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Repetition blindness in schizophrenic patients

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Abstract Repetition blindness is the failure to report the detection of repeated items in rapid visually presented lists. It can be explained in terms of either a processing limitation or an active inhibitory process. In two studies conducted in either English or German language we set out to induce repetition blindness under various conditions in a total of 47 control subjects and 30 schizophrenic patients. The patients displayed the phenomenon to at least the same degree as normal control subjects. These results render unlikely accounts of repetition blindness which involve processes known to be dysfunctional in schizophrenic patients. Moreover, the study provides an example of how the performance of schizophrenic patients can constrain theories of normal cognition.

Key words Inhibition · Perception · Psychopathology · Repetition blindness · Schizophrenia

Introduction

Experimental studies on the nature of perceptual and cognitive psychopathology found in schizophrenic patients have provided evidence for (a) a general lack of inhibitory processes (Spitzer et al. 1994; Tipper 1992) and (b) a perceptual deficit that appears to occur "early" in the time course of visual information processing (Braff 1993; Goldberg et al. 1991). In particular, it has been suggested that some form of "defective filtering" of sensory input accounts for illusions, delusional perceptions, delusions of control, and other types of "anomalous experiences" (cf. Maher and Spitzer 1993). Such defective filtering has been attributed to impaired inhibitory processes which in normal perception serves to block irrelevant information from further processing, thereby optimizing the performance of the perceptual system.

The phenomenon of repetition blindness consists of the failure to detect repeated items in a rapidly visually presented list (Kanwisher 1987; Park and Kanwisher 1994). For example, when subjects have to detect a repeated word in a list of words, each displayed for less than approximately 150 ms, they tend not to "see" the repeated word, even when it is separated by only one or two intervening words. Kanwisher (1987) has proposed that in repetition blindness, the identity and visual attributes (types) of the repeated item are successfully activated but fail to reach awareness because the repeated item is not encoded as a distinct event (token). This failure to individuate the repeated item could be explained either in terms of a simple processing limitation (e.g., Mozer 1991), or as the result of the active inhibition of token individuation for repeated items – a process which might serve the function of preventing incorrect inferences that multiple objects are present when a single stimulus object causes several recognition events (due to object movements, eye movements, or other intermittencies in the stimulus or the perceptual system).

Kanwisher (1987) attributes repetition blindness to early stages of object perception. This view has been questioned recently by two studies, which both support "off-line" processes as the cause of repetition blindness, i.e., processes occurring immediately after the perception of the targets. Fagot and Pashler (1995) hypothesized that repetition blindness is caused by memory output interference, whereas

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Whittlesea et al. (1995) claimed that repetition blindness is due to a report bias towards the second item in a sequence. Both studies share the view that repetition blindness is not a problem of perception at all, but instead of later postperceptual information processing. However, Hochhaus and Johnston (1996) produced evidence in favor of Kanwisher's (1987) perceptual account of repetition blindness by means of a single-frame experimental paradigm, which avoids problems of memory load and report bias.

The very nature of the phenomenon of repetition blindness as well as its theoretical import render it an ideal candidate for the general strategy of using measures and concepts derived from cognitive psychology for the study of psychopathology. This is the case for two reasons. Firstly, since repetition blindness represents a form of impairment in normal subjects, possibly caused by inhibitory processes, the absence of such inhibition in schizophrenic patients might cause them to perform better than normal subjects. Such "improvement-based" predictions are much stronger than "impairment-based" predictions can ever be (cf. Spitzer et al. 1993). Secondly, testing schizophrenic patients on a repetition blindness task not only may enhance psychopathological knowledge, but should also shed light on the causes of the phenomenon in normal subjects; i.e., if repetition blindness in normal subjects is caused by some kind of fundamental limitation in visual processing, the effect should be either unchanged or increased in schizophrenic patients. In contrast, if repetition blindness in normal subjects is caused by active inhibition, it should be reduced in schizophrenic patients whose inhibitory processes are impaired (Park et al. 1996; Spitzer et al. 1994); hence, the performance of schizophrenic patients can constrain theories of normal cognition.

