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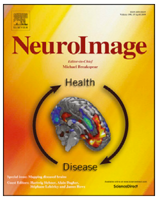

# Decision-making in schizophrenia: A predictive-coding perspective

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## ABSTRACT

Dysfunctional decision-making has been implicated in the positive and negative symptoms of schizophrenia. Decision-making can be conceptualized within the framework of hierarchical predictive coding as the result of a Bayesian inference process that uses prior beliefs to infer states of the world. According to this idea, prior beliefs encoded at higher levels in the brain are fed back as predictive signals to lower levels. Whenever these predictions are violated by the incoming sensory data, a prediction error is generated and fed forward to update beliefs encoded at higher levels. Well-documented impairments in cognitive decision-making support the view that these neural inference mechanisms are altered in schizophrenia. There is also extensive evidence relating the symptoms of schizophrenia to aberrant signaling of prediction errors, especially in the domain of reward and value-based decision-making. Moreover, the idea of altered predictive coding is supported by evidence for impaired low-level sensory mechanisms and motor processes. We review behavioral and neural findings from these research areas and provide an integrated view suggesting that schizophrenia may be related to a pervasive alteration in predictive coding at multiple hierarchical levels, including cognitive and value-based decision-making processes as well as sensory and motor systems. We relate these findings to decision-making processes and propose that varying degrees of impairment in the implicated brain areas contribute to the variety of psychotic experiences.

## Introduction

Decision-making is one of the most fundamental aspects of human behaviour. From deciding whether an approaching car is turning left or right to which career to pursue, decision-making involves processes ranging from basic sensory or sensorimotor functions to complex cognitive or value-based choices (Heekeren et al., 2008; Shadlen and Kiani, 2013; Sterzer, 2016). Dysfunctional decision-making may play role in a range of mental disorders, including schizophrenia, addiction, eating disorders, and affective disorders (So et al., 2016). In schizophrenia, impairments in decision-making have been implicated in both positive symptoms, which include delusions and hallucinations, and negative symptoms, such as apathy or social withdrawal. Positive symptoms, in particular delusions, have been related to a tendency to make decisions with certainty based on insufficient information (Evans et al., 2015; Garety and Freeman, 2013). This ‘jumping-to-conclusions’ (JTC) bias may result in a tendency to rapidly appraise ambiguous information and to thus form false beliefs based on limited evidence. In regard to negative symptoms, it has been proposed that alterations in reward processing may result in a failure to appropriately estimate and use future rewards to guide decision-making, thus resulting in lack of motivation and apathy (Deserno et al., 2017; Gold et al., 2008; Heinz and Schlagenhauf, 2010; Lee et al., 2015; Maia and Frank, 2017; Radua et al., 2015). With regard to pathophysiology, these observations raise the question whether alterations in different types of decision-making reflect distinct pathologies, or alternatively, may be explained by a more general dysfunction that has implications for multiple neural processes involved in decision-making.

A generic model of brain function that has the potential to link different levels of observation within a more general theory of schizophrenia is provided by the computational framework of predictive coding and Bayesian inference (Adams et al., 2013; Corlett et al., 2009; Fletcher and Frith, 2009). In this framework, the brain is conceived of as a hierarchical inference machine that uses predictions to infer the probable causes of the sensory signals (Friston, 2005; Lee and Mumford, 2003). Here, we propose that predictive coding provides a framework that may account for dysfunctional decision-making in schizophrenia by integrating findings from different research areas (and implicating different domains of neural processing) within one general model of brain function. We start by relating the neural processes that are thought to be involved in decision-making to the generic framework of predictive coding. We then outline the predictive-coding account of schizophrenia and review current evidence for dysfunctions that concern the domains most critically involved in decision-making, with a focus on neural data. In addition to cognitive and value-based decision-making, these domains include perceptual and sensorimotor processes (Heekeren et al., 2008;

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Shadlen and Kiani, 2013; Sterzer, 2016). Finally, we provide an integrative view of dysfunctional decision-making in schizophrenia within the framework of predictive coding and suggest directions for future research.

## Modeling decision-making within the framework of predictive coding

Decision-making is often conceptualized as neural evidence accumulation, which means that noisy pieces of evidence accumulate and give rise to a choice as soon as a decision threshold is reached (Gold and Shadlen, 2007; Heekeren et al., 2008; Mulder et al., 2014; Sterzer, 2016). The neural mechanisms that underlie this evidence accumulation have thus far been primarily considered as passive processes in which the decision maker is exposed to decision-relevant information without having to actively search for it. However, the brain not only monitors the outcomes of its decisions, but actively shapes the accumulation of evidence for these decisions on the basis of subjective expectations and preferences. The way we make decisions is thus strongly shaped by expectations and beliefs regarding the world.

Decision-making can thus be conceptualized within the notion of the brain as an inference machine, according to which prior beliefs are used to make inferences regarding the hidden states of the world (Friston and Kiebel, 2009; Summerfield and de Lange, 2014). These prior beliefs are derived from an internal "generative model" that represents knowledge about the world. The internal model is generative in the sense that it describes how sensory signals are generated by the external world. Predictive coding provides a computational framework that has been successful in modeling the neural mechanisms involved in such inference and decision-making. According to predictive coding, prior beliefs are encoded at higher hierarchical levels and are fed back as predictive signals to lower levels (Clark, 2013; den Ouden et al., 2012; Friston, 2005; Lee and Mumford, 2003; Rao and Ballard, 1999). Whenever the incoming sensory data violate these predictions, a prediction error (PE) signal is fed forward to update beliefs encoded at higher levels. In this way, PEs drive learning and enable a flexible adaptation to changes in the environment. Critically, these predictive mechanisms are thought to be organized in multiple hierarchical levels, with more abstract beliefs being encoded at higher levels of the hierarchy.

Hierarchical predictive coding may be formalized as a Bayesian inferential process in which prior beliefs ('prior') and sensory data ('likelihood') are represented in the form of probability distributions (Knill and Pouget, 2004; Lee and Mumford, 2003; O'Reilly et al., 2012; Shipp, 2016). Perceptual decisions correspond to the posterior probability, which results from the combination of prior beliefs and sensory data, weighted by their respective precisions (which is the inverse of their variance; Fig. 1). For example, if the precision of the sensory data is low relative to the precision of the prior belief (e.g., in the presence of sensory noise), the posterior will be influenced more strongly by the prior belief, which results in a perceptual decision that more strongly corresponds to the prior belief (Fig. 1A). Conversely, if the precision of the prior is low relative to the precision of the sensory data (e.g., in the absence of prior knowledge), the posterior and thus the resulting perceptual decision will be influenced more strongly by the sensory data (Fig. 1B). Bayesian models of hierarchical predictive coding as a basis for decision-making have recently been related to the notion of evidence accumulation delineated above. It has been proposed that evidence accumulation schemes can be viewed as a special case of generic Bayesian inference (FitzGerald et al., 2015; Friston et al., 2017), and recent modeling and neuroimaging work has established a link between evidence-accumulation schemes and Bayesian models of perceptual decision-making (Bitzer et al., 2014, 2015; Fard et al., 2017; FitzGerald et al., 2015). A key advantage of the use of the hierarchical Bayesian approach is that it can incorporate prior beliefs at different hierarchical levels into the modeling of the decision process. In the light of theories that have implicated altered inference as a core mechanism in schizophrenia (Adams et al., 2013; Corlett et al., 2009; Fletcher and Frith, 2009), hierarchical predictive coding may thus provide a useful framework for the understanding of dysfunctional decision-making in schizophrenia.

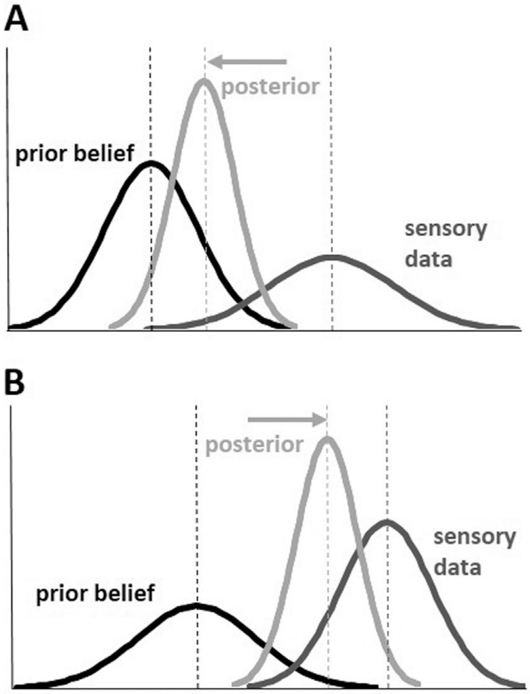

**Fig. 1. Bayesian decision-making**. The graphs show Gaussian probability distributions that represent estimates of prior beliefs (black), the likelihood of the sensory data (dark grey), and the posterior belief (light grey) for a given parameter of a sensory stimulus. The posterior determines the perceptual decision. (A) Higher precision of the prior belief shifts the posterior away from the sensory data and towards the prior belief. (B) Higher precision of the sensory data shifts the posterior towards the sensory data (modified from Adams et al., 2013).

## The predictive-coding account of schizophrenia

In recent years, hierarchical predictive coding has evolved as a promising framework to explain key symptoms of schizophrenia. It has been hypothesized that in schizophrenia, the balance between the precision of predictions and sensory data is altered, with a reduced precision of learned predictions relative to the precision in the neural representation of sensory data (Fig. 1B); and that this biases inference towards the sensory data and away from predictions, thus resulting in signaling of PEs with increased precision (Adams et al., 2013; Brown and Kuperberg, 2015; Corlett et al., 2009; Friston et al., 2014b; Mishara and Sterzer, 2015; Sterzer et al., 2018; Sterzer et al., 2016). Such inappropriate neural PE signals imbue normally inconspicuous sensory stimuli with too much salience, and it is this misattribution of salience (or 'aberrant salience') that is thought to give rise to complex accounts of why these stimuli are important (Heinz, 2002; Kapur, 2003). This theory, which was originally developed with regard to dopamine-encoded PEs (Robinson and Berridge, 1993; Schultz et al., 1997), suggests that attribution of salience to otherwise irrelevant stimuli contributes to "delusional mood", an early stage in the formation of delusions, when patients note threatening signs and messages that appear to be personally relevant (Heinz, 2002). Further studies and reviews suggested that the main neurotransmitter alterations underlying altered predictive coding in schizophrenia are a hypofunction of cortical NMDA receptors and GABAergic neurons in addition to elevated striatal dopamine turnover (Adams et al., 2013; Corlett et al., 2009; Corlett et al., 2011; Fletcher and Frith, 2009; Heinz and Schlagenhauf, 2010; Jardri et al., 2016; Juckel et al., 2006).

