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ScienceDirect
Schizophrenia Research 86 (2006) 89–98
SCHIZOPHRENIA RESEARCH
www.elsevier.com/locate/schres

Early stage vision in schizophrenia and schizotypal personality disorder

Brian F. O'Donnell [a,b,*], Andrew Bismark [b], William P. Hetrick [a], Misty Bodkins [b], Jenifer L. Vohs [a], Anantha Shekhar [b]

[a] *Department of Psychology, Indiana University, Bloomington, IN, United States*
[b] *Department of Psychiatry, Indiana University School of Medicine, Indianapolis, IN, United States*

Received 23 December 2005; received in revised form 12 May 2006; accepted 17 May 2006
Available online 10 July 2006

Abstract

Previous studies of visual perception have reported deficits in contrast sensitivity and dot motion discrimination in schizophrenia. We tested whether these deficits also appear in schizotypal personality disorder (SPD). SPD appears to be genetically and symptomatically related to schizophrenia, but without the marked psychosocial impairment associated with psychotic disorders. The present study investigated contrast sensitivity for moving and static gratings, form discrimination and dot motion discrimination in 24 patients with schizophrenia or schizoaffective disorder (SZ), 16 individuals with SPD, and 40 control subjects. SZ, but not SPD subjects, showed impairments on tests of contrast sensitivity for static and moving gratings, form discrimination in noise, and dot motion discrimination. Visual performance did not differ between medicated SZ patients and patients withdrawn from medication. These results confirm early stage visual deficits in schizophrenia regardless of medication status. SPD subjects, in contrast, show intact early stage visual processing despite the presence of marked schizotypal symptoms.

Keywords: Vision; Perception; Contrast sensitivity; Schizophrenia; Schizotypal personality disorder

1. Introduction

Schizophrenia is associated with disturbances of visual perception. Both interview and self-report scales indicate that patients with schizophrenia frequently suffer from visual distortions, which appear in the earliest stages of the illness (Bunney et al., 1999; Cutting and Dunne, 1986; Phillipson and Harris, 1985). Subjective disturbances are accompanied by visual processing deficits on psychophysical tests (Butler and Javitt, 2005). These psychophysical deficits have often been interpreted in terms of deficits in specific visual pathways or channels.

Psychophysical tests measure visual performance thresholds as a function of such factors as contrast, noise, stimulus duration, or stimulus similarity. In primates and humans, two neural pathways for visual processing have been characterized by the differing response properties of the magnocellular (M) and parvocellular (P) neurons of the lateral geniculate nucleus (Livingstone and Hubel, 1988; Wandell,

* Corresponding author. Department of Psychology, Indiana University, Bloomington, IN, United States. Tel.: +1 812 856 4164; fax: +1 812 855 4691.
E-mail address: bodonnel@indiana.edu (B.F. O'Donnell).

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doi:10.1016/j.schres.2006.05.016

1995). The M pathway is characterized by high contrast sensitivity, high temporal resolution, low spatial resolution, and insensitivity to color. The P pathway has low contrast sensitivity, low temporal resolution, high spatial resolution, and strong color opponency responses. Human psychophysical performance also suggests the existence of a transient or broad-band visual channel, whose response properties are similar to those of the M pathway, and the sustained channel, which resembles the P pathway (Legge, 1978; Livingstone and Hubel, 1988; Merigan and Maunsell, 1993). Functional differentiation continues into the cortex, with a ventral cortical pathway from the occipital to the inferior temporal lobe for the analysis of color and object properties, and a dorsal pathway from the occipital to the parietal lobe for motion and spatial relationships (Ungerleider, 1985; Van Essen and Gallant, 1994; Merigan et al., 1997). The M pathway primarily projects to the dorsal stream of the cortex, while the P pathway primarily projects to the ventral cortical stream. A number of investigators have proposed that schizophrenia is associated with a more severe disturbance of M or transient channel relative to P or sustained channel processing (Butler and Javitt, 2005; O'Donnell et al., 1996; Green et al., 1994; Kéri et al., 2004). In the following section, findings are reviewed from studies of perception of coherent dot motion and contrast sensitivity for static and moving grating stimuli in terms of visual pathway function.

One of the most consistent findings in schizophrenia has been impaired discrimination of the trajectory of moving dots in both medicated (Brenner et al., 2003; Chen et al., 2003b, 2005; Hooker and Park, 2000; Li, 2002; O'Donnell et al., 1996; Stuve et al., 1997) and unmedicated patients (Richardson et al., 1996). In a widely used paradigm, the motion coherence or dot kinetogram test, the percentage of dots moving in a uniform direction is varied (e.g. Brenner et al., 2003; Chen et al., 2003b). This percentage is usually referred to as motion coherence and constitutes the signal in the image. The percentage of motion coherence required for a specific level of discrimination performance, or threshold, is usually higher in schizophrenia than in control subjects. Li (2002) showed that this deficit was due to reduced sensitivity, rather than altered response bias. With respect to neural mechanisms, animal and human studies indicate that the dorsal visual pathway, particularly cortical region MT, is involved in the perception of coherent motion from an array of moving dots (Newsome and Pare, 1988). Chen et al. (2003b) found that patients with schizophrenia were impaired at discrimination of coherent motion of moving dots, which requires global motion processing, but not at discrimination of moving gratings, which can be accomplished by local visual cues. Chen and colleagues argued that these findings suggest a late stage motion processing deficit in schizophrenia, probably in cortical regions specialized for motion perception.

Contrast sensitivity for sinusoidal grating stimuli has also been studied in schizophrenia. Contrast sensitivity is the inverse of contrast threshold (the minimum physical contrast needed to reliably detect a stimulus). Some investigators (Butler et al., 2005; Kéri et al., 2002; Schwartz et al., 1987; Slaghuis, 1998, 2004) but not all (Chen et al., 1999a,b) have found deficient contrast sensitivity in schizophrenia. In monkeys, magnocellular lesions have their greatest impact on contrast sensitivity for low spatial frequencies (<4 cycles/degree) at temporal frequencies above 8 Hz, while parvocellular lesions have their greatest impact at temporal frequencies below 4 Hz (Merigan and Maunsell, 1993). Human psychophysical studies indicate that transient channels are insensitive at spatial frequencies above 4 cycles/degree (Legge, 1978). Consequently, a differential contrast sensitivity deficit at low spatial and high temporal frequencies would be supportive of an M deficit. This pattern has not been consistently found. Schwartz et al. (1987) reported that contrast sensitivity deficits were most reliably observed for temporally modulated gratings, rather than static gratings, suggestive of a transient channel deficit. Butler et al. (2005) reported a greater schizophrenia deficit at low compared to high spatial frequencies. Slaghuis (1998, 2004), on the other hand, found that negative symptom schizophrenic patients showed a deficit for both stationary and moving patterns for both low and high spatial frequencies. Positive symptom patients showed deficits only at medium to high spatial frequencies (Slaghuis, 1998) or no impairment at any spatial frequency (Slaghuis, 2004). Chen et al. (2003a) hypothesized that the differences among studies of contrast sensitivity may have been related to medication levels. Chen and colleagues reported that patients receiving typical anti-psychotic medications showed elevated contrast thresholds and patients receiving novel anti-psychotic medications showed unimpaired contrast sensitivity levels for a moving grating stimulus. Moreover, unmedicated patients demonstrated decreased contrast threshold levels, indicative of performance which was better than that of control subjects. Chen et al. suggested that these medication effects might be mediated by dopaminergic cells in the retina which influence contrast sensitivity. Increased