THE QUARTERLY JOURNAL OF EXPERIMENTAL PSYCHOLOGY
2006, 59 (7), 1224–1236

Psychology Press
Taylor & Francis Group

Normal auditory negative priming in schizophrenic patients

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Performance of 28 schizophrenic patients and 28 matched controls was compared in an auditory priming task. A large auditory negative priming effect was obtained for the patients as well as for the control group, and the size of the negative priming effect was approximately the same for both groups. Under the same conditions, positive or repetition priming for the patients was enhanced compared to that of the control group. The present findings from an auditory priming task are consistent with a growing body of evidence from the visual domain showing normal rather than reduced or eliminated negative priming in schizophrenic patients.

The negative priming phenomenon manifests itself in slowed-down or more error-prone reactions to recently ignored stimuli compared to those for control stimuli that are unrelated to the previous stimuli (for reviews, see Fox, 1995; May, Kane, & Hasher, 1995; Neill, Valdes, & Terry, 1995; Tipper, 2001). Several models are currently available to explain the negative priming phenomenon. Of these models, the so-called distractor inhibition model has a special status not only because it is historically the oldest model that explains this phenomenon but also because it suggests that the negative priming paradigm may be an appropriate task for testing predictions of loss-of-inhibition theories of the changes in cognitive functioning induced by schizophrenia, ageing, and other conditions. According to the version of this model proposed by Tipper (1985; see also Dalrymple-Alford & Budayr, 1966; Houghton & Tipper, 1994; Neill, 1977), negative priming reflects the operation of an inhibitory attentional selection mechanism that prevents access of recently ignored objects to overt responses by suppressing competing distractor input. This inhibitory mechanism enables more efficient responding to the current target under normal circumstances, but causes a delay in responding when, as in a negative priming laboratory task, the previously ignored (and, hence, supposedly suppressed) distractor becomes the new target.

This attentional explanation of the negative priming phenomenon is interesting for schizophrenia research because attentional impairment is one of the fundamental cognitive deficits associated with schizophrenia. In particular, increased distractibility in the presence of irrelevant information has been widely attributed to disrupted mechanisms responsible for the direction and control of attention—that is, to the impaired

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The research reported in this article was supported by a grant from the Deutsche Forschungsgemeinschaft (Bu 945/2–1).

http://www.psypress.com/qjep
DOI:10.1080/02724980543000114

functioning of inhibitory attentional mechanisms (e.g., Frith, 1979; McGhie, 1977; McGhie & Chapman, 1961). With respect to the negative priming paradigm, the assumption of impaired inhibitory attentional mechanisms in schizophrenia predicts less or no slow-down of the reactions of schizophrenic patients to previously ignored distractors compared to those of normal controls, provided that the distractor inhibition model is valid. The negative priming task itself appears particularly useful for testing the loss-of-inhibition assumption in schizophrenia because a cognitive deficit is expressed in improved performance (no reaction slow-down), rather than impoverished performance, which is usually explained by a generalized cognitive deficit rather than a specific inhibitory attentional impairment.

Indeed, Beech, Powell, McWilliam, and Claridge (1989) started off such a research programme and reported reduced negative priming in a group of schizophrenic patients compared to that in a control group. This finding was interpreted as being consistent with the assumption that schizophrenia implies a reduction in the ability of the cognitive system to inhibit and suppress irrelevant information.

However, subsequent research has produced different and somewhat inconsistent results. For instance, Laplante, Everett, and Thomas (1992) did not find reduced negative priming in schizophrenic patients relative to depressive patients and healthy control participants.[1] Parallel results have recently been reported in several studies (Baving, Wagner, Cohen, & Rockstroh, 2001; Moritz, Jacobsen, Mersmann, Kloss, & Andresen, 2000; Roesch-Ely, Spitzer, & Weisbrod, 2003; Wagner, Loeper, Cohen, & Rockstroh, in press). Other researchers also found preserved negative priming, but only in subgroups of schizophrenic patients. For instance, normal negative priming has been reported for chronic outpatients but not for acutely psychotic schizophrenic inpatients (S. Park, Lenzenweger, Püschel, & Holzman, 1996; S. Park, Püschel, Sauter, Rentsch, & Hell, 2002; see also Salo, Henik, Nordahl, & Robertson, 2002), and for medicated but not for unmedicated schizophrenic patients (Salo, Robertson, & Nordahl, 1996; Salo, Robertson, Nordahl, & Kraft, 1997), as well as conversely for unmedicated but not for medicated schizophrenic patients (David, 1995). In contrast, Moritz et al. (2001) reported that symptomatology and neuroleptic medication did not moderate the size of the negative priming effect. Interestingly, they noted that procedural details may determine whether or not negative priming is observed in schizophrenic patients. In their study, control participants and schizophrenic patients did not differ in terms of negative priming when the prime presentation duration was 250 ms, but schizophrenic patients failed to show any priming when primes were presented for 100 ms and were followed by a pattern mask, as in Beech et al. (1989). Moritz et al. argued that previous findings of no or reduced negative priming in schizophrenic patients may have been due to a procedural artifact—that is, the very short presentation durations combined with pattern masking, which simply may have impaired the patients' processing of the prime distractor.