## Cognitive biases in decision-making

When it comes to dysfunctional decision-making in schizophrenia, the first line of research that comes to mind is related to JTC. Hemsley and Garety (1986) suggested that “it is a weakening of the influence of stored memories of regularities of previous input on current perception which is basic to the schizophrenic condition”. This idea was supported empirically by evidence from the so-called “beads task” (Phillips and Edwards, 1966) an experimental paradigm that probes the integration of new evidence with stored knowledge. In the original version of this task, subjects are first presented with two jars of beads, one with mostly pink beads and a few green beads and another jar with mostly green and a few pink beads. Participants are then shown a sequence of beads and are asked after each draw whether they are ready to decide which jar the beads were drawn from. People with delusions were found to make their decisions after significantly fewer draws than controls, a finding that was interpreted as reflecting a JTC bias (Huq et al., 1988), and that was replicated in a large number of studies (Dudley et al., 2016; Evans et al., 2015; Ross et al., 2015). However, the neural processes involved in such apparently “hasty decision-making” in relation to delusions has remained controversial. A plausible explanation for JTC is that each piece of new evidence is relatively “overvalued” relative to the knowledge acquired on the basis of the previous draws, which in terms of hierarchical predictive coding points to a reduced precision of prior beliefs and, consequently, a greater influence of new evidence (Adams et al., 2016; Speechley et al., 2010). Bayesian modeling has suggested that JTC reflects greater “cognitive noise” in delusional patients (Moutoussis et al., 2011), which indeed speaks to a reduced precision of prior beliefs (Adams et al., 2013). Moreover, a recent study used a probabilistic reasoning task in conjunction with computational modeling to show that delusional ideation and hallucinatory experiences are associated with a low resilience against irrelevant information, thus giving rise to overhasty and erroneous inferences (Stuke et al., 2017).

So far, relatively little is known about the neural correlates of cognitive biases in decision-making. A functional magnetic resonance imaging (fMRI) study with a modified beads task using a block design in healthy subjects found that probabilistic reasoning preceding the decision was associated with brain activation in a prefrontal-parietal network implicated in executive functions, while during the actual decision process, functional activation was detected predominantly in regions of the dopaminergic midbrain and ventral striatum as well as anterior insular cortex (Esslinger et al., 2013). This latter finding was interpreted as potentially providing a link between salience processing and decision-making on the basis of probabilistic reasoning. In patients with schizophrenia, an fMRI study found decreased activations in prefrontal and parietal areas compared to matched controls (Krug et al., 2014), thus providing a potential neural correlate of altered probabilistic reasoning. However, this result has to be interpreted with caution, as the classical behavioral JTC effect, i.e. fewer draws to decision in patients with schizophrenia, was not replicated in this study. Moreover, it should be noted that the block design in this study makes it difficult to differentiate between the neural correlates of the two types of choices that are possible in the beads task, i.e., either to see more draws or to make a decision for one urn. JTC occurs when a choice is made in favor of choosing an urn, so brain circuits active during urn choice events are likely to be involved in JTC. Studies in healthy volunteers that directly compared fMRI responses to urn choices versus draw choices found similar functional activations as observed in the block-design contrasts, including activations in the striatum and the anterior insula associated with decision making (Esslinger et al., 2013; Furl and Averbeck, 2011). However, to our knowledge, the direct fMRI contrast between urn and draw choices has not been reported in patients with schizophrenia so far. While the neural basis for cognitive biases in decision-making therefore remains to be investigated in more detail, the interesting question arises how they are related to other decision processes involving inference.

## Value-based decision-making

Besides cognitive biases, patients with schizophrenia display alterations in value-based decision-making based on reinforcement learning. Reinforcement learning describes the fundamental processes involved in adapting our behaviour by interacting with the environment to maximize rewarding outcomes (Dayan and Daw, 2008). Based on reward signals from the environment, we learn and infer which environmental cues predict biologically significant events and which actions to perform. We have to assign and neuronally represent values to environmental cues and actions, use those values for decision-making and constantly update them by comparing the actually received reward or punishment with the expected outcome via reward PEs (Daw and O'Doherty, 2013). In this framework reward PEs (RPEs) code the differences between the predicted and the actually received reward and provide a teaching signal to update the prediction. In schizophrenia patients, altered RPE signaling and value representation have been proposed to contribute to central aspects of the disorder including aberrant salience attribution as well as motivational impairments (Heinz, 2002; Kapur, 2003). This hypothesis is supported by consistent evidence for disrupted reward anticipation and reinforcement learning on the behavioral and neural levels (Fig. 2B) (Deserno et al., 2013; Gold et al., 2008; Maia and Frank, 2017).

At the behavioral level, besides seemingly intact hedonic in-the-moment experiences of rewards (Cohen and Minor, 2010; Pankow et al., 2013), schizophrenia patients are impaired in flexible, value-based decision-making particularly in situations with changing, volatile environments (Culbreth et al., 2016; Leeson et al., 2009; Murray et al., 2008; Schlagenhauf et al., 2014; Waltz and Gold, 2007). Such studies typically use reversal-learning paradigms in which participants' choices in a decision-making task are probabilistically associated with reward (or punishment). These associations change unpredictably over the course of an experiment and it is the participants' task to adapt their behaviour accordingly. In neuroimaging experiments, probabilistic reversal learning has been successfully used to track the neural correlates of RPE signaling in the ventral striatum (Cools et al., 2002; Park et al., 2010; Schlagenhauf et al., 2013). Computational modeling indicated that patients with schizophrenia overestimate the probability that associations change and therefore show higher choice switching behaviour, possibly due to an altered belief about environmental volatility (Fig. 2A) (Culbreth et al., 2016; Schlagenhauf et al., 2014; Waltz et al., 2013). Extensions of the classical reinforcement-learning framework, inspired by Bayes optimal models of learning (Rushworth and Behrens, 2008), include estimates of the individual belief about the environmental volatility. Such (meta-)learning about the environmental volatility takes place in a trial-by-trial fashion, albeit on a hierarchically higher level

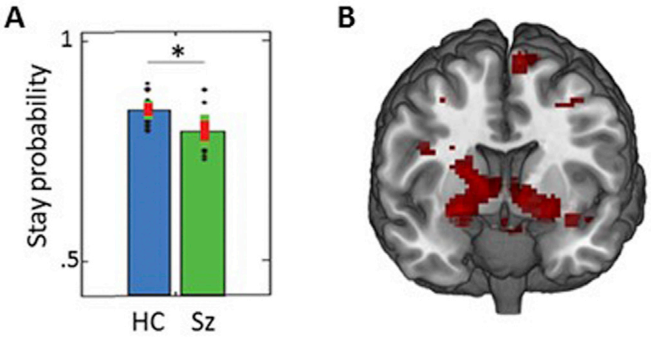

**Fig. 2. Reversal learning alterations in unmedicated schizophrenia patients**. (A) Parameter from the best fitting Hidden Markov Model (HMM) which captured the belief about how likely the task state was to remain the same on each trial. This ‘stay probability’ was significantly reduced in unmedicated schizophrenia patients compared to healthy controls. (B) Reduced coding of ventral striatal reward-prediction error in patients compared to controls (reproduced from Schlagenhauf et al., 2014, with permission).

("learning about learning"). The volatility estimates from higher levels in the learning hierarchy are supposed to influence the trial-by-trial PEs on the lower levels by scaling the learning rate. In a recent and neurobiological plausible Bayesian implementation of such hierarchical learning, the effect of PEs on learning is weighted by the belief about the current precision of the learning signal, resulting in so-called precision-weighted PEs (Mathys et al., 2011). While those precision-weighted RPEs have not been investigated in schizophrenia patients so far, application of ketamine in healthy controls, which is used to model glutamate dysfunction in schizophrenia, resulted in a disruption of the use of precision estimates to regulate reinforcement-learning parameters, which was accompanied by altered fronto-parietal activity (Vinckier et al., 2016).

At the neural level, reduced ventral striatal activation during reward anticipation was observed in unmedicated schizophrenia patients and was associated with the severity of negative symptoms (Juckel et al., 2006; Schlagenhauf et al., 2009). Both findings were confirmed in a recent meta-analysis (Radua et al., 2015). A link between negative symptoms, especially motivational deficits (avolition/apathy), with neurobiological systems implicated in motivation and reward processes is very suggestive and was observed with respect to apathy and dopamine receptor blockade induced by antipsychotic medication (Heinz et al., 1998). Negative symptoms might arise from a variety of fronto-striatal alterations including imprecise RPE encoding as well as effects of high dopamine receptor blockade, which can result in a failure to adequately learn, represent and/or use reward values to enable flexible value based decision-making (Barch and Dowd, 2010; Gold et al., 2008).

With respect to positive symptoms, chaotic or stress-dependent increases in dopamine release might be associated with aberrant salience attribution and related alterations in reinforcement learning (Heinz and Schlagenhauf, 2010). A proxy for elevated dopamine turnover is provided by dopamine synthesis capacity measured with F-DOPA and positron emission tomography, which was reliably increased in patients with schizophrenia (Howes et al., 2009; Kumakura et al., 2007). Elevated dopamine release and turnover can interfere with RPE encoding; in accordance with this hypothesis, striatal model-free PE coding was found to be inversely associated with dopamine synthesis capacity (Deserno et al., 2015; Schlagenhauf et al., 2013) and with heightened aberrant salience attribution in healthy controls (Boehme et al., 2015). Reduced coding of striatal RPE has also been observed in unmedicated schizophrenia patients (Schlagenhauf et al., 2014), however, associations with striatal dopamine turnover have not been reported to date.

Taken together, altered neural inference processes are observed in value-based decision-making, and predictive coding offers a promising framework to understand those alterations. Further studies are required to investigate the specific contribution of behavioral and neural alterations in value-based decision-making to clinical characteristics including negative and positive symptom domains over the course of the illness as well as the effects of treatment.

## Perceptual decision-making

Neurocognitive theories of schizophrenia have long linked the emergence of psychotic symptoms – not only hallucinations, but also delusions – to perceptual anomalies (e.g. (Maher, 1974),). From a clinical point of view, it is well established that subjective perceptual phenomena such as attentional capture by details in the visual field or misperceptions of color or shape, are among typically experienced disturbances referred to as "basic symptoms", which can occur already in the prodromal phase of schizophrenia (Schultze-Lutter, 2009). Such perceptual abnormalities suggest that altered inference mechanisms in schizophrenia are not confined to cognitive and value-based decision-making but represent a more pervasive alteration that also involves basic sensory processes. Indeed, several lines of evidence indicate that schizophrenia is associated with abnormalities at low hierarchical levels in the brain's inference mechanisms.

