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Impaired distractor inhibition on a selective attention task in unmedicated, depressed subjects

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ABSTRACT

Background. Impaired distractor inhibition may contribute to the selective attention deficits observed in depressed patients, but studies to date have not tested the distractor inhibition theory against the possibility that processes such as transient memory review processes may account for the observed deficits. A negative priming paradigm can dissociate inhibition from such a potentially confounding process called object review. The negative priming task also isolates features of the distractor such as colour and location for independent examination.

Method. A computerized negative priming task was used in which colour, identification and location features of a stimulus and distractor were systematically manipulated across successive prime and probe trials. Thirty-two unmedicated subjects with DSM-IV diagnoses of non-psychotic unipolar depression were compared with 32 age, sex and IQ matched controls.

Results. Depressed subjects had reduced levels of negative priming for conditions where the colour feature of the stimulus was repeated across prime and probe trials but not when identity or location was the repeated feature. When both the colour and location feature were the repeated feature across trials, facilitation in response was apparent.

Conclusions. The pattern of results supports studies that found reduced distractor inhibition in depressed subjects, and suggests that object review is intact in these subjects. Greater impairment in negative priming for colour *versus* location suggests that subjects may have greater impairment in the visual stream associated with processing colour features.

INTRODUCTION

The *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, (American Psychiatric Association, 1994), cites a diminished ability to think or concentrate as a diagnostic feature of a major depressive episode. The nature of attention deficits in patients with depressive disorder has been relatively understudied, however, despite reviews highlighting the need for further data (Mialet *et al*. 1996). Studies that are available of selective attention have suggested that an inability to inhibit distracting stimuli contributes to the observed deficits (Lemelin *et al*. 1996) but alternate accounts based on resource allocation models have also been proposed (Hasher & Zacks, 1979). This study employs a negative priming paradigm to investigate further the role of impaired distractor inhibition in depression.

Negative priming (NP) occurs when individuals selectively attend to objects while ignoring distractors. It is demonstrated as follows. Two pictures are presented in the prime component of a trial; one is a red chair, which is named (the target), the other is a green dog, which is ignored (the distractor). A red dog is next presented as the target object in the probe component of the trial, and response to it is

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slowed; this is thought to reflect inhibition of this stimulus on trial N, which must be overcome in order to respond to the stimulus on trial N + 1 (Tipper, 1985; Tipper & Cranston, 1985). An inability to inhibit distractors should therefore be associated with reduced NP, and if depressed subjects have deficits in distractor inhibition, they should have reduced levels of NP. There is evidence from the Stroop colour word task (SCWT) performance in depressed subjects to support this (Benoit *et al*. 1992).

Although NP has been useful in studying attention impairment in major depression, (Benoit *et al*. 1992; Lemelin *et al*. 1996), it has become apparent that a confound exists in many studies that makes it impossible to know whether reduced NP is, in fact, the result of reduced inhibitory capacity. Park & Kanwisher (1994) pointed out that NP effects may also be the result of a mismatch in object review, a process that is roughly analogous to brief working memory, and which was described by Kahneman *et al*. (1992). Objects review is an aspect of perceptual processing that links current and past information to produce a coherent mental picture. Negative priming effects (i.e. slower responses to previously ignored stimuli) could reflect either inhibition or perceptual mismatches that are discovered by the object review process. In the NP paradigm, the perceptual mismatch would occur when there are rapid successive demands to both 'respond to' and 'ignore' a feature of the stimulus across the prime and probe components of a trial. This mismatch would increase processing time and produce a NP effect that is unrelated to inhibition. Thus, in a clinical population where reduced NP has been interpreted as evidence for reduced inhibition, it is important to differentiate the contribution of inhibition and the object review process before concluding that one of these processes contributed to the observed NP deficit (see Kane *et al*. 1997).

