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Impaired distractor inhibition in patients with schizophrenia on a negative priming task

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ABSTRACT

Background. Numerous studies have suggested, via the interpretation of negative priming effects, that subjects with schizophrenia are less able than controls to inhibit irrelevant distracting information. Further issues concerning impairment in inhibitory processes are investigated here. First, recent research has revealed that negative priming (NP) effects can be caused by different processes, distractor inhibition or perceptual review. Therefore, conclusions concerning reduced inhibition in patients with schizophrenia are not possible from previous NP research. Secondly, previous NP studies have required subjects to identify some feature of the target. This is the first study to examine NP that uses a spatial task in patients with schizophrenia.

Method. Twenty-eight subjects with schizophrenia and 28 age and sex matched non-psychiatric control subjects completed a computerized NP task that eliminated the possible contribution of perceptual review.

Results. Subjects with schizophrenia had reduced levels of NP compared to control subjects on this spatial NP task ($t = 2{\cdot}46$, $P < 0{\cdot}02$). Current age, positive, negative or total PANNS scores did not correlate with negative priming scores, but *post hoc* analyses revealed that clozapine-treated patients had significantly greater levels of negative priming than patients receiving typical antipsychotic medications.

Conclusions. The present experiment eliminated the contribution of perceptual review to negative priming and demonstrated that when a pure measure of inhibition is taken on a localization task, patients with schizophrenia were less able to inhibit irrelevant distracting stimuli. The fact that NP was reduced in a spatial task suggested a more diffuse reduction in inhibition than previous studies that examined only identification-based responses.

INTRODUCTION

The hypothesis that selective attention deficits underlie some symptoms of schizophrenia has a long history. For example, 'The selection which attention exercises over normal sensory impressions may be reduced to zero, so that almost everything that meets the senses is registered' (Bleuler, 1911). More recently, the overload that is suggested by Bleuler's description has been described in information processing terms, primarily in the form of attention filtering mechanisms. For example, McGhie & Chapman (1961) described 'a breakdown in this selective-inhibitory function of attention' that leads to an over-inclusion of irrelevant information and then, to secondary disturbances in perception and thought. Similarly, Frith (1979) described a 'defect in the mechanism that limits and controls the contents of consciousness', which has been proposed as the basis for the positive symptoms of schizophrenia.

These observations and theoretical ideas are supported by experimental evidence; it is clear that people with schizophrenia perform poorly

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on attention tasks compared to healthy controls. Such attention deficits in schizophrenia are mainly characterized by an inability to sustain attention and to ignore irrelevant stimuli. Similar patterns are observed when high schizotype subjects are compared with low schizotypes. Thus, people with schizophrenia and high schizotypes show poorer performance than healthy controls and low schizotype subjects on a range of tasks where inhibitory control mechanisms are critical. For example, vigilance (CPT) tasks (Cornblatt *et al.* 1989; Lenzenweger *et al.* 1992; Obiols *et al.* 1993), the Wisconsin Card Sorting Task (Braff *et al.* 1991; Lenzenweger & Korfine, 1994), latent inhibition (Baruch *et al.* 1988; De la Casa *et al.* 1993), the inhibition of inappropriate meanings of ambiguous words (Chapman *et al.* 1964; Bullen & Hemsley, 1984), cross-modal set switching tasks (Spring, 1980; Wilkins & Venables, 1992) and on the simple reaction time (RT) preparatory interval cross-over (Rosenbaum *et al.* 1988).

One of the best techniques for observing inhibitory processes in attention is known as negative priming (NP) (Neill, 1977; Tipper, 1985). Negative priming is based on the following logic. During selection of a target stimulus a competing distractor can be encoded in parallel. An important mechanism for selecting the target for conscious awareness and action is to inhibit the internal representations of the competing distractor. In human subjects this inhibition cannot be observed directly, but can be studied via priming techniques. Therefore, if it is the case that the internal representations of the distractor are associated with inhibition during selection of the target, then processing of a subsequent stimulus requiring these inhibited representations will be impaired.

