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A two-stage cognitive theory of the positive symptoms of psychosis. Highlighting the role of lowered decision thresholds

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ABSTRACT

Objectives: We outline a two-stage heuristic account for the pathogenesis of the positive symptoms of psychosis.
Methods: A narrative review on the empirical evidence of the liberal acceptance (LA) account of positive symptoms is presented.
Hypothesis: At the heart of our theory is the idea that psychosis is characterized by a lowered decision threshold, which results in the premature acceptance of hypotheses that a nonpsychotic individual would reject. Once the hypothesis is judged as valid, counterevidence is not sought anymore due to a bias against disconfirmatory evidence as well as confirmation biases, consolidating the false hypothesis. As a result of LA, confidence in errors is enhanced relative to controls. Subjective probabilities are initially low for hypotheses in individuals with delusions, and delusional ideas at stage 1 (belief formation) are often fragile. In the course of the second stage (belief maintenance), fleeting delusional ideas evolve into fixed false beliefs, particularly if the delusional idea is congruent with the emotional state and provides "meaning". LA may also contribute to hallucinations through a misattribution of (partially) normal sensory phenomena. Interventions such as metacognitive training that aim to "plant the seeds of doubt" decrease positive symptoms by encouraging individuals to seek more information and to attenuate confidence. The effect of antipsychotic medication is explained by its doubt-inducing properties.
Limitations: The model needs to be confirmed by longitudinal designs that allow an examination of causal relationships. Evidence is currently weak for hallucinations.
Conclusions: The theory may account for positive symptoms in a subgroup of patients. Future directions are outlined.

1. A two-stage theory of the positive symptoms of psychosis

The basic principles of the heuristic model of positive symptoms in psychosis laid down in this article have evolved over the last decade, starting with an article in 2004 (Moritz & Woodward, 2004). This manuscript will try to bring the pieces together. While our theory hopes to provide a parsimonious explanation for the formation of positive symptoms, delusions, hallucinations and so-called first-rank symptoms (e.g., thought insertion; Schneider, 1959) by the same mechanism, our assumptions do not claim to account for every single case of psychotic experience. With Bleuler (1911/1950) who coined the plural diagnostic term *schizophrenias* we concur that psychosis is presumably a multicausal disorder.[1] Therefore, putting forward this theory does not refute or challenge prior cognitive theories (e.g., Coltheart, Langdon, & McKay, 2011; Davies, Coltheart, Langdon, & Breen, 2001; Fletcher & Frith,

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[1] In keeping with standard nomenclature we will nevertheless use the singular term *psychosis* in the remainder of the article.

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2009; Kapur, 2003; Maher, 2006). In fact, these accounts, which view aberrant input (rather than reasoning) as the driving process of delusion formation, are considered powerful explanations for overvalued ideas and autochthonous delusions (i.e., Jaspers *wahneinfall*: a sudden delusional idea that comes "out of the blue" without identifiable preceding events) following a strong sensory or neurological component ("surprising experiences demand surprising explanations", p. 360; Corlett, Taylor, Wang, Fletcher, & Krystal, 2010). We will first present our basic two-stage theory, whereby stage 1 (belief formation) is at the heart of the heuristic model. In this context, we present empirical evidence for its validity and describe how our theory may accommodate specific (at times counter-intuitive) peculiarities of positive symptoms (e.g., long course until positive symptoms are full-blown, initial inconsequentiality). We then turn to stage 2 of the account, which explains how delusional ideas may or may not evolve to incorrigible convictions, before discussing how existing treatments such as antipsychotic medication, as well as metacognitive and reasoning training, exert their effect. While our approach highlights a cognitive mechanism, we will explain why ameliorating emotion regulation and improving mood may also be important for the treatment of positive symptoms. This review will not cover genetic or brain imaging data as our focus is in the cognitive processes of positive symptoms. Another blind spot is that the review will not deal with other prominent syndromes in psychosis like disorganization and negative symptoms. While our theory might be extended to accommodate these symptoms, we will not elaborate on this subject because empirical data are presently lacking.

We also would like to acknowledge the important contribution of theorists like Daniel Freeman and Philippa Garety (Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Garety & Freeman, 1999, 2013) as well as Richard Bentall (e.g., Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001) and an early theory by Christopher Frith (1979), whose models have been very influential, especially for the second stage of our theory.

1.1. Stage 1: How false ideas enter and dominate consciousness (belief formation)

Unlike a number of theories of the positive symptoms of psychosis which posit that delusions derive as essentially normal explanations from "out-of-the-ordinary experiences" (p. 181, Maher, 2006) such as hallucinations, neuropsychological impairment or other erroneous *input* (Davies et al., 2001; Frith, 1979; Kapur, 2003; Maher, 1999, 2006), or theories that confine themselves to single core psychotic symptoms such as paranoia (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Freeman & Garety, 2014; Freeman et al., 2002), we propose that, in a subgroup of patients, the same basic pathological mechanisms are at work in all major positive symptoms.

