Imagination in human social cognition, autism, and psychotic-affective conditions

Article *in* Cognition · May 2016

DOI: 10.1016/j.cognition.2016.02.001

5 authors, including:

Bernard J Crespi
Simon Fraser University
217 PUBLICATIONS **10,294** CITATIONS
SEE PROFILE

Natalie Dinsdale
Simon Fraser University
8 PUBLICATIONS **134** CITATIONS
SEE PROFILE

Peter L Hurd
University of Alberta
75 PUBLICATIONS **1,849** CITATIONS
SEE PROFILE

Cognition 150 (2016) 181–199

Contents lists available at ScienceDirect

Cognition

journal homepage: www.elsevier.com/locate/COGNIT

Imagination in human social cognition, autism, and psychotic-affective conditions

Bernard Crespi [a,*], Emma Leach [a], Natalie Dinsdale [a], Mikael Mokkonen [a,b], Peter Hurd [c]

[a] Department of Biological Sciences, Simon Fraser University, 8888 University Drive, Burnaby, British Columbia V5A 1S6, Canada
[b] Department of Biological and Environmental Science, P.O. Box 35, 40014 University of Jyväskylä, Finland
[c] Department of Psychology, and Centre for Neuroscience, University of Alberta, 116 St. and 85 Avenue, Edmonton, Alberta T6G 2R3, Canada

ARTICLE INFO

Article history:
Received 1 June 2015
Revised 1 February 2016
Accepted 3 February 2016
Available online 18 February 2016

Keywords:
Imagination
Autism
Schizophrenia
Creativity
Polygenic risk score
Autism Spectrum Quotient

ABSTRACT

Complex human social cognition has evolved in concert with risks for psychiatric disorders. Recently, autism and psychotic-affective conditions (mainly schizophrenia, bipolar disorder, and depression) have been posited as psychological 'opposites' with regard to social-cognitive phenotypes. Imagination, considered as 'forming new ideas, mental images, or concepts', represents a central facet of human social evolution and cognition. Previous studies have documented reduced imagination in autism, and increased imagination in association with psychotic-affective conditions, yet these sets of findings have yet to be considered together, or evaluated in the context of the diametric model. We first review studies of the components, manifestations, and neural correlates of imagination in autism and psychotic-affective conditions. Next, we use data on dimensional autism in healthy populations to test the hypotheses that: (1) imagination represents the facet of autism that best accounts for its strongly male-biased sex ratio, and (2) higher genetic risk of schizophrenia is associated with higher imagination, in accordance with the predictions of the diametric model. The first hypothesis was supported by a systematic review and meta-analysis showing that Imagination exhibits the strongest male bias of all Autism Quotient (AQ) subscales, in non-clinical populations. The second hypothesis was supported, for males, by associations between schizophrenia genetic risk scores, derived from a set of single-nucleotide polymorphisms, and the AQ Imagination subscale. Considered together, these findings indicate that imagination, especially social imagination as embodied in the default mode human brain network, mediates risk and diametric dimensional phenotypes of autism and psychotic-affective conditions.

'Autism means a withdrawal into fantasy but this is not what happens in the syndrome of autism. The schizophrenic may retreat from reality into fantasy, but the autistic child does not retreat, rather he fails to develop social relationships-a crucial distinction. Furthermore, so far as one can tell, the young autistic child has a deficiency of fantasy rather than an excess.'

[Sir Michael Rutter, 1972, p. 327]

1. Introduction

Psychiatric conditions provide unique insights into the evolution, adaptive significance and genetic underpinnings of human cognition and behavior because they reflect specifiable perturbations to normally developing and functioning neurological systems (Crespi & Leach, 2015). Disorders that involve human social and self-reflexive cognition, and human imagination and creativity, are of particular interest, given that evolution along the human lineage has involved selective expansion and elaboration of the regions of higher-order association cortex subserving these functions (Buckner & Krienen, 2013; Rakic, 2009; Saxe, 2006). How, then, might social and imaginative cognition be associated with psychiatric disorders?

Two sets of disorders, the autism spectrum and the psychotic-affective spectrum, most centrally, across all psychiatric conditions, involve alterations to human sociality and imagination. The autism spectrum historically includes autism and Asperger syndrome; it usually presents from early childhood with some combination of social and communication deficits plus the presence of restricted interests and repetitive behavior (Lord & Bishop, 2015). By contrast, psychotic-affective conditions include schizophrenia, schizotypal personality disorder, bipolar disorder, depression,

* Corresponding author.
E-mail addresses: crespi@sfu.ca (B. Crespi), eleach@sfu.ca (E. Leach), nld670@mail.usask.ca (N. Dinsdale), mika@sfu.ca (M. Mokkonen), phurd@ualberta.ca (P. Hurd).

http://dx.doi.org/10.1016/j.cognition.2016.02.001
0010-0277/

borderline personality disorder, and dissociative disorders (and some related conditions), all of which overlap broadly in their symptoms and correlates, which may include, among other signs, some combination of psychosis (reality distortion), overly negative or positive (and dysregulated) mood, and dissociation (reduced integration of cognitive functions with regard to self, surroundings, memory and perception) (Balaratnasingam & Janca, 2015; Moskowitz & Heim, 2011). These disorders most commonly exhibit onset in adolescence or young adulthood, such that childhood development is completed relatively normally, although it is more likely than usual to have involved adverse social-environmental circumstances.

Kanner (1943) described nonsocial, repetitive and non-symbolic play as being central features of autistic behavior among his cohort of study children. Reductions and alterations to pretend play in autism are interesting because they span its two major dimensions, sociality and repetitive activities, whose association may otherwise remain unclear. Moreover, reduced imagination represents an important criterion for autism diagnosis (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Lord, Leventhal, & Cook, 2001; Rutter, Le Couteur, & Lord, 2003), and pretend play, and its generalization in ongoing development of the imagination (Vygotsky, 2004), represent fundamental aspects of human childhood. As such, decreases and changes to imagination hold promise as potentially-foundational aspects of autism, that may help to explain its diverse manifestations, correlates and causes, as well as directing attention to effective therapies (e.g., Woodard, Chung, & Korn, 2014; Woodard & Van Reet, 2011).

What is especially striking about alterations to imagination, in the context of its reductions in autism, is that in psychotic-affective conditions, aspects of imagination are not reduced but, instead, tend to be increased (reviews in Burns, 2007; Jamison, 1993; Kaufman, 2014; Kyaga, 2014; Nettle, 2001). Such positive associations of imagination with psychotic-affective conditions have been particularly well-documented with regard to schizophrenia, schizotypy, bipolar disorder, and depression, where individuals with milder forms and symptoms of these conditions, and close relatives of individuals with such disorders, consistently exhibit evidence of higher levels of creativity, divergent thinking, and imaginative cognition, as evidenced by diverse lines of information (e.g., Jamison, 1993; Kyaga et al., 2013; Nettle, 2001; Ruiter & Johnson, 2015; Zabelina, O'Leary, Pornpattananangkul, Nusslock, & Beeman, 2015). How, then, is imagination related to the psychological dysfunctions typical of these conditions, and what can the study of imagination tell us about the similarities and differences between autism spectrum conditions and psychotic-affective conditions (especially schizophrenia), a topic of recent increasing interest (e.g., Ciaramidaro et al., 2015; Currie, 2000; Dinsdale, Hurd, Wakabayashi, Elliot, & Crespi, 2013; Hommer & Swedo, 2015)? A primary hypothesis that we address in this context is that autism spectrum conditions, and psychotic-affective conditions, can be regarded as psychological 'opposites' to one another with regard to expressions and correlates of imagination. This hypothesis was developed by Jung (2014), who described how autism is characterized by deductive, explicit and convergent thinking that favors intelligence-mediated solving of useful problems, whereas psychosis involves abstracted, metaphorical, and divergent thinking, that mediates implicit, novel and creativity-mediated problem resolution.

In this paper we first present a simple, useful definition of imagination, and describe how it is instantiated in neurological systems. Second, we provide an overview of the extensive literatures on imagination in autism, and in psychotic-affective conditions, to evaluate the hypothesis that autism spectrum conditions and psychotic-affective conditions represent diametric (opposite) disorders (Crespi & Badcock, 2008; Jung, 2014) with regard to this core feature of human cognition. Third, we evaluate central aspects of this hypothesis using two lines of evidence: (1) data on dimensional measures of autism in healthy populations (the Autism Spectrum Quotient; Baron-Cohen et al., 2001), to evaluate gender biases in its Imagination subscale compared to other subscales, and (2) data on schizophrenia genetic risk derived from single nucleotide polymorphisms, to test for an association between genetic risk scores and imagination as quantified by the Autism Spectrum Quotient.

1.1. Imagination and its neural instantiations

The term 'imagination' is considered here as 'the faculty or action of forming new ideas, or images or concepts of external objects not present to the senses, typically derived from creative integration of past experiences, learning, or other information' (adapted from the New Oxford American Dictionary, Jewell & Abate, 2001, page 848). Imagination is conceptually most-closely related to creativity, generativity, divergent thinking, narrative production, and theory of mind, as well as to pretend play in children (Fig. 1). Production of novelty through imagination thus takes place through deriving elements of verbal or visual thought from perception and memory and combining them in new ways. Creativity can be distinguished from imagination in that it requires usefulness of the creative construct, as well as originality. Thus, for example, Stein (1953, page 311) defined a creative achievement as "a novel work that is accepted as tenable or useful or satisfying by a group in some point in time". It is important to distinguish carefully between imagination and creativity because their neural bases and psychological correlates are expected to differ to some as yet unknown degree.

Imagination can involve thinking in words and inner speech, and their condensed semiotic mental forms (Vygotsky, 2012), or thinking in pictures that are more or less reality-based (Pearson, Deeprose, Wallace-Hadrill, Heyes, & Holmes, 2013). Thinking in words, and thinking in pictures, appear to be inversely associated with one another, at least during episodes of 'mind wandering' (Stawarczyk, Cassol, & D'Argembeau, 2013). Imagination commonly exhibits self-oriented, social, and emotional content, whereby possible future scenarios are played out in the mind, or past events are processed for potential current and future salience (Schacter et al., 2012). In this regard, episodic memory and future thinking are closely associated with one another, as future mental

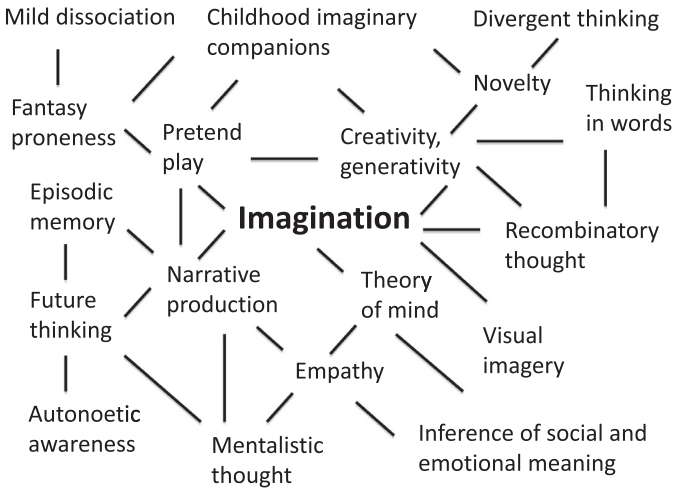

Fig. 1. Imagination, in neurotypical cognition, can be conceptualized in terms of a set of related phenomena that relate to core functions of the human brain.

'time travel' derived its components from memories of past events (Suddendorf, Addis, & Corballis, 2009).

Imagination can be conceptualized as an abstract construct semantically and psychologically, but its neural basis, predominantly instantiated in the brain's default mode network and interacting areas, is increasingly well-understood, and indeed imagination, in the context of reflexive social and self-oriented cognition, has been considered as a primary function of this key brain system (Buckner, Andrews-Hanna, & Schacter, 2008; Schilbach, Eickhoff, Rotarska-Jagiela, Fink, & Vogeley, 2008; Spreng & Grady, 2010). The default mode network represents a set of brain regions, including medial prefrontal cortex, posterior cingulate cortex, inferior parietal cortex (including regions of the temporal-parietal junction), lateral temporal cortex, and the hippocampal formation, that are preferentially coactivated during stimulus-independent thought (Agnati, Guidolin, Battistin, Pagnoni, & Fuxe, 2013; Buckner et al., 2008; Schacter et al., 2012). Thought process domains activating these regions include self-referential thought, autobiographical and other episodic memory, theory of mind, thinking about the future with regard to one's goals and possible scenarios, social scenario-building and evaluation, construction of mental 'scenes', 'mind-wandering' and 'day-dreaming' (Andrews-Hanna, Reidler, Huang, & Buckner, 2010; Andrews-Hanna, Smallwood, & Spreng, 2014; McMillan, Kaufman, & Singer, 2013; Stawarczyk & D'Argembeau, 2015) (Fig. 2). These domains share in common self-contextualized, or third-party based, self- and socially-relevant memory and imagination, with imagination based in part on inner speech and dependent on recruitment of its components from autobiographical and other episodic memory.

Activation and functional connectivity of the default mode network have thus been positively associated with the quality of remembering the past and imagining the future, both of which entail imaginative mental simulation of alternative spatial-temporal autonoetic (self in time) perspectives (Østby et al., 2012; Suddendorf & Corballis, 2007). Guided by reviews that described causes of high heterogeneity in previous analyses of the neural bases of creativity (Arden, Chavez, Grazioplene, & Jung, 2010; Dietrich & Kanso, 2010; Sawyer, 2011), more recent studies have begun to more-consistently localize imaginative, creative and divergent-thinking tasks to relatively-specific brain areas. Thus, for example, fMRI activation of default mode regions, and connectivity with other regions such as the left inferior frontal gyrus (IFG), have in recent studies consistently been linked with performance in creative and divergent thinking tasks (Beaty et al., 2014; Fink, Slamar-Halbedl, Unterrainer, & Weiss, 2012; Fink et al., 2010; Mayseless, Eran, & Shamay-Tsoory, 2015; Takeuchi et al., 2012), implicating these areas in aspects of imagination and creativity. More generally, Jung, Mead, Carrasco, and Flores (2013) described how the default mode network, though its interconnected hubs and links with other systems, demonstrates as a 'first approximation' all of the properties necessary for serving as a key brain system underlying creative cognition.

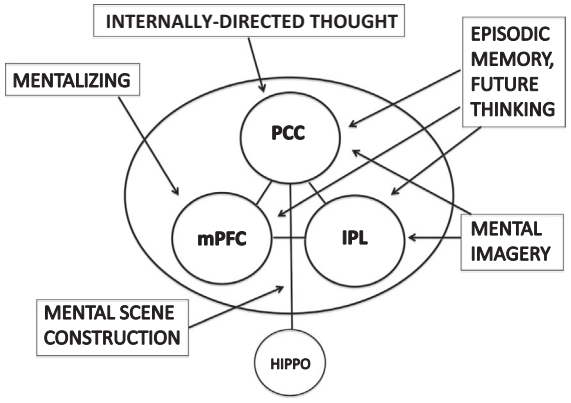

Fig. 2. The human default mode system comprises three main interacting hub regions, the posterior cingulate cortex (PCC) (including the precuneus), the medial prefrontal cortex (mPFC), and the inferior parietal lobe (IPL) (including the temporal-parietal junction), with core functions as depicted. HIPPO is the hippocampus. Arrows to specific regions represent documented functions of the region. The precise regions included in the default mode network, their specific functions, and their mediation of components of human imagination, remain active areas of research, but the primary roles of this network are well established.

Considered together, these findings present notable evidence that the default mode network represents a primary nexus of neural activity underlying imagination, given the important role of imagination in creativity. In contrast to, or in combination with, the canonical default mode, imagination that includes mental imagery recruits temporal, parietal and occipital visual brain regions that also subserve visual perception and visual memory (Lagioia, Van De Ville, Debbané, Lazeyras, & Eliez, 2010; Pearson & Kosslyn, 2015; Slotnick, Thompson, & Kosslyn, 2012). Regions activated during mental imagery thus overlap broadly with regions used for visual perception (Dentico et al., 2014; Ganis, Thompson, & Kosslyn, 2004), with mental scene construction also activating hippocampal regions (Fig. 2) (Andrews-Hanna et al., 2010; Raffard, D'Argembeau, Bayard, Boulenger, & Van der Linden, 2010). This mental imagery system is directly connected with regions of the default system, although the functional and activational associations between these two sets of regions remain largely unstudied. Mental imagery can thus be considered as a facet of imagination that involves a specific modality and system of neural instantiation, that is functionally associated with the default mode to some degree.