Perhaps one of the best established findings supporting the notion of altered low-level inference in schizophrenia is that of a reduced mismatch negativity (MMN). The MMN is an electrophysiological signal that is elicited when a sequence of identical auditory stimuli is unexpectedly interrupted by a stimulus that deviates from the standard stimulus, e.g. in in terms of intensity (for a review, see (Naatanen et al., 2007)). Importantly, the MMN also occurs when observers are not paying attention to the stimuli (e.g., performing a concurrent distractor task). The MNN therefore appears to reflect the pre-attentive, automatic detection of sensory events that violate current expectations and has been proposed to be equivalent to the signaling of sensory PEs (Lieder et al., 2013). The amplitude of the MMN was consistently found to be reduced in patients with schizophrenia, both for auditory stimuli (Erickson et al., 2016; Todd et al., 2012) and for visual stimuli (Kremlacek et al., 2016). While recent meta-analytical evidence has cast doubt on the usefulness of the MMN as a biomarker for schizophrenia (Erickson et al., 2017), it is still of theoretical relevance for the predictive coding account of schizophrenia. Interestingly, the MMN amplitude is reduced when NMDA receptor antagonists are applied in healthy humans and in animals (Javitt et al., 1996; Umbricht et al., 2000). NMDA receptor antagonists are known to elicit psychotic symptoms in healthy subjects and to increase psychotic symptoms in patients with schizophrenia (Corlett et al., 2011). As NMDA receptor function plays a major role in cortical feedback signaling (Dehaene et al., 2003; Lumer et al., 1997) and is thought to mediate the precision of prior beliefs (Adams et al., 2013; Meuwese et al., 2013; van Loon et al., 2015), a reduction in NMDA receptor signaling could account for the decreased precision of prior beliefs relative to the representation of sensory data, as outlined above. Thus, the reduction of MMN amplitude observed in individuals with schizophrenia is consistent with an impairment of predictive processes mediated by NMDA-receptor dependent feedback signaling (potentially in interaction with GABAergic inhibition) of prior beliefs (Wacongne, 2016). If predictions are imprecise, then even stimuli that should normally be predictable on the basis of learned regularities will elicit inappropriately large PE signals, thus resulting in a reduced difference between predictable and unpredictable stimuli and hence a reduced MMN. In other words, "attenuated mismatch or violation responses […] may not reflect a failure to detect surprising events but reflect a failure to detect unsurprising (predictable) events" (Adams et al., 2013). Taken together, reduced MMN amplitude is a well-replicated finding in schizophrenia, which implicates automatic stages of sensory processing and therefore strongly suggests a reduced precision of prior beliefs at low levels of the sensory processing hierarchy.

An instructive example of how perceptual inference is based on learned prior beliefs about regularities in the sensory environment are visual illusions. In line with the notion of decreased precision of prior beliefs, individuals with schizophrenia are less susceptible to some (but not all) visual illusions, that is, have a more "veridical" perception of the actual visual input (Brown et al., 2013b; Notredame et al., 2014). A well-known example of altered visual illusion perception in schizophrenia is the hollow-mask illusion (Dima et al., 2009; Keane et al., 2013; Schneider et al., 2002). When presented with the backside of a hollow mask, observers are usually unable to perceive the mask as concave, but instead report seeing a convex face. This effect has long been acknowledged as "evidence for the power of top-down knowledge for vision" (Gregory, 1970). Interestingly, patients with schizophrenia are more likely than healthy observers to experience the veridical percept of a hollow face, which supports the hypothesis of a reduced influence of prior beliefs on perception. An fMRI study investigating the neural correlates of this striking effect found differences in effective connectivity between schizophrenia patients and healthy observers (Dima et al., 2009). Using dynamic causal modeling (DCM (Friston et al., 2003);), the authors found that in healthy observers, illusory perception was best explained by a modulation of feedback connectivity between parietal cortex and the lateral occipital complex (LOC), a visual area implicated in object processing. In contrast, the data of schizophrenia patients were better explained by a model where feedforward connections from primary visual cortex (V1) to LOC were modulated, consistent with a stronger reliance on stimulus-driven processing rather than learned prior beliefs.

A reduced influence of prior beliefs in sensory processing is further indicated by altered processing of visual context in patients with schizophrenia. Context illusions (Fig. 3) are a classical demonstration of efficient predictive coding, whereby the contextual surround provides a statistical prediction of the central signal. In healthy individuals, the perceived contrast of a central stimulus is reduced when surrounded by a similar high contrast pattern, a phenomenon called surround suppression (Chubb et al., 1989). Patients with schizophrenia are less susceptible to this effect: under conditions that induce such surround suppression in healthy individuals, patients with schizophrenia have a more veridical perception of the center contrast than controls (Barch et al., 2012; Dakin et al., 2005; Tibber et al., 2013; Yang et al., 2013; Yoon et al., 2010; Yoon et al., 2009). Using fMRI and retinotopic mapping, contextual suppression of responses to a central stimulus by the spatial surround was demonstrated in V1 of healthy individuals, while such context-dependent modulation was absent in patients with schizophrenia (Seymour et al., 2013) (Fig. 3C and D). While these results are in line with the idea of a reduced influence of predictive signals in low-level perceptual inference, it should also be noted that reduced surround effects in schizophrenia

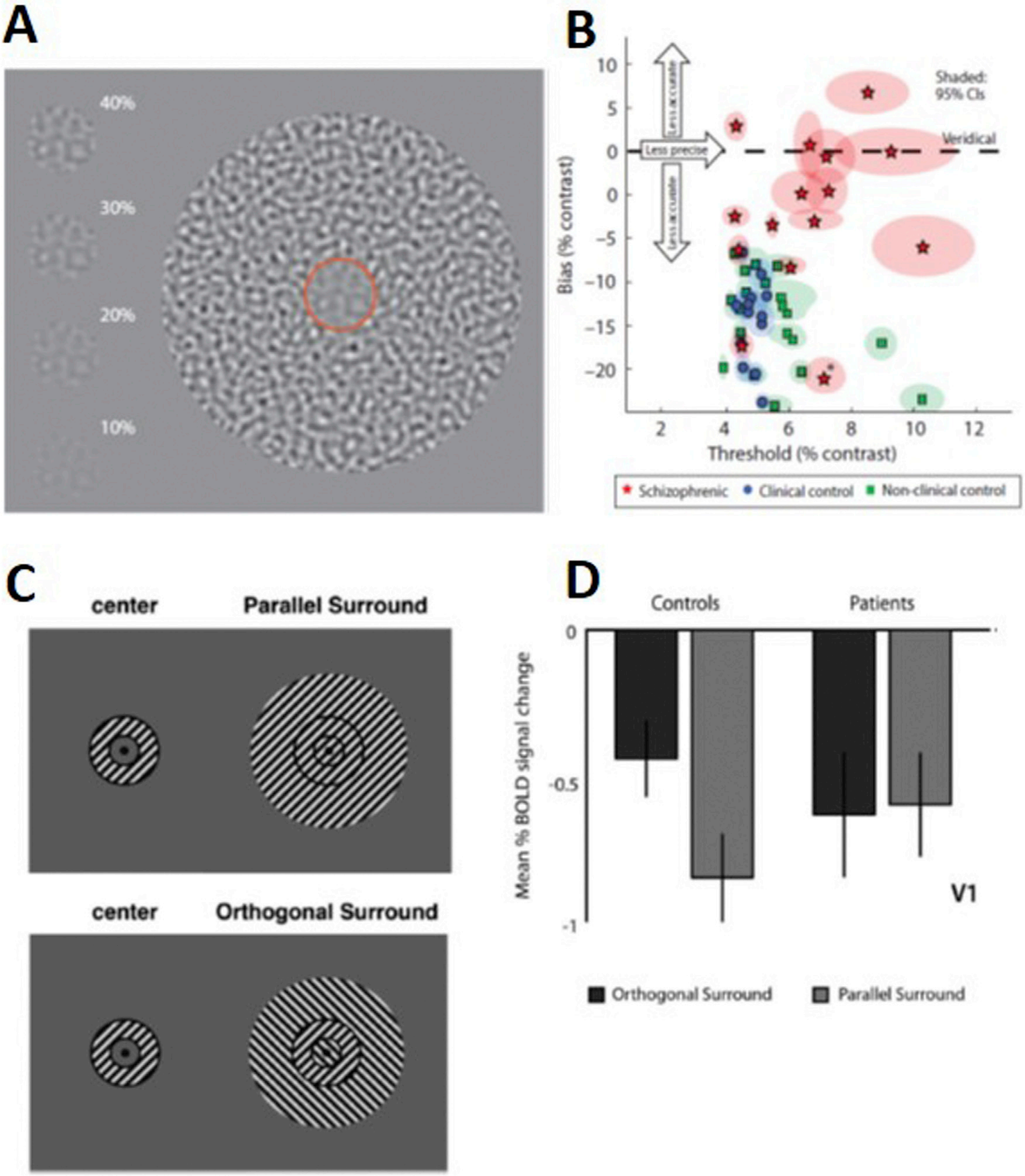

**Fig. 3. Context illusions suggest altered predictive coding in patients with schizophrenia**. (A) The perceived contrast of a central stimulus is reduced when surrounded by a similar high contrast pattern, a phenomenon called surround suppression. (B) Patients with schizophrenia (red stars) have a more veridical perception of the center contrast than healthy (green squares) and clinical controls (blue circles; reproduced from Dakin et al., 2005, with permission). (C) For grating stimuli, contextual suppression is stronger for parallel surrounds than for orthogonal surrounds. (D) Using fMRI and retinotopic mapping, such orientation-dependent contextual suppression of responses to a central stimulus by the spatial surround was demonstrated in V1 of healthy individuals, while such context-dependent modulation was absent in patients with schizophrenia (reproduced from Seymour et al., 2013, with permission).

have not been found consistently for all visual context illusions (Tibber et al., 2013; Yang et al., 2013).

These behavioral and neural findings provide support for a dysfunction of inference mechanisms at the sensory level in patients with schizophrenia. However, while visual illusions are a compelling way of investigating the influence of prior beliefs implemented at low hierarchical levels, they are characterized by a high degree of automaticity and resistance to cognitive influences. Illusions are therefore ill-suited to assess the roles of higher-level beliefs in perceptual inference. However, a full understanding of alterations in predictive neural mechanisms in schizophrenia will require the investigation of the interplay of different hierarchical levels in perceptual inference, from low-level sensory to high-level cognitive processing. An approach that is ideally suited to probe both sensory and cognitive predictions as well as the role of volatility estimation is the use of perceptually ambiguous stimuli that typically evoke bistable perception. This means that perception alternates spontaneously every few seconds between two interpretations of the same sensory input. Evidence from neuroimaging suggests that these perceptual alternations are governed by low-level sensory processes and higher-level brain circuits involved in cognitive processes (Brascamp et al., 2018; Long and Toppino, 2004; Sterzer et al., 2009). Well-known examples include the Necker cube, Rubin's face-vase illusion, ambiguous motion stimuli and binocular rivalry.

