

Attentional Inhibition in Schizophrenia and Schizotypy: A Spatial Negative Priming Study

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We investigated attentional inhibition in schizophrenia with a spatial negative priming task. In Study 1, we compared acutely psychotic schizophrenic inpatients with chronic schizophrenic outpatients in remission. Acute patients showed absence of spatial negative priming, in contrast to the chronic outpatients who displayed intact inhibition. In Study 2, we found that spatial negative priming was abolished in healthy, first-degree relatives of schizophrenic patients, indicating a possible impairment of attentional inhibition, in the absence of medication. In Study 3, we examined the individual differences in spatial negative priming in relation to schizotypy. A small reduction in negative prime effect was found in the schizotypic subjects. Reduced negative priming was much more pronounced in schizotypic women than in schizotypic men, indicating a possible influence of gender.

INTRODUCTION

One of the cognitive deficits that may characterise schizophrenia is an increase in distractibility coupled with attentional impairments (see Nuechterlein & Dawson, 1984). Patients with schizophrenia seem unable to focus on the relevant stimuli while ignoring the irrelevant (e.g. McGhie & Chapman, 1961; Shakow, 1962). Such observations have led to the formulation of hypotheses

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that focus on diminished or disrupted inhibitory processes in schizophrenia; weakened inhibition would presumably allow irrelevant stimuli to intrude during information processing (e.g. Claridge, 1967; Frith, 1992; Hemsley, 1987). There is now growing experimental evidence for the presence of inhibitory deficit during a variety of attention tasks in schizophrenic patients (e.g. Elkins & Cromwell, 1994; Nestor et al., 1992; Steffy & Galbraith, 1974).

The *negative priming paradigm* (Tipper, 1985) was originally developed to assess and quantify the inhibitory component of selective attention. Selective attention is achieved by at least two mechanisms: one involving an excitatory process associated with the target stimulus and an inhibitory mechanism that is associated with the ignored stimulus (e.g. Neill & Westberry, 1987). The negative priming paradigm involves two steps. The initial stage involves exposing subjects to irrelevant stimuli that are to be ignored. When a stimulus is ignored during a selective attention task, its internal representation is hypothesised to be associated with inhibitory processes. The second step involves the selection of previously ignored stimuli. One important consequence of such inhibitory influences is that the *later* selection of the ignored stimulus *increases* the reaction time. Hence, previous exposure retards (rather than improves) later response.

Negative priming seems to be independent of visual features of the ignored stimulus (Tipper & Driver, 1988), retinal locus (Tipper, Brehaut, & Driver, 1990), specific motor responses (Tipper, MacQueen, & Brehaut, 1988), and the modality of tasks (Tipper, Weaver, Cameron, Brehaut, & Bastedo, 1991). For example, inhibitory processes exert their influence whether the task involves identification and naming of the target stimuli or just a simple spatial localisation of the target. In addition, this effect seems to be robust and long-lasting (about 7 seconds), suggesting an active role of inhibition in selective attention (Tipper et al., 1991).

Because some of the positive symptoms of schizophrenia are associated with impaired inhibition and attentional processes (e.g. Gray, Feldon, Rawlins, Hemsley, & Smith, 1991), the negative priming paradigm is eminently suitable for assessing the degree of attentional inhibition in schizophrenic patients. Schizophrenic patients should show either absence of, or a reduced negative priming effect, compared with the normal controls; schizophrenic patients are expected to perform *faster* (not slower) than normal controls under certain circumstances, because subsequent selection of the ignored stimulus will *not* increase the reaction time, owing to impaired inhibitory processes.

Beech, Powell, McWilliam, and Claridge (1989) used a Stroop-like interference paradigm to assess the negative priming effect in medicated schizophrenic patients. Negative priming was reduced (but not abolished) in these patients. In a subsequent study, they also found that dopamine antagonists can increase cognitive inhibition (Beech, Powell, McWilliam, & Claridge, 1990) and therefore negative priming may become normalised in medicated

schizophrenic patients, in contrast to acutely ill schizophrenic patients who are presumably in a hyperdopaminergic state. In addition, positive symptoms but not negative symptoms are associated with negative priming and therefore acute schizophrenic patients may be more likely to show reduced inhibition. Similar results were obtained from individuals with schizotypal traits, as assessed by standardised personality questionnaires. Schizotypic subjects showed reduction in negative priming effect in verbal negative priming tasks (Beech & Claridge, 1987; Beech, McManus, Baylis, Tipper, & Agar, 1991; Beech et al., 1989).

Neurobiological studies of attentional inhibition point to the important role of dopaminergic action in the limbic system (Gray et al., 1991). Venables (1992) has suggested that the ability to distinguish irrelevant from relevant stimuli, mediated by the hippocampus, is disrupted in acutely ill schizophrenic patients. Dopamine antagonists increase inhibition (Beech et al., 1990; Weiner, Shofel, & Feldon, 1990) and dopamine agonists abolish or reduce inhibition (Gray et al., 1991; Gray, Pickering, Hemsley, Dawling, & Gray, 1992; Weiner, Lubow, & Feldon, 1988). The idea that exposure to an irrelevant stimulus leads to a deterioration of performance has been studied extensively in rats with "latent inhibition" paradigm. Latent inhibition is structurally similar to negative priming and indeed, dopamine agonists (e.g. amphetamine) abolish both latent inhibition and negative priming (Gray et al., 1991; Feldon, personal communication). Limbic system abnormalities, especially those involving the septo-hippocampal system, disrupt cognitive inhibition (Weiner et al., 1990; Gray et al., 1991). Baruch, Hemsley, and Gray (1988) found that latent inhibition was disrupted in acute schizophrenic patients but not in medicated, chronic patients, presumably because the acute patients were in a hyperdopaminergic state. Recently, Lyon and his colleagues have demonstrated that dopamine agonists reduce spatial negative priming effect in rats and that this effect can be reversed by a dopamine antagonist (Lyons, Karson, & Freeman, 1995). Taken together, these studies implicate the important role of dopaminergic action on the limbic system, in maintaining attentional inhibitory processes.

We examined attentional inhibition, as assessed by a spatial negative priming task in schizophrenia, as well as in populations presumed to be at risk for schizophrenia, namely the first-degree relatives of schizophrenic patients and psychometrically ascertained schizotypes. We employed a nonverbal, spatial negative priming task to minimise the linguistic and cognitive demands generated by tasks that require naming or reading. We specifically chose to assess attentional inhibition with a spatial negative priming task rather than with Stroop-like, verbal tasks employed in past studies by Beech and his colleagues, for the following reasons:

1. Spatial negative priming task is a simple localisation task, which generates almost no performance errors. Cognitive demands are low.