Picking up a metaphor used in one of our last empirical studies (Moritz, Scheu et al., 2016), we regard as a key cognitive aberration in psychosis that patients reason like "bad statisticians", that is, that they assign meaning and momentum to weakly supported evidence. A central claim is that the decision threshold for accepting hypotheses is lowered in psychosis; hypotheses that a healthy or nonpsychotic patient would reject, or put on hold until further validity checks are made, are accepted as possible (Moritz & Woodward, 2004; Moritz, Woodward, & Lambert, 2007; Moritz, Woodward, Jelinek, & Klinge, 2008; Moritz et al., 2009). Of note, strange thoughts (e.g., people are making remarks about me; feelings of being looked at) at times also occur in non-psychotic patients. What distinguishes psychotic from non-psychotic individuals is the weight these thoughts receive and the reactions they elicit (Lincoln, Möbius, Huber, Nagel, & Moritz, 2014).

1.1.1. Empirical evidence

Importantly, we assume this mechanism to be general and not confined to delusional or emotion-laden situations. Evidence for this theory comes from different lines of research.

1.1.1.1. Plausibility scores for absurd hypotheses

A recent meta-analysis (McLean, Mattiske, & Balzan, 2016) on parameters predominantly collected with the bias against disconfirmatory evidence paradigm (BADE; Buchy, Woodward, & Liotti, 2007; Sanford, Veckenstedt, Moritz, Balzan, & Woodward, 2014; Woodward, Buchy, Moritz, & Liotti, 2007; Woodward, Moritz, & Chen, 2006) suggests that individuals with schizophrenia assign higher plausibility to interpretations (verbally or nonverbally presented response options for a scenario) that nonpsychotic individuals would reject as absurd. This was typically examined using non-delusion relevant material in order to avoid tautological inferences (for results on plausibility judgements for delusional scenarios see LaRocco & Warman, 2009). In our very first study (Moritz & Woodward, 2004), which already outlined a sketch for the present theory, we used ambiguous pictures from the Thematic Apperception Test (TAT). While patients and controls did not differ with respect to well-supported interpretations of presented pictures, patients rated absurd scenarios as much more plausible than controls. Interestingly, in patients who received higher antipsychotic doses, this pattern was attenuated (we will turn to the potential significance of this and other psychopharmacological findings later).

1.1.1.2. Decision threshold for conclusions

Building upon experimental designs of Hausmann and Läge (e.g., 2008), in studies analogous to the "Who Wants to be a Millionaire" TV game show (Moritz, Woodward, & Hausmann, 2006; Moritz, Göritz, et al., 2015), we asked patients and controls to provide probability estimates to response options and then asked them, whether they would make a decision or reject any of the options/hypotheses presented, based on their subjective probability estimates. Importantly, such judgments were optional; even if participants were 99% sure, they were free to make a decision or not. In other studies (Moritz, Scheu et al., 2016; Moritz, Van Quaquebeke, & Lincoln, 2012), we adopted variants of the beads task[2] (Garety, Hemsley, & Wessely, 1991) and asked patients after each drawn item for their probability estimates, and again, whether or not they would make a decision. The "millionaire quiz" and modified beads task studies allowed us to dissociate the point of conclusion-drawing from subjective probability estimates and to determine the individual decision threshold, that is, the probability estimate an individual deems sufficient for a decision/firm judgment. In these studies (Moritz, Scheu et al., 2016; Moritz et al., 2009, 2012; Moritz, Woodward, et al., 2006; Veckenstedt et al., 2011), we found that patients based decisions on much lower probability estimates than controls (e.g. 82% relative to 93% in Moritz, Scheu et al., 2016) and this parameter proved a better discriminator between groups than the conventional jumping to conclusions

[2] Beads are drawn from one of two containers with usually opposing ratios of colors (e.g. container A: 85% green, 15% red; container B: opposite). The participant has to deduce by means of the sequence of beads from which of the two containers beads originate. Jumping to conclusions is usually defined as a (premature) decision after one or two beads.

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or draws to decision estimates (Moritz et al., 2012). In one study (Moritz, Scheu et al., 2016), we could even detect a lowered decision threshold which was correlated with more errors, while the JTC bias was absent in patients.

