Selective impairment of global motion integration, but not global form detection, in schizophrenia and bipolar affective disorder

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ABSTRACT

Recent evidence suggests that schizophrenia is associated with impaired processing of global visual motion, but intact processing of global visual form. This project assessed whether preserved visual form detection in schizophrenia extended beyond low-level pattern discrimination to a naturalistic form-detection task. We assessed both naturalistic form detection and global motion detection in individuals with schizophrenia spectrum disorder, bipolar affective disorder, and healthy controls. Individuals with schizophrenia spectrum disorder and bipolar affective disorder were impaired relative to healthy controls on the global motion task, but not the naturalistic form-detection task. Results indicate that preservation of visual form detection in these disorders extends beyond configural forms to naturalistic object processing.

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Schizophrenia is characterised by widespread perceptual abnormalities (Butler et al., 2008; Heinrichs and Zakzanis, 1998; Javitt, 2009). In particular, individuals with schizophrenia show problems with tasks requiring integration of spatiotemporally local stimulus elements into coherent context-sensitive percepts (Silverstein and Keane, 2011; Uhlhaas and Silverstein, 2005). In the temporal domain, whilst local motion perception shows no impairment, integration of local elements into a global percept of motion is compromised in schizophrenia (Chen et al., 2003). This effect could be mediated by a general failure to effectively integrate signals across both space and time; this theory suggests that as well as global motion, global form processing may be impaired in schizophrenia (e.g. Keane et al., 2014).

However, a recent comparison of form and motion perception using dot-patterns suggested a greater motion deficit than form perception deficit in schizophrenia (Brittain et al., 2010), implying some dissociation between the two domains. In the present study we investigated this question by studying a naturalistic form-detection task in schizophrenia and healthy controls, and comparing performance on this task to performance on a standard motion integration task. The form-detection task assessed detection sensitivity for two natural object types—faces and flowers—thereby also testing the hypothesis that schizophrenia is associated with a specific face processing deficit (Chen et al., 2008; Frith et al., 1983; Williams et al., 1999). In addition, we also assessed individuals with bipolar affective disorder, to test the hypothesis that these patients experience patterns of visual deficits similar to those with schizophrenia (Carter et al., submitted; Hill et al., 2014).

Participants were inpatients in an acute adult psychiatry inpatient unit in Melbourne, Australia, and were compared to age-matched healthy controls from the general population. Inpatients had a primary diagnosis of either a schizophrenia spectrum disorder (SSD; schizophrenia, schizoaffective disorder or schizophreniform disorder) or bipolar affective disorder. Diagnoses were made independently of investigators by a consultant psychiatrist and multidisciplinary clinical team using DSM-IV criteria. Exclusion criteria were intellectual disability, traumatic brain injury, stroke, or neurodegenerative disease. Additional exclusion criteria for healthy control participants were psychotropic medication or illicit substance use within two weeks of testing, or personal history of psychiatric illness. All participants had normal or corrected-to-normal vision. Participants gave informed consent, and research protocols were approved by Melbourne Health and University of Melbourne Human Research Ethics Committees. Participants’ IQ was estimated using the National Adult Reading Test (NART; Blair and Spreen, 1989; Crawford et al., 1992), and handedness was assessed using the Edinburgh Handedness Inventory (Oldfield, 1971). Benzodiazepine doses were converted to diazepam equivalents, and antipsychotic doses were converted to chlorpromazine equivalents (Bezchlibnyk-Butler et al., 2013; Woods, 2003). Table 1 presents summary demographic statistics.

In the form perception task, participants reported the presence or absence of objects (faces or flowers; Fig. 1) in visual noise arrays (Partos et al., 2015).
were 9 × 9 cm visual displays subtending 9 × 9° visual angle, No feedback was provided during testing. Participants entered responses into the testing computer. Prior to each task, participants were seated comfortably in a dimly lit room, using a chin rest 57 cm from the viewing distance. Task order counterbalanced between participants. Participants were randomly assigned to a block's target objects were faces orowers. As a result of computer error, data for one participant were unavailable for analysis.

Table 1
Participant demographic characteristics for form and motion tasks*.

