demographic and background information related to past and/or current treatment

for psychological difficulties was obtained through a semi-structured interview. Participants were then administered the SCID-I to determine their diagnostic status and inclusion into the appropriate participant group. Participants who failed to meet inclusion or exclusion criteria as a result of the SCID-I (n = 18) were informed of their ineligibility to continue and were fully debriefed.

Eligible participants next completed a series of self-report questionnaires, which included the ATQ-R, DAS-A, CCL, RRS. Participants then completed the first assessment of current mood state, which included the VAMS and two psychomotor tasks (i.e., count time and writing speed). Next, participants completed the negative Velten MIP, which was followed by a second assessment of current mood state, including the VAMS and psychomotor tasks. Participants then completed the DAS-A and ATQ-R for a second time. Because the effects of the Velten negative MIP are relatively brief in duration [46] and to account for the time required to complete the DAS-A and ATQ-R, participants underwent a brief "booster" MIP consisting of 10 negative statements, after which they completed the CCL and RSQ for a second time. Participants then underwent the positive Velten MIP, were fully debriefed and provided with general information regarding resources for depression and anxiety.

Results

Sociodemographic information

The sample consisted of 21 males and 60 females, with an average age of about 35. The sample was predominantly Caucasian (79%) and Southeast Asian/Oriental (12%), with other groups represented as: First Nations (4%), Hispanic (2%), Black (1%), and Other (1%). With regard to marital status, 51% of the sample was never married, 36% were married/common law, and 16% were widowed/ divorced/ separated. The majority of the sample had completed at least some college or technical school (58%), while 41% had completed at least some graduate or professional school, and 1% had a high school education or less. One-way ANOVA or Chi-square analyses, as appropriate, showed no significant differences across groups on the variables of gender, ethnicity, marital status, and educational history. A one-way ANOVA indicated a statistically significant difference in age across the three participant groups (formerly depressed group, M = 38.73, SD = 12.01; currently anxious group, M = 32.05, SD = 10.88; control group, M = 32.12, SD = 10.52), F (2, 78) = 3.40, p<0.05. However, no single pair-wise comparison between any two groups was statistically significant. In addition, a regression analysis indicated that age did not account for a statistically significant amount of variance in change in VAMS score from pre to post negative mood induction, F (1, 79) = .04, *ns*, and the Pearson correlation between age and VAMS shift of r = 0.02 was not statistically significant. As a result, age was not considered to confound subsequent analyses.

Effect of the velten negative mood induction procedure on mood

The effect of the Velten negative mood induction procedure on mood was assessed with three measures: a self-report rating of current mood using the VAMS, and two behavioral indices, including writing speed and count time. A series of repeated measures ANOVAs with Group as the between subjects effect and Time as the within subjects effect was performed for mean scores on each of the three mood measures pre and post negative MIP (Table 1). A significant change on VAMS score from pre to post negative MIP, indicating more negative mood, was observed across groups, F (1, 78) = 101.26, p< 0.001. A significant increase in both writing speed, F (1, 78) = 16.69, p<0.001, and count time, F (1, 78) = 13.32, p<0.001, also indicated slowed psychomotor performance following negative mood induction across groups. No significant time by group interaction was observed for either VAMS or writing speed, which indicated that the degree of change was comparable across the three participant groups. A significant time by group interaction was observed for count time, F (2, 78) = 3.11, p=0.05. A series of three follow-up paired samples t-tests (using Bonferonni correction, alpha set at .017 for each) was performed to further examine this interaction. Results indicated significant slowing for formerly depressed, t (32) = -3.8, p<0.005, but not for currently anxious or control participants.

In summary, a significant negative mood shift was observed across participant groups on two of the three mood indices employed in the current study, including self-reported VAMS score and one of the behavioral indices, writing speed. A significant reduction in count time was observed for formerly depressed but not currently anxious or control participants. Results indicate that the effect of the negative mood induction procedure was generally comparable across the three participant groups.

Cognitive Reactivity to Negative Mood Induction

Participants completed multiple measures of cognitive content and process prior to and following the negative Velten MIP measures (Table 2). A series of repeated measures ANOVAs with Group as the between subjects effect and Time as the within subjects effect was performed for mean scores on each of the cognition measures.