1.1.2. The role of confidence

A liberal decision threshold enhances chances of arriving at false assumptions. This is why in statistics, for example, the level of significance for accepting or rejecting hypotheses is usually quite strict and conventionally set at 5%, although this value is arbitrary and in some consequential situations (e.g., diagnostic decisions for severe disorders) even higher thresholds should apply. A liberal decision threshold has important implications for confidence in correct and incorrect judgments. Once a decision has been made and a judgmental *gestalt* is formed, the hypothesis is no longer openly challenged. The search for alternative hypotheses is abolished and inconsistent evidence is ignored; this response pattern is almost ubiquitous and – albeit with a lower severity – even seen in (nonpsychotic) academics (Fugelsang, Stein, Green, & Dunbar, 2004). Due to premature termination of the search process, additional valid cues for a correct hypothesis are not detected; even if a psychotic individual eventually endorses the correct hypothesis, it is held less confidently compared to a non-psychotic individual who has gathered more evidence and thus gains more confidence in the correct decision. On the other hand, the premature termination of the search process makes it more likely that evidence against the wrong hypotheses is overlooked or de-valued (Moritz & Woodward, 2006). As the search process in non-psychotic individuals is prolonged, they might still arrive at wrong conclusions but with less confidence (Patalano & LeClair, 2011). This is because inconclusive cues hold momentous affective and behavioral consequences in check via doubt (Moritz & Van Quaquebeke, 2014; for a related argument see; Yu, Pleskac, & Zeigenfuse, 2015). This pattern of results has been reported in many studies (Bhatt, Laws, & McKenna, 2010; Doré, Caza, Gingras, & Rouleau, 2007; Gawęda, Woodward, Moritz, & Kokoszka, 2013; Köther et al., 2012; Mayer, Kim, & Park, 2014; Moritz, Göritz, et al., 2015; Moritz, Ramdani, et al., 2014; Peters et al., 2007; Peters, Hauschildt, Moritz, & Jelinek, 2013); the evidence was recently summarized by Balzan (2016). In short, patients with schizophrenia display overconfidence in errors mostly in combination with lowered confidence in correct judgments, which results in "knowledge corruption" (defined as the proportion of high-confident responses that are false). Interestingly, this overconfidence bias has also been shown in participants at-risk for psychosis, or those with elevated subclinical symptoms, and is thus not confined to patients with acute psychosis (Eisenacher et al., 2015; Moritz, Göritz, et al., 2015). However, we have noted that the exact pattern varies across tasks (e.g., with task performance sometimes characterized by overall overconfidence, or underconfidence in correct responses) and seems to depend on subjective competence; a patient is more likely to adopt liberal acceptance when he or she feels competent. This is in accordance with the observation that delusional ideas are usually not random but relate to topics for which the patient has some experience, (subjective) expertise or insight (Moritz, Göritz, et al., 2015).

Relating this evidence to delusion formation, we propose that patients in stage 1 of the process are not necessarily different from non-psychotic individuals in terms of the subjective probability of their initial ideas, including ideas (i.e., *grains*) that later evolve into delusions (see Fig. 1, upper panel). However, they assign more weight/strength and validity to their hypotheses once these have achieved a certain threshold, which is lower than in non-psychotic individuals. These ideas are then deemed significant ("This is enough evidence for me"), are not rigorously challenged anymore (Fugelsang et al., 2004), and finally begin to crystalize as beliefs. As outlined before, the chance of overlooking counterarguments is high since liberal acceptance shortcuts the validating process.

A self-evident objection against this assumption is that delusions are frequently defined as *conviction* in false ideas (American Psychiatric Association, 2013). Indeed, utter conviction is often seen in patients and will be dealt with below when we turn to Stage 2 (belief maintenance). However, at least in the initial stages of delusion formation (Klosterkötter, 1992), patients have some doubt (Jaspers, 1913), and studies adopting experience sampling methodology (ESM) suggest that the severity and conviction of delusions fluctuate over time (Peters et al., 2012; Thewissen et al., 2011), and are thus not always at 100%. Some patients rest at stage 1, particularly those with bizarre and rapid changing delusions or those with so-called "double book keeping" (an expression coined by Bleuler, 1950), that is, where two seemingly counter-exclusive hypotheses flip back and forth.[3] Delusions of the stage 1 type are also the ones that are usually not acted upon and thus share some resemblance with (exaggerated) religious beliefs (the at times unclear boundaries between religion and psychosis have already been addressed by Schneider, 1928; see also McKay, 2004). Jaspers (1963) noted the inconsequentiality of many deluded patients: "With these patients, persecution does not always appear quite like the experience of people who are in fact being persecuted…. Hence the attitude of the patient to the content of his delusion is peculiarly inconsequent at times." (p. 105).

Acting upon a belief seems to be associated with belief conviction (Bjørkly, 2002; Junginger, 1996; Moritz & Van Quaquebeke, 2014) and confidence can still be low at this early stage. The delusional idea is more like a working hypothesis and increasingly absorbs the individual, a process that can take many weeks and evolves over time (Kapur, 2003; Klosterkötter, 1992).

