State and Trait Influences on Inhibition in Pre-Clinical Depression

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The cognitive symptoms of depression may be the result of the interactive effects of mood state and depressive trait factors on inhibition. Both innate (trait) and environmental (state) factors are known to be involved in depression. Previous research suggests both contribute to inhibition of emotionally valenced material. However, the use of clinical (currently depressed and remitted) populations in the literature poses a substantial problem for the study of the interaction between state and trait effects on cognitive processes. Therefore, this study used a sub-clinical population. Participants were randomly selected via the online research participation system and divided into high (BDI ≥ 9, n = 27) and low (BDI ≤ 8, n = 25) depressive trait groups. In addition a within-subjects mood induction component was used to dissociate the effects of trait and state on inhibition for valenced material. To measure inhibition for negative and positive words, participants completed the Negative Affective Priming (NAP) task. Consistent with the hypotheses, the results from a Mixed-Factor ANOVA show a significant interaction between state and trait effects. Thus, it is concluded that a reciprocal relationship between environmental and innate factors exist, resulting in depressive symptoms.

Keywords: depression, Negative Affective Priming (NAP), inhibition, mood induction

Negative content appears to be more salient to depressed individuals (Gotlib & Joormann, 2010; Phillips, Hine, & Bhullar, 2012), which indicates that unconscious priming of negative material may be occurring in depressed individuals. Consistent with this explanation, there appears to be a pattern of attentional biases unique to depression. The Negative Affective Priming (NAP) task was developed by Joormann (2004) to examine the role of inhibitory processes in depression. It requires subjects to respond to a target word (denoted by a particular colour) while ignoring a distractor word of the opposite valence (e.g., "please indicate if the blue word is positive or negative"). On some trials the target is positive, on others it is negative, and reaction times are analyzed based on the previous target valence. By subtracting reaction times on congruent (positive prime) trials from reactions times on incongruent (negative prime trials), it is possible to calculate the cost associated with previously ignoring a word of the same valence as the current target. This NAP effect indicates the strength of inhibition associated with each word type, because responding to the target requires inhibition of the distractor. This inhibition is not specific to the word presented, but rather generalizes to semantically related words. Thus, typically participants are slower to respond to a target word if it shares a valence with a previously ignored word (Joormann, 2004). However, depressed individuals do not show this expected cost for negative words (Frings, Wentura, &

Holtz, 2007; Gotlib & Joormann, 2010; Joormann, 2004), which suggests that they are unable to inhibit the negative material.

Inhibition, Negative Schema, and Rumination

A predisposition towards negative thought patterns may constitute a vulnerability towards depression. It has been proposed that a negative schema results in a lack of inhibition for negative material, which then makes negative material in the environment more salient. Consequently, more negative material enters working memory (WM), reducing the amount of space available for positive material and reinforcing the negative schema (Gotlib & Joormann, 2010). In support of this prediction, participants with high scores on the Ruminative scale of the Response Style Questionnaire (RSQ-R) showed significant inhibition of emotional material while low RSQ-R scorers showed no inhibition (Joormann, 2006). Using a 4-point Likert scale, the RSQ-R measures a person's endorsement of sad/depressed thoughts. Higher scores indicate a greater propensity to ruminate on negative material. Hence, those who tended to dwell on negative situations were less able to prevent negative material from entering WM. Of particular note, the relationship between negative patterns of cognition and rumination increased with age (LaGrange et al., 2011), which is consistent with the idea of a negative schema being continually reinforced. Interestingly, although this negative cognitive bias may predict the intensity of depressive symptoms (Carter & Garber, 2011) it does not predict the onset of depression (LaGrange et al., 2011) suggesting an interaction between an innate factor (e.g., negative schema) and some environmental trigger (e.g., negative stimuli).

State vs. Trait Effects on Depression

There is strong evidence for environmental factors playing a role in depression. For example, it is more common in isolated individuals (Sadock & Sadock, 2003), and in any given time period approximately 5% of Canadians and as much as 20% of Egyptians suffer from depression (Beshai, Dobson, & Adel, 2012). This difference may be due to political instability and the salience of conflict in Middle Eastern countries, suggesting a negative mood induction component. More importantly, depression presents differently in Middle Eastern countries. Beshai and colleagues (2012) found that Islamic patients had more somatic symptoms and fewer guilty feelings. They attributed this to cultural norms of suppressing ones emotions and externalizing guilt (compared to the Christian notion of original sin, thus internalizing guilt). This suggests that even broader situational factors (e.g., culture, religious environment, political situation, etc.) in addition to the more immediate environment (e.g., family situation, job satisfaction, social encounters, etc.) play a crucial role in determining how the internal affective state is expressed (Meyer & Hokanson, 1985).