1.2. Imagination and the default mode in autism and psychotic-affective spectrum conditions

Analyses of imagination in autism, and in psychotic-affective conditions, have thus far been conducted in virtually complete isolation from one another. Tables 1–9 summarize comparisons and contrasts between these two sets of conditions for nine major aspects and correlates of imagination: (1) pretend play, (2) creativity and generativity, (3) narrative formation and comprehension, (4) mentalizing and empathizing, (5) meaning and salience, (6) episodic memory and future thinking, (7) mental imagery, (8) sensory abilities, and (9) neural activation and connectivity. Details and citations are provided in the Table entries, and we summarize here the major points.

1.2.1. Pretend play

Pretend play, and social play, are notably reduced in children with autism (review in Jarrold, 2003), and in such children repetitive behavior tends to take the place of pretend and social play (Honey, Leekam, Turner, & McConachie, 2007; Wing & Gould, 1979). The fact that symbolic, pretend and social play can be prompted and guided in children with autism means that they are capable of engaging in it, at least to some degree, although they have reduced motivation and interest in such activities (Table 1).

Especially intense pretend play has been reported among children with dissociative disorders, and in children with imaginary companions; mild dissociation can indeed be considered as a prerequisite for imaginary social play, and higher dissociation in neurotypical children has been associated in some studies with better theory of mind skills and higher creativity (Table 1). Higher levels of childhood dissociation (often mediated by trauma), and the presence of imaginary companions, have, however, also been linked with hallucination, paranoia, borderline personality symp-

Table 1
Contrasts in pretend play between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Pretend play	Pretend play reduced on autism spectrum, though can be prompted; play usually solitary, repetitive, and focused on objects (Blanc, Adrien, Roux, & Barthélémy, 2005; Jarrold, 2003; Wolfberg, 2009) Repetitive behavior 'takes place of' play' in children with autism (Honey et al., 2007; Wing & Gould, 1979)	Children with dissociative disorders have especially intense play (McLewin & Muller, 2006; Putnam, Helmers, & Trickett, 1993; Putnam & Peterson, 1994) Children or adolescents with imaginary companions exhibit: (1) more vivid auditory and visual imagery (Gleason, Jarudi, & Cheek, 2003), (2) increased tendency to hallucinate (Feelgood & Rantzen, 1994; Fernyhough, Bland, Meins, & Coltheart, 2007; Pearson et al., 2001), (3) higher scores on measures of dissociation (Carlson, Tahiroglu, & Taylor, 2008; Dierker, Davis, & Sanders, 1995), (4) higher levels of paranoia, and psychoticism (Bonne, Canetti, Bachar, De-Nour, & Shalev, 1999) and (5) preference for fantasy over reality-based toys (Acredolo, Goodwyn, & Fulmer, 1995) Dissociation (as 'splitting') is an important feature of schizophrenia, schizotypy, and borderline personality; it is commonly associated with hallucinations, and with trauma and abuse in childhood (Bob & Mashour, 2011; Giesbrecht, Merckelbach, Kater, & Sluis, 2007; Korzekwa, Dell, Links, Thabane, & Fougere, 2009; Schacter et al., 2012) Children high in fantasy proneness exhibit more paranoid and borderline symptoms, utilize mental health services more (Waldo & Merritt, 2000)	Imaginary companions present in up to 65% of children; incidence is higher in girls than boys (Carlson et al., 2008) Imaginary play conceptualized as involving mild form of dissociation (McElroy, 1992; Shirar, 1996) In healthy children, adolescents, higher dissociation associated with increased creativity (Hoff, 2005; Seiffge-Krenke, 1997) and better theory of mind skills (Carlson, Mandell, & Williams, 2004; Taylor & Carlson, 1997)

toms, fantasy-prone thinking, and psychoticism (Table 1). Among adolescents and adults, dissociation indeed represents an important feature of schizophrenia, schizotypy, and borderline personality, one that apparently also predisposes to higher imagination and creativity (e.g., Blum, 2013; Thomson and Jaque, 2012).

Taken together, these studies suggest that imaginary companions, highly fantasy-prone play, and dissociation are associated with higher imagination on the psychotic-affective spectrum, primarily among individuals who are not subject to severe psychopathological effects. Notably, childhood imaginary companions appear to represent an 'opposite' psychological phenomenon to the deficits of social and pretend play that are characteristic of autism. Their links with imagination, creativity, and psychotic-affective phenotypes in adulthood would benefit from further study. The comparison of autism with psychotic-affective conditions with regard to pretend play has not been made previously, and should provide novel insights into how these two sets of conditions are related to one another.

1.2.2. Creativity and generativity

Creativity and generativity are reduced in autism, in the contexts of difficulties in imagining unreal, novel things, insistence on sameness, and over-selective focus on specific non-social aspects of the environment most commonly involving reality-based, deterministic, systems (Table 2). Autistic cognition thus tends to involve convergent thinking and structure-guided pattern recognition, which can be creative and combinatorial primarily or only within a narrow range of restricted interests.

Extensive bodies of research have established links of creativity and generativity with relatively mild phenotypes and symptoms on the psychotic-affective spectrum, such that increased divergent thinking, magical thinking, openness, novelty-seeking, and goal-seeking, as well as reduced latent inhibition (more diffuse and distractable attention) can foster enhanced creative accomplishments, at least among relatively high-functioning individuals (Table 2). Under a 'shared vulnerability' model (Carson, 2011), creativity and psychotic-affective conditions thus partially overlap with respect to neurological and psychological traits that contribute to both. However, in more-extreme form, or when combined with deleterious phenotypes such as childhood trauma, creativity-promoting phenotypes can also increase liability to psychotic-affective disorders.

Autism and psychotic-affective conditions appear to exhibit directly-opposite patterns with regard to convergent versus divergent thinking, and restricted interests and insistence on sameness versus novelty, fantasy, goal-seeking in mania, and openness. Despite these considerations, both divergent and convergent thinking are required for the generation of useful creative constructs (e.g., Cropley, 2006), and as such it is their interactions that should most directly mediate their production.

1.2.3. Narrative and the arts

Studies of story writing and comprehension in autistic individuals show that they exhibit reduced capacities in both areas, especially with regard to social themes, fantasy, conceptual novelty, and acquiring the 'big picture' of a narrative as opposed to local details (Table 3). Comparably, art by individuals with autism tends to be either reality based (e.g., highly realistic drawing), or it derives from rule-based systems for filling space. Autism is indeed associated, in high-functioning individuals and in relatives of people with autism, with technical professions, such as engineering and science, rather than with the humanities, literature or arts (Table 3).

On the psychotic-affective spectrum, individuals with symptoms or diagnoses of schizotypy, schizophrenia, bipolar disorder, and depression demonstrate clear tendencies toward production of poetry, fictional, socially-oriented literature, and abstract and symbolic visual and creative arts (Table 3). Relatively-severe psychotic-affective conditions can involve, however, an extreme of such phenotypes in the form of hallucinations, delusions and

Table 2
Contrasts in creativity and generativity between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Creativity & generativity	Creativity, generativity reduced in autism (Dichter, Lam, Turner-Brown, Holtzclaw, & Bodfish, 2009; Harris & Leevers, 2000; Jarrold, Boucher, & Smith, 1996); specific impairment in imagining unreal things (Scott & Baron-Cohen, 1996) Insistence on sameness represents major feature of autism (Lam, Bodfish, & Piven, 2008; Richler, Huerta, Bishop, & Lord, 2010; Van de Cruys et al., 2014) Creativity, imagination in autism are reality-based (Craig & Baron-Cohen, 1999; Craig et al., 2000); structure-guided (Mottron, Dawson, & Soulières, 2009), and focused on specific areas of non-social interest (Liu, Shih, & Ma, 2011; Ten Eycke and Müller, 2015) Attention in autism highly focused on specific non-social aspects of environment; difficulties in switching attention (Ploog, 2010)	Increased divergent thinking, creativity, generativity especially in association with positive schizotypy, hypomania, bipolar disorder, schizophrenia, depression (Barrantes-Vidal, 2004; Fink et al., 2014; Fisher et al., 2004; Folley & Park, 2005; Jamison, 1993; Kaufman, 2014; Kaufman & Paul, 2014; Lauronen et al., 2004; Murray & Johnson, 2010; Nettle, 2001; Ruiter & Johnson, 2015; Verhaeghen, Joorman, & Aikman, 2014; Zabelina et al., 2015) Increased openness, novelty-seeking associated with positive schizotypy, mania, hypomania, and dopaminergic neurotransmission (Carson, 2011; DeYoung, 2013) Creativity and psychopathological symptoms on psychotic-affective spectrum are centered mainly on people and other animate beings (e.g., Collerton, Perry, & McKeith, 2005; Crespi & Badcock, 2008); creativity is mainly fantasy-based and social-imagination based, as in magical ideation (Brugger & Mohr, 2008) More-diffuse attention, higher distractibility, loose associations, reduced latent inhibition, in schizophrenia schizotypy, bipolar disorder and depression; these traits may increase creativity (Abraham, Windmann, Daum, & Güntürkün, 2005; Foster et al., 2011; MacQueen, Galway, Goldberg, & Tipper, 2003) Overinclusiveness characteristic of schizophrenia, schizotypy and bipolar disorder, involves inability to exclude irrelevant stimuli, may increase creativity (Andreasen & Powers, 1975; Murray & Johnson, 2010; Ottemiller, Elliott, & Giovannetti, 2014)	Creativity involves both novelty and appropriateness; novelty is higher, but appropriateness is lower, with increased level of psychotic-affective psychopathology and lower level of executive function and convergent thinking (e.g., Carson, 2011; Fisher, Heller, & Miller, 2013; Ottemiller et al., 2014 Creativity, imagination and psychotic-affective psychopathology exhibit partially-overlapping causes (Carson, 2011), such that creativity and imagination are higher in association with mild psychotic-affective phenotypes, having close relatives with disorders, and being between episodes of psychosis or depression (Carson, 2011; Kyaga et al., 2013)

confabulations that commonly take narrative, 'social story', and mentalistically-based forms (e.g., Currie & Jureidini, 2003), even when personal narrative identity is reduced (e.g., in schizophrenia; Raffard, D'Argembeau, Lardi, et al., 2010).

Social narrative production and appreciation, and technical compared to literary and artistic professions, represent striking contrasts between the autism spectrum and psychotic-affective spectrum, that bear directly on reductions and enhancements of social imagination. In this context, narrative production is especially important because it connects imagination with socially-based creativity, mental imagery, autobiographical memory, future thinking, and mentalizing more generally.

1.2.4. Mentalizing

Mentalizing is considered here as cognition focused on the social world, and the drive to understand and infer and share the thoughts, feelings and intentions of other individuals. On the autism spectrum, mentalizing and empathizing are notably underdeveloped, in association with reduced skills on tasks that involve theory of mind (review in Baron-Cohen, 2009). Autistic individuals, to the extent that they indeed exhibit 'mind-blindness', thus tend to under-mentalize (use fewer mental state attributions than typical) in social interactions and stimulus-independent thought (Table 4).

The psychotic-affective spectrum is also characterized by deficits in mentalizing, except for a suite of studies that demonstrates enhanced cognitive empathy skills (better than matched controls), among females with borderline personality disorder or non-clinical depression (Dinsdale & Crespi, 2013; Harkness, Washburn, Theriault, Lee, & Sabbagh, 2011) (Table 4). However, among individuals with schizophrenia or borderline personality disorder, individuals high in schizotypy, and individuals exhibiting psychotic experiences, deficits in mentalizing often involve 'hyper-mentalizing', whereby mental states are incorrect because they are over-attributed, without objective justification. Hyper-mentalizing thus involves increased, but incorrectly applied, use of social imagination, commonly involving negatively-valenced attributions (except in mania and hypomania, where overly-positive appraisals can predominate), in association with positive symptoms of psychotic-affective conditions such as paranoia, delusions, hallucinations, jumping to conclusions, and black-or-white thinking.

The contrast between under-mentalizing in autism and over-mentalizing in some psychotic-affective symptoms and conditions demonstrates how social imagination can differ diametrically between the two sets of conditions. In this context, social imagination links directly with theory of mind, empathy, narrative production, and future thinking (Fig. 1), and the differences described here for these phenomena between autism and the psychotic-affective spectrum.

1.2.5. Meaning and salience

Autism appears to involve a reduced drive to discern, infer and imagine meanings, represented as global, whole, 'big pictures', due to enhanced perceptual functioning, detail- and parts-focused

Table 3
Contrasts in narrative formation and comprehension, and art, between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Narrative formation, comprehension; art	Narrative production and understanding reduced in autism (Barnes, 2012; King, Dockrell, & Stuart, 2014; McCabe, Hillier, & Shapiro, 2013), especially with regard to conceptual novelty, fantasy or social themes (Belmonte, 2007); story-telling biased toward local details (Barnes & Baron-Cohen, 2012); children with autism prefer to read non-fiction compared to fiction (Barnes, 2012) Art in autism mainly reality-based or involves rule-based system for producing images or patterns (Roth, 2007; Snyder & Thomas, 1997) Autism associated with technical professions and interests (Campbell & Wang, 2012; Dickerson, Pearson, Loveland, Rahbar, & Filipek, 2014; Spek & Velderman, 2013; Wheelwright & Baron-Cohen, 2001)	Psychosis (hallucinations and delusions) commonly involves imagined social narratives (Currie & Jureidini, 2003; Nettle, 2001) Narrative identity less coherent in schizophrenia (Raffard, D'Argembeau, Lardi, et al., 2010) Schizotypy, schizophrenia, bipolar disorder, and depression associated with production of poetry, literature (fiction), visual and creative arts, and humanities (e.g., Campbell & Wang, 2012; Claridge, Pryor, & Watkins, 1998; Kyaga et al., 2013; Nettle, 2006; O'Reilly et al., 2001; Verhaeghen, Joorman, & Khan, 2005; Young, Winner, & Cordes, 2013)	Narrative formation and understanding involve social creativity and imagination, and understanding of social motivations Default mode network seen as 'seat of literary creativity', based on brain imaging evidence (Wise & Braga, 2014)

attention, restricted interests, and increased focus on predictable, known patterns and systems (Table 5). This domain of cognition can thus generate some degree of consilience between the weak central coherence, empathizing-systemizing, and enhanced perceptual function theories of autism (Baron-Cohen & Belmonte, 2005).

Schizophrenia, schizotypy, and psychosis more generally are characterized by apophenia (the tendency to infer, and imagine, meaningful patterns and causal connections where none exist), in association with increased and dysregulated salience which leads to imbuing of irrelevant stimuli with meaning (Table 5). Magical ideation and fantasy proneness indeed represent clear examples of apophenia in schizotypy, which dovetail closely with increased imagination in this cognitive style.

1.2.6. Episodic memory and future thinking

Episodic memory, future thinking, and autonoetic awareness (thinking about one's past or future self) have all been reported as decreased in autism; autobiographical memory has also been reported as over-general (less specific) among individuals with Asperger syndrome (Table 6). These finding are consistent with reduced imagination in autism, given that imaginative and future-oriented cognition are directly dependent on recruiting components of thought from episodic and autobiographical memories (Schacter et al., 2012).

Episodic memory and future thinking have also been reported as reduced in schizophrenia, bipolar disorder, and depression, and episodic memory is consistently reported as over-general; autonoetic awareness is similarly reduced in schizophrenia and schizotypy in some studies, but other studies have shown that positive schizotypy is linked with higher levels of self-reported autonoetic awareness (Table 6). Psychotic-affective conditions also involve high levels of intrusive episodic memories, which are commonly associated with trauma and adversity.

Reductions in episodic memory and future thinking, and over-general memory, have been demonstrated in both autism and psychotic-affective conditions, although whether the causes of such reductions are the same remains unclear. In contrast to these deficits, enhanced episodic memory is consistently found to be associated with mixed-handedness, which is a strong correlate of schizotypy and schizophrenia (Table 6); moreover, Asperger syndrome has been shown to engender less remembering and more knowing in a task allowing these two interpretations to compete, whereas mixed handedness is linked with the opposite: more remembering and less knowing. Higher autonoetic awareness has also been associated with positive schizotypy in two studies, and reduced autobiographic memory in Asperger syndrome has been attributed to an underdeveloped sense of self (Goddard, Howlin, Dritschel, & Patel, 2007). These findings suggest that further studies of memory and future thinking in these conditions may usefully take account of handedness, confounding of remembering with knowing, the severity and forms of psychotic-affective symptoms (which may mediate general cognitive dysfunctions), and memory in autism for episodic events that do not involve the self or social interactions.