Consider the following example from a typical negative priming task (e.g. Tipper, 1985): two partially superimposed pictures are presented in different colours in a prime display. The subject is instructed to name red pictures and to ignore green pictures. On this initial prime trial, a chair is shown in green and ignored, and a dog is shown in red and named. To select the red dog, it is hypothesized that the competing representations of chair are inhibited. On the subsequent probe trial, the red target is the previously ignored drawing of a chair and the green distractor is a novel drawing of a guitar. Under normal conditions, it takes longer to name the red drawing of the chair in the probe display when it has just been ignored on the previous prime trial, than when the drawing has not been seen before. This slowing of response after a stimulus has recently been ignored has been termed the negative priming effect (Tipper, 1985).

Importantly for our current purposes, there is evidence that patients with schizophrenia show smaller NP effects than normal control subjects (Beech *et al.* 1989, 1991; LaPlante *et al.* 1992; Williams, 1995, 1996; Salo *et al.* 1996). Thus, reduced negative priming effects in schizophrenia have been interpreted as evidence for reduced cognitive inhibition in this group. This is consistent with the predominant view of attention in schizophrenia, namely, that patients with schizophrenia experience a breakdown in inhibitory control processes and are unable to inhibit distracting information.

Perceptual review or inhibition?

A problem for the above interpretation is that NP does not unambiguously reflect the inhibition of distractors. NP effects might also reflect more complex processing that could result from the discovery of perceptual differences between the prime distractor and the probe target (see Tipper, 2001 for a review of this issue). For example, Park & Kanwisher (1994) noted that there are perceptual differences between the ignored prime and subsequent probe. Thus, in the above example, the ignored picture of a chair in the prime display is green, whereas the same picture in the probe is red.

Based on ideas developed by Kahneman *et al.* (1992) it was proposed that perceptual processing was slowed by this colour mismatch. Kahneman *et al.* (1992) proposed a perceptual review process that by comparing current and immediately preceding stimuli would discover such distractor-target differences. They suggested that an automatic review of very recent perceptual events is a part of current perceptual processing that is critical for the integration of successive perceptual events. This process accesses features of objects that are no longer in view and links current and past information together to produce a coherent picture of the world. If there are mismatches, such as an object being associated with two colours (e.g. green and red) processing of the new object is slowed down.

