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Neural basis of altered physical and social causality judgements in schizophrenia

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ABSTRACT

Patients with schizophrenia (SZ) often make aberrant cause and effect inferences in non-social and social situations. Likewise, patients may perceive cause-and-effect relationships abnormally as a result of an alteration in the physiology of perception. The neural basis for dysfunctions in causality judgements in the context of both physical motion and social motion is unknown. The current study used functional magnetic resonance imaging (fMRI) to investigate a group of patients with SZ and a group of control subjects performing judgements of causality on animated collision sequences (launch-events, Michotte, 1963) and comparable "social" motion stimuli. In both types of animations, similar motion trajectories of the affected object were configured, using parametrical variations of space (angle deviation) and time (delay).

At the behavioural level, SZ patients made more physical and less social causal judgements than control subjects, and their judgements were less influenced by motion attributes (angle/time delay). In the patients group, fMRI revealed greater BOLD-responses, during both physical and social causality judgements (group × task interaction), in the left inferior frontal gyrus (L.IFG). Across conditions (main effect), L.IFG-interconnectivity with bilateral occipital cortex was reduced in the patient group.

This study provides the first insight into the neural correlates of altered causal judgements in SZ. Patients with SZ tended to over-estimate physical and under-estimate social causality. In both physical and social contexts, patients are influenced less by motion parameters (space and time) than control subjects. Imaging findings of L.IFG-disconnectivity and task-related hyper-activation in the patient group could indicate common dysfunctions in the neural activations needed to integrate external cue-information (space/time) with explicit (top–down) cause–effect judgements of object motions in physical and social settings.

1. Introduction

Patients with schizophrenia (SZ) often have aberrant views of causality, observed in delusional ideation or in ideas of reference. At the level of cognition, deviant causal inferences occur in biased (Moritz and Woodward, 2005) and impaired reasoning (Kruck et al., 2011), as well as altered inferences about other people's mental states and social interactions (Horan et al., 2009; Herrington et al., 2011). At the level of perception, psychotic patients might also experience cause and effect differently: for instance, positive symptoms of psychosis (delusions) are associated with increased impressions of physical causality in visual events (Tschacher and Kupper, 2006).

Whether abnormal cognitive and perceptual processing contributing to causal inferences in SZ have a common basis is not known. Some suggest that disturbed interactions of bottom–up sensory processing and top–down attribution (of priors, or beliefs, see e.g. Corlett et al., 2009; Fletcher and Frith, 2009) might be the common basis for psychotic symptoms (Hemsley and Garety, 1986; Grossberg, 2000; Young, 2008). Particularly, impaired beliefs (e.g. delusions) and perceptions (e.g. hallucinations) about causal relations (Corlett et al., 2006; Corlett et al., 2007) could reflect disturbed integration of relevant stimulus information (Corlett et al., 2011).

The most direct cause–effect perception arises from a physical collision, also known as a launching event (Michotte, 1963). Launching stimuli are simple animations that typically show one geometric object, e.g. a billiard-like ball, moving towards and making contact with another object which then moves on. Observers have the impression of a collision, i.e. a causal relation between the objects; i.e. A caused B to move (Scholl and Tremoulet, 2000; Scholl and Nakayama, 2002).

Similarly, the impression of a social interaction can be induced by simple moving objects, which are perceived as animate (Heider and Simmel, 1944; Blos et al., 2012). This attribution of animacy is peculiar

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to social as opposed to physical causal events (Schlottmann et al., 2006). To be interacting socially, traits of living entities like intentions must be attributed to objects; a social cognitive process referred to as mentalizing (Tremoulet and Feldman, 2006).

Stimulus motion attributes in time and space are relevant to the impressions of cause and effect in both physical and social events. In physical events, spatial and temporal violations of motion contingencies lead to more non-causal judgements of launching events (Young et al., 2005; Young and Falmier, 2008). By contrast, in social events, the same violations of motion produce more causal responses (Scholl and Tremoulet, 2000; Falmier and Young, 2008; Blos et al., 2012).

Initial behavioural research suggests, that patients with delusions tend to make different judgements of causality compared to healthy subjects in both physical (Blakemore et al., 2003; Tschacher and Kupper, 2006) and social motion events (Blakemore et al., 2003). Other perceptual deficits in "Gestalt"-domains (e.g. in perceptual grouping; see Silverstein et al., 2000) are related to disorganization symptoms in psychosis (Uhlhaas and Silverstein, 2005). These altered "Gestalt"-perceptions in SZ could reflect general dysfunctions in perceptual organisation needed to integrate stimulus-attributes, i.e. motion parameters (Tschacher and Kupper, 2006). Whether SZ patients have trouble integrating spatio-temporal parameters when judging causality in physical and social motion contexts is not known.

The neural mechanisms of cause–effect inferences are still under investigation. Evidence from brain lesion studies and neuroimaging suggests a distinction between automatic causality perception and cognitive inference (Blakemore et al., 2001; Fonlupt, 2003; Fugelsang et al., 2005; Roser et al., 2005). Recently, our group conducted a set of functional magnetic resonance imaging (fMRI) studies using healthy volunteers as participants, to investigate the neural correlates of causal judgements on simple motion stimuli in physical and social contexts (Blos et al., 2012; Wende et al., 2013). Causality judgements, as compared to judgements of movement direction, engaged a fronto-parietal network (Wende et al., 2013). Similar patterns of neural activity have been associated with explicit (top–down) inferences about visual–spatial ("perceptual") attributes during reasoning (Kranjec et al., 2012; Straube et al., 2011).