In this paper we report two studies on repetition blindness, the first carried out, on a pilot basis, in the United States, and the second carried out in Germany. Since the experiment involves the presentation of words (and hence, may be influenced by idiosyncratic language effects), and since patient populations may differ in different countries, we welcomed the opportunity to collect data at two different locations.

Materials and methods

Subjects

In the first pilot study, we investigated repetition blindness in nine schizophrenic inpatients, drawn from the Dartmouth State Hospital (Dartmouth, N.H.) and the Bedford VA Hospital (Bedford, Mass.). Diagnosis of schiozphrenia was based on clinical presentation and chart review, and established by the chief psychiatrists on the wards (G.O. and T.M.). Eleven Harvard University students served as control subjects.

The second study was performed at the Psychiatric Hospital of the University of Heidelberg, Germany. Its aim was to replicate the results of the first pilot study in German language in a larger population of schizophrenic inpatients. As in study 1, diagnosis of schizophrenia was established by the chief psychiatrist on the wards (M.S.). Healthy normal controls were recruited from the Heidelberg area. Demographic data from control subjects and patients are presented in Table 1.

Table 1 Demographic data of the subjects participating in study 2

	Controls (n = 36)	Patients (n = 21)
Age (years)	31.5 ± 10.5 (18–58)	29.9 ± 6.9 (19–41)
Gender (m/f)	22/14	8/13
Handedness (r/l)	33/3	19/2
Education score (range 1–4)[a]	2.5 ± 0.85	2.33 ± 0.86

[a] 1: Elementary school (9 years); 2: intermediate level (10 years); 3: Gymnasium (13 years); 4: university graduate

Experimental design

In both studies strings of nine words were created and presented in rapid succession on an Apple Macintosh LC II microcomputer (Apple Computer, Cupertino, CA), using customized display and data acquisition software (Study 1: MacProbe, MacProbe, version 1.5.4, written by Steven Hunt, USA; Study 2: MacLab; cf. Costin 1988). Subjects viewed the screen from a distance of approximately 50 cm. Given this distance each word subtended a visual angle of approximately 0.6° vertically and 2–4° horizontally.

Each stimulus consisted of a sequence of upper-case black words displayed on a white background. Subjects were asked to determine, by pressing one of two keys on the computer keyboard, whether each list contained one or two target words, which were defined as animal names. Subjects were told that "When there are two animal names in a sequence, they may be different or the same. You don't need to worry about this – your task is simply to decide for each list whether there were one or two animal names somewhere in that list." There were five different stimulus conditions: one single-target condition and four two-target conditions. The latter four conditions were created by crossing two factors in a 2×2 design. The first of these stimulus factors was repetition: the two targets were either the same animal name ("repeated") or they were two different animal names ("unrepeated"). The second stimulus factor was lag: either one or four items intervened between the two target items.

Each trial began when the subject pressed the space bar on the computer keyboard. A plus-sign appeared in the center of the monitor screen for 825 ms as a fixation point, followed by sequence of words presented for either 135 ms each or 305 ms. Two hundred fifty-five milliseconds later, a prompt appeared on the screen saying "One or Two?". The subjects made their first response by typing a one or two on the numeric keypad on the right side of the keyboard to indicate whether the preceding sequence contained one or two targets.

Subjects were first run on 20 practice trials, at the end of which they received feedback on their mean percent correct over the whole practice test. The trials started slowly (300 ms/item) and gradually sped up through the practice sequence. After the practice trials, subjects were run on the 96 test trials. The entire experiment lasted approximately 30 min.

