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Visual processing, social cognition and functional outcome in schizophrenia

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ABSTRACT

Visual processing deficits are well recognised in schizophrenia and have potentially important clinical implications. First, the pattern of deficits for different visual tasks may help understand the underlying pathophysiology of the visual dysfunction. Second, several studies report deficits correlating with functional outcomes, suggesting that outcome improvement is possible through visual remediation strategies. We investigated these issues in a group of 64 schizophrenia patients and matched controls with a battery of visual tasks targeting different points along the visual pathways and by examining direct and indirect relationships (via a potential mediator) of such deficits to functional outcome. The schizophrenia group was significantly worse on the visual tasks overall, with the deficit constant for low- and high-level processing. Zero-order correlations suggested minimal association between vision and outcome, however, correlations between three visual tasks and 'social perceptual' ability were found which in turn correlated with functional outcome; path analysis confirmed a significant but small and indirect effect of 'biological motion' processing ability on functional outcome mediated by 'social perception'. In conclusion, the pathophysiology of visual dysfunction affects low- and high-level visual areas similarly and the relationship between deficits and outcome is small and indirect.

1. Introduction

A large body of research indicates that individuals with schizophrenia experience visual deficits (see Butler et al., 2005). The deficits are found in relation to a variety of tasks, particularly those related to the magnocellular/dorsal stream, a pathway from retina to visual cortex and beyond linked to visual motion processing and conveying signals related to low-spatial frequencies (large scale visual detail), low contrast, and high temporal frequencies. The parvocellular/ventral system, which conveys color and high spatial frequency information, appears relatively spared. The deficits are of potential clinical interest as they may provide clues as to the underlying pathophysiology of the visual dysfunction; different visual perceptual abilities are resolved at varying points along the visual pathways so that the overall pattern of deficits may point to particular cortical locations and processes. Most previous studies have focussed on 'low-level' processing, i.e., those resolved early-on in the visual pathways, although 'higher-level' visual deficits have also been reported, revealing problems in cortical-level visual processing. Thus, deficits have been found for abilities such as 'luminance-flicker sensitivity' (Slaghuis and Bishop, 2001) static 'contrast sensitivity' (Keri et al., 2002) and 'visual masking' (Rassovsky et al., 2005) that involve early, low-level visual processing and higher-level deficits have been reported for tasks such as 'global motion' (Chen et al., 2003), requiring the integration of information across the visual field, and 'biological motion', requiring the extraction of walking figure contours (Kim et al., 2005). No studies, however, have examined a range of low- and high-level tasks within the same subjects and testing session. If visual deficits increase in magnitude from low- to high-levels of processing, as has recently been suggested (Butler et al., 2005), this would point to a mechanism affecting each level of the visual hierarchy or a low-level deficit amplified by transmission through the increasingly specialised components of the visual pathways. Alternatively, if the deficit remains constant across each level of the visual hierarchy, this would suggest a mechanism primarily affecting the earlier stages of processing.

A second potential area of clinical interest is the relation of visual deficits to functional outcomes, with a number of studies reporting significant correlations between these variables in schizophrenia. This raises the possibility that perceptual training techniques may improve the poor functional outcomes often seen in schizophrenia. However, the potential of this approach is unclear, as the few reports available disagree as to how strong the correlation might be. For example, Kim et al. (2005) found that 50% of the variance in a functional outcome measure was accounted for by perceptual ability ($r = 0.71$), Butler et al. (2005) reported figures of between 14 and 25% ($r = 0.37$–0.50) and Sergi et al. (2006) reported lower figures still of between <1% and 11% ($r = 0.03$ to $r = 0.33$). Other studies have measured the relationship

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between functional outcome and 'visual' measures within large-scale neurocognitive batteries. Some of these studies have reported significant correlations (Bowen et al., 1994) whilst others have not (Vauth et al., 2004). Furthermore, such studies tend to use tasks that, although presented visually, involve other processes such as 'sustained attention', e.g., the degraded-stimulus continuous performance task.