Previous psychophysiological research indicates that SZ patients may have altered experiences of launching events (Adams et al., 2012), but the neural basis of these observations remains unclear. Inference tasks used to investigate reasoning engage bilateral middle/inferior frontal cortex regions (Goel and Dolan, 2004; Goel, 2007; Rodriguez-Moreno and Hirsch, 2009; Prado et al., 2011; Watson and Chatterjee, 2012). In psychosis, frontal/prefrontal cortex dysfunction could be a common neural basis underlying inference deficits on external social (Lee et al., 2004) and visual–spatial information (Lee et al., 2008). A similar proposal comes from interactionist ("Bayesian") models of psychosis (Fletcher and Frith, 2009) and model psychosis studies (i.e. studies, in which healthy volunteers are induced with psychotic symptoms using psychoactive drugs like ketamine to investigate underlying neural mechanisms, see e.g. Corlett et al., 2009). This line of research links the formation of altered "heuristics", i.e. delusional beliefs, to abnormal neural responses in frontal cortices (Corlett et al., 2006). Abnormal frontal response patterns are directly related to psychotic patients' deficits in making predictive inferences (Corlett et al., 2007).

More recent imaging work associates increased neural activity in SZ patients in inferior and middle frontal brain regions, in response to impaired social inferences in visual events (Pedersen et al., 2012). Impaired social (biological) motion perceptions might reflect similar dysfunctions in integration of external visuo-spatial motion parameters to higher-order cognitive domains (Kim et al., 2005). Indeed, recent imaging evidence indicates a reduced functional connection of the frontal lobe (central to cognitive functions) and posterior cortex regions (relevant for stimulus-information processing) in psychosis (Pettersson-Yeo et al., 2011; Straube et al., 2014). Stimulus-motion attributes (space/time) provide a crucial basis for "Gestalt"-perceptions of causality, thus dysfunctions in the perceptual integration of those parameters could provoke altered judgement behaviour (Tschacher and Kupper, 2006). However, the specific effects on causality judgements based on spatial and temporal stimulus motion characteristics are not known. We are not aware of any imaging study that has investigated the judgements of physical and social causality in patients with SZ. In the context of deviant physical/social causal inferences in psychosis, it would be particularly interesting to assess patients' neural correlates in causal judgements about collisions (Michotte, 1963) and comparable social motion stimuli.

In the present study, patients and control subjects were monitored in fMRI while judging causal relationships (causal/non-causal) in contrast to movement direction (left/right, control task) of abstract moving objects. Animations were configured using equally varied spatiotemporal motion parameters (angle/time delay) for physical (collisions) and social (no collisions) contexts.

We aimed to investigate the common and distinct neural correlates of causal judgements (task effect) and context (social/physical) for patients with SZ and healthy control subjects.

Behaviourally, we expected that patients' responses would deviate from control subjects regarding the use of spatial and temporal information for their judgements, reflecting perceptual or inferential impairments of SZ patients, e.g. biases (Tschacher and Kupper, 2006).

At the neural level, we expected causality judgements to evoke common neural activity in both groups (task effect) in frontal and parietal cortex regions, a neural network confirmed to be active in tasks involving causal inferences (Kranjec et al., 2012; Watson and Chatterjee, 2012) and causality judgements (Fugelsang et al., 2005; Wende et al., 2013).

We further expected altered neural responses in the patient group located in bilateral middle/inferior frontal cortex regions associated with inferences and reasoning (Goel and Dolan, 2004; Goel, 2007; Rodriguez-Moreno and Hirsch, 2009; Prado et al., 2011; Watson and Chatterjee, 2012). Possibly, causality judgements would result in overactivation of frontal brain regions in the patient group, as recently shown for social contexts (Pedersen et al., 2012).

We additionally hypothesized reduced connections of frontal and posterior brain regions to reflect dysfunctions indicated by behavioural deviance regarding the use of motion parameters (space/time) for causal judgements in the patient group; particularly, integration of sensory information with cognitive processes (Pettersson-Yeo et al., 2011; Zalesky et al., 2011; Straube et al., 2014).

2. Methods

2.1. Subjects

Eighteen patients with schizophrenia or schizoaffective disorder and a group of eighteen control subjects (HC) matched for gender, age (SZ/HC = 35.56/34.22 years, SD = 13.48/11.09, $t = 0.32$, $p = .71$) and years of education (SZ/HC = 10.17/10.94 years, SD = 1.34/1.11, $t = -1.90$, $p = .07$), were included in the study. All patients were on stable doses of medication and none of them had acute symptoms at time of study (Andreasen et al., 2010). One patient who was diagnosed with schizotypic disorder (F21.0) was excluded from the analysis. One patient diagnosed with schizoaffective disorder (F25.2) was included as effects of interest (symptoms) are similar in both diagnoses. The exact doses of medication for two patients and the SAPS/SANS scores of one patient were missing. ICD10-diagnoses were confirmed by two independent clinical interviews with trained clinicians ($n = 16$ paranoid schizophrenia (F20.0), $n = 1$ hebephrenic schizophrenia (F20.1), and $n = 1$ schizoaffective disorder (F25.2), see Table 1). Patients were recruited at the Department of Psychiatry and Psychotherapy at the Philipps-University Marburg and SAPS and SANS ratings were used to assess their symptoms. Healthy controls were recruited via postings at the Philipps-University Marburg. All subjects had normal or corrected-