1.2.7. Mental imagery

Mental imagery in autism has been described as thinking in realistic, photographic pictures drawn from memory (Table 7). Such descriptions accord well with documented enhancements of some visual and spatial skills in autism, and highly-realistic drawing abilities as an autism-associated islet of ability, although they remain largely anecdotal.

In psychotic-affective conditions, mental imagery has been consistently self-reported as especially vivid, and individuals with schizophrenia outperform controls on some mental imagery manipulation tasks, although their abilities to maintain images in working memory is impaired (Table 7). Hallucinations have also been described in terms of confusion between perception and intrusive mental imagery, and high vividness of mental imagery may amplify their emotional content. The commonly-fantastic nature of visual hallucinations, and delusions, in psychosis attests to their imaginative content, but the degree to which mental imagery among individuals on the psychotic-affective spectrum is imaginative, compared to realistic, has yet to be determined for non-pathological contexts.

1.2.8. Sensory correlates of imagination

Sensory functions, which are necessarily directed externally, are inversely associated with internally-directed default mode functions (e.g., Greicius & Menon, 2004). A large body of evidence, centered on the enhanced perceptual function (Mottron, Dawson, Soulières, Hubert, & Burack, 2006) and intense world (Markram & Markram, 2010) theories of autism, indicates that sensory abilities are increased in autism; by contrast, a considerable suite of studies demonstrates reduced sensory function and abilities in schizophrenia (Table 8). Baron-Cohen, Ashwin, Ashwin, Tavassoli, and Chakrabarti (2009) have suggested that

Table 4
Contrasts in mentalizing and empathizing between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Mentalizing & empathizing	Reduced theory of mind skills in autism mainly due to under-mentalizing (Baron-Cohen, 2000) Reduced empathizing in autism, considered as drive to understand and share feelings of others (Baron-Cohen, 2009)	Theory of mind skills increased among individuals with borderline personality disorder or mild depression, in well-replicated studies (Dinsdale & Crespi, 2013; Harkness et al., 2011) Theory of mind skills lower in schizophrenia, bipolar, depression, individuals with psychotic experiences, due in large part to over-mentalizing especially in positive schizotypy (Clemmensen et al., 2014; Fretland et al., 2015) and in borderline personality disorder (Sharp et al., 2013) Higher empathizing relative to systemizing associated with higher paranoia and mania in healthy population (Brosnan, Ashwin, Walker, & Donaghue, 2010) Higher empathizing associated with higher rates of depression, among females subject to stressful, abusive childhood conditions (Zahn-Waxler & Van Hulle, 2011) Hyper-mentalizing in psychotic-affective conditions involves over-developed (increased and incorrect) social imagination, over-use of mental state attribution (Abu-Akel, 1999; Clemmensen et al., 2014; Dziobek et al., 2006; Frith, 2004; Sharp & Venta, 2012; Sharp et al., 2013)	Theory of mind can be considered in terms of imagination of mental states (thoughts, feelings and intentions) of others (as in Imagination subscale of Autism Quotient) (Baron-Cohen et al., 2001)

Table 5
Contrasts in meaning, salience and objects of attention between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Meaning, salience, objects of attention	Weak central coherence in autism involves reduced drive to discern, infer, imagine meanings, wholes (Frith, 2012) Increased attention to local detail in autism, less attention, understanding, imagination of global 'big picture' (Happé, 1999; Happé & Frith, 2006; Jolliffe & Baron-Cohen, 1999) Increased systemizing (attention to rule-based systems) in autism, which involves restricted interests and increased focus on predictable patterns (Baron-Cohen et al., 2011)	High levels of apophenia (tendency to infer, imagine meaningful patterns and causal connections where none exist) especially in association with schizotypy, schizophrenia, and psychosis (Brugger & Mohr, 2008; Fyfe, Williams, Mason, & Pickup, 2008; Lakis & Mendrek, 2013) Apophenia mediated by dopaminergic system (DeYoung, 2013; DeYoung, Grazioplene, & Peterson, 2012), as is salience dysregulation in schizophrenia (Winton-Brown, Fusar-Poli, Ungless, & Howes, 2014) Schizophrenia considered as 'salience syndrome' (e.g., Van Os, 2009), due to excessive and inappropriate assignment of meaning to phenomena Magical ideation, a central form of apophenia, associated with creativity (Badzakova-Trajkov, Häberling, & Corballis, 2011); schizotypy associated with fantasy-proneness (Merckelbach, Rassin, & Muris, 2000); positive association of measures of fantasy and imagination with dissociation scores (Rhue, Lynn, & Sandberg, 1995)	Less drive for meaning, and increased meaning found in rule-based systems and patterns in autism, contrast directly with over-developed drive for meaning, salience hyper-sensitivity, arbitrariness, and inappropriateness, and apophenia more generally in psychotic-affective conditions Meaning in psychotic-affective conditions commonly involves imagined scenarios, confabulations, or unacknowledged fantasy, usually with animate and social content

increased sensory abilities in autism contribute to a detail-focused and systemizing cognitive style, which may reduce imaginative cognition to the extent that detail-oriented and rule-based thought diminish the generation of cognitive novelty. In schizophrenia, psychotic symptoms have been interpreted as due in part to interpretation of imagination as perception, mediated by degradation of sensory functions; indeed, hallucinations are associated with impaired sensory processing (Javitt, 2009a, 2009b) and at an extreme, sensory deprivation induces aspects of psychosis (Table 8). Studies that directly address the issue of

Table 6
Contrasts in episodic memory and future thinking between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Episodic memory & future thinking	Episodic memory reduced in autism (Bowler, Gardiner, & Gaigg, 2007; Bowler, Gardiner, & Grice, 2000; Crane & Goddard, 2008; Crane, Pring, Jukes, & Goddard, 2012; Goddard et al., 2007; Lind & Bowler, 2010; Terrett et al., 2013); also reduced autonoetic awareness, with regard to thinking about future self (Hanson & Atance, 2014; Lind & Bowler, 2008)	Episodic memory and future thinking reduced in schizophrenia, bipolar disorder, and depression; memory tends to be overgeneral (D'Argembeau et al., 2008; Hach, Tippett, & Addis, 2014; King et al., 2011) Reduced autonoetic awareness among individuals with high schizotypy (Arzy, Mohr, Molnar-Szakacs, & Blanke, 2011) or with schizophrenia (de Oliveira, Cuervo-Lombard, Salamé, & Danion, 2009; Raffard, D'Argembeau, Bayard, Boulenger, & Van der Linden, 2010) Higher autonoetic awareness in positive schizotypy, with regard to mental time travel (Winfield & Kamboj, 2010); positive schizotypy associated with higher sense of presence (in autonoetic awareness) and perceived similarity of imagined events to past episodes (Raffard, D'Argembeau, Lardi, et al., 2010) Higher levels of intrusive episodic memories in depression, bipolar, schizophrenia, schizotypy, and with high dissociation (Brewin & Soni, 2011; Jones & Steel, 2012)	Episodic memory and future thinking are strongly positively correlated in healthy individuals (Busby & Suddendorf, 2005; D'Argembeau et al., 2008) Dissociation involved in episodic memory (current versus past self) (Suddendorf & Corballis, 1997) Less remembering and more knowing in Asperger syndrome (Bowler et al., 2000); more remembering and less knowing associated with mixed handedness (Propper & Christman, 2004) Enhanced episodic memory associated with mixed-handedness (Christman & Butler, 2011; Propper, Christman, & Phaneuf, 2005); mixed-handedness higher in schizotypy (Somers, Sommer, Boks, & Kahn, 2009) and schizophrenia (Sommer, Ramsey, Kahn, Aleman, & Bouma, 2001)

Table 7
Contrasts in mental imagery between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Mental imagery	Mental imagery in autism involves thinking in photo-realistic pictures taken from memory; abstract thoughts are converted from pictures (Grandin, 1995, 2009) Imagination in autism may be 'limited to rerunning images on visuospatial scratch-pad' (Boucher, 2007) Autism involves increased use of visuospatial processing strategies, and enhancements on some visual and spatial tasks (Sahyoun, Belliveau, Soulières, Schwartz, & Mody, 2010; Sahyoun, Soulières, Belliveau, Mottron, & Mody, 2009; Soulières et al., 2009)	Especially vivid mental imagery, or enhanced abilities at some mental imagery tasks, documented in schizotypy, schizophrenia, bipolar disorder, and dissociation (Aleman, Nieuwenstein, Böcker, & de Haan, 2000; Benson & Park, 2013; Ivins et al., 2014; Matthews, Collins, Thakkar, & Park, 2014; Meyer, Finucane, & Jordan, 2011; Murray & Johnson, 2010; Pearson et al., 2013; Rasmussen & Parnas, 2015) Hallucinations involve confusion between mental imagery and perception (Brébion et al., 2008,Currie, 2000) Mental imagery mediates striving, goal achievement, in bipolar disorder (Conway, Meares, & Standart, 2004; Meyer et al., 2011) Mental imagery is strongly positively associated with hypomanic traits, in healthy population (McGill & Moulds, 2014) Psychotic-affective conditions commonly involve intrusive, involuntary and negative mental imagery (Brewin & Soni, 2011; Holmes, Geddes, Colom, & Goodwin, 2008; Jones & Steel, 2012; Morina, Deeprose, Pusowski, Schmid, & Holmes, 2011)	Mental imagery represents one manifestation of imagination, that is negatively associated with thinking in words (inner speech) (Stawarczyk, Cassol, & D'Argembeau, 2013) Evidence for high incidence of thinking in pictures in autism remains largely anecdotal, and its effects on imagination remain unclear; to the extent that autistic visual imagery is reality-based, it may constrain imagination and creativity Mental imagery shows enhanced vividness, and confusion with reality, in psychotic-affective conditions, but the nature of its relationship with fantasy and creativity remains unspecified; to the extent that psychotic-affective mental imagery is based on fantasy and magical thinking, it should enhance creativity at least to a point Mental imagery may act as 'emotional amplifier' of positive or negative mood, and thus increase both creativity and psychopathology (Holmes et al., 2008) Visual imagery and visual perception use broadly overlapping brain regions, with information flow between them (Dentico et al., 2014; Slotnick et al., 2012)

tradeoffs between perceptual and imaginative abilities, and their neural mechanisms, should clarify the role of sensory systems in imagination and creativity, and such associations with autism and schizophrenia.

1.2.9. Neural instantiation of imagination in autism and schizophrenia

The default mode network has been indicated by numerous studies to represent the primary neural system that underlies imagination (Table 9). This system has been considered to be

Table 8
Contrasts in sensory systems between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Sensory systems	Sensory systems generally enhanced in autism, with increased sensitivities over neurotypicals, and more intense and accurate sensation (Falter, Braeutigam, Nathan, Carrington, & Bailey, 2013; Mottron et al., 2006, 2009; Tavassoli, Miller, Schoen, Nielsen, & Baron-Cohen, 2013)	Deficits in processing, filtering sensory information in schizophrenia and schizotypy; reduced sensory abilities overall (Bates, 2005; Dyck, Loughead, Gur, Schneider, & Mathiak, 2014; Javitt, 2009a, 2009b) Hallucinations in schizophrenia associated with impaired sensory processing (Javitt, 2009a, 2009b) Under mood induction, schizophrenia patients activate sensory areas less, and higher cortical areas more, than controls (Dyck et al., 2014)	Enhanced sensory abilities in autism may foster detail-oriented processing and systemizing (Baron-Cohen et al., 2009), and reduced combinatorial creativity Reduced sensory abilities in schizophrenia may potentiate apophenia and psychotic symptoms, due to confusion between poorly-perceived reality and imagination and impaired source monitoring (Brébion et al., 2008, Javitt, 2009a, 2009b); default mode network shows lower connectivity to sensory networks in schizophrenia, compared to controls (Li et al., 2015)

Table 9
Contrasts in neural instantiations of the default mode system between the autism spectrum and the psychotic-affective spectrum.

Trait	Autism spectrum (autism and Asperger syndrome)	Psychotic-affective spectrum (mainly schizophrenia, schizotypy, bipolar, depression, borderline personality, dissociative disorders)	Comments
Neural instantiation	Default system activation reduced in autism, in association with reduced self-referential and imaginative cognition (Buckner et al., 2008; Iacoboni, 2006; Kennedy & Courchesne, 2008; Kennedy et al., 2006) Temporal-parietal junction shows lower activation in autism, mediates mentalizing reductions (Kana, Libero, Hu, Deshpande, & Colburn, 2014; Lombardo, Chakrabarti, Bullmore, Baron-Cohen, & MRC AIMS Consortium, 2011) Reduced deactivation of default mode in autism, upon task initiation (Kennedy et al., 2006; Murdaugh et al., 2012; Spencer et al., 2012) Reduced connectivity within default mode in autism (Jung et al., 2014; Murdaugh et al., 2012; von dem Hagen, Stoyanova, Baron-Cohen, & Calder, 2012)	Default system over-activated in schizophrenia, in association with reality distortion and increased imaginative cognition (Buckner et al., 2008); also less deactivation of this system upon external-task initiation (Alonso-Solís et al., 2015; Landin-Romero et al., 2014) Temporal-parietal junction shows higher activation in schizophrenia, mediates some positive symptoms (Wible, 2012a, 2012b) Increased connectivity within default mode in schizophrenia (Li et al., 2015; Peeters et al., 2015; Tang et al., 2013; Whitfield-Gabrieli & Ford, 2012); comparable patterns found in depression (Manoliu et al., 2013) Originality and schizotypy exhibit similar fMRI patterns during creative ideation (reduced deactivation of right precuneus) (Fink et al., 2014) Reduced task-induced deactivation in precuneus associated with higher creativity (divergent thinking); similar pattern found in schizophrenia (Takeuchi et al., 2011) Divergent thinking, originality positively associated with activation of default mode network regions (Mayseless et al., 2015)	Default mode considered as a primary neurological system subserving imagination and creativity (Agnati et al., 2013; Beaty et al., 2014; Fink et al., 2010; Hassabis & Maguire, 2007, 2009; Jauk, Neubauer, Dunst, Fink, & Benedek, 2015; Jung et al., 2013; Kennedy et al., 2006; Mullally & Maguire, 2013; Smallwood & Schooler, 2015; Takeuchi et al., 2012; Uddin, Iacoboni, Lange, & Keenan, 2007; Wise & Braga, 2014) Reviews describe opposite nature of default mode activation, connectivity in autism versus schizophrenia (Broyd et al., 2009; Immordino-Yang et al., 2012; Karbasforoushan & Woodward, 2012), although not all findings show these patterns Creativity associated with greater functional connectivity between default mode and inferior prefrontal cortex (Beaty et al., 2014), and between regions within default mode (Wei et al., 2014); Creativity associated with gray matter volume in multiple regions of the default mode region (Jauk et al., 2015) Default mode connectivity higher in females than males (Biswal et al., 2010; Bluhm et al., 2008; Tomasi & Volkow, 2012), as expected under extreme male brain hypothesis (Baron-Cohen et al., 2011)

underactivated in autism compared to controls, based on reduced deactivation in the default mode upon initiation of an external task, which is interpreted as indicating intrinsically low activity (Kennedy, Redcay, & Courchesne, 2006; Spencer et al., 2012). By contrast, functional overactivity of the default mode has been inferred from a set of studies of schizophrenia (reviews in Buckner et al., 2008; Immordino-Yang, Christodoulou, & Singh, 2012; see also Wible, 2012a, 2012b), based in part on reduced deactivations that are interpreted as high default mode activity continuing even during engagement in externally-directed tasks (e.g., Bleich-Cohen, Kupchik, Gruberger, Kotler, & Hendler, 2012). Takeuchi et al. (2011) showed that such reduced task-induced deactivation (in the precuneus) was also associated with higher creativity (divergent thinking), noting that a similar pattern has been found in schizophrenia. Similarly, Fink et al. (2014) showed that reduced task-induced deactivation of the precuneus was associated both with higher levels of divergent thinking in a normal population, and with high schizotypy.

