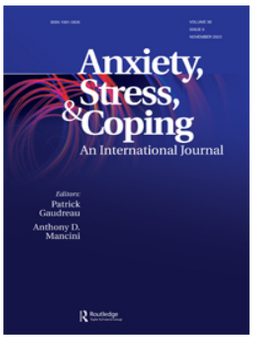

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Cognitive predictors of stress-induced mood malleability in depression

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ABSTRACT

Background & objectives: Basic attentional control, negative biases in attention and interpretation, and rumination are all cognitive processes associated with depression; however, less is known about their predictive role in depressive mood reactivity and -recovery in response to stress, and their relation to severity of depression.

Design & methods: We experimentally induced stress based on an autobiographical imagery script in a sample of 92 participants with Major Depressive Disorder with or without comorbid anxiety disorders. We used simple regression analysis for investigating the roles of state- and trait rumination, attentional networks, and attentional and interpretation biases for predicting stress-induced depressive mood reactivity and -recovery, respectively, and whether they in parallel mediated the association between cognitive processes and depression severity.

Results: Stress-induced depressive mood reactivity was predicted by better orienting ability and more state rumination. Better recovery was predicted by better orienting efficiency and lower negative interpretation bias. Furthermore, the relation between state rumination and depression severity was partially mediated by depressive mood reactivity, however limited by the lack of temporal precedence in the analysis.

Conclusions: We characterized the relation between cognitive processes and mood malleability in response to stress. Findings could refine theoretical models of depression if causality is established.

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Depression; stress; reactivity; recovery; cognition; mood

Introduction

Stressful life events have a detrimental robust and causal effect on depression (Hammen, 2005; Richter-Levin & Xu, 2018). When exposed to stress, depression has been linked with a heightened mood reactivity (i.e., *activation and intensity of mood*) and delayed mood recovery (i.e., *duration of mood*) (see Burke et al., 2005; Bylsma et al., 2008, for meta-analysis). The ability to effectively regulate one's own responses to stress and subsequent mood changes is dependent on cognitive processes (Joormann & Quinn, 2014). Aberrant cognitive processing is also implicated as in depression etiology (LeMoult

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& Gotlib, 2019). Depression has been linked with aberrant functioning in many aspects of cognition, such as reduced attentional control, increased rumination, and negative interpretation and attentional biases (Rock, Roiser, Riedel, & Blackwell, 2014). However, little is known about the relationship between these processes and mood reactivity and recovery (i.e., mood "malleability"). In turn, whether mood malleability influences the relation between cognitive processes and depression severity is not accounted for by theoretical models of depression (e.g., LeMoult & Gotlib, 2019).

Attentional processes

Some studies have investigated the prediciting role of singular cognitive processes in mood reactivity and recovery, and suggest that attentional processes influence the relation between stress and exacerbation of negative mood. Sanchez et al. (2013) studied the relation between attentional disengagement and stress in an experimental setting, including both participants with depression and control participants. In the overall sample, longer time to disengage attention from sad faces predicted increased sad mood during anticipatory stress. In addition, prolonged attentional disengagement also predicted sustained sad mood in the recovery period, although only in the depressed group. Using a stressful Stroop-challenge, Ellenbogen et al. (2006) found that slow disengagement from dysphoric pictures was associated with higher negative mood reactivity in both a mixed sample of patients with anxiety and depression and in healthy controls. In another study by Sanchez et al. (2017), they found that the link between stress recovery and depression symptoms after a stress-inducing speech task was mediated by attentional processes. More specifically, they found that reduced attentional disengagement *from* and extended engagement *with* negative stimuli, alongside longer fixation duration to negative relative to positive stimuli, was associated with increased depressive symptoms. Thus, mood reactivity and recovery after stress seems to be delayed in individuals that have an underlying vulnerability for dysregulated mood and show impairments in attentional disengagement.

Mood malleability as mediator

Attentional processes and stress may also relate to other cognitive factors that are relevant to depression. Ruscio et al. (2011) found that repetitive negative thoughts (RNT, i.e., rumination and worry) were associated with increased mood reactivity, and RNT also predicted persistence of negative affect after success feedback, suggesting that perseverative thoughts may hinder mood recovery after stress. Further nuancing the relation between attention and depression, Kertz et al. (2017) found a serial indirect effect of RNT and mood recovery on the relation between self-reported attentional control and trait anxiety and depression. A few additional studies have also investigated whether mood malleability could be a mediating factor between cognitive vulnerabilities and depression-related outcomes. In a study of children and adolescents differing in risk for depression, ability to regulate emotions was found to mediate the relationship between cognitive biases and depressive symptoms (Sfäerla et al., 2021). Furthermore, mood reactivity rather than cognitive reactivity, was found to predict depressive relapses (van Rijsberg et al., 2013). Together, these studies might point towards the need to refine current theoretical models of the relation between cognition and depression severity by including mood malleability as mediating process.