There is evidence suggesting that innate and external factors interact. For example, higher stress levels lead to increased depressive symptoms in people who are prone to negative cognitions (i.e., the glass if half empty; Carter & Garber, 2011). Similarly, following a negative mood induction, low self-esteem subjects remembered more negative material, thus demonstrating more mood congruent recall (Joormann & Siemer, 2004). However, the model proposed by Gotlib and Joormann (2010) does not fully account for this possible interaction. More specifically it is unclear how this external factor can exert its influence on the inhibitory processes central to their model. The NAP paradigm shows a very robust NAP effect for negative words in depressive trait individuals, such that a general lack of inhibition for negative words in high trait individuals compared to controls is found (Frings et al., 2007; Gotlib & Joormann, 2010; Joormann, 2004) However, several problems exist with the operational definition of

high depressed trait. Firstly, using a clinically depressed population is problematic since an ongoing depressive episode is not simply an effect of innate factors (disease diagnosis), but there is also an ongoing environmental component (severity of symptoms). This is due to a tendency to withdraw from the world, which translates into less engagement in pleasurable activities (Veale, 2008). Thus, their world becomes progressively more negative, which acts as a constant negative mood induction.

The fact that Positron Emissions Tomography (PET) data show similar brain regions are activated in remitted patients following a negative mood induction and currently depressed individuals (Liotti, Mayberg, McGinnis, Brannan, & Jerabek, 2002), suggests that the remission stage of the disorder is in fact the depressive trait with state effects removed. However, these populations show highly mixed results on the NAP task. Joormann (2004) showed no negative priming for negative words in the previously depressed groups. However, the remitted group in Gotlib and Joorman (2010), showed the exact opposite. In addition, remitted patients seem particularly affected by environmental cues (Harkness, Jacobson, Duong, & Sabbagh, 2010; Scher, Ingram, & Segal, 2005). Consequently, when a self-referential component was added to the NAP task, inhibition for negative words was further reduced in the previously depressed group (Joormann, 2004). Therefore the mixed result may be due to the depressive trait and mood state being conflated in some studies but not in others. Thus, this population does not provide the necessary control needed to properly examine the state-trait interaction in inhibition.

The Current Study

To simulate this interaction within the laboratory a mood induction paradigm devised by Goeleven, De Raedt, and Koster (2007) was superimposed on the NAP task as described by Joormann (2004). Thus, two specific questions could be addressed: 1) is there a similarly robust effect of mood state as there is for depressive trait on inhibition as measured by the NAP task? 2) Is the interaction between trait and state evident when these factors are not confounded as they are in clinical populations?

Three a priori hypotheses were generated based on previous research: 1) the effect of depressive trait found in the original Joormann (2004) paper would also be found. Thus, the high depressed trait (H-BDI) group would show a reduced overall NAP effect for negative words compared to positive words, 2) the low depressed trait (L-BDI) group would show a reduced NAP effect for both positive and negative words (Pnap and Nnap) following the positive mood induction (based on findings by Goeleven et al., 2007), and 3) that an interaction between depressive trait and mood state would exist. If this is true then not only would trait influence the material entering WM, but mood state would also have an effect. Specifically it was hypothesized that in the H-BDI group, the positive mood induction would reduce inhibition for positive material (decreased Pnap), while the negative mood induction would further reduce inhibition for negative material (decreased Nnap).

Methods

Participants

A total of 56 undergraduate students at Simon Fraser University, with normal or corrected to normal vision, received course credit for their participation. A BDI cut-off of 9 was used to split subjects into L-BDI (BDI $\leq$ 8) and H-BDI (BDI $\geq$ 9) groups. This cut-off has no clinical significance, but represented a logical split between two clusters of BDI scores in the current sample. Four participants were dropped from the analysis due to extremely low accuracy. Thus, the final analysis included 27 H-BDI (females n = 22, males n = 5, average BDI = 14, average age = 20) and 25 L-BDI (females n = 20, males n = 5, average BDI = 4, average age = 20) indi-

viduals. Students with a history of depression or anxiety (as reported on the medical questionnaire) were excluded from the study to avoid any unnecessary distortions of the results. It should be noted that for ethical and practical reasons subjects were not excluded based on ethnic and linguistic background. Instead, subjects with less than 60% accuracy on all words types were excluded.

