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Dual-process theory, conflict processing, and delusional belief

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Abstract

Many reasoning biases that may contribute to delusion formation and/or maintenance are common in healthy individuals. Research indicating that reasoning in the general population proceeds via analytic processes (which depend upon working memory and support hypothetical thought) and intuitive processes (which are autonomous and independent of working memory) may therefore help uncover the source of these biases. Consistent with this possibility, recent studies imply that impaired conflict processing might reduce engagement in analytic reasoning, thereby producing reasoning biases and promoting delusions in individuals with schizophrenia. Progress toward understanding this potential pathway to delusions is currently impeded by ambiguity about whether any of these deficits or biases is necessary or sufficient for the formation and maintenance of delusions. Resolving this ambiguity requires consideration of whether particular cognitive deficits or biases in this putative pathway have causal primacy over other processes that may also participate in the causation of delusions. Accordingly, the present manuscript critically evaluates whether impaired conflict processing is the primary initiating deficit in the generation of reasoning biases that may promote the development and/or maintenance of delusions. Suggestions for future research that may elucidate mechanistic pathways by which reasoning deficits might engender and maintain delusions are subsequently offered.

Keywords

Delusions; Dual-process theory; Dual stream modulation failure; Conflict detection; Reasoning bias

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Contributors

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1. Introduction

Delusions can be defined as fixed false beliefs that are idiosyncratic or held by very few others in one's cultural reference group (American Psychiatric Association, 2013). Approximately 60 to 90% of individuals with schizophrenia experience delusional beliefs during the course of their illness (Harrow, MacDonald III, Sands, & Silverstein, 1995; Robinson, 1988). Recent research has identified a host of potentially delusion-promoting reasoning biases, including the bias toward discounting evidence against one's beliefs (Sanford, Veckenstedt, Moritz, Balzan, & Woodward, 2014; Woodward, Moritz, Cuttler, & Whitman, 2006), the bias toward increased consideration of evidence that matches one's hypotheses (Speechley, Whitman, & Woodward, 2010), the bias toward reduced data gathering before forming conclusions (Moritz & Woodward, 2005), and the bias toward lower hypothesis-acceptance thresholds (Moritz & Woodward, 2004). Research strongly suggests that these biases may contribute to the formation and/or maintenance of delusions (e.g., Broyd, Balzan, Woodward, & Allen, 2017; Eisenacher & Zink, 2017; McLean, Mattiske, & Balzan, 2017; Moritz et al., 2017). However, it has not been definitively determined how these biases might arise, how they might be interrelated, and whether any of them plays a causal role in forming or maintaining delusions.

Progress toward answering these important questions might be made by recognizing that these reasoning biases are not unique to individuals with delusions. Each of these biases can be observed to varying degrees in members of the general population. For instance, the exaggerated discounting of disconfirmatory evidence in delusional individuals may be an extension of the normal human tendency to resist belief modification. Human cognition appears organized to resist belief modification (e.g., Altemeyer, 2002; Kaplan, Gimbel, & Harris, 2016), particularly with respect to core beliefs that explain a wide range of experiences. Accordingly, humans generally display reasoning biases, such as confirmation bias and bias against disconfirmatory evidence (Balzan, Delfabbro, Galletly, & Woodward, 2013; Bronstein, Dovidio, & Cannon, 2017; Buchy, Woodward, & Liotti, 2007), that help to maintain core beliefs. These same biases are associated with resistance to belief modification in delusional individuals (Balzan et al., 2013; Sanford et al., 2014). Indeed, belief inflexibility appears to be a feature of personally meaningful beliefs in both delusional and healthy individuals, though in delusional individuals this lack of flexibility may extend to other beliefs as well (Colbert, Peters, & Garety, 2010). The quotidian nature of the bias against disconfirmatory evidence and other delusion-promoting reasoning biases suggests that the formation and/or persistence of delusional thoughts can be at least partially understood within the context of research on reasoning processes in the general population.

One of the richest traditions in this research area is the study of dual-process reasoning. For decades, it has been theorized that human cognition makes use of two types of reasoning (Epstein, 1994; James, 1890; Thompson, 2009; Wason & Evans, 1975; though this hypothesis remains subject to some debate, see Evans & Stanovich, 2013, for a review). The first, intuitive (Type I) reasoning, is autonomous and does not require working memory. The second, analytic (Type II) reasoning, relies on working memory and supports hypothetical thinking (Evans & Stanovich, 2013). Analytic reasoning engagement may depend upon the detection of and ensuing neurocognitive response to conflict. Detection of conflict between

competing intuitive responses is thought to initiate analytic reasoning (De Neys, 2012; Pennycook, 2017). Once analytic reasoning is initiated, it may produce outputs that further conflict with intuitions, potentially prompting additional analytic thinking (Evans, 2007). Ultimately, analytic reasoning may be employed in an attempt either to override initial (often intuitive) responses, or to increase confidence in initial responses (i.e., to rationalize or engage in motivated reasoning; Pennycook, Fugelsang, & Koehler, 2015). Furthermore, the success of either venture is likely to depend upon the mobilization of neurocognitive resources (see Goel & Dolan, 2003; Stollstorff, Vartanian, & Goel, 2012) in response to conflict detection.

The importance of conflict detection in analytic reasoning engagement has led several recent studies to examine the effectiveness of conflict monitoring in nonclinical populations. These studies have found that response conflict tends to increase reaction times and reduce response confidence, even when individuals give biased or normatively incorrect answers to reasoning problems (De Neys, Cromheeke, & Osman, 2011; De Neys & Glumicic, 2008; Pennycook, Fugelsang, & Koehler, 2015). Mirroring these findings, response conflict provokes increased activity in the anterior cingulate cortex (a brain region involved in conflict detection; see Carter & Van Veen, 2007) and an increased amplitude of the centro-parietal N2 electroencephalogram (EEG) waveform regardless of whether individuals' responses are normatively accurate (Bago et al., 2018; Vartanian et al., 2018). Taken together, these neuroimaging and behavioral studies suggest that conflict monitoring is typically quite effective in individuals from the general population.

Relative to these individuals, people with schizophrenia may exhibit deficits in conflict monitoring and/or in the subsequent mobilization of neurocognitive resources that support analytic reasoning. According to one recent theoretical account, Dual Stream Modulation Failure (Speechley & Ngan, 2008), these deficits may discourage analytic reasoning and thereby engender delusion-inspiring cognitive biases. For example, Dual Stream Modulation Failure hypothesizes that these deficits may encourage bias against disconfirmatory evidence by reducing individuals' drive to reconcile conflicting information, and may lead individuals to jump to conclusions in scenarios where available evidence supports multiple working hypotheses (Speechley, 2012). The former hypothesis is consistent with evidence that delusional individuals with schizophrenia make an especially large number of errors on syllogistic reasoning tasks when preexisting beliefs and deductive reasoning suggest conflicting appraisals of syllogism validity (Speechley, Whitman, & Woodward, 2010; Speechley, Woodward, & Ngan, 2013). The latter hypothesis is consistent with research suggesting that jumping to conclusions may be associated with reduced engagement in analytic reasoning processes (Ross et al., 2016; Ward, Peters, Jackson, Day, & Garety, 2017).

A more recent model employing dual-process theory to account for delusion-inspiring reasoning impairments (Ward & Garety, 2017) also posits that reduced engagement of analytic reasoning may contribute to delusions. More specifically, this model suggests that distressing delusions (e.g., those involving paranoia) may arise when interpretations of anomalous experiences and ambiguous/negative events are colored by over-reliance on intuitive reasoning and failure to override intuitions using analytic reasoning processes.

Over-reliance on intuitive reasoning processes is thought to color these interpretations by encouraging jumping-to-conclusions, while the reduction in interventionist analytic reasoning is thought to color them by decreasing belief flexibility (the metacognitive capacity to reflect on one's beliefs, revise them in response to evidence, and generate and consider alternatives; Ward & Garety, 2017). Alongside these theories linking delusions to dual-process reasoning, interventions such as "Thinking Well" (Waller et al., 2015) and "SlowMo" (Ward & Garety, 2017) have been developed with the goal of ameliorating the deficit in analytic reasoning observed in individuals with distressing delusions (see Freeman, Lister, & Evans, 2014).

This body of literature provides preliminary evidence that the formation and/or persistence of delusions may be impacted by reasoning biases that might arise from dual-process reasoning deficits, thereby suggesting that increased understanding of the links between these deficits and reasoning biases might herald useful treatments for delusions. Unfortunately, progress toward understanding potential pathways from dual-process reasoning deficits to reasoning biases and delusions is currently impeded by ambiguity regarding whether any of these deficits or biases is necessary or sufficient for the formation and maintenance of delusions.

Resolving this ambiguity requires consideration of whether a particular reasoning deficit or bias in these putative pathways has causal primacy over other processes that may also participate in the causation of delusions. For example, Dual Stream Modulation Failure (Speechley & Ngan, 2008; Speechley, Whitman, & Woodward, 2010) asserts that deficits in the detection of conflict *per se* (hereafter: "conflict detection"), in the process of engaging analytic reasoning following conflict detection (hereafter: "conflict responsiveness"), or both (hereafter: "conflict processing") might contribute to the formation and maintenance of delusions. If impaired conflict processing can be considered the primary initiating deficit in delusional individuals, it would be expected to precede and predict the emergence of delusions and to account fully for the relationships between delusions and reasoning biases that could conceivably result from failures of conflict detection and responsiveness, including reduced engagement in analytic reasoning and increased bias against disconfirmatory evidence. Further, this impairment would be expected to account for clinically-observable features of delusions. For instance, impaired conflict processing might be expected to result in an increased openness to unusual explanations (i.e., liberal acceptance), which in turn could help to explain the bizarre content of many delusions. Other contributory factors, such as stress and neurocognitive impairments (e.g., deficits in working memory and cognitive control), via their effects on the detection of conflict and/or its downstream consequences. Whatever its sources, deficits in conflict processing would result in a combination of reasoning biases expected to produce and maintain delusional beliefs, including reduced analytic engagement, liberal acceptance, bias against disconfirmatory evidence, and jumping to conclusions.

The present manuscript critically evaluates whether impaired processing of cognitive conflict serves as the primary initiating deficit in the generation of reasoning biases relevant to delusions and in the development and/or maintenance of delusions themselves. This evaluation begins by examining several potential pathways from impaired conflict

processing to reasoning deficits or biases and delusions (see Fig. 1). It continues by highlighting critical gaps in knowledge and avenues of future work that may help differentiate among competing theoretical alternatives.