The present study

Summing up, a few studies, primarily conducted in various non-clinical populations, have examined the effects of specific cognitive factors on either stress-related mood reactivity, mood recovery, or depression in isolation (LeMoult & Gotlib, 2019), but to our knowledge no study had examined several of these factors simultaneously in a clinical sample and investigated their predictive role in both mood reactivity and recovery. Also, the mediating role of mood malleability in the relation

between cognitive processes and depression severity is underexplored. Hence, the present study investigates the interplay among three cognitive processes derived from the framework by LeMoult and Gotlib (2019) (i.e., attentional control, cognitive biases, rumination) and their relation to mood malleability in the context of stress in a clinical sample, and their relation to depression severity.

First, we investigate cognitive predictors of stress-induced mood malleability in a clinical population. We predict that reduced basic attentional control, negative biases in attention, negative interpretation bias, and increased state rumination, are associated with increased emotional reactivity and impaired recovery, respectively. Secondly, we examine whether mood malleability mediates the relation between cognitive processes and depression severity.

Methods

Participants

The present study is based on baseline data from a randomized controlled trial of attentional bias modification (ABM) for depression ($n = 92$; female $n = 67$) clinicaltrials.gov #NCT04137367 (Bø et al., 2023), including data from five participants randomized to the assessment only condition, a condition that was later discontinued due to restrictions posed by Covid-19.

Participants were recruited through local advertisements and in social media. All participants were assessed using the MINI International Neuropsychiatric Interview PLUS 5.0.0 (M.I.N.I). Inclusion criteria were a diagnosable major depressive disorder, with or without anxiety and with or without alcohol use disorder and age between 18 and 65. Exclusion criteria were mania, psychosis, and neurological disorder, but not hypomania, thus allowing participants with Bipolar-II to take part in this study.

Procedures

The study was approved by the Regional Committees for Medical and Health Research Ethics (REK Sør-Øst 2019/330) and conducted in accordance with the Helsinki declaration. All participants were given oral and written information about the study and provided informed written consent to take part in the study. The study took place in the neuropsychological laboratory at the Department of Psychology, University of Oslo.

In the first session, we conducted a diagnostic interview identifying diagnosable psychopathology, and an interview about an autobiographically stressful event, in accordance with the procedure described in Sinha and Tuit (2012). This session lasted about 60 min. Based on the interview about an autobiographically stressful event, researchers created an audiotaped imagery script to be used in the stress induction procedure in the next session (see Stress induction for details). Two weeks later, in the second session, participants completed self-report questionnaires and cognitive tests, and were subjected to the stress induction procedure. This session lasted for about 2.5 h.

Stress induction

The stress induction was based on Lang's theory of emotional imagery (1979) and was developed according to the manual of imagery script development by Sinha and Tuit (2012). An interview about details concerning a recent autobiographically stressful event that made them "sad, mad, or upset" and their associated bodily sensations, formed the basis for the script development. Participants rated the situation they described in terms of perceived stress on a 10-point Likert scale from 1 = not at all stressful and 10 = most stress they felt in the past year. Only situations rated 8 or above were considered suitable for script development. Stressful situations that included alcohol or drugs were not permitted.

After the interview, the researcher combined the obtained information into an imagery script, that was recorded in the voice of the researcher. By interweaving feelings, thoughts, urges and actions from the interview with physical sensations derived from a checklist of commonly experienced bodily sensations associated with emotional activation, the script gradually builds up tension, and ends at the most intense point of the event. The stress induction was personalized by autobiographical content, as one type of stressor is not distressing to all. For example, for some a quarrel with a friend could result in deep despair, for others the event would pass without any lasting effect on mood.

The stress induction procedure and associated assessment of mood is depicted in Figure 1. First, participants completed self-reports of mood and stress. Then, to induce stress, the participants used headphones to listen to the audiotaped imagery script. Participants were alone while listening to the script (using headphones) and instructed to have their eyes closed and visualize what they heard. After the recording ended (approx. three to four minutes), they were prompted to stay in the visualized situation and to think about their feelings and how they react. Listening and visualization lasted for a total of seven minutes. Immediately after, participants completed new self-reports of mood and stress. Then, participants completed a repeated session of ANT (not reported in this study), before they watched a National Geographic movie for 20 min alone. Finally, participants completed new self-reports of mood and stress, and were given the possibility to do a debrief. The total length of the procedure was approx. 65 min.