1.2. Stage 2: Factors prompting conviction

Stage 1 cannot explain utter delusional conviction and the maintenance of delusional beliefs. To explain this transformation (i.e., enhancement of confidence) and consolidation (i.e., resistance to counterevidence), further processes come into play, some of which have been previously described by other authors (e.g., Freeman & Garety, 2014; Freeman et al., 2002). We will refer to this as stage 2, which will be subdivided into stage 2a (consolidation of the primary delusional belief) and stage 2b (neglect of competing hypotheses). Both stages are depicted in Fig. 1.

1.2.1. Confirmation bias and bias against disconfirmatory evidence

One very important cognitive process for stage 2a is the confirmation bias (see Fig. 1, lower panel), that is, the tendency to look for cues and facts supporting one's hypothesis (Nickerson, 1998). While this bias has long been studied in healthy people (Oswald & Grosjean, 2004), evidence suggests that it is elevated in people with psychosis (Balzan, Delfabbro, Galletly, & Woodward, 2013). This process may be driven by a hypersalience of evidence-hypothesis matches, where people with delusions overvalue and attach excessive weight to hypothesis congruent evidence (Speechley, Whitman, & Woodward, 2010). Another important facet is the disregard of evidence that conflicts with one's beliefs, and a recent meta-analysis suggests that this *bias against disconfirmatory evidence* (BADE) distinguishes patients with psychosis

[3] Of note, a lowered decision threshold can not only explain why patients jump to conclusions on the aforementioned beads task but also why they switch more often to an alternative option than controls once contrary evidence occurs instead of being stuck onto one option.

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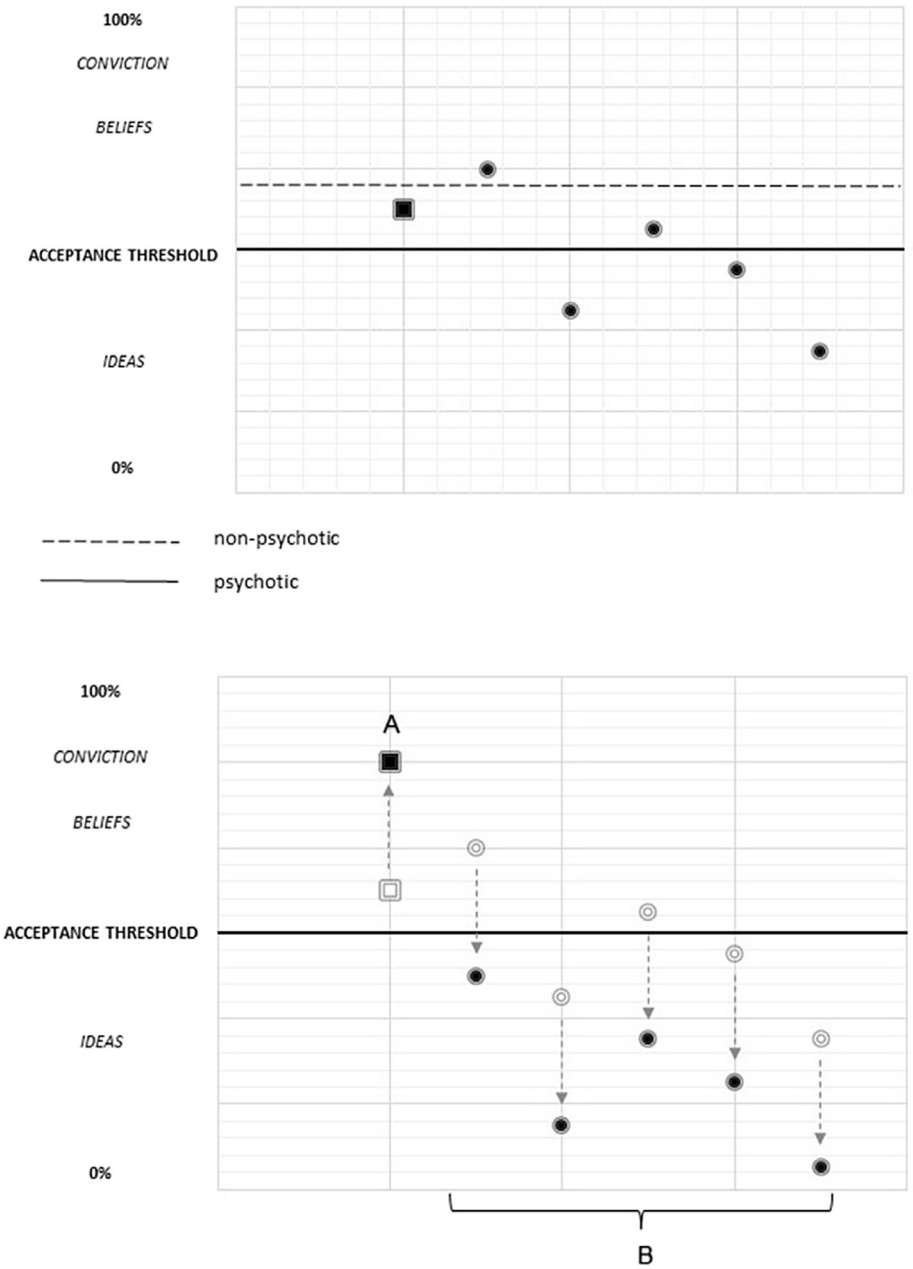