2. Impaired conflict processing in delusional individuals with schizophrenia

Several lines of evidence suggest that individuals with schizophrenia as a group (irrespective of delusional status) are more likely than healthy controls to fail to detect conflicts that arise during reasoning. Electrophysiological studies suggest that individuals with schizophrenia have an attenuated N450 and no significant conflict sustained potential (SP) during the Stroop task, implying a deficit in conflict detection per se (McNeely, West, Christensen, & Alain, 2003). Further evidence for such a deficit comes from functional magnetic resonance imaging (fMRI) studies showing that individuals with schizophrenia (vs. controls) exhibit a reduced effect of conflict on the blood-oxygen-level-dependent (BOLD) signal in the anterior cingulate cortex (Dehaene et al., 2003; Kerns et al., 2005). Because anterior cingulate cortex activity might be sensitive to sources of conflict within but not outside of conscious awareness (Dehaene et al., 2003), the attenuation of this effect is consistent with the possibility that individuals with schizophrenia may have a specific deficit in conscious conflict monitoring; this possibility is strongly consistent with research showing that while motor conflict induced by a masked prime slows reaction times in both individuals with schizophrenia and healthy controls, conflicts induced by an unmasked prime only impact reaction times (and anterior cingulate cortex activity) in healthy controls (Dehaene et al., 2003). Although no prospective, longitudinal studies have examined these EEG or fMRI-based conflict detection measures prior to onset of full psychosis, those at clinical high risk who later develop full psychosis show progressive thinning of regions of superior and medial prefrontal cortex, including those likely to be involved in conflict monitoring (Cannon et al., 2015), which is at least circumstantially consistent with the hypothesis that progressive impairment in the ability to detect conflict might contribute to the development of clinically significant psychosis symptoms, including delusions.

Research on cognitive conflict has also more specifically examined delusional individuals with schizophrenia. A key corpus of this research examines belief bias, which reflects the tendency of individuals to evaluate the validity of an argument by examining the accordance of its conclusion with preexisting beliefs rather than by examining its logical structure (Evans & Curtis-Holmes, 2005). Belief bias is often assessed using syllogistic reasoning tasks in which preexisting beliefs and available evidence sometimes suggest conflicting appraisals of syllogism validity and other times suggest the same appraisal (e.g., Goel & Dolan, 2003; Klauer, Musch, & Naumer, 2000). Research employing these tasks indicates that delusional individuals with schizophrenia are more susceptible to belief bias than healthy controls and exhibit less of an increase in central executive network activation than healthy controls when deductive reasoning and preexisting beliefs suggest conflicting appraisals of syllogism validity (Speechley et al., 2013; Speechley, Whitman, & Woodward, 2010). These observations in delusional individuals with schizophrenia could be explained by impaired conflict detection, impaired conflict responsiveness, impaired ability to inhibit

intuitive beliefs in favor of less intuitive alternatives (see Directions for Future Research), or some combination of these deficits.

Taken together, the results reviewed in this section suggest that delusional individuals with schizophrenia are likely to have deficits in conflict detection and potentially also in conflict responsiveness. As developed more fully in the next section, these conflict processing deficits are likely to limit delusional individuals' engagement in analytic reasoning.

3. A direct path between impaired conflict processing and decreased analytic reasoning

Studies observing a correlation between the effect of conflict on reaction times and the degree to which individuals in the general population tend to engage in analytic reasoning (Pennycook, Cheyne, Barr, Koehler, & Fugelsang, 2014) indicate that the detection of conflict between competing responses may play an important role in the engagement of analytic reasoning. The putative role of conflict in analytic reasoning engagement is central to recent theoretical accounts of reasoning in the general population (De Neys, 2012; Pennycook, Fugelsang, & Koehler, 2015). It is also central to theory which posits that a reduced influence of conflict on the engagement of analytic reasoning encourages delusional beliefs (Speechley & Ngan, 2008). These theories and corresponding empirical evidence suggest that the conflict-processing impairments outlined in the previous section may directly result in reduced analytic reasoning in delusional individuals with schizophrenia.

Evidence supporting this suggestion can be derived by synthesizing research on syllogism evaluation in the general population and in delusional individuals with schizophrenia. In the general population, manipulations of the amount of time individuals have to complete syllogism evaluation tasks suggest that accurate syllogism evaluation in the face of conflicting cues regarding syllogism validity is partially the result of increased engagement in analytic reasoning following conflict detection. For example, when individuals are provided with less time to evaluate syllogisms (which should particularly impair relatively more effortful and, therefore, slow analytic reasoning; Bago & De Neys, 2017), they tend to give more incorrect answers in the face of conflicting cues regarding syllogism validity (Evans & Curtis-Holmes, 2005). One might therefore expect that if the conflict-processing impairments outlined in the previous section directly result in less analytic reasoning in delusional individuals with schizophrenia, these individuals might perform particularly poorly and under-recruit neural resources that might support analytic reasoning when faced with conflicting cues regarding syllogism validity. In accordance with this expectation, delusional individuals with schizophrenia in this situation exhibit particularly impaired syllogism evaluation performance and reduced activation of the central executive network (Speechley et al., 2013).

The evidence reviewed above favors a model in which impaired conflict processing leads to less engagement in analytic thinking among delusional individuals with schizophrenia. Future research could more conclusively evaluate this causal model via prospective, longitudinal studies examining whether conflict processing impairments precede and predict reduced engagement in analytic reasoning. If impaired conflict processing is causally

primary to reduced analytic thinking in the genesis and/or maintenance of delusions, impaired conflict processing could exert its influence on analytic reasoning directly (as when the lack of conflict “signal” results in failure to activate analytic reasoning). It could also influence analytic reasoning indirectly by promoting premature acceptance of implausible explanations, a phenomenon referred to as liberal acceptance. This possibility is discussed in the next section.

4. A direct path between impaired conflict processing and liberal acceptance

Liberal acceptance may promote delusions by causing premature acceptance of implausible explanations for events that are at first tentatively believed but are later elevated to delusional levels of conviction through processes including confirmation bias and bias against disconfirmatory evidence (Moritz et al., 2017). Two facets of liberal acceptance (which a recent meta-analysis suggests is characteristic of individuals with delusions and is not a characteristic of those with psychopathology generally; McLean et al., 2017) that may contribute to delusions in this manner are an increased willingness to consider the absurd and lowered decision thresholds. Delusional individuals with schizophrenia make decisions on the basis of less evidence than controls (Moritz et al., 2009; Veckenstedt et al., 2011). These lowered decision thresholds are thought to increase the chances that delusional individuals accept implausible response options. Consistent with this notion, delusional individuals with schizophrenia give higher plausibility ratings to absurd response options across multiple tasks in which liberal acceptance is thought to influence performance (e.g., Moritz et al., 2009; Moritz & Woodward, 2004; Moritz, Woodward, Jelinek, & Klinge, 2008; Sanford et al., 2014).

Conflict processing impairments can potentially account for both of these facets of liberal acceptance. Increased willingness to consider implausible explanations is thought to result from delusional individuals’ failure to detect inconsistencies between their hypotheses and available evidence (Moritz & Woodward, 2004). Given the putative role of conflict processing in the engagement of analytic reasoning (Pennycook, Fugelsang, & Koehler, 2015; Speechley & Ngan, 2008), failure to detect and appropriately respond to these inconsistencies may preserve confidence in implausible explanations by reducing the chances that delusional individuals with schizophrenia subject these explanations to additional scrutiny by analytic reasoning processes. Consistent with this notion, in the general population the absence (vs. presence) of conflicts between intuitive responses is associated with increased response confidence (De Neys et al., 2011), which in turn is associated with decreased engagement in analytic reasoning (Thompson, Turner, & Pennycook, 2011). Moreover, decreased analytic thinking is associated with a variety of outcomes that are conceptually related to liberal acceptance (see Pennycook & Rand, 2019 for a review), such as belief in fake news (Bronstein, Pennycook, Bear, Rand, & Cannon, 2019; Pennycook & Rand, 2018) and conspiracy theories (Swami, Voracek, Stieger, Tran, & Furnham, 2014), as well as receptivity to pseudo-profound bullshit (Pennycook, Cheyne, Barr, Koehler, & Fugelsang, 2015).

The decreased decision thresholds characteristic of liberal acceptance can also potentially be accounted for by conflict-processing-related impairments. In healthy individuals, theta band activity over the medial prefrontal cortex (mPFC) increases following conflict detection (Cavanagh et al., 2011). This activity elevates decision thresholds in the context of response conflict via a mechanism mediated by connections between the mPFC and the subthalamic nucleus (Cavanagh et al., 2011). Impaired conflict processing in delusional individuals with schizophrenia may attenuate this theta-band increase, resulting in the lower decision thresholds in these individuals seen in studies of liberal acceptance (e.g., Moritz et al., 2009; Moritz & Woodward, 2004; Veckenstedt et al., 2011). Consistent with the notion that response conflict may fail to inspire appropriate decision thresholds in these individuals, research has found that delusional individuals with schizophrenia are more willing than healthy controls to make decisions when the option they selected was only marginally better appraised than competing alternatives (Moritz et al., 2009; Veckenstedt et al., 2011). This failure of delusional individuals with schizophrenia to increase decision thresholds in the face of response conflict may be exacerbated by reduced modulation of frontal theta power by task demands in individuals with schizophrenia generally (Schmiedt, Brand, Hildebrandt, & Basar-Eroglu, 2005).

The reduced decision thresholds engendered by these deficits may discourage analytic thinking in two ways. Lower decision thresholds may cause earlier cessation of decision making, limiting the role of slower analytic processes relative to their faster intuitive counterparts (sometimes referred to as “reflexive open-mindedness”; see Pennycook & Rand, 2019). Lower decision thresholds may also discourage analytic reasoning by increasing confidence in incorrect relative to correct responses (i.e., by reducing the “confidence gap;” see Moritz et al., 2008). This effect may lead liberal acceptance to curtail consideration of incorrect responses by analytic processes, which are less often engaged in connection to responses given with greater confidence (Thompson et al., 2011). Accordingly, the reduction of analytic thinking in delusional individuals with schizophrenia directly resulting from impaired detection of cognitive conflict may be compounded by the indirect effects of this impairment that act via liberal acceptance.

The results reviewed above suggest that conflict processing deficits may have causal primacy with respect to liberal acceptance. However, it is important to consider the possibility that liberal acceptance may instead come first in this causal chain. If this were the case, one would predict that liberal acceptance would emerge prior to deficits indicative of impaired conflict processing. The possibility that the latter deficits may be evident during the schizophrenia prodrome is supported indirectly and circumstantially by evidence of accelerated thinning of regions of superior and medial prefrontal cortex involved in conflict monitoring and decreased conflict-related activation in the dorsolateral prefrontal cortex in at-risk cases who convert to psychosis (Cannon et al., 2015; Colibazzi et al., 2016). It is not clear whether liberal acceptance is present in individuals at risk of developing psychosis. Studies employing the bias against disconfirmatory evidence (BADE) task suggest that individuals at risk for psychosis do not liberally accept implausible response options (Eisenacher et al., 2016). This suggestion conflicts somewhat with meta-memory studies examining the confidence gap in at-risk individuals. Specifically, research indicates that at-risk individuals exhibit a greater confidence gap than healthy controls (Eisenacher et al.,

2015), implying that at-risk individuals may indeed exhibit liberal acceptance (which is thought to increase confidence gaps, Moritz et al., 2008). Future research should focus on clarifying whether liberal acceptance is indeed present in the schizophrenia prodrome using paradigms that more directly examine liberal acceptance (e.g., that in Moritz et al., 2008 or Moritz, Woodward, & Hausmann, 2005).