Materials

Words were selected from the Affective Norms for English Words (ANEW) database. A total of 64 positive and 64 negative words were selected based on valence rating, and controlled for length and arousal rating. Words with a valence rating above six were considered for the positive list, and words with a valence rating below four were considered for the negative list. Words that may be associated with fear (such as snake, serpent, spider, etc.) were excluded from consideration. The final lists had an average valence of 7.54 (*SD* = .48) for positive words and 2.55 (*SD* = .66) for negative words. Average word length was 6.55 characters (positive = 6.80, negative = 6.30), while average arousal rating was 5.40 (positive = 5.60, negative = 5.20). T-Tests showed that positive and negative words did not significantly differ with regards to length (p = .68) or arousal (p = .88).

BDI-II. The Beck Depression Inventory-II (BDI-II) is used to measure the severity of depressive symptoms during the preceding two weeks. It consists of 21 multiple choice questions worded such that it reflects phrases often used by depressed individuals to describe their own symptoms. The BDI-II was used to assign participants to either the L-BDI or H-BDI groups.

PANAS-SF. The Positive Affect and Negative Affect Scale –Short Form (PANAS-SF) is a short 10-item questionnaire designed to measure a person's current mood state. This was used to document the current mood state prior to completing the NAP task.

STAI. The State-Trait Anxiety Index (STAI) is a 40-item anxiety scale consisting of trait anxiety and state anxiety subscales. This was used simply to collect independent measures of state and trait anxiety, to further describe the sample.

Design

The computerized NAP task was completed in a quiet, secluded room containing only a cathode ray tube (CRT) monitor and a few tables. The task was controlled from a computer in the next room to avoid unnecessary distractions. The same monitor was used for all subjects, who were positioned approximately 60 cm from the screen.

During the task participants saw a fixation cross appearing for 500 ms, alternating with response slides containing two words. Response slides remained on screen until the subject responded (see Figure 1). Letter dimensions were approximately 1 cm x 1 cm, and words were presented 1 cm apart in the centre of the screen. Each slide contained both a red and a blue word indicating which word was to be ignored and which was to be attended to. The attended colour was counterbalanced such that half of the participants attended to the blue word and half attended to the red word. The colour was independent of valence. Hence, though each slide always contained both a positive and a negative word, the status as either target or distractor was randomly assigned. Each trial was analyzed relative to the valence of the previous target since each slide primed the subsequent slide. Thus, there are two positive prime conditions where the target valence corresponded to the previous target valence and two negative prime conditions where they were incongruent. The order of the four conditions was randomized, with the proviso that each appeared 32 times in each block. Thus, each block contained 128 trials for a total of 384 experimental trials and 16 practice trials. Reaction time and accuracy of responses to the target

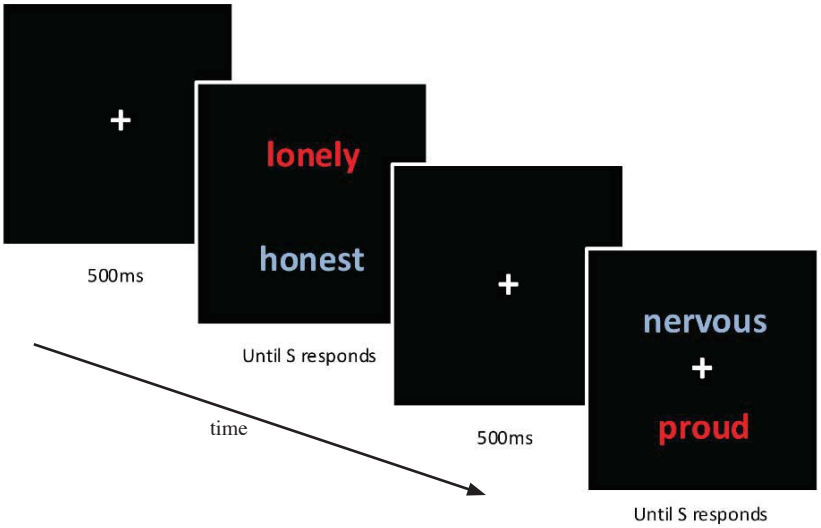

Figure 1. Example of the NAP task. Subjects respond to each set of words. Thus each slide is both a probe for the previous slide as well as a prime for the next. The first slide of each block was discarded from analysis as no priming occurred prior to its presentation.

word were recorded with button click.