Fig. 1. Upper panel: Stage 1. In individuals with psychosis, ideas are more easily accepted due to a lowered decision threshold (dotted line: threshold of nonpsychotic individuals; full line: threshold of individuals with psychosis). In the example, nonpsychotic individuals pursue further only one (nondelusional) hypothesis. Lower panel: Stage 2. Stage 2a: The delusional belief is augmented to a conviction by means of a confirmation bias and/or emotional factors (see text) [A]. Stage 2b: Alternative hypotheses decrease in strength by means of a bias against disconfirmatory evidence (BADE) and, for example, withdrawal/avoidance (lack of corrective experiences) [B]. Square: delusional idea; circles: nondelusional idea(s).

from psychiatric and non-psychiatric controls (McLean et al., 2016). It has also been detected in high-risk 'delusion-prone' populations (Buchy et al., 2007; Eisenacher et al., 2016; Woodward, Moritz, Menon, & Klinge, 2008). The BADE further contributes to the systematization of the delusional idea as (challenging) alternative scenarios are less and less contemplated (stage 2b). Isolation as well as active social withdrawal/avoidance, which limit opportunities for critical social feedback and corrective experiences (Fett et al., 2012; Freeman et al., 2002; Zimbardo, 1999), are additional fertile ground for the subsequent "delusional work" (*Wahnarbeit*) as Jaspers named it (Jaspers, 1913).

1.2.2. The role of emotions

Another important criticism seems at place. A purely cognitive theory faces the difficulty to explain the specific contents and themes of psychological disorders and their prevalent affective tones (e.g., fear, depression). Why do patients arrive at ideas about persecution instead of more peaceful ones? In line with research on anxiety disorders (Soares, Esteves, Lundqvist, & Öhman, 2009), we argue a threatening and emotionally charged explanation has an evolutionary processing advantage and will receive more weight than a neutral one, because, if true, it would be associated with momentous and life-threatening consequences (de Jong, 2015).

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Therefore, "emotionally charged" hypotheses are considered first and individuals may get stuck with them because of their temporal precedence, especially when such hypotheses are primed by negative core beliefs following adverse events (such as bullying or abuse) in childhood and adolescence (Bentall et al., 2014). Indeed, first hypotheses are generally judged as more plausible and therefore have a higher chance to prevail (Moritz & Woodward, 2004), which is seen both in healthy subjects and patients, and seems to represents a special case of the "primacy effect" described for memory processes (Vinokur & Ajzen, 1982). Beyond that, we concur with Freeman et al. (2002) that delusions reflect and pick up the dominant emotional state. If a patient is anxious, a persecutory content is more probable than when a patient is in an elated mood. In the latter case grandiose delusions will more likely occur.

Another potential mechanism for stage 2 should be mentioned. An accepted theory is also more likely to prevail if it has a benefit and/or serves a purpose for the individual (Westermann, Cavelti, Heibach, & Caspar, 2015).[4] As already highlighted by founders of modern psychiatry (Jaspers, 1913; Kraepelin, 1919), delusional ideas often bring some "insight relief" (Kapur, 2003). Although the individual may feel persecuted and experience other unpleasant feelings, he or she now "knows" his or her opponent, has an explanation for previous emotional turmoil and often feels elated because the alleged opponents are people or organizations of a certain rank or power imparting the patient with importance in return (Moritz, Rietschel, et al., 2016; Moritz, Favrod, et al., 2013; Sundag, Lincoln, Hartmann, & Moritz, 2014). Religious or secret service organizations are particularly attractive targets, because these have the alleged power to induce the many disturbances a patient usually suffers from. The greater the explanatory power of the delusional superstructure the more stable it is. Moreover, if the patient has a lot to lose if their beliefs turn out to be false (e.g., self-esteem, structure, purpose), they might be more prone to disregard counterevidence.