2. Nonverbal tasks render comparison with animal studies of attentional inhibition more feasible, allowing neurobiological basis of negative priming to be inferred indirectly.
3. Negative priming tasks that have been used in schizotypy research require linguistic-processing and articulatory responses (i.e. left hemisphere-dependent in right-handed males). But language may be less lateralised in women than in men (McGlone, 1980) and in schizotypes than in control subjects (Broks, 1984; Rawlings & Claridge, 1984). We administered the spatial negative priming task specifically to minimise any linguistic demands on the subjects.

STUDY 1: SPATIAL NEGATIVE PRIMING IN ACUTE INPATIENTS AND CHRONIC OUTPATIENTS

In the first study, we recruited both acute schizophrenic inpatients and chronic outpatients in remission. We hypothesised that acute schizophrenic inpatients would fail to show negative priming because they are assumed to be in hyperdopaminergic state but that chronic schizophrenic outpatients in remission would show intact negative priming.

Method

Subjects

Normal controls: 28 volunteer subjects (15 men and 13 women) from Cambridge, Massachusetts and Zürich, Switzerland were tested. Subjects were clerical workers, nurses, cleaning staff, and students. The mean age was 34.8 (SD = 10.6) and the mean number of years of education was 12.8 (SD = 1.4). All subjects were screened for medication and history of mental illness personally or in their family.

Chronic outpatients in remission: 18 schizophrenic outpatients (10 women and 8 men), who had not been hospitalised for at least one year, were recruited from a private hospital outpatient unit. These subjects met criteria for DSMIII-R diagnosis of schizophrenia, as determined from the Structured Clinical Interview for DSMIII-R (Spitzer & Williams, 1985), administered by a staff psychiatrist or a psychologist, and a chart review. These patients had no evidence of organic brain pathology. Their mean age was 33.4 (SD = 6.5), the mean number of years of education was 13.4 (SD = 1.2), and the mean duration of illness was 13.1 years (SD = 6.5). All patients were taking antipsychotic medications (haloperidol, perphenazine, clozapine, fluphenazine, or thioridazine). Four patients were also taking anti-anxiety drugs (propranolol) and five patients were receiving antidepressants (fluoxetine or desipramine).

Acutely ill inpatients within one week of hospitalisation: 19 acute schizophrenia patients (4 women and 15 men) were recruited from the

Psychiatric Clinic of the University Hospital Zürich. These subjects met criteria for a DSMIII-R diagnosis of schizophrenia in a structured clinical interview, administered by a staff psychiatrist, and a chart review. They had no evidence of organic brain pathology. Their mean age was 33.0 (SD=9.4), the mean number of years of education was 12.2 years (SD=1.5), and the mean duration of illness was 10.9 years (SD=7.6). Three patients were unmedicated because they were refusing to take any medication at the time of testing. The other 16 patients had been receiving antipsychotic medication (haloperidol or clozapine) since their hospital admission (1 week or less).

The three groups did not differ significantly in age [$F(2,62)=0.23$, $P>0.79$]. The two patient groups did not differ significantly in the duration of illness [$F(1,35)=0.85$, $P>0.36$]. There was a significant difference in the number years of education [$F(2,62)=3.4$, $P<0.04$]. The chronic outpatients had more years of education (13.4) than did the acute patients (12.2). However, there was no difference between the acute patients and the normal controls (12.8) in the number of years of education ($P>0.15$). It must be noted that the education system in Switzerland is different from that in the United States. Therefore, a one-year difference in the duration of education, although statistically significant, may not reflect a meaningful difference in the general levels of education attained by the patients. American patients were paid a modest honorarium for their participation. Swiss patients were not paid, in accordance with the customs of the clinic.

Apparatus and Stimuli

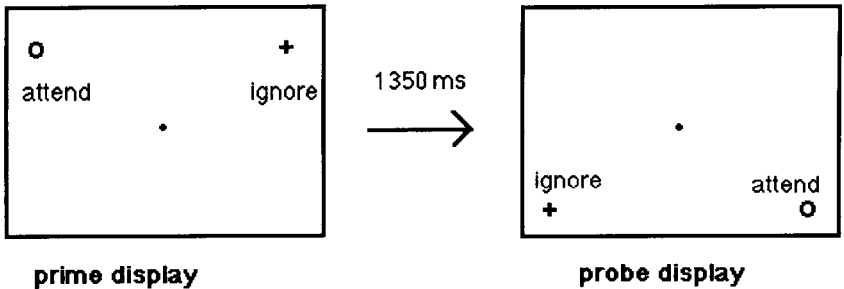
The stimuli were presented on a Macintosh computer. There were four locations on the screen where the target (O) or the distractor (+) could appear. The four positions were placed on the corners of an imaginary square. The visual angle between the horizontal or the vertical positions was 7.8 degrees (see Fig. 1). The stimuli (O or +) subtended 0.6 by 0.6 degrees of visual angle. Subjects sat 45cm from the screen; a chin rest was used to minimise head movement. Each trial was started by the experimenter only when the subject was fixating in the centre.

Design

All procedures and stimuli were constructed to approximate Tipper et al.'s experiment 3 (1991) as closely as possible. Each trial consisted of a pair of prime and probe displays. There were four positions at which the stimuli could be presented. Each trial consisted of a pair of prime and probe displays; each prime display was always followed by a probe display. Subjects were asked to locate the target and ignore the distractor (see Fig. 2).

There were two types of trials, all consisting of pairs of prime and probe displays: control (C) and ignored repetition (IR). In the control trials, the

Control trial



Ignored Repetition trial

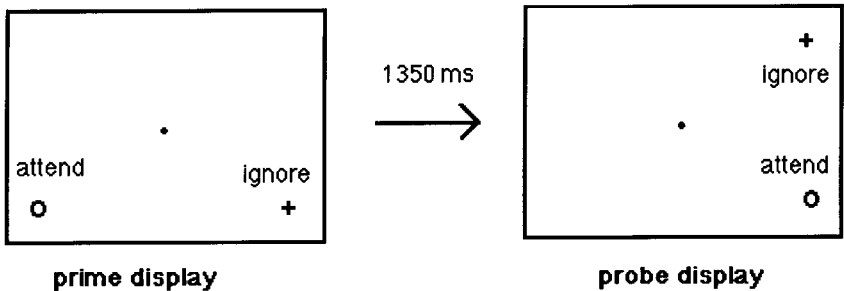


FIG. 1. Control and ignored repetition trials.

positions of the target and the distractor in the prime and the probe displays were all different, whereas in the ignored repetition trials, the location of the *target in the probe display* was identical to the location of the *distractor in the prime display*. Therefore, in the ignored repetition probe trials, subjects were required to respond to the location that they had just ignored. A negative priming effect is indicated by longer reaction times (RTs) in the ignored repetition probe condition than in the control probe condition. Negative priming was examined within subjects by comparing the RTs to the probe displays of the ignored repetition trials and the RTs to the probe displays of the control trials.

