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Early visual processing deficits in patients with schizophrenia during spatial frequency-dependent facial affect processing

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ABSTRACT

Abnormal facial emotion recognition is considered as one of the key symptoms of schizophrenia. Only few studies have considered deficits in the spatial frequency (SF)-dependent visual pathway leading to abnormal facial emotion recognition in schizophrenia. Twenty-one patients with schizophrenia and 19 matched healthy controls (HC) were recruited for this study. Event-related potentials (ERP) were measured during presentation of SF-modulated face stimuli and their source imaging was analyzed. The patients showed reduced P100 amplitude for low-spatial frequency (LSF) pictures of fearful faces compared with the HC group. The P100 amplitude for high-spatial frequency (HSF) pictures of neutral faces was increased in the schizophrenia group, but not in the HC group. The neural source activities of the LSF fearful faces and HSF neutral faces led to hypo- and hyper-activation of the frontal lobe of subjects from the schizophrenia group and HC group, respectively. In addition, patients with schizophrenia showed enhanced N170 activation in the right hemisphere in the LSF condition, while the HC group did not. Our results suggest that deficits in the LSF-dependent visual pathway, which involves magnocellular neurons, impair early visual processing leading to dysfunctional facial emotion recognition in schizophrenia. Moreover, it suggests impaired bottom-up processing rather than top-down dysfunction for facial emotion recognition in these patients.

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1. Introduction

Abnormal facial affect perception and processing have been widely documented in patients with schizophrenia, both behaviorally and physiologically, as measured by event-related potentials (ERPs). Over the last few decades, studies have revealed that face-related ERPs generally show reduced or delayed activation in patients with schizophrenia during facial emotion recognition or tasks related to face perception (McCleery et al., 2014). P100, a component reflecting early visual perception, was reduced in response to facial stimuli in patients (Campanella et al., 2006; Caharel et al., 2007), while some studies reported no differences between normal controls (Herrmann et al., 2004; Wynn et al., 2008; Jung et al., 2012). The N170 component has been consistently reported to show reduced amplitude and delayed latency that reflects altered decoding stages of facial features (Herrmann et al., 2004; Johnston et al., 2005; Turetsky et al., 2007; Lee et al., 2010). Some studies report that the late ERP components of facial emotional processing (N250 or P300) are altered in schizophrenia (Streit et al., 2001; Turetsky et al., 2007; Wynn et al., 2008); however, there are also results suggesting intact amplitudes and latencies in such components (Herrmann et al., 2004; Johnston et al., 2005; Turetsky et al., 2007).

Facial affect recognition involves complex visual processing that combines the global emotional expression of the face with detailed features. Such visual features are transferred from the retina to the visual cortex through two major parallel pathways: the magnocellular pathway and parvocellular pathway (Livingstone and Hubel, 1988; Tobimatsu and Celesia, 2006). Each pathway processes different aspects of facial features; global information and coarse emotional cues are related to the low-spatial frequency (LSF) features and thus, more dominantly processed through the magnocellular pathway, whereas high-spatial frequency (HSF) features like precise recognition of identity and detailed analysis of facial traits is more dominantly processed by the parvocellular pathway (Obayashi et al., 2009; Silverstein et al., 2010; Calderone et al., 2013; Laprevote et al., 2013).

Researchers have found meaningful abnormalities of such visual pathways in schizophrenia patients. These deficits include increased visual thresholds (Schechter et al., 2003; Caharel et al., 2007), greater sensitivity to backward masking (Saccuzzo and Braff, 1986; Braff, 1993; Butler et al., 1996; Green and Nuechterlein, 1999; Schechter et al., 2003), decreased contrast sensitivity (Slaghuis and Curran,

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1999; Keri et al., 2002; Butler et al., 2005), or motion perception (Chen et al., 1999; Schwartz et al., 1999; Li, 2002). Such findings indicate impairments in both magnocellular and parvocellular pathway, but a more dominant impairment in the magnocellular pathway seems to be evident. Thus, the deficit in perceptual organization or ability to process global form information may be responsible for inaccurate facial expression recognition in schizophrenia (Frith et al., 1983; Turetsky et al., 2007; Laprevote et al., 2010; Silverstein et al., 2010). However, there is insufficient evidence to confirm this hypothesis.

Since the parvocellular and magnocellular pathways have different dominancy in processing spatial frequencies (SF), it is possible to arouse each visual pathway using SF-manipulated facial images according to their dominancy. To the best of our knowledge, only one ERP study has used SF-manipulated facial images to investigate which visual pathway is responsible for abnormal emotion recognition in schizophrenia (Obayashi et al. (2009). Although they found that schizophrenia patients have deficits in SF-dependent visual processing, they failed to demonstrate any change in emotional processing due to SF differences. Therefore, the relationship between visual pathway deficits and altered facial affect recognition by patients with schizophrenia is still unclear.

In the current study, we investigated how deficits in the visual pathway alter face affect processing in schizophrenia using SF-manipulated facial images as stimuli. Patients with schizophrenia show more inaccurate understanding in negative emotions (Bell et al., 1997); therefore, we used fear and neutral facial pictures in different SF conditions. We hypothesized that visual pathway deficiencies would be dominant for LSF fearful face pictures expressed as reduced amplitude of the ERP components or reduced source activation of pathway-related brain areas.