In study 1 stimulus presentation duration was a between-subject factor. This resulted in 32 single-target trials and in 64 two-target trials. The results of the pilot study suggested the possibility of taking fewer measurements per condition. We therefore used a complete within-subject design for the second study resulting in a $2 \times 2 \times 2$ design with the factors repetition, lag, and stimulus presentation duration, i.e., each patient and each healthy subject was tested in the fast sequence mode of 135 ms as well as in the slow mode of 305 ms. In each mode 48 trials (16 single target condition, 4×8 two-target conditions) were presented. Study 2 lasted for approximately as long as study 1.

Table 2 Data from study 1. Percentage of "2–" responses per group, stimulus duration, and condition

English stimulus words	Controls		Patients	
Stimulus presentation duration (ms)	Rapid (135) (n = 6)	Slow (305) (n = 5)	Rapid (135) (n = 6)	Slow (305) (n = 3)
Single target	16.7	2.5	12.6	4.4
Two targets				
Unrepeated target (lag 1)	81	100	31.9	88.2
Repeated target (lag 1)	60	95.7	12.5	86.5
Amount of repetition blindness (lag 1)	21***	4.3	19.4**	1.7
Unrepeated target (lag 4)	90	96.4	54.2	94.3
Repeated target (lag 4)	94	98	37.1	89.7
Amount of repetition blindness (lag 4)	–4	–1.6	17.1*	4.6

NOTE: Significant repetition-blindness effects are indicated by asterisks: * F (1,5) = 5.6, $p < 0.05$; ** F (1,5) = 7.2, $p < 0.025$; *** F (1,5) = 13.9, $p < 0.01$; all other repetition blindness effects were not significant

Results

Study 1

The relative frequency of "2"-responses for each group, stimulus duration, and condition is displayed in Table 2. Note that these frequencies report error rates in the single target condition, but percentage of correct responses in all the four cases of two targets, unrepeated as well as repeated. In addition, Table 2 indicates repetition blindness effects, i.e., the differences between unrepeated and repeated two-target conditions, for lags 1 and 4. As can be seen from performance on single and unrepeated targets, the control subjects were able to perform the task, especially when stimuli were displayed for 305 ms. They also show a clear repetition blindness effect of 21% in the lag 1 condition when stimuli were presented rapidly (i.e., for 135 ms each). With either a long stimulus duration and/or four intervening words between the two repeated targets no substantial repetition blindness is found.

Schizophrenic patients were able to do the task with ease at the slow presentation duration, as can be infered from the small number of false-positive responses (4.4%) and the detection rates for two targets in the 80–90% range. Like normal controls, schizophrenic patients showed no repetition blindness effect under such conditions. When stimuli were presented rapidly, the patients' performance dropped significantly. However, significant repetition blindness effects of 19.4% (lag 1) and 17.1% (lag 4), respectively, were found. Due to the small number of subjects, we refrained from further analysis of the data.

Study 2

Normal subjects generally answered faster and more accurately than the patients. Mean reaction times across all conditions, as measured from the start of the question panel "one or two" at the end of each sequence, were 340 ± 142 ms in the control group and 600 ± 278 ms in the patient group ($t(54) = 4.6$, $p < 0.0001$; unpaired t-test). Mean error rates were 18.1 ± 8.9 and 28.7 ± 11.9%, respectively ($t(54) = 3.8$, $p < 0.0005$). Percentage of "2"-responses for each study group, stimulus duration, and condition are given in Table 3. Normal subjects were able to perform the rapid task (presentation time 135 ms) as well as the slow task (presentation time 305 ms). In general, the number of correct responses was higher in the slow task. As in study 1, a repetition blindness effect (31.6%) only occurred in the rapid task with a lag 1, but not in the rapid task with a lag 4, as can be seen from Table 3. In the patient group a similar result was observed, i.e., a repetition blindness effect of 23.4%, despite a generally lower accuracy: No repetition blindness effects were found in either group in the slow task.

