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Holding on to false beliefs: The bias against disconfirmatory evidence over the course of psychosis

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ABSTRACT

Background and Objectives: The ability to integrate evidence into a reasoning process is crucial in order to react to changing information, e.g. to adapt one's beliefs according to new evidence or to generate new beliefs when facing better alternatives. Evidence integration ability is thus associated with belief flexibility. A specific bias of evidence integration, a bias against disconfirmatory evidence (BADE), can be found in patients with schizophrenia and has been linked to delusion development and maintenance. Knowledge about whether the BADE occurs already in risk constellations of psychosis can clarify its role in the pathogenesis of psychosis.
Methods: This article reviews the current literature on BADE. Many studies demonstrate BADE over the course of illness, ranging from healthy controls with subclinical properties of schizotypy, over patients with at-risk mental states (ARMS) and patients with a first episode of psychosis to patients with chronic schizophrenia. These data allow a comparison of competences and deficits over the course of illness. Underlying mechanisms of BADE are discussed, including interrelations with neurocognitive performance and dopaminergic processes.
Results: The BADE could be found in different phases of psychosis development and can be regarded as a cognitive marker of the beginning psychotic state.
Limitations: The presented findings are derived from independent cross-sectional studies. So far, no comprehensive longitudinal assessment has been published.
Conclusions: Treatments of metacognitive deficits in general and as early as in the ARMS might interfere with the cognitive pathogenesis of psychosis, and thereby ameliorate, postpone or even prevent the transition to psychosis.

Contents

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1. Introduction

The diagnostic category "schizophrenia" represents a spectrum of psychotic disorders characterized by disturbances in perception, thought, language, emotion and sense of self (Owen, Sawa, & Mortensen, 2016). The presence of delusions, hallucinations and formal thought disorders, the so-called positive symptoms, is crucial for the diagnosis according to international classifications. The definition of delusions goes as far back as to Karl Jaspers in 1913 who defined three criteria of delusional thinking. He specified delusions as beliefs held with strong conviction, incorrigible even in view of counterarguments and impossible or implausible in their content (Jablensky, 2013). Today, the view that delusions are unamenable to change, for example by psychotherapy, is not supported anymore. In contrast, a lot of evidence underlines the changeability of delusional beliefs (e.g. Mehl, Werner, & Lincoln, 2015; Moritz, Andreou et al., 2014; Wykes, Steel, Everitt, & Tarrier, 2008). In more detail, delusions reflect a compound of cognitive processes that are responsible for changes in thinking and reasoning (Miller & Karoni, 1996). In recent years, cognitive models have been promoted in the attempt to understand cognitive mechanisms of delusions and of positive symptomatology in general (e.g. Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). These models add to the before much more investigated physical and environmental factors to schizophrenia (van Os, Kenis, & Rutten, 2010) and have opened a wide field of new experimental research. Cognitive models of delusions have mainly concentrated on monitoring and control processes, so-called metacognitive processes (Flavell, 1979) and their relation to reasoning, decision making, memory processing and information integration (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Freeman et al., 2004; Garety & Freeman, 1999). It is suspected that a combination of several biased cognitive processes contribute to the formation and maintenance of beliefs (Garety et al., 2005). These include, for example, a metamemory bias (Eifler et al., 2015; Eisenacher et al., 2015), a jumping to conclusion bias (Rausch et al., 2014, 2015, 2016), a Theory of Mind-deficit (Bora, Yucel, & Pantelis, 2009; Mier et al., 2010; Thompson et al., 2012) and attributional biases (e.g. an external-personal attributional style) (Berry, Bucci, Kinderman, Emsley, & Corcoran, 2015; Randjbar, Veckenstedt, Vitzthum, Hottenrott, & Moritz, 2011).

To understand the role of cognitive biases in the pathogenesis of psychosis, it is not sufficient to investigate patients with an established diagnosis of schizophrenia. Psychotic symptoms have been found dimensionally distributed along a continuum. They range from healthy people with psychosis-like experiences and high scores of schizotypy over patients with at-risk mental states (ARMS), those with a first episode of psychosis to patients with multiple episodes of psychosis (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). Especially the early detection and treatment of those patients with risk constellations for psychosis has attracted attention in recent years because of the potential to attenuate, delay or even prevent a first psychotic episode (Schmidt et al., 2015; Schultze-Lutter et al., 2015). Amongst other diagnostic instruments the Early Recognition Inventory based on the Iraos (ERIraos) has been established and evaluated as a comprehensive tool for the assessment of basic symptoms (BS), attenuated psychotic symptoms (APS) and brief limited intermittent psychotic symptoms (BLIPS) with high sensitivity (Eisenacher, Rausch, Ainser, et al., 2016; Maurer, Zink, Rausch, & Häfner, 2016; Rausch et al., 2013). Accordingly, the investigation of metacognitive processes along the continuum of psychosis renders the possibility for an early detection and treatment of cognitive mechanisms and a mediation of the development of positive symptoms.

2. Belief flexibility and evidence integration

Once a belief has been formed, it is reasonable to steadily consider new information, to adapt one's belief according to changing evidence if necessary, and to generate new beliefs on the basis of better alternatives. This metacognitive ability has been termed belief flexibility (Garety et al., 2005). The opposite, not being able to change beliefs in the presence of new knowledge, may lead to an adherence of false beliefs. It was hypothesized that safety behaviors, such as avoiding potentially fearful situations or preparing for an attack, pronounce this lack of belief flexibility (Garety et al., 2001). A disturbance in the flexibility towards and integration of disconfirmatory evidence is especially crucial in the discussion of delusional thinking because it entails holding a false belief with strong conviction though it has been proven to be false. This feature is a hallmark of delusions as defined by Karl Jaspers (Jablensky, 2013). In this sense, evidence integration disturbances are assumed to be associated with delusional thinking.

Many recent studies have concentrated on metacognitive abilities in schizophrenia and have found an inflexibility of beliefs (Colbert, Peters, & Garety, 2010; So et al., 2012), biases in evidence integration (e.g. Colbert et al., 2010; Speechley, Ngan, Moritz, & Woodward, 2012) and specifically biases in the integration of disconfirmatory evidence (BADE) (Eifler et al., 2014; Woodward, Moritz, Cuttler, & Whitman, 2006; Woodward, Moritz, Menon, & Klinge, 2008). The BADE constitutes a failure to integrate new evidence, which contradicts a held belief, into a reasoning and decision making process. A practical example illustrates this: Jenny is 33 years old, has a diagnosis of schizophrenia and has experienced three phases of acute psychosis. She is convinced that her neighbour secretly comes into her apartment at night and steals from her. She is even more convinced when she cannot find her credit card in her wallet. When she finally finds her credit card loosely in her purse which she usually uses only for grocery shopping, Jenny is not able to recognize this as evidence against her belief about her neighbour.

2.1. Assessing evidence integration

The research group around Woodward (Woodward, Moritz, Cuttler, et al., 2006) developed a specific paradigm to experimentally investigate BADE in patients with schizophrenia. At first, the