Milliken *et al*. (1994) devised a NP task that can isolate the relative contributions of inhibition and the object review process to NP. By systematically manipulating the location, colour and identity of a letter stimulus, they showed that both inhibition and the object review process can contribute to NP. In this paradigm subjects select the target letter by matching its colour with a colour cue presented on a computer screen. They respond by pointing to the location of the correct letter. Thus, colour is an important selection variable while location is an important response variable. Key NP trials are those in which the location, identity and colour of a stimulus are held constant. On such trials, the effect of inhibition and object review oppose each other, and Milliken *et al*. (1994) demonstrated that suppression of object review leads to large inhibitory effects in this condition. If object review is not suppressed, the review process in this condition facilitates the response, and the opposing inhibition and object review result in NP values near zero. Thus, large levels of NP in this condition indicate suppressed or reduced object review, while facilitatory effects in this condition imply intact object review but absent or reduced inhibitory effects. Finally, identity in this task is an irrelevant variable; that is, neither selecting the appropriate target nor making the correct response depend on processing of the identity feature. Assessing the identity feature for the presence of NP, however, allows us to determine whether depressed subjects have a problem with inhibition such that the inhibitory mechanism is present but inefficient, misapplying inhibition to features of a stimulus that are irrelevant for task completion. Control subjects have virtually no NP in the identity trials on this paradigm; if depressed subjects have an inability to direct inhibition towards relevant features of the distractor, they would have a greater amount of NP on identity trials.

In addition to sorting out whether inhibitory or object review deficits account for reduced NP in depressed subjects, being able to study stimulus features in isolation is important because of increasing evidence for specialization of function between the two major visual pathways in the visual cortex (Mishkin *et al*. 1983; Ungerleider & Haxby, 1994). The ventral pathway codes features of objects such as colour, orientation, and texture and culminates in identification of objects in the inferior temporal cortex. The dorsal pathway codes representation of space and motion and may be important in the spatial control of actions (Milner & Goodale, 1995). Isolating the features of the distractor so that colour and location are independently manipulated allowed us to assess the extent to which impaired responding might be specific to

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either the colour feature (ventral stream) or spatial feature (dorsal pathway) of the distractor.

METHOD

Subjects

Thirty-two depressed subjects met criteria for major depressive disorder without psychotic features, as measured by the Structured Clinical Interview for DSM-IV (SCID; Spitzer & Williams, 1994), and had Hamilton Depression Rating Scores (Ham-D) $\geqslant 16$ on the 21-item version (Hamilton, 1967). Depressed subjects were free of psychotropic medication for a minimum of 2 weeks, except for fluoxetine, which could not have been consumed within the past 5 weeks. Subjects were permitted short acting benzodiazepines, but were required to refrain from taking the medication within 24 hours of testing.

Control subjects ($N = 32$) were healthy volunteers recruited, according to hospital guidelines, from the community and hospital staff by advertisements. Control subjects were selected to be age, sex, and IQ matched (as measured by the National Adult Reading Test, NART: Nelson, 1982) with depressed subjects. Control subjects had no history of major psychiatric illness as measured by the SCID, and had a score < 7 on the Ham-D administered on the day of testing.

Depressed and control subjects were excluded from testing if they met any of the following criteria: (*i*) substance related disorder within the past 6 months as measured by the SCID; (*ii*) concurrent social phobia, panic disorder or obsessive–compulsive disorder as measured by the SCID; (*iii*) history of manic or hypomanic episodes as measured by the SCID; (*iv*) persistent specific reading disorder, reflected by the