Neural connectivity within the default mode shows evidence of contrasting patterns between autism and schizophrenia, with lower connectivity reported in most studies of autism, whereas most analyses of schizophrenia describe higher connectivity (reviewed in Broyd et al., 2009; Karbasforoushan & Woodward, 2012; Peeters et al., 2015; Table 9). It is important to bear in mind that, despite the conclusions of these and other authors that connectivity patterns are opposite in autism and schizophrenia, there is considerable heterogeneity in results across studies. The overall finding of diametric patterns are also supported, however, by psy-

Table 10
Nine of the ten questions on the imagination subscale of the Autism Spectrum Quotient refer to validated functions of the default mode. Note that some questions are reversed (lower endorsement is considered more autistic).

Default mode function	AQ-Imagination subscale question	Citations for default mode function
Social narrative	I find making up stories easy	Wise and Braga (2014)
	I don't particularly enjoy reading fiction	Smallwood et al. (2013a) and Smallwood, Ruby, and Singer (2013b)
	I would rather go to the theatre than a museum	Barnes (2012)
Pretend play/imagining	When I was young, I used to enjoy playing games involving pretending with other children	Agnati et al. (2013)
		Schacter et al. (2012)
	I find it very easy to play games with children that involve pretending	Østby et al. (2012)
Mentalizing	I find it difficult to imagine what it would be like to be someone else	Amft et al. (2015)
	When I'm reading a story, I find it difficult to work out the character's intention	Denny, Kober, Wager, and Ochsner (2012)
		Mars et al., 2012
Mental Imagery	If I try to imagine something, I find it very easy to create a picture in my mind	Zvyagintsev et al. (2013)
		Daselaar, Porat, Huijbers, and Pennartz (2010)
	When I'm reading a story, I can easily imagine what the characters might look like	
Not clearly associated	I like to collect information about categories of things	

chiatric correlates of default mode connectivity; for example, increased default mode connectivity has been associated with higher positive-symptoms scores in schizophrenia (Tang et al., 2013), and with lower scores on the Autism Quotient among individuals with ASD and in healthy controls (Jung et al., 2014). Higher default mode connectivity has also been found in neurotypical females compared to neurotypical males (Biswal et al., 2010; Bluhm et al., 2008; Tomasi & Volkow, 2012), as expected under Baron-Cohen's extreme male brain hypothesis (Baron-Cohen et al., 2011). Finally, higher functional connectivity between default mode and inferior prefrontal cortex (Beaty et al., 2014), and between regions within the default mode (Wei et al., 2014), has been associated with enhanced creativity.

Considered together, these findings suggest that autism and schizophrenia exhibit diametric patterns of default mode activation and connectivity, and that imagination and creativity mediate at least some of these differences. However, sets of individuals with these two conditions have yet to be studied together by the same research group using the same protocols, and there is considerable variation in populations, methodologies, and specific research findings with regard to default mode functioning in autism and schizophrenia. Imaging studies that quantify aspects of imagination among individuals with autism and schizophrenia, in comparison to controls, will be especially useful to address the hypotheses proposed here.

1.3. Evaluating the diametric model for imagination using dimensional autism in healthy populations

We used two lines of empirical evidence to evaluate the hypothesis that imagination represents a central and diametric aspect of autism and psychotic-affective conditions. First, in Study 1, we evaluated the extent to which reduced imagination in autism spectrum cognition is male-biased (compared to other facets of autism), and may thus exhibit a differentially strong impact in accounting for its strong male bias overall (Baron-Cohen et al., 2011). Understanding the causes of the strong male bias in autism represents a key question, that could aid in both refining conceptual cognitive frameworks for the disorder (e.g, Baron-Cohen et al., 2011) and developing better therapies, such as those that target imaginative capacities (Woodard et al., 2014). In particular, to the extent that reduced imagination disproportionately accounts for the strong male bias in autism, it is expected to exhibit an especially important role in the causal underpinnings of autistic cognition.

Second, in Study 2, we tested the hypothesis that summed genetic risk of schizophrenia mediates increased imagination. This hypothesis predicts in particular that individuals with more schizophrenia risk alleles should exhibit more-imaginative cognition, at least by self-report. To our knowledge, this is the first study to test for genetic links of imagination *per se* with schizophrenia, rather than testing for links with creativity, which as noted above includes both imagination and usefulness.

For both of these tests, we used the Imagination subscale of the Autism Spectrum Quotient (Baron-Cohen et al., 2001), since nine of the ten items on this subscale provide clear measures of brain default mode functions (Table 10), and involve aspects of social imagination, the core phenotype postulated to be under-expressed in autism and over-expressed in psychotic-affective spectrum conditions.

2. Study 1

2.1. Methods

2.1.1. Background

We tested the hypothesis that the Imagination subscale of the Autism Spectrum Quotient exhibits an especially strong male bias in scores, compared to the other subscales, by conducting a systematic review and meta-analysis of gender differences on the AQ among non-clinical populations.

Epidemiological studies indicate that autism spectrum conditions are diagnosed substantially more frequently in males than in females, with gender prevalence on the order of 2:1 males to females for severe autism, and 10:1 for autism without intellectual disability (Fombonne, 2009). These findings, and the extensive evidence for the exaggerated male-typical cognition in autism (the extreme male brain hypothesis; Baron-Cohen et al., 2011), indicate that causal factors more-directly and strongly underlying autism are expected to exhibit higher expression in males than females.

The Autism Spectrum Quotient was developed by Baron-Cohen et al. (2001) as a means to quantify self-report autism, in both non-clinical and clinical populations. This questionnaire comprises five subscales that are designed to tap different facets of autism (Social Skills, Communication, Attention Switching, Attention to Detail, and Imagination); for the Imagination subscale, higher scores represent less-developed imagination, especially social imagination, as found in autism (Table 10). As such, to the extent that imagination represents an especially important factor contributing to autism, if should exhibit an especially strong male bias, in the same way that low empathizing and high systemizing are highly male-typical and thus represent central factors underlying autism by Baron-Cohen's extreme male brain theory (Baron-Cohen, 2009; Baron-Cohen et al., 2011).

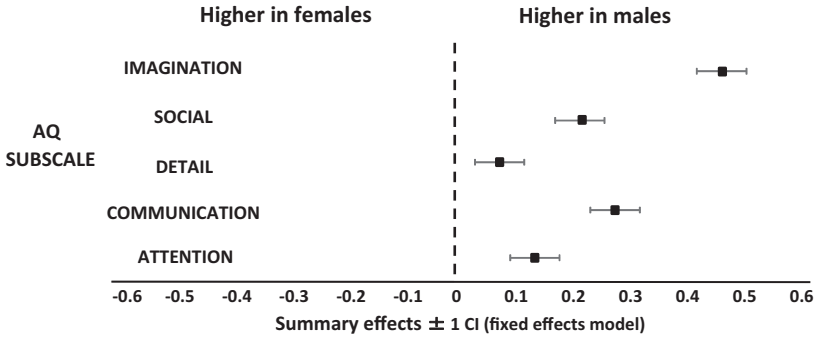

Fig. 3. Results from a fixed-effect meta-analysis of a systematic review, testing for overall gender differences in scores on the five subscales of the Autism Spectrum Quotient test.

2.1.2. Systematic review

All papers (N = 1010, as of the 27 March 2015 access date) that cited Baron-Cohen et al. (2001) in the Web of Science database were included in the meta-analysis if they met the following criteria: (1) the study was based on a non-clinical population, including individuals who were unrelated to individuals with autism; (2) at least 25 individuals of each sex were measured in the study; (3) mean values, standard deviations and sample sizes were reported for the focal AQ subscales (Attention, Communication, Detail, Imagination and Social) under standard coding for this test. Eleven samples from nine studies matched these inclusion criteria. For each subscale, we recorded the average male and female scores per study, as well as their standard deviations and sample sizes. These values are recorded in Supplementary File 1.

2.1.3. Meta-analytic methods

We conducted the meta-analysis of sex differences in AQ subscales according to the procedure outlined in Crawley (2013) for fixed-effect models. Each subscale was analyzed separately for summary effects. First, we calculated the effect sizes for each subscale by measuring the mean difference between male and female response variables. We then calculated the within-study pooled standard deviation, and scaled the effect sizes by this pooled standard deviation. Hedges' g was calculated to correct for any potential biases in mean differences. Weights for each effect were derived by taking the inverse of the variance of the bias-corrected mean difference, which then were multiplied by the corrected effect size (g). The sum of these weighted effect sizes was divided by the sum of the weights to arrive at the summary statistic for a given AQ subscale. The variance of the summary effect was the inverse of the summed weights, while the standard error of the summary effect was simply the square root of this variance. Z-statistics were calculated by dividing the summary statistic by the standard error, whereby significance was assessed.

Our analyses utilized the fixed-effect model to assess differences between the sexes, as we assumed there was a true effect size (Crawley, 2013). To verify the analysis, we utilized R (version 3.1.1) software with the 'meta' package (R Core Team, 2014; Schwarzer, 2014) and 'metacont' function that utilized the inverse variance method. These results closely matched with the initial analysis (Supplementary File 1).

2.2. Results

This analysis demonstrated that Imagination showed a substantially and significantly (greater than two standard errors) higher gender difference, with males higher, than did any of the other AQ subscales (Fig. 3 and Supplementary File 1). Indeed, mean effect sizes for Imagination were about twice as high as those of the next-highest subscales, Social Skills and Communication, both of which also involve aspects of sociality, mainly forms of mentalizing, that are also mediated in part by default mode brain regions. These findings of an exceptionally strong male bias to the AQ subscale Imagination are concordant with evidence for higher female than male connectivity between default mode regions (Biswal et al., 2010; Bluhm et al., 2008; Tomasi & Volkow, 2012) as described above, and with the evidence in Table 10 showing default-mode functions for nine of ten questions on this subscale.

3. Study 2

3.1. Methods

3.1.1. Background

Schizophrenia exhibits a heritability of about 60–80% (Schwab & Wildenauer, 2013), and the heritability of creative achievement has been reported to show similar levels, of about 40–70% (Piffer & Hur, 2014). Under the hypothesis evaluated here, some proportion of alleles underlying schizophrenia risk represent 'imagination' or 'creativity' alleles, which should mediate the expression of imagination and other default-mode functions. This hypothesis was tested for a single genetic locus by Kéri (2009), who found that individuals who carried a genotype linked in other studies with high psychosis risk ('TT', for the Neuregulin1 (NRG1) gene single nucleotide polymorphism rs6994992) showed higher levels of questionnaire-based creative achievement than did individuals with the lower psychosis-risk genotypes, among subjects of relatively-high intellect. More recently, Power et al. (2015) calculated polygenic schizophrenia risk scores (sums of risk alleles across many loci) and demonstrated, with large sample sizes, that higher polygenic risk scores were associated with membership in artistic societies or creative professions. Considered together, these findings indicate that 'schizophrenia risk' alleles can also be considered as alleles mediating aspects of creativity, as postulated from many years of non-genetic work.

These outstanding studies by Kéri (2009) and Power et al. (2015) used creative achievement or profession as their proxies for creativity; by contrast, we focus here on imagination as quantified by the Autism Quotient Imagination subscale which, as described above, appears to closely reflect imaginative default mode network functions. We thus tested a primary prediction of the hypothesis that schizophrenia risk genes mediate human imagination by genotyping a large set of non-clinical individuals for a set of schizophrenia risk loci previously-established by meta-analyses (the Szgene database; Allen et al., 2008), and computing polygenic risk scores that represent the summed number of risk alleles found in each individual (summed 'genetic risk' of schizophrenia). By the hypothesis analyzed here, higher summed

genetic risk of schizophrenia should be linked with increased imagination, as quantified using the Autism Spectrum Quotient Imagination subscale.

3.1.2. Participants

Questionnaire data and samples for DNA extraction were collected from Caucasian undergraduate students (355 males and 644 females) at both University of Alberta and Simon Fraser University. Protocols were conducted according to guidelines established by the ethics boards of both universities, with approval from both governing bodies and informed consent provided by all participants. Samples were collected from two cohorts, and analyzed separately as well as pooled as described below. Further details concerning the populations and sampling methods used can be found in Dinsdale et al. (2013) and Leach, Hurd, and Crespi (2013).

3.1.3. Molecular-genetic methods

Genomic DNA was extracted from mouthwash samples using standard phenol-chloroform extractions. By this method, DNA comes predominantly from lymphocytes.

We genotyped 33 common single nucleotide polymorphisms (SNPs) (with minor allele frequencies >0.1) from the 24 top-ranked genes listed by the Schizophrenia Gene (SZGene) database. These genes represent a list prioritized by strength of meta-analytic evidence, across 118 meta-analyses (Allen et al., 2008); the database has been used extensively (with 560 citations to date in Web of Science), and many of the genes included exhibit strong functional links with schizophrenia risk.

Genes, SNPs and risk alleles (in parentheses) included in this study were: AHI1 (rs1154801(C), rs2064430(T)), AKT1 (rs3803300(A)), C6orf217 (rs10223338(C)), CCKAR (rs1800857 (C)), DAOA (rs3916971(C), rs778293(A)), DISC1 (rs999710(T)), DRD2 (rs6275(T), rs6277(C)), DTNBP1 (rs1474605(T), rs3213207 (T)), GABRB2 (rs1816072(C)), GWA_11p14.1 (rs1602565(C)), GWA_16p13.12 (rs7192086(T)), HIST1H2BJ (rs6913660(C)), HTR2A (rs6311(A)), MDGA1 (rs11759115(T), rs12191311(T)), NOTCH4 (rs2071287(G)), NRG1 (rs10503929(T)), NRGN (rs12807809(T)), PDE4B (rs910694(T)), PPP3CC (rs10108011(G)), PRSS16 (rs13219354(T), rs6932590(T)), RELN (rs262355(A), rs7341475 (G)), RGS4 (rs2661319(G)), RPP21 (rs3130375(C)), TPH1 (rs1799913(A), rs1800532(A)),and ZNF804A (rs1344706(T)). Many of these loci (e.g., DISC1, DTNBP1, GABRB2, NRG1, RELN, ZNF804A) have been subject to extensive replication and functional validation as mediating risk of schizophrenia and its phenotypes; by contrast, more-recent SNPs inferred from GWAS have yet to be subject to such functional analyses to any degree. Genotyping was performed by Genome Québec (Montréal, Canada) using Sequenom technology, and genotypes were in Hardy-Weinberg equilibrium for all loci analyzed.

3.1.4. Analytic methods

Polygenic risk scores represent summations of genetic risk across multiple risk loci, such that each individual can be quantified for their estimated level of overall risk (review in Wray et al., 2014). In our analysis, polygenic risk scores were computed under three genetic models: (1) additive (score of one for each risk allele, at each locus), (2) dominant (individuals with one or two risk alleles receive a score of one, compared to individuals with no risk alleles who receive a score of zero), or (3) recessive (individuals with two risk alleles receive a score of one, compared to individuals with one or no risk alleles who receive a score of zero). We then evaluated the product-moment correlations of polygenic risk scores with AQ-Imagination scores, separately for males and females. The sexes were analyzed separately because of previous results showing sex-differential associations of polygenic schizophrenia risk on mental rotation ability (Leach et al., 2013), because the sexes differ notably in AQ-Imagination scores (with males lower, as described above), and because sex differences in cognitive and psychological traits are extensive in schizophrenia (e.g., Mendrek & Stip, 2011; reviews in Leung & Chue, 2000; Abel et al., 2010; Mendrek, 2015). However, we did not have *a priori* predictions concerning patterns expected in each of the two sexes, so interpretation of sex-specific results was subjected to twofold Bonferroni adjustment.

Table 11
Product-moment correlations of schizophrenia polygenic risk scores with Autism Quotient Imagination scores, under three different genetic models for schizophrenia allelic risk effects.

Model	Sex	Correlation	*t*	*p*
Additive	Males	−0.13	−2.42	0.016 *
Additive	Females	0.01	0.34	0.73
Dominant	Males	−0.05	−0.87	0.39
Dominant	Females	−0.01	−0.24	0.81
Recessive	Males	−0.14	−2.65	0.0085 **
Recessive	Females	0.03	0.72	0.47

* $p < 0.05$.
** $p < 0.01$.

3.2. Results

For males, polygenic risk score was significantly negatively correlated with AQ-Imagination score, under both the additive and recessive models (Table 11); by these two analyses, variation in polygenic risk score thus accounted for about 1.5–2% of variation in AQ-Imagination score. A recessive model is commonly considered as most appropriate for psychiatric 'risk' genes (e.g., Harrison, Gamsiz, Berkowitz, Nagpal, & Jerskey, 2015), and as such may be expected to represent the best model in this case. Correlations for females were small and non-significant, as was that for males under the dominant model (Table 11).