A statistically significant main effect for time was observed for four of the five cognition measures, including the ATQ-N, DAS-A, CCL-A, CCL-D, and RRS. Scores on the ATQ-N indicated a significant increase in negative automatic thoughts from pre to post negative mood induction across the three participant groups, F (1, 78) = 31.12, p<0.001. DAS-A scores revealed a significant increase in dysfunctional attitudes, from pre to post negative mood induction across groups, F (1, 78) = 22.77, p<0.001. Both anxiety- and depression-related cognitions increased from pre to post negative mood induction across groups, as evidenced by a significant main effect for Time for scores on the CCL-A, F (1, 78) = 7.24, p<0.05, and the CCL-D, F (1, 78) = 13.62, p<0.001, respectively. Finally, a significant main effect for Time was observed for RRS scores, as a result of an increase in ruminative cognitive style from pre to post negative mood induction across groups, F (1, 78) = 14.52, p<0.001. The main effect for Time for ATQ-P indicated a nonsignificant trend toward reduced positive thoughts across participant groups following the negative MIP, F (1, 78) = 3.54, p=0.06. In summary, significant increases in negative automatic thoughts, dysfunctional attitudes, anxiety- and depression-related cognition, and ruminative cognitive style were reported across participant groups.

Interaction effects were also examined to identify any significant group differences in degree of cognitive change following negative mood induction. No significant time by group interaction was observed for either positive or negative automatic thoughts, or for either anxiety- and depression-related cognitions. A significant time by group interaction was observed, however, for change in dysfunctional attitudes, as assessed by the DAS-A, F (2, 78) = 3.82, p<0.05. A series of three follow-up paired samples t-tests (using Bonferonni correction, alpha set at 0.017 for each) was performed to further examine this interaction. Results indicated a significant increase in reported dysfunctional attitudes among formerly depressed following negative mood induction, t (32) = -4.55, p<0.001, but not for currently anxious

Measure	Group	Pre Negative MIP		Post Negative MIP	
		Mean (SD)	Range	Mean (SD)	Range
VAMS	Formerly Depressed	66.09 (14.51)	33-92	44.97 (17.65)	7-75
	Currently Anxious	57.95 (10.24)	43-86	45.77 (13.32)	21-79
	Control	71.08 (12.51)	42-91	54.31 (13.84)	29-80
Count Time	Formerly Depressed	5.57 (2.24)	1.32-11.22	6.45 (2.90)	1.60-14.25
	Currently Anxious	5.70 (1.79)	2.35-9.75	5.72 (1.69)	2.68-9.19
	Control	5.91 (2.54)	1.62-12.25	6.59 (3.23)	1.68-14.50
Writing Speed	Formerly Depressed	45.64 (9.37)	31-67	48.09 (9.73)	33-70
	Currently Anxious	44.59 (10.25)	23-63	46.09 (11.44)	26-73
	Control	48.12 (7.80)	30-65	51.46 (8.50)	36-67

Table 1: Mean Scores by Group from Pre to Post Negative MIP on Mood Measures.

Measure	Group					
	Formerly Depressed *n* = 33		Anxious *n* = 22		Control *n* = 26	
	Pre-MIP Mean (*SD*)	Post-MIP Mean (*SD*)	Pre-MIP Mean (*SD*)	Post-MIP Mean (*SD*)	Pre-MIP Mean (*SD*)	Post-MIP Mean (*SD*)
ATQ-P	27.76 (10.13)	25.45 (9.05)	27.18 (9.37)	26 (9.02)	29.85 (9.52)	30.19 (8.82)
ATQ-N	50.03 (18.00)	61.58 (24.18)	60.50 (20.11)	68.41 (20.60)	42.04 (13.03)	46.00 (16.01)
DAS	119.64 (31.16)	135.18 (34.54)	135.77 (27.22)	142.00 (28.82)	106.88 (28.73)	111.54 (31.91)
CCL-D	11.06 (8.46)	15.42 (12.69)	11.64 (5.64)	14.27 (8.00)	5.23 (5.61)	6.15 (7.41)
CCL-A	8.21 (5.86)	11.70 (10.18)	12.95 (7.34)	14.32 (8.20)	5.12 (4.68)	5.62 (6.08)
RRS	49.39 (8.57)	53.55 (9.28)	49.09 (9.70)	50.09 (7.84)	41.08 (12.93)	42.35 (13.31)

