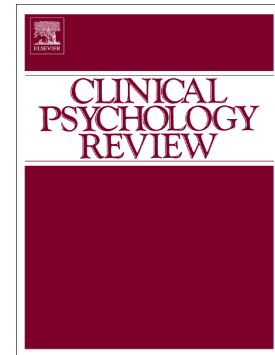# Journal Pre-proof

Aberrant memory and delusional ideation: A pernicious partnership?

William N. Koller, Tyrone D. Cannon

Aberrant memory and delusional ideation: A pernicious partnership?

William N. Koller[1] and Tyrone D. Cannon[1]

[1]Department of Psychology, Yale University

2 Hillhouse Avenue, New Haven, CT 06520-8205

Author Note: We have no conflict of interest to disclose. Correspondence concerning this article should be addressed to William N. Koller, Yale University, P.O. Box 208205, 2 Hillhouse Avenue, New Haven, CT 06520-8205. Email: william.koller@yale.edu

Declaration of Interest: none

Abstract

Delusions can be conceptualized as beliefs that are both at odds with consensus reality and espoused with high conviction. While delusions represent a cardinal symptom of schizophrenia, delusion-like beliefs can be found in the general population. Do similar cognitive mechanisms support delusionality across this spectrum? If so, what are they? Here, we examine evidence for a mechanistic role of the (associative) memory system in the formation and maintenance of delusions and delusion-like beliefs. While general neurocognitive metrics do *not* tend to associate with delusionality, our scoping review of the clinical and subclinical literature reveals several subdomains of memory function that do. These include a propensity to commit errors of commission (i.e., false alarms and intrusions), source memory biases, and metamemory impairment. We discuss how several of these effects may stem from aberrant associative memory function and offer recommendations for future research. Further, we propose a state/trait interaction model in which underlying *traits* (i.e., impaired associative and metamemory function) may become coupled with delusionality during *states* of acute psychosis, when

memory function is particularly challenged by aberrant salience attribution and noisy perceptual input. According to this model, delusions may arise as explanations to high-salience (but low-source) mnemonic content that is endorsed with high confidence.

Schizophrenia is a disorder characterized by symptoms ranging from "low-level" perceptual disturbances (Horga & Abi-Dargham, 2019; Uhlhaas & Mishara, 2007) to "higher-level" alterations of one's explicit, declarative beliefs (Butler & Braff, 1991; Kiran & Chaudhury, 2009). Delusions, which are often conceptualized as rigidly held and objectively untenable beliefs, represent one such "higher-level" symptom of schizophrenia that can cause significant distress and impairment (Freeman, 2016). However, our understanding of the cognitive mechanisms by which delusions are formed and maintained is incomplete. Here, we critically examine the human associative memory system as a potential "hub" in the formation of delusional beliefs. Given its involvement in the integration of basic perceptual input, affective information, and temporospatial context into subjectively meaningful cognitive structures (Behrendt, 2013), the human associative memory system may be uniquely situated to play an intermediary role in the translation of perceptual experience to higher-order "beliefs". Accordingly, systematic aberrancies in the function of the associative memory system may help to create the conditions under which patently false, but subjectively meaningful, beliefs may develop and be held with high conviction.

Notably, delusional ideation extends across a continuum, such that similarly themed but clinically subthreshold beliefs are not uncommon in the general population (Freeman et al., 2011). Memory biases similarly extend into the general population and covary with these delusion-like beliefs (Koller & Cannon, 2021, in revision; Koller et al., in prep; Ongchoco et al.,

in prep; Sahakyan & Kwapil, 2019). This provides a basis for reviewing the role of the associative memory system in the formation and maintenance of delusional ideation not only in the context of schizophrenia, but also across the full spectrum of delusion-like beliefs. As such, we organize the present review around the following guiding question: are aberrancies in the associative memory system *reliably and differentially associated with delusional* or *delusion-like* beliefs and if so, which aspect(s) in particular may play a mechanistic role?

Associative Memory, Belief, and Delusion

Before turning to this guiding question, we must first offer definitions of associative memory, belief, and delusion.

Associative Memory

The human associative memory system, broadly defined, encodes, monitors, and retrieves various components of experience (e.g., simple percepts, affective information, contextual details) according to spatial, temporal, or other relationships between these components (Eichenbaum, 2017; Mayes et al., 2007; Turk-Browne, 2019). Via this system, simple elements of experience become tied to one another, such that presentation of a single aspect of the resulting associative network can prompt auto-associative recall of its other elements (Treves & Rolls, 1994). Importantly, this allows us to *contextualize* ongoing experience in reference to stored representations – in other words, when encountering an object in our environment (e.g., a dog), we can retrieve relevant associations (e.g., past experiences with this particular dog, or with dogs in general) that help to orient current action (e.g., deciding whether or not to pet the dog). In this way, associative memory allows us to make sense of and respond to the vast array of information which arrives into consciousness on a daily basis.

Belief

In this way, associative memory may facilitate the extraction of subjective meaning from simple perceptual inputs. At the level of processing that occurs in early sensory cortices, percepts are themselves largely without declarative "meaning". For example, consider a single percept, such as a red circle. In isolation, this stimulus likely conveys very little meaning, perhaps related only to its redness and its geometric properties. Yet if this same percept has been incorporated into a broader associative network (thus becoming capable of triggering auto-associative recall of other elements of this network), it gains the capability of conveying a much richer meaning (e.g., activating threat systems, if this stimulus was previously paired with shock as in Pavlovian conditioning; Maren & Holt, 2000). In this way, our beliefs – which we roughly define here as subjectively meaningful assertions that are used to model or explain some objective state of the world (Connors & Halligan, 2014) – may rely on these associative networks that are maintained in memory. For example, following Pavlovian conditioning, one may learn that red circles are associated with and temporally precede unpleasant shocks, resulting in a new associative "belief" (Gawronski & Bodenhausen, 2011; Klein Flugge et al., 2019) that acts as a working model of the contingency "if red circle, then shock". One's more explicit, declarative belief about red circles may arise in part as an *explanatory response* to this simple association: in other words, if one becomes aware that red circles seem to coincide with feelings of fear, how does one make sense of this experience? This resulting belief could be immensely simple, such as "red circles precede a shock". Or it could take the form of a more abstract idea, such as "red circles are dangerous" (closer to what might be called a "propositional" belief; Buchanan, 2012). In either case, this belief may represent the end result of the process of drawing meaning from underlying associations – a process that allows these simple associations to be consciously reported, shared, or collaborated on. This model of belief formation could thus be described as "explanationist" in

nature, in that it suggests that higher-order beliefs grow out of explanatory responses to lower-order networks of associations that may help to orient action but are themselves without declarative meaning *per se*.

Delusion

This brings us to the definition of delusion – a construct which, like belief, is notoriously hard to define (Spitzer, 1990). Delusions are often defined as "fixed false beliefs" in the fields of clinical psychology and psychiatry, largely based on Karl Jaspers' suggestion that delusions are characterized by: 1) false or impossible content, 2) conviction and 3) resistance to counterevidence (Jaspers, 1913/1997). However, as a comprehensive definition of delusion, these three criteria have been found to be insufficient. Common critiques include the fact that the content of the delusion may not always be false, an individual's conviction in a delusion often fluctuates over time, and other beliefs that are widely accepted to be non-delusional (e.g., political or religious beliefs) can be held with similar certitude or otherwise be resistant to counterevidence. In other words, it is surprisingly difficult to define a "delusional belief" with high discriminant validity (Spitzer, 1990) – and more phenomenological accounts suggest that delusions are not "beliefs" at all, but may rather represent changes in core aspects of reality experience (Feyaerts et al., 2021). Also counter to Jasper's tripartite designation is a continuum model of delusions (Strauss, 1969), which ascribes to the position that symptoms of psychosis occupy an extreme end of a spectrum that extends into the general population (van Os et al., 2000; van Os & Reininghaus, 2016). This model is supported by epidemiological studies (van Os et al., 2009) and those concerning the construct of schizotypy (Lenzenweger, 2010), though it is not without its challenges (e.g., see Feyaerts et al., 2021). Importantly, a continuum model of

psychosis raises the possibility that delusions are formed and maintained by similar (or even identical) mechanisms as other forms of beliefs.

Here, we draw from this continuum framework to explore whether associative memory processes carry explanatory power for belief formation and maintenance across a spectrum of delusional and delusion-like beliefs. This framing invites the interpretation that, just as memory may provide a substrate for "normative" beliefs, aberrant memory may set the stage for "non-normative" beliefs. If our beliefs represent explanatory frameworks that draw meaning from associative networks (a model that coincides with "explanationist" accounts of delusions; e.g., Maher, 1999), what happens if these underlying networks are degraded or haphazardly connected? This may result in decontextualized internal models of the world that are not reflective of its external structure, inviting interpretations of meaning ("beliefs") that are both at odds with consensus reality (i.e., "impossible" in content) and held with high conviction/resistant to updating (e.g., due perhaps to their explanatory function).

Review of Theoretical Precedents

A number of extant models of delusional belief, both historical and contemporary, suggest that memory and memory-related brain structures play key roles in the formation and maintenance of delusions. Gray et al. (1991) and Hemsley (1993) posited that changes to the hippocampal formation and its memory-related functions feature heavily in the pathogenesis of delusions. Hemsley (1993) wrote that insult to the hippocampus may result in a "weakening of the influence of stored memories of regularities of previous input on current perception" (p. 635), which may in turn lead to the intrusion of redundant or irrelevant features of the environment into consciousness, thus setting the stage for delusional beliefs. Bogerts (1997) similarly pointed to aberrant function within temperolimbic regions (e.g., left hippocampus and parahippocampal

gyrus) as fueling delusional beliefs. Drawing from lesion studies and structural and functional imaging, Bogerts suggested that a breakdown in the integrative and associative functions conducted by these regions leads to the "distorted interpretations of the external reality" (p. 424) that are characteristic of delusions. Other theorists have since built on this general framework, pointing to a role of "decontextualized" memory in delusional beliefs (Boyer et al., 2007; Moskowitz et al., 2008). These authors suggested that the retrieval of affect-laden yet decontextualized memories may result in their conflation with the current context, producing a sense of unease and anxiety that contributes to the "delusional atmosphere" often associated with the psychosis prodrome (Mishara, 2010). Many of these theories share certain features with Gestalt conceptions of psychosis. For instance, Silverstein and Uhlhaas (2004) suggest that the positive symptoms of schizophrenia arise from a breakdown in the ability to process information as a unified *whole* rather than a collection of disconnected parts – a dysfunction that could manifest in various domains of cognition, including perception or memory.

More contemporary theories of delusions have formally connected hippocampally-mediated memory processes to the experience of *hypersalience* – in which innocuous features of one's environment appear unduly significant or otherwise demand one's attention (Kapur, 2003). Hypersalience has been associated with a hyperdopaminergic state (Winton-Brown et al., 2017). According to Lodge and Grace (2011), hyperdominergia in psychosis may stem from disinhibition of the ventral tegmental area (VTA) – a site of origin for dopaminergic pathways in the brain's mesocortical and mesolimbic systems – due to upstream hyperactivation of the ventral hippocampus. This hypothesis is supported by animal models of schizophrenia (Perez & Lodge, 2014), and the underlying hippocampal-VTA circuit has been put forward as a novel target for pharmaceutical intervention (Kätzel et al., 2020). Further, several contemporary

theories make specific predictions about the involvement of hippocampal subfields in the formation of positive symptoms such as delusions. Tamminga and colleagues have theorized that impaired dentate gyrus function and elevated cornu ammonis 3 activity in schizophrenia result in reduced *pattern separation* relative to *pattern completion* (Tamminga et al., 2012; Tamminga et al., 2010) – the competing hippocampal computations that support *encoding* and *retrieval*, respectively (O'Reilly & McClelland, 1994). These authors suggest that the imbalance of these mnemonic processes gives rise to the spurious associations characteristic of delusions. There is some support for this model (Das et al., 2014), though the data are not entirely conclusive with respect to the specific impairment of dentate gyrus function *per se* (Koller & Cannon, under review; Martinelli & Shergill, 2015). Finally, prediction error theories (Corlett et al., 2010; Fletcher & Frith, 2009) draw from Bayesian models of the brain to suggest that aberrant prediction error – or anomalous experience of mismatch between outcome and expectation – represents the core deficit that gives rise to delusions. While not strictly mnemonic in nature, these theories are germane to this review insofar as memory-related processes are involved in the definition of the "prior" against which incoming data is compared to generate an error signal, as well as in the process of belief updating and consolidation following prediction errors (e.g., see Corlett et al., 2013). In other words, prediction errors occur against a backdrop of expectation – the genesis, maintenance, and retrieval of which is likely facilitated in part by associative memory systems. In turn, prediction errors influence the formation and updating of memory (e.g., Bein et al., 2020; Rouhani et al., 2020; Sinclair et al., 2021).