Recently, the interplay between low- and high-level predictive processing was investigated in relation to delusional ideation both in healthy individuals with varying levels of delusion proneness and in patients with schizophrenia (Schmack et al., 2013, 2015, 2017). In these studies, an ambiguous visual motion stimulus that yields alternating perception of either leftward or rightward rotation was used to probe the influence of prior beliefs implemented either at low or high levels of the visual processing hierarchy. First, the hypothesis that delusion proneness is associated with a reduced influence of low-level predictions (Adams et al., 2013; Corlett et al., 2009; Fletcher and Frith, 2009) was tested with intermittent presentation of the ambiguous stimulus. In healthy controls, such intermittent presentation leads to a survival of the current percept between stimulus presentations, likely resulting from a build-up of prior beliefs implemented within sensory cortices (Pearson and Brascamp, 2008; Sterzer and Rees, 2008). The survival probability of percepts across temporary stimulus removals was found to be reduced both in delusion-prone healthy individuals and, in a subsequent study, also in patients with schizophrenia (Schmack et al., 2015), thus indicating a reduced influence of low-level priors. In a second step, expectations about the appearance of the ambiguous stimulus were manipulated experimentally (cf. (Sterzer et al., 2008)) in order to probe the effect of higher-level cognitive beliefs. Interestingly, the strength of the belief-induced perceptual bias correlated positively with delusion proneness and was strongest in those individuals who had shown evidence for weak sensory predictions in the first experiment. At the neural level, the influence of higher level beliefs on perception was paralleled by an association of delusional ideation with enhanced functional connectivity between frontal areas and visual areas encoding perception. Moreover, the effect of beliefs on perception was reflected in visual cortical fMRI signal patterns as decoded with multi-voxel pattern analysis (MVPA (Haynes, 2015);). These findings support the idea that when predictions within lower-level sensory processing stages are reduced in precision, perceptual inference relies more strongly on predictions from higher-level non-sensory areas that encode beliefs (Sterzer et al., 2018). Confirming this interpretation, a recent study using the same experimental paradigm again found enhanced functional connectivity between frontal areas and visual areas in medicated and clinically stable patients with schizophrenia (Schmack et al., 2017).

An overly strong influence of learned high-level beliefs in delusion-prone individuals and those with early psychosis was also reported for the perception of so-called two-tone images, in which sensory information is impoverished and perceptual inference therefore relies more strongly on prior beliefs (Teufel et al., 2015). Differential roles of learned predictions at low and high hierarchical levels are also suggested by recent work relating delusion-proneness in healthy individuals to reduced usage of prior beliefs in perceptual but not cognitive decision-making (Stuke et al., 2018). However, the neural processes involved in these latter observations remain to be elucidated. Together with evidence for a cognitive bias against disconfirmatory evidence (McLean et al., 2017; Moritz and Woodward, 2006), the emerging picture is thus that prior beliefs implemented at high levels of the processing hierarchy may by enhanced in schizophrenia, perhaps compensating for the perceptual uncertainty caused by a reduced influence of more automatic predictive mechanisms at low hierarchical levels. This way, perception may be sculpted into conformity with high-level beliefs, which may be an important mechanism contributing to the tenacious persistence of delusions.

Taken together, there is converging behavioral, electrophysiological and fMRI evidence supporting the notion that altered predictive coding plays an important role in dysfunctional perceptual processes in schizophrenia. However, little is still known about the role of these alterations in perceptual decision-making, which is typically studied under conditions of perceptual uncertainty (Heekeren et al., 2008; Shadlen and Kiani, 2013). Investigating the neural correlates of perceptual decision-making under uncertainty in relation to altered predictive coding in schizophrenia is a promising avenue for future research.

## Sensorimotor control

Imprecision of prior beliefs may also help to explain impairments in the self-attribution of one's own actions in schizophrenia: Whenever we move a part of our body, we constantly have to decide which of the changes of our body state occur as a consequence of our actions. The feeling that oneself controls one's own actions, and, through them, events in the external world is referred to as sense of agency (SoA). It involves an interplay between predictions based on internal motor signals (i.e., prior beliefs regarding the sensory consequences of an action) and retrospective inferences based on sensory feedback resulting from the action (Moore and Fletcher, 2012; Synofzik et al., 2013).

Disturbances in SoA are frequent in schizophrenia (Frith, 1992; Nelson et al., 2014a; b). The common feature of these experiences – referred to as *Ichstörungen* ("self-disturbance") (Gruhle, 1929; Schneider, 1946) – is an altered experience of control over one's own actions, emotions and thoughts (Heinz, 2014; Jeannerod, 2009; Sterzer et al., 2016; Vosgerau and Voss, 2014). The disordered sense of agency in schizophrenia has been linked to deficits in internal monitoring processes (e.g. (Fourneret et al., 2002; Franck et al., 2001; Lindner et al., 2005)) and, more specifically, to a failure to predict one's own actions and their consequences in the environment (Blakemore et al., 2002; Shergill et al., 2005). In schizophrenia, such prediction deficits may be compensated for by more strongly weighting sensory, afferent information about action outcomes retrospectively (Chambon et al., 2011; Synofzik et al., 2010; Voss et al., 2010; Werner et al., 2014).

Recent research on SoA has been dominated by a theoretical framework originally developed for computational motor control (Frith et al., 2000; Wolpert et al., 1995). At heart, this framework uses an internal forward model to predict the sensory consequences of actions based on internal action-related information such as an efference-copy of a motor command (von Holst and Mittelstadt, 1950), corollary discharge (Sperry, 1950) and/or proprioception (Bell, 2001). These predictions can be used to cancel or attenuate incoming sensory information that arises from one's own action. Cancelling or attenuating such non-informative sensory feedback is conventionally held to prevent cognitive overload during action, and to free limited cognitive resources to attend to non-predicted, external events in the environment (Bays and Wolpert, 2007; Voss et al., 2006; Voss et al., 2008). With respect to experiencing a sense of agency, it has been proposed that this cancellation process may serve to distinguish between events that arise from one's own actions, and external events in the environment (Bays et al., 2005; Bell, 2001; Blakemore et al., 2002;

Lindner et al., 2005; Synofzik et al., 2006). The comparison between predicted and actual sensory feedback may thus be a fundamental process to experience oneself as the author of an action: if predicted and actual state are congruent, the sensory event is attenuated and attributed to one's own agency. If sensory feedback and sensory prediction are incongruent, sensory feedback is not attenuated and an external attribution results. Disorders of agency like passivity phenomena in schizophrenia have been explained within this framework: imprecise predictions about action consequences may lead to an erroneous mismatch signal at the comparator, which in turn leads to a false attribution of self-initiated actions to external sources (Frith et al., 2000).

More recently, predictive coding or active inference models were introduced as an alternative framework to explain abnormalities in perceptual decision-making as well as in sensorimotor control and action generation in schizophrenia (Fletcher and Fotopoulou, 2015). In contrast to the comparator model, which focuses on motor commands related to mismatch between predicted and actual state, active inference models focus on proprioceptive predictions, and actions are performed to resolve proprioceptive prediction errors. Attenuation of sensory signals resulting from movement is therefore not due to an absence of mismatch between predicted and actual states (as conceptualized in the comparator model framework), but a result of reduced precision (or weighting) of sensory evidence during movement in order to minimise prediction errors (Brown et al., 2013a). This distinction is important, as it opens new possibilities to understand both perception and action as being tightly bound together in the light of minimising prediction errors – and symptoms of schizophrenia resulting from alterations in this mechanism.

Several neuroimaging studies investigated neural correlates of the sense of agency and its disturbances (for a review, see (David et al., 2008)). Most studies focused on identifying the brain regions involved in the detection of sensorimotor mismatch, i.e. differences between predicted and actual sensory feedback resulting from voluntary action. Findings of these studies converge on the activation of posterior brain areas including the superior parietal and inferior parietal lobules, the cerebellum (Blakemore and Sirigu, 2003; Farrer et al., 2003; Farrer and Frith, 2002) as well as the angular gyrus (Chambon et al., 2015; Chambon et al., 2013; Farrer et al., 2004) for the detection of sensorimotor mismatch. These areas possibly integrate afferent information from various sources (vision, proprioception, kinematics and efference copy), also with respect to the "self-other" distinction. By contrast, the insula was rather recruited during sensorimotor matching (Farrer et al., 2003; Farrer and Frith, 2002). There is also evidence that the prefrontal cortex (PFC) may be required at the level of conscious agency monitoring (Slachevsky et al., 2001). Indeed, the dorsolateral prefrontal cortex (DLPFC) has been implicated in conflict monitoring and detection such as between one's own intended action and the sensory outcome (e.g. (Fink et al., 1999; Schnell et al., 2008),). In schizophrenia patients, hyper-activation in the inferior parietal lobule (IPL) was found in correspondence with an increase in false detections of visuomotor incongruence (Schnell et al., 2008). Furthermore, a correlation between passivity experiences and a differential effect of monitoring on activation in the posterior cingulate cortex (PCC) was interpreted as a result of a greater effort to monitor one's own actions in patients suffering from delusions of control compared to healthy volunteers.

In addition to re-afferent or predicted information about action outcomes, signals arising during action selection and prior to execution of a motor command can directly influence the SoA (e.g. (Chambon et al., 2013; Sidarus et al., 2013; Wenke et al., 2010),). In experiments where subliminal primes influence participant's choice regarding which of two possible actions to perform, response activations are primarily increased, followed by an inhibition of this response tendency (Eimer and Schlaghecken, 2003). Primes can thus either delay or facilitate the selection of an appropriate action in response to action cues, or can bias free selection. Interestingly, subliminal primes that are compatible with action cues also induce a stronger feeling of control over an external outcome of action, compared to primes that are incompatible with action cues (Chambon et al., 2013; Wenke et al., 2010). In schizophrenia, such influence of action selection signals on later agency judgements seems to be impaired. A recent study (Voss et al., 2017) shows that subliminal primes had a strong effect on response times in both patients and healthy participants, whereas effects on subsequent agency judgements were only seen in the group of healthy participants (see Fig. 4). Neuroimaging results showed a distinctive pattern of connectivity between the angular gyrus and frontal action selection areas related to priming effects on self-agency (Chambon et al., 2013). Patients with schizophrenia showed normal activation of frontal action selection areas but no priming-related activation of angular gyrus, and no changes in fronto-parietal connectivity related to priming effects (Voss et al., 2017). These findings are consistent with hypotheses that emphasize disconnection of functional neuronal networks as a hallmark of schizophrenia (e.g. (Barbalat et al., 2011; Friston, 1998),).