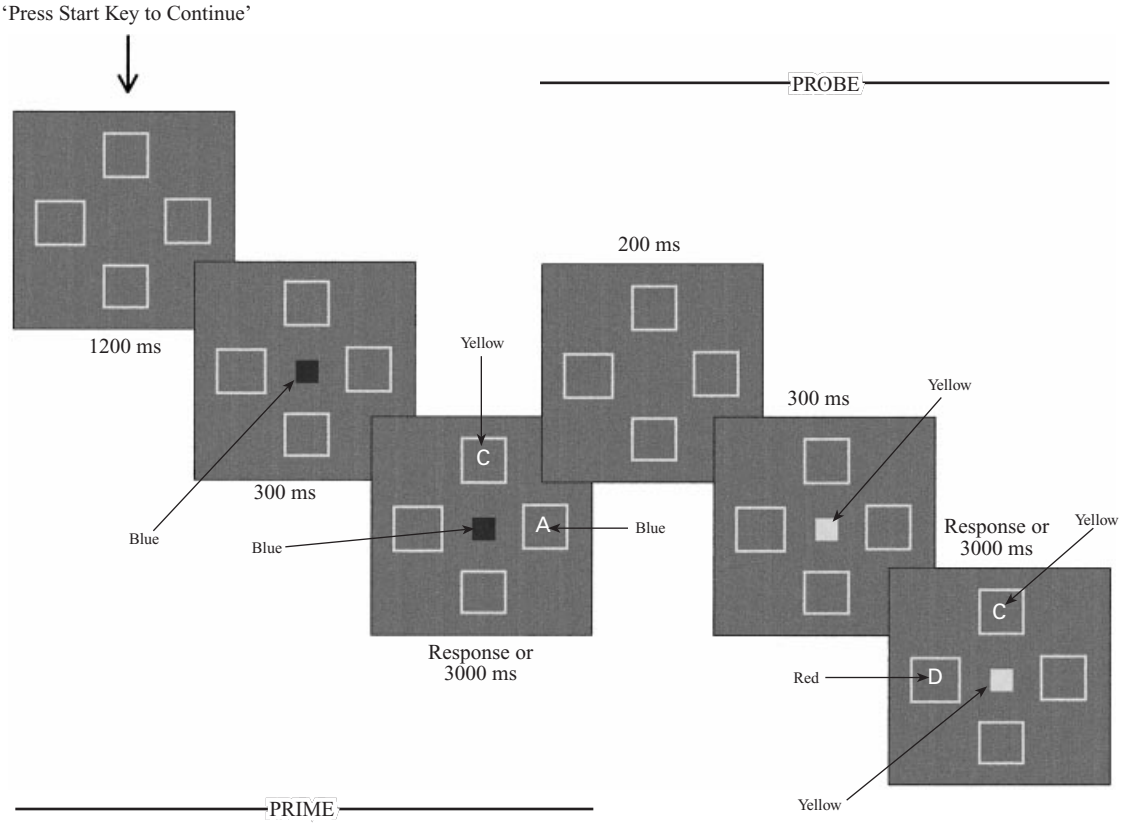


FIG. 1. A typical NP trial type consists of the initial prime display, followed by a probe component. This is an example of a LIC trial type in which all features of the distractor stimulus in the prime (the yellow colour, letter C and location above the cue) are repeated as the target stimulus in the probe.

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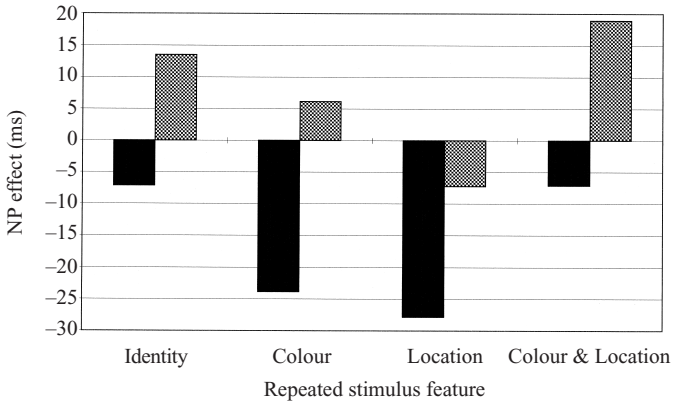


FIG. 2. Negative priming effects when the identity, colour and location features of the stimuli were repeated as the distractor in the prime component of a trial and the target in the probe component of the trial. NP effects were calculated by subtracting response times on each NP trial type from the response time on control trials (in which no features of the prime distractor are repeated as the probe target), so that large negative values reflect large NP effects. Control subjects (■) show the expected NP effects in the colour and location trial types, but these effects are greatly reduced for depressed subjects (▨).