In sum, past theories point to a breakdown in contextual binding and a weakened or otherwise aberrant influence of stored representations on current processing as key drivers of delusion formation. This disorganization of the associative memory system might interact with

predictive processing or salience attribution functions, contributing to inappropriate prediction error and/or the experience of hypersalience (e.g., as mediated by over-influence of the hippocampus on the VTA). Finally, the hippocampal subfields responsible for encoding new information and retrieving relevant representations may be imbalanced, contributing to the generation of spurious associations and a loss of effective referencing of incoming information. Together, these changes in the memory system may contribute to an internal model of the world that lacks associative coherence and, accordingly, experiences of the external world that are confusing, overwhelming, or otherwise fraught with unsolved meaning – conditions ripe for the formation of delusional beliefs.

Is Aberrant Memory Reliably and Differentially Associated with Delusional Ideation?

A review of memory-related models of delusions reveals robust theoretical precedents for considering a role of memory in the formation and maintenance of delusions and delusion-like beliefs. If disruptions in particular aspects of associative memory *do* contribute to delusions, there should be evidence of a robust correlation between them, and differentially so with respect to other symptoms. To address this possibility, we conducted a scoping review of studies that report direct associations (null or otherwise) between memory-related measures and delusion-related constructs (including "delusions", "positive symptoms", "positive schizotypy", "psychotic symptoms", "paranoia", "magical ideation", "unusual experiences"). Notably, given its focus on delusions and delusion-like beliefs, this review touches only cursorily on the larger body of work on memory in schizophrenia more broadly, given that patient samples used in case-control studies vary in their reporting of clinical state characteristics (i.e., presence of active delusional symptoms). This review was conducted between September 2021 and May 2022.

Associations with Positive, Negative, and Disorganized Symptoms

The symptoms of schizophrenia and related constructs such as schizotypy are often sorted into three clusters: disorganized, negative, and positive (Lenzenweger & Dworkin, 1996; Liddle, 1987). Disorganized symptoms (e.g., incoherent speech and thought disorder) are characterized by a loss of organization of language, thought, or behavior. Negative symptoms (e.g., amotivation and anhedonia) are characterized by a reduction or diminution of some premorbid function, while positive symptoms (e.g., delusions and hallucinations) represent an excess or distortion of some premorbid function.

When compared to healthy controls, individuals with schizophrenia consistently perform more poorly on a wide array of neurocognitive tests, with deficits in episodic memory counting among the most consistent findings (Reichenberg & Harvey, 2007; Schaefer et al., 2013).[1] Yet within schizophrenia spectrum disorders themselves, differential relationships also emerge between neurocognition and positive, negative and disorganized symptom dimensions (e.g., Basso et al., 1998; O'Leary et al., 2000). Historically, the association between neurocognition and *positive* symptoms, such as delusions, has received little attention. This is justified in part by several systematic reviews and meta-analyses which suggest that associations between general measures of neurocognition (including various visual, verbal, and working memory measures) and positive symptoms (including delusions) are small (Ventura et al., 2010) or null (Dibben et al., 2009; Dominguez et al., 2009). These analyses reveal that general measures of neurocognition are instead more closely associated with the negative and disorganized symptom clusters of schizophrenia. A similar pattern can be seen in a meta-analysis of the memory literature, in which general memory deficits appear to be more strongly associated with negative as opposed to positive symptoms (Aleman et al., 1999). The fact that watershed metrics of

---

[1]Although cf. recent work (Moritz et al., 2021) which suggests that this literature may underestimate the influence of secondary factors such as motivation.

memory performance that collapse across error types (e.g., *d'* or the "g" factor of neuropsychological tests) tend to track with negative and disorganized symptoms stands to reason. For instance, one common negative symptom is amotivation, which may confer widespread impairments in cognitive testing (Fortgang et al., 2020; Moritz et al., 2021). Further, generalized cognitive deficits represent both risk factors and possible consequences of conversion to psychosis (Seabury & Cannon, 2020; Sheffield et al., 2018) and accordingly may be most closely associated with its more chronic (i.e., negative and disorganized) symptoms as opposed to more state-like positive symptoms.

Taken at face value, this pattern of findings suggests that positive symptoms such as delusions are only very weakly related to memory function – a conclusion that poses a clear challenge to the notion that aberrant memory is reliably and differentially associated with delusional belief. However, a closer read of the underlying literature (as reviewed below) reveals a number of memory-related processes that *do* seem to track more closely with positive symptoms. This indicates a more heterogeneous relationship between memory and delusionality than meta-analyses involving general measures of memory performance might suggest. More specifically, this observation raises the possibility that the relationship between *specific* memory biases and delusionality may be obscured by the analysis of *general* trends in neurocognition and memory.

Memory Biases Associated with Delusionality

In contrast to watershed metrics of memory performance (e.g., *d'*), there are three domains of memory in particular that show promise as having a robust and differential association with delusionality. These include a tendency to make intrusion and false alarm errors,

source memory deficits, and metamemory impairment. We review these three domains in turn below.

Intrusions and False Alarms. A number of studies have reported impairments in working memory, recognition, and free recall performance in association with delusionality among patients with schizophrenia spectrum disorders (Bhatt et al., 2010; Brébion et al., 1999; Brébion, Smith, Amador, et al., 1997; Caligiuri et al., 2005; Ibanez-Casas et al., 2013; Schott et al., 2015; Schröder et al., 1996; Stip et al., 2007) and in the general population (Dagnall & Parker, 2009; Dehon et al., 2008; Evans et al., 2019; Koller & Cannon, 2021, in revision; Koller et al., in prep; Laws & Bhatt, 2005; Meyersburg et al., 2009; Ongchoco et al., in prep; Rodriguez-Ferreiro et al., 2020; Rossi et al., 2016; Sahakyan & Kwapil, 2019; Saunders et al., 2012; Sugimori & Tanno, 2010). Interestingly, many (but not all; e.g., Elvevag et al., 2004; Fisher et al., 2007; Heinrichs & Vaz, 2004; Moritz et al., 2006) of the studies in this literature report associations between positive symptoms and a specific genre of memory error – namely, *errors of commission*. In other words, these studies indicate that participants with more severe positive symptoms are more likely to falsely recognize or recall material that was either never presented during the study or was presented in a context that is irrelevant to their current task. In the language of Signal Detection Theory (SDT), this is referred to as a "false alarm", or the erroneous detection of "signal" in "noise" (Stanislaw & Todorov, 1999). In the domain of memory, "signal" can be understood as evidence that an item has been previously encountered in a relevant context (i.e., that an item is "old", or is a target). Conversely, "noise" represents evidence that an item does *not* correspond to previous relevant experience (i.e., that an item is "new", or is a lure). Thus, in a memory task, a false alarm – or the judgment of "old" for an item

that is in fact "new" – is characterized by the intrusion of task- or context-irrelevant content into the retrieval process.

In Schizophrenia Spectrum Disorders. A number of studies have demonstrated that positive symptom severity among patients with schizophrenia is associated with intrusion errors. Brébion, Smith, Amador, et al. (1997) found that higher scores on the positive subscale of the Positive and Negative Syndrome Scale for Schizophrenia (PANSS; Kay et al., 1987) corresponded to 1) a bias towards false alarms in recognition and 2) inter-list intrusions in free recall – i.e., when items from previously presented word lists are falsely remembered as having appeared in the currently tested list. Brébion et al. (1999) subsequently showed that scores on delusion-related items of the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984) positively correlated with extra-list intrusions – i.e., items that were *never* presented in the study and yet were falsely remembered as having appeared in the currently tested list – as well as inter-list intrusions in free recall. Stip et al. (2007) similarly found that scores on the Magical Ideation Scale (MIS; Eckblad & Chapman, 1983) were positively associated with extra-list errors in a patient sample. Further, Bhatt et al. (2010) demonstrated that actively delusional patients exhibited elevated rates of false alarms during recognition in a Deese-Roediger-McDermott paradigm (DRM; Deese, 1959; Roediger & McDermott, 1995). In a DRM paradigm, participants are presented with a list of highly semantically related words (e.g., BED, REST, AWAKE). Following a delay, they are asked to recognize or recall these words. Under these conditions, participants tend to be more likely to falsely recognize semantically related lures (e.g., SLEEP) or falsely recall semantically related words that were not presented during encoding (Gallo, 2010). Thus, a DRM false alarm represents the intrusion of a semantically related, but non-presented, item into the retrieval process. In a case-control fMRI study, Schott et al. (2015) also

found elevated false alarm rates on a recognition task among patients with paranoid schizophrenia who were actively experiencing positive symptoms (as indexed by SAPS scores). Moreover, while a strong positive correlation emerged between hippocampal BOLD activation and delayed recognition performance in the control group, the patient group showed *no* such association between novelty-related activation in the hippocampus and memory performance. This general pattern of findings has also been found among first-episode patients: in recent work from Seabury et al. (2021), positive symptoms were associated with high-confidence errors (including false alarms) in a relational recognition memory task involving word pairs and their corresponding images. Finally, there is evidence of a similar memory bias in delusional disorder, which is characterized by the presence of delusional beliefs in the absence of other psychotic symptoms such as hallucinations (American Psychiatric Association, 2013). In a case-control study, Ibanez-Casas et al. (2013) demonstrated that individuals with delusional disorder performed more poorly than healthy controls on a number of memory tasks while controlling for sex, age, educational level, and premorbid IQ. Notably, these deficits included elevated false alarms in recognition and intrusions in free recall (although note that other types of error were also seen in this study – i.e., delusional disorder was also associated with *decreased* recognition and recall of target stimuli).

Notably, in a number of these studies, elevated false alarms and intrusion errors were *differentially* associated with positive symptoms, over and above other symptom clusters. In the work of Brébion and colleagues, no parallel associations with false alarms or intrusion errors emerged for depressive (Brébion, Smith, Amador, et al., 1997) or negative symptoms (which were in fact negatively associated with intrusion errors; Brébion et al., 1999; Brébion, Smith, Amador, et al., 1997). Bhatt et al. (2010) showed a similar dissociation, in which the actively

delusional patient group made close to double the number of false alarms when compared to both the healthy control group as well as a non-delusional patient group. Seabury et al. (2021) also found a double dissociation in which positive symptoms, but *not* negative symptoms, were associated with high-confidence memory errors (including false alarms). Taken together, this suggests some degree of specificity between errors of commission and positive symptoms of schizophrenia such as delusions.

   In the General Population. A similar pattern of results can be found among members of the general population (including community members, undergraduates, and online workers) who endorse symptoms of positive schizotypy, subclinical delusionality, and paranoia. Many studies conducted in this population used DRM paradigms to assess false recognition or false recall. For example, both Dehon et al. (2008) and Meyersburg et al. (2009) reported elevated susceptibility to the classic DRM intrusion effects among those endorsing symptoms of positive schizotypy. In Dehon et al. (2008), undergraduates who scored higher on the Peter Delusions Inventory (PDI; Peters et al., 1999) – a scale designed to index delusionality in general and clinical samples alike – made more extra-list intrusions of semantically related words. Meyersburg et al. (2009) found a similar effect in a community sample that included individuals reporting recovered memories of a past life (e.g., as a member of the British royal family, a space alien, or a 19th century newspaper boy). Compared to a control group, those reporting past lives were both more vulnerable to DRM intrusion/false alarm effects (in free recall/recognition) and reported more magical ideation on the MIS. MIS scores correlated directly with false alarms of semantically related lures in recognition (but not intrusions in free recall). No parallel effects emerged for semantically unrelated lures. Saunders et al. (2012) observed a similar pattern among undergraduates who endorsed more "Unusual Experiences" (a measure of positive

schizotypy) on the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE; Mason &

Claridge, 2006). Here, the high-Unusual-Experiences group made significantly more extra-list

intrusions of highly semantically related words. Saunders and colleagues also included several

weakly semantically related words on the study list. Interestingly, the high-Unusual-Experiences

group successfully recalled *more* of these weakly semantically related words, despite recalling

less of the other (semantically unrelated) studied items in free recall. Together, these findings

suggest that errors of commission in this population may be related to a heightened spread of

semantic activation – a process by which related concepts activate one another in network

models of semantic memory (e.g., Anderson & Pirolli, 1984; Collins & Loftus, 1975).

However, some DRM-based studies found elevated rates of commission errors in

conjunction with positive schizotypy or delusionality, even among semantically unrelated words.