Videos were used as a mood induction at the beginning of each block. These were matched for length (~3 min each) and featured light verbal interaction between characters. Thus, the only significant difference was valence. The block order was counterbalanced such that half of the subjects received negative first and half positive first with the neutral mood block always presented in the middle to establish a baseline and remove any residual effects of the initial mood induction. At the beginning and end of each block participants were asked to indicate how they felt on a 7-point scale, with 1 being *very sad*, and 7 being *very happy*. This served as a manipulation check to determine the strength and duration of the mood induction.

Procedure

Informed consent was obtained from all participants, after which they completed the BDI-II, a medical history questionnaire, and the PANAS-SF. They then received a set of instructions for the NAP task. Subjects were not made aware of the priming aspect of the study. Following these instructions, participants completed 16 practice trials, before proceeding with the main experiment. Following the experiment, participants were asked to complete the STAI, before being debriefed.

Statistical Analysis

A mixed-factor analysis of variance (one between-factor and two within-factors) was performed on the self-report ratings of mood state following the positive and negative mood inductions to confirm the effectiveness of the inductions. T-tests were conducted on each of the demographic variables to compare the H-BDI and L-BDI conditions on relevant details.

A second mixed-factor analysis of variance (one between-factor and 3 within-factors) was conducted on the reaction times. This was done to determine the differences in response times to positive and negative words on positive priming vs. negative priming trials, depending on which mood induction preceded the block for the H- and L-BDI groups. Two follow-up within-factor analysis of variance tests

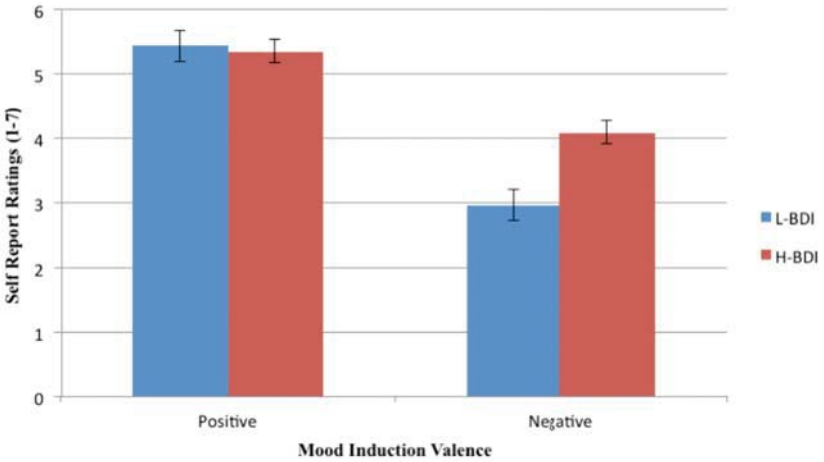

Figure 2. Interaction between mood induction valence and BDI group. The high BDI group reported being much more affected by the negative mood induction than the low BDI group.

were conducted with the between-factor (group) removed to examine the interactions in more detail. T-tests were also conducted comparing the overall Nnap and Pnap, as well as Nnap and Pnap within each mood induction for the H- and L-BDI groups. The probability level for significant effects was set at $\alpha = .05$ for all tests.

Results

Mood Induction Analysis

A global, mixed-factor ANOVA conducted on the self-report rating of mood state following the mood inductions showed main effects for mood induction and time, $F(1, 50) = 63.94$, $p < .001$, and $F(1, 50) = 78.99$, $p < .001$ respectively. Thus, participants responded more strongly to the positive mood induction compared to the negative mood induction (5.4 ± .13 vs. 3.53 ± .17). They also reported higher values immediately following the mood induction compared to after completing the block (4.99 ± .11 vs. 3.94 ± .12). This suggests that people were more willing to endorse the positive mood compared to the negative mood and the effect of the mood induction declined towards the end of the block.

A main effect for BDI group was also evident, $F(1, 50) = 7.21$, $p = .01$. The H-BDI group seemed to be more affected by the mood inductions compared to the L-BDI group (4.72 ± .14 vs. 4.2 ± .14). This was corroborated by a statistically significant Mood Induction x Group interaction, $F(1, 50) = 6.82$, $p = .012$, $p\varepsilon^2 = .12$. Means for the positive mood induction did not differ between the H- and L-BDI groups (5.35 ± .18 vs. 5.44 ± .19). However, the H-BDI group reported being much more affected by the negative mood induction than the L-BDI group (4.09 ± .24 vs. 2.96 ± .19). This difference is illustrated in Figure 2.