Procedure

Subjects were seated in front of the computer and were asked to read the instructions on the screen. Subjects were told that they must pay attention to the target and to ignore the distractor. They were asked to indicate the location of the target O by pressing the corresponding key on a keyboard and ignore the

distractor +. They were asked to identify the target as quickly and as accurately as possible. Subjects initiated each block of trials by pressing the spacebar. There were 18 trials (i.e. 18 pairs of prime and probe displays) in each block. When subjects were ready, they pressed the spacebar to initiate a block of trials. A trial began with the prime display which stayed on until the subject responded to it by locating the target. Then, there was a 1350msec pause before the second display (probe) was presented. During the final 800msec of the pause, the fixation point appeared at the centre to prepare subjects for the next response. When the subject responded to the probe display by locating the target, a pattern mask was presented and it stayed on the screen. At this point, the subjects were asked if they were ready to go on to the next trial. When ready, the subject pressed the spacebar, after which there was always a 6.4-second delay before the next trial was presented. The mask stayed on the screen during this period. Therefore, there was always a rest period of *at least* 6.4 seconds and, in practice, the inter-trial interval was about 8–10 seconds. This lengthy inter-trial interval was necessary because Tipper et al. (1991) reported that negative priming may last up to about 7 seconds and we wanted to remove any residual priming effect between trials. During the final 800msec of the pause, a fixation point was presented in the centre of the screen to prepare the subject for the next prime display (see Fig. 2 for a schematic diagram of the procedure). Subjects were given 10 practice trials before the beginning of the testing. The order of presentation of different conditions was random within each block. There were four blocks of trials.

Results

The accuracy in locating the target was 100% for almost all the subjects. The average error rate was less than 1%. Negative priming effect is indicated by longer RTs in the ignored repetition probe trials than in the control probe trials. We calculated a raw negative prime score for each subject by subtracting the mean RT of ignored repetition probe trials from the main RT of control probe trials. Negative raw score indicates the presence of negative priming whereas positive raw score indicates absence of negative priming (disinhibition). We counted the number of subjects who show negative raw scores (presence of negative priming). By this count, 36% of acute schizophrenic patients, 72% of the chronic schizophrenic outpatients, and 82% of the control subjects showed negative priming effect.

The mean raw negative prime score for the acute schizophrenic inpatients was +47.0msec (SE = 30.9), which means acute inpatients were faster in the ignored repetition probe trials than in the control probe trials (disinhibition, absence of negative priming). Chronic outpatients' mean raw negative prime score was -34.7msec (SE = 17.9) and that for the normal control subjects was -26.9msec (SE = 5.5). Both chronic outpatients and normal controls showed an

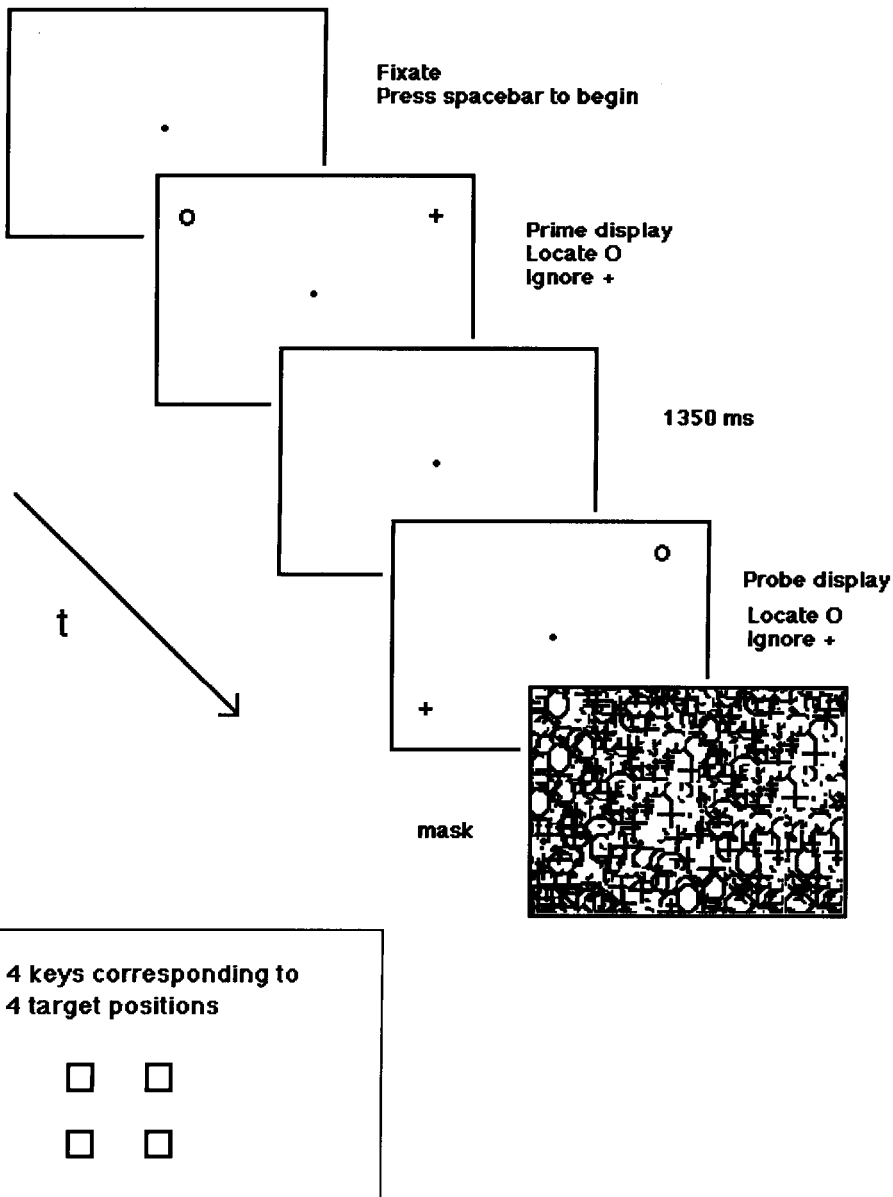


FIG. 2. Sequence of instructions presented on the computer screen.

increase in mean RTs for the ignored repetition probe trials (inhibition, presence of negative priming).