Measures and assessment

Momentary measures of stress and mood

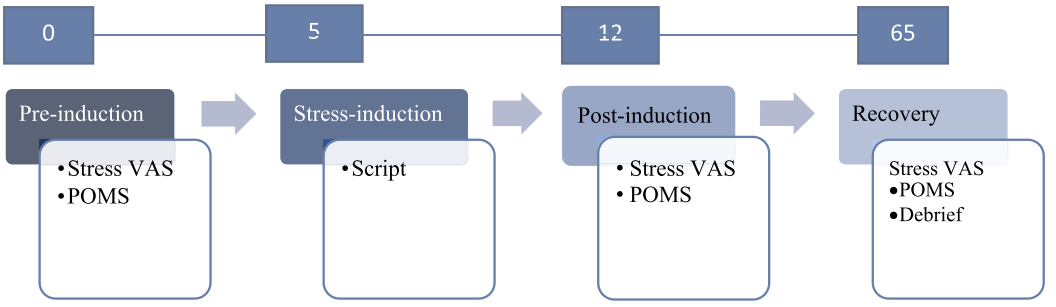
We employed a visual analogue scale (VAS) in measuring subjective stress, asking participants to indicate how stressed they felt in the moment on a scale from 0 to 100 (0 = not at all, 100 = extremely stressed).

The shortened version of the Profile of Mood States (POMS; Shacham, 1983) includes 37-item and has six sub-scales: depression, anxiety, fatigue, activity, confusion, and anger. POMS depression (Cronbach's α: pre-induction = .932, post induction = .898, recovery = .934) was used as an indicator of negative mood.

Symptom measures

Depressive symptoms were investigated by means of the Beck Depression Inventory–II (BDI-II; Beck, Steer, & Carbin, 1988; Cronbach's α = .870). Anxiety was measured by means of the Beck Anxiety

Overview of the stress induction procedure.



Note. Numbers represent the timeline in minutes. BSRI = Brief state rumination inventory, POMS = Profile of mood states, VAS = Visual analogue scale.

Figure 1. Overview of the stress induction procedure.

Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988; Cronbach's α = .886). Alcohol consumption last 30 days was assessed by means of Timeline Follow Back (Sobell & Sobell, 1992).

Cognitive measures

Interpretation biases. In the Facial Expression Recognition Task (FERT; Harmer et al., 2009), facial expressions of the six basic emotions (sad, disgust, fear, surprise, happy, anger) are morphed in increments of 10% from neutral (0%) to prototypical expression (100%). A total of 250 stimuli was shown (6 emotions x 10 intensities x 4 examples + 10 neutral expressions). Facial stimuli were presented for 500 msec in random order on a computer screen and then replaced by a blank screen. By pressing one of seven keys labeled with the different emotions (included neutral) on the keyboard, participants indicate what emotion that was displayed. Participants were asked to respond as fast and accurate as possible.

Based on accuracy (i.e., the number of faces correctly identified as containing any intensity of a particular emotion identified divided by total number of faces containing that emotion) and misclassifications (i.e., false alarms), we computed the β (Grier, 1971) for sad and happy facial expressions respectively (emotions central to depression, e.g., Bourke et al., 2010). Compared to simple accuracy measures, the β accounts for expectations and previous knowledge when making decisions on how to interpret stimuli, thereby measuring the top-down process of how much evidence a person requires before he or she is willing to report that the emotion is present. The lower the value from 0 up to, but not including 1 the more liberal the person is when responding that the signal is present.

Attentional bias. We used a computerized visual dot probe task for estimating attentional bias (Browning, Holmes, Charles, Cowen, & Harmer, 2012). Paired stimuli of face images in three valences: positive (happy), negative (fearful and angry), and neutral was presented vertically on the computer screen. Angry and fearful faces are known to evoke amygdala activation which is suggested to trigger the neurobiological foundation of attentional biases (Shackman et al., 2016). One or two dots (a probe) were presented in the same location on the screen where the stimuli were previously displayed. The task was to fast and accurately indicate the correct number of dots in the probe. The task included 96 trials with equal number of each stimulus pair. The stimuli were randomly presented for either 500 *ms* or 1000 *ms* before the probe appeared. By calculating the difference in reaction time in milliseconds between trials where the probe replaced the relatively more negative face vs. the more positive face, we derived the attentional bias score (see Jonassen et al., 2019 for calculations). Hence, a negative score reflects an attentional bias towards negative stimuli.

Attentional control. Attentional functioning was assessed along the lines of Posner's theory of attentional networks, namely alerting, orienting, and executive control using the Attentional Network Task (ANT; Fan et al., 2002). Participants are required to respond to right- or left-pointing arrows shown on a screen by pressing the corresponding button on a keyboard as fast and accurate as possible, recording reaction time in msec. Each trial consists of a fixation cross, a cue (no cue, center cue, double cue, or spatial cue), a fixation cross, and then the target (central arrow pointing left or right, flankered by congruent or incongruent arrows or neutral stimuli). ANT consists of a practice block and three experimental blocks á 96 trials presented in random order. See Fan et al. (2002) for details regarding experimental procedure.