RT Analysis

The global mixed-design ANOVA returned main effects of Trial type and Word type, $F(1, 50) = 13.83$, $p < .001$, and $F(1, 50) = 39.44$, $p < .001$, respectively. The former confirmed that the NAP task worked as expected, with participant taking on average 23.54 ms longer to respond to emotional words after attending to a word of the opposite valence on the preceding trial (negative priming) relative to attending to a word of the same valence (positive priming) (874.12 ± 21.26 ms vs. 850.58 ± 19.67 ms). The Word type effect was due to participants being on average 50 ms slower to respond to negative than

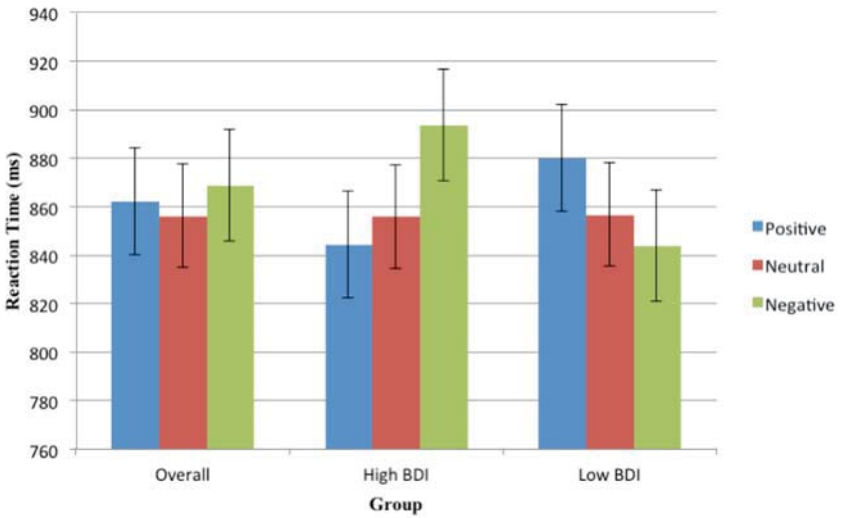

Figure 3. Main effects for mood induction across groups. Note the inverse effects of mood induction in the H-BDI vs. L-BDI groups.

to positive words (887.37 ± 22.34 ms compared to 837.33 ± 18.75 ms) independent of Trial type or Group. The main effects of Mood Induction and Group were not statistically significant, $F(2, 100) = .34$, $p = .71$, and $F(1, 50) = .012$, $p = .91$.

The statistically significant main effects were qualified by a number of interactions. Of particular interest here are the interactions involving BDI group. First, Mood Induction x Group was statistically significant, $F(2, 100) = 4.05$, $p = .020$, $p\varepsilon^2 = .075$. This took the form of a state-dependent emotional interference effect with the H-BDI group being slower while in the sad state relative to the happy state (893.58 ± 31.77 ms vs. 844.29 ± 30.28 ms), while the L-BDI group was slower while in the happy state relative to the sad state (880.11 ± 31.47 ms vs. 843.81 ± 33.01 ms; see Figure 3). The neutral induction yielded very similar responses across groups (856.55 ± 30.77 ms vs. 855.77 ± 29.61 ms). This effect was best captured after computing a "state emotional interference index" (S-EI) as the mean difference between Sad Mood and Happy Mood RT in the H- and L-BDI groups (43.33 ± 121.42 ms vs. -29.87 ± 133.17 ms), $t(51) = 2.04$, $p = .046$.

Neither the 2-way Word type x Group nor the 3-way Trial type x Word type x Group interactions were statistically significant, $F(1, 50) = 3.80$, $p = .057$, $p\varepsilon^2 = .071$, and $F(1, 50) = 3.65$, $p = .062$, $p\varepsilon^2 = .068$ respectively, most likely due to insufficient power. However, there is some evidence suggesting that the word valence asymmetry differed across BDI status and that Negative Priming differed as a function of word valence and BDI Group, supporting the second hypothesis of this study. None of the other interactions approached statistical significance. In particular, the 4-way interaction of Mood Induction x Trial type x Word type x Group was not statistically significant, $F(2, 100) = 1.57$, $p = .21$, $p\varepsilon^2 = .030$.

In order to explain some of the above group interactions, and bring support to our hypotheses, restricted ANOVAs were carried out in each BDI Group separately.