Similar to perceptual processes, there is thus strong behavioral and

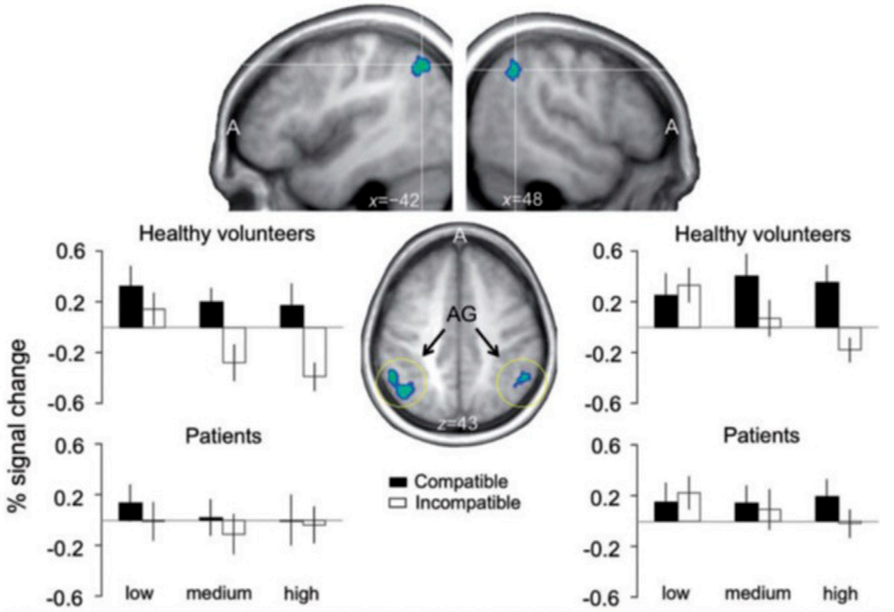

**Fig. 4. Parametric modulation of angular gyrus activation during action selection by subjective control ratings on cued-choice trials.** In this fMRI experiment, subliminal arrow primes were used to influence key-press responses to a subsequent left, right or double-headed arrow cue. Targets were preceded by a subliminal prime that was either compatible or incompatible with the target. In compatible trials, the direction of the prime corresponded to the direction of the mask/target, and hence signaled the same response. In incompatible trials, prime and mask/target pointed in different directions. Participants' responses caused a color to appear after a variable delay and subjects were asked to indicate the level of control they felt over the action effect. Results are shown for each combination of group and compatibility. fMRI responses in left and right angular gyrus are differentially modulated by subsequent control ratings (low, medium, and high control ratings) in healthy volunteers, but not patients, depending on how fluent action selection is. A = anterior; AG = angular gyrus (reproduced from Voss et al., 2017 with permission).

neuroimaging evidence for altered predictive coding in sensorimotor circuits in schizophrenia. These abnormalities have been convincingly related to passivity phenomena (Synofzik et al., 2013). Sensorimotor processes are increasingly acknowledged as playing an important role in decision-making, whereby the preparation and execution of motor actions are thought to influence decision processes (Cisek and Pastor-Bernier, 2014; Lepora and Pezzulo, 2015). It is therefore tempting to speculate that aberrant sensorimotor processing may also affect decision-making in schizophrenia, but empirical evidence for this link is lacking to date.

## Summary and conclusions

Dysfunctional decision-making in schizophrenia can be understood in the context of a more general concept of the brain as an organ of inference and the computational framework of hierarchical predictive coding (Friston et al., 2014a). There are abnormalities in cognitive inference and decision-making that are well-documented at the behavioral level, but are only beginning to be understood in neural terms (Adams et al., 2013). In contrast, there is already a large body of neuroimaging research available to support the view that altered mechanisms of value-based decision-making and in particular aberrant PE signaling play an important role in schizophrenia, especially in relation to negative symptoms including motivational deficits (Deserno et al., 2017; Maia and Frank, 2017). These findings are well in line with the more general notion within the predictive coding framework that an imbalance between the precision of predictions and PEs, with a reduced precision of predictions and enhanced precision-weighted prediction error signaling, is a key neurocomputational mechanism in schizophrenia (Adams et al., 2013; Corlett et al., 2009). This idea is also supported by evidence from various lines of research indicating alterations in low-level sensory processing: Electrophysiological findings showing reduced mismatch signals (Erickson et al., 2016) and behavioral as well as neuroimaging studies showing a reduced susceptibility to visual illusions can be accounted for by a reduced precision of predictions at sensory processing levels (Notredame et al., 2014). On the other hand, stronger influence of priors compared to perceptual input might contribute to hallucinations (Corlett et al., 2009), a theory which recently received empirical support in the context of conditioned hallucinations (Powers et al., 2017). In addition, recent evidence points towards different roles of predictive signals originating from low and high levels of the predictive coding hierarchy, whereby the reduced precision of lower-level predictions may be compensated by an increased precision of higher-level predictions (Schmack et al., 2013; Schmack et al., 2017; Schmack et al., 2015; Stuke et al., 2018). This mechanism may lead to perceptual experiences that are strongly shaped by delusional beliefs, thereby contributing to the fixity of delusions. Finally, research into the mechanisms of sensorimotor control again point towards a reduced precision of predictive signals, which leads to a mismatch between the predicted and the actual sensory consequences of motor actions (Frith, 1992; Voss et al., 2017). This mismatch is thought to result in the external attribution of one's own voluntary actions, thus leading to an impaired SoA.

The emerging picture from this research is thus that the neurocomputational mechanisms involved in the psychopathology of schizophrenia may be due to similar impairments in the functional brain systems implicated in various aspects of decision-making, including sensory, motor, cognitive and motivational systems and involving both cortical and subcortical levels. This hypothesis does not rule out that there may also be some degree of informational encapsulation in light of the well-documented regional differences in the functional architecture and neurotransmitter systems across the brain (Fodor, 1983). For example, it is conceivable that predictive coding in sensory systems may be disrupted in a manner relatively independent from predictive coding of actions and cognitive beliefs. It is an intriguing challenge for future research to determine the degree to which similar mechanisms are involved in different functional brain systems and how they interact with each other. Another important question is to which degree impairments in these different systems may account for the clinical heterogeneity of the disease. It is quite possible that the pattern of impairments of functional brain systems as well as differences in the degree to which different hierarchical levels are affected may be related to clinical symptomatology at the phenomenological level. For example, dysfunction in value-based learning has been linked to negative symptoms (Heinz and Schlagenhauf, 2010; Radua et al., 2015), whereas predictive coding deficits in sensorimotor control can account for self-disturbances such as the experience of alien control and thought insertion (Heinz et al., 2012; Sterzer et al., 2016; Vosgerau and Voss, 2014).

As we have argued, hierarchical predictive coding may provide a useful framework for the understanding of decision-making processes in general and for dysfunctional decision-making in schizophrenia in particular. However, while broad empirical evidence is available for functional and neural alterations in all domains considered relevant in decision-making, research directly addressing the role of these alterations in decision-making processes is still rather scarce. The field of decision neuroscience offers a range of concepts and approaches that may shed new light on the pathophysiology of schizophrenia. For example, decision neuroscience has a longstanding tradition of research into the neural mechanisms of decision-making under perceptual uncertainty (Heekeren et al., 2008; Shadlen and Kiani, 2013), which is an area that may be highly relevant for the understanding of aberrant inference and learning, but that has been largely neglected in schizophrenia research. The framework of Bayesian inference and predictive coding is a promising approach for the integration of decision-making alterations across brain systems within a generic theory of brain function. Importantly, computational modeling in the predictive coding framework also provides the means for rigorous hypothesis testing in a quantitative way and for linking behavioral and neural abnormalities to specific model parameters, thereby facilitating a mechanistic understanding of neural dysfunctions in schizophrenia (Friston et al., 2014a; Stephan et al., 2017).

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## References

Adams, R.A., Huys, Q.J., Roiser, J.P., 2016. Computational Psychiatry: towards a mathematically informed understanding of mental illness. J. Neurol. Neurosurg. Psychiatry 87, 53–63.
Adams, R.A., Stephan, K.E., Brown, H.R., Frith, C.D., Friston, K.J., 2013. The computational anatomy of psychosis. Front. Psychiatr. 4, 47.
Barbalat, G., Chambon, V., Domenech, P.J., Ody, C., Koechlin, E., Franck, N., Farrer, C., 2011. Impaired hierarchical control within the lateral prefrontal cortex in schizophrenia. Biol. Psychiatr. 70, 73–80.
Barch, D.M., Carter, C.S., Dakin, S.C., Gold, J., Luck, S.J., MacDonald, A., Ragland, J.D., Silverstein, S., Strauss, M.E., 2012. The clinical translation of a measure of gain control: the contrast-contrast effect task. Schizophr. Bull. 38, 135–143.
Barch, D.M., Dowd, E.C., 2010. Goal representations and motivational drive in schizophrenia: the role of prefrontal-striatal interactions. Schizophr. Bull. 36, 919–934.
Bays, P.M., Wolpert, D.M., 2007. Computational principles of sensorimotor control that minimize uncertainty and variability. J. Physiol. 578, 387–396.
Bays, P.M., Wolpert, D.M., Flanagan, J.R., 2005. Perception of the consequences of self-action is temporally tuned and event driven. Curr. Biol. 15, 1125–1128.
Bell, C.C., 2001. Memory-based expectations in electrosensory systems. Curr. Opin. Neurobiol. 11, 481–487.
Bitzer, S., Bruineberg, J., Kiebel, S.J., 2015. A bayesian attractor model for perceptual decision making. PLoS Comput. Biol. 11 e1004442.
Bitzer, S., Park, H., Blankenburg, F., Kiebel, S.J., 2014. Perceptual decision making: drift-diffusion model is equivalent to a Bayesian model. Front. Hum. Neurosci. 8, 102.
Blakemore, S.J., Sirigu, A., 2003. Action prediction in the cerebellum and in the parietal lobe. Exp. Brain Res. 153, 239–245.

Blakemore, S.J., Wolpert, D.M., Frith, C.D., 2002. Abnormalities in the awareness of action. Trends Cognit. Sci. 6, 237–242.

Boehme, R., Deserno, L., Gleich, T., Katthagen, T., Pankow, A., Behr, J., Buchert, R., Roiser, J.P., Heinz, A., Schlagenhauf, F., 2015. Aberrant salience is related to reduced reinforcement learning signals and elevated dopamine synthesis capacity in healthy adults. J. Neurosci. 35, 10103–10111.

Brascamp, J., Sterzer, P., Blake, R., Knapen, T., 2018. Multistable perception and the role of the frontoparietal cortex in perceptual inference. Annu. Rev. Psychol. 69, 77–103.

Brown, H., Adams, R.A., Parees, I., Edwards, M., Friston, K., 2013a. Active inference, sensory attenuation and illusions. Cognit. Process. 14, 411–427.

Brown, H., Adams, R.A., Parees, I., Edwards, M., Friston, K., 2013b. Active inference, sensory attenuation and illusions. Cognit. Process. 14, 411–427.

Brown, M., Kuperberg, G.R., 2015. A hierarchical generative framework of language processing: linking language perception, interpretation, and production abnormalities in schizophrenia. Front. Hum. Neurosci. 9, 643.

Chambon, V., Moore, J.W., Haggard, P., 2015. TMS stimulation over the inferior parietal cortex disrupts prospective sense of agency. Brain Struct. Funct. 220, 3627–3639.

Chambon, V., Pacherie, E., Barbalat, G., Jacquet, P., Franck, N., Farrer, C., 2011. Mentalizing under influence: abnormal dependence on prior expectations in patients with schizophrenia. Brain 134, 3728–3741.

Chambon, V., Wenke, D., Fleming, S.M., Prinz, W., Haggard, P., 2013. An online neural substrate for sense of agency. Cerebr. Cortex 23, 1031–1037.

Chubb, C., Sperling, G., Solomon, J.A., 1989. Texture interactions determine perceived contrast. Proc. Natl. Acad. Sci. Unit. States Am. 86, 9631–9635.

Cisek, P., Pastor-Bernier, A., 2014. On the challenges and mechanisms of embodied decisions. Philos. Trans. R. Soc. Lond. B Biol. Sci. 369.