Correct response rates from all trials with two targets were submitted to a repeated-measures analysis of variance (ANOVA) with one between-group factor (group) and three within-subject factors, presentation time, repeti-

Table 3 Data from study 2. Percentage of "2–" responses per group, stimulus duration, and condition

German stimulus words	Controls		Patients	
Stimulus presentation duration (ms)	Rapid (135) (n = 36)	Slow (305) (n = 36)	Rapid (135) (n = 20)	Slow (305) (n = 20)
Single target	16.1	4.3	16.6	9.7
Two targets				
Unrepeated target (lag 1)	64.6	89.6	52.1	74.4
Repeated target (lag 1)	33.0	94.8	28.7	79.4
Amount of repetition blindness (lag 1)	31.6*	–5.2	23.4**	–5
Unrepeated target (lag 4)	84	91.7	68.1	82.5
Repeated target (lag 4)	84.4	97.6	63.7	90
Amount of repetition blindness (lag 4)	–0.4	–5.9	4.4	–7.5

NOTE: Significant repetition-blindness effects (results of two-tailed t-tests) are indicated by asterisks: * t (35) = 6.8; $p < 0.0001$; ** t (19) = 6.3; $p < 0.0001$; all other repetition-blindness effects were not signifcant

tion of the target, and lag. A main effect was found for subject population ($F(1,54) = 12.9$; $p < 0.001$) as well as for presentation time ($F(1,54) = 113.0$; $p < 0.0001$), and for lag ($F(1,54) = 241.9$; $p < 0.0001$), but there was no significant main effect of repetition ($F(1,54) = 3.73$; $p > 0.05$). There was no significant four-way interaction, but two significant three-way interactions were found. The three within-subject factors presentation time, repetition of the target, and lag ($F(3,54) = 25.0$; $p < 0.0001$) interacted significantly. Post-hoc analysis (Scheffé) revealed that in the fast presentation mode (135 ms) a clear repetition blindness effect occurred in the lag-1 condition only (significant difference in error rates comparing the repeated targets with the non-repeated targets: 30.9 vs 58.4%; $p < 0.0001$). This repetition blindness effect was neither present in the lag-4 condition (74.1 vs 76.1%; n.s.) nor in the slow presentation mode for either lag.

A second interaction was found for the group factor, presentation time, and lag ($F(3,54) = 9.83$; $p < 0.005$) and did not regard the factor of target repetition. It was therefore not further analyzed.

Discussion

The results on the normal subjects are in line with previous results (Kanwisher 1987; Park and Kanwisher 1994), adding to the body of evidence that repetition blindness is a robust phenomenon, which occurs at rapid presentation rates and under conditions of one or two intervening words. It was neither found under conditions of slow stimulus presentation nor long lag (i.e., four intervening words between the two target words). Since comparable effects were observed in both studies (i.e., in English and German language), the repetition blindness effect is not dependent on language-related idiosyncrasies.

Patients can do the task. They perform generally worse than control subjects, but they nevertheless show a comparable repetition blindness effect, which was present in both studies. Like normal subjects, the patients showed repetition blindness only when stimuli were presented rapidly and in close succession.

The results of both studies suggest that the rate bottleneck for individuation of repeated tokens is similar in schizophrenic and normal subjects, i.e., their perceptual processes are normal in this respect. According to the "processing limitation" hypothesis of repetition blindness, our findings suggest the presence of a similar limitation in schizophrenic patients. We currently plan further experiments to clarify whether the perceptual deficits shown by schizophrenic patients (possibly seen in our experiment as increased general error rates in a perceptual task) can be attributed to some form of general slowness or noisiness of visual processing. Positive findings would suggest a general information processing deficit, as has been suggested by several authors (c.f. Meehl 1989).

In summary, our results question the existence of a specific defective in early visual processing in schizophrenic patients.

Finally, the fact that repetition blindness occurs in schizophrenic patients to at least the same extent as in control subjects is informative with respect to normal psychological theories about the causes of repetition blindness.

Firstly, if we take some form of lack of inhibition in early perceptual processes in schizophrenic patients for granted, then our results rule out the idea that repetition blindness is caused by such inhibitory processes. A similar argument has been made on the basis of the finding that repetition blindness is not decreased in older adults, who have also been claimed to be impaired in inhibitory processes (MayKay et al. 1994).