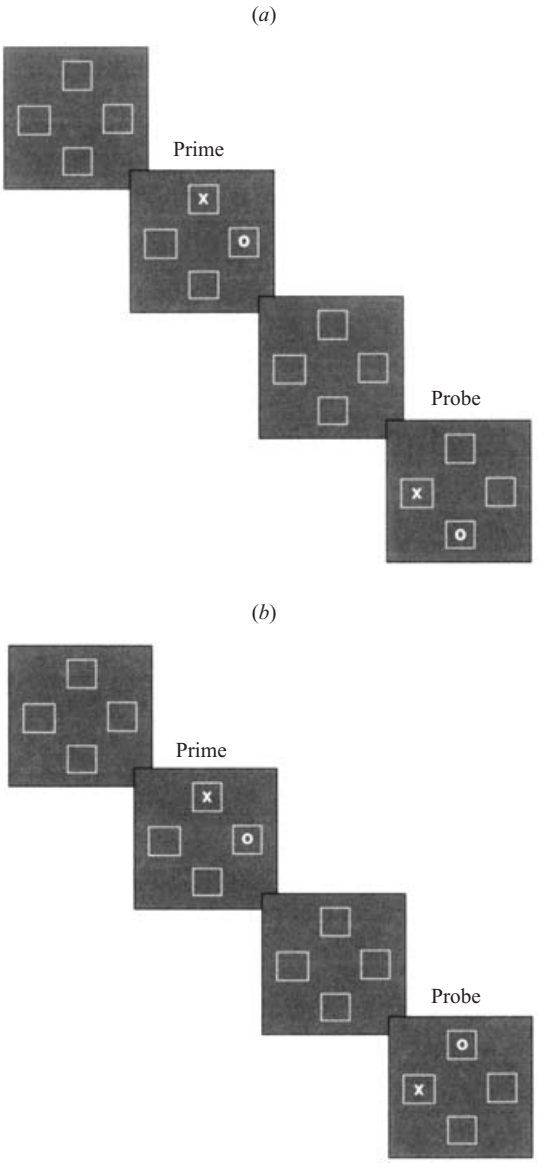

FIG. 1. Illustration of the experimental displays used in previous studies of spatial negative priming. The target was defined as the O and the distractor as the X. On control trials (Panel *a*) the probe target and distractor appeared in previously empty loci. On ignored repetition trials (Panel *b*), the probe target appeared in the same location as the prime distractor. Note that the stimuli appearing in this location change identity from X to O. It is this switch in identity that is assumed to slow response to the probe, according to Park & Kanwisher (1994).

A similar example of such mismatching is shown in Fig. 1. In this task target (O) and distractor (X) can be presented in one of four locations (see Tipper *et al.* 1990; Tipper & McLaren, 1991). The subject's task is simply to report the location of the target 'O' with a keypress or joystick motion, while ignoring the distractor. Based on the logic outlined above, the distractor stimulus (X) is inhibited as a means of selecting the target. Therefore, processing of a subsequent stimulus that appears in the previously inhibited location will be impaired (ignored repetition condition) as compared to a probe presented in a previously empty location (control condition). However, a perceptual mismatch account explains this slower responding by the change in stimulus identity. In the prime the object is an X, but at the same location in the probe display the object is now an O. This mismatch would again impair processing.

Therefore, because this mismatch problem exists in all previous studies examining negative priming in schizophrenia, it is not possible to conclude that people with schizophrenia have reduced inhibitory control. Consequently, reduced NP in patients with schizophrenia may not reflect reduced inhibition, but instead may be due to a reduced ability to review recent perceptual inputs. This possibility is particularly relevant to one theory of schizophrenia (Hemsley, 1987, 1994) that proposes that a perceptual integration deficit may underlie some of the symptoms that patients experience. The experimental technique described below (Milliken *et al.* 1994; Tipper *et al.* 1995) is designed to prevent review processes contributing to NP. This technique may reveal whether it is a failure of inhibition, rather than perceptual review, that underlies reduced NP in subjects with schizophrenia.

New methodologies

As discussed above, in the O–X task (Tipper *et al.* 1990) the subject's task is to 'point to' the location of the 'O' target while ignoring the 'X' distractor. This procedure elicits NP when the probe target occurs in the location that was occupied by the distractor in the preceding prime display. The prolonged latencies have been attributed to inhibition associated with the location of the ignored object, but could just as easily be ascribed to perceptual mismatches (Park & Kanwisher, 1994).

In order to avoid this potential confound between inhibition and perceptual mismatching as causes of negative priming, a localization task in which the prime distractor and the probe target are perceptually identical has been developed

