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Crowding, grouping, and gain control in schizophrenia

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ABSTRACT

Visual paradigms are versatile tools to investigate the pathophysiology of schizophrenia. Contextual modulation refers to a class of paradigms where a target is flanked by neighbouring elements, which either deteriorate or facilitate target perception. It is often proposed that contextual modulation is weakened in schizophrenia compared to controls, with facilitating contexts being less facilitating and deteriorating contexts being less deteriorating. However, results are mixed. In addition, facilitating and deteriorating effects are usually determined in different paradigms, making comparisons difficult. Here, we used a crowding paradigm in which both facilitation and deterioration effects can be determined all together. We found a main effect of group, i.e., patients performed worse in all conditions compared to controls. However, when we discounted for this main effect, facilitation and deterioration were well comparable to controls. Our results indicate that contextual modulation can be intact in schizophrenia patients.

1. Introduction

The obvious symptoms of schizophrenia are hallucinations, delusions, and cognitive dysfunctions. However, schizophrenia patients have many other abnormalities, including visual impairments (Kraepelin, 1893; Chapman, 1966; Silverstein and Keane, 2011; Chen, 2011). Visual paradigms are versatile tools of schizophrenia research because patients' deficits are often very pronounced (Espeseth et al., 2007; Chkonia et al., 2010; Silverstein and Keane, 2011; Bakanidze et al., 2013).

One class of interesting visual paradigms relates to contextual modulation, where perception of a target is strongly influenced by surrounding elements. For example, all spatial illusions are versions of contextual modulation. Other examples are surround suppression, contour integration, and crowding, which all are abnormal in schizophrenia patients. Dakin et al. (2005) presented a medium contrast patch together within a high-contrast surround. Controls perceived the patch as of much lower than the true contrast. In schizophrenia patients this effect was strongly diminished, i.e., patients reported a value closer to the *true* contrast. Interestingly, contrast discrimination itself is strongly deteriorated in schizophrenia patients (Slaghuis, 1998; Keri et al., 2002). Another example of contextual modulation is crowding, where perception of a target deteriorates when flanked by neighbouring elements (see Fig. 1). As in the previous paradigm, patients show less interference by the neighbouring elements (Robol et al., 2013).

Contextual modulation is usually explained by interactions between neighbouring neurons that mutually influence each other, for example via gain control or long range excitation, which are proposed to be weaker than in controls (e.g., Butler et al., 2008; Phillips and Silverstein, 2013). Accordingly, it seems that contextual modulation is in general weaker in patients, i.e., patients benefit less from helpful contexts but are also less affected by deleterious ones (Robol et al., 2013). These diminished neural interactions are also in agreement with the broader claim that contextual processing is deteriorated in general, including in non-visual examples such as verbal and cognitive context memory (e.g., Phillips and Silverstein, 2003; Uhlhaas et al., 2006; Phillips and Silverstein, 2013). It might be that all sorts of abnormal contextual modulation are due to higher levels of cognitive disorganisation, as already proposed by Bleuler (1911).

However, the situation is more complex. For example, Tibber et al. (2013) used a similar paradigm to Dakin et al. (2005) mentioned above, together with a paradigm where the

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orientation of target lines was modulated by surrounding lines. As in Dakin et al. (2005), contextual modulation for the contrast was weaker in the patients compared to controls but there were no differences in the orientation paradigm. Hence, not all types of contextual modulation are affected in schizophrenia patients (see also Yang et al., 2013). Moreover, Yoon et al. (2009) reported that patients show weaker modulation when target and surround have the same orientation but no differences when they are orthogonal. In addition to mixed findings, results are often hard to interpret because facilitating (release of deterioration) and deteriorating effects are not determined in one, but in different paradigms.

Related to the neural causes, the neural mechanisms of contextual modulation are under debate. For example, we have shown that in healthy participants contextual modulation cannot be explained by simple local interactions between neighbouring neurons. To the contrary, complex Gestalt aspects determine processing (e.g., crowding: Malania et al., 2007; Pelli and Tillman, 2008; Whitney and Levi, 2011; Manassi et al., 2012; surround modulation: Saarela and Herzog, 2008; visual masking: Herzog and Fahle, 2002). Particularly interesting is crowding, where we developed a task which allows one to test contextual facilitation and deterioration within one paradigm. We presented a vernier, which comprises two vertical bars that are offset slightly either to the left or right. Observers indicated the offset direction. Performance strongly deteriorated when the vernier was flanked by arrays of lines of the same length as the vernier (Fig. 1). This is a classic crowding effect. However when we presented arrays of shorter or longer lines, performance improved, challenging most models of crowding and of contextual modulation in general. Particularly, models cannot explain why longer lines, with more stimulus energy, improve performance compared to the equal length condition (e.g., Malania et al., 2007; Manassi et al., 2012). We proposed that grouping, rather than low level mechanisms, determine crowding. When the vernier groups with the flankers (same length lines) crowding is strong. When the vernier ungroups from the flankers grouping is weaker (shorter or longer lines).