Clark, A., 2013. Whatever next? Predictive brains, situated agents, and the future of cognitive science. Behav. Brain Sci. 36, 181–204.

Cohen, A.S., Minor, K.S., 2010. Emotional experience in patients with schizophrenia revisited: meta-analysis of laboratory studies. Schizophr. Bull. 36, 143–150.

Cools, R., Clark, L., Owen, A.M., Robbins, T.W., 2002. Defining the neural mechanisms of probabilistic reversal learning using event-related functional magnetic resonance imaging. J. Neurosci. 22, 4563–4567.

Corlett, P.R., Frith, C.D., Fletcher, P.C., 2009. From drugs to deprivation: a Bayesian framework for understanding models of psychosis. Psychopharmacology (Berlin) 206, 515–530.

Corlett, P.R., Honey, G.D., Krystal, J.H., Fletcher, P.C., 2011. Glutamatergic model psychoses: prediction error, learning, and inference. Neuropsychopharmacology 36, 294–315.

Culbreth, A.J., Gold, J.M., Cools, R., Barch, D.M., 2016. Impaired activation in cognitive control regions predicts reversal learning in schizophrenia. Schizophr. Bull. 42, 484–493.

Dakin, S., Carlin, P., Hemsley, D., 2005. Weak suppression of visual context in chronic schizophrenia. Curr. Biol. 15, R822–R824.

David, N., Newen, A., Vogeley, K., 2008. The "sense of agency" and its underlying cognitive and neural mechanisms. Conscious. Cognit. 17, 523–534.

Daw, N.D., O'Doherty, J.P., 2013. Multiple systems for value learning. In: Glimcher, P.W., Fehr, E. (Eds.), Neuroeconomics. Elsevier, pp. 393–410.

Dayan, P., Daw, N.D., 2008. Decision theory, reinforcement learning, and the brain. Cognit. Affect Behav. Neurosci. 8, 429–453.

Dehaene, S., Sergent, C., Changeux, J.P., 2003. A neuronal network model linking subjective reports and objective physiological data during conscious perception. Proc. Natl. Acad. Sci. U. S. A. 100, 8520–8525.

den Ouden, H.E., Kok, P., de Lange, F.P., 2012. How prediction errors shape perception, attention, and motivation. Front. Psychol. 3, 548.

Deserno, L., Boehme, R., Heinz, A., Schlagenhauf, F., 2013. Reinforcement learning and dopamine in schizophrenia: dimensions of symptoms or specific features of a disease group? Front. Psychiatr. 4, 172.

Deserno, L., Heinz, A., Schlagenhauf, F., 2017. Computational approaches to schizophrenia: a perspective on negative symptoms. Schizophr. Res. 186, 46–54.

Deserno, L., Huys, Q.J., Boehme, R., Buchert, R., Heinze, H.J., Grace, A.A., Dolan, R.J., Heinz, A., Schlagenhauf, F., 2015. Ventral striatal dopamine reflects behavioral and neural signatures of model-based control during sequential decision making. Proc. Natl. Acad. Sci. U. S. A. 112, 1595–1600.

Dima, D., Roiser, J.P., Dietrich, D.E., Bonnemann, C., Lanfermann, H., Emrich, H.M., Dillo, W., 2009. Understanding why patients with schizophrenia do not perceive the hollow-mask illusion using dynamic causal modelling. Neuroimage 46, 1180–1186.

Dudley, R., Taylor, P., Wickham, S., Hutton, P., 2016. Psychosis, delusions and the "jumping to conclusions" reasoning bias: a systematic review and meta-analysis. Schizophr. Bull. 42, 652–665.

Eimer, M., Schlaghecken, F., 2003. Response facilitation and inhibition in subliminal priming. Biol. Psychol. 64, 7–26.

Erickson, M.A., Albrecht, M., Ruffle, A., Fleming, L., Corlett, P., Gold, J., 2017. No association between symptom severity and MMN impairment in schizophrenia: a meta-analytic approach. Schizophr Res Cogn 9, 13–17.

Erickson, M.A., Ruffle, A., Gold, J.M., 2016. A meta-analysis of mismatch negativity in schizophrenia: from clinical risk to disease specificity and progression. Biol. Psychiatr. 79, 980–987.

Esslinger, C., Braun, U., Schirmbeck, F., Santos, A., Meyer-Lindenberg, A., Zink, M., Kirsch, P., 2013. Activation of midbrain and ventral striatal regions implicates salience processing during a modified beads task. PLoS One 8 e58536.

Evans, S.L., Averbeck, B.B., Furl, N., 2015. Jumping to conclusions in schizophrenia. Neuropsychiatric Dis. Treat. 11, 1615–1624.

Fard, P.R., Park, H., Warkentin, A., Kiebel, S.J., Bitzer, S., 2017. A bayesian reformulation of the extended drift-diffusion model in perceptual decision making. Front. Comput. Neurosci. 11, 29.

Farrer, C., Franck, N., Frith, C.D., Decety, J., Georgieff, N., d'Amato, T., Jeannerod, M., 2004. Neural correlates of action attribution in schizophrenia. Psychiatr. Res. 131, 31–44.

Farrer, C., Franck, N., Georgieff, N., Frith, C.D., Decety, J., Jeannerod, M., 2003. Modulating the experience of agency: a positron emission tomography study. Neuroimage 18, 324–333.

Farrer, C., Frith, C.D., 2002. Experiencing oneself vs another person as being the cause of an action: the neural correlates of the experience of agency. Neuroimage 15, 596–603.

Fink, G.R., Marshall, J.C., Halligan, P.W., Frith, C.D., Driver, J., Frackowiak, R.S., Dolan, R.J., 1999. The neural consequences of conflict between intention and the senses. Brain 122 (Pt 3), 497–512.

FitzGerald, T.H., Moran, R.J., Friston, K.J., Dolan, R.J., 2015. Precision and neuronal dynamics in the human posterior parietal cortex during evidence accumulation. Neuroimage 107, 219–228.

Fletcher, P.C., Fotopoulou, A., 2015. Sense of Agency and its disruptions: clinical and computational perspectives. In: Haggard, P., Eitam, B. (Eds.), The Sense of Agency. Oxford University Press.

Fletcher, P.C., Frith, C.D., 2009. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. Nat. Rev. Neurosci. 10, 48–58.

Fodor, J.A., 1983. The Modularity of Mind: an Essay on Faculty Psychology. MIT Press, Cambridge, MA.

Fourneret, P., de Vignemont, F., Franck, N., Slachevsky, A., Dubois, B., Jeannerod, M., 2002. Perception of self-generated action in schizophrenia. Cognit. Neuropsychiatry 7, 139–156.

Franck, N., Farrer, C., Georgieff, N., Marie-Cardine, M., Dalery, J., d'Amato, T., Jeannerod, M., 2001. Defective recognition of one's own actions in patients with schizophrenia. Am. J. Psychiatr. 158, 454–459.

Friston, K., FitzGerald, T., Rigoli, F., Schwartenbeck, P., Pezzulo, G., 2017. Active inference: a process theory. Neural Comput. 29, 1–49.

Friston, K., Harrison, L., Penny, W., 2003. Dynamic causal modelling. Neuroimage 19, 1273–1302.

Friston, K., Kiebel, S., 2009. Predictive coding under the free-energy principle. Philos. Trans. R. Soc. Lond. B Biol. Sci. 364, 1211–1221.

Friston, K., Schwartenbeck, P., FitzGerald, T., Moutoussis, M., Behrens, T., Dolan, R.J., 2014a. The anatomy of choice: dopamine and decision-making. Philos. Trans. R. Soc. Lond. B Biol. Sci. 369.

Friston, K.J., 1998. The disconnection hypothesis. Schizophr. Res. 30, 115–125.

Friston, K.J., 2005. A theory of cortical responses. Philos. Trans. R. Soc. Lond. B Biol. Sci. 360, 815–836.

Friston, K.J., Stephan, K.E., Montague, R., Dolan, R.J., 2014b. Computational psychiatry: the brain as a phantastic organ. Lancet Psychiatry 1, 148–158.

Frith, C.D., 1992. The Cognitive Neuropsychology of Schizophrenia. Lawrence Erlbaum, Sussex, UK.

Frith, C.D., Blakemore, S., Wolpert, D.M., 2000. Explaining the symptoms of schizophrenia: abnormalities in the awareness of action. Brain Res Brain Res Rev 31, 357–363.

Furl, N., Averbeck, B.B., 2011. Parietal cortex and insula relate to evidence seeking relevant to reward-related decisions. J. Neurosci. 31, 17572–17582.

Garety, P.A., Freeman, D., 2013. The past and future of delusions research: from the inexplicable to the treatable. Br. J. Psychiatry 203, 327–333.

Gold, J.I., Shadlen, M.N., 2007. The neural basis of decision making. Annu. Rev. Neurosci. 30, 535–574.

Gold, J.M., Waltz, J.A., Prentice, K.J., Morris, S.E., Heerey, E.A., 2008. Reward processing in schizophrenia: a deficit in the representation of value. Schizophr. Bull. 34, 835–847.

Gregory, R.L., 1970. The Intelligent Eye. McGraw-Hill.

Gruhle, H.W., 1929. Psychologie der Schizophrenie. In: Berze, J. (Ed.), Psychologie der Schizophrenie. Springer, Wien, pp. 73–168.

Haynes, J.D., 2015. A primer on pattern-based approaches to fMRI: principles, pitfalls, and perspectives. Neuron 87, 257–270.

Heekeren, H.R., Marrett, S., Ungerleider, L.G., 2008. The neural systems that mediate human perceptual decision making. Nat. Rev. Neurosci. 9, 467–479.

Heinz, A., 2002. Dopaminergic dysfunction in alcoholism and schizophrenia–psychopathological and behavioral correlates. Eur. Psychiatr. 17, 9–16.

Heinz, A., 2014. Der Begriff der psychischen Krankheit. Suhrkamp/Insel Verlag, Berlin.

Heinz, A., Bermpohl, F., Frank, M., 2012. Construction and interpretation of self-related function and dysfunction in Intercultural Psychiatry. Eur. Psychiatr. 27 (Suppl. 2), S32–S43.

Heinz, A., Knable, M.B., Coppola, R., Gorey, J.G., Jones, D.W., Lee, K.S., Weinberger, D.R., 1998. Psychomotor slowing, negative symptoms and dopamine receptor availability–an IBZM SPECT study in neuroleptic-treated and drug-free schizophrenic patients. Schizophr. Res. 31, 19–26.

Heinz, A., Schlagenhauf, F., 2010. Dopaminergic dysfunction in schizophrenia: salience attribution revisited. Schizophr. Bull. 36, 472–485.

Hemsley, D.R., Garety, P.A., 1986. The formation of maintenance of delusions: a Bayesian analysis. Br. J. Psychiatr. 149, 51–56.