Wide Range Achievement Test (Jastak & Wilkinson, 1984); (*v*) concurrent major medical illness; (*vi*) consumption of alcohol within 48 hours; (*vii*) electroconvulsive therapy within the past 2 years. All subjects provided written, informed consent.

Diagnostic and symptom assessment

The SCID was administered by a trained rater to each subject for diagnosis and assessment of co-morbidity. Subjects were administered the Ham-D on the day of cognitive testing. Medical chart records, family members, treating clinicians and life charts were also consulted when available to determine the most accurate diagnoses and demographic data.

Cognitive assessment

Eligible subjects completed the computer task that required them to attend to a coloured cue appearing on a computer monitor and point with a joystick to the location of a letter of the same colour as the cue. The features of the stimuli varied in terms of the colour (drawn in blue, yellow, purple or green), location (whether it appeared to the right or left, above or below the colour cue) or identity (i.e. A, B, C, D) of the target to be selected, but the task of pointing to the letter whose colour matched the square cue was the same across all trials. Trials began with a prompt instructing subjects to 'Press start key to continue'. The prime component of a trial began after subjects pressed the start key; first, four boxes appeared on the screen indicating the position in which the target and distractors would later appear (see Fig. 1). A coloured square next appeared in the centre of the screen for 300 ms; this cued the subject to the colour of letter to be selected. A letter of the same colour as the square (the target) and a letter of a different colour (the distractor) then appeared on the screen until subjects moved the joystick in the direction of the letter whose colour matched the square, completing the prime component of the trial. The probe component followed immediately and included a similar presentation of coloured cue followed by target and distractor letters. If subjects did not respond to either the prime or probe letters within 3000 ms, the trial proceeded.

There were approximately 160 trials as trials proceeded until subjects made 20 correct responses in each of the following conditions: a control condition (Ctrl) in which the prime and probe target letter shared no features (different colour, identity and location of presentation); the probe target occured in the same location (L) as the distractor in the prime; the probe target had the same colour (C) as the distractor in the prime; the probe target had the same identity (I) as the prime distractor; the probe target shared two features with the prime distractor (LI, LC, or IC); the probe target was identical to the prime distractor (LIC). Number of errors and reaction times (RTs) were recorded automatically.

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Statistical analysis

Demographic characteristics of the patient and control groups were compared using independent *t* tests. The 20 RTs obtained for each subject in each trial type (Ctrl, L, LI, C, CI, I, LC, LIC) were collapsed and represented by median RTs (to minimize the effect of erroneous long RTs). Because there may be differences in strategy between individuals in terms of a speed – accuracy trade off (increase the number of errors to decrease overall response time), incorrect responses were excluded from the calculation of overall RTs for each trial type within subject. Differences in baseline RTs were assessed by comparing depressed and control subjects on the Ctrl trial types. Finally, planned comparisons were utilized to compare performance of depressed and control subjects in key NP trial types.

RESULTS

Characteristics of the sample

Both groups had 11 males (34·4%). Control subjects were 35·7 years of age on average (range 20–53), while control subjects were 36·3 (range 18–56), a non-significant difference. The NART-estimated full scale IQs were 116·3 (S.D. = 7·4) for non-depressed subjects and 115·1 (S.D. = 10·1) for depressed subjects, which were not different. The 21 item Ham-D scores were 1·45 (S.D. = 2·0) for control subjects and 21·4 (S.D. = 4·1) for depressed subjects, which were significantly different.

Performance on tests of cognitive processing

Reaction times

Table 1 demonstrates that depressed subjects were slower to respond on the baseline Ctrl trial types ($t = 3{\cdot}9$, $P < 0{\cdot}001$) and in the other trial types as well. Slower response times on attention tasks have been well documented in depressed patients and may reflect either a motor slowing in depression or a slowing of central processing. There is evidence that depression contributes to an increase in perceptuomotor response time that is independent of motor control (Cooper *et al*. 1994), but the factors that contribute to this slowed reaction time remain to be elucidated. Given that our patients were unmedicated, and no subjects had ingested alcohol or other substances for at least 24 h, this slowing is unlikely to be the result of psychotropic drug effects.