The variation in reported correlation strengths between visual processing and functional outcome might reflect the fact that different studies have probed different levels of the visual system. Tasks probing low-level visual processing may be less directly linked to functional outcome than tasks probing higher levels. This might explain why studies such as Kim et al. (2005) which used a high-level, biological motion stimulus, found a strong association whilst studies such as Sergi et al. (2006) using a low-level visual masking paradigm found lower correlations. Another explanation might be the effect of certain high-level visual tasks such as biological motion perception inadvertently tapping into another, unmeasured factor, related to outcome, 'social cognition'; thereby inflating the apparent strength of relation between vision and function. Social cognition includes varied domains such as emotion perception, social perception and social knowledge (Green et al., 2005a), and has been proposed to act as an intermediary variable between several classes of neurocognition (including vision) and functional outcome (Brekke et al., 2005). This theory is based in part on the link between neurocognition and social cognition (Bozikas et al., 2004) and between social cognition and functional outcome (Penn et al., 2002) and it seems plausible that if visual deficits produce a relative inability to pick up socially important visual cues, i.e., micro-expressions or subtle gestures, this could be a path through which visual deficits impact on functional outcome in schizophrenia. One study thus far has found evidence for this causal chain (Sergi et al., 2006).

Here, we set out to address the above issues. First, we have studied the pattern of visual deficits in individuals with schizophrenia compared to controls across different levels of visual processing in tasks which, at least partially, recruit the magnocellular/dorsal stream. Second, we have studied the relationship between vision and functional outcome in schizophrenia and attempted to address the question of why previous studies have found varying strengths of association. A battery of visual tests probing different levels of the visual hierarchy was undertaken by a cohort of patients and control subjects together with measures of social perception and, in the patient group, functional outcome.

2. Methods

2.1. Subjects

Sixty-four persons with a Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) diagnosis of schizophrenia and 65 control subjects were recruited. All but five of the patients were taking antipsychotic medication (50 atypical, seven typical, two mixed). The patient group were recruited from outpatient and long-term assisted living settings in South London. Diagnosis was confirmed by their treating clinician, chart review and the psychotic symptoms subsection of the Structured Clinical Interview for DSM-IV (SCID) Patient Edition (First et al., 2002b). Control subjects with no self-reported history of psychiatric illness were recruited from the community. The psychotic screening subsection from the SCID Non-patient edition (First et al., 2002a) was used to check for lifetime presence of psychotic illness. Potential control subjects were excluded if any of their first-degree relatives had a history of psychotic illness. Exclusion criteria applied to both groups included: current drug or alcohol dependency, a reading or sensory disability, an identifiable neurological condition and having best-corrected visual acuity below 0.80 decimal, measured using the Freiburg Visual Acuity test (Bach, 2007). The experimental protocol was approved by the Institute of Psychiatry ethics committee. Subjects gave written consent and were paid for their participation.

2.2. Stimuli and procedure

Tasks were presented on a 21 inch gamma corrected ViewSonic G220f monitor in a dimly lit room (10–12 lx). The monitor's mean luminance was 87.7 cd/m^2. The following resolutions and frame rates were applied: contrast sensitivity (*CS*; 1024*768, 85 Hz), visual masking (*MASK*; 800*600, 160 Hz), global motion (*GM*; 800*600, 100 Hz), biological motion (*BM*; 1024*768, 120 Hz) and Half-PONS (*HP*; 1024*768, 85 Hz). The CS task was run from a Mac Tower. The GM and BM tasks were run from a Centrex PC through a VSG 2/5 graphics card. The MASK and HP tasks were run from a Dell laptop. Subjects gave their responses by speaking aloud and the test administrator entered responses on the computer. All tasks were viewed binocularly and were presented in a random order with the exception of the contrast sensitivity task which was performed during an initial visual acuity screening session. Viewing distance for the MASK, GM, BM and HP tasks was 100 cm, and for the visual tasks was kept constant with a chin rest. The CS task viewing distance was 200 cm and no chin rest was used. All tasks were forced choice procedures and were preceded by a practise period.