Laws and Bhatt (2005) found that undergraduates who scored higher on the PDI made more

extra-list intrusion errors during free recall of words in a DRM paradigm, with the high-

delusionality group erroneously recalling over four times as many non-presented words than the

low-delusionality group (8.04% versus 1.95%). These intrusions occurred for both semantically

related *and* unrelated words (although the effect was larger for the former). In a subsequent

recognition memory test, the high-delusionality group similarly made more high-confidence

false alarms. A parallel pattern was found by Evans et al. (2019) among undergraduates who

completed a DRM-like image recognition task, in which a subset of the lures shared a category

with a previously presented image. Here, higher scores on the Unusual Experiences scale of the

O-LIFE were associated with more false alarms in recognition. Similar to Laws and Bhatt

(2005), Evans and colleagues found elevated false alarms among *both* same-category and

unrelated lures. Importantly, elevated false alarm rates were also seen in association with scores

on the PDI, indicating a relationship with delusionality more specifically. Finally, Rodriguez-Ferreiro et al. (2020) found that undergraduates scoring higher on the Distortion of Reality subscale (analogous to positive schizotypy) of the Esquizo-Q-A (Fonseca-Pedrero et al., 2010) made more false alarms in a word recognition DRM task. However, this was *only* the case among weakly semantically related words and unrelated words – there was *no* significant association between positive schizotypy and false alarms on the highly semantically related lures characteristic of a DRM paradigm. Corlett et al. (2009) similarly showed that undergraduates scoring higher in positive schizotypy (via the MIS) were *no more susceptible* to DRM errors than their lower-positive-schizotypy counterparts (although those endorsing more magical ideation did display a more liberal response bias). Together, these studies challenge the notion that false alarms and intrusion errors are driven solely by abnormal spreading of semantic activation among those experiencing positive symptoms in the general population.

Further, the association between positive schizotypy or delusionality and elevated false alarm rates persists outside of the context of DRM paradigms altogether. Sugimori and Tanno (2010) reported an association between subclinical delusionality (indexed by the Delusional Identification Checklist; Tanno et al., 2000) and false alarms in a word recognition memory test administered to undergraduates. Here, however, elevated false alarms were only found among *delusion-congruent* lures. In other words, "positive" delusionality (e.g., grandiosity) was associated with false recognition of adjectives with positive connotations (e.g., "adventurous"); "negative" delusionality (e.g., persecutory beliefs) with adjectives with negative connotations (e.g., "reckless"). In a large sample of adolescents (*n*=4744; ages 17-18), Rossi et al. (2016) found that the presence of psychotic experiences (i.e., delusions or hallucinations, as established by semi-structured interview; Zammit et al., 2013) coincided with greater odds of making false

alarms on a digit-based working memory task. This was true of both 2-back and 3-back versions of the working memory task, and effects persisted when controlling for IQ, drug use, and other psychiatric diagnoses. Further, Sahakyan and Kwapil (2019) combined samples of undergraduates and Amazon MTurk workers (total $n = 826$) to show that positive schizotypy (i.e., the positive factor of the Wisconsin Schizotypy Scales-Brief; Winterstein et al., 2011) was associated with elevated false alarms during recognition of both verbal and visual stimuli (words and faces, respectively). Moreover, mirroring several studies conducted in patient populations (Brébion et al., 1999; Brébion, Smith, Amador, et al., 1997; Seabury et al., 2021), Sahakyan and Kwapil found that this effect was specific to positive symptoms: in a double dissociation, positive schizotypy was associated with more false alarms (but not less correct recognition), while negative symptoms were conversely associated with less correct recognition (but not greater false alarms). Drawing from tenets of SDT, Sahakyan and Kwapil suggested that this pattern of effects could be explained by a *reduction of signal* in negative schizotypy but an *increase in noise* in positive schizotypy. Finally, in our own work, we found a similar pattern in the context of subclinical paranoia (Koller & Cannon, 2021). In an MTurk sample ($n=317$), we demonstrated that paranoia (i.e., via the Revised Green Paranoid Thoughts Scale B; R-GPTS-B; Freeman et al., 2019) was associated with elevated false alarms (but not decreased correct recognition) in word recognition. Notably, this effect was especially prominent for lures with high rates of extra-experimental exposure (i.e., common words in the English language, such as "bike") compared to less common pseudowords (i.e., nonsense words, such as "rimp"). This suggests that those high in paranoia may rely in part on context-nonspecific feelings of familiarity as a source of signal in recognition memory (although note that associations with false alarms were still seen among pseudowords, albeit to a lesser extent).

Intrusions and False Alarms – Summary. Together, these studies suggest that

delusionality is associated with increased acceptance of non-presented or task-irrelevant stimuli

in both schizophrenia spectrum disorders and in the general population. This effect has been

found among stimuli of various modalities (i.e., verbal, visual, and numeric). Further, this

finding has been replicated in multiple domains of memory (i.e., recall, recognition, and working

memory). Finally, this effect does *not* seem to be driven by abnormal spread of semantic

activation alone (as evidenced by errors of commission occurring even for semantically unrelated

lures or outside of the context of DRM tasks altogether). In fact, there are reports of impaired

recognition memory and/or elevated false alarms in these populations despite showing non-

elevated (Rodriguez-Ferreiro et al., 2020) or even reduced (Dagnall & Parker, 2009)

susceptibility to the DRM semantic intrusion effect, which corroborates the notion that increased

semantic spreading is unlikely to be the sole driver of these memory errors.

Source Memory. This pattern of performance, in which non-presented or task-irrelevant

material is interpreted as having been encountered in a task-relevant context, may represent an

excess of "noise" in the memory system that is being interpreted as meaningful signal (an idea

discussed in more detail by Salekyan & Kwapil, 2019). But what generates this noise? One

hypothesis is that this "noise" stems in part from deficits in the associative memory system

leading to breakdowns in the *contextual organization* of memory (Criss & Shiffrin, 2004; Dennis

& Humphreys, 2001). This may manifest as impaired "source memory", or a decreased ability to

attribute a given stimulus to its context (Johnson et al., 1993).

Reduced contextual organization of the memory system may bias one towards the use of

familiarity over recollective processes in memory retrieval (Yonelinas, 2002). To illustrate this

point, consider an associative memory system in which item-item and item-context associations

are formed and updated with high fidelity. When presented with a stimulus in the environment, this memory system will be capable of accessing relevant associations with this stimulus through auto-associative recall. This may include surrounding stimuli that co-occurred in time or space with the item in question or the background context within which it was presented. This recollective process represents one mechanism via which the memory system helps to determine the relevance of a given item to the current context. In a recognition memory task, this could mean determining whether an item is "old" – if it *does* call up task-relevant contextual associations – or "new" – if it does *not*. Accordingly, if one's memory system has *not* formed an accurate network of associative links between items and their spatiotemporal co-occurrents and contexts, one may have to rely on other processes (such as assessments of familiarity) to determine relevance (or to make old/new decisions in a recognition memory task). This may introduce additional "noise" to the system given that there are many potential reasons that a given item may feel familiar), leaving one vulnerable to the intrusion of irrelevant content into the retrieval process. This effect may help explain the results of Koller and Cannon (2021), whereby the association between paranoia and false alarm rates was greater among words that frequently appeared in other contexts (i.e., real words versus pseudowords). This suggests that higher-paranoia participants used cues other than item-item or item-context associations to determine whether a given stimulus was "old" or "new". Namely, they may have relied instead on a general sense of familiarity (i.e., whether they had *ever* seen a given item, without regard for the contexts in which it had been encountered), leaving them particularly vulnerable to false recognition. Importantly, a breakdown in contextual organization (and compensatory reliance on other cues such as familiarity) may represent a common mechanism for commission errors across various stimulus types (e.g., verbal, visual, etc.) insofar as it would be expected to

manifest not only among *semantic* associations (e.g., in DRM paradigms), but also among associations formed within and between other modalities.

Imbalances of familiarity versus recollection processes have been frequently observed in case-control studies of schizophrenia (Libby et al., 2013), as have associative memory deficits (e.g., see Achim & Weiss, 2008; Waters, 2004). This points to a chronic impairment of the associative memory system that would otherwise support recollective modes of retrieval. Evidence for a more specific relationship between source memory errors and delusionality can also be found in a number of studies. For one, many of the previously reviewed studies can be re-interpreted within an associative/source memory framework (Brébion et al., 1999; Brébion, Smith, Amador, et al., 1997; Koller & Cannon, 2021; Seabury et al., 2021). However, similar results have also been reported in more explicit tests of source memory.

In Schizophrenia Spectrum Disorders. In a case-control study, Brébion, Smith, Gorman, et al. (1997) showed that the positive subscales of the PANSS and the SAPS were positively associated with source errors. Namely, more severe positive symptoms corresponded to a greater likelihood of mistaking orally presented material as having been presented visually. Importantly, this bias was also specifically associated with scores on delusion-specific items of the PANSS. Anselmetti et al. (2007) similarly found that an actively delusional subset of a patient group made more source errors (internally versus externally generated) than their non-delusional counterparts. Doré et al. (2007) tested source memory among adolescents with psychosis, including memory for both the person who generated a given stimulus (i.e., experimenter versus participant) and the temporal context in which it originally appeared (i.e., list 1 or list 2). Compared to a healthy control group, adolescents with psychosis showed poorer performance on both aspects of the source task. Positive symptoms (via the PANSS) were correlated with the

tendency to misattribute internally generated words to an external source (but not temporal context errors). Finally, in an fMRI paradigm, Thoresen et al. (2014) similarly found that delusionality within a patient sample (indexed by scores on delusion-related items of the PANSS) was associated with a greater tendency to mistake internally generated stimuli as having been externally presented. Following an encoding phase during which verbal prompts were either accompanied by an image or visualized by the participant, participants with more severe delusions were more likely to misremember imagined stimuli as having been presented in image form. Importantly, this relationship persisted when controlling for other PANSS symptoms, suggesting specificity to delusions. Further, delusion severity was strongly negatively associated with left hippocampal activity during this task: when attempting to recall the source of imagined stimuli, left hippocampal activity was attenuated among those with more severe delusions. This supports the notion that a breakdown in associative memory (a core function of the hippocampus; e.g., see Hannula & Ranganath, 2008) may be driving errors in source monitoring.

In the General Population A relationship between delusionality and source memory error also appears in the general population. Dehon et al. (2008) found that PDI scores among undergraduates were associated with lower scores on a source memory metric (in addition to elevated rates of intrusion errors, as reviewed in the previous section). After completing a DRM procedure, Dehon and colleagues asked participants to list all of the words that came to mind during testing but which they did *not* report because they had no memory of the experimenter producing it. This allowed for the estimation of the number of *potential* DRM intrusions that had been successfully rejected due to accurate source memory judgments. Using this estimate, the source memory metric was calculated by dividing the number of actual DRM intrusion errors by the total number of actual plus potential DRM intrusions (with higher scores on this metric

corresponding to worse source memory). PDI scores positively correlated with this metric, indicating that delusionality was associated with a greater proportion of DRM intrusions that *failed* to be rejected using source memory. Mirroring Anselmetti et al. (2007), Humpston et al. (2017) demonstrated that positive schizotypy (indexed by the O-LIFE Unusual Experiences subscale) was associated with elevated source memory errors. These included internal source errors – i.e., confusing which acts were performed versus imagined by the participant – and internal-external source errors – i.e., confusing which acts were imagined or performed by the participant versus performed by the experimenter. In a community sample, Hegelstad et al. (2020) further showed that error in a *spatial* source memory task was associated with positive schizotypy (via the positive subscale of the Community Assessment of Psychic Experiences; CAPE-42; Stefanis et al., 2002). In this task, participants attempted to recall where a stimulus had previously appeared in a 2D space. Error in this task was indexed by the degree of displacement between a participant's estimate and the original position of the stimulus. Here, positive schizotypy was associated with greater displacement error, suggesting impaired binding between item and spatial context.

Finally, we have demonstrated that individuals higher in paranoia show systematic impairment in a temporal source memory task (Koller, Ongchoco, et al., in prep). In this preregistered study, MTurkers (*n*=250) first completed a learning phase in which they observed a stream of images one after another and completed trial-wise attention checks. Later, they completed an old/new recognition test. Importantly, if they judged an item to be "old", they were asked to use a timeline to mark *when* they remembered this item as having appeared during the learning phase. This allowed us to calculate a highly sensitive measure of *temporal displacement* error – the distance between a participant's estimate on the timeline and the original position of

the item during the learning phase. Paranoia (via the R-GPTS-B) was strongly positively associated with temporal displacement, indicating that higher-paranoia participants tended to systematically remember recognized items as having occurred *more recently* than they actually did. Notably, this effect was associated with paranoia above and beyond other aspects of schizotypy (i.e., disorganized and negative symptoms, via the Multidimensional Schizotypy Scale; Kwapil et al., 2018) and various covariates (age, education, race, and attention check performance). Finally, there was no parallel association between paranoia and a non-temporal control judgment made on a similar slider, suggesting that these results are unlikely to be driven by non-mnemonic processes such as pseudoneglect (Liouta et al., 2008). Further work by Ongchoco, Koller et al., (in prep) has revealed an association between paranoia and attenuated event segmentation in working memory, suggesting that breakdowns in contextual organization of memory may be related to changes in how individuals parse continuous experience into discrete episodes.