Table 2: Mean Scores and Standard Deviations for Cognition Measures Pre and Post Negative MIP by Participant Group.

or control participants. A significant time by group interaction was also observed for change in reported rumination, as assessed by the RRS, $F(2, 78) = 3.57$, $p<0.05$. Follow-up paired samples t-tests indicated that the increase in reported rumination was statistically significant for the formerly depressed group, $t(32) = -4.55$, $p<0.001$, but not for currently anxious or control participants.

In summary, a significant effect from the MIP was observed for most of the cognition measures, including negative automatic thoughts, dysfunctional attitudes, anxiety- and depression-related cognition, and reported ruminative cognitive style, across the three participant groups. Time by group interactions were significant for measures of dysfunctional attitudes and ruminative cognitive style, but not for measures of negative automatic thoughts and anxiety- and depression-related cognition. Follow-up tests indicated that the degree of change for both dysfunctional attitudes and ruminative cognitive style was statistically significant only for the formerly depressed group, and not the currently anxious or control groups.

Discussion

The present study examined the specificity of cognitive reactivity to a depressive mood induction procedure (MIP) to individuals theoretically vulnerable to depression due to a history of Major Depressive Disorder (MDD), compared to a clinical control group of participants with a current anxiety disorder, and a community control sample including individuals with no history of depression or current anxiety disorder diagnosis. The use of these participant groups allowed comparison of cognitive reactivity to depressed mood across clinical populations, as well as between clinical and nonclinical populations. It was hypothesized that an increase in negative automatic thoughts and a decrease in positive automatic thoughts would be observed across participant groups; that formerly depressed participants would exhibit greater increase in dysfunctional attitudes, depression-related cognitions, and rumination than both currently anxious and control participants; and that currently anxious participants would exhibit greater increase in anxiety-related cognition than formerly depressed and control participants.

The mood induction procedure employed in the current study was effective, as a significant negative shift in mood was observed from pre- to post-mood induction procedure across the three participant groups on self-reported mood and writing speed. Significant slowing was also observed for the formerly depressed group on the psychomotor task of count time. Further, the extent of that the mood induction was comparable across groups, so observed group differences on measures of cognitive reactivity cannot be attributed to differential mood shift.

The primary result of the current study was related to cognitive reactivity to the MIP. Scores on the majority of the cognition measures changed significantly from pre- to post-mood induction procedure. Specifically, a significant increase in reported negative automatic thoughts, and anxiety- and depression-related cognition was observed from pre- to post-negative mood induction procedure, and these effects were comparable across the three participant groups. A significant increase in reported dysfunctional attitudes and rumination was also observed from pre- to post-negative mood induction; however, Time by Group interactions were also significant for both of these cognitive variables. Follow-up tests indicated that the increase in these measures was significant for the formerly depressed group only.

The majority of the results were as predicted, and build on and extend previous research on cognitive vulnerability in depression. A central theoretical proposition of cognitive models of depression [3,6] is the concept of cognitive vulnerability, which as discussed above argues that individuals vulnerable to depression possess a cognitive vulnerability to the disorder in the form of latent dysfunctional schema that are activated by stressful situations. Further, it was posited that such cognitive reactivity is unique to individuals vulnerable to depression, and contributes to the cognitive processes that precipitate depressive onset, relapse, and recurrence [3,6].

Empirical tests of cognitive vulnerability and reactivity in depression have relied heavily on mood priming designs, often utilizing mood induction procedures, among remitted depressed individuals who are theoretically vulnerable to depression due to possessing latent dysfunctional schema. Indeed, the assessment of attitudes with the Dysfunctional Attitude Scale (DAS) before and after laboratory mood induction has been identified as the "gold standard" procedure for indexing cognitive reactivity [48]. In the present study, the hypothesis that formerly depressed participants would exhibit greater increase in dysfunctional attitudes than both currently anxious and control participants was supported. Past research examining cognitive reactivity to depressive mood induction with the DAS has employed remitted depressed participants [14,15,49] or compared remitted depressed to never depressed participants [9,10,17,28]. As such, the present study extends the literature by suggesting specificity of cognitive reactivity as assessed with the DAS to formerly depressed individuals.