Efficiency of the alerting network is examined by changes in reaction time resulting from a warning signal (i.e., no cue vs double cue). Efficiency of the orienting network is examined by changes in the reaction time associated with cues indicating that and where the target will occur (i.e., center cue vs spatial cue). The efficiency of the executive network is examined by subtracting reaction times to central arrows surrounded by congruent from incongruent flankers. A high executive score represents poorer functioning, while lower values for alerting and orienting represent poorer performance (DeJong et al., 2019).

Rumination. Trait rumination was measured using the Ruminative Responses scale, which is a 22-item scale containing three factors: depression, brooding, and reflective pondering. Each item is

rated on a 4-point Likert scale ranging from 1 (never) to 4 (always). We report results of the brooding scale (Cronbach's α = .761), which, together with reflective pondering, are considered unconfounded by depression content (Treynor et al., 2003) and found to have a more pronounced role compared to reflective pondering for depression-relevant processing (e.g., Allard & Yaroslavsky, 2019). State rumination was measured using the Brief State Ruminative Inventory (BSRI; Marchetti et al., 2018; Cronbach's α = .857). The BSRI consists of 8 items (e.g., Right now, I am thinking: Why do I have problems other people don't have?) and is a valid and reliable measure of state rumination. Each item is rated on a 100-mm visual analogue scale (VAS) ranging from "completely disagree" to "completely agree", and we report the sum score. While state and trait rumination are moderately correlated, previous research has shown that state, but not trait rumination is specifically linked to reactivity to a stressor (Hilt et al., 2015), and that state rumination is linked to recovery from a stressor, even when trait rumination is controlled for (LeMoult et al., 2013).

Statistical analysis

Descriptive statistics were used to characterize the sample. Pearson correlations were used to assess the relationships between the cognitive processes and BDI-II. To conduct manipulation checks of the stress induction, one-way repeated measures ANOVAs were used for examining changes in subjective stress and depressed mood from pre to post induction and from post induction to recovery.

To analyze the predictive role of cognitive variables on mood reactivity and recovery, we estimated simple linear regression models for each of the predictor variables (FERT variables, ANT-variables, dot probe, brooding, and state rumination). Mood reactivity was operationalized as residual change in negative mood from pre to post induction, while mood recovery was operationalized as residual change in negative mood from post induction to recovery. Higher scores reflect greater mood reactivity (i.e., increases in negative mood from pre to post induction) and better mood recovery. Relying on residual change scores of mood reactivity and mood recovery controls for baseline levels of negative mood and is a reliable method for investigating mood change (Segal et al., 2006). Compared to other mood change scores (e.g., difference scores), this method allows us to investigate variability among residuals as independent from the variability of previous mood scores (Cohen et al., 2002). Raw score equivalents were obtained by summing the residuals and the negative mood change scores to facilitate interpretations of the results.

Six participants had missing data on reactivity and recovery due to technical problems during data acquisition. In addition, there was missing data on RRS ($n = 1$), FERT ($n = 1$), and ANT ($n = 4$).

Finally, to further elucidate the role between cognitive factors and depression severity, we investigated whether mood malleability (i.e., reactivity and recovery, respectively) in parallel mediated the significant correlations between cognitive variables and BDI-II, even when temporal precedence was not settled. This was done by means of the PROCESS macro in SPSS (Hayes, 2017) testing the traditional a, b, and c′ paths. In addition, the PROCESS estimates the direct and indirect effects using 5000 bias-corrected bootstrapped 95% confidence intervals (*CI*s). A *CI* that does not contain zero indicates an effect that is statistically different from zero at $p < .05$.

All statistical analysis were performed using IBM SPSS 27.

Results

Participant characteristics and predictor variables

Table 1 shows the characteristics of the sample. The majority of the sample were middle-aged females with recurrent and moderate to severe depression symptoms, comorbid anxiety disorders, and an education level equivalent to a bachelor's degree or above.

Table 2 shows the descriptive and correlation among predictor variables and depression severity. Most predictor variables were not significantly correlated, suggesting that they capture different

Table 1. Sample characteristics.

Gender (*n*)	92 (100%)
Male	25 (27%)
Female	67 (73%)
Age in years	43.4 (11.3)
Education (ISCED)	5.6 (1.4)
BDI-II[a]	24.4 (9.9)
Minimal (0–13) (*n*)	16 (18%)
Mild (14–19) (*n*)	11 (12%)
Moderate (20–28) (*n*)	34 (37%)
Severe (29–63) (*n*)	30 (33%)
BAI[a]	14.6 (8.6)
Minimal (0–7) (*n*)	21 (23%)
Mild (8–15) (*n*)	35 (38%)
Moderate (16–25) (*n*)	23 (25%)
Severe (26–63) (*n*)	12 (13%)
Major depressive disorder (*n*)	92 (100%)
Previous depressive episode	88 (96%)
Current depressive episode	40 (43%)
Median number of episodes	5 (3–18)
Current comorbid anxiety disorder[b] (*n*)	66 (72%)
Social phobia	28 (30%)
Specific phobia	11 (12%)
Agoraphobia	18 (20%)
Panic disorder	14 (15%)
Current dysthymia (*n*)	20 (22%)
Current obsessive-compulsive disorder (*n*)	7 (8%)
Current post traumatic stress disorder (*n*)	6 (7%)
Current alcohol use disorder (*n*)	17 (18%)
Current substance use disorder (*n*)	7 (8%)
Previous hypomanic episode (*n*)	9 (10%)
Antidepressant medication[a] (*n*)	36 (39%)
SSRI	30 (33%)
SNRI	3 (3%)
Atypical	3 (3%)
Number of alcohol units last 30 days	16.4 (19.4)