Source Memory – Summary. In summary, a number of studies point to a specific association between source memory deficits and delusionality in both schizophrenia spectrum disorders and in the general population. These associations have been found in the context of internal-external judgments, spatial source memory, and temporal source memory. Together, these studies generally support the notion that a breakdown in the contextual organization of memory along spatiotemporal dimensions may represent one source of "noise" that contributes to memory error among those experiencing delusionality. However, few studies directly speak to the relationship between impaired source memory and other aspects of memory error such as false alarms and intrusions (i.e., this has been assessed only tangentially by Dehon et al., 2008; Koller & Cannon, 2021). Relatedly, the literature offers no firm conclusions about whether

internal-external source monitoring is undergirded by the same associative memory mechanisms that may support spatiotemporal source judgments. Clearly, while associative memory deficits may represent a common mechanism of memory error in these populations, more research is needed to disentangle the associations between the contextual organization of memory, errors of commission, and delusionality.

Metamemory. Up to this point, we have reviewed associations between delusionality and "Type-I" memory performance (e.g., "Is this item old or new?"). Yet effective memory systems are also defined by their ability to conduct "Type-II" appraisals of these judgments (e.g., "How *confident* am I that this item is old or new?"). This capacity is referred to as *metamemory*, and represents a subdomain of metacognition – or the process of thinking about one's thinking (Dunlosky & Metcalfe, 2008). Metamemory is often measured using confidence ratings, in which participants not only make a memory judgment (e.g., old/new) but also report how confident they are in this judgment (e.g., sure old, maybe old, maybe new, sure new). Accordingly, impaired metamemory is often characterized as underconfidence in correct responses or overconfidence in incorrect responses. Metamemory impairment is germane to the study of delusions in that it represents the uncritical acceptance of false or irrelevant material that may give rise to beliefs which are untenable yet held with high degrees of conviction.

Metamemory biases have been frequently reported in case-control studies within the schizophrenia literature (Moritz et al., 2004; Moritz et al., 2008; Moritz et al., 2003), including in contexts where Type-I memory performance is intact (Eisenacher et al., 2015; Moritz et al., 2006). However, many of these studies found *no* specific or differential associations between metamemory impairment and positive symptoms (though cf. Eisenacher et al., 2015). In this section, we return to several of the studies reviewed in previous sections to assess the evidence

for specificity in the relationship between metamemory impairment and delusionality in this body of literature.

In Schizophrenia Spectrum Disorders. Several of the previously reviewed patient studies also collected metamemory measures (e.g., self-reported confidence ratings). In Bhatt et al. (2010), *both* patient groups (i.e., those actively experiencing delusions and those who were not) endorsed false alarms with higher confidence than controls in a DRM recognition task. Interestingly, the active-delusion group endorsed their "miss" errors (i.e., responses of "new" to an "old" stimulus) with higher confidence than the non-delusional group. Seabury et al. (2021) found more consistent and specific associations between high-confidence errors and positive symptoms: here, positive symptoms (but not negative symptoms) were associated with elevated confidence on incorrect responses in a relational recognition memory task (i.e., both false alarms and misses). Finally, Doré et al. (2007) found no specific associations between positive symptoms and confidence ratings in their source memory task, although the patient group made less high-confident correct responses and more high-confident incorrect responses overall.

In the General Population. In Laws and Bhatt (2005), participants high in delusionality (as indexed by the PDI) made more high-confident errors during recognition memory testing. In particular, they were specifically overconfident in false alarms (but not in misses). Evans et al. (2019) similarly found that both O-LIFE Unusual Experience and PDI scores were positively associated with a tendency to endorse false alarm errors with the highest confidence response. This was true for both same-category and unrelated lures. No parallel effect was seen for misses. Interestingly, the authors also found an association between delusionality and confidence on "hits" (i.e., responses of "old" to an "old" stimulus). Corlett et al. (2009) similarly found elevated confidence in DRM false alarms in association with positive schizotypy (via the MIS), despite

finding no evidence for elevated *rates* of false alarms. No parallel associations emerged for other response types (i.e., hits, misses, correct rejections). Importantly, this relationship was specific to positive symptoms – no parallel associations were seen for other measured aspects of schizotypy (including physical and social anhedonia). Koller and Cannon (2021) found that paranoia (via the R-GPTS-B) was associated with generally impaired metamemory, as captured by meta-*d'* (an SDT-compatible metric of metacognitive sensitivity; Fleming, 2017; Maniscalco & Lau, 2012). More specifically, higher-paranoia participants showed elevated confidence on error trials during recognition, including both false alarms and misses. Interestingly, overconfidence on false alarms was accentuated among real word versus pseudo-word lures, suggesting that extra-experimental familiarity may have been a driver of both recognition *and* metamemory judgments among higher-paranoia participants. Unexpectedly, paranoia was also associated with higher confidence on correct rejections (i.e., responses of "new" on a "new" trial). Rodriguez-Ferreiro et al. (2020) found a slightly different effect, in which participants high in positive schizotypy (as indexed by the Esquizo-Q-A) showed *decreased* confidence on correct rejections but no difference in confidence on false alarms. Finally, Hegelstad et al. (2020) found no significant relationships between positive schizotypy (via the CAPE-42) and confidence in spatial memory judgments (as indexed by a selected area of the 2D task space within which participants could be sure that the target originally appeared).

Metamemory – Summary. Altogether, there is strong evidence for a relationship between metamemory impairments and schizophrenia in general (i.e., in case-control studies), and mixed evidence for a more specific relationship between metamemory and delusionality. Some of the aforementioned studies point to strong and/or differential relationships between positive symptoms such as delusionality and overconfidence in memory errors (Eisenacher et al., 2015;

Koller & Cannon, 2021; Seabury et al., 2021) – especially for errors of commission (Evans et al., 2019; Laws & Bhatt, 2005) – or underconfidence in correct responses (Rodriguez-Ferreiro et al., 2020). Others revealed generally impaired metamemory in schizophrenia, but no specific associations with positive symptoms (Bhatt et al., 2010; Moritz et al., 2004; Moritz et al., 2008; Moritz et al., 2006; Moritz et al., 2003). Finally, Hegelstad et al. (2020) showed no association between metamemory and symptoms of schizotypy whatsoever.

General Summary. In contrast to more general neurocognitive metrics (which tend to track more closely with negative or disorganized symptoms; e.g. Aleman et al., 1999; Ventura et al., 2010), the results of a scoping review of the literature reveal several subdomains of memory which are associated with positive symptoms such as delusionality. These include errors of commission (i.e., false alarms and intrusion errors), source memory deficits, and to some extent metamemory impairment.

Alternate Explanations of Null Effects

Yet even within these three subdomains of memory, there are clear inconsistencies in the literature. For example, not all studies find errors of commission to be specifically associated with delusionality: some find them to be associated with hallucinations (e.g., Brébion et al., 1998; Brébion et al., 2020), others with symptoms of disorganization (e.g., Brébion et al., 2020; Fridberg et al., 2010). Many studies report no such association at all (e.g., Elvevag et al., 2004; Fisher et al., 2007; Heinrichs & Vaz, 2004; Moritz et al., 2006). Similarly, while case-control studies suggest that schizophrenia is characterized by general impairments in associative/source memory (Achim & Lepage, 2003; Achim & Weiss, 2008; Armstrong et al., 2012) and metamemory (Eisenacher et al., 2015; Moritz et al., 2004; Moritz et al., 2008; Moritz et al., 2003), more specific associations with delusionality are not always evident (e.g., Moritz et al.,

2006; Ongür et al., 2006). Similar inconsistencies can be seen in the general population, with some studies finding associative memory deficits in negative but not positive schizotypy (Sahakyan et al., 2019). How do we reconcile these mixed findings? Here, we attempt to address several of these inconsistencies and offer recommendations for future research.

Delusions, Hallucinations, or Disorganization?

A number of the studies reviewed here report gross measures of positive symptoms of schizophrenia or schizotypy, which collapse across various phenomena (i.e., delusions and hallucinations) that may have unique cognitive correlates. Further, some studies consider symptoms of disorganization a positive symptom (e.g., Aleman et al., 1999), while others measure these symptoms as a separate cluster. The reliance on positive subscale scores reflecting heterogenous symptoms introduces uncertainty regarding the specific relationships between delusionality and memory. To what extent are these associations driven by hallucinations versus delusions? Could null associations with one phenomenon mask true associations with the other? Examples of these dissociations are evident in the literature. For instance, Brébion et al. (1998) found that the PANSS positive subscale was associated with a bias towards false alarms in a patient sample, but subsequent analysis revealed the association to be driven by hallucinations and not delusions. On the contrary, Brébion et al. (1999) found intrusion errors to be associated with delusions and thought disorganization, but not hallucinations. Finally, Brébion et al. (2020) demonstrated that intrusion errors were associated with hallucinations and thought disorganization, but not delusions. While hallucinations often co-occur with delusions and have been hypothesized to be a causal factor in delusion formation (Maher, 2006; Smeets et al., 2012) and/or reinforced by delusions (Krabbendam et al., 2004), it is important to tease apart whether memory biases are more closely related to one symptom or the other or rather represent a shared

cognitive mechanism. Similar can be said of disorganization symptoms, which are associated with neurocognitive impairment (Ventura et al., 2010) and tend to correlate with many other symptom clusters in psychotic illness (Peralta et al., 2020), including delusionality (Bentham et al., 1996). Future research should thus assess to what extent the identified memory biases covary with specific symptoms such as delusions, hallucinations, and symptoms of disorganization (e.g., formal thought disorder).

Finally, even metrics that are more proximal to delusionality (e.g., R-GPTS-B, PDI, MIS, etc.) are composed of items that correspond to a wide array of delusion-like or delusion-adjacent experiences. These range from superstitious beliefs that are relatively common in the general population (e.g., "I have sometimes been fearful of stepping on sidewalk cracks", from the MIS; Eckblad & Chapman, 1983) to persecutory ideation (e.g., "I was convinced there was a conspiracy against me", from the R-GPTS B; Freeman et al., 2019). While these experiences generally fall under the umbrella of delusion-like thinking, they may reflect various subfactors of delusionality or may not even be characteristic of delusions *per se*. As such, it will be critical for future research to compare and contrast multiple scales that measure delusion-like symptoms in order to test more specific relationships between aspects of delusionality and memory performance. Dimensionality reduction techniques (e.g., principle components analysis) may also prove useful in identifying scale subfactors that are meaningfully related to cognitive processes of interest.

Medication and Other Confounding Variables

Further, when reckoning with the spread within the literature on memory and delusions, it is important to consider potentially confounding third variables. Antipsychotic medication is one such variable, with medication confounds representing a longstanding issue in the field of

schizophrenia research (Blanchard & Neale, 1992). Antipsychotics are a first-line treatment for many individuals with schizophrenia, with D2 receptor antagonism representing a common mechanism of action (Lally & MacCabe, 2015). Given their anti-dopaminergic mechanism, the majority of antipsychotics have robust effects on positive symptoms (Leucht et al., 2009). However, their effects on cognitive symptoms such as memory deficits are mixed. Some reports suggest that neither first nor second-generation antipsychotics tend to produce clinically meaningful improvements to cognition (Hill et al., 2010). Other studies point to small but statistically significant cognitive improvements across several domains, including memory (Weickert et al., 2003), irrespective of the type of medication employed (Keefe et al., 2007). Past work has also revealed that antipsychotic medication affects metamemory performance among patients with schizophrenia (Moritz et al., 2016). Taken together, this suggests that associations between delusion and memory may be muddied by the high medication rates characteristic of the patient studies reviewed in this manuscript. As such, future research should seek to determine whether antipsychotic medication moderates the relationship between delusionality and memory dysfunction. Other potential confounders include variance in task difficulty across studies, measurement (im)precision (e.g., assessing the presence versus absence of memory errors may be less sensitive than measuring degree of error, as in Hegelstad et al., 2020; Koller et al., in prep), and power issues (i.e., many case-control studies have low sample sizes).