None of the autism subscales other than Imagination (for which we did not have *a priori* predictions of any kind), or total AQ scores, showed significant associations with genetic risk scores), nor did any analyses that pooled the sexes demonstrate significance (Supplementary File 2). These findings provide evidence that, among males, higher genetic risk of schizophrenia is associated with enhanced imagination, since higher scores on AQ-Imagination indicate reduced imaginative function.

4. Discussion

We have described three lines of evidence, from narrative review, meta-analysis, and schizophrenia genetic risk scores, salient to predictions of the hypothesis that imagination, as conceptualized here, is notably reduced in autism spectrum conditions and dysfunctionally increased in psychotic-affective conditions. These convergent findings connect human evolution with psychiatric conditions in that imagination, primarily instantiated in the brain's default mode network, represents a highly human-elaborated phenotype that is expected to be subject, like other biological systems, to alterations toward either decreased or increased expression. Considered together, these results support Jung's (2014) models of autism and schizophrenia as opposite with regard to creative cognition, and the diametric model for autism and psychotic-affective conditions developed by Crespi and Badcock (2008), which posits that these two sets of disorders result from opposite alterations to central facets of human cognition.

Our analyses and findings are novel in that they represent the first joint, comprehensive study of imagination in autism and in psychotic-affective conditions, and link psychological and psychi-

atric with neurological and genetic results. They are subject, however, to important further considerations and caveats.

4.1. Narrative review

Our narrative review of the literature on core aspects of imagination in autism, and in psychotic-affective conditions, shows that with regard to pretend play, creativity and generativity, narrative and the arts, mentalizing, meaning and salience, future thinking and episodic memory, mental imagery, and sensory systems, autism and psychotic-affective conditions exhibit considerable evidence of opposite alterations. These findings suggest that the Crespi and Badcock (2008) diametric-disorders hypothesis, which has previously emphasized altered balances in mentalistic, compared to mechanistic, cognitive phenotypes in psychotic-affective conditions compared with autism, can be extended and generalized to encompass psychological phenotypes, such as creativity, conceptualization of novelty, salience detection, mental imagery, and sensory perception in relation to imagery, that are broader than social cognition *per se*. As such, these phenotypes represent components of the default mode and closely-interacting networks that underlie non-social as well as social functions.

The emphasis described here on neurologically-based default mode functions is notably supported by (1) extensive, though not fully unambiguous, evidence for reduced default mode connectivity in autism and increased connectivity in schizophrenia, (2) reduced activation of the temporal-parietal junction in autism but increased activation in schizophrenia, and (3) consideration of the validated social as well as non-social functions of this network, which centrally include imagination. In this general context, our review has identified mental imagery in autism compared to schizophrenia, and the relation of dissociation to 'overdevelopment' of pretend play and imagination, as especially interesting and important areas for future research.

A limitation of the results from our narrative review is that few studies have directly compared groups of individuals with autism versus psychotic-affective conditions, in relation to control groups, using the same metrics. Moreover, both psychotic-affective conditions, which include schizophrenia, bipolar disorder, depression, borderline personality, dissociative disorders, and autism spectrum conditions, are notably diverse in the psychological and psychiatric phenotypes expressed, such that particular findings regarding imagination may apply only to subsets of individuals or diagnoses in each general category. Default mode cognitive functions may also differ between neurotypical individuals and individuals with disorders, such that, for example, 'mind wandering' and 'day dreaming' in autism may involve thinking about one's specific ('restricted') non-social interests, rather than the self or others (Lombardo & Baron-Cohen, 2011; Uddin, 2011). Similarly, mental imagery mediated by the same brain regions may be predominantly realistic in autism but mainly fancifully-imagined, and intrusive, in schizophrenia, and intermediate in form of expression among neurotypical individuals. In such contexts, it becomes more accurate, in some respects, to consider imagination as different in autism, and in psychotic-affective conditions, rather than just lowered or raised to the point of dysfunctional effects. Finally, general or specific cognitive or neurological deficits may also affect individuals with autistic or psychotic-affective conditions, which complicates or confounds the empirical connection of imagination-related adaptations with maladaptations due to under- or over-development.

Despite these caveats, the close dovetailing of our narrative review results between psychiatric, psychological and neurological lines of evidence suggests that the default mode network in general, and imagination in particular, represent phenotypes whose alteration can generate a wide spectrum of psychological and psychiatric variation from autism, to neurotypical individuals, to psychotic-affective conditions. Most broadly, the default mode can be considered as the brain network that is most frequently active during ongoing human thought (Buckner et al., 2008), and as such represents a core substrate for alteration and variation in both psychiatric conditions and dimensional cognitive variation. Indeed, Shin et al. (2015) describe a novel cognitive model of schizophrenia based on increased mind-wandering, and demonstrate significant positive associations of a psychological measure of mind-wandering with both positive and total schizophrenia symptom scores, among individuals with schizophrenia. Their model is also supported by studies showing that primary cognitive 'costs' of mind-wandering include reduced working memory and lower sustained attention, both of which exhibit especially-notable reductions in schizophrenia (Mooneyham & Schooler, 2013; Silver & Feldman, 2005).

4.2. Gender biases in AQ subscales

Our second line of evidence, much stronger male biases to the Imagination subscale on the Autism Quotient questionnaire than for the other four subscales, by meta-analysis, is relevant to reduced imagination in autism. In particular, these results suggest that differences between males and females in imagination represent the strongest psychological factor, of the five AQ subscales, contributing to the male bias in autism. As such, and given that imagination may broadly underlie and subserve the social, communicative, and attentional functions that are represented by the other AQ subscales, reduced imagination among males may especially-strongly contribute to their higher dimensional and categorical manifestations of autism spectrum disorders.

The interpretation of these results is predicated on the strong male sex ratio biases found in autism (Fombonne, 2009), in conjunction with the extensive data that support Baron-Cohen's extreme male brain hypothesis in this epidemiological context (Baron-Cohen et al., 2011). Given that the AQ-Imagination subscale taps self-report psychological variation in default mode functions (Table 2), these results are concordant with the findings from our narrative review, as well as with evidence of lower default mode connectivity among males than among females (Biswal et al., 2010; Bluhm et al., 2008; Tomasi & Volkow, 2012).

Do the sexes differ, then, in imagination from previous relevant studies, and does the default mode mediate any differences? Abraham, Thybusch, Pieritz, and Hermann (2014) showed that males and females differ in brain activation during two creative thinking tasks, conceptual expansion and divergent thinking, with females (but not males) showing high activation in regions such as the medial PFC and temporal parietal junction that represent important components of the default mode network. Ryman et al. (2014) similarly described notable sex differences in patterns of brain activity during creative cognition, for regions that include the default mode network as well as other areas. In neither study did the sexes differ in performance. Considered together, these studies suggest that even when performance does not differ, males and females exhibit different neurocognitive strategies during creative tasks, with females apparently using social and default-mode regions more than do males (Abraham, 2015). Preferential female use of social-cognitive regions in creative tasks would be concordant with their higher AQ-Imagination scores compared to males, given that AQ Imagination mainly involves aspects of social imagination (Table 10). Given the differences between creativity and imagination, however, with the latter being a necessary component of the former, additional tests for sex differences in aspects of imagination *per se* are required for robust interpretation of these inferences.

A primary caveat for interpreting these meta-analytic results is that they derive from non-clinical populations, so their relevance to individuals with autism remains unclear. By contrast, the primary advantage of non-clinical populations is that they are not influenced by secondary, more or less pathological, effects as in clinical populations, or by ascertainment biases that may raise diagnostic thresholds for females compared to males (Dworzynski, Ronald, Bolton, & Happé, 2012). Moreover, very few studies have reported AQ subscale scores separately by sex for individuals with autism. The degree to which imagination-related phenotypes can be considered as exerting stronger causal influences than effects from other AQ subscale domains in autism also remains unclear; however, strong links of imagination with theory of mind, empathizing, perceptual and sensory abilities, and salience as relevant to weak central coherence, as summarized in Tables 1–9, all suggest that psychological theory for autism based in imagination (e.g., Woodard & Van Reet, 2011) is highly compatible with previous psychological theories for autism.

4.3. Genetic risk of schizophrenia in relation to AQ-Imagination

The third line of evidence investigated here has involved collection of empirical data on schizophrenia risk genes in relation to AQ-Imagination, to test the hypothesis that such 'risk' genes represent, in part, genes whose alleles mediate variation among individuals in imaginative cognition. This test is based on the supposition that a substantial number of 'schizophrenia risk' alleles, especially common polymorphisms, exhibit functions associated with higher imagination in non-clinical populations, rather than simply representing deleterious alleles that affect the incidence of disease. Such a prediction follows from several considerations: (1) the high heritability of schizophrenia and the substantial proportion of its heritability that is underlain by common alleles (Ripke et al., 2014), (2) the evidence showing that imagination and creativity tend to be enhanced in close relatives of individuals with schizophrenia rather than in the affected individuals themselves (Table 2), (3) the fact that such alleles predominantly exert their cognitive effects in non-clinical populations, who represent on the order of 99% of all individuals, and (4) previous studies, described above, that have linked schizophrenia risk alleles with measures of high creativity (Kéri, 2009; Power et al., 2015).

In our study, we genotyped non-clinical individuals for a set of SNPs that have been associated with schizophrenia by meta-analyses in previous work, and found that under two of three genetic models, and for males only, higher summed genetic risk of schizophrenia was significantly associated with lower scores on AQ-Imagination, which represents better imaginative cognition. These results thus provide partial support for the prediction, in that the observed effects were sex specific and model specific, and as such they suggest that further studies along these lines would be informative. Findings limited to one sex are frequently observed in studies of schizophrenia or schizotypy (e.g., Mendrek & Stip, 2011), and in this instance they may be related in some way to the well-established gender differences in default mode network connectivity discussed above, and in social brain functions such as reading emotions from facial features such as eyes (e.g., Kirkland, Peterson, Baker, Miller, & Pulos, 2013).

Our genetic study is substantially limited by the small number of SNPs used, although it represents an objectively ascertained set of loci ranked by meta-analyses of odds ratios. Moreover, recent genome-wide analysis studies have identified many additional risk loci for schizophrenia (Ripke et al., 2014); our study indicates that these recently-developed loci should be tested for association with measures of imagination (rather than just creative achievement), in part because the cognitive deficits concomitant to schizophrenia and its risk alleles may tend to reduce or preclude achievement, while imagination, instantiated in a highly active and connected default mode network, may still be increased.

Given our results, which can be regarded as tentative yet intriguing, additional studies using these recent sets of risk alleles are thus warranted, to determine if imagination is prominent among the normal evolved functions of alleles that also mediate risk of schizophrenia. In particular, a notable proportion of schizophrenia risk alleles, and autism risk alleles, are expected by our theory and results to modulate default mode network activation patterns and connectivity, with measureable effects on components of imagination. For example, Rose et al. (2012) showed that a schizophrenia risk allele in the nitric oxide synthase gene NOS1 was associated, in healthy individuals, with failure to disengage regions of the default mode network, as expected under the hypotheses addressed here.

4.4. Conclusions

Human psychiatric conditions derive from alterations to adaptive cognitive systems, and imagination can be considered as one of the most fundamental such systems to evolve and become highly elaborated along the human lineage. In this regard, Vygotsky (2004) indeed stated that "absolutely everything around us that was created by the hand of man, the entire world of human culture, is the product of human imagination and of creation based on this imagination". As such, we contend that human imagination, and its bases in the default mode network, represents a key substrate that underlies, and structures, disorders of human cognition.

The integrated psychological, neurological, and genetic perspective used in this paper has suggested that both decreased and increased imagination may mediate symptoms of psychiatric disorders, and that imagination appears to underlie central diametric features of autism in relation to psychotic-affective conditions. A primary implication of these considerations is that imagination, and its default mode instantiations, may serve as primary foci for cognitive and behavioral therapeutic interventions, as described in previous work for autism (Woodard & Van Reet, 2011; Woodard et al., 2014), depression (Brewin et al., 2009; Ivins, Di Simplicio, Close, Goodwin, & Holmes, 2014), bipolar disorder (Holmes et al., 2011; Murray & Johnson, 2010) and schizophrenia (Morrison et al., 2002; Ricarte, Hernández-Viadel, Latorre, Ros, & Serrano, 2014). The hypothesis developed and evaluated here also makes clear, testable predictions regarding future work, with important implications for understanding human imagination from evolutionary, neurological and psychological perspectives.

Acknowledgements

We are grateful to The Natural Sciences and Engineering Research Council of Canada and the Academy of Finland for financial support.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.cognition.2016.02.001.

References

Abel, K. M., Drake, R., & Goldstein, J. M. (2010). Sex differences in schizophrenia. *International Review of Psychiatry, 22*, 417–428.

Abraham, A. (2015). Gender and creativity: An overview of psychological and neuroscientific literature. *Brain Imaging and Behavior*. in press.

Abraham, A., Thybusch, K., Pieritz, K., & Hermann, C. (2014). Gender differences in creative thinking: Behavioral and fMRI findings. *Brain Imaging and Behavior, 8*, 39–51.

Abraham, A., Windmann, S., Daum, I., & Güntürkün, O. (2005). Conceptual expansion and creative imagery as a function of psychoticism. *Consciousness and Cognition, 14*, 520–534.

Abu-Akel, A. (1999). Impaired theory of mind in schizophrenia. *Pragmatics & Cognition, 7*, 247–282.

Acredolo, L., Goodwyn, S., & Fulmer, A. (1995). Why some children create imaginary companions: Clues from infant and toddler play preferences. *Poster presented at the biennial meetings of the Society for Research in Child Development*, Indianapolis, IN.

Agnati, L. F., Guidolin, D., Battistin, L., Pagnoni, G., & Fuxe, K. (2013). The neurobiology of imagination: Possible role of interaction-dominant dynamics and default mode network. *Frontiers in Psychology, 4*, 296.

Aleman, A., Nieuwenstein, M. R., Böcker, K. B., & de Haan, E. H. (2000). Mental imagery and perception in hallucination-prone individuals. *The Journal of Nervous and Mental Disease, 188*, 830–836.

Allen, N. C., Bagade, S., McQueen, M. B., Ioannidis, J. P. A., Kavvoura, F. K., Khoury, M. J., ... Bertram, L. (2008). Systematic meta-analyses and field synopsis of genetic association studies in schizophrenia: The SZGene database. *Nature Genetics, 40*, 827–834.

Alonso-Solís, A., Vives-Gilabert, Y., Grasa, E., Portella, M. J., Rabella, M., Sauras, R. B., ... Corripio, I. (2015). Resting-state functional connectivity alterations in the default network of schizophrenia patients with persistent auditory verbal hallucinations. *Schizophrenia Research, 161*, 261–268.

Amft, M., Bzdok, D., Laird, A. R., Fox, P. T., Schilbach, L., & Eickhoff, S. B. (2015). Definition and characterization of an extended social-affective default network. *Brain Structure and Function, 220*, 1031–1049.

Andreasen, N. J., & Powers, P. S. (1975). Creativity and psychosis: An examination of conceptual style. *Archives of General Psychiatry, 32*, 70–73.

Andrews-Hanna, J. R., Reidler, J. S., Huang, C., & Buckner, R. L. (2010). Evidence for the default network's role in spontaneous cognition. *Journal of Neurophysiology, 104*, 322–335.

Andrews-Hanna, J. R., Smallwood, J., & Spreng, R. N. (2014). The default network and self-generated thought: Component processes, dynamic control, and clinical relevance. *Annals of the New York Academy of Sciences, 1316*, 29–52.

Arden, R., Chavez, R. S., Grazioplene, R., & Jung, R. E. (2010). Neuroimaging creativity: A psychometric view. *Behavioural Brain Research, 214*, 143–156.

Arzy, S., Mohr, C., Molnar-Szakacs, I., & Blanke, O. (2011). Schizotypal perceptual aberrations of time: Correlation between score, behavior and brain activity. *PLoS One, 6*, e16154. http://dx.doi.org/10.1371/journal.pone.0016154.

Badzakova-Trajkov, G., Häberling, I. S., & Corballis, M. C. (2011). Magical ideation, creativity, handedness, and cerebral asymmetries: A combined behavioural and fMRI study. *Neuropsychologia, 49*, 2896–2903.