Just as impaired conflict processing is likely to encourage liberal acceptance, failure to detect and adequately respond to cognitive conflict may encourage bias against disconfirmatory evidence, a bias against revising beliefs when confronted with evidence that disconfirms them, in delusional individuals with schizophrenia. This possibility is explored in detail in the next section.

5. Impaired conflict processing may encourage BADE

Bias against disconfirmatory evidence is generally measured via the BADE task (Woodward et al., 2006; Woodward, Buchy, Moritz, & Liotti, 2007), in which individuals are lured into forming a belief that is then violated by increasing amounts of evidence. BADE is thought to allow delusional beliefs to persist in the face of evidence against them. Consistent with this notion, the number of delusion-like beliefs that individuals in the general population experience correlates with BADE (Bronstein & Cannon, 2017; Buchy et al., 2007). Also consistent with this notion, BADE is elevated in delusional individuals compared to both psychiatric and healthy controls (e.g., Sanford et al., 2014; Speechley, Ngan, Moritz, & Woodward, 2012). A recent meta-analysis suggests that BADE may afflict individuals with delusions specifically rather than psychiatric populations in general (McLean et al., 2017). However, research published after this meta-analysis was completed indicates that depressed and socially anxious individuals may exhibit BADE when revising emotion-laden beliefs about themes such as social rejection and failure (Everaert, Bronstein, Cannon, & Joormann, 2018).

Research employing the BADE task (the current standard for quantifying BADE) is consistent with the possibility that BADE may result from impaired conflict processing. In the BADE task, multiple scenarios are presented along with a series of explanations for the events they detail. These explanations can be placed into three categories: Lure explanations, which are initially most plausible but become less so; True explanations, which are initially moderately plausible but most plausible by the end of the scenario; and Absurd explanations, which remain implausible throughout the scenario. Recent research using confirmatory factor analysis to model BADE task performance suggests that Evidence Integration Impairment, the component of BADE task data associated with failure to revise beliefs in the presence of disconfirmatory evidence (Bronstein & Cannon, 2018; also called Evidence Integration by Sanford et al., 2014; Speechley et al., 2012), can be effectively modeled using only individuals' final ratings of Lure explanations along with all ratings for Absurd explanations (Bronstein & Cannon, 2018). Impaired conflict processing is likely to influence final ratings of Lure explanations: at the end of each scenario, the perceived plausibility of these explanations is subject to strong conflict between prior beliefs established at the start of the scenario and available evidence. Absurd explanation ratings are also likely to be influenced by the ability to detect and respond to cognitive conflict. These ratings, by their

implausible nature, are likely subject to the influence of liberal acceptance, which (as mentioned earlier) can be tied to impaired conflict processing. Notably, this analysis is consistent with theory suggesting that liberal acceptance may cause BADE (Sanford et al., 2014). In accordance with this theory, liberal acceptance (lowered decision thresholds) correlates with increased BADE (Veckenstedt et al., 2011).

If impaired conflict processing does encourage BADE, it may do so via two indirect pathways going through liberal acceptance. Impaired conflict processing may cause liberal acceptance and, in turn, BADE (as suggested by research reviewed in this and previous sections). Impaired conflict processing could also indirectly impact BADE by increasing liberal acceptance and thereby reducing engagement in analytic reasoning (see previous section). The viability of this second pathway depends upon the hypothesis that analytic reasoning engagement can influence BADE. In the general population, this hypothesis is supported by the negative correlation between BADE and analytic reasoning that is observed when statistically controlling for response bias (Bronstein, Everaert, Castro, Joormann, & Cannon, 2019). Support for this hypothesis can also be gleaned from observations that the belief bias effect (i.e., the impairment in syllogism evaluation when available evidence and prior beliefs conflict) is exaggerated in delusional individuals with schizophrenia (Speechley, 2012; Speechley, Whitman, & Woodward, 2010). The exaggeration of this effect in these individuals suggests that they are less able to engage deliberative reasoning processes when prior belief and available evidence suggest conflicting responses. Dual Stream Modulation Failure theory posits that this impairment may encourage BADE in delusional individuals with schizophrenia by reducing their drive to reconcile conflicting evaluations, thereby allowing erroneous explanations to coexist with logical ones and to remain unmodified even in the face of mounting disconfirmatory evidence (Speechley, 2012; Speechley, Murray, McKay, Munz, & Ngan, 2010). Future research should more thoroughly examine these potential indirect pathways from impaired conflict processing to BADE. Future research should also examine the possibility that impaired conflict processing might encourage BADE more directly.

Although the evidence reviewed above most strongly supports the notion that impaired conflict processing might result in BADE, it is nevertheless important to consider an alternative model in which BADE is the primary deficit in the causal chain. An appeal to temporal priority suggests that this alternative model is unlikely: as stated earlier, deficits that may be consistent with impaired conflict processing are present during the schizophrenia prodrome (Cannon et al., 2015; Colibazzi et al., 2016). The one study on BADE in at-risk individuals suggests that BADE may not be present during the prodrome (Eisenacher et al., 2016). Notably, this study observed that at-risk individuals as a group do not liberally accept absurd response options in the BADE task, although it remains unclear whether those who go on to develop full psychosis exhibit lower decision thresholds prior to psychosis onset. Given evidence suggesting that liberal acceptance may cause BADE (Sanford et al., 2014; Veckenstedt et al., 2011), this observation strengthens the case that BADE may be absent in at-risk individuals. Stronger evidence regarding the presence or absence of BADE in prodromal individuals could be obtained in a prospective, longitudinal study that examines BADE in at-risk individuals who later convert to psychosis.

6. Insufficient engagement in analytic reasoning may encourage individuals to jump to conclusions

Just as reduced engagement in analytic reasoning may cause delusional individuals with schizophrenia to exhibit a bias against disconfirmatory evidence, research indicates that less analytic reasoning may encourage these individuals to make decisions after gathering less data (i.e., it may encourage them to jump to conclusions). Research on jumping to conclusions has generally been conducted using the “Beads task” (but has increasingly employed the “Lakes task”). In both these tasks, individuals see two jars (lakes) filled with different proportions of colored beads (fish). Individuals can ask to see a series of beads (fish) that they are told came from one of the jars (lakes). Studies employing these tasks generally find that, unlike healthy controls, delusional individuals decide where the series came from after as few as one such request (e.g., Moritz & Woodward, 2005). This data gathering bias may result in delusional ideation by making it harder to maintain the skepticism of one’s working hypotheses necessary to critically evaluate presently available evidence for these hypotheses in relation to prior beliefs (see Langdon, Still, Connors, Ward, & Catts, 2014).

Consistent with the notion that this data gathering bias can engender delusions, changes in the jumping to conclusions bias have been linked to changes in delusional ideation (Winton-Brown et al., 2015; Woodward, Munz, LeClerc, & Lecomte, 2009). However, it is unclear whether this bias is present only in delusional individuals. Some research supports this notion (McLean et al., 2017; Peters, Thornton, Siksou, Linney, & MacCabe, 2008), while other studies suggest that jumping to conclusions may be associated with schizophrenia more generally (e.g., Dudley, Taylor, Wickham, & Hutton, 2016). The jumping to conclusions bias may even generalize beyond individuals diagnosed with schizophrenia; it has been observed in individuals with increased genetic risk for schizophrenia (Van Dael et al., 2005). As such, jumping to conclusions may be best conceptualized as a factor that increases vulnerability for delusional beliefs.

In situations where the available evidence supports multiple working hypotheses, jumping to conclusions is thought to result from failure to modulate toward analytic reasoning when the responses suggested by intuitive and analytic reasoning conflict (Speechley, 2012; Speechley, Murray, et al., 2010). This hypothesis suggests that the reduced reliance on analytic reasoning putatively produced by impaired conflict processing might be an important factor that inspires jumping to conclusions. Consistent with this notion, research employing a dual-process framework strongly suggests that jumping to conclusions is associated with reduced reliance on analytic reasoning. Reduced reliance on analytic reasoning (during an interview examining appraisals of anomalous experiences) has been associated (at a trend level) with jumping to conclusions in a sample of individuals with persistent psychotic experiences (Ward et al., 2017). Additionally, because analytic processes are thought to rely upon working memory, the relationship between working memory impairment and jumping to conclusions in delusional individuals (see Freeman, Lister, & Evans, 2014; Garety et al., 2013) is consistent with the notion that reduced reliance on analytic processes may encourage this data gathering bias. In the general population, an

association between reduced engagement in analytic reasoning and jumping to conclusions has also been observed (Ross et al., 2016).

Less direct evidence for the notion that reduced reliance on analytic reasoning may be responsible for jumping to conclusions in delusional individuals with schizophrenia is similarly abundant. For instance, this notion is consistent with evidence that factors that may cause individuals to jump to conclusions, such as liberal acceptance, hypersalient evidence-hypothesis matches, and acute stress (see Moritz et al., 2010; Moritz, Woodward, & Lambert, 2007; Speechley, Whitman, & Woodward, 2010), might also cause them to rely less on analytic thinking. The lower decision thresholds characteristic of liberal acceptance (Moritz et al., 2007) may attenuate the role of analytic reasoning processes due to these processes' generally slow nature. This attenuation may be amplified by the hypersalience of evidence-hypothesis matches in delusional individuals, which may increase confidence in responses that match individuals' working hypotheses, thereby decreasing the chances that these responses are subjected to analytic reasoning (see Thompson et al., 2011). Acute stress, which reduces analytic thinking (see Margittai et al., 2016; Otto, Raio, Chiang, Phelps, & Daw, 2013), may do so by depleting cognitive resources (cognitive control, working memory) on which analytic thinking may depend (Arnsten, 2015; Qin, Hermans, van Marle, Luo, & Fernández, 2009). The potential for all three of these putative causes of jumping to conclusions to reduce analytic thinking suggests that this reduction may be a common mechanism by which these factors engender this reasoning bias.

In summary, a variety of evidence suggests that reduced engagement in analytic thinking may encourage delusional individuals with schizophrenia to jump to conclusions. This evidence suggests that impaired conflict processing in these individuals may encourage jumping to conclusions by both directly and indirectly (e.g., through liberal acceptance) reducing engagement in analytic reasoning. Nevertheless, it is important to consider whether jumping to conclusions might be the primary deficit in this causal pathway. The possibility that jumping to conclusions might cause impaired conflict processing in delusional individuals with schizophrenia cannot be ruled out by considering temporal priority. Just as there is evidence of impaired conflict processing in prodromal individuals (Cannon et al., 2015; Colibazzi et al., 2016), some evidence suggests that putatively prodromal individuals may jump to conclusions (Rausch et al., 2015; Rausch et al., 2016). However, the earlier of these studies also found that these same individuals do not jump to conclusions when a modified task version is employed (Rausch et al., 2015). Another study (Broome et al., 2007) found that putatively prodromal individuals jump to conclusions only when task difficulty increases beyond that used in the aforementioned studies.