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenia spectrum disorder</th>
<th>Bipolar affective disorder</th>
<th>Healthy control</th>
</tr>
</thead>
<tbody>
<tr>
<td>n completing form task</td>
<td>24a</td>
<td>11b</td>
<td>13</td>
</tr>
<tr>
<td>n completing motion task</td>
<td>14a</td>
<td>8a</td>
<td>35</td>
</tr>
<tr>
<td>Gender (form)</td>
<td>17 M, 5 F</td>
<td>5 M, 6 F</td>
<td>6 M, 7 F</td>
</tr>
<tr>
<td>Gender (motion)</td>
<td>11 M, 3 F</td>
<td>5 F, 3 M</td>
<td>20 M, 15 F</td>
</tr>
<tr>
<td>Handedness (form)</td>
<td>21R, OL, 1A</td>
<td>10R, 1 L</td>
<td>13R, 0 L, 0A</td>
</tr>
<tr>
<td>Handedness (motion)</td>
<td>13R, 0 L, 1A</td>
<td>8R, 0 L</td>
<td>32R, 2 L, 1A</td>
</tr>
<tr>
<td>Age [SEM] (motion)</td>
<td>38.9 ± 3.06</td>
<td>40.43 ± 3.80</td>
<td>40.38 ± 3.81</td>
</tr>
<tr>
<td>Age [SEM] (form)</td>
<td>40.00 ± 3.94</td>
<td>41.75 ± 4.67</td>
<td>39.11 ± 2.28</td>
</tr>
<tr>
<td>IQ estimate' [SEM] (form)</td>
<td>103.23 ± 2.55</td>
<td>102.23 ± 3.61</td>
<td>111.6 ± 2.61</td>
</tr>
<tr>
<td>IQ estimate' [SEM] (motion)</td>
<td>98.26 ± 2.48</td>
<td>104.01 ± 4.57</td>
<td>108.87 ± 3.50</td>
</tr>
<tr>
<td>Chlorpromazine dose [mg] [SEM] (form)</td>
<td>569.93 ± 65.68</td>
<td>404.09 ± 66.58</td>
<td>--</td>
</tr>
<tr>
<td>Chlorpromazine dose [mg] [SEM] (motion)</td>
<td>552.25 ± 97.65</td>
<td>364.00 ± 55.64</td>
<td>--</td>
</tr>
<tr>
<td>Benzodiazepine dose [mg] [SEM] (form)</td>
<td>19.89 ± 7.31</td>
<td>15.45 ± 5.11</td>
<td>--</td>
</tr>
<tr>
<td>Benzodiazepine dose [mg] [SEM] (motion)</td>
<td>19.11 ± 8.17</td>
<td>17.5 ± 6.75</td>
<td>--</td>
</tr>
<tr>
<td>DOI° [days] [SEM] (form)</td>
<td>4168.8 ± 684.31</td>
<td>3620.82 ± 1107.83</td>
<td>--</td>
</tr>
<tr>
<td>DOI° [days] [SEM] (motion)</td>
<td>4420.89 ± 942.85</td>
<td>3563.13 ± 1003.33</td>
<td>--</td>
</tr>
</tbody>
</table>

* All inpatients completed the form task, and a subset of inpatients also completed the motion task. Two healthy control participants completed both form and motion tasks.

† Including 18 with schizophrenia, three with schizoaffective disorder, and one with schizophreniform psychosis.
‡ Including ten participants in a manic episode at time of testing, and one in a depressed episode.
§ Including seven participants in a manic episode at time of testing, and one in a depressed episode.
‖ Due to dyslexia or illiteracy, IQ estimates were not available for two individuals in the schizophrenia group. In addition, two healthy control participants did not complete the NART.
† Duration of illness (DOI), calculated as elapsed days from assignment of current diagnosis to day of testing for patients with more than one admission; for first episode patients DOI was calculated from the day of admission to day of testing.

et al., submitted). Stimuli were 12.3 × 12.3 cm 8-bit greyscale arrays, subtending 12.3 × 12.3° visual angle. Sensitivity was measured as $d'$, and response bias as $c$ (Stanislaw and Todorov, 1999; Tanner and Swets, 1954).

In the motion integration task, participants reported the direction of motion (left or right) of coherently moving ‘signal’ dots amongst randomly moving ‘noise’ dots (Newsome and Pare, 1988). Stimuli were 9 × 9 cm visual displays subtending 9 × 9° visual angle, containing 300 black and white dots on a grey background (Fig. 2). Sensitivity was calculated as the inverse of coherence threshold.

Form and motion tasks were completed in separate sessions, with task order counterbalanced between participants. Participants were seated comfortably in a dimly lit room, using a chin rest 57 cm from the monitor. Participants verbally reported responses to a researcher, who entered responses into the testing computer. Prior to each task participants completed a practice block with accurate task feedback. No feedback was provided during testing.