A one-way analysis of variance showed that there was a significant main effect of the subject groups in the raw negative prime score [$F(2,62) = 5.56$, $P < 0.006$]. Chronic schizophrenic outpatients and normal controls displayed the negative priming effect, whereas there was an absence of negative priming effect in acute schizophrenic inpatients (see Table 1).

The three groups differed significantly in their baseline RTs [$F(2,62) = 7.1$, $P < 0.0008$]. Normal controls were twice as fast as the acute and chronic schizophrenic patients regardless of the trial type. To take account of the large difference in baseline RTs, we computed a percent negative prime score as follows:

$$\% \text{ Negative Prime Score} = \frac{\text{Raw Negative Prime Score}}{\text{RT (Control Probe Trials)}} \times 100$$

An analysis of variance showed that the three groups differ significantly on their percentage in negative prime scores [$F(2,62) = 6.2$, $P < 0.004$]. The acute inpatients showed a 3.4% decrease in the ignored repetition RT (disinhibition or absence of negative priming). The chronic outpatients showed 4.7% increase in the ignored repetition RT (inhibition, presence of negative priming), which is not statistically different from the 5.4% increase shown by the normal control subjects (see Fig. 3).

Acute inpatients showed less percentage in negative priming than did the outpatients [$F(1,35) = 5.24$, $P < 0.03$; Scheffe F -test = 5.24]. Acute inpatients showed significantly less percentage in negative priming effect than did the control group [$F(1,45) = 14.6$, $P < 0.0004$; Scheffe F -test = 14.6]. Outpatients did not differ from the normal controls in percentage in negative priming [$F(1,43) = 0.066$, $P > 0.79$; Scheffe F -test = 0.066].

TABLE 1
Negative Prime Effect in Schizophrenic Patients and Control Subjects

Reaction Times (msec)	Acute Inpatients ($n = 19$)	Chronic Outpatients ($n = 18$)	Normal Controls ($n = 28$)
Control	828.0 (SE = 85.3)	809.7 (SE = 117.1)	481.0 (SE = 10.7)
Ignored repetition	781.0 (SE = 68.6)	844.4 (SE = 121.8)	507.9 (SE = 13.6)
Negative prime raw score	+47.0 (SE = 30.9)	-34.7 (SE = 17.9)	-26.9 (SE = 5.5)
Negative prime score (%)	+3.4% (SE = 2.3)	-4.7% (SE = 2.7)	-5.4% (SE = 1.1)

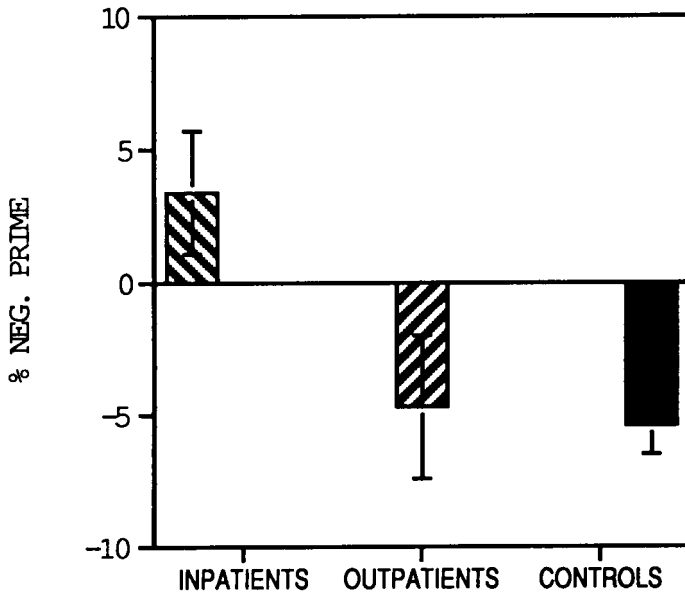


FIG. 3. Percentage of negative prime scores of schizophrenic patients and controls.

Discussion

Acute schizophrenia inpatients, tested within one week of hospital admission, showed absence of negative priming, in contrast to chronic schizophrenia outpatients who displayed a negative priming effect in much the same way as did the normal controls, even though their overall reaction times were slowed down to the same level as the acute patients.

What factors are responsible for the intact negative priming effect in the chronic schizophrenia outpatients? Abolition of negative priming in the acute patients may be examined in the context of their positive symptoms. All acute patients were floridly psychotic at the time of testing. Positive symptoms have been associated with impairments of attentional inhibition (Gray et al., 1991). They have also been associated with attentional dysfunction *per se* (e.g. Cornblatt, Lenzenweger, Dworkin, & Erlenmeyer-Kimling, 1985). It is not clear whether absence of inhibition eventually leads to some of the clinical symptoms such as formal thought disorder, or whether cognitive disinhibition occurs in parallel to other clinical symptoms. It is also possible that a third variable accounts for both (i.e. dopaminergic dysfunction).

Negative priming in animals and humans can be abolished or reduced by dopamine agonists (Gray et al., 1991, 1992; Weiner et al., 1988). Dopamine antagonists increase or restore negative priming (Beech et al., 1990; Weiner et

al., 1990). Moreover, absence of negative priming may indicate abnormalities of the limbic system (e.g. Venables, 1992). All the chronic outpatients were complying with medication instructions and had been out of the hospital for at least one year prior to testing. It is possible that attentional inhibition is restored in chronic outpatients in remission by steady intake of neuroleptic medication (i.e. dopamine antagonist). Sixteen out of the 19 acute patients were also receiving antipsychotic medication at the time of testing but they had been unmedicated (noncompliance or off-medication) prior to the hospitalisation, which was within one week of testing. Therefore, the extent of the dopaminergic action in these inpatients is unclear. One limitation of this study is that we do not have measures of the symptoms. Therefore, we cannot examine the correlation between some measure of positive symptoms and the negative priming scores.

We found that negative priming effect was absent in acute schizophrenic inpatients but fully present in chronic schizophrenic outpatients. We do not know if the absence of negative priming is a trait variable that is ‘‘restored’’ or ‘‘normalised’’ by the neuroleptic treatment. To address this question, we recruited unaffected, first-degree relatives of schizophrenia patients and a new group of matched control subjects.