Finally, Hoenig, Hochrein, Müller, and Wagner (2002) reported normal levels of identity negative priming in schizophrenic patients in a task that required selection by stimulus identity, but the patients showed reduced spatial negative priming when spatial position was incidental and not task relevant. However, MacQueen, Galway,

[1] The reported negative priming effects were descriptively large for schizophrenic patients classified as negative (255 ms, $N = 10$) or positive (100 ms, $N = 8$), for depressive patients (228 ms, $N = 21$), and for control participants (69 ms, $N = 35$). An analysis of variance (ANOVA) showed that the size of the effect did not differ significantly between groups. However, when the authors tested whether the effect was significant within a particular group, statistically significant results emerged only for the depressive and control participants. The problem with these group-specific tests and the conclusions drawn from them is that the sample size and, hence, the statistical power were so much smaller in the schizophrenic than in the other groups that results favouring the statistical null hypothesis ("no negative priming") are highly expected on purely methodological grounds and must not be interpreted as indicating no negative priming in the schizophrenic groups.

Goldberg, and Tipper (2003) reported reduced spatial negative priming of schizophrenic patients compared to control participants when spatial position was relevant and not incidental.

Given this pattern of findings, the main purpose of the present research was to add further evidence to the body of available findings on the relation between negative priming and schizophrenia. In particular, we wanted to extend the range of available findings from the visual to the auditory domain. While this empirical extension into a new sensory modality may be regarded as interesting in its own right, another purpose for moving into the auditory modality was to provide for a potentially more sensitive test of the hypothesis of reduced negative priming in schizophrenia. Specifically, Banks, Roberts, and Ciranni (1995) argued that attending to a tone while avoiding auditory distraction must operate almost entirely by internal processing mechanisms. Visual selection, in contrast, may be supported by peripheral mechanisms such as eye or head movements. Auditory selection by internal distractor inhibition may thus be regarded as much more demanding, leaving little or no room for compensating for any inhibitory deficits. As a consequence, reductions in the efficiency of inhibitory mechanisms may be more clearly apparent in the auditory than in the visual domain, so that group differences in negative priming may be expected to be stronger and more reliable in the auditory than in the visual domain. Furthermore, given the widespread occurrence of auditory hallucinations in schizophrenia, the auditory domain seemed especially relevant for the investigation of attentional mechanisms in this patient group.

EXPERIMENT

Method

Pilot study

In the task used here, participants heard pairs of tones displayed via headphones. One tone was presented to each ear. A click indicated the ear that had to be attended. Participants were asked to classify, by an appropriate key press, the attended tone as originating from an instrument or an animal. Each trial consisted of a prime pair and a probe pair of stimuli. Trials were separated by a pause.

A preliminary version of this task using experimental parameters that had previously been found appropriate for use with elderly participants (Buchner & Mayr, 2004) was implemented in a pilot study to check its adequacy for a clinical sample with diagnoses of schizophrenia. In this pilot experiment, the participants' task was to classify the attended tones as belonging to either the "wind instrument" (flute, trumpet, and saxophone) or the "string instrument" (piano, balalaika, and pizzicato violin) category by responding with an appropriate key press. Each trial consisted of a prime pair and a probe pair of stimuli, with a cue–target interval of 500 ms and an interval of 500 ms between reaction to prime and the following probe cue (click).

The main result of the pilot study was that the clinical sample had great difficulties performing a task that had been shown to be clearly feasible for elderly participants. In general, the patients produced huge numbers of errors. What is more, of the 46 clinical participants, 12 prematurely terminated the experiment because they felt overtaxed. For the experiment reported here, therefore, stimuli were selected from categories that were easier to distinguish (instruments and animal sounds), and the temporal parameters between experimental events were modified to ensure that patients could comply with the task without being overtaxed, so as to avoid serious and unwanted selection bias. It also seemed important to take these steps in order to avoid possible perceptual problems that might prevent the patients from fully processing the prime distractors, since this is thought to be a variable that could explain the observation of no or reduced negative priming in schizophrenic patients (cf. Moritz et al., 2001).