One potential strategy to address several of these confounds lies in the dimensional model of schizophrenia. Studying schizotypy in the general population may allow us to assess relationships between various schizophrenia-relevant cognitive processes while simultaneously avoiding medication confounds and maximizing power (i.e., through large-scale community or online sampling). Notably, in our review there was in fact more consistent evidence for an

association between false recognition and delusionality in the general population than there was in patient samples. A similar pattern emerged among metamemory studies. Granted, there are undoubtedly other factors that may influence this pattern of results (e.g., stage of illness effects, differences in demographic variables). Yet nonetheless, this pattern points to a need for greater statistical power and a firmer understanding of the moderating effects of third variables such as antipsychotic medication on the relationship between memory function and delusionality.

State Versus Trait

Measurement issues and confounding variables aside, the inconsistencies in the literature may also have broader theoretic implications. When assessing overall trends in the reviewed literature, there are two broad patterns of inconsistencies that emerge. The first is in the realm of errors of commission. Here, there is substantial evidence for an association with positive symptoms, yet many studies report *no* evidence for errors of commission in schizophrenia or schizotypy more broadly (e.g., Elvevag et al., 2004; Fisher et al., 2007; Heinrichs & Vaz, 2004; Moritz et al., 2006). Conversely, associative/source memory and metamemory are consistently linked to schizophrenia and schizotypy, but are *not* always associated with positive symptoms (Achim & Lepage, 2003; Achim & Weiss, 2008; Armstrong et al., 2012; Moritz et al., 2004; Moritz et al., 2008; Moritz et al., 2003). In other words, whereas errors of commission are often associated with positive symptoms but are not always seen in schizophrenia, source and metamemory deficits are often seen in schizophrenia but are not always associated with positive symptoms.

One possible explanation of these inconsistencies lies in the differentiation of *state*-based memory function from *trait*-like memory impairments (Cannon, 2021). For example, errors of commission may be tied to *state*-based changes in memory function (in that they are consistently

associated with positive symptoms, but *not* always found in case-control studies). During acute psychotic states, cascading changes to cognitive and perceptual functioning may push the memory system into a mode of processing in which irrelevant content has a greater likelihood of crossing a decision threshold. This may be influenced by elevated attribution of salience to innocuous material (i.e., hypersalience) or the presence of noisy perceptual input. The notion that memory function may be particularly altered during acute psychotic states is supported in part by studies on ketamine – an NMDA antagonist often used as a drug model of schizophrenia (Corlett et al., 2013; Frohlich & Van Horn, 2014). Ketamine, which generates dose-dependent psychotic-like symptoms, has also been found to produce memory impairments that roughly resemble those seen in schizophrenia, including impaired source memory and reliance on familiarity over recollective processes during memory retrieval (see Fletcher & Honey, 2006 for a review).[2] Thus, during periods of more acute psychosis, individuals may rely more heavily on compensatory cues such as familiarity, resulting in elevated susceptibility to false recognition. On the other hand, associative/source and metamemory impairments may be more *trait*-like in nature (in that they are consistently found in case-control studies but *not* always associated with positive symptoms). While relevant longitudinal studies are limited, this general notion is supported by studies reporting associative memory deficits in unaffected first degree relatives (Oertel et al., 2019) and in early psychosis (Avery et al., 2019; Avery et al., 2021), as well as those reporting metamemory impairment in early psychosis (Eisenacher et al., 2015).

Critically, these state and trait-like memory features may interact with one another. During psychotic states, which are characterized by high sensory noise (Horga & Abi-Dargham, 2019; Rolls et al., 2008) and aberrant prediction error (Corlett et al., 2007), otherwise infrequent

---

[2] Though note that these memory impairments do *not* necessarily include elevated false alarms in recognition memory (e.g., see Morgan et al., 2004).

or innocuous mental events (such as instances of false recognition or intrusions of decontextualized thought) may occur with higher frequency and/or take on a heightened sense of urgency. Normal interpretation of these high-salience cognitions may be frustrated by an associative memory system that struggles to untangle the web of connections that called this content to mind. Further, trait-like impairments to metamemory functioning may reduce the likelihood that this content is flagged as unreliable or low-confidence. Altogether, this may result in high-salience (but low-source) cognitions that defy conventional explanation and yet are accompanied with high conviction (Cannon, 2015). To make sense of these odd experiences, one may begin to form new connections between these decontextualized mental events. This may result in the genesis of "beliefs" which, while fulfilling a key explanatory function for salient internal phenomena, have little bearing on the objective state of the world. Thus, the "loosened associations" characteristic of schizophrenia (Bleuler, 1911/1950) may reflect an attempt to explain *decontextualized mnemonic content* that is generated during periods of *heightened salience*. A recent review further suggests that such loosened associations may be instantiated by shallow hippocampal cognitive maps (Musa et al., 2022). In this way, associations between more stable, trait-like memory biases and positive symptoms may come online only during more acute psychotic states during which one's memory system is flooded by high-salience mental events that demand interpretation.

Conclusion

Here, we performed a scoping review of the literature on memory and delusionality in order to assess the extent to which aberrant memory is reliably and differentially associated with delusional ideation across the psychosis spectrum. Standing in contrast to reviews of general neurocognitive metrics (which tend to track more closely with negative or disorganized

symptoms; e.g., Aleman et al., 1999; Ventura et al., 2010), we identified several memory biases that are more tightly coupled with positive symptoms such as delusionality. These include a propensity to commit errors of commission (i.e., false alarms and intrusion errors), source memory biases, and metamemory impairment (e.g., overconfidence in memory errors, underconfidence in correct responses). However, even within these three subdomains of memory, associations with delusionality are inconsistent. This may be due to heterogeneity in the measurement of positive symptoms of schizophrenia, third-variable confounds such as antipsychotic medication, and complex interactions between state and trait-like phenomena.

Relevance to Delusionality

Drawing from explanatory models of delusionality, we posit that these three aspects of altered memory function may mutually reinforce one another and help to seed content that is ripe for delusional interpretation. Breakdowns in the associative memory system, via which items are relationally bound to both co-occurring items and their spatiotemporal context, may manifest as source memory deficits and contribute to the conditions under which errors of commission are likely to occur. Metamemory impairments may reduce the odds that this erroneous content gets flagged as such. These dynamics may be particularly relevant to positive symptoms during periods of acute psychosis, during which time the memory system must additionally contend with noisy perceptual input and/or experiences of aberrant salience. In such a state, context-irrelevant or erroneous mnemonic content may take on a heightened sense of meaning, demanding explanation. Due to underlying trait-like memory deficits, this high-salience (but low-source) content may be rendered uninterpretable by conventional means (i.e., due to failures in associative memory) and/or endorsed with high conviction (i.e., due to failures in metamemory). This may form the basis for delusional beliefs that serve to organize and explain

these aberrant mental phenomena, but which are not themselves reflective of the structure of the external world.

Recommendations for Future Research

Based on our review, we suggest that delusionality is robustly and differentially associated with several specific memory biases. Our read of the literature suggests that these memory biases may be important drivers of delusionality, insofar as they seed the mental content from which delusional beliefs are born. However, they are likely not in and of themselves sufficient as they may require aberrant attributions of salience in order to coalesce into full-blown delusions (as opposed to fleeting moments of intrusive thought, mind wandering, or odd associations that are normative and universal experiences). Future research should focus on disentangling the associations between these memory biases and the various positive symptoms of schizophrenia, as well as identifying the role of antipsychotic medication in the expression of these associations. Finally, we recommend testing hypotheses generated by the proposed state-trait interaction model of memory and delusion: namely, that the co-occurrence of trait-like risk factors (i.e., associative and metamemory function) with certain state-like features (i.e., mnemonic errors of commission and aberrant salience) will strongly predict delusionality. In closing, standing in contrast to historical reports of a lack of association between delusionality and general neurocognition, this review affirms a potentially pernicious connection between particular aspects of associative memory function and the genesis and maintenance of delusional and delusion-like beliefs.

References

Achim, A. M., & Lepage, M. (2003). Is associative recognition more impaired than item recognition memory in Schizophrenia? A meta-analysis. *Brain and Cognition, 53*(2), 121-124. https://doi.org/10.1016/s0278-2626(03)00092-7

Achim, A. M., & Weiss, A. P. (2008, Sep). No evidence for a differential deficit of reality monitoring in schizophrenia: a meta-analysis of the associative memory literature. *Cogn Neuropsychiatry, 13*(5), 369-384. https://doi.org/10.1080/13546800802299476

Aleman, A., Hijman, R., de Haan, E. H. F., & Kahn, R. S. (1999). Memory impairment in schizophrenia: A meta-analysis. *Am J Psychiatry, 156*(9), 1358-1366.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Publishing. https://doi.org/10.1176/appi.books.9780890425596

Anderson, J. R., & Pirolli, P. L. (1984). Spread of activation. *J Exp Psychol Learn Mem Cogn, 10*(4), 791-798. https://doi.org/10.1037/0278-7393.10.4.791

Andreasen, N. C. (1984). Scale for the assessment of positive symptoms (SAPS).

Anselmetti, S., Cavallaro, R., Bechi, M., Angelone, S. M., Ermoli, E., Cocchi, F., & Smeraldi, E. (2007, Feb 28). Psychopathological and neuropsychological correlates of source monitoring impairment in schizophrenia. *Psychiatry Res, 150*(1), 51-59. https://doi.org/10.1016/j.psychres.2005.12.004

Armstrong, K., Williams, L. E., & Heckers, S. (2012, Jul). Revised associative inference paradigm confirms relational memory impairment in schizophrenia. *Neuropsychology, 26*(4), 451-458. https://doi.org/10.1037/a0028667

Avery, S. N., Armstrong, K., Blackford, J. U., Woodward, N. D., Cohen, N., & Heckers, S. (2019, Oct). Impaired relational memory in the early stage of psychosis. *Schizophr Res, 212*, 113-120. https://doi.org/10.1016/j.schres.2019.07.060

Avery, S. N., Armstrong, K., McHugo, M., Vandekar, S., Blackford, J. U., Woodward, N. D., & Heckers, S. (2021, Jan 23). Relational Memory in the Early Stage of Psychosis: A 2-Year Follow-up Study. *Schizophr Bull, 47*(1), 75-86. https://doi.org/10.1093/schbul/sbaa081

Basso, M. R., Nasrallah, H. A., Olson, S. C., & Bornstein, R. A. (1998). Neuropsychological correlates of negative, disorganized and psychotic symptoms in schizophrenia. *Schizophr Res, 31*(2-3), 99-111. https://doi.org/https://doi.org/10.1016/S0920-9964(98)00023-1

Behrendt, R. P. (2013). Conscious experience and episodic memory: hippocampus at the crossroads. *Front Psychol, 4*, 304. https://doi.org/10.3389/fpsyg.2013.00304

Bein, O., Duncan, K., & Davachi, L. (2020). Mnemonic prediction errors bias hippocampal states. *Nat Commun, 11*(3451). https://doi.org/https://doi.org/10.1038/s41467-020-17287-1

Bentham, A. M., McKay, A. P., Quemada, I., Clare, L., Eastwood, N., & McKenna, P. J. (1996, Nov 1). Delusions in schizophrenia: a phenomenological and psychological exploration. *Cogn Neuropsychiatry, 1*(4), 289-304. https://doi.org/10.1080/135468096396451

Bhatt, R., Laws, K. R., & McKenna, P. J. (2010, Jul 30). False memory in schizophrenia patients with and without delusions. *Psychiatry Res, 178*(2), 260-265. https://doi.org/10.1016/j.psychres.2009.02.006

Blanchard, J. J., & Neale, J. M. (1992). Medication effects: Conceptual and methodological issues in schizophrenia research. *Clin Psychol Rev, 12*(3), 345-361. https://doi.org/https://doi.org/10.1016/0272-7358(92)90141-T

Bleuler, E. (1911/1950). *Dementia praecox or the group of schizophrenias*. International Universities Press.

Bogerts, B. (1997). The temperolimbic system theory of positive schizophrenic symptoms. *Schizophr Bull, 23*(3), 423-435.

Boyer, P., Phillips, J. L., Rousseau, F. L., & Ilivitsky, S. (2007, Apr). Hippocampal abnormalities and memory deficits: new evidence of a strong pathophysiological link in schizophrenia. *Brain Res Rev, 54*(1), 92-112. https://doi.org/10.1016/j.brainresrev.2006.12.008

Brébion, G., Amador, X., Smith, M. J., Malaspina, D., Sharif, Z., & Gorman, J. M. (1999). Opposite links of positive and negative symptomatology with memory errors in schizophrenia. *Psychiatry Res, 88*, 15-24.