STUDY 2: SPATIAL NEGATIVE PRIMING IN THE RELATIVES OF SCHIZOPHRENIA PATIENTS

In Study 2, we tested whether clinically unaffected relatives of schizophrenic patients show negative priming abnormalities, in the absence of illness and medication effect. Studies of first-degree relatives of schizophrenic patients, including twins discordant for schizophrenia, have been very important in broadening our concept of the phenotype of schizophrenia. Past studies of the relatives of schizophrenic patients have revealed many shared attributes with schizophrenic patients, such as a high incidence of eye-tracking abnormalities (e.g. Holzman, 1985; Levy, Holzman, Matthyse, & Mendell, 1993), presence of formal thought disorder (Shenton, Solovay, Holzman, Coleman, & Gale, 1989), working memory deficit (Park, Holzman, & Levy, 1993; Park, Holzman, & Goldman-Rakic, 1995), sensory gating deficit (Waldo et al., 1991), among others. Presence of negative priming abnormalities in the first-degree relatives would indicate subtle deficits in attentional inhibition in otherwise healthy individuals who are at risk for schizophrenia.

Method

Subjects

Normal controls: 33 volunteer subjects (15 men and 18 women) from Cambridge, Massachusetts and Zürich, Switzerland were tested. Subjects were mental health workers, clerical workers, and students. The mean age was 34.2

(SD = 10.1) and the mean number of years of education was 15.8 (SD = 3.2). All subjects were screened for medication and history of mental illness personally or in their family.

Relatives of schizophrenic patients: 14 unaffected (no DSM-III-R Axis I diagnosis), medication-free siblings, and parents of schizophrenic patients (6 men and 8 women) participated in the study. They were from nine different families. They were recruited from the McLean Hospital Collaborative Schizophrenia Research Study. Their mean age was 36.5 (SD = 7.8) and the average number of years of education was 15.5 (SD = 1.4). All subjects except one were employed (1 social worker, 2 lab technicians, 1 physicist, 3 students, 1 engineer, 3 businessmen, 2 teachers).

There were no statistically significant differences between the two subject groups in age and education level. Written informed consent was obtained from all subjects. American subjects were paid a modest honorarium for their participation.

Design and Procedure

All the experimental details were the same as in Study 1.

Results

The accuracy in locating the target was 100% for most subjects. The average error rate was less than 1%. The raw negative prime score was calculated for each subject by subtracting the mean RTs of the ignored repetition probe trials from the mean of the RTs of control probe trials, as in Study 1. Negative raw score indicates the presence of negative priming whereas positive raw score indicates absence of negative priming (disinhibition). We counted the number of subjects who show negative raw scores (presence of negative priming). By this count, 57% of the relatives and 100% of the control subjects showed a negative priming effect.

There was a significant group difference in the raw negative prime score [$F(1,45) = 8.4, P < 0.006$]. The mean raw score for the relatives was +8.7msec (SE = 19.0) and that for the normal controls was -31.0msec (SE = 4.0). Normal control subjects were faster in the control trials than in the ignored repetition trials (hence presence of negative priming), whereas the relatives showed the opposite pattern overall (absence of negative priming) (see Table 2). All of the control subjects exhibited negative priming (the negative prime score was *negative*) whereas only 57% of the relatives showed negative priming effect. The raw score for the normal controls was comparable to that obtained from the control subjects in Study 1 (-26.9msec) (see Table 2).

There was a significant difference in the mean RTs between the two groups [$F(1,45) = 13.8, P < 0.0006$]. Although they were not clinically ill or medicated, the relatives (657.5msec) were slower than the control subjects (491.3msec).

TABLE 2
Negative Prime Effect in the First-degree Relatives of Schizophrenic Patients
and in Controls

<i>Reaction Times (msec)</i>	<i>Relatives (n = 14)</i>	<i>Normal Controls (n = 33)</i>
Control	657.5 (SE = 68.0)	475.8 (SE = 12.2)
Ignored repetition	648.8 (SE = 52.6)	506.8 (SE = 13.9)
Negative prime raw score	+ 8.7 (SE = 19.0)	- 31.0 (SE = 4.0)
Negative prime score (%)	- 0.39 (SE = 1.68)	- 6.47 (SE = 0.79)

To take account of the large difference in the baseline RTs between the two groups, we computed a percentage negative prime score, as in Study 1. A one-way analysis of variance showed a significant difference [$F(1,45)=13.9$, $P<0.0005$] between the relatives (-0.39%) and the normal controls (-6.5%) (see Fig. 4).

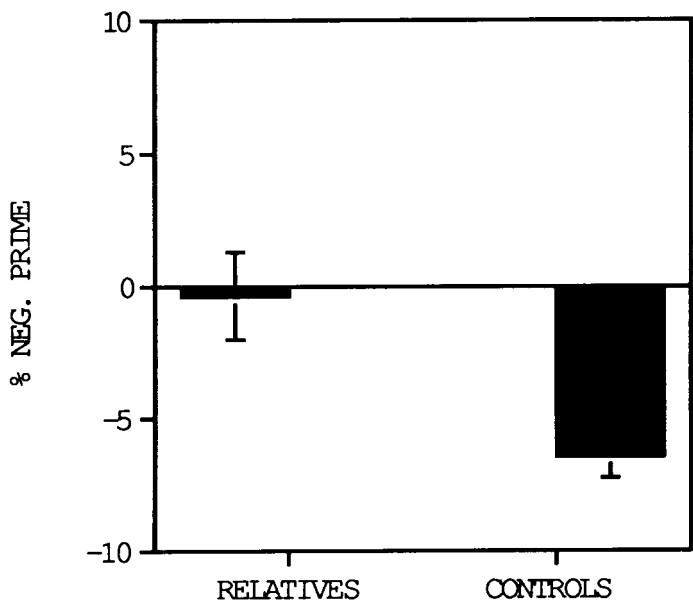


FIG. 4. Percentage of negative prime scores of relatives and controls.

Discussion

Past research has already established the significant increase in the morbid risk for schizophrenia in the first-degree relatives of schizophrenic patients. The reduction of spatial negative priming in the relatives of schizophrenic patients suggests that the inhibitory processes during a selective attention task may be impaired in the absence of medication. This result emphasises the importance of studying the healthy relatives in order to further our understanding of the attentional impairments in schizophrenia.

These relatives were healthy and medication-free, yet about half of them showed more disinhibition than did chronic schizophrenia patients from Study 1. It is possible that some of the relatives may have schizotypal personality, which has been shown to associate with absence of negative priming. We were not able to assess the personality profiles of the relatives of schizophrenic patients. However, the question of schizotypy and its influence on negative priming has been studied before by Stroop-like negative priming paradigms (e.g. Beech & Claridge, 1987; Beech et al., 1991; Peters, Pickering, & Hemsley, 1994). It has also been suggested that positive symptomatology is an important factor for negative priming (Peters et al., 1994). In the third study, we examined individual differences in spatial negative priming in relation to schizotypy, as assessed by the Perceptual Aberration Scale (PAS) (Chapman, Chapman, & Raulin, 1978), which is associated with the positive symptomatology of schizotypy.