Participants

Participants for the clinical group were 28 psychiatric patients (10 female) who were recruited from

three different psychiatric hospitals and met the diagnostic criteria for schizophrenia according to ICD-10 (Dilling, Mombour, & Schmidt, 1993). Six of the patients were outpatients.[2] Diagnoses were established by experienced clinicians at the hospitals. Furthermore, one of the authors and the responsible ward psychiatrists and psychologists carefully screened all available clinical records in order to exclude patients with previous or concurrent diagnoses that were incompatible with an unambiguous diagnosis of schizophrenia. For an unambiguous diagnosis of schizophrenia, only patients without double psychiatric diagnoses, schizoaffective disorders, or potentially drug-induced psychoses were accepted. On average the first known diagnosis of schizophrenia had occurred 5.9 years (minimum, 0.5 years; maximum, 26 years; $SD = 5.7$) before the time at which the experiment was conducted. Thus, the sample of patients had the advantage of being relatively heterogeneous with respect to the duration of their illness, rendering it less likely that the findings of this experiment could be due to the specific characteristics of a highly selective group of patients. The responsible clinicians were requested to rate the patients on the Brief Psychiatric Rating Scale's (BPRS, Overall & Gorham, 1962) German version (Collegium Internationale Psychiatriae Scalarum, 1996) based on their daily interaction with patients and previous clinical interviews. The total sum of the 18 individual ratings can be interpreted as an approximate total pathology score. For our clinical sample, the BPRS total score mean was 48 ($SD = 9.9$). All patients received neuroleptic medication. Chlorpromazine equivalents were calculated for 27 patients according to Jahn and Mussgay (1989) for conventional antipsychotic medication and according to Woods (2003) for newer atypical antipsychotic medication. The mean daily dosage in chlorpromazine units was 449.6 (minimum, 41; maximum, 1,949), and the distribution of dosages was positively skewed. A total of 10 patients also received other medication (tranquillizers, sedatives, or antidepressants), and 6 were also taking anticholinergic medication. Controls were 28 healthy participants (10 female) recruited mainly from the university staff and without a history of psychiatric illness. Controls and schizophrenia participants were matched for age, gender, and years of education as a measure of overall intellectual level, premorbid in the case of the patients (see Table 1). None of the participants used hearing aids, and controls and patients did not differ with respect to their self-reported hearing ability (using a 3-point scale with "above average", "normal", and "below average"), $z = 1.38$, $p > .17$. None of the patients and control persons was older than 45 years so that possible confusion with age-related effects on the size of the negative priming phenomenon (cf. Verhaeghen & De Meersman, 1998) could be excluded, although in retrospect this additional control may have been unnecessary given that a more recent meta-analysis suggests that there is no age-related difference in negative priming (Gamboz, Russo, & Fox, 2002).

All participants were paid for their participation and gave informed consent previous to their participation in the experiment. They were also explicitly informed that they could terminate the experiment at any time if they so wished.

Materials

The stimuli were six digitized tones, which could be identified and categorized easily and unambiguously as "musical instruments" (piano, guitar, and clarinet) or "animal sounds" (duck, lamb, and frog). Each tone was 300 ms long, complete with attack and decay. The participants heard the tones over earphones that were fitted with high-isolation hearing protection covers and plugged directly into an Apple PowerBook computer.

[2] Note that removing the outpatients from the patient group did not change any of the statistical conclusions reported in this article, except for one secondary result pertaining to the supplementary analysis of the error data: The overall difference in errors between ignored repetition (IR) and ignored repetition control (IRC) trials for the entire sample of schizophrenic patients and healthy control participants was just barely statistically significant when the outpatients were included in the sample, and it narrowly missed the preset level of statistical significance when the six outpatients were removed (see the Results section).

Table 1. *Data characterizing the matched patient and control groups*

	Age				*Gender*		*Years of education*				*Hearing ability*			
	Min	*Mean*	*Max*	*SD*	*No. female*	*No. male*	*Min*	*Mean*	*Max*	*SD*	*Min*	*Mean*	*Max*	*SD*
Patients	19	32.3	45	7.7	10	18	8	10.1	13	1.7	1	2.1	3	0.5
Controls	18	32.2	45	7.3	10	18	9	10.1	13	1.3	1	1.9	3	0.5

Trials consisted of a prime and a probe pair of stimuli. There were four basic types of trial: ignored repetition trials (IR), ignored repetition control trials (IRC), attended repetition trials (AR), and attended repetition control trials (ARC).