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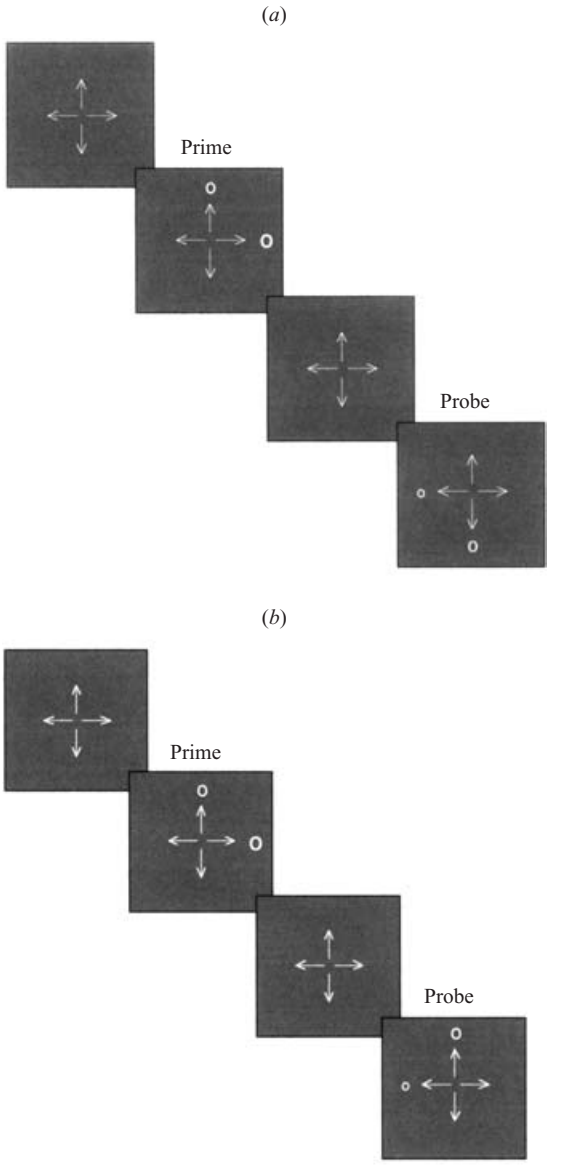

FIG. 2. Illustration of the experimental displays. The target was defined as the larger of two circles. The prime target was always size 3 (with size 2 distractor) and the probe target was always size 2 (with size 1 distractor). On control trials (Panel *a*) the probe target and distractor appeared in previously empty loci. On ignored repetition trials (Panel *b*), the probe target appeared in the same location as the identical prime distractor.

(Tipper *et al.* 1995). The subject's task is to point to or locate the larger of two circles in a prime and subsequent probe display (by making a spatially compatible movement on a computer joystick). As seen in Fig. 2, on ignored repetition trials, the smaller distractor circle on the prime display is identical to, and occurs in the same location as, the larger target circle on the probe display. The significant negative priming that is observed with this task (Tipper *et al.* 1995) cannot therefore be attributed to a perceptual mismatch between the prime distractor and the probe target because both objects are identical. Such an observation provides strong evidence that NP does indeed reflect an inhibitory mechanism of selection. This task is used here to assess inhibitory mechanisms in schizophrenia.

METHOD

Subjects

Subjects were stable out-patients recruited from the Hamilton Program for Schizophrenia, a case management, intensive treatment community clinic. Twenty-eight individuals who met criteria for schizophrenia, as measured by the Structured Clinical Interview for DSM-IV (SCID) (Spitzer & Williams, 1994) completed the task. All subjects provided written informed consent and had the Positive and Negative Symptom Scale administered within 1 week of completing the NP task. Chart records and clinical history were reviewed with subjects and treating clinicians when necessary to ascertain the age of illness onset, last hospitalization, living situation and current medication profile of each participant.

Control subjects ($N=28$) were healthy volunteers recruited, according to hospital guidelines, from the community and hospital staff by advertisements. Control subjects were selected to be age and sex matched with subjects with schizophrenia. Control subjects had no history of major psychiatric illness as measured by the SCID and were not on psychotropic medication at the time of testing. They had no known first-degree relatives with a psychotic disorder. Both control subjects and those with schizophrenia were excluded if there was a history of substance abuse or dependence. Demographic characteristics of the subjects are described in Table 1.

Apparatus

The experiment was programmed on an IBM-compatible microcomputer with stimuli presented on a VGA colour monitor. All stimuli were presented in VGA medium resolution graphics mode. Responses were made by a joystick

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Table 1. *Demographic and clinical characteristics of the subjects*