Although the possibility that jumping to conclusions causes impaired conflict processing cannot be ruled out by considering temporal priority, this possibility is somewhat inconsistent with evidence suggesting that liberal acceptance might cause jumping to conclusions (Moritz et al., 2007). As mentioned earlier, it is most likely that liberal acceptance is caused by conflict processing impairments (and not viceversa). If conflict processing impairments do cause liberal acceptance, and liberal acceptance causes jumping to conclusions, this would clearly make conflict processing impairment causally primary to jumping to conclusions. Future research should examine this potential causal pathway in

greater detail. Future research might also build upon the evidence (reviewed in this section) that reduced analytic reasoning (secondary to impaired conflict processing) encourages jumping to conclusions. This future research might manipulate analytic reasoning using a paradigm similar to that of Evans and Curtis-Holmes (2005). It might also employ Pennycook and colleagues' (2015) rapid-response base-rate task to examine the association between analytic reasoning engagement specifically due to conflict and jumping to conclusions.

Future research should also examine whether jumping to conclusions contributes to delusions in a manner that goes above and beyond the contribution of related constructs, including analytic reasoning, liberal acceptance, and hypersalient evidence-hypothesis matches. This future work might build upon previous research examining the relationships between these constructs and the jumping to conclusions bias. Previous research indicates that delusional individuals completing the Lakes task find the matches between available evidence and working hypotheses hypersalient, which may encourage these individuals to jump to conclusions when available evidence uniformly supports a single response option (Speechley, Whitman, & Woodward, 2010). Previous research also indicates that liberal acceptance might cause individuals to jump to conclusions (Moritz et al., 2007). In light of this previous research, longitudinal studies examining whether changes in liberal acceptance and hypersalient evidence-hypothesis matches precede and predict changes in data gathering behavior (e.g., jumping to conclusions) would be a reasonable first step toward determining whether or not the tendency to jump to conclusions is simply a behavioral manifestation of these other constructs and makes no unique contribution to delusions.

7. Other sources of reduced analytic reasoning in delusional individuals with schizophrenia

Several factors might compound the reduction in analytic reasoning that may be primarily attributable to impaired conflict processing in delusional individuals with schizophrenia. One such factor is dysregulated dopamine transmission, which may lead delusional individuals with schizophrenia to overvalue evidence that matches a given working hypothesis (i.e., to find evidence-hypothesis matches hypersalient), potentially leading these individuals to jump to conclusions (Broyd et al., 2017; Evans, Averbeck, & Furl, 2015). This possibility is supported by research employing the Lakes task, which indicates that delusional individuals with schizophrenia give higher plausibility ratings (than healthy and psychiatric controls) to whichever response option is best supported by the most recently presented piece of evidence, but do not differ from other groups in their ratings of the non-supported response option (Speechley et al., 2010).

This hypersalience effect may increase individuals' confidence in intuitive responses. This hypothesis is broadly consistent with Speechley and colleagues' (2010) observation that delusional individuals with schizophrenia display hypersalient evidence-hypothesis matches in contexts where serially-presented evidence uniformly supports a single response option, which could be expected to make the consistently-supported response option intuitive. The hypothesis that hypersalient evidence-hypothesis matches may increase intuitive response

confidence is more strongly consistent with the higher initial plausibility ratings given to Lure explanations (which are, at first, the most intuitive) by delusional individuals with schizophrenia in a BADE task analogue (Balzan et al., 2013; though in the original BADE task higher initial Lure ratings have not been consistently observed, see Sanford et al., 2014). Given that greater initial response confidence is associated with reduced engagement in analytic thinking (Thompson et al., 2011), this putative effect of hypersalient evidence-hypothesis matches on confidence in intuitive responses may curtail engagement in analytic thinking.

Like hypersalient evidence-hypothesis matches, general neurocognitive impairments might reduce analytic reasoning in delusional individuals with schizophrenia. These impairments include the reduced recruitment of neural resources during tasks involving executive function (meta-analysis: Minzenberg, Laird, Thelen, Carter, & Glahn, 2009). Consistent with the notion that reduced recruitment of these resources might decrease analytic reasoning, reduced engagement of the central executive network in delusional individuals with schizophrenia has been associated with reduced analytic thinking in the belief bias task (Speechley et al., 2013). Working memory deficits are a second neurocognitive impairment that may reduce analytic thinking in delusional individuals with schizophrenia. A meta-analysis of studies examining memory in individuals with schizophrenia suggests that these individuals have difficulty early in the process of maintaining information in working memory (Lee & Park, 2005). This difficulty may reduce analytic reasoning – and, specifically, responsiveness to conflict – given its putative dependence on working memory (Evans, 2003).

The potential for impairments in working memory and executive function to reduce analytic thinking in delusional individuals with schizophrenia is notable because it suggests that biological risk factors for schizophrenia might lead to reductions in analytic thinking. Evidence suggests that genes associated with schizophrenia risk impair both cognitive control and working memory (see Zheutlin et al., 2016; Skitskoorn et al., 2004). For example, recent research employing machine-learning algorithms suggests that genes associated with schizophrenia in genome-wide association studies (GWAS) are reliably associated with performance on visual, verbal, and working memory tasks (Zheutlin et al., 2018). Obstetric complications associated with schizophrenia may also impair working memory and cognitive control. Maternal infection is linked to impaired performance on cognitive control tasks (Brown et al., 2009) and, in animal models, is linked to impaired working memory (Meyer, Knuesel, Nyffeler, & Feldon, 2010). Low birth weight has also been associated with impaired working memory and cognitive control (Freedman et al., 2012). Fetal hypoxia is associated with reduced hippocampal volume (van Erp et al., 2002), which in turn is associated with reduced performance on verbal declarative memory tasks (Seidman et al., 2002).

More speculatively, impairments related to consciousness, which is hypothesized to be closely intertwined with working memory (Baars & Franklin, 2003), may also reduce analytic reasoning in delusional individuals with schizophrenia. It has been hypothesized that long-range functional connectivity is essential to consciousness (Baars, 2005). Multiple biological processes associated with schizophrenia might enervate the long-range neural

connections that partially determine this connectivity. For example, decreased *N*-methyl-D-aspartate (NMDA) receptor function, which is thought to impede synaptic plasticity, could prevent long-range connections from becoming strong enough to avoid falling victim to over-zealous pruning in individuals with schizophrenia (Stephan, Friston, & Frith, 2009). In this manner, decreased NMDA receptor function could amplify the deleterious effects of genetic factors, like Neuregulin-1 (Mei & Xiong, 2008), on long-range neural connections in individuals with schizophrenia. The efficiency of long-range connections that survive these insults could be limited by the reduced myelination present in schizophrenia (Flynn et al., 2003). Given that analytic reasoning processes (but not their intuitive counterparts) are thought to operate mainly (but perhaps not entirely) within conscious awareness (Evans, 2003; Evans & Stanovich, 2013), these impairments to consciousness may reduce analytic reasoning in delusional individuals with schizophrenia.

Research indicates that acute stress may also decrease analytic reasoning in delusional individuals with schizophrenia. Acute stress may reduce analytic thinking by increasing liberal acceptance, decreasing the availability of resources on which analytic thinking may depend, and by reducing the sensitivity of the ventromedial prefrontal cortex (VMPFC) to changes in outcome value. The potential for acute stress to increase liberal acceptance stems from observations that acute stress reduces the ability of processes like working memory to modulate prefrontal theta activity (Gärtner, Rohde-Liebenau, Grimm, & Bajbouj, 2014). As mentioned earlier, modulation of prefrontal theta activity (over mPFC) may be necessary to raise decision thresholds following conflict detection (Cavanagh et al., 2011). The reduction of frontal theta modulation following acute stress may therefore lead to liberal acceptance in the face of cognitive conflict. This increase in liberal acceptance may reduce analytic thinking by limiting the time necessary to make a decision, which might decrease analytic thinking given its relatively slow nature. Acute stress may also reduce analytic thinking by decreasing the availability of resources upon which analytic processes putatively depend. For instance, acute stress may reduce working memory capacity and encourage the allocation of resources away from networks involved in executive function (Qin et al., 2009). Genetic vulnerabilities over-represented in individuals with schizophrenia (e.g., Disrupted in Schizophrenia 1 [*DISC1*], mutations in the phosphodiesterase encoding gene *PDE4A*) may decrease the ability of these individuals to resist these deleterious effects of stress (Arnsten, 2015). Finally, the release of norepinephrine and glucocorticoids elicited by acute stress may reduce the role of analytic processes in decision making by decreasing the sensitivity of the VMPFC, which is more active when individuals engage in belief-based responding that may be mediated by intuitive processes (Goel & Dolan, 2003), to changes in outcome value (see Arnston, 2015).

In general, the possibility that these risk factors contribute to delusions via their effects on reduced analytic thinking ability has not been investigated. As a first step toward investigating this possibility, future research could employ serial mediation models to test whether these risk factors contribute to delusion-relevant reasoning biases by reducing analytic reasoning. For example, one might examine whether fetal hypoxia's aforementioned effect on hippocampal volumes might decrease working memory capacity, thereby impairing analytic reasoning and encouraging jumping to conclusions. The latter portion of this serial mediation pathway is consistent with research indicating that reduced working memory

capacity might encourage jumping to conclusions (e.g., Freeman, Lister, & Evans, 2014; Garety et al., 2013). It is also consistent with evidence suggesting that insufficient engagement in analytic reasoning might encourage jumping to conclusions (e.g., Ross et al., 2016).

8. Directions for future research

The forgoing sections of this manuscript favor an account in which impaired conflict processing (in tandem with other factors) reduces analytic thinking, leading to reasoning biases and delusions in individuals with schizophrenia (see Fig. 1). This examination was conducted in a nascent research area; it offers preliminary support for the causal primacy of conflict processing impairments in pathways leading to delusions. Accordingly, it is particularly important to consider primary competing hypotheses that future research may pit against the account described thus far.

One possible competing hypothesis is that deficits in conflict processing are secondary to neurocognitive deficits that can be observed even in the absence of response conflict. For example, individuals with schizophrenia have difficulty early in the process of maintaining information in working memory (Lee & Park, 2005). This deficit may impair analytic reasoning, which is thought to depend upon working memory (see Evans, 2003), making analytic reasoning less likely to yield desirable outcomes. As a result, delusional individuals with schizophrenia may learn to avoid analytic reasoning because the benefit it offers may often be outweighed by the effort it requires, particularly in light of the general tendency of individuals with schizophrenia to avoid exerting effort in cognitive tasks (Gorrisen, Carlos Sans, & Schmand, 2005; Fortgang & Srihari, 2017). This learning may manifest as a tendency to allocate less attention to cognitive conflict, which under normal circumstances leads to analytic reasoning engagement (Pennycook, Fugelsang, & Koehler, 2015). Reduced attentiveness to cognitive conflict may decrease analytic reasoning engagement and could explain the conflict processing deficits observed in delusional individuals with schizophrenia. This account is depicted in Fig. 2.