Each IR trial corresponded to one IRC trial in terms of the tone configuration except for the ignored prime, which differed but was taken from the same category. Similarly, each AR trial corresponded to one ARC trial in terms of the tone configuration except for the attended prime, which differed but was taken from the same category. This is illustrated in Table 2. If negative, or positive, priming occurs with this arrangement of corresponding trials, then it must be due to processes operating at the level of the stimulus identity of the tone and cannot be due to processes related to the response category. To illustrate, the guitar tone is ignored in the IR prime pair example given in Table 2, and the piano tone is ignored in the corresponding IRC prime pair. Both belong to the same category of instruments. If the reaction to the guitar tone was slowed in the IR relative to the IRC probe pair, then the processes causing the slow-down must operate on the stimulus identity of the ignored prime, because that is the only difference between the IR and its corresponding IRC trial. The same holds for the AR and their corresponding ARC trials.

For each prime and each probe pair of stimuli, only combinations of tones from the two categories (animal sound or musical instrument) were used in order to necessitate real selection for each category. In other words, combinations of stimuli from one category were never presented together. Combining all six different tones in the way illustrated in Table 2 yields 72 unique trials of each type: IR, IRC, AR, and ARC, resulting in a total of 288 different trials. Of this original pool of stimuli, a subset of 144 combinations (36 per trial type) was selected in such a way that the absolute frequencies of the different tones were equal overall as well as within each trial type. Furthermore, the frequencies of the combinations of attended and ignored tones were identical overall and within trial types.

An important difference between IR and IRC trials on the one side and AR and ARC trials on the other is that the required response always changed in the former and remained the same in the latter types of trial. For the experimental procedure, this has the desired consequence that the required reaction to the attended probe could not

Table 2. *Examples of stimulus configurations*

	Ignored repetition		*Ignored repetition control*		*Attended repetition*		*Attended repetition control*	
	Attended ear	*Ignored ear*	*Attended ear*	*Ignored ear*	*Attended ear*	*Ignored ear*	*Attended ear*	*Ignored ear*
Prime	Frog	Guitar	Frog	Piano	Frog	Guitar	Lamb	Guitar
Probe	Guitar	Lamb	Guitar	Lamb	Frog	Piano	Frog	Piano

be predicted from the prime. For the data analysis, the consequence is that IR trials can only be compared to IRC trials, and AR trials can only be compared to ARC trials.

Procedure

The task was introduced as a tone categorization task. Extensive practice was provided in order to familiarize the participants with the task. They were first introduced to the tones and the reaction keys by hearing each tone individually, and by reacting to single tones for each category presented first to one ear, and then to the other. A metronome click indicated the ear at which the tone would be presented. Following this, participants were asked to react to 24 randomly presented single tones. The last part of the practice session introduced the actual experimental task. Here two different tones were presented to the right and left ear, and the preceding 20-ms metronome click now indicated which ear was to be attended to (determined at random). Participants reacted to consecutive pairs of stimuli—that is, a prime pair and a probe pair. After a 1,000-ms cue–target interval, a pair of tones was presented, one to the left and one to the right ear. The interval between prime reaction and probe cue was 500 ms. Participants reacted to the attended tone by pressing, as quickly as possible, the "instrument" or the "animal" key, depending on the category to which the tone belonged.

The experiment itself consisted of 144 trials, each of which was composed of a prime and a probe pair of tones as in the final phase of the practice session. The participants initiated each trial at their own pace. A trial began with a brief countdown followed by the click indicating the ear on which the to-be-attended tone would be presented. The instructions emphasized correctness, but reactions were also to be made as quickly as possible. The interval between participants' reactions and the click preceding the probe pair of tones was 500 ms. The probe tone pair was presented with the same temporal parameters as those for the prime tone pair.

The to-be-attended ear for the prime pair of stimuli was always selected at random. The to-be-attended probe tone was presented either to the same ear as the prime or to the other ear. The relation of the to-be-attended prime and probe location was varied in blocks. The blockwise presentation was a consequence of the pilot study that had shown that schizophrenic patients find it difficult to cope with attended primes and probes appearing unpredictably at the same or at different ears. One half of the participants in each group first received the block of randomly ordered trials in which the attended primes and probes were presented to the same ear, followed by the remaining trials in which the attended primes and probes were presented to different ears. For the other half or the participants in each group this sequence of blocks was reversed. The ordering of blocks was counterbalanced over subjects. Within each block, the ear at which the to-be-attended prime was presented was thus randomly selected but predicted the ear of the to-be-attended probe. Within each trial type, the attended primes and probes were presented to the same ear on 18 trials, whereas attended primes and probes were presented to different ears for the other 18 trials. This implies that on IR trials, the ignored prime changed location in the former case, but did not change location in the latter case.