Demographic/clinical variable	Patients with schizophrenia	Control subjects
Age, mean	33·8	34·0
Sex	22 M/6 F	22 M/6 F
Age of illness onset, years	22·4 (6·5)	NA
Accommodation, *N* (%)		
Independent	10 (35·7)	28 (100)
With family	2 (7·4)	
Boarding home	6 (21·4)	
Supported apartment	4 (14·3)	
Other	6 (21·4)	
PANSS, mean (S.D.)		
Total score	62·3 (17·6)	NA
Positive	16·5 (6·4)	NA
Negative	19·0 (10·5)	NA
Duration of illness, mean (S.D.)	11·9 (7·1)	NA
Months since last hospitalization	41·3 (40·8)	NA

interfaced with the computer via a standard gameport. Reaction times were computed with Boven's & Brysbaert's (1990) TIMEX function. Response times and error rates were collected automatically for each trial. The viewing distance to the monitor was approximately 45 cm. Stimuli for the circle task were a series of small (4 mm), medium (8 mm) and large (16 mm) circles. Circles were drawn in solid black presented on a white background in one of four possible loci marked by central black arrows (see Fig. 2).

Procedure

Subjects were told that during each trial, they would see two circles on the screen and that one circle would be larger than the other. The circles would appear in two of four possible locations, with four arrows (pointing up, down, left and right from the centre of the screen) used to mark the four possible locations. The subject's task was to indicate the location of the larger circle as quickly as possible by making a spatially compatible movement of the joystick (up, down, left or right).

Each pair of trials (prime-probe) began with a prompt for the subjects to press the start key. Immediately after the start key was pressed, the four arrows appeared on the screen. The arrows remained visible throughout each trial. The distance from the tip of the left pointing arrow to the tip of the right pointing arrow was 4·07°, as was the distance between the tip of the upward pointing arrow to the tip of the downward pointing arrow.

Then, 1500 ms after the start key had been pressed, the first of two displays (the prime) was presented. One circle was displayed at the tip of each of two arrows. In the prime display, the larger (target) circle was always a size 3 circle and the smaller circle was always size 2. The prime display remained on the screen until a response was made (i.e. the subject moved the joystick up, down, left or right). Auditory feedback allowed the subject to distinguish between correct and incorrect responses throughout the procedure. Following the response, the display vanished, leaving only the arrows on the screen for 357 ms. The probe display was then presented. As in the prime, there were two circles, one larger than the other. The larger circle was always size 2 and the smaller circle was always size 1. The probe display circles also remained on the screen until the subject made a joystick response. The screen was then cleared and the prompt to press the start key reappeared.

Design

The probe trials contained a within-subject priming trials factor; trials were divided between control and ignored repetition (IR). On control trials, the two probe circles appeared in the two locations that had not been occupied by either circle on the previous prime trial. On ignored repetition trials, the probe target (size 2 circle) always appeared in the location that had been occupied by the identical prime distractor. There were 18 initial practice trials, followed by 90 experimental trials. There were twice as many control trials ($N=60$) as NP trials ($N=30$).

RESULTS

Reaction times

A repeated measures analysis of variance was used to compare RTs of patients *versus* controls across prime trial types (Control *v*. Ignored repetition). There was a significant effect of subject group ($F(43, 1)=15{\cdot}33$, $P<0{\cdot}001$) and trial type ($F(86, 2)=6{\cdot}63$, $P=0{\cdot}002$), but no significant interaction between subject group and trial type ($F(86, 2)=0{\cdot}79$, $P=0{\cdot}457$). Table 2 provides a summary of all RTs and errors.

NP effects

Because the RTs of the patients were consistently and significantly slower in this task, a

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Table 2. *Reaction times and errors across all experimental conditions. Clozapine-treated patients with matched controls and patients treated with typical antipsychotics with matched controls are shown separately as* post hoc *analyses suggested that the clozapine-treated group display NP effects that are distinct from patients treated with other antipsychotic medications*

Experimental condition	Clozapine-treated subjects ($N=7$)	Clozapine-treated control subjects ($N=7$)	Subjects receiving other antipsychotic medications ($N=21$)	Control subjects ($N=21$)
Prime RT (ms)	604	507	746	518
Prime error (%)	1·3	0·4	0·6	0·6
Control RT (ms)	601	521	773	491
Control error (%)	2·4	0·0	1·6	0·8
IR RT (ms)	635	552	774	518
IR error (%)	1·7	0·8	0·7	1·2