It is currently unclear whether this account represents a strong alternative to theories in which conflict processing is the causally primary deficit leading to delusions. This account predicts that neurocognitive deficits observable even in the absence of response conflict should be correlated with delusion-related cognitive biases. The association between working memory deficits and both BADE (Eifler et al., 2014; but, see Eisenacher et al., 2016 and Riccaboni et al., 2012) and jumping to conclusions (Freeman, Lister, & Evans, 2014; Garety et al., 2013) is therefore broadly consistent with this account. However, the account depicted in Fig. 2 also predicts that cognitive training targeting working memory would reduce BADE and delusional ideation in individuals with schizophrenia. Evidence against this account therefore comes from a small treatment study in which cognitive remediation alone did not reduce BADE, nor did reductions in BADE following combined metacognitive training and cognitive remediation associate with improved neurocognitive performance (Buonocore et al., 2015).

Future research should examine the account depicted in Fig. 2 to better ascertain its plausibility. For example, treatment studies employing a larger sample than that in Buonocore et al. (2015) could better determine whether cognitive training yields improvements in conflict processing and delusions. Future treatment studies could also examine whether interventions that increase effort expenditure in individuals with schizophrenia yield a significant improvement in conflict processing and delusions. These improvements could be expected under the account depicted in Fig. 2 given that cognitive deficits in individuals with schizophrenia may largely result from decreased effort expenditure (Fortgang & Srihari, 2017). Finally, prospective, longitudinal studies could examine the course of both conflict processing deficits and working memory impairments in at-risk individuals (e.g., in a cohort with a family history of psychosis and a personal history of pregnancy and birth complications) who go on to develop schizophrenia (and delusions). If working memory impairments did not precede and predict deficits in conflict processing in such a study, the account depicted in Fig. 2 would require revision. If the account depicted in Fig. 2 proves plausible, future research should examine the merits of this account in relation to accounts in which conflict processing deficits are causally primary.

In addition to investigating alternative accounts like the one depicted in Fig. 2, future research examining delusional individuals with schizophrenia should attempt to more fully differentiate between deficits at different stages of conflict processing. For example, the attenuated central executive network activity that may explain these individuals' reduced engagement in analytic reasoning in the presence of conflict (Speechley et al., 2013) may result from impaired conflict detection, impaired conflict responsiveness, or both. Additional clarity regarding the cause of this attenuated activity may be gleaned from research examining whether changes in conflict detection ability in delusional individuals with schizophrenia can fully account for the emergence of reduced central executive network activation in the presence of conflict. This future research could measure conflict detection ability using techniques employed in previous studies of dual-process reasoning in the general population (e.g., Bago et al., 2018). Future studies attempting to better differentiate between deficits at various stages of conflict processing could also take advantage of computational modeling techniques. Relatively few studies have employed computational models of dual-process reasoning (one example: Caplin & Martin, 2016), perhaps because of the challenges inherent in implementing these models (see Evans, 2005). Nevertheless, computational models that include parameters describing various stages of conflict processing could help researchers ascertain the stage(s) of conflict processing at which causally primary impairments in pathways leading from dual-process reasoning deficits to delusions are most likely to reside.

Progress toward discerning how deficits at different stages of conflict processing might contribute to delusions could also be made by examining cognitive decoupling, the process of inhibiting a response in favor of a less intuitive or completely novel alternative (see Pennycook, Fugelsang, & Koehler, 2015), in delusional individuals with schizophrenia. Given that individuals with schizophrenia generally exhibit structural and functional perturbations of brain regions that have inhibitory roles in reasoning (Potkin et al., 2008; Wang et al., 2014), such as the dorsolateral and right lateral prefrontal cortex (Greene, Nystrom, Engell, Darley, & Cohen, 2004; Stollstorff et al., 2012), decoupling seems likely to

be inefficient in these individuals. Notably, inefficient decoupling (rather than or in addition to deficits in conflict processing) could explain the exaggerated effect of conflict on syllogism evaluation performance in delusional individuals with schizophrenia observed by Speechley et al. (2012), Speechley et al., 2013). In simple syllogism evaluation tasks, intuitions regarding syllogism validity may be generated according to both preexisting beliefs and deductive logic (De Neys, 2012). When these intuitions suggest conflicting responses, inefficient decoupling may cause delusional individuals to give incorrect evaluations of syllogism validity (i.e., those driven by pre-existing beliefs) more often than healthy controls, leaving these individuals more susceptible to belief bias.

The contributions of inefficient decoupling and impaired conflict processing to delusion-related reasoning biases could be examined simultaneously using Pennycook and colleagues' (2015) rapid-response base-rate task. This task is modeled off of the famous "lawyer-engineer problem" (Kahneman & Tversky, 1973). The task consists of many problems in which a fictional individual is ostensibly drawn at random from a sample containing two groups (e.g., farmers and doctors). Each problem reports the composition of the sample (the base-rate probability; e.g., 995 farmers and 5 doctors) as well as a stereotypical description of the fictional individual (e.g., Person A is intelligent). On the basis of these two sources of information, a decision must be rapidly made regarding the fictional individual's group membership. Half of the problems are "conflict problems," in which these sources of information each suggest that the fictional individual belongs to a different group. Half are "non-conflict problems," in which both information sources suggest that the fictional individual belongs to the same group.

Reaction times for correct responses to non-conflict problems can be used as a baseline when calculating two indices of analytic reasoning engagement. An index of analytic reasoning engagement in the presence of conflict can be derived by comparing this baseline to reaction times for stereotypical responses to conflict problems. Comparing this baseline to reaction times for base-rate responses to conflict problems yields an index of decoupling efficiency. These indices could be used to clarify whether delusional individuals with schizophrenia (vs. healthy controls) exhibit an attenuated effect of conflict on analytic reasoning engagement, inefficient decoupling, or both. If, as expected, both impairments are present in delusional individuals with schizophrenia, multiple regression models including the aforementioned indices could be used to determine whether either impairment is uniquely associated with delusion-related cognitive biases (e.g., BADE).

These lines of future research could be complemented by attempts to more definitively determine whether conflict processing impairments are causally primary in encouraging and maintaining delusions. Significant progress in this area could be made by examining whether impaired conflict processing encourages liberal acceptance, which can potentially account for many constructs hypothesized to be downstream of impaired conflict processing on the path to delusions (e.g., BADE, jumping to conclusions, reduced analytic reasoning, and delusions themselves [see Moritz et al., 2017]). Prospective, longitudinal studies might examine whether these constructs are causally related by determining whether changes in conflict processing precede and predict changes in liberal acceptance (as would be expected if conflict processing impairments cause liberal acceptance). Because conflict processing

may become progressively impaired during the transition to psychosis (see Cannon et al., 2015), these studies might do well to recruit clinical high-risk individuals with a follow-up period that allows sufficient time for potential conversion to psychosis.

The nature of the putative association between conflict processing impairments and liberal acceptance could also be evaluated by building upon evidence that a “hyperdirect pathway” linking the prefrontal cortex to the subthalamic nucleus modulates decision thresholds (Cavanagh & Frank, 2014). Via this pathway, mPFC theta activity might increase decision thresholds in the presence of high amounts of response conflict by modulating subthalamic nucleus activity (Cavanagh et al., 2011; Cavanagh & Frank, 2014). Accordingly, future research could examine whether conflict-related mPFC theta activity less strongly elevates decision thresholds in delusional individuals with schizophrenia (vs. healthy and psychiatric controls), perhaps because of dysconnectivity within the hyperdirect pathway.

After probing the relationship between conflict processing impairments and liberal acceptance, future research could clarify the causal primacy of conflict processing impairments by examining whether experimental manipulations that either increase (e.g., the therapies described by Waller et al., 2015; Ward & Garety, 2017) or decrease (e.g. acute stress, time pressure, and cognitive load) analytic reasoning engagement effect behavior impacted by conflict processing (e.g., De Neys et al., 2011). Positive results from such studies could indicate that impaired conflict processing is not causally primary to reduced analytic reasoning. In contrast, null results (with adequate statistical power) would strengthen the case that conflict processing impairments are a causally primary deficit leading to delusions.

Beyond these suggestions, future research could examine the following specific predictions:

- 1) Compared to controls, individuals with damage to the dorsal anterior cingulate (dACC) will exhibit reduced engagement in analytic reasoning, more BADE, and a greater tendency to jump to conclusions.
- 2) There should be a negative correlation between delusions and both behavioral and neuroimaging-related measures of dACC-mediated conflict detection ability. This may be particularly true for persecutory delusions (extant literature most strongly links these delusions to reductions in analytic reasoning).
- 3) There are significant individual differences in the cognitive biases reviewed in this manuscript. In more highly biased individuals, delusions may result from neuropsychological insults akin to those conceptualized as forming “Factor 1” in two-factor theories of delusional belief (e.g., Coltheart, 2010). That is, no second insult would be necessary in these individuals.

9. Concluding remarks

The present manuscript suggests that conflict processing deficits have the potential to be causally primary in putative pathways leading to reasoning biases and delusions. By subjecting these putative pathways to further scrutiny in future research, greater conceptual clarity regarding the cognitive operations underlying delusions and their associated

reasoning biases may be achieved. To echo previous authors (Ward & Garety, 2017), it is hoped that this greater conceptual clarity may facilitate theory refinement and the development of more effective treatments for psychosis.

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References

- Altemeyer B. (2002). Dogmatic behavior among students: Testing a new measure of dogmatism. *The Journal of Social Psychology*, 142(6), 713–721. [PubMed: 12450346]
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.
- Arnsten AF (2015). Stress weakens prefrontal networks: Molecular insults to higher cognition. *Nature Neuroscience*, 18(10), 1376–1385. [PubMed: 26404712]
- Baars BJ (2005). Global workspace theory of consciousness: Toward a cognitive neuroscience of human experience. *Progress in Brain Research*, 150, 45–53. [PubMed: 16186014]
- Baars BJ, & Franklin S. (2003). How conscious experience and working memory interact. *Trends in Cognitive Sciences*, 7(4), 166–172. [PubMed: 12691765]