It is important to note that differences in NP between the depressed and control group cannot be accounted for by the overall increased RTs in the depressed group. The NP effect is calculated by taking a difference score between RTs in the Ctrl and NP trial types, where the effect of NP is to slow responding relative to the control condition. When NP is reduced or absent, response time on NP trials becomes almost as fast as responding on the control trials. The overall slowing in absolute RTs in depressed subjects therefore, cannot contribute to the relatively faster response that occurs when NP is impaired.

NP trials

We first compared NP scores on I trial types to determine whether there was evidence for inefficient application of inhibition to irrelevant features of the distractor stimulus (see Table 1). There was no difference between control and depressed subjects in NP on these trial types ($t = 1{\cdot}3$; $P = 0{\cdot}19$). Furthermore, there was no evidence of an interaction with identity and the other stimulus features. Identity was an irrelevant feature of the stimulus as it controlled neither selection of the target nor direction of response, and it was treated as an irrelevant stimulus equally by both depressed and control subjects. For further analyses we therefore averaged subjects' scores in L and LI trial types, C and CI trial types, and LC and LIC trial types to obtain composite location, colour and location/colour scores (see Milliken *et al*. 1994).

The most notable finding was that there were significant differences between groups in trials where the selection variable colour was repeated across prime and probe trials ($t = 3{\cdot}0$, $P = 0{\cdot}004$). Depressed subjects showed no evidence of inhibition on these trial types.

There was also a trend for a difference in the location trial types ($t = -1{\cdot}99$, $P = 0{\cdot}053$). Thus, when the location feature of the stimulus was repeated across trials, there were consistent trends for reduced NP in the depressed group, but these effects were less pronounced than when colour was the manipulated variable.

Finally, there was a difference between groups

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Table 1. *Reaction times (ms) for depressed and control subjects on each NP trial type. NP values for depressed and controls subjects on each trial type calculated as Ctrl-trial type for each combination of stimulus features*

Trial type	Control RTs Mean (S.E.M.)	Control NP Mean (S.E.M.)	Depressed RT Mean (S.E.M.)	Depressed NP (Mean (S.E.M.)
Ctrl	454·9 (11·4)		556·2 (23·6)	
I	455·7 (10·9)	−0·8 (4·6)	542·7 (20·1)	−13·5 (9·6)
L	486·7 (12·0)	−31·8 (4·2)	561·8 (19·1)	−5·6 (10·4)
LI	479·2 (11·1)	−24·3 (6·3)	565·1 (19·4)	−8·9 (10·3)
C	476·0 (10·6)	−21·1 (5·0)	548·0 (19·4)	−8·2 (8·4)
CI	481·7 (12·0)	−26·8 (5·3)	552·0 (17·0)	−4·2 (5·3)
LC	468·0 (12·9)	−13·1 (7·3)	534·5 (17·9)	+22·1 (10·3)
LIC	456·3 (12·9)	−1·4 (6·9)	540·0 (22·8)	+16·2 (14·0)

on key trials where location and colour were both manipulated ($t = 2{\cdot}1$, $P = 0{\cdot}038$). Of note, the overall pattern of NP observed in the control subjects is a clear replication of that obtained by Milliken *et al.* (1994).

DISCUSSION

A major finding of the study was that depressed patients had reduced ability to inhibit features of a distractor, supporting previous studies with similar conclusions. The data are not consistent with an explanation that relies on deficits in an object review process. In studies of non-psychiatric subjects (Milliken *et al.* 1994; Tipper *et al.* 1995), experimentally suppressing the review process resulted in large NP effects in the LC and LIC conditions that were interpreted as the effect of inhibition alone. If the review process was impaired in depressed subjects, response in the LC or LIC trial types should mimic responding when object review is suppressed in control subjects. In our depressed subjects the opposite pattern is apparent: response on these trial types is facilitated, suggesting that the object review process is intact but the inhibitory process is reduced. As object review may resemble a form of brief memory, these results are consistent with other recent findings that working memory is intact in depressed subjects (Purcell *et al.* 1997, 1998; Zakzanis *et al.* 1999).