The DAS has become the most common measure of cognitive vulnerability and reactivity in priming research [3], and numerous studies have used it exclusively as a measure of depressive beliefs or schema [9,10,14,15,17]. The current study, however, extended past research through an examination of additional aspects of cognitive vulnerability and reactivity. The use of the Automatic Thoughts Questionnaire-Revised (ATQ-R) allowed for examination of the hypothesis that an increase in negative automatic thoughts would be observed across participant groups as a result of the negative mood induction, and this hypothesis was supported. The hypothesis of decreased positive automatic thoughts was not supported, though a nonsignificant trend was observed. Thus, while negative mood shift appears to be related to increased negative automatic thoughts and to a lesser extent decreased positive thought content, cognitive reactivity at the level of automatic thoughts was not found to be related to diagnostic status and as hypothesized, was not specific to individuals vulnerable to depression. These results are consistent with recent research conducted by Lau, Haigh, Christensen, Segal, and Taube-Schiff [50], who found following a negative mood induction procedure an increase in reported automatic thoughts (using the ATQ) across two groups including formerly and never depressed participants, while an increase in reported dysfunctional beliefs (using the DAS) was observed only for formerly depressed participants.

The Cognition Checklist (CCL) was also utilized as a measure of cognitive content and to assess the impact of mood shift on reporting of depression- and anxiety-related cognition. A significant increase in depression- and anxiety-related cognitive content was observed from pre to post mood induction procedure across the three participant groups, with no significant difference in extent of change observed across groups. These results, along with those found for negative automatic thoughts, suggest that the Velten mood induction procedure contributes to similar degree of increase in negative, depression- and anxiety-related cognition at the level of automatic thoughts across individuals with a history of depression, current anxiety, or neither.

Finally, the hypothesis that formerly depressed participants would exhibit greater increase in rumination, as assessed with the Ruminative Response Scale (RRS), than both currently anxious and control participants was supported, as a significant increase in rumination scores from pre to post mood induction procedure was observed only for formerly depressed participants. The response styles theory of depression hypothesizes that repetitive and passive thoughts about one's negative emotions, combined with a focus on the symptoms and meaning of one's distress, exacerbates and prolongs depression. Some of the mechanisms which have been implicated in this process include enhanced negative thinking, activation of negative cognition and memories, impaired active problem solving, increased pessimism and fatalism, interference with instrumental behavior, increased stress, and reduced social support, all of which are proposed to further contribute to depression [40,51]. Considerable evidence supports a link between rumination and increased negative thinking, poor problem solving, diminished instrumental behavior, and reduced social support among dysphoric and clinically depressed individuals [51]. Rumination has been found to predict depression [40,42,52], particularly onset of depressive symptoms and disorders [51], and anxiety symptoms [41]. Little attention has been paid to rumination as a marker of cognitive reactivity. Moulds et al. [48] found a positive relationship between self-reported cognitive reactivity and rumination after controlling for current symptoms of depression, but research has not examined the effect of depressive mood state following laboratory mood induction on reported rumination. The current study suggests that negative mood state is associated with increased self-report of rumination, and that this relationship is specific to individuals with a history of depression. This result is consistent with theoretical accounts that emphasize the contribution of rumination in onset and persistence of depressive symptoms [51] and warrants further investigation and replication.

In summary, the current results support cognitive reactivity in the form of increased reports of dysfunctional attitudes and rumination in response to negative mood states following laboratory mood induction. While mood-related negative, depressive, and anxious automatic thoughts were influenced by negative mood state regardless of past or current experience with depression or anxiety, increased reports of dysfunctional attitudes and ruminative cognitive style were specific to individuals with a history of depression. The present results extend past research in cognitive vulnerability and reactivity [3,6], and provide support for specificity of cognitive reactivity to individuals vulnerable to clinical depression, which is consistent with the content specificity hypothesis of the cognitive model of depression.