Howes, O.D., Montgomery, A.J., Asselin, M.C., Murray, R.M., Valli, I., Tabraham, P., Bramon-Bosch, E., Valmaggia, L., Johns, L., Broome, M., McGuire, P.K., Grasby, P.M., 2009. Elevated striatal dopamine function linked to prodromal signs of schizophrenia. Arch. Gen. Psychiatr. 66, 13–20.

Huq, S.F., Garety, P.A., Hemsley, D.R., 1988. Probabilistic judgements in deluded and non-deluded subjects. Q. J. Exp. Psychol. 40, 801–812.

Jardri, R., Hugdahl, K., Hughes, M., Brunelin, J., Waters, F., Alderson-Day, B., Smailes, D., Sterzer, P., Corlett, P.R., Leptourgos, P., Debbane, M., Cachia, A., Deneve, S., 2016.

Are hallucinations due to an imbalance between excitatory and inhibitory influences on the brain? Schizophr. Bull. 42, 1124–1134.

Javitt, D.C., Steinschneider, M., Schroeder, C.E., Arezzo, J.C., 1996. Role of cortical N-methyl-D-aspartate receptors in auditory sensory memory and mismatch negativity generation: implications for schizophrenia. Proc. Natl. Acad. Sci. U. S. A. 93, 11962–11967.

Jeannerod, M., 2009. The sense of agency and its disturbances in schizophrenia: a reappraisal. Exp. Brain Res. 192, 527–532.

Juckel, G., Schlagenhauf, F., Koslowski, M., Wustenberg, T., Villringer, A., Knutson, B., Wrase, J., Heinz, A., 2006. Dysfunction of ventral striatal reward prediction in schizophrenia. Neuroimage 29, 409–416.

Kapur, S., 2003. Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. Am. J. Psychiatr. 160, 13–23.

Keane, B.P., Silverstein, S.M., Wang, Y., Papathomas, T.V., 2013. Reduced depth inversion illusions in schizophrenia are state-specific and occur for multiple object types and viewing conditions. J. Abnorm. Psychol. 122, 506.

Knill, D.C., Pouget, A., 2004. The Bayesian brain: the role of uncertainty in neural coding and computation. Trends Neurosci. 27, 712–719.

Kremlacek, J., Kreegipuu, K., Tales, A., Astikainen, P., Poldver, N., Naatanen, R., Stefanics, G., 2016. Visual mismatch negativity (vMMN): a review and meta-analysis of studies in psychiatric and neurological disorders. Cortex 80, 76–112.

Krug, A., Cabanis, M., Pyka, M., Pauly, K., Kellermann, T., Walter, H., Wagner, M., Landsberg, M., Shah, N.J., Winterer, G., Wolwer, W., Brinkmeyer, J., Muller, B.W., Kargel, C., Wiedemann, G., Herrlich, J., Vogeley, K., Schilbach, L., Rapp, A., Klingberg, S., Kircher, T., 2014. Attenuated prefrontal activation during decision-making under uncertainty in schizophrenia: a multi-center fMRI study. Schizophr. Res. 152, 176–183.

Kumakura, Y., Cumming, P., Vernaleken, I., Buchholz, H.G., Siessmeier, T., Heinz, A., Kienast, T., Bartenstein, P., Grunder, G., 2007. Elevated [18F]fluorodopamine turnover in brain of patients with schizophrenia: an [18F]fluorodopa/positron emission tomography study. J. Neurosci. 27, 8080–8087.

Lee, J.S., Jung, S., Park, I.H., Kim, J.J., 2015. Neural basis of anhedonia and amotivation in patients with schizophrenia: the role of reward system. Curr. Neuropharmacol. 13, 750–759.

Lee, T.S., Mumford, D., 2003. Hierarchical Bayesian inference in the visual cortex. J Opt Soc Am A Opt Image Sci Vis 20, 1434–1448.

Leeson, V.C., Robbins, T.W., Matheson, E., Hutton, S.B., Ron, M.A., Barnes, T.R., Joyce, E.M., 2009. Discrimination learning, reversal, and set-shifting in first-episode schizophrenia: stability over six years and specific associations with medication type and disorganization syndrome. Biol. Psychiatr. 66, 586–593.

Lepora, N.F., Pezzulo, G., 2015. Embodied choice: how action influences perceptual decision making. PLoS Comput. Biol. 11 e1004110.

Lieder, F., Daunizeau, J., Garrido, M.I., Friston, K.J., Stephan, K.E., 2013. Modelling trial-by-trial changes in the mismatch negativity. PLoS Comput. Biol. 9 e1002911.

Lindner, A., Thier, P., Kircher, T.T., Haarmeier, T., Leube, D.T., 2005. Disorders of agency in schizophrenia correlate with an inability to compensate for the sensory consequences of actions. Curr. Biol. 15, 1119–1124.

Long, G.M., Toppino, T.C., 2004. Enduring interest in perceptual ambiguity: alternating views of reversible figures. Psychol. Bull. 130, 748–768.

Lumer, E.D., Edelman, G.M., Tononi, G., 1997. Neural dynamics in a model of the thalamocortical system. I. Layers, loops and the emergence of fast synchronous rhythms. Cerebr. Cortex 7, 207–227.

Maher, B.A., 1974. Delusional thinking and perceptual disorder. J. Indiv. Psychol.

Maia, T.V., Frank, M.J., 2017. An integrative perspective on the role of dopamine in schizophrenia. Biol. Psychiatr. 81, 52–66.

Mathys, C., Daunizeau, J., Friston, K.J., Stephan, K.E., 2011. A Bayesian foundation for individual learning under uncertainty. Front. Hum. Neurosci. 5.

McLean, B.F., Mattiske, J.K., Balzan, R.P., 2017. Association of the jumping to conclusions and evidence integration biases with delusions in psychosis: a detailed meta-analysis. Schizophr. Bull. 43, 344–354.

Meuwese, J.D., van Loon, A.M., Scholte, H.S., Lirk, P.B., Vulink, N.C., Hollmann, M.W., Lamme, V.A., 2013. NMDA receptor antagonist ketamine impairs feature integration in visual perception. PLoS One 8 e79326.

Mishara, A.L., Sterzer, P., 2015. Phenomenology is Bayesian in its application to delusions. World Psychiatr. 14, 185–186.

Moore, J.W., Fletcher, P.C., 2012. Sense of agency in health and disease: a review of cue integration approaches. Conscious. Cognit. 21, 59–68.

Moritz, S., Woodward, T.S., 2006. A generalized bias against disconfirmatory evidence in schizophrenia. Psychiatr. Res. 142, 157–165.

Moutoussis, M., Bentall, R.P., El-Deredy, W., Dayan, P., 2011. Bayesian modelling of jumping-to-conclusions bias in delusional patients. Cognit. Neuropsychiatry 16, 422–447.

Mulder, M.J., van Maanen, L., Forstmann, B.U., 2014. Perceptual decision neurosciences - a model-based review. Neuroscience 277, 872–884.

Murray, G.K., Cheng, F., Clark, L., Barnett, J.H., Blackwell, A.D., Fletcher, P.C., Robbins, T.W., Bullmore, E.T., Jones, P.B., 2008. Reinforcement and reversal learning in first-episode psychosis. Schizophr. Bull. 34, 848–855.

Naatanen, R., Paavilainen, P., Rinne, T., Alho, K., 2007. The mismatch negativity (MMN) in basic research of central auditory processing: a review. Clin. Neurophysiol. 118, 2544–2590.

Nelson, B., Whitford, T.J., Lavoie, S., Sass, L.A., 2014a. What are the neurocognitive correlates of basic self-disturbance in schizophrenia? Integrating phenomenology and neurocognition: Part 2 (aberrant salience). Schizophr. Res. 152, 20–27.

Nelson, B., Whitford, T.J., Lavoie, S., Sass, L.A., 2014b. What are the neurocognitive correlates of basic self-disturbance in schizophrenia?: Integrating phenomenology and neurocognition. Part 1 (Source monitoring deficits). Schizophr. Res. 152, 12–19.

Notredame, C.E., Pins, D., Deneve, S., Jardri, R., 2014. What visual illusions teach us about schizophrenia. Front. Integr. Neurosci. 8, 63.

O'Reilly, J.X., Jbabdi, S., Behrens, T.E., 2012. How can a Bayesian approach inform neuroscience? Eur. J. Neurosci. 35, 1169–1179.

Pankow, A., Friedel, E., Sterzer, P., Seiferth, N., Walter, H., Heinz, A., Schlagenhauf, F., 2013. Altered amygdala activation in schizophrenia patients during emotion processing. Schizophr. Res. 150, 101–106.

Park, S.Q., Kahnt, T., Beck, A., Cohen, M.X., Dolan, R.J., Wrase, J., Heinz, A., 2010. Prefrontal cortex fails to learn from reward prediction errors in alcohol dependence. J. Neurosci. 30, 7749–7753.

Pearson, J., Brascamp, J., 2008. Sensory memory for ambiguous vision. Trends Cognit. Sci. 12, 334–341.

Phillips, L.D., Edwards, W., 1966. Conservatism in a simple probability inference task. J. Exp. Psychol. 72, 346–354.

Powers, A.R., Mathys, C., Corlett, P.R., 2017. Pavlovian conditioning-induced hallucinations result from overweighting of perceptual priors. Science 357, 596–600.

Radua, J., Schmidt, A., Borgwardt, S., Heinz, A., Schlagenhauf, F., McGuire, P., Fusar-Poli, P., 2015. Ventral striatal activation during reward processing in psychosis: a neurofunctional meta-analysis. JAMA psychiatry 72, 1243–1251.

Rao, R.P., Ballard, D.H., 1999. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. Nat. Neurosci. 2, 79–87.

Robinson, T.E., Berridge, K.C., 1993. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Brain Res Rev 18, 247–291.

Ross, R.M., McKay, R., Coltheart, M., Langdon, R., 2015. Jumping to conclusions about the beads Task? A meta-analysis of delusional ideation and data-gathering. Schizophr. Bull. 41, 1183–1191.

Rushworth, M.F., Behrens, T.E., 2008. Choice, uncertainty and value in prefrontal and cingulate cortex. Nat. Neurosci. 11, 389–397.

Schlagenhauf, F., Huys, Q.J., Deserno, L., Rapp, M.A., Beck, A., Heinze, H.J., Dolan, R., Heinz, A., 2014. Striatal dysfunction during reversal learning in unmedicated schizophrenia patients. Neuroimage 89, 171–180.

Schlagenhauf, F., Rapp, M.A., Huys, Q.J., Beck, A., Wustenberg, T., Deserno, L., Buchholz, H.G., Kalbitzer, J., Buchert, R., Bauer, M., Kienast, T., Cumming, P., Plotkin, M., Kumakura, Y., Grace, A.A., Dolan, R.J., Heinz, A., 2013. Ventral striatal prediction error signaling is associated with dopamine synthesis capacity and fluid intelligence. Hum. Brain Mapp. 34, 1490–1499.

Schlagenhauf, F., Sterzer, P., Schmack, K., Ballmaier, M., Rapp, M., Wrase, J., Juckel, G., Gallinat, J., Heinz, A., 2009. Reward feedback alterations in unmedicated schizophrenia patients: relevance for delusions. Biol. Psychiatr. 65, 1032–1039.