Notes: Data are shown as frequency (%), mean (SD), and median (IQR). BAI = Beck Anxiety Inventory, BDI = Beck Depression Inventory–II, SNRI = Selective noradrenaline reuptake inhibitor, SSRI = Selective serotonin reuptake inhibitor.
[a]Missing data for one participant.
[b]Number does not add up, as some participants have more than one disorders.

aspects of cognitive processing. BDI-II was moderately correlated with RRS brooding and BSRI, only, and they were in turn moderately correlated with each other.

Manipulation check

There was a significant effect of the stress induction on self-reported stress, F (2,88) = 72.46, $p < .001$, Wilks' $\lambda = .38$. Post hoc tests indicated that stress levels increased after stress induction: mean stress level was 23.9 ($SE = 2.1$) pre-induction, and 47.3 ($SE = 2.8$) post induction. Stress levels decreased from post induction to recovery ($M = 17.9$, $SE = 17.6$, $p < .01$). There was also a decrease in stress levels from pre-induction to recovery ($p < .05$). On average, stress increased by 98% from pre to post induction. Thirteen percent of the sample reported decreased or no change in stress in response to the stress induction.

There was a significant effect of the stress induction on negative mood, F (2,84) = 47.33, $p < .001$, Wilks' $\lambda = .47$. Post hoc tests indicated that negative mood increased from pre-induction ($M = 0.69$, $SE = 0.1$) to post induction ($M = 1.34$, $SE = 0.10$, $p < .001$). There was a significant difference between negative mood at post induction and at recovery ($M = 0.63$, $SE = 0.8$, $p < .001$). There was no difference in negative mood between pre-induction and recovery. On average, negative mood increased by 95% from pre to post induction. Twenty-four percent of the sample reported decreased

Table 2. Descriptives and Pearson's correlations among predictor variables.

	Mean	SD	1	2	3	4	5	6	7	8
1 Negative interpretation bias	0.6	0.2								
2 Positive interpretation bias	0.9	0.1	−.29** [.09, .47]							
3 Attentional bias	0.5	37.4	−.16 [−.33, .05]	−.20 [−.39, .01]						
4 Alerting	26.5	32.7	−.10 [−.31, .11]	.03 [−.19, .24]	−.19 [−.39, .0,]					
5 Orienting	64.9	38.9	−.26* [−.45, −.05]	−.02 [−.23, .12]	−.15 [−.25, .07]	.02 [−.20, .29]				
6 Executive	138.4	80.1	.01 [−.20, .22]	.22* [.01, .41]	−.13 [−.33, .08]	.11 [−.10, .32]	−.19 [−.38, .03]			
7 Trait rumination	12.3	3.6	.03 [−.18, .23]	−.08 [−.28, .13]	−.14 [−.34, .07]	.06 [−.15, .27]	.01 [−.21, .22]	−.01 [−.22, .21]		
8 State rumination	365.9	183.7	−.07 [−.27, .14]	−.04 [−.24, .17]	.09 [−.12, .29]	.10 [−.12, .30]	−.04 [−.25, .17]	.05 [−.16, .26]	.30** [.10, .47]	
9 BDI-II	24.4	9.9	−.18 [−.38, .03]	−.01 [−.22, .19]	.03 [−.18, 23]	.02 [−.19, .23]	−.02 [−.24, .19]	−.11 [−.31, .10]	.42** [.23, .58]	.31** [.11, .49]

Notes: Values in square brackets indicate the 95% confidence interval for each correlation. BDI-II = Beck's depression inventory–II, SD = standard deviation.
*$p < .05$, **$p < .01$.

or no change in negative mood in response to the stress induction. See Figure S1 in Supplementary Materials for details regarding changes in other mood factors as measured by POMS.

By qualitatively assessing the content of a random sample of scripts ($n = 10$) we found that all described a situation where they felt upset, and half of them also reported being either sad or mad.