Balaratnasingam, S., & Janca, A. (2015). Normal personality, personality disorder and psychosis: Current views and future perspectives. *Current Opinion in Psychiatry, 28*, 30–34.

Barnes, J. L. (2012). Fiction, imagination, and social cognition: Insights from autism. *Poetics, 40*, 299–316.

Barnes, J. L., & Baron-Cohen, S. (2012). The big picture: Storytelling ability in adults with autism spectrum conditions. *Journal of Autism and Developmental Disorders, 42*, 1557–1565.

Baron-Cohen, S. (2009). Autism: The Empathizing-Systemizing (E-S) theory. *Annals of the New York Academy of Sciences, 1156*, 68–80.

Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: Hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 364*, 1377–1383.

Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen year review. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds.), *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed., pp. 3–20). New York, NY, USA: Oxford University Press. xix, 530 pp.

Baron-Cohen, S., & Belmonte, M. K. (2005). Autism: A window onto the development of the social and the analytic brain. *Annual Review of Neuroscience, 28*, 109–126.

Baron-Cohen, S., Lombardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B., & Knickmeyer, R. (2011). Why are autism spectrum conditions more prevalent in males? *PLoS Biology, 9*, e1001081.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The Autism Spectrum Quotient (AQ): Evidence from Asperger syndrome/high functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders, 31*, 5–17.

Barrantes-Vidal, N. (2004). Creativity & madness revisited from current psychological perspectives. *Journal of Consciousness Studies, 11*, 58–78.

Bates, T. C. (2005). The panmodal sensory imprecision hypothesis of schizophrenia: Reduced auditory precision in schizotypy. *Personality and Individual Differences, 38*, 437–449.

Beaty, R. E., Benedek, M., Wilkins, R. W., Jauk, E., Fink, A., Silvia, P. J., ... Neubauer, A. C. (2014). Creativity and the default network: A functional connectivity analysis of the creative brain at rest. *Neuropsychologia, 64*, 92–98.

Belmonte, M. K. (2007). Human, but more so: What the autistic brain tells us about the process of narrative. *Autism Represent*, 166–180.

Benson, T. L., & Park, S. (2013). Exceptional visuospatial imagery in schizophrenia; implications for madness and creativity. *Frontiers in Human Neuroscience, 7*.

Biswal, B. B., Mennes, M., Zuo, X., Gohel, S., Kelly, C., Smith, S. M., ... Milham, M. P. (2010). Toward discovery science of human brain function. *Proceedings of the National Academy of Sciences USA, 107*, 4734–4739.

Blanc, R., Adrien, J. L., Roux, S., & Barthélémy, C. (2005). Dysregulation of pretend play and communication development in children with autism. *Autism, 9*, 229–245.

Bleich-Cohen, M., Kupchik, M., Gruberger, M., Kotler, M., & Hendler, T. (2012). Never resting region—mPFC in schizophrenia. *Schizo Res, 140*, 155–158.

Bluhm, R. L., Osuch, E. A., Lanius, R. A., Boksman, K., Neufeld, R. W. J., Théberge, J., et al. (2008). Default mode network connectivity: Effects of age, sex, and analytic approach. *NeuroReport, 19*, 887–891.

Blum, H. (2013). Dissociation and its disorders. *Psychoanalytic Inquiry, 33*, 427–438.

Bob, P., & Mashour, G. A. (2011). Schizophrenia, dissociation, and consciousness. *Consciousness and Cognition, 20*, 1042–1049.

Bonne, O., Canetti, L., Bachar, E., De-Nour, A. K., & Shalev, A. (1999). Childhood imaginary companionship and mental health in adolescence. *Child Psychiatry and Human Development, 29*, 277–286.

Boucher, J. (2007). Memory and generativity in very high functioning autism: A firsthand account, and an interpretation. *Autism, 11*, 255–264.

Bowler, D. M., Gardiner, J. M., & Gaigg, S. B. (2007). Factors affecting conscious awareness in the recollective experience of adults with Asperger's syndrome. *Consciousness and Cognition, 16*, 124–143.

Bowler, D. M., Gardiner, J. M., & Grice, S. J. (2000). Episodic memory and remembering in adults with Asperger syndrome. *Journal of Autism and Developmental Disorders, 30*, 295–304.

Brébion, G., Ohlsen, R. I., Pilowsky, L. S., & David, A. S. (2008). Visual hallucinations in schizophrenia: Confusion between imagination and perception. *Neuropsychol, 22*, 383.

Brewin, C. R., & Soni, M. (2011). Gender, personality, and involuntary autobiographical memory. *Memory, 19*, 559–565.

Brewin, C. R., Wheatley, J., Patel, T., Fearon, P., Hackmann, A., Wells, A., ... Myers, S. (2009). Imagery rescripting as a brief stand-alone treatment for depressed patients with intrusive memories. *Behaviour Research and Therapy, 47*, 569–576.

Brosnan, M., Ashwin, C., Walker, I., & Donaghue, J. (2010). Can an 'Extreme Female Brain' be characterised in terms of psychosis? *Personality and Individual Differences, 49*, 738–742.

Broyd, S. J., Demanuele, C., Debener, S., Helps, S. K., James, C. J., & Sonuga-Barke, E. J. S. (2009). Default-mode brain dysfunction in mental disorders: A systematic review. *Neuroscience and Biobehavioral Reviews, 33*, 279–296.

Brugger, P., & Mohr, C. (2008). The paranormal mind: How the study of anomalous experiences and beliefs may inform cognitive neuroscience. *Cortex, 44*, 1291–1298.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network. *Annals of the New York Academy of Sciences, 1124*, 1–38.

Buckner, R. L., & Krienen, F. M. (2013). The evolution of distributed association networks in the human brain. *Trends in Cognitive Sciences, 17*, 648–665.

Burns, J. (2007). *The descent of madness – Evolutionary origins of psychosis and the social brain*. New York: Routledge.

Busby, J., & Suddendorf, T. (2005). Recalling yesterday and predicting tomorrow. *Cognitive Development, 20*, 362–372.

Campbell, B. C., & Wang, S. S. (2012). Familial linkage between neuropsychiatric disorders and intellectual interests. *PLoS One, 7*, e30405.

Carlson, S. M., Mandell, D. J., & Williams, L. (2004). Executive function and theory of mind: Stability and prediction from ages 2 to 3. *Developmental Psychology, 40*, 1105.

Carlson, S. M., Tahiroglu, D., & Taylor, M. (2008). Links between dissociation and role play in a nonclinical sample of preschool children. *Journal of Trauma & Dissociation, 9*, 149–171.

Carson, S. H. (2011). Creativity and psychopathology: A shared vulnerability model. *Canadian Journal of Psychiatry, 56*, 144–153.

Christman, S. D., & Butler, M. (2011). Mixed-handedness advantages in episodic memory obtained under conditions of intentional learning extend to incidental learning. *Brain and Cognition, 77*, 17–22.

Ciaramidaro, A., Bölte, S., Schlitt, S., Hainz, D., Poustka, F., Weber, B., ... Walter, H. (2015). Schizophrenia and autism as contrasting minds: Neural evidence for the hypo-hyper-intentionality hypothesis. *Schizophrenia Bulletin, 41*, 171–179.

Claridge, G., Pryor, R., & Watkins, G. (1998). *Sounds from the bell jar: Ten psychotic authors*. Cambridge MA: Malor Books.

Clemmensen, L., van Os, J., Skovgaard, A. M., Væver, M., Blijd-Hoogewys, E. M. A., Bartels-Velthuis, A. A., et al. (2014). Hyper-Theory-of-Mind in children with psychotic experiences. *PLoS One, 9*, e113082.

Collerton, D., Perry, E., & McKeith, I. (2005). Why people see things that are not there: A novel perception and attention deficit model for recurrent complex visual hallucinations. *Behavioral and Brain Sciences, 28*, 737–757.

Conway, M., Meares, K., & Standart, S. (2004). Images and goals. *Memory, 12*, 525–531.

Craig, J., & Baron-Cohen, S. (1999). Creativity and imagination in autism and Asperger syndrome. *Journal of Autism and Developmental Disorders, 29*(4), 319–326.

Craig, J., Baron-Cohen, S., & Scott, F. (2000). Story-telling ability in autism: A window into the imagination. *Israel Journal of Psychiatry, 37*, 64–70.

Crane, L., & Goddard, L. (2008). Episodic and semantic autobiographical memory in adults with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 38*, 498–506.

Crane, L., Pring, L., Jukes, K., & Goddard, L. (2012). Patterns of autobiographical memory in adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 42*, 2100–2112.

Crawley, M. J. (2013). *The R book*. West Sussex, UK: John Wiley and Sons, Ltd..

Crespi, B., & Badcock, C. (2008). Psychosis and autism as diametrical disorders of the social brain. *Behavioral and Brain Sciences, 31*, 241–261.

Crespi, B., & Leach, E. (2015). The evolutionary biology of human neurodevelopment: Evo-neuro-devo comes of age. In J. Boughner & C. Rolian (Eds.), *Evolutionary developmental anthropology*. Hoboken NJ: Wiley.

Cropley, A. (2006). In praise of convergent thinking. *Creativity Research Journal, 18*, 391–404.

Currie, G. (2000). Imagination, delusion and hallucinations. *Mind & Language, 15*, 168–183.

Currie, G., & Jureidini, J. (2003). Art and delusion. *Monist*, 556–578.

D'Argembeau, A., Raffard, S., & Van der Linden, M. (2008). Remembering the past and imagining the future in schizophrenia. *Journal of Abnormal Psychology, 117*, 247.

Daselaar, S. M., Porat, Y., Huijbers, W., & Pennartz, C. M. A. (2010). Modality-specific and modality-independent components of the human imagery system. *NeuroImage, 52*, 677–685.

de Oliveira, H., Cuervo-Lombard, C., Salamé, P., & Danion, J. (2009). Autonoetic awareness associated with the projection of the self into the future: An investigation in schizophrenia. *Psychiatry Research, 169*, 86–87.

Denny, B. T., Kober, H., Wager, T. D., & Ochsner, K. N. (2012). A meta-analysis of functional neuroimaging studies of self-and other judgments reveals a spatial gradient for mentalizing in medial prefrontal cortex. *Journal of Cognitive Neuroscience, 24*, 1742–1752.

Dentico, D., Cheung, B. L., Chang, J., Guokas, J., Boly, M., Tononi, G., et al. (2014). Reversal of cortical information flow during visual imagery as compared to visual perception. *NeuroImage, 100*, 237–243.

DeYoung, C. G. (2013). The neuromodulator of exploration: A unifying theory of the role of dopamine in personality. *Frontiers in Human Neuroscience, 7*.

DeYoung, C. G., Grazioplene, R. G., & Peterson, J. B. (2012). From madness to genius: The Openness/Intellect trait domain as a paradoxical simplex. *Journal of Research in Personality, 46*(1), 63–78.

Dichter, G. S., Lam, K. S. L., Turner-Brown, L. M., Holtzclaw, T. N., & Bodfish, J. W. (2009). Generativity abilities predict communication deficits but not repetitive behaviors in autism spectrum disorders. *Journal of Autism and Developmental Disorders, 39*, 1298–1304.

Dickerson, A. S., Pearson, D. A., Loveland, K. A., Rahbar, M. H., & Filipek, P. A. (2014). Role of parental occupation in autism spectrum disorder diagnosis and severity. *Research in Autism Spectrum Disorders, 8*, 997–1007.

Dierker, L. C., Davis, K. F., & Sanders, B. (1995). The imaginary companion phenomenon: An analysis of personality correlates and developmental antecedents. *Dissociation, 4*, 220–228.

Dietrich, A., & Kanso, R. (2010). A review of EEG, ERP, and neuroimaging studies of creativity and insight. *Psychological Bulletin, 136*, 822.

Dinsdale, N. L., & Crespi, B. J. (2013). The borderline empathy paradox: Evidence and conceptual models for empathic enhancements in borderline personality disorder. *Journal of Personality Disorders, 27*, 172–195.

Dinsdale, N. L., Hurd, P. L., Wakabayashi, A., Elliot, M., & Crespi, B. J. (2013). How are autism and schizotypy related? Evidence from a non-clinical population. *PLoS One, 8*, e63316.

Dworzynski, K., Ronald, A., Bolton, P., & Happé, F. (2012). How different are girls and boys above and below the diagnostic threshold for autism spectrum disorders? *Journal of the American Academy of Child and Adolescent Psychiatry, 51*, 788–797. http://dx.doi.org/10.1016/j.jaac.2012.05.018.

Dyck, M., Loughead, J., Gur, R. C., Schneider, F., & Mathiak, K. (2014). Hyperactivation balances sensory processing deficits during mood induction in schizophrenia. *Social Cognitive and Affective Neuroscience, 9*, 167–175.

Dziobek, I., Fleck, S., Kalbe, E., Rogers, K., Hassenstab, J., Brand, M., ... Convit, A. (2006). Introducing MASC: A movie for the assessment of social cognition. *Journal of Autism and Developmental Disorders, 36*, 623–636.

Falter, C. M., Braeutigam, S., Nathan, R., Carrington, S., & Bailey, A. J. (2013). Enhanced access to early visual processing of perceptual simultaneity in autism spectrum disorders. *Journal of Autism and Developmental Disorders, 43*, 1857–1866.

Feelgood, S. R., & Rantzen, A. S. (1994). Auditory and visual hallucinations in university students. *Personality and Individual Differences, 17*, 293–329.

Fernyhough, C., Bland, K., Meins, E., & Coltheart, M. (2007). Imaginary companions and young children's responses to ambiguous auditory stimuli: Implications for typical and atypical development. *Journal of Child Psychology and Psychiatry, 48*, 1094–1101.

Fink, A., Grabner, R. H., Gebauer, D., Reishofer, G., Koschutnig, K., & Ebner, F. (2010). Enhancing creativity by means of cognitive stimulation: Evidence from an fMRI study. *NeuroImage, 52*, 1687–1695.

Fink, A., Slamar-Halbedl, M., Unterrainer, H. F., & Weiss, E. M. (2012). Creativity: Genius, madness, or a combination of both? *Psychology of Aesthetics, Creativity, 6*, 11–18.

Fink, A., Weber, B., Koschutnig, K., Benedek, M., Reishofer, G., Ebner, F., ... Weiss, E. M. (2014). Creativity and schizotypy from the neuroscience perspective. *Cognitive, Affective, & Behavioral Neuroscience, 14*, 378–387.

Fisher, J. E., Heller, W., & Miller, G. A. (2013). Neuropsychological differentiation of adaptive creativity and schizotypal cognition. *Personality and Individual Differences, 54*, 70–75.

Fisher, J. E., Mohanty, A., Herrington, J. D., Koven, N. S., Miller, G. A., & Heller, W. (2004). Neuropsychological evidence for dimensional schizotypy: Implications for creativity and psychopathology. *Journal of Research in Personality, 38*, 24–31.

Folley, B. S., & Park, S. (2005). Verbal creativity and schizotypal personality in relation to prefrontal hemispheric laterality: A behavioral and near-infrared optical imaging study. *Schizophrenia Research, 80*, 271–282.

Fombonne, E. (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research, 65*, 591–598.

Foster, P. S., Yung, R. C., Branch, K. K., Stringer, K. L., Ferguson, B. J., Sullivan, W., et al. (2011). Increased spreading activation in depression. *Brain and Cognition, 77*, 265–270.

Fretland, R. A., Andersson, S., Sundet, K., Andreassen, O. A., Melle, I., & Vaskinn, A. (2015). Theory of mind in schizophrenia: Error types and associations with symptoms. *Schizophrenia Research*.

Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*, 672–686.

Frith, U. (2012). Why we need cognitive explanations of autism. *The Quarterly Journal of Experimental Psychology, 65*, 2073–2092.

Fyfe, S., Williams, C., Mason, O. J., & Pickup, G. J. (2008). Apophenia, theory of mind and schizotypy: Perceiving meaning and intentionality in randomness. *Cortex, 44*, 1316–1325.

Ganis, G., Thompson, W. L., & Kosslyn, S. M. (2004). Brain areas underlying visual mental imagery and visual perception: An fMRI study. *Cognitive Brain Research, 20*, 226–241.

Giesbrecht, T., Merckelbach, H., Kater, M., & Sluis, A. F. (2007). Why dissociation and schizotypy overlap: The joint influence of fantasy proneness, cognitive failures, and childhood trauma. *The Journal of Nervous and Mental Disease, 195*, 812–818.