The NP deficit was most apparent on trials in which the colour feature of the stimulus was manipulated. Initial studies with this NP paradigm using colour as a selection cue and location as a response cue suggested that the NP in conditions where the selection cue was manipulated was secondary to the object review process (Milliken *et al.* 1994). However, a recent study (Simone *et al.* 1998) reported inhibition to a colour selection cue using a similar paradigm, and a number of previous studies have suggested that inhibition is not limited to, or dependent upon, response-associated features of the target (Tipper *et al.* 1988). Given this, and the apparent reduction of inhibition in the LC and LIC conditions, the most parsimonious explanation for the reduced NP on colour trial types is that inhibition is impaired on these trials as well. Furthermore, the depressed subjects' pattern of reduced NP on trials where location of the stimulus is manipulated also suggests a decreased ability to inhibit these features of the distractor.

Although depressed subjects had reduced NP when either colour or location were manipulated, the greater effect in the colour condition relative to the spatial location condition suggests that depressed subjects may be particularly poor at suppressing features processed by the ventral visual processing stream that terminates in the inferior temporal cortex. There is controversy regrading the validity of this two visual streams model (Schroeder, 1995) and the extent to which it may apply to distractor stimuli as well as targets, but studies have shown a dissociation between distractor features on tasks of distractor inhibition. Connelly & Hasher (1993) demonstrated that normal elderly subjects are efficient when the spatial features of the object are important but are less able to inhibit other features of a stimulus. The relatively greater deficit in our depressed subjects when the colour feature of a stimulus was manipulated suggests that information processed by structures related to the ventral processing stream may be most affected by depression. An increasing number of

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neuropsychological studies have found that depressed subjects are impaired on tasks thought to be dependent on structures of the temporal lobe (Denny & Hunt, 1992; Bazin *et al*. 1994), and there is currently significant interest in the relation of temporal lobe structures to the pathophysiology of depression (see Bremmer, 1999 for review). Speculatively, the finding that depressed subjects showed the greatest differences on the colour feature of the task may be further evidence that neural pathways involving the temporal lobe are differentially, although not exclusively, vulnerable in depression.

In a comprehensive review, Mialet and colleagues suggested that the two major methodological problems in studies of attention in depressed patients were a failure to define an appropriate population for study and a failure to assess specific components of attention using theoretically strong tests that allow for *a priori* hypotheses (Mialet *et al*. 1996). This study attended to both these details. The population was rigidly selected to include only unmedicated unipolar depressed patients with no known medical or psychiatric co-morbidity. The control group was rigorously assessed for the absence of psychiatric symptomatology, and potential effects of age, sex and pre-morbid IQ were controlled. The NP task allowed for systematic examination of specific distractor features and enabled us to test competing accounts of why NP is impaired in depression. Studies using other tasks of visual attention are needed to confirm this finding, but if this deficit is in fact more specific to stimuli processed by the ventral visual stream, such data would be consistent with current studies emphasizing the temporal lobe changes in unipolar depression.

The limitations of the study are that the cross sectional nature of the assessment does not allow for any information regarding the extent to which these changes in NP are transient and related to mood state or a reflection of a more enduring process associated with long-term neuronal changes that may occur with depression. Furthermore, while we have some evidence that subjects with psychotic disorders do not have the same pattern of impairment on such a NP task, no studies have directly compared across patients with various primary psychiatric diagnoses to confirm the extent to which these findings are specific for depression.

Changes in cognitive processes such as attention and memory are recognized as core features of major depression. These symptoms remain understudied, however, and consequently little is done clinically to assess or treat these symptoms, although they are distressing to patients. Further studies using paradigms from cognitive neuroscience may lead to a better understanding of the cognitive deficits associated with depression and ultimately to an understanding of how the pathophysiological processes leading to depression also result in these cognitive changes.

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