The ANOVA conducted in the H-BDI group showed the main effect of Mood induction was statistically significant, $F(2, 52) = 3.21$, $p = .049$, $p\varepsilon^2 = .11$, although the contrast between Sad and Happy mood

induction did not attain statistical significance as a paired t-test (893.58 ± 36.12 ms vs. 844.29 ± 24.76 ms, $t(26) = .42$, $p = .52$). As expected, the main effects of Trial type and Word type were statistically significant, $F(1, 26) = 7.74$, $p = .010$, $p\varepsilon^2 = .23$ and $F(1, 26) = 28.48$, $p < .001$, $p\varepsilon^2 = .52$, respectively, confirming the presence of a general NAP effect (on average 28.1 ms) and the valence asymmetry with slower RTs to negative words (on average 65.59 ms). More importantly, the interaction Trial type x Word type was statistically significant, $F(1, 26) = 7.04$, $p = .013$, $p\varepsilon^2 = .21$. There was no difference in RT to negative words regardless of the trial type (positive prime = 895.59 ms, negative prime = 899.09 ms). However reaction times were faster on positive prime trials than on negative prime trials (805.44 ± 25.06 ms vs. 858.07 ± 26.21 ms). This interaction effect was better captured after separately calculating a NAP score (difference in RT between negative prime and positive prime trials) for Positive and Negative words as previously done in the literature. Consistent with our hypothesis, H-BDI participants had significantly greater NAP scores for positive than negative words, Pnap: 52.63 ± 12.24 ms; Nnap: 3.50 ± 15.00 ms, $t(27) = 2.65$, $p = .013$. In other words, it is difficult for high depressed trait individuals to switch attention away from previous negative target word in order to respond to a current positive word, resulting in a sizable negative priming effect to positive words. In contrast, there is no cost associated with switching attention away from a previous positive target word to a current negative word, resulting in a negligible NAP for negative words.

Finally, the Mood Induction x Trial type x Word type interaction was not significant, $F(2, 52) = 1.25$, $p = .29$, $p\varepsilon^2 = .046$. While the hypothesis of an influence of mood induction on the size of the NAP effect was not supported, inspection of the data suggest that the effect may be present, however it may be more complex than anticipated. In particular, Figure 4 shows that, consistent with the hypothesis, in the sad mood state a facilitation effect was present for negative words (Nnap = -13.98 ms), while in the positive mood state the Pnap was reduced (Pnap = 40.34 ms). However, the positive mood state also reinstated the Nnap (Nnap = 22.21 ms), which contradicts the hypotheses. Regardless, it appears that there is an effect of mood induction that interacts with BDI group, but this effect was too weak to yield a significant three-way interaction, likely due to lack of power (see Figure 4).

The ANOVA in the L-BDI group showed the main effect of Mood Induction was not significant, $F(2, 48) = 1.34$, $p = .27$, $p\varepsilon^2 = .053$. However, the main effects of Trial type and Word type were significant, $F(1, 24) = 6.66$, $p = .016$ and $F(1, 24) = 12.15$, $p = .002$, respectively, confirming the presence of global negative priming effect (NAP: averaging 16.03 ms) and the faster overall RTs to positive than negative valence words (842.90 ± 28.21 ms vs. 877.41 ± 29.61 ms).

However, unlike the BDI group, there was no significant Trial type x Word type interaction, $F(1, 24) = .015$, $p = .90$. When NAP scores for each emotional valence were calculated, there was no difference between Pnap and Nnap scores (Pnap: 20.01 ± 9.04 ms; Nnap: 18.05 ± 12.49 ms, $t(24) = .12$, $p = .90$, suggesting that the L-BDI group experienced the same degree of negative priming for positive and negative valenced words.

Finally, the 3-way interaction of Mood Induction x Trial type x Word type was not significant, $F(2, 48) = 1.11$, $p = .34$, $p\varepsilon^2 = .044$. However, the relationship between the Nnap and Pnap was inverted in the negative mood induction compared to the positive mood induction. This suggests that mood induction did have an effect on the L-BDI group, however the manipulation may not have been strong enough to detect a difference.