Here, we applied this paradigm to schizophrenia patients. Patients performed worse compared to controls in all conditions. However, when we discounted for this main effect of group, patients performed similarly as controls, indicating that contextual modulation is intact.

2. Methods

2.1. Participants

Sixteen schizophrenia patients and 15 healthy controls participated in this study. All observers had normal or corrected-to-normal vision i.e., visual acuity was ≥ 0.8 (corresponding to 20/25) at least in one eye, as determined with the Freiburg Visual Acuity Test (Bach, 1996).

Schizophrenia patients were recruited from the Tbilisi Mental Health Hospital or the psycho-social rehabilitation centre where they had been admitted because of an acute episode of their disease. They were invited to participate in the study when they had recovered sufficiently and were estimated to be able to endure the study procedure. Among the patients group, there were three inpatients and 13 outpatients. Healthy controls were recruited from the general population. General exclusion criteria were drug or alcohol abuse, neurological or other somatic illnesses influencing subjects' mental state. Participants were no older than 55 years.

Ethics approval was obtained in Tbilisi from the Georgian National Council on Bioethics. All subjects signed informed consent and were informed that they could quit the experiments at any time.

Patients were diagnosed according to DSM-IV, by means of an interview based on the SCID, information of the staff, and the study of the records. Psychopathology of the schizophrenia patients was assessed by an experienced psychiatrist (EC) by Scales for the Assessment of Negative Symptoms and Scales for the Assessment of Positive Symptoms (SANS, SAPS (Andreasen, 1983, 1984). Group characteristics are depicted in Table 1.

All patients were receiving neuroleptic medication. Chlorpromazine equivalents are indicated in Table 1.

2.2. Apparatus and procedure

We determined thresholds for vernier offset discrimination. Verniers were presented alone or neighboured by flanker configurations. Verniers consisted of two vertical lines slightly offset to the left or right. The task of the observers was to discriminate the vernier offset direction.

The experimental room was dimly illuminated 0.5 lx Stimuli were generated on a Pentium-based computer and displayed on a Siemens Fujitsu P796–1 monitor (31.0 cm (H) × 23.3 cm (V), 1024 × 768 resolution). White stimuli were presented on a black background and the luminance of the screen was below 1 cd/m^2. Luminance of stimuli was 100 cd/m^2 approximately. Refresh rate was 100 Hz and viewing distance was 350 cm.

The vernier consisted of two vertical 10′ (arcmin) long lines separated by a vertical gap of 1′. Observers were instructed to fixate the vernier. Vernier and flankers were presented simultaneously for 150 ms.

Observers were asked to indicate the vernier offset direction by pressing one of two push buttons. Auditory feedback was provided after incorrect or omitted responses. An adaptive staircase procedure (Taylor and Creelman, 1967) was used to determine the threshold for which observers reached 75% correct responses. Thresholds were determined after fitting a cumulative Gaussian to the data using probit and likelihood analyses. The starting offset was 1.67′.

After each trial, the screen remained blank for a maximum period of 3 s during which the observer was required to make a response. The screen was blank for 400 ms between the response and the next trial. In every block of 80 trials, the number of left and right offsets was balanced.

2.3. Stimulus configurations

The vernier was presented alone or flanked by two arrays of 16 vertical lines, one on each side (Fig. 1). The directly neighbouring lines were always placed at a distance of 3.33′ from the vernier. Inter-flanker spacing was also 3.33′. Three different flanker lengths were used: short (5′), equal (10.5′), and long (21′) (Fig. 1). Each condition was presented in separate blocks of 80 trials. All conditions, including the vernier alone condition, were measured twice (i.e., 160 trials in total). The order of the flanker and vernier configurations was randomized across observers. To compensate for potential learning effects, performance in all conditions was measured once and then, the order of conditions was reversed in the second run.