Cognitive processes predicting mood reactivity and -recovery

Regarding mood reactivity, orienting $b = .004$, $t = 2.285$, $p = .025$ was a significant predictor, yielding a significant regression equation, $F(1, 80) = 5.221$, $p = .025$ with an R^2 of .061. (Described in raw score equivalents, this implies that 1 *SD* increase in orienting is associated with an increase in reactivity of .24). State rumination was also a significant predictor of reactivity, $b = .001$, $t = 2.190$, $p = .031$, yielding a significant regression equation $F(1, 84) = 4.797$, $p = .031$, with an R^2 of .054. (+1 *SD* in state rumination = 0.2 mood reactivity units). Alerting was a marginally significant predictor, $b = .004$, $t = 1.798$, $p = .076$, yielding a marginally significant regression equation $F(1, 80) = 3.232$, $p = .076$, with an R^2 of .061. Positive interpretation bias (being more conservative when reporting positive emotions) was a marginally significant predictor, $b = 1.816$, $t = 1.919$, $p = .058$, yielding a marginally significant regression equation $F(1, 83) = 3.681$, $p = .058$, with an R^2 of .042. None of the three other predictors reached significance, all t's < 1.7., all p's > .1.

Regarding mood recovery, attentional bias was a significant predictor, $b = .004$, $t = 2.563$, $p = .012$, yielding a significant regression equation, $F(1,84) = 6.569$, $p = .012$, with an R^2 of .073. (+1 *SD* in attentional bias = 0.27 mood recovery units). Negative interpretation bias (being more conservative when reporting sad emotions) was a significant predictor, $b = .892$, $t = 2.945$, $p = .004$, yielding a marginally significant regression equation, $F(1, 83) = 8.671$, $p = .004$, with an R^2 of .095. (+1SD in negative interpretation bias = 0.27 mood recovery units). None of the other predictors reached significance,

all t's < +/- 1.25., all p's > .2. Imputing the mean for the missing values rendered the results unchanged.

Mood malleability as mediator between cognitive processes and depression severity

Two separate parallel mediation analyses, limited by lack of temporal precedence, were conducted to examine the mediating effect of mood malleability on state- and trait rumination (the only two factors significantly associated with BDI-II, see Table 2), and depression severity.

For state rumination, the total effect of the model was found to be significant, b = .017, *se* = .0056, *t* = 3.01, *CI* [.0057, .00282], p = .004. There was a statistically significant direct effect, b = .0113, *SE* = .0054, *t* = 2.10, *CI* [.0006, .0220], p = .04. A statistically significant total indirect effect of mood malleability was not found, b = .0057, CI [-.0007, .0128], however, there was a partial mediation effect of reactivity, b = .0005, CI [.0001, .0011], but not recovery, b = .000, CI [-.0006, .0006]. These results suggest that reactivity, but not recovery, partially mediated the relation between state rumination and depression severity.

For trait rumination, the total effect of the model was significant, *b* = 1.190, *se* = .28, *t* = 4.25, *CI* [.634, 1.75], *p* = .0001. There was a statistically significant direct effect, *b* = .968, *SE* = .26, *t* = 3.73, *CI* [.452, 1.48], p = .0004, but not a statistically significant total indirect effect of mood malleability, *b* = .220, *CI* [-.038, .551]. These results suggest that mood malleability did not mediate the relation between trait rumination and depression severity (Figure 2).

Discussion

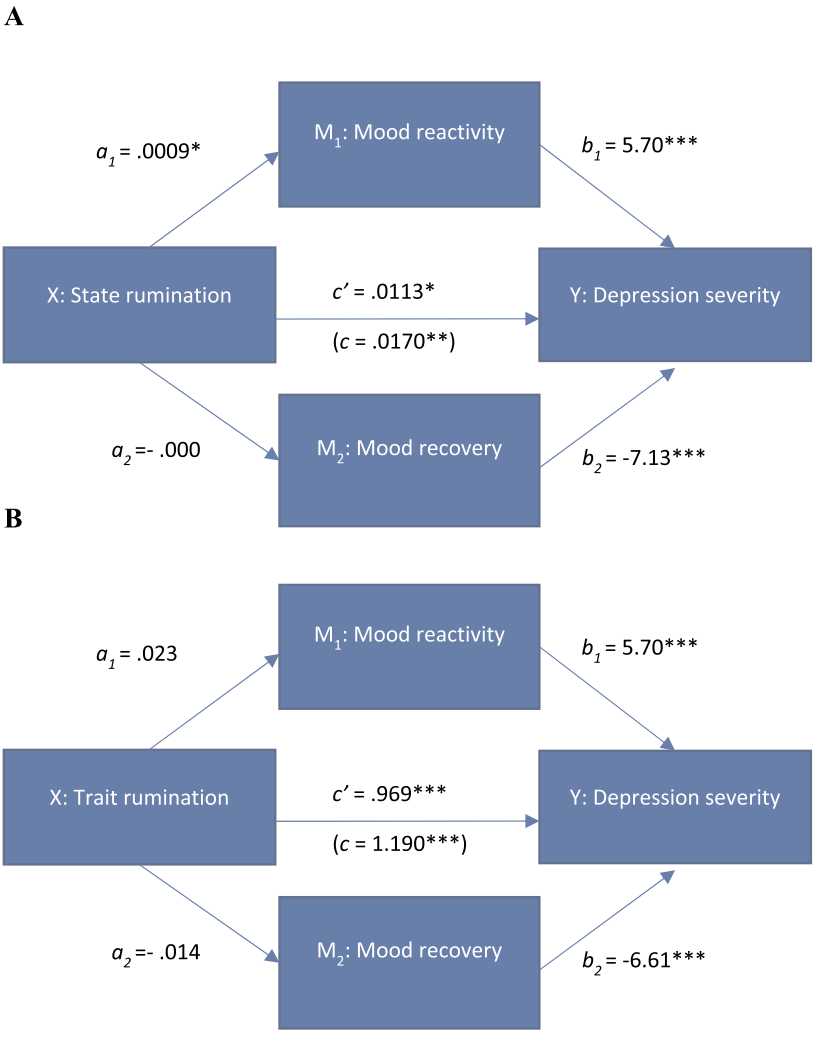
Theoretical models have pointed to the relevance of various cognitive processes in concert with stressful life events for the exacerbation and maintenance of depressed mood, however few studies have been conducted in clinical samples, and theoretical models (e.g., LeMoult & Gotlib, 2019) have not included mood reactivity and recovery as relevant predictors of depression-related outcomes. This study emphasized the predictive role of cognitive processes in the exacerbation of, and recovery from, depressed mood in response to stress, and investigated whether mood malleability mediated the relation between cognitive processes and severity of depression.