STUDY 3: INDIVIDUAL DIFFERENCES IN SPATIAL NEGATIVE PRIMING IN RELATION TO SCHIZOTYPY AS ASSESSED BY THE PAS

Previous studies suggest that hypothetically “psychosis-prone” individuals (see Chapman & Chapman, 1985) within the general population may carry a latent liability for schizophrenia although they may never become ill (see Lenzenweger & Loranger, 1989a,b; Meehl, 1990). In Study 3, we examined attentional inhibition in relation to schizotypy. We chose the Perceptual Aberration Scale (PAS) as a measure of schizotypy due to the prominence attached to body-image and perceptual distortions in schizotypy, as defined by both Rado and Meehl (see also Chapman et al., 1978; Lenzenweger, 1993). Rado (1960) suggested that the body-image distortions and perceptual anomalies reflect the psychological experience of the schizotype, deriving fundamentally from the putative proprioceptive (kinesthetic) diathesis. Meehl (1964, 1990) referred to body-image distortions as an outgrowth of the spatial-kinesthetic-vestibular system aberrations deriving from the hypothesised, ubiquitous central nervous system anomaly (i.e. schizotaxia), and the related associative loosening.

In nonclinical university samples, individuals who score high on the PAS exhibit psychotic-like symptoms (Allen, Chapman, Chapman, Vuchetich, &

Frost, 1987; Chapman, Edell, & Chapman, 1980), cognitive slippage (i.e. mild thought disorder) (Allen, Chapman, & Chapman, 1987), Rorschach deviance comparable to that often observed among schizophrenic patients (Edell & Chapman, 1979), sustained attentional deficit (Cornblatt & Erlenmeyer-Kimling, 1985; Lenzenweger, Cornblatt, & Putnick, 1991), impaired performance on the Wisconsin Card Sorting Test (Lenzenweger & Korfine, 1994), and working memory deficit (Park, Holzman, & Lenzenweger, 1995) among others.

Schizophrenic patients also reveal PAS elevations (Chapman et al., 1978). Finally, taxometric analyses of the PAS (Korfine & Lenzenweger, 1995; Lenzenweger & Korfine, 1992) reveal the scale taps into a taxonic latent entity with a general population base rate of approximately 10% for the schizotypy taxon as conjectured by Meehl (1990). Thus, multiple converging lines of evidence show that the PAS is a valid, although fallible, psychometric indicator of schizotypy (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994; Cronbach & Meehl, 1955). However, the specificity and long-term predictive criterion validity of the PAS for detecting schizotypy remains to be firmly established.

Previous research has also shown that attentional disinhibition is associated with schizotypy (e.g. Beech & Claridge, 1987; Beech et al., 1991), particularly in relation to their positive symptoms (Peters et al., 1994). In Study 3, we hypothesised that those individuals who score high on the PAS would show *less* negative priming than those scoring low on the PAS.

An additional factor that may influence the relationship between schizotypy and cognitive inhibition is gender (Claridge, Clark, & Beech, 1991). In negative priming research, the relationship between schizotypy and disinhibition has been most reliably demonstrated in all-male samples (see Beech, 1987). Claridge et al. (1991) failed to find correlation between negative priming and schizotypy in women, whereas they found the expected difference between schizotypes and controls in the male sample. We hypothesised that the negative priming difference between the male schizotypic and control subjects would be greater than that obtained from the female subjects.

Method

Subjects

These were drawn from a large *randomly ascertained* sample of first-year undergraduates from Cornell University who voluntarily completed a 250-item objective psychological inventory entitled "Attitudes, Feelings, and Experiences Questionnaire" that included the PAS (Chapman et al., 1978). We chose this approach in order to maximise diversity within our pool of potential study subjects as well as to minimise the effects of both subject self-selection factors and group-related test-taking attitudes.

Initially, 2000 subjects were selected at random from an exhaustive university roster. A team of trained research assistants individually asked each of the potential study participants to complete the psychological inventory within 48 hours. The completed inventories were collected by study staff in sealed envelopes. The subjects were informed that their inventory responses would remain confidential and would be used for research purposes only.

Of the 2000 students who were asked to complete the inventory, 1684 (51.3% women, 48.7% men) did so. To control for pseudo-random responding and invalid test-taking attitudes, a 14-item version of Jackson's (1984) Infrequency Scale from his *Personality Research Form* was included in the screening inventory. Subjects scoring more than 3 on the Infrequency Scale were dropped from the sample ($n=35$, 2.1% of the sample). Three of the subjects were dropped due to extensive missing data on the inventory. The resulting final sample consisted of 1646 cases.

Two subject groups were composed from the overall pool of 1646 subjects. Separate group means and standard deviations for males and females on the PAS were computed and served as the basis for subject selection. Following Chapman and Chapman (1985), potential schizotypic subjects were required to have scored at least 2.0 standard deviations (SDs) above the group mean on the PAS, whereas normal controls were required to have scored no higher than 0.5 SDs above the group mean. Study subjects for each of the two groups were selected at random from the two subsamples of subjects meeting the specified criteria.

Using these criteria, 30 (15 female) high PAS (i.e. schizotypic) subjects and 25 (15 female) low PAS (i.e. normal control) subjects were selected for the study. The proportions of male and female subjects across the two subject groups did not differ significantly [$\chi^2(1)=0.22$, $n=55$, $P=0.64$]. The mean age and mean PAS score of the schizotypic subjects were 19.00 years ($SD=0.52$) and 19.00 ($SD=6.35$), respectively; age and PAS score means of the controls were 18.96 years ($SD=0.53$) and 0.77 ($SD=0.99$), respectively. There were no differences between the two groups in terms of agreement to participate in the study.

Every subject provided us with a formal release of information that allowed us to obtain their official Scholastic Aptitude Test (SAT) scores, verbal and quantitative, from their Cornell record. There was no difference between the two subject groups in the SAT scores.

The potential subjects had been initially preselected from the general population for academic achievement (i.e. university admission) but academic ability does not preclude a risk to psychopathology (Depue, Krauss, Spont, & Arbis, 1989; Rimmer, Halikas, & Schuckit, 1978; Stangler & Printz, 1980). This sample was probably screened for particularly early-onset variants of severe psychopathology, but this does not preclude the liability for later onset psychoses or schizophrenia spectrum-related personality disorders. Further

details concerning subject selection have been published elsewhere (Park et al., 1995).