- Bago B, & De Neys W. (2017). Fast logic?: Examining the time course assumption of dual process theory. *Cognition*, 158, 90–109. [PubMed: 27816844]
- Bago B, Frey D, Vidal J, Houdé O, Borst G, & De Neys W. (2018). Fast and slow thinking: Electrophysiological evidence for early conflict sensitivity. *Neuropsychologia*, 117, 483–490. [PubMed: 30025789]
- Balzan R, Delfabbro P, Galletly C, & Woodward T. (2013). Confirmation biases across the psychosis continuum: The contribution of hypersalient evidence-hypothesis matches. *British Journal of Clinical Psychology*, 52(1), 53–69. [PubMed: 23398112]
- Bronstein MV, & Cannon TD (2017). Bias against disconfirmatory evidence in a large nonclinical sample: Associations with schizotypy and delusional beliefs. *Journal of Experimental Psychopathology*, 8(3), 288–302.
- Bronstein MV, & Cannon TD (2018). Measuring bias against disconfirmatory evidence: An evaluation of BADE task scoring methods and the case for a novel method. *Psychiatry Research*, 261, 535–540. [PubMed: 29407719]
- Bronstein MV, Dovidio JF, & Cannon TD (2017). Both bias against disconfirmatory evidence and political orientation partially explain the relationship between dogmatism and racial prejudice. *Personality and Individual Differences*, 105, 89–94.
- Bronstein MV, Everaert J, Castro A, Joormann J, & Cannon TD (2019). Pathways to paranoia: Analytic thinking and belief flexibility. *Behaviour Research and Therapy*, 113, 18–24. [PubMed: 30580159]
- Bronstein MV, Pennycook G, Bear A, Rand DG, & Cannon TD (2019). Belief in fake news is associated with delusionality, dogmatism, religious fundamentalism, and reduced analytic thinking. *Journal of Applied Research in Memory and Cognition*, 8(1), 108–117.
- Broome MR, Johns LC, Valli I, Woolley JB, Tabraham P, Brett C, ... McGuire PK (2007). Delusion formation and reasoning biases in those at clinical high risk for psychosis. *The British Journal of Psychiatry*, 191(51), s38–s42.
- Brown AS, Vinogradov S, Kremen WS, Poole JH, Deicken RF, Penner JD, ... Schaefer CA (2009). Prenatal exposure to maternal infection and executive dysfunction in adult schizophrenia. *American Journal of Psychiatry*, 166(6), 683–690. [PubMed: 19369317]
- Broyd A, Balzan RP, Woodward TS, & Allen P. (2017). Dopamine, cognitive biases and assessment of certainty: A neurocognitive model of delusions. *Clinical Psychology Review*, 54, 96–106. [PubMed: 28448827]
- Buchy L, Woodward TS, & Liotti M. (2007). A cognitive bias against disconfirmatory evidence (BADE) is associated with schizotypy. *Schizophrenia Research*, 90(1), 334–337. [PubMed: 17215108]
- Buonocore M, Bosia M, Riccaboni R, Bechi M, Spangaro M, Piantanida M, ... Cavallaro R. (2015). Combined neurocognitive and metacognitive rehabilitation in schizophrenia: Effects on bias against disconfirmatory evidence. *European Psychiatry*, 30(5), 615–621. [PubMed: 25758155]
- Cannon TD, Chung Y, He G, Sun D, Jacobson A, Van Erp TG, ... Cornblatt B. (2015). Progressive reduction in cortical thickness as psychosis develops: A multisite longitudinal neuroimaging study of youth at elevated clinical risk. *Biological Psychiatry*, 77(2), 147–157. [PubMed: 25034946]
- Caplin A, & Martin D. (2016). The dual-process drift diffusion model: Evidence from response times. *Economic Inquiry*, 54(2), 1274–1282.
- Carter CS, & Van Veen V. (2007). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cognitive, Affective, & Behavioral Neuroscience*, 7(4), 367–379.
- Cavanagh JF, & Frank MJ (2014). Frontal theta as a mechanism for cognitive control. *Trends in Cognitive Sciences*, 18(8), 414–421. [PubMed: 24835663]
- Cavanagh JF, Wiecki TV, Cohen MX, Figueroa CM, Samanta J, Sherman SJ, & Frank MJ (2011). Subthalamic nucleus stimulation reverses mediofrontal influence over decision threshold. *Nature Neuroscience*, 14(11), 1462–1467. [PubMed: 21946325]
- Colbert SM, Peters ER, & Garety PA (2010). Delusions and belief flexibility in psychosis. *Psychology and Psychotherapy: Theory, Research and Practice*, 83(1), 45–57.

- Colibazzi T, Horga G, Wang Z, Huo Y, Corcoran C, Klahr K, ... Peterson BS (2016). Neural dysfunction in cognitive control circuits in persons at clinical high-risk for psychosis. *Neuropsychopharmacology*, 41(5), 1241–1250. [PubMed: 26354046]
- Coltheart M. (2010). The neuropsychology of delusions. *Annals of the New York Academy of Sciences*, 1191(1), 16–26. [PubMed: 20392273]
- De Neys W. (2012). Bias and conflict: A case for logical intuitions. *Perspectives on Psychological Science*, 7(1), 28–38. [PubMed: 26168420]
- De Neys W, Cromheeke S, & Osman M. (2011). Biased but in doubt: Conflict and decision confidence. *PLoS One*, 6(1), e15954.
- De Neys W, & Glumicic T. (2008). Conflict monitoring in dual process theories of thinking. *Cognition*, 106(3), 1248–1299. [PubMed: 17631876]
- Dehaene S, Artiges E, Naccache L, Martelli C, Viard A, Schürhoff F, ... Martinot J. (2003). Conscious and subliminal conflicts in normal subjects and patients with schizophrenia: The role of the anterior cingulate. *Proceedings of the National Academy of Sciences*, 100(23), 13722–13727.
- Dudley R, Taylor P, Wickham S, & Hutton P. (2016). Psychosis, delusions and the “jumping to conclusions” reasoning bias: A systematic review and meta-analysis. *Schizophrenia Bulletin*, 42(3), 652–665. [PubMed: 26519952]
- Eifler S, Rausch F, Schirmbeck F, Veckenstedt R, Englisch S, Meyer-Lindenberg A, ... Zink M. (2014). Neurocognitive capabilities modulate the integration of evidence in schizophrenia. *Psychiatry Research*, 219(1), 72–78. [PubMed: 24880580]
- Eisenacher S, Rausch F, Mier D, Fenske S, Veckenstedt R, Englisch S, ... Kirsch P. (2016). Bias against disconfirmatory evidence in the ‘at-risk mental state’ and during psychosis. *Psychiatry Research*, 238, 242–250. [PubMed: 27086240]
- Eisenacher S, & Zink M. (2017). Holding on to false beliefs: The bias against disconfirmatory evidence over the course of psychosis. *Journal of Behavior Therapy and Experimental Psychiatry*, 56, 79–89. [PubMed: 27608522]
- Epstein S. (1994). Integration of the cognitive and the psychodynamic unconscious. *American Psychologist*, 49(8), 709. [PubMed: 8092614]
- van Erp TG, Saleh PA, Rosso IM, Huttunen M, Lönnqvist J, Pirkola T, ... Cannon TD (2002). Contributions of genetic risk and fetal hypoxia to hippocampal volume in patients with schizophrenia or schizoaffective disorder, their unaffected siblings, and healthy unrelated volunteers. *American Journal of Psychiatry*, 159(9), 1514–1520. [PubMed: 12202271]
- Evans JSB (2003). In two minds: Dual-process accounts of reasoning. *Trends in Cognitive Sciences*, 7(10), 454–459. [PubMed: 14550493]
- Evans JSB (2005). Modelling thinking and reasoning: The challenge ahead. *Modeling Language, Cognition and Action* (pp. 411–419). .
- Evans JSB (2007). On the resolution of conflict in dual-process theories of reasoning. *Thinking & Reasoning*, 13(4), 321–339.
- Evans SL, Averbeck BB, & Furl N. (2015). Jumping to conclusions in schizophrenia. *Neuropsychiatric Disease and Treatment*, 11, 1615. [PubMed: 26170674]
- Evans JSB, & Curtis-Holmes J. (2005). Rapid responding increases belief bias: Evidence for the dual-process theory of reasoning. *Thinking & Reasoning*, 11(4), 382–389.
- Evans JSB, & Stanovich KE (2013). Dual-process theories of higher cognition: Advancing the debate. *Perspectives on Psychological Science*, 8(3), 223–241. [PubMed: 26172965]
- Everaert J, Bronstein MV, Cannon TD, & Joormann J. (2018). Looking through tinted glasses: Depression and social anxiety are related to both interpretation biases and inflexible negative interpretations. *Clinical Psychological Science*, 6(4), 517–528 (2167702617747968).
- Flynn SW, Lang DJ, Mackay AL, Goghari V, Vavasour IM, Whittall KP, ... Falkai P. (2003). Abnormalities of myelination in schizophrenia detected in vivo with MRI, and post-mortem with analysis of oligodendrocyte proteins. *Molecular Psychiatry*, 8(9), 811. [PubMed: 12931208]
- Fortgang R, & Srihari V. (2017). Cognitive disengagement and task switching in patients with schizophrenia. *Schizophrenia Bulletin*, 43(Suppl. 1), S23.

- Freedman D, Bao Y, Kremen WS, Vinogradov S, McKeague IW, & Brown AS (2012). Birth weight and neurocognition in schizophrenia spectrum disorders. *Schizophrenia Bulletin*, 39(3), 592–600. [PubMed: 22378899]
- Freeman D, Lister R, & Evans N. (2014). The use of intuitive and analytic reasoning styles by patients with persecutory delusions. *Journal of Behavior Therapy and Experimental Psychiatry*, 45(4), 454–458. [PubMed: 25000504]
- Garety P, Joyce E, Jolley S, Emsley R, Waller H, Kuipers E, ... Freeman D. (2013). Neuropsychological functioning and jumping to conclusions in delusions. *Schizophrenia Research*, 150(2), 570–574. [PubMed: 24075604]
- Gärtner M, Rohde-Liebenau L, Grimm S, & Bajbouj M. (2014). Working memory-related frontal theta activity is decreased under acute stress. *Psychoneuroendocrinology*, 43, 105–113. [PubMed: 24703176]
- Goel V, & Dolan RJ (2003). Explaining modulation of reasoning by belief. *Cognition*, 87(1), B11–B22. [PubMed: 12499108]
- Greene J, Nystrom LE, Engell AD, Darley JM, & Cohen JD (2004). The neural basis of cognitive conflict and control in moral judgment. *Neuron*, 44, 389–400. [PubMed: 15473975]
- Harrow M, MacDonald AW III, Sands JR, & Silverstein ML (1995). Vulnerability to delusions over time in schizophrenia and affective disorders. *Schizophrenia Bulletin*, 21(1), 95–109. [PubMed: 7770745]
- James W. (1890). *The principles of psychology*. Vol. 1 1950.
- Kahneman D, & Tversky A. (1973). On the psychology of prediction. *Psychological Review*, 80(4), 237.
- Kaplan JT, Gimbel SI, & Harris S. (2016). Neural correlates of maintaining one's political beliefs in the face of counterevidence. *Scientific Reports*, 6, 39589.
- Kerns JG, Cohen JD, MacDonald AW III, Johnson MK, Stenger VA, Aizenstein H, & Carter CS (2005). Decreased conflict-and error-related activity in the anterior cingulate cortex in subjects with schizophrenia. *American Journal of Psychiatry*, 162(10), 1833–1839. [PubMed: 16199829]
- Klauer KC, Musch J, & Naumer B. (2000). On belief bias in syllogistic reasoning. *Psychological Review*, 107(4), 852–884. [PubMed: 11089409]
- Langdon R, Still M, Connors MH, Ward PB, & Catts SV (2014). Jumping to delusions in early psychosis. *Cognitive Neuropsychiatry*, 19(3), 241–256. [PubMed: 24215351]
- Lee J, & Park S. (2005). Working memory impairments in schizophrenia: A meta-analysis. *Journal of Abnormal Psychology*, 114(4), 599. [PubMed: 16351383]
- Margittai Z, Nave G, Strombach T, van Wingerden M, Schwabe L, & Kalenscher T. (2016). Exogenous cortisol causes a shift from deliberative to intuitive thinking. *Psychoneuroendocrinology*, 64, 131–135. [PubMed: 26658173]
- McLean BF, Mattiske JK, & Balzan RP (2017). Association of the jumping to conclusions and evidence integration biases with delusions in psychosis: A detailed meta-analysis. *Schizophrenia Bulletin*, 43(2), 344–354. [PubMed: 27169465]
- McNeely HE, West R, Christensen BK, & Alain C. (2003). Neurophysiological evidence for disturbances of conflict processing in patients with schizophrenia. *Journal of Abnormal Psychology*, 112(4), 679–688. [PubMed: 14674879]
- Mei L, & Xiong WC (2008). Neuregulin 1 in neural development, synaptic plasticity and schizophrenia. *Nature reviews. Neuroscience*, 9(6), 437. [PubMed: 18478032]
- Meyer U, Knuesel I, Nyffeler M, & Feldon J. (2010). Chronic clozapine treatment improves prenatal infection-induced working memory deficits without influencing adult hippocampal neurogenesis. *Psychopharmacology*, 208(4), 531–543. [PubMed: 20041229]
- Minzenberg MJ, Laird AR, Thelen S, Carter CS, & Glahn DC (2009). Meta-analysis of 41 functional neuroimaging studies of executive function in schizophrenia. *Archives of General Psychiatry*, 66(8), 811–822. [PubMed: 19652121]
- Moritz S, Burnette P, Sperber S, Köther U, Hagemann-Goebel M, Hartmann M, & Lincoln TM (2010). Elucidating the black box from stress to paranoia. *Schizophrenia Bulletin*, 37(6), 1311–1317. [PubMed: 20513651]