Prime or probe reactions faster than 100 ms and slower than 4,000 ms were counted as invalid, and the entire trial was repeated. After each trial, participants were given visual and acoustic feedback on the correctness of their reactions to the prime and probe (visual feedback indicated whether the reactions to prime and probe had been correct or false, and acoustic feedback was "very good" for two correct responses, "almost" for one correct response, and "too bad" for two incorrect responses). A summary feedback about error percentages and average reaction times was provided after every 12th trial, and to further motivate the participants they were informed of how they had performed compared to the previous block of 12 trials. Once again, after the summary feedback, participants continued the experiment at their own discretion. On completion of the experiment, participants were given the

opportunity to be informed about the purpose of the experiment.

Design

The design consisted of two 2 × 2 × 2 subdesigns. The ignored repetition subdesign comprised IR versus IRC trials as the levels of the within-subject priming factor and same versus different presentation sides of the attended prime and probe as the levels of the within-subject presentation side factor. The attended repetition subdesign differed by having AR versus ARC trials as the levels of the priming factor. Both subdesigns also comprised the two levels, patients versus matched control persons, of the quasi-experimental between-subjects variable. The primary dependent variables were participants' reaction times, but error probabilities were also analysed.

A power analysis with respect to the negative (or positive) priming effect showed that given a sample size of $n = 28$ in each of the two groups (i.e., $N = 56$), and $\alpha = .05$, effects of size $d_z = .45$ (cf. Cohen, 1977) could be detected with a probability of $(1-\beta) = .95$ (the sample negative priming effect actually turned out to be much larger than this assumed population value, see below).[3] Further, with $n_{\text{patient}} = n_{\text{control}} = 28$, and $\alpha = .05$, effects of size $d = .80$ ("large" effects in terms of the conventions suggested by Cohen, 1977) could be detected with a probability of $(1-\beta) = .91$ for the one-tailed test of whether negative priming is smaller in patients than in controls. Assuming a population effect size of $d = .80$ for the difference in negative priming between patients and controls seemed conservative and reasonable given that the sample effect sizes in studies that reported an overall difference between these groups were typically much larger and reached $\hat{d} = 2.11$ for the data reported by Beech et al. (1989) and $\hat{d} = 0.91$ for the data reported by MacQueen et al. (2003).

The level of α was set to .05 for all analyses reported in this article.

Results

Probe reaction times were evaluated only for trials in which both the probe and the prime reactions were correct. The means of participants' average reaction times and the corresponding error rates are presented in the upper and lower panels of Figure 1, respectively.

Patients reacted more slowly than controls. Reaction times on IR trials were longer than reaction times on IRC trials, and they were longer when the attended prime was presented to a different ear from that of the attended probe. Corresponding to these descriptive data, a 2 × 2 × 2 analysis of variance (ANOVA) with participant group (schizophrenic patients vs. controls) as between-subjects variable, and priming (IR vs. IRC) as well as presentation side (same vs. different) as within-subject variables showed significant main effects of patient group, $F(1, 54) = 11.22$, $p < .001$, of priming, $F(1, 54) = 64.71$, $p < .001$, and of presentation side, $F(1, 54) = 68.17$, $p < .001$. The critical interaction of participant group and priming was not significant, $F(1, 54) = 0.22$, $p > .64$, as were all other interactions, Fs$(1, 54) < 1.90$, $p > .17$. Follow-up t tests showed that negative priming was significant for both the schizophrenic patients, $t(27) = 5.68$, $p < .001$, and the control participants, $t(27) = 5.73$, $p < .001$. In terms of the reaction times, the mean and standard deviation of the negative priming effect was somewhat larger for the patients ($M = 88$ ms, $SD = 82$ ms) than for the controls ($M = 78$ ms, $SD = 72$ ms). In terms of standardized effect sizes, the sample effects for patients and control participants were quite large and very similar, $\hat{d}_z = 1.07$ and $\hat{d}_z = 1.08$, for patients and control participants, respectively. This very close match across groups can be considered strong evidence in favour of the hypothesis that the negative priming effect does not differ between groups.

The error data were generally consistent with the reaction time data although only the

[3] The power calculations were conducted using the G·Power program (Buchner, Faul, & Erdfelder, 1996; Erdfelder, Faul, & Buchner, 1996).