The Perceptual Aberration Scale as Schizotypy Indicator

The PAS is a 35-item true-false self-report measure of disturbances and distortions in perceptions of body image as well as other objects (Chapman et al., 1978). It includes items like ‘‘Occasionally I have felt as though my body did not exist’’ (keyed *true*) and ‘‘I have never felt that my arms or legs have momentarily grown in size’’ (keyed *false*). The items included in the PAS were selected for inclusion only after being found to be sufficiently unaffected by social desirability or acquiescence response set (Chapman et al., 1978). Internal consistency analyses typically reveal coefficient alphas around 0.90, and stability of PAS scores is high ($r = 0.75$, Chapman & Chapman, 1985).

Procedures

Potential study participants were contacted by telephone. All subjects were paid for their participation. Because a well-developed complex coding scheme was employed to disguise the group status of the subjects, all study staff were blind to a subject’s group membership throughout both subject recruitment, testing, and scoring. The procedure, design, and methods of the spatial negative priming task were identical to those described in Study 1.

Results

The accuracy in locating the target was 100% for all subjects. The raw negative prime score was calculated for each subject, as in Studies 1 and 2. *Negative* raw scores indicate the presence of negative priming, whereas *positive* raw scores indicate the absence of negative priming (disinhibition). We counted the number of subjects who showed *negative* raw prime scores (i.e. presence of negative priming). By this count, 63% of the high PAS (schizotypic) subjects and 80% of the low PAS subjects (control group) showed the negative priming effect.

The raw mean score for the high PAS (schizotypic) subjects was -7.3 msec ($SE = 5.3$) and that for the control subjects was -16.3 msec ($SE = 4.8$) (see Table 3). These means are very small compared with the results obtained in Studies 1 and 2. An analysis of variance showed that there was a trend towards a group difference in the raw negative prime score and although it was not significant, the effect size was tending towards ‘‘medium’’ [$F(1,53) = 1.52$, $P < 0.11$; effect size $d = 0.34$].

On examining the raw scores, it appears that the control subjects in Study 3 showed much less negative priming than did the control subjects in Studies 1 and 2. The control subjects in Studies 1 and 2 showed an average raw negative

TABLE 3
Negative Prime Effect in High and Low PAS Groups

<i>Reaction Times (msec)</i>	<i>High PAS (Schizotypes)</i> (<i>n</i> = 30)	<i>Low PAS (Controls)</i> (<i>n</i> = 25)
Control	442.1 (SE = 11.4)	409.5 (SE = 13.6)
Ignored repetition	449.4 (SE = 13.1)	420.2 (SE = 13.3)
Negative prime raw score	-7.3 (SE = 5.3)	-16.3 (SE = 4.8)
Negative prime score (%)	-1.47 (SE = 1.17)	-4.40 (SE = 1.33)

prime score of -26.9 msec and -31.0 msec, respectively. The subjects in Studies 1 and 2 were older than the controls in Study 3, but as ageing diminishes the negative priming effect in adults (Tipper, 1991), age is unlikely to be the reason why control subjects in Study 3 showed a smaller negative prime effect.

The difference between the high PAS (schizotypic) subjects and low PAS (control) group became more apparent when we compared their percentage in the negative prime scores (see Table 3). The high PAS (schizotypic) subjects showed a -1.4% ($SE = 1.2$) negative prime effect, compared with -4.4% ($SE = 1.3$) for the control subjects. This difference was statistically significant [$F(1,53) = 2.7$, $P < 0.05$, 1-tailed]. The effect size was medium ($d = 0.45$) (see Fig. 5). In addition, the correlation between the PAS and the percentage in negative prime scores was 0.26 ($P < 0.05$, 1-tailed). Those with a high PAS score tended to show less negative priming.

Gender has been suggested to be a moderator variable in experimental studies of schizotypy (Claridge et al., 1991). Some studies have reported gender effects in relation to schizotypy in that experimental measures in relation to schizotypy are shown more reliably in male subjects (e.g. Beech, 1987; Claridge et al., 1991). Therefore, we examined the men and women separately as they were selected on the bases of different means of the PAS scores. We expected a greater difference between schizotypic and control subjects in the male population than in the female population, based on past observations (Beech, 1987; Claridge et al., 1991).

Our results did not confirm Claridge et al.'s results. There was a significant difference between the high PAS ($n = 16$) and low PAS women ($n = 15$) in both the raw [$F(1,29) = 4.24$, $P < 0.049$] and the percentage in negative prime scores [$F(1,29) = 5.08$, $P < 0.032$]. High PAS (schizotypic) female subjects showed -1.3 msec ($SE = 7.4$) raw negative prime score, which is close to *no* negative priming. Low PAS (control) female subjects showed -20.8 msec ($SE = 5.9$) raw

negative prime score. The percentage in the negative prime score for the high PAS (schizotypic) female subjects was 0.0% (SE = 1.7) and that for the low PAS (control) female subjects was -5.6% (SE = 1.7).

The pattern of results from the men was surprisingly different. There was no difference between the high PAS (schizotypic) male subjects ($n=15$) and low PAS (control) male subjects ($n=10$), whether we examined the raw negative prime scores [$F(1,23)=0.03$, $P>0.85$] or the percentage in negative prime scores [$F(1,23)=0.004$, $P>0.95$]. The mean raw negative prime score for the male high PAS (schizotypic) subjects was -11.5msec (SE = 7.5) and that for the male low PAS (control) subjects was -9.4msec (SE = 7.9). The percentage in the negative prime score was almost identical for both groups: -2.4% (SE = 1.5) for the schizotypes and -2.5% (SE = 2.1) for the controls. Hence, the male high PAS (schizotypic) subjects are virtually indistinguishable from the male low PAS (control) subjects in the extent of negative priming (see Table 4).

Discussion

We found a smaller negative priming effect in the schizotypic (high PAS) group than in the control subjects (low PAS). Among men, there was no difference between the high and low PAS subjects in the extent of negative priming, but in women there was a difference between the schizotypic and the control subjects.