Schmack, K., Gomez-Carrillo de Castro, A., Rothkirch, M., Sekutowicz, M., Rossler, H., Haynes, J.D., Heinz, A., Petrovic, P., Sterzer, P., 2013. Delusions and the role of beliefs in perceptual inference. J. Neurosci. 33, 13701–13712.

Schmack, K., Rothkirch, M., Priller, J., Sterzer, P., 2017. Enhanced predictive signalling in schizophrenia. Hum. Brain Mapp.

Schmack, K., Schnack, A., Priller, J., Sterzer, P., 2015. Perceptual instability in schizophrenia: probing predictive coding accounts of delusions with ambiguous stimuli. Schizophr Res Cogn Epub.

Schneider, K., 1946. Klinische Psychopathologie. Thieme, Stuttgart.

Schneider, U., Borsutzky, M., Seifert, J., Leweke, F., Huber, T., Rollnik, J., Emrich, H., 2002. Reduced binocular depth inversion in schizophrenic patients. Schizophr. Res. 53, 101–108.

Schnell, K., Heekeren, K., Daumann, J., Schnell, T., Schnitker, R., Moller-Hartmann, W., Gouzoulis-Mayfrank, E., 2008. Correlation of passivity symptoms and dysfunctional visuomotor action monitoring in psychosis. Brain 131, 2783–2797.

Schultz, W., Dayan, P., Montague, P.R., 1997. A neural substrate of prediction and reward. Science 275, 1593–1599.

Schultze-Lutter, F., 2009. Subjective symptoms of schizophrenia in research and the clinic: the basic symptom concept. Schizophr. Bull. 35, 5–8.

Seymour, K., Stein, T., Sanders, L.L.O., Guggenmos, M., Theophil, I., Sterzer, P., 2013. Altered contextual modulation of primary visual cortex responses in schizophrenia. Neuropsychopharmacology 38, 2607–2612.

Shadlen, M.N., Kiani, R., 2013. Decision making as a window on cognition. Neuron 80, 791–806.

Shergill, S.S., Samson, G., Bays, P.M., Frith, C.D., Wolpert, D.M., 2005. Evidence for sensory prediction deficits in schizophrenia. Am. J. Psychiatr. 162, 2384–2386.

Shipp, S., 2016. Neural elements for predictive coding. Front. Psychol. 7, 1792.

Sidarus, N., Chambon, V., Haggard, P., 2013. Priming of actions increases sense of control over unexpected outcomes. Conscious. Cognit. 22, 1403–1411.

Slachevsky, A., Pillon, B., Fourneret, P., Pradat-Diehl, P., Jeannerod, M., Dubois, B., 2001. Preserved adjustment but impaired awareness in a sensory-motor conflict following prefrontal lesions. J. Cognit. Neurosci. 13, 332–340.

So, S.H., Siu, N.Y., Wong, H.L., Chan, W., Garety, P.A., 2016. 'Jumping to conclusions' data-gathering bias in psychosis and other psychiatric disorders - two meta-analyses of comparisons between patients and healthy individuals. Clin. Psychol. Rev. 46, 151–167.

Speechley, W.J., Whitman, J.C., Woodward, T.S., 2010. The contribution of hypersalience to the "jumping to conclusions" bias associated with delusions in schizophrenia. JPN (J. Psychiatry Neurosci.): JPN (J. Psychiatry Neurosci.) 35, 7.

Sperry, R.W., 1950. Neural basis of the spontaneous optokinetic response produced by visual inversion. J. Comp. Physiol. Psychol. 43, 482–489.

Stephan, K.E., Schlagenhauf, F., Huys, Q.J., Raman, S., Aponte, E.A., Brodersen, K.H., Rigoux, L., Moran, R.J., Daunizeau, J., Dolan, R.J., Friston, K.J., Heinz, A., 2017. Computational neuroimaging strategies for single patient predictions. Neuroimage 145, 180–199.

Sterzer, P., 2016. Moving forward in perceptual decision making. Proc. Natl. Acad. Sci. U. S. A. 113, 5771–5773.

Sterzer, P., Adams, R.A., Fletcher, P.C., Frith, C., Lawrie, S.M., Muckli, L., Petrovic, P., Uhlhaas, P.J., Voss, M., Corlett, P.R., 2018. The predictive coding account of psychosis. Biol. Psychiatr. https://doi.org/10.1016/j.biopsych.05.015 (Epub).

Sterzer, P., Frith, C., Petrovic, P., 2008. Believing is seeing: expectations alter visual awareness. Curr. Biol. 18, R697–R698.

Sterzer, P., Kleinschmidt, A., Rees, G., 2009. The neural bases of multistable perception. Trends Cognit. Sci. 13, 310–318.

Sterzer, P., Mishara, A.L., Voss, M., Heinz, A., 2016. Thought insertion as a self-disturbance: an integration of predictive coding and phenomenological approaches. Front. Hum. Neurosci. 10, 502.

Sterzer, P., Rees, G., 2008. A neural basis for percept stabilization in binocular rivalry. J. Cognit. Neurosci. 20, 389–399.

Stuke, H., Stuke, H., Weilnhammer, V.A., Schmack, K., 2017. Psychotic experiences and overhasty inferences are related to maladaptive learning. PLoS Comput. Biol. 13 e1005328.

Stuke, H., Weilnhammer, V.A., Sterzer, P., Schmack, K., 2018. Delusion proneness is linked to a reduced usage of prior beliefs in perceptual decisions. Schizophr. Bull.

Summerfield, C., de Lange, F.P., 2014. Expectation in perceptual decision making: neural and computational mechanisms. Nat. Rev. Neurosci. 15, 745–756.

Synofzik, M., Thier, P., Leube, D.T., Schlotterbeck, P., Lindner, A., 2010. Misattributions of agency in schizophrenia are based on imprecise predictions about the sensory consequences of one's actions. Brain 133, 262–271.

Synofzik, M., Thier, P., Lindner, A., 2006. Internalizing agency of self-action: perception of one's own hand movements depends on an adaptable prediction about the sensory action outcome. J. Neurophysiol. 96, 1592–1601.

Synofzik, M., Vosgerau, G., Voss, M., 2013. The experience of agency: an interplay between prediction and postdiction. Front. Psychol. 4, 127.

Teufel, C., Subramaniam, N., Dobler, V., Perez, J., Finnemann, J., Mehta, P.R., Goodyer, I.M., Fletcher, P.C., 2015. Shift toward prior knowledge confers a perceptual advantage in early psychosis and psychosis-prone healthy individuals. Proc. Natl. Acad. Sci. U. S. A. 112, 13401–13406.

Tibber, M.S., Anderson, E.J., Bobin, T., Antonova, E., Seabright, A., Wright, B., Carlin, P., Shergill, S.S., Dakin, S.C., 2013. Visual surround suppression in schizophrenia. Front. Psychol. 4.

Todd, J., Michie, P.T., Schall, U., Ward, P.B., Catts, S.V., 2012. Mismatch negativity (MMN) reduction in schizophrenia—impaired prediction-error generation, estimation or salience? Int. J. Psychophysiol. 83, 222–231.

Umbricht, D., Schmid, L., Koller, R., Vollenweider, F.X., Hell, D., Javitt, D.C., 2000. Ketamine-induced deficits in auditory and visual context-dependent processing in healthy volunteers: implications for models of cognitive deficits in schizophrenia. Arch. Gen. Psychiatr. 57, 1139–1147.

van Loon, A.M., Fahrenfort, J.J., van der Velde, B., Lirk, P.B., Vulink, N.C., Hollmann, M.W., Steven Scholte, H., Lamme, V.A., 2015. NMDA Receptor Antagonist Ketamine Distorts Object Recognition by Reducing Feedback to Early Visual Cortex. Cereb Cortex.

Vinckier, F., Gaillard, R., Palminteri, S., Rigoux, L., Salvador, A., Fornito, A., Adapa, R., Krebs, M.O., Pessiglione, M., Fletcher, P.C., 2016. Confidence and psychosis: a neuro-computational account of contingency learning disruption by NMDA blockade. Mol. Psychiatr. 21, 946–955.

von Holst, E., Mittelstadt, H., 1950. Das reafferenzprinzip. Naturwissenschaften 37, 464–476.

Vosgerau, G., Voss, M., 2014. Authorship and control over thoughts. Mind Lang. 29, 534–565.

Voss, M., Chambon, V., Wenke, D., Kühn, S., Haggard, P., 2017. In and out of control: brain mechanisms linking fluency of action selection to self-agency in patients with schizophrenia. Brain.

Voss, M., Ingram, J.N., Haggard, P., Wolpert, D.M., 2006. Sensorimotor attenuation by central motor command signals in the absence of movement. Nat. Neurosci. 9, 26–27.

Voss, M., Ingram, J.N., Wolpert, D.M., Haggard, P., 2008. Mere expectation to move causes attenuation of sensory signals. PLoS One 3, e2866.

Voss, M., Moore, J., Hauser, M., Gallinat, J., Heinz, A., Haggard, P., 2010. Altered awareness of action in schizophrenia: a specific deficit in predicting action consequences. Brain 133, 3104–3112.

Wacongne, C., 2016. A predictive coding account of MMN reduction in schizophrenia. Biol. Psychol. 116, 68–74.

Waltz, J.A., Gold, J.M., 2007. Probabilistic reversal learning impairments in schizophrenia: further evidence of orbitofrontal dysfunction. Schizophr. Res. 93, 296–303.

Waltz, J.A., Kasanova, Z., Ross, T.J., Salmeron, B.J., McMahon, R.P., Gold, J.M., Stein, E.A., 2013. The roles of reward, default, and executive control networks in set-shifting impairments in schizophrenia. PLoS One 8 e57257.

Wenke, D., Fleming, S.M., Haggard, P., 2010. Subliminal priming of actions influences sense of control over effects of action. Cognition 115, 26–38.

Werner, J.D., Trapp, K., Wustenberg, T., Voss, M., 2014. Self-attribution bias during continuous action-effect monitoring in patients with schizophrenia. Schizophr. Res. 152, 33–40.

Wolpert, D.M., Ghahramani, Z., Jordan, M.I., 1995. An internal model for sensorimotor integration. Science 269, 1880–1882.

Yang, E., Tadin, D., Glasser, D.M., Hong, S.W., Blake, R., Park, S., 2013. Visual context processing in schizophrenia. Clinical Psychological Science 1, 5–15.

Yoon, J.H., Maddock, R.J., Rokem, A., Silver, M.A., Minzenberg, M.J., Ragland, J.D., Carter, C.S., 2010. GABA concentration is reduced in visual cortex in schizophrenia and correlates with orientation-specific surround suppression. J. Neurosci. 30, 3777–3781.

Yoon, J.H., Rokem, A.S., Silver, M.A., Minzenberg, M.J., Ursu, S., Ragland, J.D., Carter, C.S., 2009. Diminished orientation-specific surround suppression of visual processing in schizophrenia. Schizophr. Bull. 35, 1078–1084.