- Moritz S, Pfuhl G, Lüdtke T, Menon M, Balzan RP, & Andreou C. (2017). A two-stage cognitive theory of the positive symptoms of psychosis. Highlighting the role of lowered decision thresholds. *Journal of Behavior Therapy and Experimental Psychiatry*, 56, 1220.
- Moritz S, Veckenstedt R, Randjbar S, Hottenrott B, Woodward TS, Eckstaedt FV, ... Lincoln TM (2009). Decision making under uncertainty and mood induction: Further evidence for liberal acceptance in schizophrenia. *Psychological Medicine*, 39(11), 1821–1829. [PubMed: 19426569]
- Moritz S, & Woodward TS (2004). Plausibility judgment in schizophrenic patients: Evidence for a liberal acceptance bias. *German Journal of Psychiatry*, 7(4), 66–74.
- Moritz S, & Woodward TS (2005). Jumping to conclusions in delusional and non-delusional schizophrenic patients. *British Journal of Clinical Psychology*, 44(2), 193–207. [PubMed: 16004654]
- Moritz S, Woodward TS, & Hausmann D. (2005). Incautious reasoning as a pathogenetic factor for the development of psychotic symptoms in schizophrenia. *Schizophrenia Bulletin*, 32(2), 327–331. [PubMed: 16339971]
- Moritz S, Woodward TS, Jelinek L, & Klinge R. (2008). Memory and metamemory in schizophrenia: A liberal acceptance account of psychosis. *Psychological Medicine*, 38(6), 825–832. [PubMed: 18205963]
- Moritz S, Woodward TS, & Lambert M. (2007). Under what circumstances do patients with schizophrenia jump to conclusions? A liberal acceptance account. *The British Journal of Clinical Psychology*, 46(2), 127–137. [PubMed: 17524208]
- Otto AR, Raio CM, Chiang A, Phelps EA, & Daw ND (2013). Working-memory capacity protects model-based learning from stress. *Proceedings of the National Academy of Sciences*, 110(52), 20941–20946.
- Pennycook G. (2017). A perspective on the theoretical foundation of dual-process models In W.I. M. De Neys. *Dual-process theory 2.0* (pp. 13–35). [Routledge].
- Pennycook G, Cheyne JA, Barr N, Koehler DJ, & Fugelsang JA (2014). Cognitive style and religiosity: The role of conflict detection. *Memory & Cognition*, 42(1), 1–10. [PubMed: 23784742]
- Pennycook G, Cheyne JA, Barr N, Koehler DJ, & Fugelsang JA (2015). On the reception and detection of pseudo-profound bullshit. *Judgment and Decision making*, 10, 549–563. 10.3389/fpsyg.2013.00279.
- Pennycook G, Fugelsang JA, & Koehler DJ (2015). What makes us think? A three-stage dual-process model of analytic engagement. *Cognitive Psychology*, 80, 34–72. [PubMed: 26091582]
- Pennycook G, & Rand DG (2018). Lazy, not biased: Susceptibility to partisan fake news is better explained by lack of reasoning than by motivated reasoning. *Cognition*. 10.1016/j.cognition.2018.06.011.
- Pennycook G, & Rand DG (2019). Who falls for fake news? The roles of bullshit receptivity, overclaiming, familiarity, and analytic thinking. *Journal of Personality*. 10.1111/jopy.12476.
- Peters ER, Thornton P, Siksou L, Linney Y, & MacCabe JH (2008). Specificity of the jump-to-conclusions bias in deluded patients. *British Journal of Clinical Psychology*, 47(2), 239–244. [PubMed: 17988432]
- Potkin SG, Turner JA, Brown GG, McCarthy G, Greve DN, Glover GH, ... Ford JM (2008). Working memory and DLPFC inefficiency in schizophrenia: The FBIRN study. *Schizophrenia Bulletin*, 35(1), 19–31. [PubMed: 19042912]
- Qin S, Hermans EJ, van Marle HJ, Luo J, & Fernández G. (2009). Acute psychological stress reduces working memory-related activity in the dorsolateral prefrontal cortex. *Biological Psychiatry*, 66(1), 25–32. [PubMed: 19403118]
- Rausch F, Eisenacher S, Elkin H, Englisch S, Kayser S, Striepens N, ... Gaebel W. (2016). Evaluation of the ‘jumping to conclusions’ bias in different subgroups of the at-risk mental state: From cognitive basic symptoms to UHR criteria. *Psychological Medicine*, 46(10), 2071–2081. [PubMed: 27094404]
- Rausch F, Mier D, Eifler S, Fenske S, Schirmbeck F, Englisch S, ... Zink M. (2015). Reduced activation in the ventral striatum during probabilistic decision-making in patients in an at-risk mental state. *Journal of Psychiatry & Neuroscience*, 40(3), 163. [PubMed: 25622039]

- Riccaboni R, Fresi F, Bosia M, Buonocore M, Leiba N, Smeraldi E, & Cavallaro R. (2012). Patterns of evidence integration in schizophrenia and delusion. *Psychiatry Research*, 200(2–3), 108–114. [PubMed: 22578403]
- Robinson ADT (1988). A century of delusions in south West Scotland. *The British Journal of Psychiatry*, 153(2), 163–167. [PubMed: 3076491]
- Ross RM, Pennycook G, McKay R, Gervais WM, Langdon R, & Coltheart M. (2016). Analytic cognitive style, not delusional ideation, predicts data gathering in a large beads task study. *Cognitive Neuropsychiatry*, 21(4), 300–314. [PubMed: 27341507]
- Sanford N, Veckenstedt R, Moritz S, Balzan RP, & Woodward TS (2014). Impaired integration of disambiguating evidence in delusional schizophrenia patients. *Psychological Medicine*, 44(13), 2729–2738. [PubMed: 25065271]
- Schmiedt C, Brand A, Hildebrandt H, & Basar-Eroglu C. (2005). Event-related theta oscillations during working memory tasks in patients with schizophrenia and healthy controls. *Cognitive Brain Research*, 25(3), 936–947. [PubMed: 16289526]
- Seidman LJ, Faraone SV, Goldstein JM, Kremen WS, Horton NJ, Makris N, ... Tsuang MT (2002). Left hippocampal volume as a vulnerability indicator for schizophrenia: A magnetic resonance imaging morphometric study of nonpsychotic first-degree relatives. *Archives of General Psychiatry*, 59(9), 839–849. [PubMed: 12215084]
- Sitskoorn MM, Aleman A, Ebisch SJ, Appels MC, & Kahn RS (2004). Cognitive deficits in relatives of patients with schizophrenia: a meta-analysis. *Schizophrenia research*, 71(2), 285–295. [PubMed: 15474899]
- Speechley WJ (2012). Dual-stream modulation failure, cognitive biases and delusions in schizophrenia. University of British Columbia: Doctoral dissertation.
- Speechley WJ, Murray CB, McKay RM, Munz MT, & Ngan ETC (2010). A failure of conflict to modulate dual-stream processing may underlie the formation and maintenance of delusions. *European Psychiatry*, 25(2), 80–86. [PubMed: 19699616]
- Speechley WJ, & Ngan ET (2008). Dual-stream modulation failure: A novel hypothesis for the formation and maintenance of delusions in schizophrenia. *Medical Hypotheses*, 70(6), 1210–1214. [PubMed: 18328633]
- Speechley WJ, Ngan ETC, Moritz S, & Woodward TS (2012). Impaired evidence integration and delusions in schizophrenia. *Journal of Experimental Psychopathology*, 3, 688–701.
- Speechley WJ, Whitman JC, & Woodward TS (2010). The contribution of hypersalience to the “jumping to conclusions” bias associated with delusions in schizophrenia. *Journal of Psychiatry & Neuroscience: JPN*, 35(1), 7. [PubMed: 20040242]
- Speechley WJ, Woodward TS, & Ngan ET (2013). Failure of conflict to modulate central executive network activity associated with delusions in schizophrenia. *Frontiers in Psychiatry*, 4, 113. [PubMed: 24069005]
- Stephan KE, Friston KJ, & Frith CD (2009). Dysconnection in schizophrenia: From abnormal synaptic plasticity to failures of self-monitoring. *Schizophrenia Bulletin*, 35(3), 509–527. [PubMed: 19155345]
- Stollstorff M, Vartanian O, & Goel V. (2012). Levels of conflict in reasoning modulate right lateral prefrontal cortex. *Brain Research*, 1428, 24–32. [PubMed: 21684531]
- Swami V, Voracek M, Stieger S, Tran US, & Furnham A. (2014). Analytic thinking reduces belief in conspiracy theories. *Cognition*, 133(3), 572–585. [PubMed: 25217762]
- Thompson VA (2009). *Dual-process theories: A metacognitive perspective Two minds: Dual processes and beyond..* Oxford: Oxford University Press.
- Thompson VA, Turner JAP, & Pennycook G. (2011). Intuition, reason, and metacognition. *Cognitive Psychology*, 63(3), 107–140. [PubMed: 21798215]
- Van Dael F, Versmissen D, Janssen I, Myin-Germeys I, Van Os J, & Krabbendam L. (2005). Data gathering: Biased in psychosis? *Schizophrenia Bulletin*, 32(2), 341–351. [PubMed: 16254066]
- Vartanian O, Beatty EL, Smith I, Blackler K, Lam Q, Forbes S, & De Neys W. (2018). The reflective mind: Examining individual differences in susceptibility to base rate neglect with fmri. *Journal of Cognitive Neuroscience*, 30(7), 1011–1022. [PubMed: 29668391]