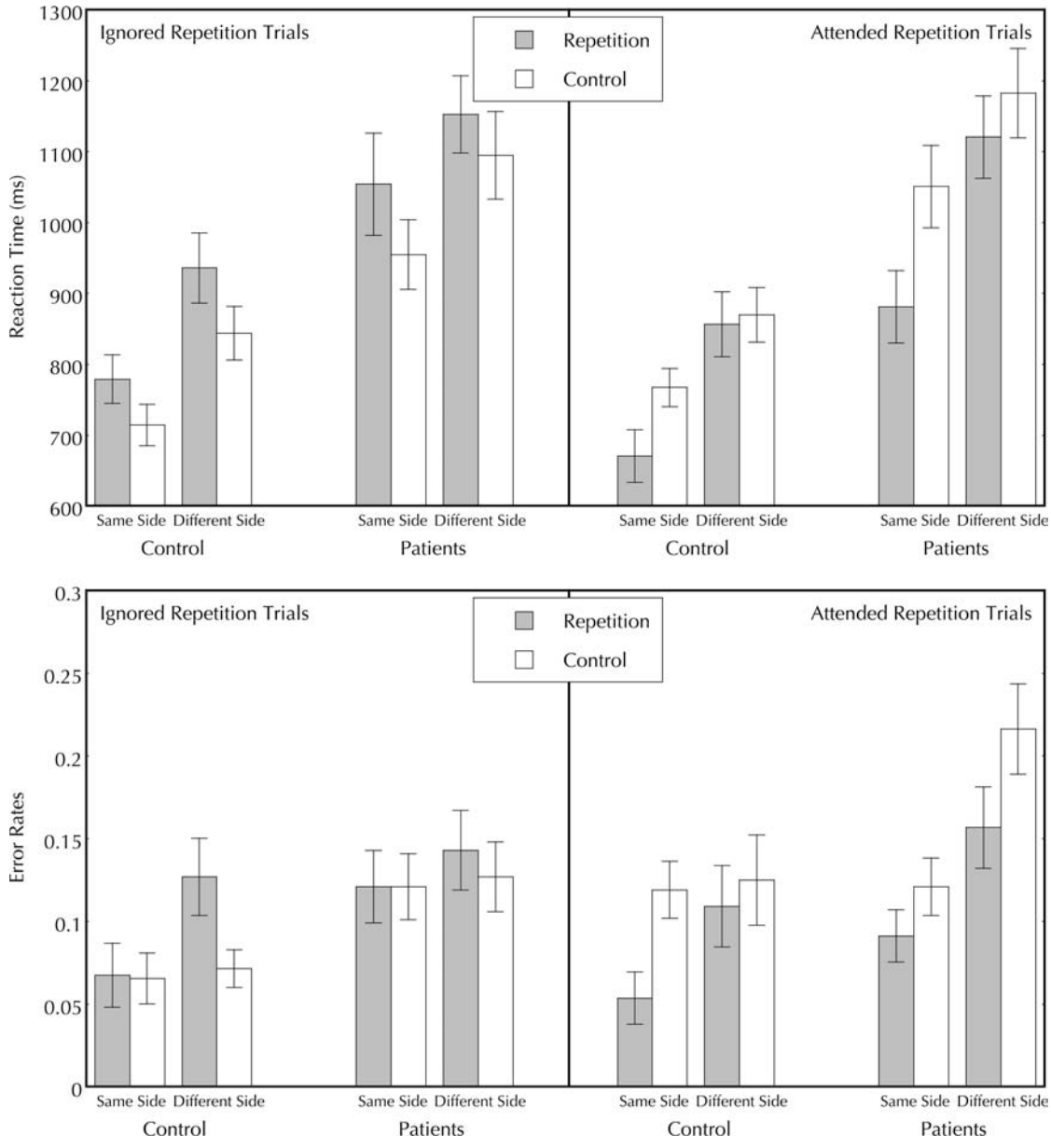

Figure 1. *Reaction times (upper panel) and error rates (lower panel) as a function of trial type and presentation side for schizophrenic patients and control participants. The error bars depict the standard errors of the means.*

difference in errors between IR and IRC trials was statistically significant, $F(1, 54) = 4.45$, $p = .04$.

Turning to the positive priming subdesign, we find that reactions on AR trials were faster than reactions on ARC trials. Again patients showed slower reaction times than the control persons, and reaction times were longer when the attended primes and probes were presented to different ears.