1.3. Applying the account to hallucinations and disturbances of ego boundaries

We will now turn to hallucinations and also briefly outline how the aforementioned processes, particularly liberal acceptance, may explain disturbances of the ego boundaries (e.g., thought insertion, thought broadcasting). This aspect of the theory is perhaps the one with the least empirical foundation. As mentioned before, unlike other models of psychosis (Davies et al., 2001; Kapur, 2003; Maher, 1974, 1999), we do not assume that aberrant sensory or sensual input in psychosis is a necessary driving factor neither for hallucinations nor for the formation of most delusions (see also Bell, Halligan, & Ellis, 2008; Chapman & Chapman, 1988), although it may shape the content of the delusion. We concur with Morrison (2001) that the main factor is a faulty *interpretation* of intrusions. Having said this we again would like to acknowledge that "biological" models of psychosis may well explain certain important delusional subtypes, particularly over-valued ideas or conditions with a strong hallucinatory component ("out-of-the-ordinary" experiences according to Maher) that are the brink of neurology and psychiatry, such as Capgras syndrome.

At times, loud, alien, vivid and persistent intrusive thoughts emerge in nonclinical individuals, too (Laroi et al., 2014; Vellante et al., 2012), and are very frequently observed in nonpsychotic conditions such as depression and obsessive-compulsive disorder (OCD). Approximately 50% of depressed patients (Moritz, Hörmann, et al., 2014) and even 75% of OCD patients have intrusions of some perceptual quality (Moritz, Claussen, Hauschildt, & Kellner, 2016; Röhlinger, Wulf, Fieker, & Moritz, 2015), that is, patients can "hear" their inner critic or see upcoming catastrophes. Although such phenomena are considered strange and somehow alien by its holder, the nonpsychotic individual can still acknowledge "authorship" (i.e., hold them for unreal and self-generated). In psychosis, the same input is externalized and equipped with a delusional superstructure. We found (Moritz, Rietschel, et al., 2016; Moritz & Larøi, 2008) that the "voices" in patients with psychosis are often not as *a*coustic, *a*lien (i.e., appears as non-self), *a*utonomous (i.e., beyond subjective control) and *a*uthentic (i.e., appears like a real voice; the four A's of hallucinations) as one would expect from contemporary definitions of hallucinations. The main difference is that patients – according to our account – liberally accept a bizarre explanation which externalizes self-generated phenomena. Patients with disorders outside the psychotic spectrum ultimately acknowledge their aberrant experiences as a mere "as if" feeling (e.g., "it is *as if* there is someone talking to me") due to their higher standards for evaluating hypotheses (high decision threshold). Psychotic patients, in contrast, contemplate the false inference, which receives further impact and eventually prevails by means of stage 2 processes. Of note, it is often easier to provide further evidence in favor of a delusional explanation than for a medical one as the true origins of, for example, loud thoughts, tinnitus etc. are not well understood. Interestingly, delusions pick up the *zeitgeist* and progress with scientific knowledge (Stompe, Ortwein-Swoboda, Ritter, & Schanda, 2003); today, voice-hearers are no longer preoccupied with telephones as they were in the 1920s (Steinebrunner & Scharfetter, 1976) showing that patients are able to refute hypotheses that have become utterly absurd. In many cases delusions are not impossible but often just improbable by current scientific standards.

The same mechanism may also apply for dysfunctions of ego boundaries. For example, lack of attention resulting in derailed or blocked thoughts might bring about ideas of a brain disorder, dementia or paranoid content. Again, such "as if" intrusions are not pathological per se but only if accepted and remain subsequently unchallenged. Prominent other first-rank symptoms deal with the permeability of ego boundaries (e.g., thought broadcasting, thought insertion) and thought-action fusion. While such symptoms may indeed be considered bizarre, they are by no means confined to people with schizophrenia and are thus not pathognomonic (Carpenter, Strauss, & Muleh, 1973). Some of these symptoms are observed in, for example, nonpsychotic OCD patients. However, unlike in patients with psychosis, such ideas are held in check by doubt (feeling that one's thoughts could bring misfortune to others). The person with OCD separately seeks for an explanation, which usually does not bring forward a delusional idea or if so, the individual is usually able to acknowledge the absurdity of the intrusion.

1.4. The role of (cold) neuropsychological dysfunctions – aggravating but not necessary conditions

Neuropsychological deficits increase the chances of false inferences but do not represent a sufficient or even necessary condition for the formation of positive symptoms according to our account. While neuropsychological performance dysfunctions undoubtedly exist in patients with psychosis (for reviews and meta-analyses see Fatouros-Bergman, Cervenka, Flyckt, Edman, & Farde, 2014; Heinrichs & Zakzanis, 1998; Keefe & Harvey, 2012;

[4] The related idea that delusions serve an "ego defense" function, that is, they guard one's self-esteem or lift it (Bentall et al., 2001; Lincoln, Stahnke, & Moritz, 2014) is currently not well supported (Galbraith & Manktelow, 2015; Mackinnon, Newman-Taylor, & Stopa, 2011), which might be owing to methodological problems (Moritz, Werner, & von Collani, 2006).