The present study had several limitations that warrant further consideration. The clinical samples of formerly depressed participants with no diagnosable anxiety disorder and currently anxious participants with no history of or current depression were defined in order to minimize the overlap between groups in terms of clinical symptomatology and to provide the strongest possible test of group differences in predictors of and cognitive reactivity to negative mood induction. However, given that "pure" depressive states may in fact be quite rare and comorbidity between anxiety and depression appears to be the rule, rather than the exception [53,54] the group definitions employed in the present study preclude the generalization of the current results to a significant portion of individuals with depression who also experience comorbid anxiety.

The degree to which the effects of laboratory-induced mood states generalize to naturally occurring mood has been a matter of some debate [29]. For example, the intentional nature of laboratory mood

induction procedures may limit generalizability to naturally occurring negative mood states. Further, the cognitive nature of the Velten MIP, which requires individuals to focus on a series of negative self-referent statements may also in and of itself impact reporting of cognition following the mood induction procedure. However, this potential limitation is mitigated by research finding increased reporting of dysfunctional attitudes following naturally occurring negative mood shifts [17], and mood induction procedures that do not rely on self-referent statements [9].

Although the Velten mood induction procedure is one of the most widely researched MIPs, numerous alternatives are available [14,15,55]. The use of the Velten MIP in the present research limits the ability to generalize the present results to other mood induction research that has employed alternate techniques.

Finally, there are generalizability issues related to the samples employed that warrant consideration. The sample was predominantly Caucasian and educated to the postsecondary level. Generalization to more diverse populations may be limited. Although all participants in the currently anxious group met diagnostic criteria for one of the anxiety disorders that constituted one of the inclusion criteria, a minority of participants also received treatment in the form of medication or psychotherapy for their anxiety and their anxiety scores fell within the "mild" range [56]. The degree to which the current results might apply to individuals with greater severity of anxiety symptomatology remains an issue for further study.

The limitations of the present study suggest several directions for future research. Given the high degree of comorbidity between depression and anxiety disorders [53,54] and theoretical questions concerning the relationship between the two diagnostic categories, research may include participants with comorbid depression and anxiety to further elucidate common and differential aspects of cognitive reactivity in depression and anxiety. The inclusion of participants with comorbid anxiety and depression would extend the mood induction literature beyond "purely" depressed populations or "purely" anxious samples, as examined in the present study, and as such would have significant implications for theoretical models of psychopathology. The external validity of such research would also be enhanced by more accurately reflecting clinical presentations, which often includes comorbidity.

Future research could examine cognitive reactivity to naturally occurring mood fluctuations. As discussed above, work by Miranda et al., [17] has found cognitive reactivity to naturally occurring negative mood states among individuals with a history of depression. Wenze, Gunthert, and Forand [57] also found that "naturally occurring" cognitive reactivity predicted depressive symptomatology at 6-month follow-up in a student sample. Future studies could examine additional aspects of cognitive reactivity, including both cognitive products and processes, as well as specificity to formerly depressed compared to additional clinical (e.g., anxious) samples. Future research could also evaluate the influence of various mood induction procedures on cognitive reactivity to mood induction.

The idea that cognitive reactivity to a negative mood induction procedure predicts relapse in remitted depressed individuals [6,14] has substantial clinical implications [3]. A growing body of research has identified cognitive reactivity as a predictor of depressive relapse and further, has begun to examine the relationships among cognitive intervention, cognitive reactivity, and vulnerability for depressive relapse. For example, Jarrett et al. [58] recently reported that among a sample of individuals with depression who responded to a course of cognitive therapy, increased unprimed dysfunctional attitudes following CT predicted depressive. Further, Van Rijsbergen et al. [59] similarly found that unprimed dysfunctional beliefs predicted relapse over 5.5 year follow-up following a course of preventive cognitive therapy (PCT), but found that while mood reactivity predicted relapse in their sample, cognitive reactivity did not . These authors do note, however, that increase in cognitive reactivity over time was associated with greater risk for relapse, suggesting that cognitive reactivity may need to be assessed over time to accurately examine its relationship with relapse. Further elucidation of the nature of cognitive reactivity in depression and its relationship with future risk for depression, could ultimately inform efforts aimed at both intervention and prevention.

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