3. Results

First, we performed a 2 × 4 repeated measures analysis with Group as between-subjects factor (patients vs. controls) and Condition as within-subjects factor (unflanked, equal size flankers, short size flankers, long size flankers). There was a significant effect of Condition ($F[1,55]=12.6$; $p \leq 0.0005$), where performance was best in the unflanked condition and worst in the equal sized flankers condition, reproducing previous results. There was no significant interaction. Schizophrenia patients had, on average, higher thresholds than controls in all conditions ($F[1,29]=56.3$; $p \leq 0.0005$). For example, mean thresholds for the unflanked vernier discrimination were 16.2′ ± 8.6 and 27.0′ ± 13.1 in controls and patients, respectively. To account for this base deficit, we normalised performance by dividing the “crowding” threshold for each observer by his/her threshold in the unflanked condition, i.e., we determined performance in terms of threshold elevation. These values were subjected to a 2 × 3 repeated measures ANOVA with factors Group (patients, controls) and Condition (equal size, short size, long size flankers). There was a main effect of Condition ($F[1,42]=11.9$; $p \leq 0.0005$) but no significant main effect of group ($F[1,29]=0.02$; $p=0.9$) and no significant interaction ($F[1,42]=0.2$; $p=0.75$).

Hence, there is nonspecific deterioration of patients in all conditions. When this effect is controlled for, performance of patients is about on the same level as the one for controls.

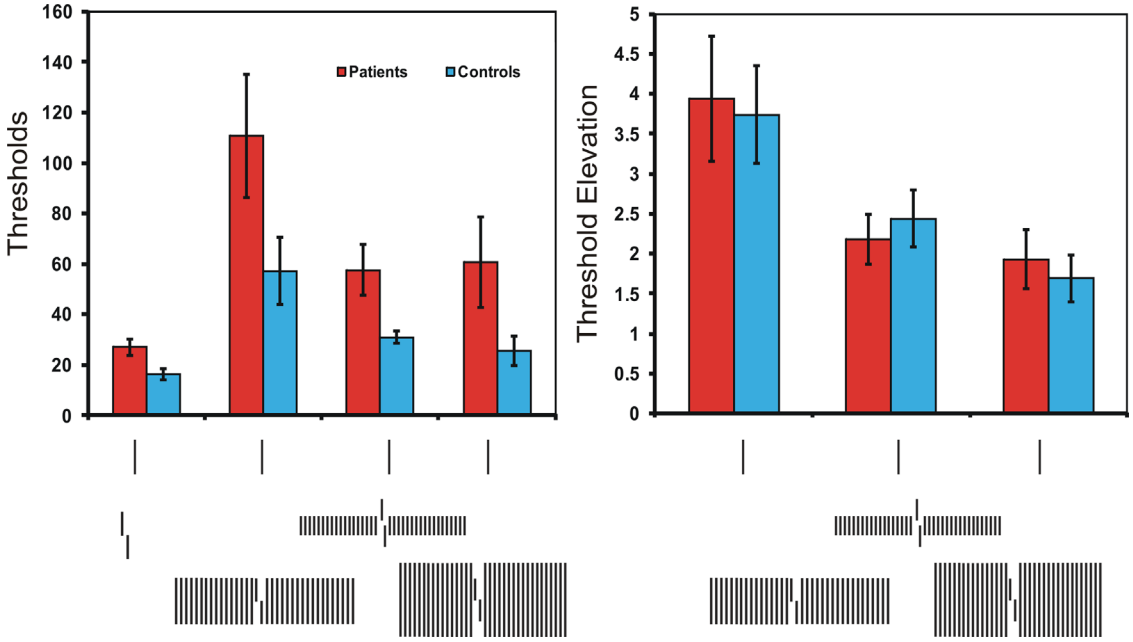

Fig. 1. (A) Mean thresholds for the unflanked (vernier is presented alone) condition and for three crowding conditions (vernier is flanked by 2 × 8 equal, short or long flankers). When the vernier was flanked by 2 × 8 same-length flankers, thresholds increased dramatically compared to the other conditions. Performance in patients is worse in all conditions compared to controls. (B) We discounted for the main effect of Group by dividing the thresholds of each observer in the crowding conditions by the threshold in the unflanked condition of this observer (threshold elevation). After discounting, performance is very similar in patients and controls. Error bars indicate the standard error of the mean for 16 schizophrenia patients and 15 healthy controls.

Table 1
Average statistics (± S.D.) of schizophrenia patients and healthy controls.

	Schizophrenia patients	Healthy controls
N	16	15
Age (years)	40.3 ± 8.1	38.9 ± 6.6
Gender (f/m)	3/13	2/13
Education level (years)	13.4 ± 1.6	15.6 ± 2.1
Duration of illness (years)	14.7 ± 8.5	
SANS	8.9 ± 5.3	
SAPS	7.8 ± 2.8	
CPZ	439.6 ± 76.2	
Degraded CPT	2.8 ± 1.0	4.0 ± 0.08
WCST (categories)	3.2 ± 1.5	4.5 ± 1.8
Handedness (R/L)	15/1	14/1
Visual acuity	1.3 ± 0.3	1.7 ± 0.4

The analysis for the CPT-DS was conducted on average number of correct responses ("hit rate") across trials, perceptual sensitivity (d-prime) (referring to an individual's ability to discriminate target stimuli from non-targets stimuli) and the total number of errors (WCST-Err) for the WCST.