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Schaefer, Giangrande, Weinberger, & Dickinson, 2013) their multicausal origin and their putative specificity are subject to an ongoing debate. Not all neurocognitive impairments reflect primary brain dysfunction but can be caused by the effects of medication such as anticholinergic agents (Vinogradov et al., 2009) and benzodiazepines (Barker, Greenwood, Jackson, & Crowe, 2004) as well as by distraction due to voice hearing and poor motivation (Fervaha et al., 2014). We therefore regard neurocognitive impairments as factors which can worsen the severity of the illness, but are probably not causal factors. First, many studies assert that positive symptoms are not correlated with simple cognitive deficits (Keefe & Harvey, 2012). Some theorists have even put forward the idea that paranoid ideas require the capability of constructing complex systems and that paranoid patients therefore have even better than average intelligence (Frith, 1979). Moreover, many patients with other mental and neurological disorders who do not display positive symptoms show neuropsychological impairments of similar or larger magnitude. In addition, if patients are aware of their compromised cognitive faculties (i.e., have good metacognitive awareness; Balzan, Neaves, Denson, Liu, & Galletly, 2014), momentous errors are not to be expected. In this case, patients will still commit errors but will attach doubt to them (referred to as "not trustworthy tags"; Moritz, Woodward, & Ruff, 2003).

2. Explanation why pharmacological and non-pharmacological treatments may work

We believe that a central element of the treatment of psychosis is to "plant the seeds of doubt", that is, to get patients to reflect on their judgment, decrease overconfidence in errors and look for further evidence before making momentous and firm judgments. Indeed, a new line of research shows that metacognitive (Eichner & Berna, 2016; Moritz, Andreou, et al., 2014) and reasoning training (Waller et al., 2015) decrease delusions and other positive symptoms at a small to medium effect size (Eichner & Berna, 2016). Preliminary evidence suggests that this might be owing to decreasing overconfidence (Köther et al., submitted) and raising the decision threshold (Andreou et al., 2016) but the mechanism of action is not fully elucidated and our hypothesis remains speculative at this point.

Mounting evidence suggests that apart from emotional detachment (Mizrahi et al., 2006; Moritz, Andreou, Klingberg, Thoering, & Peters, 2013) one of the mechanisms of action of antipsychotic agents, whose common denominator is a dopamine receptor antagonism, is the induction of "doubt" (however see also Mizrahi et al., 2006). This has been shown both at a subjective (Moritz et al., 2013) as well as objective level in dopamine challenge studies (Andreou, Moritz, Veith, Veckenstedt, & Naber, 2013; Andreou et al., 2015). In line with these findings, several correlational studies also found a relationship between the reduction of confidence and antipsychotic drug dose (e.g., Moritz et al., 2008, 2003) and less liberal acceptance under higher antipsychotic medication (Moritz & Woodward, 2004). It deserves to be investigated further why patients with acute schizophrenia generally respond better than those with more chronic symptoms (Salimi, Jarskog, & Lieberman, 2009). One reason might be that psychological motifs and processes (see above) are more relevant in patients with a longer duration of illness or more systematized delusional ideas (typical for stage 2 in our model), which are less sensitive to the effects of medication.

Recent research shows that working on emotional problems may also reduce paranoia (Freeman et al., 2015). As described above, a delusional idea will more likely gain strength and persist if it is congruent with the person's present emotional state, if it serves an "ego function", particularly to lift one's self-esteem, and if it resolves ambiguity. In our view, removing emotional problems may not strongly impact stage 1 delusions but may improve stage 2 delusions.

3. How does our theory relate to other accounts of positive symptoms

As acknowledged above, some of our assumptions build upon prior work. For example, our view of stage 2 positive symptoms largely overlaps with psychological accounts by, for example, Freeman et al. (2002). Stage 2 also bears strong resemblance to the second step of the delusion theory by Coltheart (e.g., Coltheart et al., 2011) which aims to explain how a delusion is adopted and maintained; Galbraith and Manktelow (2015) already noted that the second stage of Coltheart's model is consistent with the aforementioned BADE findings, where patients show resistance to disconfirmatory data.

It is also tempting to link our account with early assumptions made by Karl Jaspers (1963). Stage 1 delusions seem analogous to what Jaspers referred to as delusional ideas that are not yet solid. Many of these carry the character of autochthonous delusions, for which the "bottom up" accounts of Maher, Coltheart and Kapur (see above) present powerful theories. At first sight, stage 2 delusions seem close to Jaspers' concept of "delusion proper", in the sense that the belief is held with utter conviction. A difference is that Jaspers assigned diagnostic importance to bizarre delusional ideas to qualify as delusion proper. However, bizarre ideas are often very volatile (e.g., Dollfus & Petit, 1995) and therefore more typical of stage 1 delusions (some bizarre delusions may be convictions, however). Moreover, as elaborated above, stage 2 delusions in our view are more than stage 1 delusions determined by psychological (i.e., partially willed/conscious) processes contrary to Jaspers who considered true delusions as "un-understandable".