Brébion, G., Smith, M. J., Amador, X., Malaspina, D., & Gorman, J. M. (1997). Clinical correlates of memory in schizophrenia: Differential links between depression, positive and negative symptoms, and two types of memory impairment. *Am J Psychiatry, 154*(1), 1538-1543. https://doi.org/https://doi.org/10.1176/ajp.154.11.1538

Brébion, G., Smith, M. J., Amador, X., Malaspina, D., & Gorman, J. M. (1998). Word recognition, discrimination accuracy, and decision bias in schizophrenia: association with positive symptomatology and depressive symptomatology. *The Journal of Nervous and Mental Disease, 186*(10), 604-609. https://doi.org/10.1097/00005053-199810000-00003

Brébion, G., Smith, M. J., Gorman, J. M., & Amador, X. (1997). Discrimination accuracy and decision biases in different types of reality monitoring in schizophrenia. *The Journal of Nervous and Mental Disease, 185*(4), 247-253. https://doi.org/10.1097/00005053-199704000-00005

Brébion, G., Stephan-Otto, C., Cuevas-Esteban, J., Usall, J., & Ochoa, S. (2020, Jun). Impaired memory for temporal context in schizophrenia patients with hallucinations and thought disorganisation. *Schizophr Res, 220*, 225-231. https://doi.org/10.1016/j.schres.2020.03.014

Buchanan, R. (2012). Is belief a propositional attitude? *Philosophers' Imprint, 12*(1-20).

Butler, R. W., & Braff, D. L. (1991). Delusions: a review and integration. *Schizophr Bull, 17*(4), 633-647.

Caligiuri, M. P., Hellige, J. B., Cherry, B. J., Kwok, W., Lulow, L. L., & Lohr, J. B. (2005, Dec 15). Lateralized cognitive dysfunction and psychotic symptoms in schizophrenia. *Schizophr Res, 80*(2-3), 151-161. https://doi.org/10.1016/j.schres.2005.07.030

Cannon, T. D. (2015, Dec). How Schizophrenia Develops: Cognitive and Brain Mechanisms Underlying Onset of Psychosis. *Trends Cogn Sci, 19*(12), 744-756. https://doi.org/10.1016/j.tics.2015.09.009

Cannon, T. D. (2021, Dec 14). Psychosis, schizophrenia, and states vs. traits. *Schizophr Res*. https://doi.org/10.1016/j.schres.2021.12.001

Collins, A. M., & Loftus, E. F. (1975). A spreading activation theory of semantic memory. *Psychol Rev, 82*(6), 407-428. https://doi.org/10.1037/0033-295X.82.6.407

Connors, M. H., & Halligan, P. W. (2014). A cognitive account of belief: a tentative road map. *Front Psychol, 5*, 1588. https://doi.org/10.3389/fpsyg.2014.01588

Corlett, P. R., Cambridge, V., Gardner, J. M., Piggot, J. S., Turner, D. C., Everitt, J. C., Arana, F. S., Morgan, H. L., Milton, A. L., Lee, J. L., Aitken, M. R., Dickinson, A., Everitt, B. J., Absalom, A. R., Adapa, R., Subramanian, N., Taylor, J. R., Krystal, J. H., & Fletcher, P. C. (2013). Ketamine effects on memory reconsolidation favor a learning model of delusions. *PLoS One, 8*(6), e65088. https://doi.org/10.1371/journal.pone.0065088

Corlett, P. R., Murray, G. K., Honey, G. D., Aitken, M. R., Shanks, D. R., Robbins, T. W., Bullmore, E. T., Dickinson, A., & Fletcher, P. C. (2007, Sep). Disrupted prediction-error

signal in psychosis: evidence for an associative account of delusions. *Brain, 130*(Pt 9), 2387-2400. https://doi.org/10.1093/brain/awm173

Corlett, P. R., Simons, J. S., Pigott, J. S., Gardner, J. M., Murray, G. K., Krystal, J. H., & Fletcher, P. C. (2009). Illusions and delusions: relating experimentally-induced false memories to anomalous experiences and ideas. *Front Behav Neurosci, 3*(53), 1-9. https://doi.org/10.3389/neuro.08.053.2009

Corlett, P. R., Taylor, J. R., Wang, X. J., Fletcher, P. C., & Krystal, J. H. (2010, Nov). Toward a neurobiology of delusions. *Prog Neurobiol, 92*(3), 345-369. https://doi.org/10.1016/j.pneurobio.2010.06.007

Criss, A. H., & Shiffrin, R. M. (2004, Jul). Context noise and item noise jointly determine recognition memory: a comment on Dennis and Humphreys (2001). *Psychol Rev, 111*(3), 800-807. https://doi.org/10.1037/0033-295X.111.3.800

Dagnall, N., & Parker, A. (2009, Mar). Schizotypy and false memory. *J Behav Ther Exp Psychiatry, 40*(1), 179-188. https://doi.org/10.1016/j.jbtep.2008.08.001

Das, T., Ivleva, E. I., Wagner, A. D., Stark, C. E., & Tamminga, C. A. (2014, Oct). Loss of pattern separation performance in schizophrenia suggests dentate gyrus dysfunction. *Schizophr Res, 159*(1), 193-197. https://doi.org/10.1016/j.schres.2014.05.006

Deese, J. (1959). On the prediction of occurrence of particular verbal intrusions in immediate recall. *Journal of Experimental Psychology, 58*(1), 17-22. https://doi.org/10.1037/h0046671

Dehon, H., Bastin, C., & Larøi, F. (2008). The influence of delusional ideation and dissociative experiences on the resistance to false memories in normal healthy subjects. *Personality and Individual Differences, 45*(1), 62-67. https://doi.org/10.1016/j.paid.2008.02.020

Dennis, S., & Humphreys, M. S. (2001). A context noise model of episodic word recognition. *Psychol Rev, 108*(2), 452-478. https://doi.org/10.1037/0033-295x.108.2.452

Dibben, C. R., Rice, C., Laws, K., & McKenna, P. J. (2009, Mar). Is executive impairment associated with schizophrenic syndromes? A meta-analysis. *Psychol Med, 39*(3), 381-392. https://doi.org/10.1017/S0033291708003887

Dominguez, M. G., Viechtbauer, W., Simons, C. J., van Os, J., & Krabbendam, L. (2009, Jan). Are psychotic psychopathology and neurocognition orthogonal? A systematic review of their associations. *Psychol Bull, 135*(1), 157-171. https://doi.org/10.1037/a0014415

Doré, M. C., Caza, N., Gingras, N., & Rouleau, N. (2007, Nov). Deficient relational binding processes in adolescents with psychosis: evidence from impaired memory for source and temporal context. *Cogn Neuropsychiatry, 12*(6), 511-536. https://doi.org/10.1080/13546800701614098

Dunlosky, J., & Metcalfe, J. (2008). *Metacognition*. Sage Publications.

Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *J Consult Clin Psychol, 51*(2), 212-225. https://doi.org/10.1037//0022-006x.51.2.215

Eichenbaum, H. (2017, Oct). Time (and space) in the hippocampus. *Curr Opin Behav Sci, 17*, 65-70. https://doi.org/10.1016/j.cobeha.2017.06.010

Eisenacher, S., Rausch, F., Ainser, F., Mier, D., Veckenstedt, R., Schirmbeck, F., Lewien, A., Englisch, S., Andreou, C., Moritz, S., Meyer-Lindenberg, A., Kirsch, P., & Zink, M. (2015, Nov). Investigation of metamemory functioning in the at-risk mental state for psychosis. *Psychol Med, 45*(15), 3329-3340. https://doi.org/10.1017/S0033291715001373

Elvevag, B., Fisher, J. E., Weickert, T. W., Weinberger, D. R., & Goldberg, T. E. (2004). Lack of false recognition in schizophrenia: a consequence of poor memory? *Neuropsychologia, 42*(4), 546-554. https://doi.org/10.1016/j.neuropsychologia.2003.08.013

Evans, L. H., McCann, H. M., Isgar, J. G., & Gaston, A. (2019, Mar). High delusional ideation is associated with false pictorial memory. *J Behav Ther Exp Psychiatry, 62*, 97-102. https://doi.org/10.1016/j.jbtep.2018.09.005

Feyaerts, J., Henriksen, M. G., Vanheule, S., Myin-Germeys, I., & Sass, L. A. (2021). Delusions beyond beliefs: a critical overview of diagnostic, aetiological, and therapeutic schizophrenia research from a clinical-phenomenological perspective. *The Lancet Psychiatry, 8*(3), 237-249. https://doi.org/10.1016/s2215-0366(20)30460-0

Fisher, J. E., Heller, W., & Miller, G. A. (2007, Mar 2). Semantic associations, lateralized frontal function, and context maintenance in schizotypy. *Neuropsychologia, 45*(4), 663-672. https://doi.org/10.1016/j.neuropsychologia.2006.07.017

Fleming, S. M. (2017). HMeta-d: hierarchical Bayesian estimation of metacognitive efficiency from confidence ratings. *Neurosci Conscious, 2017*(1), nix007. https://doi.org/10.1093/nc/nix007

Fletcher, P. C., & Frith, C. D. (2009, Jan). Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. *Nat Rev Neurosci, 10*(1), 48-58. https://doi.org/10.1038/nrn2536

Fletcher, P. C., & Honey, G. D. (2006, Apr). Schizophrenia, ketamine and cannabis: evidence of overlapping memory deficits. *Trends Cogn Sci, 10*(4), 167-174. https://doi.org/10.1016/j.tics.2006.02.008

Fonseca-Pedrero, E., Paino, M., Lemos-Giráldez, S., Vallina-Fernández, O., & Muñiz, J. (2010). ESQUIZO-Q: Un Instrumento para la Valoración del "Alto Riesgo Psicométrico" a la Psicosis. *Clínica y Salud, 21*(3), 255-269. https://doi.org/10.5093/cl2010v21n3a4

Fortgang, R. G., Srihari, V., & Cannon, T. D. (2020, May). Cognitive effort and amotivation in first-episode psychosis. *J Abnorm Psychol, 129*(4), 422-431. https://doi.org/10.1037/abn0000509

Freeman, D. (2016). Persecutory delusions: a cognitive perspective on understanding and treatment. *The Lancet Psychiatry, 3*(7), 685-692. https://doi.org/10.1016/s2215-0366(16)00066-3

Freeman, D., Loe, B. S., Kingdon, D., Startup, H., Molodynski, A., Rosebrock, L., Brown, P., Sheaves, B., Waite, F., & Bird, J. C. (2019, Nov 20). The revised Green et al., Paranoid Thoughts Scale (R-GPTS): psychometric properties, severity ranges, and clinical cut-offs. *Psychol Med, 49*(15), 1-10. https://doi.org/10.1017/S0033291719003155

Freeman, D., McManus, S., Brugha, T., Meltzer, H., Jenkins, R., & Bebbington, P. (2011, May). Concomitants of paranoia in the general population. *Psychol Med, 41*(5), 923-936. https://doi.org/10.1017/S0033291710001546

Fridberg, D. J., Brenner, A., & Lysaker, P. H. (2010, Aug 30). Verbal memory intrusions in schizophrenia: associations with self-reflectivity, symptomatology, and neurocognition. *Psychiatry Res, 179*(1), 6-11. https://doi.org/10.1016/j.psychres.2010.06.026

Frohlich, J., & Van Horn, J. D. (2014, Apr). Reviewing the ketamine model for schizophrenia. *J Psychopharmacol, 28*(4), 287-302. https://doi.org/10.1177/0269881113512909

Gallo, D. A. (2010, Oct). False memories and fantastic beliefs: 15 years of the DRM illusion. *Mem Cognit, 38*(7), 833-848. https://doi.org/10.3758/MC.38.7.833

Gawronski, B., & Bodenhausen, G. V. (2011). The associative-propositional evaluation model. In *Advances in Experimental Social Psychology* (Vol. 44, pp. 59-127). https://doi.org/10.1016/b978-0-12-385522-0.00002-0

Gray, J. A., Feldon, J., Rawlins, J. N. P., Hemsley, D. R., & Smith, A. D. (1991). The neuropsychology of schizophrenia. *Behav Brain Sci, 14*(1), 1-20. https://doi.org/https://doi.org/10.1017/S0140525X00065055

Hannula, D. E., & Ranganath, C. (2008, Jan 2). Medial temporal lobe activity predicts successful relational memory binding. *J Neurosci, 28*(1), 116-124. https://doi.org/10.1523/JNEUROSCI.3086-07.2008

Hegelstad, W. T. V., Kreis, I., Tjelmeland, H., & Pfuhl, G. (2020). Psychosis and Psychotic-Like Symptoms Affect Cognitive Abilities but Not Motivation in a Foraging Task. *Front Psychol, 11*, 1632. https://doi.org/10.3389/fpsyg.2020.01632

Heinrichs, R. W., & Vaz, S. M. (2004). Verbal memory errors and symptoms in schizophrenia. *Cog Behav Neurol, 17*(2), 98-101. https://doi.org/10.1097/01.wnn.0000116252.78804.73

Hemsley, D. R. (1993). A simple (or simplistic?) cognitive model for schizophrenia. *Behav Res Ther, 31*(7), 633-645. https://doi.org/https://doi.org/10.1016/0005-7967(93)90116-C

Hill, S. K., Bishop, J. R., Palumbo, D., & Sweeney, J. A. (2010, Jan). Effect of second-generation antipsychotics on cognition: Current issues and future challenges. *Expert Rev Neurother, 10*(1), 43-57. https://doi.org/10.1586/ern.09.143

Horga, G., & Abi-Dargham, A. (2019, Dec). An integrative framework for perceptual disturbances in psychosis. *Nat Rev Neurosci, 20*(12), 763-778. https://doi.org/10.1038/s41583-019-0234-1

Humpston, C. S., Linden, D. E. J., & Evans, L. H. (2017, Apr). Deficits in reality and internal source monitoring of actions are associated with the positive dimension of schizotypy. *Psychiatry Res, 250*, 44-49. https://doi.org/10.1016/j.psychres.2017.01.063

Ibanez-Casas, I., De Portugal, E., Gonzalez, N., McKenney, K. A., Haro, J. M., Usall, J., Perez-Garcia, M., & Cervilla, J. A. (2013). Deficits in executive and memory processes in delusional disorder: a case control study. *PLoS One, 8*(7), e67341. https://doi.org/10.1371/journal.pone.0067341

Jaspers, K. (1913/1997). *General psychopathology* (7th ed.). The University of Chicago Press.