4. Discussion

It is often proposed that contextual modulation is diminished in schizophrenia, i.e., patients are less influenced by both facilitating and deteriorating contexts (Must et al., 2004; Dakin et al., 2005; Robol et al., 2013). On a neuronal level, these effects are attributed to abnormal gain control (Phillips and Silverstein, 2013). However, a comparison between the facilitating and deleterious context conditions is often hard to determine because, in most paradigms, both effects are not determined in one paradigm. In addition, important conditions, such as a target-only-condition, are missing (Keri et al., 2002; Must et al., 2004) and stimuli are sometimes backward masked, confusing contextual modulation and the well-known masking effects in schizophrenia (Kraehenmann et al., 2012; Robol et al., 2013).

Here, we tested crowding in a paradigm where we could determine both facilitating and deteriorating effects of the context. First, in line with previous studies, we found that shorter and longer flanking lines lead to better performance than lines of the same length as the vernier. These results hold true for both controls and patients and cannot be explained by classic models of crowding, which are based on local interactions. Gestalt is key. Second, patients are deteriorated in all conditions, i.e., there is a main effect of group. Particularly, performance was strongly deteriorated in the vernier only condition. However, main effects are of no avail for schizophrenia research (patients are deteriorated in most paradigms) and in particular for contextual modulation, where only *changes* in performance relative to context are of interest. When we discounted for the main effect of Group, facilitation and deterioration were very similar to controls (Fig. 1). Hence, contextual modulation and, as we like to argue, Gestalt processing, are intact in our paradigm. These results are reminiscent of our work on backward masking, where patients also performed comparable to controls, when we accounted for a main effect of Group (Herzog et al., 2004; Schütze et al., 2007; Roinishvili et al., 2008). In this line, Tschacher et al. (2008) showed that apparent motion process, another Gestalt phenomenon, is intact in schizophrenia patients. Parnas et al. (2001) found even improved performance in a texture task for prodromal patients (see also Knight et al., 2000; Uhlhaas et al., 2006; Uhlhaas and Silverstein, 2005).

Where does the main effect come from? We argued previously that patients have problems to enhance weak neural signals when they are of task relevance. For example, fine grained vernier offset are of no relevance for the visual system in normal conditions and are therefore suppressed by default. Only in certain tasks, this information might be of interest and needs to be enhanced. We attributed such enhancement deficits to abnormalities in the cholinergic system, where we found a mutation (Herzog et al., 2013; Bakanidze et al., 2013).

We found that schizophrenia patients were not different from controls in our crowding paradigm. Other studies have found contextual modulation deficits in patients (see Cohen et al., 1999; Dakin et al., 2005; Barch and Ceaser, 2012). How can these

differences be explained? Three factors may play a role. First, outpatients may perform better than inpatients. For example, Barch et al. (2012) could not replicate the results of Dakin et al. (2005). One crucial difference in the studies was that Dakin et al. (2005) investigated intpatients whereas in Barch et al. (2012), outpatients participated. Similar results were found by Silverstein et al. (2013), who reported a strong deterioration in size constancy in acute patients but no difference between stable outpatients and controls. In our study, mainly outpatients participated (13/16). However, it needs to be noted that we found a main effect of performance, which is different to Tibber et al. (2013), where no effects at all were found. A second important factor is task difficulty depending on the spatial configuration of the stimuli. For example, patients' performance is similar to controls when contextual elements are clearly visible, such as a smooth contour. However, performance is different when contours are fragmented (Chey and Holzman, 1997; Silverstein et al., 1998; Knight et al., 2000). Similarly, in our study, the flankers themselves were clearly visible and thus was their grouping as flanker arrays. It may be that contextual effects become weaker when grouping is attenuated, for example, by increasing the spacing between flankers. In a final note, in many studies, it is impossible to determine whether performance deficits of patients are due to a main effect, i.e., deterioration in all conditions, or a context specific effect.

In conclusion, first, our results suggest that key mechanisms of contextual modulation need to be rethought. Even though visual mechanisms are well investigated, models are not always firmly proved and thus need to be taken cautiously. For example, complex Gestalt vision plays a role, which was neglected for almost half century. Second, in line with other studies, it seems that spatial contextual processing is intact in schizophrenia patients at least when clinically stable, as were the patients in this study (see also, Tibber et al., 2013; Silverstein et al., 2013). This result holds true independent of whatever the underlying mechanism are. An important question is why other forms of contextual modulation are impaired in schizophrenia patients? Third, our paradigm offers a simple way to test both deterioration and facilitation (release of deterioration) in one paradigm.

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