An even more parsimonious (one-stage) theory has been published by Fletcher and Frith (2009) that tries to explain positive symptoms by a single deficit, defined as a disturbance in error-dependent updating of inferences and beliefs about the world (i.e., an extension of Kapur's 'aberrant salience' model). Although this prediction-error theory has been viewed as fundamentally different from Coltheart and colleagues two-factor model, Miyazono et al. (2015) have suggested that it is possible to incorporate both approaches into a single theoretical framework. This conciliatory strategy might overcome the inherent problems of both theories when considered in isolation (e.g., unspecific nature of the secondfactor; aberrant prediction-error signals are sometimes dissociable from delusions and the theory does not say much about maintenance). The specific relationship between our account and Miyazono's incorporation of the prediction-error theory into the two-factor framework needs to be tested further.

4. Open questions and future directions

While some parts of our theory are well established (e.g. overconfidence in errors), many pieces of the puzzle are still preliminary and require thorough validation by independent studies. For example, evidence suggests (see Lüdtke et al., this issue) that hasty decision making waxes and wanes and that the decision threshold may therefore be flexible. We suspect that liberal acceptance is not ubiquitous in patients with psychosis but may be aggravated under defined circumstances. One of these candidate circumstances is stress. There is indeed some evidence to implicate stress and emotional involvement in hasty decision making (Lincoln, Peter, Schäfer, & Moritz, 2010; Lincoln, Salzmann, Ziegler, & Westermann, 2011; Moritz, Köther, Hartmann, & Lincoln, 2015; Moritz et al., 2011) but independent replications are lacking.

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Likewise, while we have not found any strong associations between a lowered decision threshold and symptom severity, which tentatively suggests that the parameter is a trait vulnerability marker, more research is needed. Affairs are very complicated, as patients at stage 2 often show overall higher probability ratings (Moritz, Scheu et al., 2016) and therefore no liberal acceptance. Moreover, most studies mentioned above highlighted the JTC bias which is related to but, as argued before, is not the same as liberal acceptance (Moritz, Scheu et al., 2016) - liberal acceptance may at times even delay decision making (Moritz et al., 2007). Finally, as shown antipsychotics may induce doubt and thus heavily confound experiments examining confidence.

More studies on the specificity of the BADE, overconfidence and liberal acceptance are needed. It is unclear if these biases are confined to people with schizophrenia psychosis or also work in patients with psychotic features but different primary diagnoses (Gawęda, Mikuła, Szelenbaum, & Kokoszka, 2014; McLean et al., 2016). To illustrate, we have to address the question if, for example, the false belief (sometimes held with high confidence) that the individual might die or become crazy in patients with OCD is governed by similar processes to delusional ideas in psychosis.

In the future, we also need to be more strict with respect to terminology. For example, in some of our studies, we used terms like *decision* and *acceptance* (thresholds), or *probability* and *confidence,* almost interchangeably. However, when individuals are asked to make a *probability judgment* (on a 0–100% scale) for a response option, this may provide different results compared to asking the individual to judge whether they are *sure* or *unsure*, because the latter judgment may merge the conclusion process with a probability judgment (Balzan, 2016). A related methodological problem is that, if individuals are asked to provide probability estimates and make decisions, the two may not be as independent as would be desirable (e.g., participants may provide increased probability estimates once a decision is made; post-decisional confidence). We need new paradigms to verify the validity of this distinction and to investigate the generalizability of our findings. We must also acknowledge that we still do not know under which circumstances overconfidence in errors – a central feature of our theory – occurs and when it does not.

While our studies render motivational deficits unlikely as explanations for lowered decision thresholds, we must still entertain the possibility that the proposed cognitive abnormalities are an epiphenomenon caused by a hidden variable. As mentioned above, we think it is particularly interesting to take a closer look at the time course of decision thresholds and their moderators such as the potential cost of false conclusions (e.g., patients are perhaps less adaptive in their threshold when weighing risks and potential costs in case of false decisions).

We also think it is worthwhile to further pursue the hypothesis that stage 2 delusions, in particular, involve psychological processes that are amenable to psychological understanding. In our view, neither a biological model deriving positive symptoms from aberrant input nor a pure psychological perspective attempting to attribute positive symptoms mono-causally to biographical and social factors (e.g., parenting, social exclusion) can solve the riddle of psychosis.

We believe our theory has strengths and limitations. It provides some testable assumptions and will hopefully bring the field forward, regardless of whether it is (essentially) valid or a stage-1 delusional idea itself.

5. Declaration of interest

None.

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