2.2.1. Visual tasks

We focussed on tasks with sensitivity to magnocellular/dorsal stream function, given the previous evidence of this channels dysfunction in schizophrenia (Butler and Javitt, 2005). Each section below initially details the aspect of the magnocellular/dorsal stream targeted by the visual task. Tests of low-level visual processing included contrast detection and visual masking. Intermediate-level visual processing was examined using a test of global motion perception. The highest level of visual processing was assessed using a test of biological motion perception.

2.2.1.1. Contrast sensitivity (CS). Magnocellular neurons have lower contrast thresholds (Livingstone and Hubel, 1988) and preferentially activate to low-spatial frequency stimuli below about 1.5 cycles/° (Legge, 1978). The Freiburg Contrast Test version 5.6.1 (Bach, 2007) was used to assess contrast detection thresholds. A 'Landolt C' optotype is used where the gap of the 'C' can have one of four orientations (Fig. 1). Subjects were required to name the orientation (up, down, left, right). The figure subtended 5.7° of visual angle and the gap had an equivalent spatial frequency of 0.5 cycles/°. Threshold is estimated with a 'Best PEST' ('parameter estimation by sequential testing') algorithm and an adaptive-staircase procedure. The 'contrast sensitivity' result is the logarithmized inverse of the threshold contrast, defined with the Michelson contrast (Lmax − Lmin)/(Lmax + Lmin). There were 28 trials. Higher values indicated better sensitivity to contrast.

2.2.1.2. Visual masking (MASK). Magnocellular neurons are sensitive to transient stimuli (Breitmeyer and Julesz, 1975) and have short latencies (Breitmeyer, 1975). Furthermore, the 'location' masking task used here is thought to further increase magnocellular involvement (Cadenhead et al., 1998). The masking procedures and stimuli (Fig. 2) were those used by Green et al. (2002). The 'target' was a square with a small gap in one of its sides (the gap was irrelevant for the version of the task used here). The square could appear in any one of four locations on the screen (upper left, upper right, lower left, lower right). For each trial, the subjects were asked to state the location of the target. Each target subtended 0.27° of visual angle and was located 1.03° of visual angle from the fixation cross, which was presented 400 ms before each target presentation for 300 ms. The target's contrast was set for each subject with a thresholding procedure. This produces a grey scale value called the 'critical stimulus intensity' at which all subjects can see the unmasked target on approximately 84% of trials. The 'mask' consisted of a 4×4 array of adjacent boxes (a 2×2 array in each quadrant) that appeared together in the same spatial locations as the target could appear, i.e., target and mask were superimposed in one of the quadrants. The duration of the target was 12.5 ms and the duration of the mask was 25 ms. The 'stimulus onset asynchrony' (SOA) is the time between the onset of the stimulus and the onset of the mask. Twelve SOAs ranging from −75 (forward masking) to 75 ms (backward masking) were presented in randomized fashion, 12 trials were presented for each SOA and the four possible locations of the target were counterbalanced (an SOA of 0 where mask and target are displayed simultaneously was also presented but was not used in the analysis). Percentage correct rates for each of the six backward and six forward making SOAs were averaged and used as the masking performance score, as analysis revealed there was no differential performance between groups on the forward versus backward trials. Higher values indicated better performance. A 'no-mask' condition was randomly interspersed within the masking trials and the observed equal performance between groups suggested that attentional problems in the patient group would not account for any deficit on this task.

2.2.1.3. Global motion (GM). Global motion is determined in cortical area V5/MT (Newsome and Paré, 1988), a dorsal stream area (Ungerleider and Haxby, 1994). The stimulus was a 10° of visual angle circular area containing 100 moving dots. Each dot was 8.5 min arc in diameter. A percentage of the dots moved upwards or downwards (signal dots) at 2.86°/s, whilst the remaining dots (noise dots) moved in random

Fig. 1. Illustration of 'Landolt C' figures used in contrast sensitivity testing. The orientation of the gap varies on each trial. The luminance contrast of the figure against the background varies until threshold is approximated.