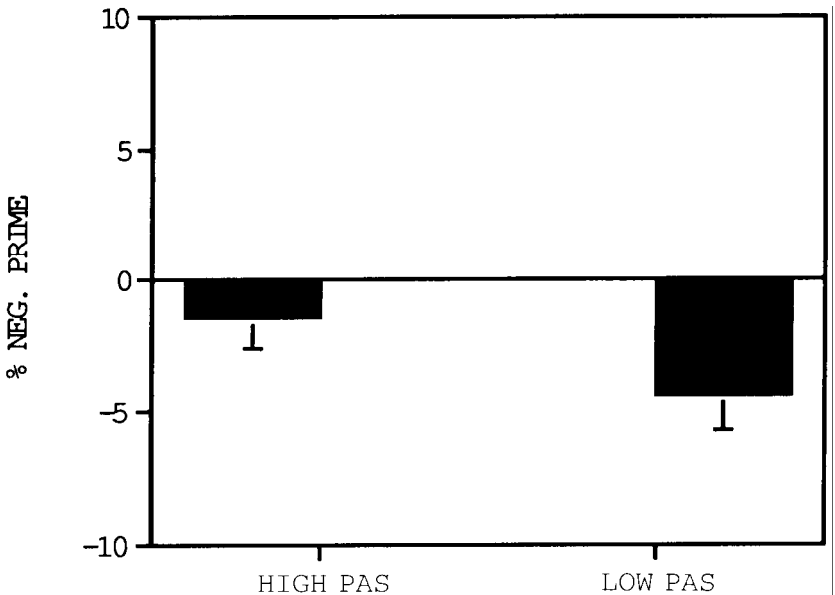


FIG. 5. Percentage of negative prime scores of high PAS (schizotypic) and low PAS (control) groups.

TABLE 4
Negative Prime Effect in Men (M) and Women (W)

	<i>High PAS (Schizotypes)</i> (<i>n</i> = 30)		<i>Low PAS (Controls)</i> (<i>n</i> = 25)	
	<i>M</i> (<i>n</i> = 15)	<i>W</i> (<i>n</i> = 10)	<i>M</i> (<i>n</i> = 10)	<i>W</i> (<i>n</i> = 15)
Raw negative prime	-11.5 (SE = 7.5)	-3.1 (SE = 7.6)	-9.4 (SE = 7.9)	-20.8 (SE = 5.9)
Negative prime score (%)	-2.4 (SE = 1.5)	-0.5 (SE = 1.8)	-2.5 (SE = 2.1)	-5.6 (SE = 1.7)

These results partially support previous studies that have demonstrated an absence of negative priming in schizotypal subjects (e.g. Beech & Claridge, 1987). Our results differed from Beech and Claridge's study in that we did not find a reduction of negative priming in the male schizotypic (high PAS) subjects.

It should be noted that we selected our schizotypic group using a fallible psychometric indicator with imperfect validity (i.e. the PAS) that probably generated an admixture of compensated schizotypes and an unknown proportion of false positives. Thus, it is possible that we identified individuals representing a diversity of schizotypic liability, a diversity that was associated with a range of negative priming scores.

The negative priming task may be sensitive to gender. Contrary to Beech (1987) and Claridge et al. (1991), we found that the negative priming effect in relation to schizotypy is better demonstrated in women than in men. We were surprised to find that the male control subjects showed very little negative priming effect compared with the female control subjects in this study, who, in turn, did not differ from the control subjects from Studies 1 and 2. This observation may be a reflection of the psychometric scale that we chose to detect schizotypic characteristics. Women score higher than men on the PAS and other scales that assess "positive" schizotypic features, whereas women tend to score lower than men on scales that assess "negative" symptomatology (Muntaner, Garcia-Sevilla, Fernandez, & Torrubia, 1988; Raine, 1992). It is possible that the PAS may detect schizotypic characteristics in female populations better than in male populations and, as result, we may not have been able to separate clearly the schizotypic men from the control subjects on the basis of their scores on the PAS, but this study cannot adequately address this question.

An additional point concerns the laterality and language effects in negative priming tasks. Most of the negative priming tasks that have been used in schizotypy research require linguistic-processing and articulatory responses (i.e. left hemisphere-dependent in right-handed males). But language seems to be

less lateralised in women than in men (McGlone, 1980), and in high schizotypes than in control subjects (Broks, 1984; Rawlings & Claridge, 1984). Therefore, Beech's (1987) and Claridge et al.'s (1991) findings may not be strictly comparable to our study, as we chose the spatial negative priming task specifically to minimise any linguistic demands on the subjects. It is interesting to note that with verbal negative priming tasks, the differences between schizotypes and controls are better demonstrated in male subjects than in women; with the spatial negative priming task, the converse is true.

Finally, we pooled all the control subjects from the three studies and examined the gender effect. There was a significant effect of gender on both the raw [$F(1,80) = 5.8, P < 0.02$] and the percentage [$F(1,80) = 5.9, P < 0.02$] negative prime scores. Overall, women controls showed about twice as much negative priming effect as men in the spatial negative priming task. Although we cannot offer a definite conclusion concerning the gender effect in negative priming and schizotypy, it would be prudent to view gender as a potential source of individual differences in schizotypy research.

GENERAL DISCUSSION

Some clinical symptoms of schizophrenia have long been associated with a breakdown of the selective and inhibitory mechanisms of attention (e.g. Gray et al., 1991; Nestor et al., 1992; Nuechterlein & Dawson, 1984). In this project, we focused on a simple task of spatial selective attention and assessed inhibition associated with the location of the ignored distractor stimulus. Successful performance on this task was seen in the increased reaction time to locate the target, if it appeared in the exact position that had been previously occupied by the ignored stimulus (i.e. presence of a negative priming effect).

We found that negative priming was abolished in acute schizophrenia inpatients but it was present in chronic schizophrenia outpatients in remission. This difference may be due to the effect of neuroleptics in the outpatients who benefit from a reduction in positive symptoms and perhaps some normalisation of the limbic system function. The acute inpatients were likely to be in a hyperdopaminergic state during testing, in spite of the recent (less than a week) neuroleptic treatment, and as result may have failed to show negative priming. Future research is needed to examine the relationship between specific symptom characteristics and reduced inhibition in detail. We are in the process of documenting detailed aspects of positive and negative symptoms in acute patients, as they are admitted to the hospital, three weeks after the admission and three months later, in relation to negative priming and neuropsychological profiles.

We also found that the relatives of schizophrenia patients and female schizotypic subjects were disinhibited on the spatial negative priming task, in the absence of medication. Because negative priming is abolished by

dopamine agonists and normalised by dopamine antagonists, it has been suggested that the effects may be primarily mediated by the limbic system (e.g. Gray et al., 1991; Venables, 1992; Weiner et al., 1990). Indeed, there is growing evidence implicating the presence of limbic system abnormalities in schizophrenia from both postmortem studies (e.g. Benes, 1989; Bogerts, Meertz, & Schonfeldt-Bausch, 1985; Falkai & Bogerts, 1986) and *in vivo* (e.g. Shenton et al., 1992).

One of the limitations of our studies is that we did not document symptom scores for the patient population in relation to their attentional inhibition. Another limitation might be that the relatives were not assessed for any DSM axis 2 disorders. Future studies with larger sample sizes should address these issues in order to further our understanding of cognitive deficits in relation in clinical symptoms of schizophrenia.

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