Secondly, our results do not support the "off-line" hypothesis of repetition blindness proposed by Fagot and Pashler (1995) who suggested that the phenomenon is caused by memory retrieval processes. Schizophrenic patients are known to suffer from working memory dysfunctions (Goldman-Rakic 1991). Hence, they should display either less or more repetition blindness than normal subjects, independent of the mode of presentation, if the effect were memory related. The fact that schizophrenic patients show a comparable amount of repetition blindness as healthy control subjects, and that this effect is restricted to the fast presentation mode and the close succession of the items, rules out any theories of repetition blindness that engage functions known to be disrupted in schizophrenic patients.

To conclude, repetition blindness is a perceptual phenomenon that occurs in normal subjects and in schizophrenic patients as well. From a psychopathological perspective, this finding rules out a major deficit in the early perceptual process of token individuation as the cause of schizophrenic perceptual aberrations. Within a normal psychological framework, it renders unlikely accounts of repetition blindness which involve processes known to be dysfunctional in schizophrenic patients (such as decision making and working memory retrieval).

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References

Braff DL (1993) Information processing and attention dysfunctions in schizophrenia. Schizophr Bull 19:233–259

Costin D (1988) MacLab: a Macintosh system for psychology labs. Behav Res Methods Instruments Comput 20:197–200

Fagot C, Pashler H (1995) Repetition blindness: perception or memory failure? J Exp Psychol Human Percept Perf 21:275–292

Goldberg TE, Gold JM, Braff DL (1991) Neuropsychological functioning and time-linked information processing in schizophrenia: In: Tasman A, Goldfinger SM (eds) Review of psychiatry, vol 10. Washington DC, Psychiatric Press, pp 60–78

Goldman-Rakic PS (1991) Prefrontal cortical dysfunction in schizophrenia: the relevance of working memory. In: Carroll BJ, Barrett JE (eds) Psychopathology and the brain. Raven Press, New York, pp 1–23

Hochhaus L, Johnston JC (1996) Perceptual repetition blindness effects. J Exp Psychol Human Percep Perf 22:355–366

Kanwisher N (1987) Repetition blindness: type recognition without token individuation. Cognition 27 : 117–143
MacKay DG, Miller M, Schuster SP (1994) Repetition blindness and aging: evidence for a binding deficit involving a single, theoretically specified connection. Psychol Aging 9 : 251–258
Maher BA, Spitzer M (1993) Delusions. In: Sutker PB, Adams HE (eds) Comprehensive handbook of psychopathology, 2nd edn. Plenum Press, New York London, pp 263–293
Meehl PE (1989) Schizotaxia revisited. Arch Gen Psychiatry 46 : 935–944
Mozer MC (1991) The perception of multiple objects: a connectionist approach. MIT Press, Cambridge, Mass.
Park J, Kanwisher N (1994) Determinants of repetition blindness. J Exp Psychol Human Percep Perf 20 : 500–519
Park S, Lenzenweger MF, Püschel J, Holzman PS (1996) Attentional inhibition in schizophrenia and schizotypy: a spatial priming study. Cogn Neuropsychiatry 1 : 125–149
Spitzer M, Braun U, Maier S, Hermle L, Maher BA (1993) Indirect semantic priming in schizophrenic patients. Schizophr Res 11 : 71–80
Spitzer M, Weisker I, Maier S, Hermle L, Maher BA (1994) Semantic and phonological priming in schizophrenia. J Abnorm Psychol 103 : 485–494
Tipper SP (1992) Selection for action: the role of inhibitory mechanisms. Curr Direct Psychol Sci 1 : 105–109
Whittlesea BWA, Dorken MD, Podrouzek KW (1995) Repeated events in rapid lists: Part 1. Encoding and representation. J Exp Psychol Learn Memory Cogn 21 : 1670–1688