A 2 × 2 × 2 ANOVA with participant group (patients vs. control participants) as between-subjects variable, and priming (AR vs. ARC) as well as presentation side (same vs. different) as within-subject variables showed significant main effects of participant group, $F(1, 54) = 14.27$, $p < .001$, of priming, $F(1, 54) = 39.55$, $p < .001$, and of presentation side, $F(1, 54) = 90.35$, $p < .001$. There was also an interaction between the participant group and priming, $F(1, 54) = 4.66$, $p < .04$, reflecting the fact that positive priming was larger for schizophrenic patients ($M = 113$ ms, $SD = 104$ ms, $\hat{d}_z = 1.09$) than for control participants ($M = 55$ ms, $SD = 95$ ms, $\hat{d}_z = 0.58$); for both groups the priming scores were significantly different from zero, $t(27)$s > 3.06, $p < .01$. The interaction between priming and presentation side was also significant, $F(1, 54) = 26.71$, $p < .001$, indicating that positive priming was larger when attended primes and probes were presented to the same ear ($M = 133$ ms, $SD = 105$ ms) than when they were presented to different ears ($M = 35$ ms, $SD = 142$ ms). No other interactions were significant, $F(1, 54) < 1.37$, $p > .24$.

With respect to the error data, there were significant main effects of priming, $F(1, 54) = 16.14$, $p < .001$, and of presentation side, $F(1, 54) = 21.55$, $p < .001$. An interaction between participant group and presentation side, $F(1, 54) = 4.30$, $p < .05$, reflected that the presentation side effect was larger for schizophrenic patients ($M = .06$, $SD = .20$) than for control participants ($M = .03$, $SD = .14$).

Discussion

The main result of the present experiment is that schizophrenic patients and control persons showed the same amount of negative priming in this auditory priming task, as is most obvious from the very close match of these groups in terms of the standardized negative priming effect. Considering that reductions in the efficiency of inhibitory mechanisms may be more clearly apparent in the auditory than in the visual domain (Banks et al., 1995), these results represent important conceptual replications and extensions of previous findings of normal visual negative priming in schizophrenic patients (e.g., Baving et al., 2001; Moritz et al., 2001; Roesch-Ely et al., 2003; Wagner et al., in press). If we also consider that the original demonstration of reduced negative priming in schizophrenic patients (Beech et al., 1989) seems to have been confounded with atypically impoverished presentation conditions (cf. Moritz et al., 2001), then the conclusion suggests itself that attentional deficits associated with schizophrenia may not after all be reflected in a paradoxically "faster" performance on ignored repetition trials. Instead, schizophrenic patients seem to show a slow-down in reactions to previously ignored distractors in the negative priming paradigm, which is comparable to that found with healthy participants. Does this necessarily have to be counted as evidence against the assumption of an impaired functioning of inhibitory attentional mechanisms in schizophrenia? It does not, for several reasons.

First, according to the inhibition model of negative priming (Dalrymple-Alford & Budayr, 1966; Houghton & Tipper, 1994; Neill, 1977) the characteristic slow-down when reacting to a probe that was previously ignored is caused by the need to overcome the inhibition imposed during prime selection. Therefore, one possible conclusion is that the kinds of inhibitory processes thought to be impaired in schizophrenia ("a defect in the mechanism that controls and limits the contents of consciousness", cf. Frith, 1979, p. 225) are actually different from those involved in the suppression of perceptual distractors in negative priming tasks. Indeed, it may be an oversimplification to conceive of only one homogeneous type of inhibitory process.

Second, one could assume that negative priming tasks do not really measure inhibition at all, and indeed there are alternative explanations of the basic negative priming phenomenon of which feature mismatch, temporal discrimination, and episodic retrieval appear to be most relevant (see Fox, 1995; May et al., 1995; Milliken, Joordens, Merikle, & Seiffert, 1998; Neill et al., 1995). For instance, J. Park and Kanwisher (1994) assumed

that negative priming was caused by a feature mismatch between prime and probe—that is, by a change in the bindings of symbol identities to locations between the prime and probe. In the present experiments, bindings of tone identities (e.g., the piano tone) to locations (e.g., the left ear) changed when, on ignored repetition trials, the prime distractor presented to one ear was also the probe target subsequently presented to the other ear (mismatch condition). In contrast, the bindings stayed the same on those ignored repetition trials on which prime and probe distractors were presented to the same ear (no mismatch condition). Obviously, the size of the negative priming effect was completely unaffected by whether or not a mismatch occurred, adding the present study to the growing body of evidence suggesting that feature mismatching does not play an important role in negative priming (Baylis, Tipper, & Houghton, 1997; Buchner & Mayr, 2004; Buchner & Steffens, 2001; Buchner, Steffens, & Berry, 2000; Fuentes, Humphreys, Agis, Carmona, & Catena, 1998; Milliken, Tipper, & Weaver, 1994; Tipper, Weaver, & Houghton, 1994).