In form detection, a 3 × 2 mixed-design ANOVA revealed no significant effect of diagnostic group (schizophrenia spectrum disorder, bipolar, control) on sensitivity, $F(2,42) = 1.07, p = .35$ (Fig. 3a). However, there was a main effect of image type, with poorer sensitivity for faces ($M = 1.32, SEM = 0.07$) than flowers ($M = 1.46, SEM = 0.09$), $F(1,42) = 4.44, p < .05, \eta^2 = .10$. However, the interaction between diagnostic group and image type was not significant, $F(2,42) = 1.01, p = .37$, indicating that this pattern was observed across all diagnostic groups and is therefore likely to reflect differences in stimulus properties (such as symmetry) rather than visual pathology. This is consistent with the view that schizophrenia is associated with extensive general visual impairments, rather than a specific face-processing deficit (Darke et al., 2013). A second 3 × 2 mixed-design ANOVA revealed a main effect of image type on response bias, $F(1,42) = 7.07, p < .05$, with participants more likely to report seeing faces than flowers. However, there was no main effect of diagnostic group on response bias, $F(2,42) = 0.23, p = .79$, .
In motion integration, a Kruskal–Wallis test revealed significant differences in motion coherence sensitivity between the three diagnostic groups (schizophrenia spectrum disorder, bipolar, control) $\chi^2(2, n = 57) = 33.93, p < .001$ (Fig. 3b). Post-hoc Mann–Whitney U tests with Bonferroni adjustment for multiple comparisons revealed that sensitivity was significantly poorer amongst both participants with schizophrenia spectrum disorder, $U = 7$, $p < .001$, and bipolar, $U = 26$, $p < .001$, relative to healthy control participants, but did not differ significantly between the two patient groups, $U = 40$, $p = .28$.

Multiple linear regression indicated that form and motion task performance were unrelated to possible confounds including medication dosage, IQ, or illness duration (all $p > .05$).

In summary, the present study found that inpatients with schizophrenia spectrum disorder or bipolar diagnoses were impaired in global motion detection, but not naturalistic global form detection, relative to healthy controls. This is consistent with the finding that low-level visual form perception was intact amongst individuals with schizophrenia who displayed impaired global motion perception (Brittain et al., 2010). However, our results are somewhat incongruous with past research demonstrating abnormal form processing in schizophrenia spectrum disorders (Chapman, 1966; Klosterkötter et al., 2001). This apparent inconsistency may be the result of different task demands in different studies of form processing in schizophrenia spectrum disorder. Rather than identification or discrimination of forms, the present study assessed the ability to detect forms embedded within visual noise. As such, the task may have assessed a relatively early stage of form processing, prior to integration of information into a coherent percept. It is the latter function which is thought to be particularly compromised in schizophrenia spectrum disorder, even where early stages of form processing are intact (Keane et al., 2014; Uhlhaas and Silverstein, 2005).

The present study found for the first time that this pattern of differential visual dysfunction was also present in individuals with bipolar affective disorder, consistent with previous findings showing that across diagnoses, the presence or absence of psychotic symptoms gives a more parsimonious explanation of patterns of sensory impairment than diagnosis (Carter et al., submitted). However, although patterns of sensory impairment were similar between schizophrenia spectrum disorder and bipolar in the present study, previous research has shown that in other psychophysical tasks, these disorders are associated with distinct patterns of sensory deficits (e.g. Chen et al., 2006). Moreover, the bipolar sample in the present study was relatively small, and as such it is possible that meaningful differences in form detection between these disorders may become apparent in future studies involving larger patient numbers. In addition, whereas some previous research has observed lower IQs in schizophrenia spectrum disorder than bipolar affective disorder (Seidman et al., 2002), the present study found no significant
difference in IQ between these disorders; it is therefore unclear how our results will generalise to the wider patient population. Finally, we note that since variability in visual acuity within the normal range can account for significant differences in visual task performance (Keane et al., 2015), we cannot rule out subtle differences in acuity between diagnostic groups as a possible confound of results.

The results of the present study demonstrate that individuals with schizophrenia spectrum disorder and bipolar affective disorder display a dissociation between form and motion processing. This pattern is of particular interest since a prominent theory proposes that the primate visual system is divided into ventral ‘what’ (or form) and dorsal ‘where’ (or motion) cortical pathways (Mishkin and Ungerleider, 1982). One interpretation of our results is that individuals with schizophrenia spectrum disorder and bipolar affective disorder may be more impaired in functions subserved by the latter pathway. Such a deficit might be the product of an impairment of magnocellular relative to parvocellular signalling in schizophrenia (Brittain et al., 2010; Doniger et al., 2002). However, we also note that this simple anatomy–behaviour link has been disputed (Skottun and Skoyles, 2007), and it is also possible that a generalised processing deficit in schizophrenia and bipolar affective disorder may have more strongly affected performance on the motion task (Chapman and Chapman, 1978). Alternatively, a dissociation between form and motion processing might result from a general deficit in sensory integration, since motion processing introduces an additional dimension (time) across which to judge context.

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Contributors

Authors SS and OC designed and supervised the project. Authors DB and AD collected data. Authors DB, SJC and TP contributed to task design and data analysis. Authors DB, SJC, SS and OC contributed substantially to writing the manuscript.

Conflict of Interest

The authors declare that they have no conflicts of interest to report.

Conflict of interest statement

Dr Carter has received research and salary support from the Australian National Health and Medical Research Council. Dr Sundram has received consulting fees, advisory board fees, research support, speaker’s honoraria or travel support from AstraZeneca, Bristol-Myers Squibb, Eli Lilly, Flack trust, GlaxoSmithKline, the Australian National Health and Medical Research Council, One-in-Five Association, Pfi zer and Roche. All other authors declare that they have no conflicts of interest.

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