Johnson, M. K., Hashtroudi, S., & Lindsay, D. S. (1993). Source monitoring. *Psychol Bull, 114*(1), 3-28. https://doi.org/10.1037/0033-2909.114.1.3

Kapur, S. (2003). Psychosis as a State of Aberrant Salience: A Framework Linking Biology, Phenomenology, and Pharmacology in Schizophrenia. *Am J Psychiatry, 160*(1), 13-23.

Kätzel, D., Wolff, A. R., Bygrave, A. M., & Bannerman, D. M. (2020). Hippocampal Hyperactivity as a Druggable Circuit-Level Origin of Aberrant Salience in Schizophrenia. *Front Pharmacol, 11*, 486811. https://doi.org/10.3389/fphar.2020.486811

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophr Bull, 13*(2), 261-276. https://doi.org/https://doi.org/10.1093/schbul/13.2.261

Keefe, R. S. E., Bilder, R. M., Davis, S. M., Harvey, P. D., Palmer, B. W., Gold, J. M., Meltzer, H. Y., Green, M. F., Capuano, G., Stroup, T. S., McEvoy, J. P., Swartz, M. S., Rosenheck, R. A., Perkins, D. O., Davis, C. E., Hsiao, J. K., & Lieberman, J. A. (2007). Neurocognitive effects of antipsychotic medications in patients with chronic schizophrenia

in the CATIE trial. *Arch Gen Psychiatry, 64*(6), 633-647.
https://doi.org/10.1001/archpsyc.64.6.633

Kiran, C., & Chaudhury, S. (2009, Jan). Understanding delusions. *Ind Psychiatry J, 18*(1), 3-18.
https://doi.org/10.4103/0972-6748.57851

Klein-Flugge, M. C., Wittmann, M. K., Shpektor, A., Jensen, D. E. A., & Rushworth, M. F. S.
(2019, Oct 23). Multiple associative structures created by reinforcement and incidental
statistical learning mechanisms. *Nat Commun, 10*(1), 4835.
https://doi.org/10.1038/s41467-019-12557-z

Koller, W. N., & Cannon, T. D. (2021, Jan 25). Paranoia is associated with impaired novelty
detection and overconfidence in recognition memory judgments. *J Abnorm Psychol,
130*(3), 273-285. https://doi.org/10.1037/abn0000664

Koller, W. N., & Cannon, T. D. (in revision). Paranoia is associated with reduced sensitivity to
preceding contextual cues during mnemonic discrimination.

Koller, W. N., Ongchoco, J. D. K., Bronstein, M. V., Scholl, B., & Cannon, T. D. (in prep).
Temporal source memory and paranoia.

Krabbendam, L., Myin-Germeys, I., Hanssen, M., Bijl, R. V., Graaf, R. d., Vollebergh, W., Bak,
M., & Os, J. v. (2004). Hallucinatory experiences and onset of psychotic disorder:
evidence that the risk is mediated by delusion formation. *Acta Psychol (Scand), 110*(4),
264-272. https://doi.org/10.1111/j.1600-0447.2004.00343.x

Kwapil, T. R., Gross, G. M., Silvia, P. J., Raulin, M. L., & Barrantes-Vidal, N. (2018, Mar).
Development and psychometric properties of the Multidimensional Schizotypy Scale: A
new measure for assessing positive, negative, and disorganized schizotypy. *Schizophr
Res, 193*, 209-217. https://doi.org/10.1016/j.schres.2017.07.001

Lally, J., & MacCabe, J. H. (2015, Jun). Antipsychotic medication in schizophrenia: a review. *Br
Med Bull, 114*(1), 169-179. https://doi.org/10.1093/bmb/ldv017

Laws, K. R., & Bhatt, R. (2005). False memories and delusional ideation in normal healthy
subjects. *Personality and Individual Differences, 39*(4), 775-781.
https://doi.org/10.1016/j.paid.2005.03.005

Lenzenweger, M. F. (2010). *Schizotypy and schizophrenia: The view from experimental
psychopathology*. Guilford Press.

Lenzenweger, M. F., & Dworkin, R. H. (1996). The dimensions of schizophrenia
phenomenology: Not one or two, at least three, perhaps four. *Br J Psychiatry, 168*(4),
432-440. https://doi.org/10.1192/bjp.168.4.432

Leucht, S., Arbter, D., Engel, R. R., Kissling, W., & Davis, J. M. (2009, Apr). How effective are
second-generation antipsychotic drugs? A meta-analysis of placebo-controlled trials. *Mol
Psychiatry, 14*(4), 429-447. https://doi.org/10.1038/sj.mp.4002136

Libby, L. A., Yonelinas, A. P., Ranganath, C., & Ragland, J. D. (2013, May 15). Recollection
and familiarity in schizophrenia: a quantitative review. *Biol Psychiatry, 73*(10), 944-950.
https://doi.org/10.1016/j.biopsych.2012.10.027

Liddle, P. F. (1987). The symptoms of chronic schizophrenia: A re-examination of the positive-
negative dichotomy. *Br J Psychiatry, 151*(2), 145-151.
https://doi.org/https://doi.org/10.1192/bjp.151.2.145

Liouta, E., Smith, A. D., & Mohr, C. (2008, Mar). Schizotypy and pseudoneglect: A critical
update on theories of hemispheric asymmetries. *Cogn Neuropsychiatry, 13*(2), 112-134.
https://doi.org/10.1080/13546800801936698

Lodge, D. J., & Grace, A. A. (2011, Sep). Hippocampal dysregulation of dopamine system function and the pathophysiology of schizophrenia. *Trends Pharmacol Sci, 32*(9), 507-513. https://doi.org/10.1016/j.tips.2011.05.001

Maher, B. A. (1999). Anomalous experience in everyday life: Its significance for psychopathology. *The Monist, 82*(4), 547-570. https://doi.org/10.5840/monist199982428

Maher, B. A. (2006). The relationship between delusions and hallucinations. *Curr Psychiatr Rep, 8*, 179-183. https://doi.org/10.1007/s11920-006-0021-3

Maniscalco, B., & Lau, H. (2012, Mar). A signal detection theoretic approach for estimating metacognitive sensitivity from confidence ratings. *Conscious Cogn, 21*(1), 422-430. https://doi.org/10.1016/j.concog.2011.09.021

Maren, S., & Holt, W. (2000). The hippocampus and contextual memory retrieval in Pavlovian conditioning. *Behav Brain Res, 110*(1-2), 97-108. https://doi.org/10.1016/S0166-4328(99)00188-6

Martinelli, C., & Shergill, S. S. (2015, Aug). Clarifying the role of pattern separation in schizophrenia: the role of recognition and visual discrimination deficits. *Schizophr Res, 166*(1-3), 328-333. https://doi.org/10.1016/j.schres.2015.06.004

Mason, O., & Claridge, G. (2006, Feb 28). The Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE): further description and extended norms. *Schizophr Res, 82*(2-3), 203-211. https://doi.org/10.1016/j.schres.2005.12.045

Mayes, A., Montaldi, D., & Migo, E. (2007, Mar). Associative memory and the medial temporal lobes. *Trends Cogn Sci, 11*(3), 126-135. https://doi.org/10.1016/j.tics.2006.12.003

Meyersburg, C. A., Bogdan, R., Gallo, D. A., & McNally, R. J. (2009, May). False memory propensity in people reporting recovered memories of past lives. *J Abnorm Psychol, 118*(2), 399-404. https://doi.org/10.1037/a0015371

Mishara, A. L. (2010, Jan). Klaus Conrad (1905-1961): delusional mood, psychosis, and beginning schizophrenia. *Schizophr Bull, 36*(1), 9-13. https://doi.org/10.1093/schbul/sbp144

Moritz, S., Kolbeck, K., & Andreou, C. (2016, Aug). Antipsychotics decrease response confidence. *J Psychopharmacol, 30*(8), 831-833. https://doi.org/10.1177/0269881116650404

Moritz, S., Silverstein, S. M., Dobo, T., Özaslan, Z., Zink, M., & Gallinat, J. (2021). Much of the neurocognitive impairment in schizophrenia is due to factors other Than schizophrenia itself: Implications for research and treatment. *Schizophrenia Bulletin Open, 2*(1). https://doi.org/10.1093/schizbullopen/sgaa034

Moritz, S., Woodward, T. S., Cuttler, C., Whitman, J. C., & Watson, J. M. (2004, Apr). False memories in schizophrenia. *Neuropsychology, 18*(2), 276-283. https://doi.org/10.1037/0894-4105.18.2.276

Moritz, S., Woodward, T. S., Jelinek, L., & Klinge, R. (2008, Jun). Memory and metamemory in schizophrenia: a liberal acceptance account of psychosis. *Psychol Med, 38*(6), 825-832. https://doi.org/10.1017/S0033291707002553

Moritz, S., Woodward, T. S., & Rodriguez-Raecke, R. (2006, May). Patients with schizophrenia do not produce more false memories than controls but are more confident in them. *Psychol Med, 36*(5), 659-667. https://doi.org/10.1017/S0033291706007252

Moritz, S., Woodward, T. S., & Ruff, C. C. (2003, Jan). Source monitoring and memory confidence in schizophrenia. *Psychol Med, 33*(1), 131-139. https://doi.org/10.1017/s0033291702006852

Moskowitz, A., Nadel, L., Watts, P., & Jacobs, W. J. (2008). Delusional Atmosphere, The Psychotic Prodrome and Decontextualized Memories. In *Psychosis, Trauma and Dissociation* (pp. 65-78). https://doi.org/10.1002/9780470699652.ch5

Musa, A., Khan, S., Mujahid, M., & El-Gaby, M. (2022). The shallow cognitive map hypothesis: A hippocampal framework for thought disorder in schizophrenia. *Schizophrenia, 8*(1). https://doi.org/10.1038/s41537-022-00247-7

O'Leary, D. S., Flaum, M., Kesler, M. L., Flashman, L. A., Arndt, S., & Andreasen, N. C. (2000). Cognitive correlates of the negative, disorganized, and psychotic symptom dimensions of schizophrenia. *J Neuropsychiatry Clin Neurosci, 12*(1), 4-15. https://doi.org/https://doi.org/10.1176/jnp.12.1.4

O'Reilly, R. C., & McClelland, J. L. (1994). Hippocampal conjunctive encoding, storage, and recall: avoiding a trade-off. *Hippocampus, 4*(6), 661-682.