Regarding our first aim, we found that mood reactivity and mood recovery were associated with different cognitive processes. Mood reactivity was predicted by increased orienting ability and state rumination, and alerting and positive interpretation bias were marginal predictors, whereas better recovery was predicted by less negative interpretation bias and negative attentional bias. Regarding our second aim, we found that mood reactivity, but not recovery, partially mediated the relation between state rumination, but not trait rumination, and depression severity. This finding served to expand current theoretical models of the relation between rumination and depression (e.g., LeMoult & Gotlib, 2019), and suggests that stress-induced mood malleability perhaps should be included as predictor of depression severity and that reactivity, if replicated, should be included as partial mediator of the relation between state rumination and depression.

Mood reactivity

The relationship between superior orienting ability and mood reactivity has previously not been discussed in the literature. Increased orienting efficiency might imply that individuals whose attentional systems are aligned in favor of externally stimuli are more prone to experiencing negative mood during stress. Furthermore, this may downstream lead to activation of negative thoughts. Accordingly, we found that increased state rumination (i.e., reporting an increased focus on negative thoughts and emotions) was related to mood reactivity. Alternatively, the aforementioned finding may be explained by the general effect that increased orienting may have on

Graphical representation of mediation analysis.



Note. *Direct and indirect effects of **A** state rumination and **B** trait rumination on depression severity.* There is a lack of temporal precedence supporting this analysis. Numbers are unstandardized coefficients. * $p = .05$, *** $p = .001$.

Figure 2. Graphical representation of mediation analysis.

perception. Individuals who are more externally oriented may be more impacted by all types of external stimuli, also stress-related stimuli. Such a general effect on perception may explain the observed relationship between increased orienting efficiency and mood reactivity. This may be especially relevant for our stress induction procedure, where participants were actively instructed to focus on what is heard in the audiotape (external focus) and the feelings which are present (a process similar to rumination – hence, the relation between state rumination and reactivity was a rather tautological finding).

Mood recovery

Negative interpretation bias was associated with worse recovery. The finding may indicate that negative interpretation bias may hinder recovery and maintain negative mood states through continuous feeding of negative material into working memory. Accordingly, among high dysphoric persons, difficulties disengaging attention from emotional expressions have been related to deficient mood recovery (LeMoult et al., 2013). This finding begs the question of whether interventions targeting cognitive processes may be effective in treating depression. Since negative interpretation bias is known to be causally related to depression (Mathews & MacLeod, 2005), is modifiable and associated with stress reactivity (Joormann et al., 2015), it would be worth investigating the causal nature between interpretation bias and recovery, and whether change in bais towards more positive interpretations could improve recovery, and subsequent the severity of depression. On a contradicotry note, however, positive interpretation bias was unrelated to mood malleability, but this could potentially be related to lack of variance in response style attributable to happy faces.

We found a significant relation between attentional bias and recovery after stress, and this corresponds to previous findings among unselected populations (Applehans & Luecken, 2006; Fox et al., 2010). Perhaps the most striking findings, however, is the absence of association between cognitive control, and mood malleability. There is a vast literature on the role of cognitive control deficits in emotion regulation and depression. However, we did not assess cognitive control during stress, and it is possible that dysfunctions are more prominent during stress activation (e.g., Quinn et al., 2020; Quinn & Joormann, 2020). We also did not find a significant relation between state and trait rumination and recovery, and this contradicts findings from studies with samples of adolescents and university students (Hilt et al., 2015; LeMoult et al., 2013; Shapero et al., 2017). This lack of correspondence to our results might simply caution against generalizing finding from healthy to clinical samples or that we assessed reactivity in the form of self-report rather than by physiological indices.