Gleason, T. R., Jarudi, R. N., & Cheek, J. M. (2003). Imagination, personality, and imaginary companions. *Social Behavior and Personality, 31*, 721–737.

Goddard, L., Howlin, P., Dritschel, B., & Patel, T. (2007). Autobiographical memory and social problem-solving in Asperger syndrome. *Journal of Autism and Developmental Disorders, 37*, 291–300.

Grandin, T. (1995). *Thinking in pictures*. New York: Vintage Press Random House.

Grandin, T. (2009). How does visual thinking work in the mind of a person with autism? A personal account. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 364*, 1437–1442.

Greicius, M. D., & Menon, V. (2004). Default-mode activity during a passive sensory task: Uncoupled from deactivation but impacting activation. *Journal of Cognitive Neuroscience, 16*, 1484–1492.

Hach, S., Tippett, L. J., & Addis, D. R. (2014). Neural changes associated with the generation of specific past and future events in depression. *Neuropsychologia, 65*, 41–55.

Hanson, L. K., & Atance, C. M. (2014). Brief report: Episodic foresight in autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*, 674–684.

Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences, 3*, 216–222.

Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders, 36*, 5–25.

Harkness, K. L., Washburn, D., Theriault, J. E., Lee, L., & Sabbagh, M. A. (2011). Maternal history of depression is associated with enhanced theory of mind in depressed and nondepressed adult women. *Psychiatry Research, 189*, 91–96.

Harris, P. L., & Leevers, H. J. (2000). Pretending, imagery, and self-awareness in autism. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from developmental cognitive neuroscience* (2nd ed., pp. 182–202). Oxford: Oxford University Press.

Harrison, A. J., Gamsiz, E. D., Berkowitz, I. C., Nagpal, S., & Jerskey, B. A. (2015). Genetic variation in the oxytocin receptor gene is associated with a social phenotype in autism spectrum disorders. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 168*, 720–729.

Hassabis, D., & Maguire, E. A. (2007). Deconstructing episodic memory with construction. *Trends in Cognitive Sciences, 11*, 299–306.

Hassabis, D., & Maguire, E. A. (2009). The construction system of the brain. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 364*, 1263–1271.

Hoff, E. V. (2005). Imaginary companions, creativity, and self-image in middle childhood. *Creativity Research Journal, 17*, 167–180.

Holmes, E. A., Deeprose, C., Fairburn, C. G., Wallace-Hadrill, S. M., Bonsall, M. B., Geddes, J. R., et al. (2011). Mood stability versus mood instability in bipolar disorder: A possible role for emotional mental imagery. *Behaviour Research and Therapy, 49*, 707–713.

Holmes, E. A., Geddes, J. R., Colom, F., & Goodwin, G. M. (2008). Mental imagery as an emotional amplifier: Application to bipolar disorder. *Behaviour Research and Therapy, 46*, 1251–1258.

Hommer, R. E., & Swedo, S. E. (2015). Schizophrenia and autism-related disorders. *Schizophrenia Bulletin, 41*, 313–314.

Honey, E., Leekam, S., Turner, M., & McConachie, H. (2007). Repetitive behaviour and play in typically developing children and children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37*, 1107–1115.

Iacoboni, M. (2006). Failure to deactivate in autism: The co-constitution of self and other. *Trends in Cognitive Sciences, 10*, 431–433.

Immordino-Yang, M. H., Christodoulou, J. A., & Singh, V. (2012). Rest is not idleness: Implications of the brain's default mode for human development and education. *Perspectives on Psychological Science, 7*, 352–364.

Ivins, A., Di Simplicio, M., Close, H., Goodwin, G. M., & Holmes, E. (2014). Mental imagery in bipolar affective disorder versus unipolar depression: Investigating cognitions at times of 'positive' mood. *Journal of Affective Disorders, 166*, 234–242.

Jamison, K. R. (1993). *Touched with fire: Manic-depressive illness and the creative temperament*. New York: The Free Press.

Jarrold, C. (2003). A review of research into pretend play in autism. *Autism, 7*, 379–390.

Jarrold, C., Boucher, J., & Smith, P. K. (1996). Generativity deficits in pretend play in autism. *British Journal of Developmental Psychology, 14*, 275–300.

Jauk, E., Neubauer, A. C., Dunst, B., Fink, A., & Benedek, M. (2015). Gray matter correlates of creative potential: A latent variable voxel-based morphometry study. *NeuroImage, 111*, 312–320.

Javitt, D. C. (2009a). When doors of perception close: Bottom-up models of disrupted cognition in schizophrenia. *Annual Review of Clinical Psychology, 5*, 249–275.

Javitt, D. C. (2009b). Sensory processing in schizophrenia: Neither simple nor intact. *Schizophrenia Bulletin, 35*, 1059–1064.

Jewell, E. J., & Abate, F. R. (Eds.). (2001). *New oxford American dictionary* (1st ed.. Oxford: Oxford University Press.

Jolliffe, T., & Baron-Cohen, S. (1999). A test of central coherence theory: Linguistic processing in high-functioning adults with autism or Asperger syndrome: Is local coherence impaired? *Cognition, 71*, 149–185.

Jones, V., & Steel, C. (2012). Schizotypal personality and vulnerability to involuntary autobiographical memories. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 871–876.

Jung, R. E. (2014). Evolution, creativity, intelligence, and madness: "Here Be Dragons". *Frontiers in Psychology, 5*, 784.

Jung, M., Kosaka, H., Saito, D. N., Ishitobi, M., Morita, T., Inohara, K., ... Iidaka, T. (2014). Default mode network in young male adults with autism spectrum disorder: Relationship with autism spectrum traits. *Molecular Autism, 5*, 35.

Jung, R. E., Mead, B. S., Carrasco, J., & Flores, R. A. (2013). The structure of creative cognition in the human brain. *Frontiers in Human Neuroscience, 7*.

Kana, R. K., Libero, L. E., Hu, C. P., Deshpande, H. D., & Colburn, J. S. (2014). Functional brain networks and white matter underlying Theory-of-Mind in autism. *Social Cognitive and Affective Neuroscience, 9*, 98–105.

Kanner, L. (1943). Autistic disturbances of affective contact. *The Nervous Child. Acta Paedopsychiatr, 35*, 100–136. Reprinted (1968).

Karbasforoushan, H., & Woodward, N. D. (2012). Resting-state networks in schizophrenia. *Current Topics in Medicinal Chemistry, 12*, 2404–2414.

Kaufman, J. C. (2014). *Creativity and mental illness*. Cambridge: Cambridge University Press.

Kaufman, S. B., & Paul, E. S. (2014). Creativity and schizophrenia spectrum disorders across the arts and sciences. *Frontiers in Psychology, 5*, 1145.

Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self- and other-reflection in autism. *Social Cognitive and Affective Neuroscience, 3*, 177–190.

Kennedy, D. P., Redcay, E., & Courchesne, E. (2006). Failing to deactivate: Resting functional abnormalities in autism. *Proceedings of the National Academy of Sciences USA, 103*, 8275–8280.

Kéri, S. (2009). Genes for psychosis and creativity: A promoter polymorphism of the Neuregulin 1 gene is related to creativity in people with high intellectual achievement. *Psychological Science, 20*, 1070–1073.

King, D., Dockrell, J., & Stuart, M. (2014). Constructing fictional stories: A study of story narratives by children with autistic spectrum disorder. *Research in Developmental Disabilities, 35*, 2438–2449.

King, M. J., Williams, L., MacDougall, A. G., Ferris, S., Smith, J. R. V., Ziolkowski, N., et al. (2011). Patients with bipolar disorder show a selective deficit in the episodic simulation of future events. *Consciousness and Cognition, 20*, 1801–1807.

Kirkland, R. A., Peterson, E., Baker, C. A., Miller, S., & Pulos, S. (2013). Meta-analysis reveals adult female superiority in "Reading the Mind in the Eyes Test". *North American Journal of Psychology, 15*.

Korzekwa, M. I., Dell, P. F., Links, P. S., Thabane, L., & Fougere, P. (2009). Dissociation in borderline personality disorder: A detailed look. *Journal of Trauma & Dissociation, 10*, 346–367.

Kyaga, S. (2014). *Creativity and mental illness: The mad genius in question*. Hampshire: Palgrave Macmillan.

Kyaga, S., Landén, M., Boman, M., Hultman, C. M., Långström, N., & Lichtenstein, P. (2013). Mental illness, suicide and creativity: 40-year prospective total population study. *Journal of Psychiatric Research, 47*, 83–90.

Lagioia, A., Van De Ville, D., Debbané, M., Lazeyras, F., & Eliez, S. (2010). Adolescent resting state networks and their associations with schizotypal trait expression. *Frontiers in System Neuroscience, 4*.

Lakis, N., & Mendrek, A. (2013). Individuals diagnosed with schizophrenia assign emotional importance to neutral stimuli: An FMRI study. *ISRN Psychiatry*. 965428.

Lam, K. S. L., Bodfish, J. W., & Piven, J. (2008). Evidence for three subtypes of repetitive behavior in autism that differ in familiality and association with other symptoms. *Journal of Child Psychology and Psychiatry, 49*, 1193–1200.

Landin-Romero, R., McKenna, P. J., Salgado-Pineda, P., Sarró, S., Aguirre, C., Sarri, C., ... Pomarol-Clotet, E. (2014). Failure of deactivation in the default mode network: A trait marker for schizophrenia? *Psychological Medicine*, 1–11.

Lauronen, E., Veijola, J., Isohanni, I., Jones, P. B., Nieminen, P., & Isohanni, M. (2004). Links between creativity and mental disorder. *Psychiatry, 67*, 81–98.

Leach, E. L., Hurd, P. L., & Crespi, B. J. (2013). Schizotypy, cognitive performance, and genetic risk for schizophrenia in a non-clinical population. *Personality and Individual Differences, 55*, 334–338.

Leung, A., & Chue, P. (2000). Sex differences in schizophrenia, a review of the literature. *Acta Psychiatrica Scandinavica. Supplementum, 401*, 3–38.

Li, M., Deng, W., He, Z., Wang, Q., Huang, C., Jiang, L., ... Li, T. (2015). A splitting brain: Imbalanced neural networks in schizophrenia. *Psychiatry Research, 232*, 145–153.

Lind, S. E., & Bowler, D. M. (2008). Episodic memory and autonoetic consciousness in autistic spectrum disorders: The roles of self-awareness, representational abilities and temporal cognition. *Memory in Autism: Theory and Evidence*, 166–187.

Lind, S. E., & Bowler, D. M. (2010). Episodic memory and episodic future thinking in adults with autism. *Journal of Abnormal Psychology, 119*, 896.

Liu, M., Shih, W., & Ma, L. (2011). Are children with Asperger syndrome creative in divergent thinking and feeling? A brief report. *Research in Autism Spectrum Disorders, 5*, 294–298.

Lombardo, M. V., & Baron-Cohen, S. (2011). The role of the self in mindblindness in autism. *Consciousness and Cognition, 20*, 130–140.

Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., & Baron-Cohen, S.MRC AIMS Consortium. (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. *NeuroImage, 56*, 1832–1838.

Lord, C., & Bishop, S. L. (2015). Recent advances in autism research as reflected in DSM-5 criteria for Autism Spectrum Disorder. *Annual Review of Clinical Psychology, 11*, 53–70.

Lord, C., Leventhal, B. L., & Cook, E. H. (2001). Quantifying the phenotype in autism spectrum disorders. *American Journal of Medical Genetics, 105*, 36–38.

MacQueen, G. M., Galway, T., Goldberg, J. O., & Tipper, S. P. (2003). Impaired distractor inhibition in patients with schizophrenia on a negative priming task. *Psychological Medicine, 33*, 121–129.

Manoliu, A., Meng, C., Brandl, F., Doll, A., Tahmasian, M., Scherr, M., ... Sorg, C. (2013). Insular dysfunction within the salience network is associated with severity of symptoms and aberrant inter-network connectivity in major depressive disorder. *Frontiers in Human Neuroscience, 7*.

Markram, K., & Markram, H. (2010). The intense world theory – A unifying theory of the neurobiology of autism. *Frontiers in Human Neuroscience, 4*.

Mars, R. B., Neubert, F., Noonan, M. P., Sallet, J., Toni, I., & Rushworth, M. F. S. (2012). On the relationship between the "default mode network" and the "social brain". *Frontiers in Human Neuroscience, 6*.

Matthews, N. L., Collins, K. P., Thakkar, K. N., & Park, S. (2014). Visuospatial imagery and working memory in schizophrenia. *Cognitive Neuropsychiatry, 19*, 17–35.

Mayseless, N., Eran, A., & Shamay-Tsoory, S. G. (2015). Generating original ideas: The neural underpinning of originality. *Neuroimage, 116*, 232–239.

McCabe, A., Hillier, A., & Shapiro, C. (2013). Brief report: Structure of personal narratives of adults with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 43*, 733–738.

McElroy, L. P. (1992). Early indicators of pathological dissociation in sexually abused children. *Child Abuse and Neglect, 16*, 833–846.

McGill, B., & Moulds, M. L. (2014). Characteristics of autobiographical memories and prospective imagery across a spectrum of hypomanic personality traits. *Memory, 22*, 1139–1148.

McLewin, L. A., & Muller, R. T. (2006). Attachment and social support in the prediction of psychopathology among young adults with and without a history of physical maltreatment. *Child Abuse and Neglect, 30*, 171–191.

McMillan, R. L., Kaufman, S. B., & Singer, J. L. (2013). Ode to positive constructive daydreaming. *Frontiers in Psychology, 4*, 626.

Mendrek, A. (2015). Is it important to consider sex and gender in neurocognitive studies? *Front Psychiatry, 6*, 83.

Mendrek, A., & Stip, E. (2011). Sexual dimorphism in schizophrenia: Is there a need for gender-based protocols? *Expert Review of Neurotherapeutics, 11*, 951–959.

Merckelbach, H., Rassin, E., & Muris, P. (2000). Dissociation, schizotypy, and fantasy proneness in undergraduate students. *The Journal of Nervous and Mental Disease, 188*, 428–431.

Meyer, T. D., Finucane, L., & Jordan, G. (2011). Is risk for mania associated with increased daydreaming as a form of mental imagery? *Journal of Affective Disorders, 135*, 380–383.

Mooneyham, B. W., & Schooler, J. W. (2013). The costs and benefits of mind-wandering: A review. *Canadian Journal of Experimental Psychology, 67*, 11–18.

Morina, N., Deeprose, C., Pusowski, C., Schmid, M., & Holmes, E. A. (2011). Prospective mental imagery in patients with major depressive disorder or anxiety disorders. *Journal of Anxiety Disorders, 25*, 1032–1037.

Morrison, A. P., Beck, A. T., Glentworth, D., Dunn, H., Reid, G. S., Larkin, W., et al. (2002). Imagery and psychotic symptoms: A preliminary investigation. *Behaviour Research and Therapy, 40*, 1053–1062.

Moskowitz, A., & Heim, G. (2011). Eugen Bleuler's dementia praecox or the group of schizophrenias (1911): A centenary appreciation and reconsideration. *Schizophrenia Bulletin, 37*, 471–479.

Mottron, L., Dawson, M., & Soulières, I. (2009). Enhanced perception in savant syndrome: Patterns, structure and creativity. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 364*, 1385–1391.

Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. A. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders, 36*, 27–43.

Mullally, S. L., & Maguire, E. A. (2013). Memory, imagination, and predicting the future: A common brain mechanism? *Neuroscientist, 20*, 220–234.

Murdaugh, D. L., Shinkareva, S. V., Deshpande, H. R., Wang, J., Pennick, M. R., & Kana, R. K. (2012). Differential deactivation during mentalizing and classification of autism based on default mode network connectivity. *PLoS One, 7*, e50064. http://dx.doi.org/10.1371/journal.pone.0050064.