Discussion

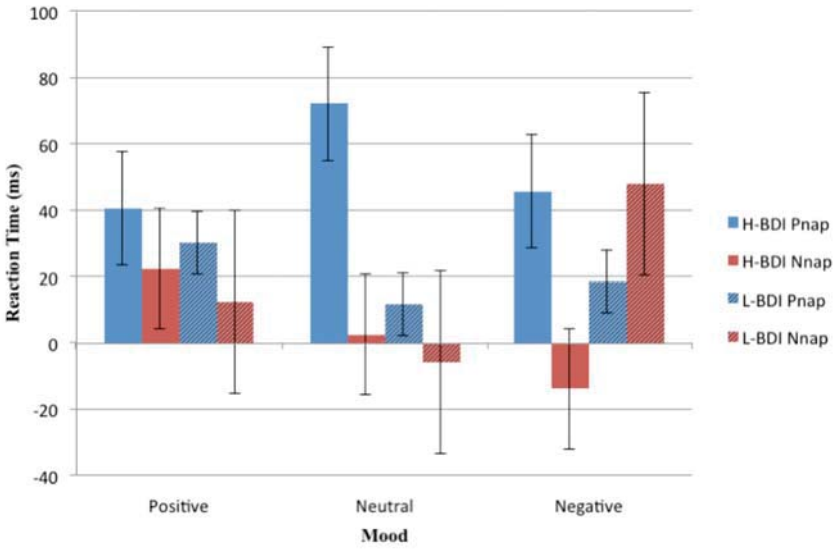

Figure 4. Main effects for NAP across groups. The Nnap effects in the H-BDI group show the expected pattern of results, although these effects are not significant. However, the difference between the Nnap and Pnap in the neutral condition was significant.

The hypothesis that the H-BDI group would show less inhibition for negative words than for positive words was supported. A t-test confirmed that the overall Nnap was significantly reduced compared to the overall Pnap in the H-BDI group. This is in line with previous research that consistently shows decreased inhibition for negative words in high depressive trait individuals (Frings et al., 2007; Joormann, 2006). In particular this replicates the results from the original NAP study by Joormann (2004) suggesting the criterion used to split subjects into the H-BDI and L-BDI groups was appropriate.

No main effect for mood induction was found in the L-BDI group. Thus, the prediction that the positive mood induction would decrease inhibition for all material in the L-BDI group was not supported. A visual inspection of the NAP effects in the L-BDI group confirm that contrary to the results found by Goeleven et al. (2007), the expected reduction in Nnap and Pnap was not present. However, there appeared to be an inversion of the NAP effects between the positive and negative mood inductions (see Figure 4) suggesting the mood induction did have an effect on inhibition, though the manipulation may not have been strong enough to produce statistically significant results. This pattern is consistent with results from Gotlib and Joorman (2010) that showed the inverted Nnap and Pnap in the remitted group compared to the never depressed group. However, care should be taken when comparing clinical and non-clinical populations. The authors suggested that individuals in remission are engaging in compensatory behaviour to negate their negative affect. Similarly, the L-BDI group in the present study may be combatting the negative mood state induced by the negative mood induction. Interestingly, results from the H-BDI condition corroborate this interpretation. The significant interaction between Word type and Group showed that H-BDI individuals were faster to respond to positive words than the L-BDI group, suggesting they may be compensating for the negative bias associated with high depressive trait.

The third hypothesis was not supported. The Group x Word type x Trial type x Mood Induction interaction was not significant, possibly due to a lack of power. However, several results suggest that mood state and depressive traits interact. For example, no main effect for mood induction was found, suggesting the positive and negative mood states affected the H- and L-BDI groups differently. Also, both groups showed mood congruent interference. Thus, the H-BDI group was slowest following a negative mood induction, while the L-BDI group was slowest following a positive mood induction. It appears that trait (either H-BDI or L-BDI) determines what material enters WM resulting in mood congruent rumination. This is consistent with the schema-based model proposed by Gotlib and Joormann (2010) as well as the mood congruent memory bias associated with depression (Joormann & Siemer, 2006). In addition, a significant Mood x Group interaction was evident in the analysis of the mood induction ratings indicating that the H-BDI group responded more strongly to the negative mood induction than the L-BDI group. More importantly however, a visual inspection of the NAP data showed the expected pattern of results was present in the H-BDI group. The Nnap is reduced following a negative mood induction but increased following the positive mood induction, while the Pnap was reduced following the positive induction, compared to the neutral and negative inductions (see Figure 4). This indicates that the positive induction reduces inhibition for positive material, which allowed more positive material to enter WM. With more positive material in WM, there is less space for negative material. This results in more inhibition for negative material, decreasing the amount of negative material entering WM.

Based on these results it was concluded that the modifications to the 3-Factor Model are warranted since the individual's schema and their environment both contribute to the amount of inhibition for valenced material, and consequently what enters WM. However, the lack of a main effect for mood induction in the presence of the robust effect for group does indicate that mood state is the less important factor in this interaction. The manipulation may simply not have been strong enough to produce noticeable effects. Research shows that previously depressed individuals are more susceptible to the effects of a mood induction (Harkness et al., 2010). Thus, with a weak induction it is no surprise that only the H-BDI group was affected. Consistent with this explanation, only the H-BDI group showed a main effect for mood induction. The H-BDI group also rated themselves as more sad after the negative mood induction compared to the L-BDI group. Therefore, given that the pattern of NAP effects is consistent with the proposed interaction, using a stronger manipulation such as autobiographical memories might produce stronger results.