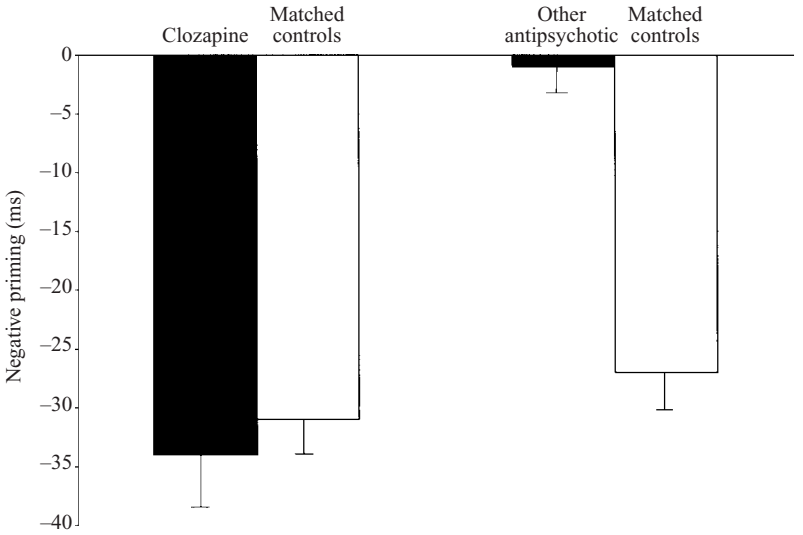

FIG. 3. Negative priming effects in the experimental task.

transformation was applied to the NP data, such that the raw NP effect (in ms) was divided by the raw RT (in ms) to control for variation between groups. An independent samples t test found a significant difference in NP, with patients having reduced levels of NP ($t=2{\cdot}46$, $P=0{\cdot}018$). NP in this task is thought to be secondary to inhibitory effects only, suggesting that the reduction in NP apparent in the patient group is secondary to reduced levels of inhibition.

Error rates

A repeated measures analysis of variance was used to examine the pattern of error rates in this task. There was no significant effect of subject group ($F=2{\cdot}13$, $P=0{\cdot}15$) or trial type ($F=1{\cdot}02$, $P=0{\cdot}37$) neither was there a subject group by trial type interaction ($F=2{\cdot}48$, $P=0{\cdot}09$).

Correlations

There was a relation between subjects' age and reaction times on this task ($r^2=0{\cdot}30$, $P=0{\cdot}04$ for the prime trials, $r^2=0{\cdot}26$, $P=0{\cdot}08$ for the probe control trials, and $r^2=0{\cdot}25$, $P=0{\cdot}09$ for the probe ignored repetition trial types). Importantly, age did not predict magnitude of the NP effect in this spatial localization task (see also Tipper & McLaren, 1991), neither did other clinical variables predict performance in the patient group.

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Medication effects

We examined the potential relation between current treatment and NP in an exploratory manner, interested in whether some of the variance in NP observed in the patient group might be accounted for by differences in treatment, specifically differences in patients receiving typical *versus* atypical agents. When patients who were treated with clozapine were compared against patients treated with typical antipsychotic medications, there was a significant effect of clozapine treatment on NP effect (Fig. 3). Clozapine treated patients had greater levels of NP than other patients ($t=2{\cdot}50$, $P=0{\cdot}02$) and did not differ from matched control subjects in NP levels. Patients who were treated with clozapine also had faster reaction times in the prime trial ($t=2{\cdot}53$, $P=0{\cdot}02$), probe control trial ($t=2{\cdot}13$, $P=0{\cdot}05$) and probe ignored repetition trial ($t=2{\cdot}2$, $P=0{\cdot}04$). The effect of clozapine on raw RTs may have been confounded by age as in our sample patients treated with clozapine were 5 years younger on average, and age did predict raw RT performance. The effect of clozapine treatment on NP, however, is not likely to be accounted for by an age effect as there was no relation between age and NP effect in this task, either in the patient group ($r^2=0{\cdot}29$, $P=0{\cdot}15$) or in the overall sample.