2. Methods

2.1. Participants

We recruited 21 patients (10 women) aged 37.57 ± 11.37 years who were diagnosed with schizophrenia (SPR) based on the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) Axis I Psychiatric Disorders (First et al., 1997b). All patients were on stable doses of atypical antipsychotic medications. The patient's psychiatric symptoms were evaluated using the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1987).

Eighteen healthy controls (HC; 10 women) were recruited from the local community through advertisements in local newspapers and posters. Their mean age was 40.83 ± 12.07 years. They were initially screened for any signs that might have affected the experiment. After initial screening, controls were interviewed using the SCID for DSM-IV Axis II Disorders to exclude those with any personality disorders (First et al., 1997a).

The details of the study were explained to the participants and they signed a written consent form, approved by the Institutional Review Board of Inje University Ilsan Paik Hospital. Detailed demographic data of our participants are listed in Table 1.

Table 1
Demographic data and symptom ratings of the participants.

		Schizophrenia ($n = 21$)	Healthy controls ($n = 18$)	p value
Age (years)		37.57 ± 11.37	40.83 ± 12.07	0.391
Male:Female		11:10	8:10	0.751
Education duration (years)		12.44 ± 2.33	14.17 ± 4.13	0.133
Number of accepted epochs				
BSF Fear		39.14 ± 11.62	40.56 ± 12.43	0.716
BSF Neutral		39.76 ± 12.43	42.11 ± 8.46	0.502
HSF Fear		37.38 ± 12.69	41.06 ± 9.74	0.323
HSF Neutral		37.19 ± 11.84	41.39 ± 9.20	0.230
LSF Fear		38.00 ± 11.41	40.33 ± 10.42	0.512
LSF Neutral		38.67 ± 11.72	40.17 ± 9.93	0.672
Number of hospitalizations		3.23 ± 7.83		
Dosage of medication (CPZ equivalents, mg)		394.12 ± 283.88		
PANSS	Positive score	15.70 ± 6.11		
	Negative score	20.20 ± 8.06		
	General score	39.55 ± 12.38		
	Total score	75.45 ± 23.86		

2.2. Stimuli

A total of 104 faces (52 fearful and 52 neutral facial expressions) were selected from the Korea University Facial Expression Collection (KUFEC) (Lee et al., 2006). The pictures were converted into gray scale. HSF and LSF pictures were obtained by applying high-pass (>24 cycles/image) and low-pass (<8 cycles/image) filters, respectively. The filtering procedures were done using the MATLAB software version 7.9 (The MathWorks, Natick, MA, USA). A statistical test using repeated measures ANOVA was used to ensure that the average intensity of each gray scale image did not differ between the SF and emotion (SF: ($F[1.730,88.210] = 3.149$, $p = 0.055$; emotion: $F[1,51] = 1.959$, $p = 0.168$). Example images and their amplitude spectrum of the facial stimuli used in the current study are illustrated in Fig. 1.

2.3. Experimental procedure

The participants were seated in a comfortable chair facing a 17-in. CRT monitor in a sound-attenuated room. The experiment consisted of three sessions. In each session, the participant was presented with two facial stimuli, a fearful and a neutral face, and an irrelevant stimulus (picture of a chair). The SF of the facial stimuli in a session was set to be either broad spatial frequency (BSF), HSF, or LSF, which was maintained during each session. The participants were instructed to focus on the stimuli appearing on the screen. To ensure that the participants were concentrating on the stimulus, they were instructed to press a button whenever a chair stimulus were presented. Each session was composed of 100 facial (50 fearful and 50 neutral faces) and 20 chair stimuli appearing in a random order. Each trial began with a fixation cross for 200 ms, followed by a blank screen for 500 ms. The face or chair stimuli were presented for 500 ms afterwards, followed by a blank screen with an interval between 1200 and 1800 ms.

2.4. EEG acquisition

EEG were amplified and recorded using NeuroScan SynAmps2 amplifier (Compumedics USA, El Paso, TX, USA). We recorded EEG using QuikCap (Compumedics USA, El Paso, TX, USA) with two additional bipolar electrodes to record vertical and horizontal electrooculogram. The signals were referenced to both mastoids and grounded to AFz. Impedances were maintained below 5 kΩ for all electrodes. The sampling rate was set to 1000 Hz and a 0.1–100 Hz online filter with 60 Hz bandstop filter were applied to the initial recording.

2.5. EEG preprocessing and ERP analysis

All preprocessing was done with the Scan 4.3 software (Compumedics USA, El Paso, TX, USA). A trained person inspected the recordings to reject artifact blocks induced by gross movements or other possible artifacts. Ocular artifacts were reduced using the mathematical procedure implemented in the preprocessing software (Semlitsch et al., 1986). The artifact-corrected data were re-referenced to average reference and epoched from −200 ms pre-stimulus to 900 ms post-stimulus. Each epoch was baseline-corrected and filtered using a 0.1 Hz to 30 Hz bandpass filter. Any epoch with amplitude exceeding ±75 μV was considered as a physiological artifact and was rejected from the analysis. Finally, the remaining artifact-free epochs were averaged across each stimulus condition and group. The number of epochs used

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