In contrast, the episodic retrieval model suggested by Neill and colleagues (Neill & Valdes, 1992; Neill et al., 1995; Neill, Valdes, Terry, & Gorfein, 1992) could explain the current data pattern. According to this model the probe target cues the retrieval of the perceptually similar prime display in which the distractor representation contains the information that no response was (to be) made to that stimulus. This remembered nonresponse information conflicts with the response requirements implied by the probe target and in that way slows down the probe responses. If such low-capacity, short-term retrieval as specified by this model were intact in schizophrenia, then we would expect the pattern of findings reported in the present experiment.[4]

Thus, the only conclusion we can come to from the present findings is that negative priming as an empirical phenomenon is not reduced in schizophrenia. We cannot reject, based on these findings, the assumption that attentional inhibitory functions are impaired in schizophrenia, either because there may be more than one type of inhibitory process, or because the characteristic slow-down to previously ignored stimuli in negative priming tasks actually may not measure inhibition. We have reason to believe that the latter alternative is quite unlikely, because existing evidence suggests that inhibitory processes are at least to some extent involved in the generation of the negative priming phenomenon for the present experimental task (Buchner & Steffens, 2001). However, it could well be that negative priming tasks do not yield the best measure of inhibition, or at least not an exclusive one.

Another result worth mentioning is that the positive priming effects were significantly larger in schizophrenic patients than in healthy controls. This replicates a data pattern first reported by Baving et al. (2001; see also Wagner et al., in press) for a situation with relatively long stimulus presentations in which both the prime and the

[4] The so-called temporal discrimination account (Milliken, Joordens, Merikle, & Seiffert, 1998) may, in principle, also explain the present data, but past experiments suggest that the discrimination account does not seem adequate to explain negative priming in the present paradigm, and it also does not add to the understanding of negative priming phenomena beyond what can already be explained by the distractor inhibition and episodic retrieval models (Buchner & Steffens, 2001; Buchner, Zabal, & Mayr, 2003) which is why it is not mentioned in the main text. Within this framework, two classes of process are assumed to occur when a probe response is generated. First, if the probe can be categorized as old, then automatic processes are likely to determine the response in that the prior action is simply retrieved and executed. This explains fast responses on attended repetition trials. Second, if the probe target is categorized as new so that prior learning is an inappropriate basis for action, complete perceptual analysis of the stimulus is necessary in order to arrive at a response. This takes more time than simply retrieving a recent response and corresponds to control trials. A third situation is given on ignored repetition trials. The target has been presented as part of the prime display, but it has not been fully attended. Thus, the probe target is somewhat familiar so that it cannot be quickly categorized as new, but it is also not quite familiar enough to be immediately categorized as old. According to Milliken et al. (1998), "this ambiguity in the temporal discrimination process for ignored repetition trials is presumed to underlie negative priming" (p. 210). There is no reason to assume that the ambiguity should be eliminated in schizophrenia, so that, in principle, this account could explain intact negative priming in schizophrenia.

probe stimuli required a response. As Baving et al. also noted, positive priming is normal or reduced with short stimulus presentations and without the requirement to respond to the primes. Thus, just as with negative priming, the relative size of the positive priming effect in schizophrenic patients seems to depend, among other things, on whether the conditions for perceiving the stimuli are impoverished or not.

The investigation of the causes of such positive priming or repetition effect has a long history. It dates back to the work of Bertelson (e.g., Bertelson, 1961, 1965) who already argued that the effect was primarily caused by processes located at the level of response mechanisms and not by a speedup of signal processing. More recently, Pashler and Baylis (1991) reaffirmed Bertelson's earlier reasoning and refined it, concluding that, first, the repetition effect was very stimulus specific and, second, that it was due to transient short-cuts in response selection for immediately repeated stimuli (presumably a direct translation from early stimulus representations all the way to fairly specific responses) and not due to faster perceptual processing of the repeated stimuli. Thus, schizophrenic patients seem to benefit more from such "short-cuts" than do control persons.

To summarize, then, the present findings from the auditory domain are consistent with a growing body of evidence from the visual domain showing normal rather than reduced or eliminated negative priming in schizophrenic patients when stimulus presentation parameters allow proper perceptual identification by the patients, while at the same time positive or repetition priming is enhanced.

Original manuscript received 20 December 2004
Accepted revision received 2 March 2005
PrEview proof published online 8 August 2005

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