Oertel, V., Kraft, D., Alves, G., Knochel, C., Ghinea, D., Storchak, H., Matura, S., Prvulovic, D., Bittner, R. A., Linden, D. E. J., Reif, A., & Stablein, M. (2019). Associative Memory Impairments Are Associated With Functional Alterations Within the Memory Network in Schizophrenia Patients and Their Unaffected First-Degree Relatives: An fMRI Study. *Front Psychiatry, 10*, 33. https://doi.org/10.3389/fpsyt.2019.00033

Ongchoco, J. D. K., Koller, W. N., Bronstein, M. V., Yates, T. S., Cannon, T. D., & Scholl, B. (in prep). The influence of perceived event segmentation on temporal memory is diminished for individuals high in paranoia

Ongür, D., Cullen, T. J., Wolf, D. H., Rohan, M., Ferreira, P., Zalesak, M., & Heckers, S. (2006). The neural basis of relational memory deficits in schizophrenia. *Arch Gen Psychiatry, 63*(4), 356-365. https://doi.org/10.1001/archpsyc.63.4.356

Peralta, V., Gil-Berrozpe, G. J., Librero, J., Sánchez-Torres, A., & Cuesta, M. J. (2020). The symptom and domain structure of psychotic disorders: A network analysis approach. *Schizophrenia Bulletin Open, 1*(1). https://doi.org/10.1093/schizbullopen/sgaa008

Perez, S. M., & Lodge, D. J. (2014). New approaches to the management of schizophrenia: focus on aberrant hippocampal drive of dopamine pathways. *Drug Des Devel Ther, 8*, 887-896. https://doi.org/10.2147/DDDT.S42708

Peters, E. R., Joseph, S. A., & Garety, P. A. (1999). Measurement of delusional ideation in the normal population: Introducing the PDI (Peters et al. Delusions Inventory). *Schizophr Bull, 25*(3), 553-576. https://doi.org/10.1093/oxfordjournals.schbul.a033401

Reichenberg, A., & Harvey, P. D. (2007, Sep). Neuropsychological impairments in schizophrenia: Integration of performance-based and brain imaging findings. *Psychol Bull, 133*(5), 833-858. https://doi.org/10.1037/0033-2909.133.5.833

Rodriguez-Ferreiro, J., Aguilera, M., & Davies, R. (2020). Positive Schizotypy Increases the Acceptance of Unpresented Materials in False Memory Tasks in Non-clinical Individuals. *Front Psychol, 11*, 262. https://doi.org/10.3389/fpsyg.2020.00262

Roediger, H. L., & McDermott, K. B. (1995). Creating false memories: Remembering words not presented in lists. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 21*(4), 803-814. https://doi.org/10.1037/0278-7393.21.4.803

Rolls, E. T., Loh, M., Deco, G., & Winterer, G. (2008, Sep). Computational models of schizophrenia and dopamine modulation in the prefrontal cortex. *Nat Rev Neurosci, 9*(9), 696-709. https://doi.org/10.1038/nrn2462

Rossi, R., Zammit, S., Button, K. S., Munafo, M. R., Lewis, G., & David, A. S. (2016). Psychotic experiences and working memory: A population-based study using signal-detection analysis. *PLoS One, 11*(4), 1-16. https://doi.org/10.1371/journal.pone.0153148

Rouhani, N., Norman, K. A., Niv, Y., & Bornstein, A. M. (2020, Oct). Reward prediction errors create event boundaries in memory. *Cognition, 203*, 104269. https://doi.org/10.1016/j.cognition.2020.104269

Sahakyan, L., & Kwapil, T. R. (2019, Aug). Hits and false alarms in recognition memory show differential impairment in positive and negative schizotypy. *J Abnorm Psychol, 128*(6), 633-643. https://doi.org/10.1037/abn0000441

Sahakyan, L., Kwapil, T. R., Lo, Y., & Jiang, L. (2019, Sep). Examination of relational memory in multidimensional schizotypy. *Schizophr Res, 211*, 36-43. https://doi.org/10.1016/j.schres.2019.07.031

Saunders, J., Randell, J., & Reed, P. (2012, Jun). Recall of false memories in individuals scoring high in schizotypy: memory distortions are scale specific. *J Behav Ther Exp Psychiatry, 43*(2), 711-715. https://doi.org/10.1016/j.jbtep.2011.10.005

Schaefer, J., Giangrande, E., Weinberger, D. R., & Dickinson, D. (2013, Oct). The global cognitive impairment in schizophrenia: consistent over decades and around the world. *Schizophr Res, 150*(1), 42-50. https://doi.org/10.1016/j.schres.2013.07.009

Schott, B. H., Voss, M., Wagner, B., Wustenberg, T., Duzel, E., & Behr, J. (2015). Fronto-limbic novelty processing in acute psychosis: disrupted relationship with memory performance and potential implications for delusions. *Front Behav Neurosci, 9*, 1-13. https://doi.org/10.3389/fnbeh.2015.00144

Schröder, J., Tittel, A., Stockert, A., & Karr, M. (1996). Memory deficits in subsyndromes of chronic schizophrenia. *Schizophr Res, 21*(1), 19-26. https://doi.org/https://doi.org/10.1016/0920-9964(96)00027-8

Seabury, R. D., Bearden, C. E., Ventura, J., Subotnik, K. L., Nuechterlein, K. H., & Cannon, T. D. (2021). Confident memory errors and disrupted reality testing in early psychosis. *Schizophr Res, 238*, 170-177. https://doi.org/10.1016/j.schres.2021.10.007

Seabury, R. D., & Cannon, T. D. (2020, Dec). Memory Impairments and Psychosis Prediction: A Scoping Review and Theoretical Overview. *Neuropsychol Rev, 30*(4), 521-545. https://doi.org/10.1007/s11065-020-09464-2

Sheffield, J. M., Karcher, N. R., & Barch, D. M. (2018, Dec). Cognitive deficits in psychotic disorders: A lifespan perspective. *Neuropsychol Rev, 28*(4), 509-533. https://doi.org/10.1007/s11065-018-9388-2

Silverstein, S. M., & Uhlhaas, P. J. (2004). Gestalt psychology: The forgotten paradigm in abnormal psychology. *The American Journal of Psyhology, 117*(2), 259-277.

Sinclair, A. H., Manalili, G. M., Brunec, I. K., Adcock, R. A., & Barense, M. D. (2021, Dec 21). Prediction errors disrupt hippocampal representations and update episodic memories. *Proc Natl Acad Sci U S A, 118*(51). https://doi.org/10.1073/pnas.2117625118

Smeets, F., Lataster, T., Dominguez, M. D., Hommes, J., Lieb, R., Wittchen, H. U., & van Os, J. (2012, May). Evidence that onset of psychosis in the population reflects early hallucinatory experiences that through environmental risks and affective dysregulation become complicated by delusions. *Schizophr Bull, 38*(3), 531-542. https://doi.org/10.1093/schbul/sbq117

Spitzer, M. (1990). On defining delusions. *Compr Psychiatry, 31*(5), 377-397. https://doi.org/10.1016/0010-440X(90)90023-L

Stanislaw, H., & Todorov, N. (1999). Calculation of signal detection theory measures. *Behav Res Methods, 31*(1), 137-149. https://doi.org/https://doi.org/10.3758/BF03207704

Stefanis, N., Hanssen, M., Smirnis, N., Avramopoulos, Evdokimidis, I., Verdoux, H., Osl, J. v., & Stefanis, C. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychol Med*(347-58), 347-358. https://doi.org/10.1017/s0033291701005141

Stip, E., Corbiere, M., Boulay, L. J., Lesage, A., Lecomte, T., Leclerc, C., Ricard, N., Cyr, M., & Guillem, F. (2007, Mar). Intrusion errors in explicit memory: their differential relationship with clinical and social outcome in chronic schizophrenia. *Cogn Neuropsychiatry, 12*(2), 112-127. https://doi.org/10.1080/13546800600809401

Strauss, J. S. (1969). Hallucinations and delusions as points on continua function. *Arch Gen Psychiatry, 21*(5), 581-586. https://doi.org/10.1001/archpsyc.1969.01740230069010

Sugimori, E., & Tanno, Y. (2010, Apr 1). Effects of positive and negative delusional ideation on memory. *Int J Psychol, 45*(2), 90-101. https://doi.org/10.1080/00207590903030287

Tamminga, C. A., Southcott, S., Sacco, C., Wagner, A. D., & Ghose, S. (2012, Sep). Glutamate dysfunction in hippocampus: relevance of dentate gyrus and CA3 signaling. *Schizophr Bull, 38*(5), 927-935. https://doi.org/10.1093/schbul/sbs062

Tamminga, C. A., Stan, A. D., & Wagner, A. D. (2010). The hippocampal formation in schizophrenia. *Am J Psychiatry, 167*(10), 1178-1193.

Tanno, Y., Ishigaki, T., & Sugiura, Y. (2000). Construction of scales to measure thematic tendencies of paranoid ideation. *Jpn J Psychol, 71*(5), 379-386. https://doi.org/10.4992/jjpsy.71.379

Thoresen, C., Endestad, T., Sigvartsen, N. P., Server, A., Bolstad, I., Johansson, M., Andreassen, O. A., & Jensen, J. (2014). Frontotemporal hypoactivity during a reality monitoring paradigm is associated with delusions in patients with schizophrenia spectrum disorders. *Cogn Neuropsychiatry, 19*(2), 97-115. https://doi.org/10.1080/13546805.2013.776495

Treves, A., & Rolls, E. T. (1994). Computational analysis of the role of the hippocampus in memory. *Hippocampus, 4*(3), 374-391. https://doi.org/10.1002/hipo.450040319

Turk-Browne, N. B. (2019, Dec). The hippocampus as a visual area organized by space and time: A spatiotemporal similarity hypothesis. *Vision Res, 165*, 123-130. https://doi.org/10.1016/j.visres.2019.10.007

Uhlhaas, P. J., & Mishara, A. L. (2007, Jan). Perceptual anomalies in schizophrenia: integrating phenomenology and cognitive neuroscience. *Schizophr Bull, 33*(1), 142-156. https://doi.org/10.1093/schbul/sbl047

van Os, J., Hanssen, M., V.Bijl, R., & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res, 45*(1-2), 11-20. https://doi.org/10.1016/S0920-9964(99)00224-8

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009, Feb). A systematic review and meta-analysis of the psychosis continuum: Evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychol Med, 39*(2), 179-195. https://doi.org/10.1017/S0033291708003814

van Os, J., & Reininghaus, U. (2016). Psychosis as a transdiagnostic and extended phenotype in the general population. *World Psychiatry, 15*(2), 118-124. https://doi.org/10.1002/wps.20310

Ventura, J., Thames, A. D., Wood, R. C., Guzik, L. H., & Hellemann, G. S. (2010, Aug). Disorganization and reality distortion in schizophrenia: a meta-analysis of the

relationship between positive symptoms and neurocognitive deficits. *Schizophr Res, 121*(1-3), 1-14. https://doi.org/10.1016/j.schres.2010.05.033

Waters, F. (2004). Context memory and binding in schizophrenia. *Schizophrenia Research, 68*(2-3), 119-125. https://doi.org/10.1016/s0920-9964(03)00221-4

Weickert, T. W., Goldberg, T. E., Marenco, S., Bigelow, L. B., Egan, M. F., & Weinberger, D. R. (2003, Aug). Comparison of cognitive performances during a placebo period and an atypical antipsychotic treatment period in schizophrenia: critical examination of confounds. *Neuropsychopharmacology, 28*(8), 1491-1500. https://doi.org/10.1038/sj.npp.1300216

Winterstein, B. P., Silvia, P. J., Kwapil, T. R., Kaufman, J. C., & Reiter-Palmon, R. (2011). Brief assessment of schizotypy: Developing short forms of the Wisconsin Schizotypy Scales. *Person Individ Diff, 51*(8), 920-924. https://doi.org/10.1016/j.paid.2011.07.027

Winton-Brown, T., Schmidt, A., Roiser, J. P., Howes, O. D., Egerton, A., Fusar-Poli, P., Bunzeck, N., Grace, A. A., Duzel, E., Kapur, S., & McGuire, P. (2017, Oct 3). Altered activation and connectivity in a hippocampal-basal ganglia midbrain circuit during salience processing in subjects at ultra high risk for psychosis. *Transl Psychiatry, 7*(10), e1245. https://doi.org/10.1038/tp.2017.174

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of research. *Journal of Memory and Language, 46*(3), 441-517. https://doi.org/10.1006/jmla.2002.2864

Zammit, S., Kounali, D., Cannon, M., David, A. S., Gunnell, D., Heron, J., Jones, P. B., Lewis, S. n., Sullivan, S., Wolke, D., & Lewis, G. (2013). Psychotic experiences and psychotic disorders at age 18 in relation to psychotic experiences at age 12 in a longitudinal population-based cohort study. *Am J Psychiatry, 170*(7), 742-750. https://doi.org/10.1176/appi.ajp.2013.12060768

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Contributors
Author WNK and TDC devised the review concept. Author WNK performed the literature review and wrote the first draft of the manuscript. Author TDC provided critical feedback and approved the final manuscript.

Conflict of Interest
We have no conflict of interest to disclose.

Highlights

- Delusionality associates with false alarms, source memory biases, and metamemory deficits.
- Several of these effects may stem from breakdowns in the associative memory system.
- In psychotic states, trait memory biases may fuel high-salience/low-source thought.