DISCUSSION

This study investigated whether NP is reduced in subjects with schizophrenia after removal of the perceptual mismatch confound present in all previous investigations of this issue. The decline in NP observed in patients with schizophrenia does not appear to be secondary to abnormal perceptual mismatch processes, because in this experiment the ignored distractor in the prime display is physically identical to the subsequent target in the probe display. Confirming other research (e.g. Milliken *et al.* 1994; Tipper *et al.* 1994, 1995), perceptual mismatching does not seem to be the main mechanism mediating NP effects in such tasks. It thus appears that patients with schizophrenia do have reduced inhibitory control processes, as revealed by declines in NP effects.

A second issue of importance in this task is that it required report of a target's spatial location, rather than its identity. To our knowledge, this is the first report of reduced NP in people with schizophrenia in such a task; all previous work required report of some identity feature of the target (e.g. naming colour, reading words or naming pictures). These latter identification tasks are mediated by the temporal lobe, whereas target localization appears to be mediated by the parietal lobe (e.g. Mishkin *et al.* 1983). Thus, the inhibitory deficits in schizophrenia are not isolated to cortical systems processing object meaning. Rather the deficits are diffuse, appearing in brain networks encoding and directing action towards target location.

It appears likely that patients with schizophrenia produce less NP because the inhibitory mechanisms acting on the competing distractor are less efficient. An alternative explanation is that levels of inhibition are normal, but there is a weakness in retrieval of the inhibition during probe processing or perhaps inhibition decays more rapidly in patients. These possibilities need to be considered, but there are three reasons to doubt this latter memory failure hypothesis: first, in this experiment the memory period is very brief (356 ms RSI), hence there is little chance for decay. Very long-term NP effects have now been observed (e.g. DeSchepper & Treisman, 1996), so it is not typically a transient phenomenon. Secondly, the ignored prime and subsequent target probe are identical objects. Therefore, the proposed processes that retrieve prior inhibition associated with a stimulus should be most efficient. And third, such short-term memory deficits have not been established in schizophrenia (in contrast, even working memory deficits have a much longer time frame), whereas a substantial literature has identified failures of inhibitory control. Nonetheless, the memory failure hypothesis is worthy of further consideration in future research (see Tipper, 2001 for a more detailed discussion of competing hypotheses of negative priming).

Concerning the apparent effect of medication, the finding that clozapine-treated patients performed more like control subjects in NP tasks is compelling but needs to be interpreted with caution. It is unlikely that the difference in response observed here is because clozapine-treated patients had a less severe form of illness, first because the PANSS scores were equivalent in clozapine-treated patients compared to those

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treated with other antipsychotic medications; secondly, patients are not generally prescribed clozapine unless they have a severe or resistant form of illness. Literature supports the notion of improvement in aspects of cognition with clozapine treatment (Meltzer & McGurk, 1999), and these results suggest that one mechanism for this improvement could be because clozapine improves inhibitory control. Overall, the evidence appears to suggest that antipsychotic medications in general have a salutary effect on vigilance tasks (Goldberg & Weinberger, 1996), and previous studies of NP do support the notion that inhibitory processes are enhanced by antipsychotic medication (Beech *et al*. 1990). In our study, however, treatment with other antipsychotic medications did not restore levels of NP, but treatment with clozapine specifically was associated with levels of NP equivalent to those of non-psychiatric control subjects.

In summary, our results confirm that inhibitory processes are reduced in patients with schizophrenia. The deficits apparent on these tasks are not likely the result of failure in the perceptual review process. These results support the notion that inhibitory deficits may be diffuse and not confined to neural systems required for object identification. Finally, clozapine-treated patients had levels of NP that approximated non-psychiatric control subjects. Future studies examining the performance of patients with schizophrenia on NP tasks may provide insight into the mechanisms underlying the attention deficits in these patients, and also into the mechanisms by which antipsychotic medications such as clozapine may improve attention processes in patients with schizophrenia.

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