Mood malleability as mediator

We found that increased reactivity and deficient recovery from psychological stress, predicted depression severity. *Increased* reactivity contradicts previous findings, in that depression typically has been associate with *blunted* reactivity and recovery compared to healthy controls (Burke et al., 2005; Bylsma et al., 2008). The categorical approach to psychopathology taken in former studies (i.e., comparing healthy controls and clinical groups), may have masked the correlation between mood malleability and depression severity, and this study therefore provides support to dimensional understandings of psychopathology (e.g., RDoC- initiative; Cuthbert & Insel, 2013). In addition, we found a relationship between depression severity and trait and state rumination a finding that is well characterized in the literature (Nolen-Hoeksema, 2000). Interestingly, mood reactivity was partially found to mediate the relationship between state rumination and depression severity, a finding that could expand current theoretical models of depression if replicated. Trait rumination was unrelated to mood malleability, and this could be due to the aggregated events underlying the assessment of trait rumination, whereas state rumination, on the other hand, had an episode-specific relationship to the stress induction. A cautionary note relates to the temporal precedence in the mediation analysis, as measures of depression severity were adopted prior to assessment of mood malleability. Furthermore, replications in clinical samples are indeed needed, as we failed to replicate the serial indirect effect from repeated negative thoughts to recovery to depression and anxiety by Kertz et al. (2017). Alternatively, this lack of correspondence to our results might relate to the failure of generalizing findings from healthy to clinical samples. It may also suggest that studies aimed at understanding how depression is maintained and how it is related to the severity of episodes indeed require clinical samples.

Furthermore, we found that mood variability in response to stress (i.e., increased reactivity and impaired recovery), after controlling for rumination, was predictive of depression severity. Indeed,

impaired ability to recover from negative mood after stress, has been strongly related to depression outcomes (Burke et al., 2005). The relation between reactivity and depression outcomes, on the other hand, seems more complex (e.g., Perez & Rohan, 2021; Vanderhasselt et al., 2016), and may potentially be related to the operationalization of reactivity, and how reactivity interacts with context.

Future studies and limitations

Stress inductions vary in their potential for eliciting a stress response (Ferreira, 2019). The current stress induction procedure was based on imagery scripts personalized in line with autobiographical memories of a recent stressful life event (Sinha & Tuit, 2012). Results showed that this procedure led to increased self-reported stress and negative mood for most of the participants. At recovery, mean negative mood recovered to pre-induction levels, and mean stress level decreased below pre-induction levels, suggesting that this is a tolerable procedure for inducing stress in clinical populations. A minority reported no change or decreased negative mood and stress in response to the induction. Non-response is quite typical to stress induction procedures, but still, due to the personalized format of this stress induction, we would have expected fewer non-responders. The personalized procedure was contrasting the typically employed stress induction procedures, the Trier Stress Test (Kirschbaum et al., 1993) that induces social-evaluative threat and uncountability, both of which the current paradigm lacked. On the other hand, the current paradigm reflected a more naturalistic account of the daily stress events experienced by the participants, the procedure is well-validated, and has been shown to elicit stress-related physiological changes compared to exposure to neutral imagery (Sinha, 2009). While we cannot fully ascertain that the procedure affected all participants equally, the qualitative assessment of a minority of the scripts found that all described an event that made them upset, with or without reference to being sad or mad.

The generalizability of our findings is uncertain. To draw firm conclusions, the study needs replication, preferably also by including a healthy control group to establish whether the findings are specific to clinical populations. Future studies should also establish whether the association between cognitive processes, mood malleability and depression severity are causal, include objective measures of stress, and investigate how biological stress and cognitive factors interact in intensifying depressed mood (see LeMoult, 2020).

Conclusion

This study expands our knowledge on the association between cognitive processes, stress, and depressed mood, and serves to nuance which cognitive processes that are associated with reactivity and recovery of depressed mood in response to stress in a clinical sample. Mood reactivity was associated with attentional orienting and state rumination, whereas mood recovery was associated with negative interpretation bias and attentional bias. Mood reactivity partially mediated the relation between state rumination and depression severity, a finding that requires further investigation with temporal precedence. If causality is established, interventions targeting these processes may improve resilience to stress by attenuating mood deterioration in clinical populations.

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Disclosure statement

NIL has received consultancy fees and travel expenses from Lundbeck outside this work. CJH has received consultancy fees from P1vital, Lundbeck, Sage Therapeutics, Compass Pathways, Zogenix outside of this work. The remaining author (s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Data availability statement

Data are not publicly available.

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