- Veckenstedt R, Randjbar S, Vitzthum F, Hottenrott B, Woodward TS, & Moritz S. (2011). Incurrigibility, jumping to conclusions, and decision threshold in schizophrenia. *Cognitive Neuropsychiatry*, 16(2), 174–192. [PubMed: 21271413]
- Waller H, Emsley R, Freeman D, Bebbington P, Dunn G, Fowler D, ... Garety P. (2015). Thinking well: A randomized controlled feasibility study of a new CBT therapy targeting reasoning biases in people with distressing persecutory delusional beliefs. *Journal of Behavior Therapy and Experimental Psychiatry*, 48, 82–89. [PubMed: 25770671]
- Wang X, Xia M, Lai Y, Dai Z, Cao Q, Cheng Z, ... Li K. (2014). Disrupted restingstate functional connectivity in minimally treated chronic schizophrenia. *Schizophrenia Research*, 156(2), 150–156. [PubMed: 24794395]
- Ward T, & Garety PA (2017). Fast and slow thinking in distressing delusions: A review of the literature and implications for targeted therapy. *Schizophrenia Research*, 203, 80–87. [PubMed: 28927863]
- Ward T, Peters E, Jackson M, Day F, & Garety PA (2017). Data-gathering, belief flexibility, and reasoning across the psychosis continuum. *Schizophrenia Bulletin*, 44(1), 126–136.
- Wason PC, & Evans JSB (1975). Dual-processes in reasoning? *Cognition*, 3(2), 141–154.
- Winton-Brown TT, Broome MR, Allen P, Valli I, Howes O, Garety PA, ... McGuire P. (2015). Misattributing speech and jumping to conclusions: a longitudinal study in people at high risk of psychosis. *European Psychiatry*, 30(1), 32–37. [PubMed: 25511317]
- Woodward TS, Buchy L, Moritz S, & Liotti M. (2007). A bias against disconfirmatory evidence is associated with delusion proneness in a nonclinical sample. *Schizophrenia Bulletin*, 33(4), 1023–1028. [PubMed: 17347526]
- Woodward TS, Moritz S, Cuttler C, & Whitman JC (2006). The contribution of a cognitive bias against disconfirmatory evidence (BADE) to delusions in schizophrenia. *Journal of Clinical and Experimental Neuropsychology*, 28(4), 605–617. [PubMed: 16624787]
- Woodward TS, Munz M, LeClerc C, & Lecomte T. (2009). Change in delusions is associated with change in “jumping to conclusions”. *Psychiatry Research*, 170(2), 124–127. [PubMed: 19906443]
- Zheutlin AB, Chekroud AM, Polimanti R, Gelernter J, Sabb FW, Bilder RM, ... Cannon TD (2018). Multivariate pattern analysis of genotype–phenotype relationships in schizophrenia. *Schizophrenia Bulletin*, 44(5), 1045–1052. [PubMed: 29534239]
- Zheutlin AB, Viehman RW, Fortgang R, Borg J, Smith DJ, Suvisaari J, ... Cannon TD (2016). Cognitive endophenotypes inform genome-wide expression profiling in schizophrenia. *Neuropsychology*, 30(1), 40. [PubMed: 26710095]

HIGHLIGHTS

- Reasoning biases linked to delusion formation/maintenance appear to be common.
- Dual-process theories of reasoning may help uncover the source of these biases.
- These theories state that impaired conflict processing may limit analytic reasoning.
- Conflict processing problems may therefore promote cognitive biases and delusions.
- Evidence for this is reviewed and suggestions for future research are offered.

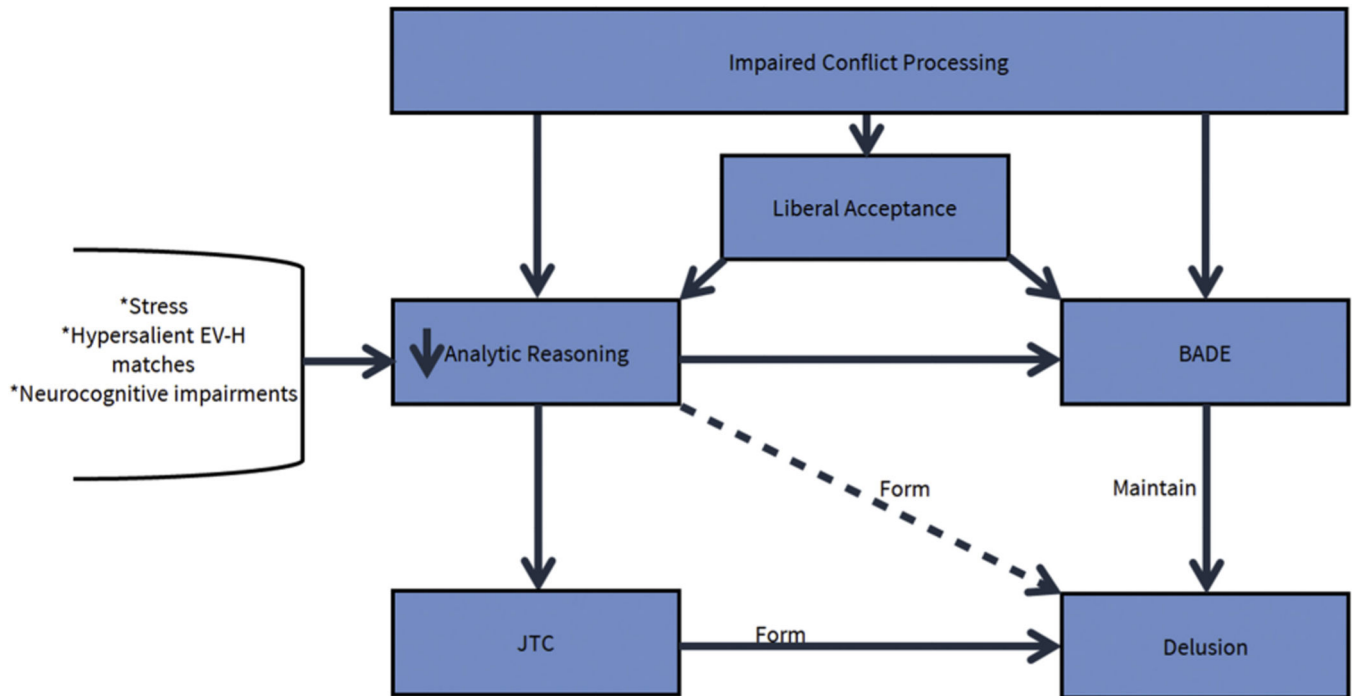


Fig. 1.

Potential pathways from impaired conflict processing to biased reasoning to the formation and maintenance of delusions. The dotted line from analytic reasoning to delusions is included because it is currently unclear whether JTC exerts a unique causal effect on delusions above and beyond the effects of factors such as hypersalient evidence-hypothesis matches, liberal acceptance, and reduced engagement in analytic reasoning. The dotted line represents the alternative pathway to consider if JTC does not exert a unique causal effect (in which case the pathway through JTC would be superfluous). In this figure, the term “neurocognitive impairments” refers specifically to deficits in the machinery underlying analytic reasoning (e.g., impairments in cognitive control and working memory).

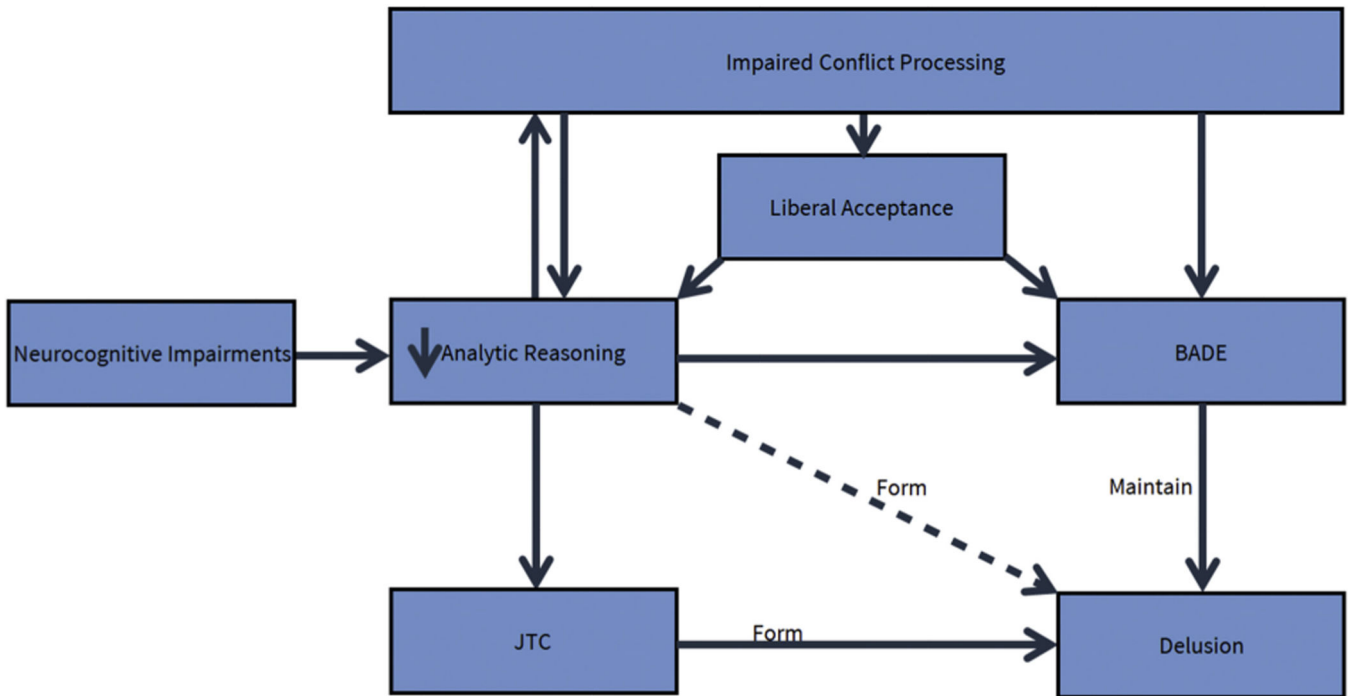


Fig. 2.

A possible competing account of delusion formation (to be compared with that depicted in Fig. 1). In this account, general neurocognitive deficits (i.e., ones that can be observed even in the absence of conflict), rather than conflict processing impairments, are causally primary in the pathways leading to delusions. In this figure, the term “neurocognitive impairments” refers specifically to deficits in the machinery underlying analytic reasoning (e.g., impairments in working memory).