This interaction may explain the efficacy of various treatment options. Many therapeutic approaches centre on increasing the amount of positive material a person is exposed to (i.e., behavioural activation, laughter therapy). For example, behavioural activation (BA) encourages individuals to engage in pleasurable activities such as the exercise of socializing (Hundt, Mignogna, Underhill, & Cully, 2013). BA focuses less on the internal causes of depression (Turner & Leach, 2012), and instead takes a pragmatic approach, identifying specific avoidance behaviours and providing specific solutions (Veale, 2008). Oftentimes these solutions revolve around changing behavioural patterns, and thus changing the contextual factors influencing the individual's affective state (Turner & Leach, 2012). This requires a permanent change in lifestyle, suggesting that it acts as a constant positive mood induction. BA is almost as effective as cognitive behavioural therapy (CBT) and continued medication at a 2-year follow-up (Dobson

et al., 2008) and acts on similar regions in the prefrontal cortex as CBT (Dichter, Felder, & Smoski, 2010). However, as with pharmacological treatment regimens, BA is only effective as long as a person continues with the treatment (Dobson et al., 2008), and it is only with adjunct therapies (such as CBT) that an individual truly enjoy the long-term benefits of treatment (Dobson et al., 2008; Huijbers et al., 2012; Padesky & Greenberger, 1995; Siddique, Chung, Brown, & Miranda, 2012). The current study suggests that this mood induction component may be crucial to the treatment of depression, since it temporarily relieves the individual from the negative ruminatory cycle.

CBT takes this one step further and aims to permanently change a person's maladaptive thoughts and actions by training them in more adaptive ways that they can then employ outside the therapeutic setting (Padesky & Greenberger, 1995) In addition to engaging in more positive activities (Corsini, 1984), clients are guided to recognize the maladaptive nature, origin, and consequence of current patterns (Phares, 1984; Richardson & Marshal, 2012). The behavioural component acts as a positive mood induction, providing a window wherein cognitive treatment can be effective in producing long-term effects. This dual action immediately reduces depressive symptoms, as well as protects against relapse (Feng et al., 2012). Thus, the efficacy of CBT lies in the fact that it targets both the negative schema and the negative mood that is conflated in depressed individuals.

Limitations with the study design suggest a degree of caution should be taken when considering these conclusions. Firstly, to avoid conflating currently and previously depressed populations with depressed traits, the low cut-off for the H-BDI group (≥ 9) was used. However this divide reflects depressive tendencies, and not depression as a disorder. For that reason, without knowing how these high trait individuals relate to currently and previously depressed persons, the generalizing of results to the clinical population may not be warranted. It follows from Gotlib and Joormann's (2010) model that there is a positive correlation between the severity of depressive symptoms (particularly cognitive ones) and the negativity of their schema. This would obviously distort incoming information, and thus distort how the individual perceives the mood induction. Future research should examine this interaction in clinical populations to determine to what extent these results are generalizable.

Secondly, the mood inductions used in this study also prevent generalizing the results to the therapeutic situation. The use of videos may not provide a strong enough induction and is not consistent with the types of behavioural changes required of people in therapy. Exercise and social interaction are both much stronger manipulations than watching a brief video clip. Thus, in order to fully determine if the behavioural or pharmaceutical components of therapy do act as a positive mood induction and produce the same effects as is suggested by this study, other mood induction paradigms should be studied. Specifically, paradigms that correspond to therapeutic approaches (e.g., laughter, exercise, social activities, etc.) need to be examined.

Lastly, there is also a logical inconsistency in the original paradigm as developed by Joormann (2004). It requires subtracting reaction times on control trials (which are in fact positive priming trials) from reaction time on negative prime trials. This could lead to an unnatural inflation of the NAP effect. Also, since there are always a positive and a negative word on the screen, the effects of positive and negative words are never truly independent of one another. Thus, it is impossible to fully distinguish between inhibition and priming effects for positive and negative words. The use of neutral words has been adopted by several researchers (e.g., Got-

lib and Joorman, 2010), although for simplicity they were not included in the present study. Future research should include neutral words to completely differentiate between inhibitory effects for positive and negative words.

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