Murray, G., & Johnson, S. L. (2010). The clinical significance of creativity in bipolar disorder. *Clinical Psychology Review, 30*, 721–732.
Nettle, D. (2001). *Strong imagination: Madness, creativity and human nature*. New York: Oxford University Press.
Nettle, D. (2006). The evolution of personality variation in humans and other animals. *American Psychologist, 61*, 622.
O'Reilly, T., Dunbar, R., & Bentall, R. (2001). Schizotypy and creativity: An evolutionary connection? *Personality and Individual Differences, 31*, 1067–1078.
Østby, Y., Walhovd, K. B., Tamnes, C. K., Grydeland, H., Westlye, L. T., & Fjell, A. M. (2012). Mental time travel and default-mode network functional connectivity in the developing brain. *Proceedings of the National Academy of Sciences USA, 109*, 16800–16804.
Ottemiller, D. D., Elliott, C. S., & Giovannetti, T. (2014). Creativity, overinclusion, and everyday tasks. *Creativity Research Journal, 26*, 289–296.
Pearson, D. G., Deeprose, C., Wallace-Hadrill, S. M. A., Heyes, S. B., & Holmes, E. A. (2013). Assessing mental imagery in clinical psychology: A review of imagery measures and a guiding framework. *Clinical Psychology Review, 33*, 1–23.
Pearson, J., & Kosslyn, S. M. (2015). The heterogeneity of mental representation: Ending the imagery debate. *Proceedings of the National Academy of Sciences, 112* (33), 10089–10092.
Pearson, D., Rouse, H., Doswell, S., Ainsworth, C., Dawson, O., Simms, K., et al. (2001). Prevalence of imaginary companions in a normal child population. *Child: Care, Health and Development, 27*(1), 13–22.
Peeters, S. C., van de Ven, V., Gronenschild, E. H., Patel, A. X., Habets, P., Goebel, R., ... Marcelis, M. (2015). Default mode network connectivity as a function of familial and environmental risk for psychotic disorder. *PLoS One, 10*, e0120030.
Piffer, D., & Hur, Y. (2014). Heritability of creative achievement. *Creativity Research Journal, 26*, 151–157.
Ploog, B. O. (2010). Stimulus overselectivity four decades later: A review of the literature and its implications for current research in autism spectrum disorder. *Journal of Autism and Developmental Disorders, 40*, 1332–1349.
Power, R. A., Steinberg, S., Bjornsdottir, G., Rietveld, C. A., Abdellaoui, A., Nivard, M. M., ... Cesarini, D. (2015). Polygenic risk scores for schizophrenia and bipolar disorder predict creativity. *Nature Neuroscience, 18*, 953–955.
Propper, R., & Christman, S. (2004). Mixed-versus strong right-handedness is associated with biases towards "remember" versus "know" judgements in recognition memory: Role of interhemispheric interaction. *Memory, 12*, 707–714.
Propper, R. E., Christman, S. D., & Phaneuf, K. A. (2005). A mixed-handed advantage in episodic memory: A possible role of interhemispheric interaction. *Memory & Cognition, 33*, 751–757.
Putnam, F. W., Helmers, K., & Trickett, P. K. (1993). Development, reliability, and validity of a child dissociation scale. *Child Abuse and Neglect, 17*, 731–741.
Putnam, F. W., & Peterson, G. (1994). Further validation of the child dissociative checklist. *Dissociation: Progress in the Dissociative Disorders*.
R Core Team (2014). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria. <http://www.R-project.org/>.
Raffard, S., D'Argembeau, A., Bayard, S., Boulenger, J., & Van der Linden, M. (2010). Scene construction in schizophrenia. *Neuropsychology, 24*, 608.
Raffard, S., D'Argembeau, A., Lardi, C., Bayard, S., Boulenger, J., & Van der Linden, M. (2010). Narrative identity in schizophrenia. *Consciousness and Cognition, 19*, 328–340.
Rakic, P. (2009). Evolution of the neocortex: A perspective from developmental biology. *Nature Reviews Neuroscience, 10*, 724–735.
Rasmussen, A. R., & Parnas, J. (2015). Pathologies of imagination in schizophrenia spectrum disorders. *Acta Psychiatrica Scandinavica, 131*, 157–161.
Rhue, J. W., Lynn, S. J., & Sandberg, D. (1995). Dissociation, fantasy and imagination in childhood: A comparison of physically abused, sexually abused, and non-abused children. *Contemporary Hypnosis*.
Ricarte, J. J., Hernández-Viadel, J. V., Latorre, J. M., Ros, L., & Serrano, J. P. (2014). Effects of specific positive events training on autobiographical memories in people with schizophrenia. *Cognitive Therapy and Research, 38*, 407–415.
Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with Autism Spectrum Disorders. *Development and Psychopathology, 22*, 55–69.
Ripke, S., Neale, B. M., Corvin, A., Walters, J. T., Farh, K. H., Holmans, P. A., ... Pers, T. H. (2014). Biological insights from 108 schizophrenia-associated genetic loci. *Nature, 511*, 421.
Rose, E. J., Greene, C., Kelly, S., Morris, D. W., Robertson, I. H., Fahey, C., ... Bokde, A. (2012). The NOS1 variant rs6490121 is associated with variation in prefrontal function and grey matter density in healthy individuals. *Neuroimage, 60*(1), 614–622.
Roth, I. (2007). Autism and the imaginative mind. In I. Roth (Ed.). *Proceedings of the British academy: Imaginative minds* (Vol. 147, pp. 277–306). Oxford: Oxford University Press.
Ruiter, M., & Johnson, S. L. (2015). Mania risk and creativity: A multi-method study of the role of motivation. *Journal of Affective Disorders, 170*, 52–58.
Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Developmental Disorders, 2*, 315–337.
Rutter, M., Le Couteur, A., & Lord, C. (2003) *ADI-R: autism diagnostic interview – Revised*. Western Psychological Services, Los Angeles, CA.
Ryman, S. G., van den Heuvel, M. P., Yeo, R. A., Caprihan, A., Carrasco, J., Vakhtin, A. A., ... Jung, R. E. (2014). Sex differences in the relationship between white matter connectivity and creativity. *NeuroImage, 101*, 380–389.
Sahyoun, C. P., Belliveau, J. W., Soulières, I., Schwartz, S., & Mody, M. (2010). Neuroimaging of the functional and structural networks underlying visuospatial vs. linguistic reasoning in high-functioning autism. *Neuropsychologia, 48*, 86–95.
Sahyoun, C. P., Soulières, I., Belliveau, J. W., Mottron, L., & Mody, M. (2009). Cognitive differences in pictorial reasoning between high-functioning autism and Asperger's syndrome. *Journal of Autism and Developmental Disorders, 39*, 1014–1023.
Sawyer, K. (2011). The cognitive neuroscience of creativity: A critical review. *Creativity Research Journal, 23*, 137–154.
Saxe, R. (2006). Uniquely human social cognition. *Current Opinion in Neurobiology, 16*, 235–239.
Schacter, D. L., Addis, D. R., Hassabis, D., Martin, V. C., Spreng, R. N., & Szpunar, K. K. (2012). The future of memory: Remembering, imagining, and the brain. *Neuron, 76*, 677–694.
Schäfer, I., Fisher, H. L., Aderhold, V., Huber, B., Hoffmann-Langer, L., Golks, D., ... Harfst, T. (2012). Dissociative symptoms in patients with schizophrenia: Relationships with childhood trauma and psychotic symptoms. *Comprehensive Psychiatry, 53*, 364–371.
Schilbach, L., Eickhoff, S. B., Rotarska-Jagiela, A., Fink, G. R., & Vogeley, K. (2008). Minds at rest? Social cognition as the default mode of cognizing and its putative relationship to the "default system" of the brain. *Consciousness and Cognition, 17*, 457–467.
Schwab, S. G., & Wildenauer, D. B. (2013). Genetics of psychiatric disorders in the GWAS Taera: An update on schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience, 263*(Suppl 2), S147–S154. http://dx.doi.org/10.1007/s00406-013-0450-z.
Schwarzer, G. (2014). *Meta: Meta-analysis with R*. R package version 3.7-1. <http://CRAN.R-project.org/package=meta>.
Scott, F. J., & Baron-Cohen, S. (1996). Imagining real and unreal things: Evidence of a dissociation in autism. *Journal of Cognitive Neuroscience, 8*, 371–382.
Seiffge-Krenke, I. (1997). Imaginary companions in adolescence: Sign of a deficient or positive development? *Journal of Adolescence, 20*, 137–154.
Sharp, C., Ha, C., Carbone, C., Kim, S., Perry, K., Williams, L., et al. (2013). Hypermentalizing in adolescent inpatients: Treatment effects and association with borderline traits. *Journal of Personality Disorders, 27*, 3–18.
Sharp, C., & Venta, A. (2012). Mentalizing problems in children and adolescents. In N. Midgley & I. Vrouva (Eds.), *Minding the child: Mentalization-based interventions with children, young people and their families* (pp. 35–53). New York: Routledge.
Shin, D. J., Lee, T. Y., Jung, W. H., Kim, S. N., Jang, J. H., & Kwo, J. S. (2015). Away from home: The brain of the wandering mind as a model for schizophrenia. *Schizophrenia Research, 165*, 83–89.
Shirar, L. (1996). *Dissociative children: Bridging the inner and outer worlds*. New York: WW Norton & Company.
Silver, H., & Feldman, P. (2005). Evidence for sustained attention and working memory in schizophrenia sharing a common mechanism. *Journal of Neuropsychiatry and Clinical Neurosciences, 17*, 391–398.
Slotnick, S. D., Thompson, W. L., & Kosslyn, S. M. (2012). Visual memory and visual mental imagery recruit common control and sensory regions of the brain. *Cognitive Neuroscience, 3*, 14–20.
Smallwood, J., Gorgolewski, K. J., Golchert, J., Ruby, F. J. M., Engen, H., Baird, B., ... Margulies, D. S. (2013a). The default modes of reading: Modulation of posterior cingulate and medial prefrontal cortex connectivity associated with comprehension and task focus while reading. *Frontiers in Human Neuroscience, 7*.
Smallwood, J., Ruby, F. J. M., & Singer, T. (2013b). Letting go of the present: Mind-wandering is associated with reduced delay discounting. *Consciousness and Cognition, 22*, 1–7.
Smallwood, J., & Schooler, J. W. (2015). The science of mind wandering: Empirically navigating the stream of consciousness. *Annual Review of Psychology, 66*, 487–518.
Snyder, A. W., & Thomas, M. (1997). Autistic artists give clues to cognition. *Perception London, 26*, 93–96.
Somers, M., Sommer, I. E., Boks, M. P., & Kahn, R. S. (2009). Hand-preference and population schizotypy: A meta-analysis. *Schizophrenia Research, 108*, 25–32.
Sommer, I., Ramsey, N., Kahn, R., Aleman, A., & Bouma, A. (2001). Handedness, language lateralisation and anatomical asymmetry in schizophrenia meta-analysis. *British Journal of Psychiatry, 178*, 344–351.
Soulières, I., Dawson, M., Samson, F., Barbeau, E. B., Sahyoun, C. P., Strangman, G. E., ... Mottron, L. (2009). Enhanced visual processing contributes to matrix reasoning in autism. *Human Brain Mapping, 30*, 4082–4107.
Spek, A. A., & Velderman, E. (2013). Examining the relationship between Autism Spectrum Disorders and technical professions in high functioning adults. *Research in Autism Spectrum Disorders, 7*, 606–612.
Spencer, M. D., Chura, L. R., Holt, R. J., Suckling, J., Calder, A. J., Bullmore, E. T., et al. (2012). Failure to deactivate the default mode network indicates a possible endophenotype of autism. *Molecular Autism, 3*, 1–9.
Spreng, R. N., & Grady, C. L. (2010). Patterns of brain activity supporting autobiographical memory, prospection, and theory of mind, and their relationship to the default mode network. *Journal of Cognitive Neuroscience, 22*, 1112–1123.
Stawarczyk, D., Cassol, H., & D'Argembeau, A. (2013). Phenomenology of future-oriented mind-wandering episodes. *Frontiers in Psychology, 4*.

Stawarczyk, D., & D'Argembeau, A. (2015). Neural correlates of personal goal processing during episodic future thinking and mind-wandering: An ALE meta-analysis. *Human Brain Mapping, 36*, 2928–2947.
Stein, M. I. (1953). Creativity and culture. *Journal of Psychology, 36*, 311–322.
Suddendorf, T., Addis, D. R., & Corballis, M. C. (2009). Mental time travel and the shaping of the human mind. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 364*, 1317–1324.
Suddendorf, T., & Corballis, M. C. (1997). Mental time travel and the evolution of the human mind. *Genetic, Social, and General Psychology Monographs, 123*, 133–167.
Suddendorf, T., & Corballis, M. C. (2007). The evolution of foresight: What is mental time travel, and is it unique to humans? *Behavioral and Brain Sciences, 30*, 299–313.
Takeuchi, H., Taki, Y., Hashizume, H., Sassa, Y., Nagase, T., Nouchi, R., et al. (2011). Failing to deactivate: The association between brain activity during a working memory task and creativity. *NeuroImage, 55*, 681–687.
Takeuchi, H., Taki, Y., Hashizume, H., Sassa, Y., Nagase, T., Nouchi, R., et al. (2012). The association between resting functional connectivity and creativity. *Cerebral Cortex, 22*, 2921–2929.
Tang, J., Liao, Y., Song, M., Gao, J., Zhou, B., Tan, C., ... Chen, X. (2013). Aberrant default mode functional connectivity in early onset schizophrenia. *PLoS One, 8*, e71061.
Tavassoli, T., Miller, L. J., Schoen, S. A., Nielsen, D. M., & Baron-Cohen, S. (2013). Sensory over-responsivity in adults with autism spectrum conditions. *Autism*. 1362361313477246.
Taylor, M., & Carlson, S. M. (1997). The relation between individual differences in fantasy and theory of mind. *Child Development, 68*, 436–455.
Ten Eycke, K. D., & Müller, U. (2015). Brief report: New evidence for a social-specific imagination deficit in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 45*, 213–220.
Terrett, G., Rendell, P. G., Raponi-Saunders, S., Henry, J. D., Bailey, P. E., & Altgassen, M. (2013). Episodic future thinking in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 43*, 2558–2568.
Thomson, P., & Jaque, S. V. (2012). Dancing with the Muses: Dissociation and flow. *Journal of Trauma & Dissociation, 13*, 478–489.
Tomasi, D., & Volkow, N. D. (2012). Gender differences in brain functional connectivity density. *Human Brain Mapping, 33*, 849–860. http://dx.doi.org/10.1002/hbm.21252.
Uddin, L. Q. (2011). The self in autism: An emerging view from neuroimaging. *Neurocase, 17*, 201–208.
Uddin, L. Q., Iacoboni, M., Lange, C., & Keenan, J. P. (2007). The self and social cognition: The role of cortical midline structures and mirror neurons. *Trends in Cognitive Sciences, 11*, 153–157.
Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., et al. (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review, 121*, 649.
Van Os, J. (2009). 'Salience syndrome' replaces 'schizophrenia' in DSM-V and ICD-11: Psychiatry's evidence-based entry into the 21st century? *Acta Psychiatrica Scandinavica, 120*, 363–372.
Verhaeghen, P., Joorman, J., & Aikman, S. N. (2014). Creativity, mood, and the examined life: Self-reflective rumination boosts creativity, brooding breeds dysphoria. *Psychology of Aesthetics, Creativity, 8*, 211.
Verhaeghen, P., Joorman, J., & Khan, R. (2005). Why we sing the blues: The relation between self-reflective rumination, mood, and creativity. *Emotion, 5*, 226.
von dem Hagen, E. A. H., Stoyanova, R. S., Baron-Cohen, S., & Calder, A. J. (2012). Reduced functional connectivity within and between 'social' resting state networks in autism spectrum conditions. *Social Cognitive and Affective Neuroscience, nss053*.
Vygotsky, L. S. (2004). Imagination and creativity in childhood. *Journal of Russian & East European Psychology, 42*, 7–97.
Vygotsky, L. S. (2012). *Thought and language*. Cambridge MA: MIT press.
Waldo, T. G., & Merritt, R. D. (2000). Fantasy proneness, dissociation, and DSM-IV Axis II symptomatology. *Journal of Abnormal Psychology, 109*, 555.
Wei, D., Yang, J., Li, W., Wang, K., Zhang, Q., & Qiu, J. (2014). Increased resting functional connectivity of the medial prefrontal cortex in creativity by means of cognitive stimulation. *Cortex, 51*, 92–102.
Wheelwright, S., & Baron-Cohen, S. (2001). The link between autism and skills such as engineering, maths, physics and computing – A reply to Jarrold and Routh. *Autism, 5*, 223–227.
Whitfield-Gabrieli, S., & Ford, J. M. (2012). Default mode network activity and connectivity in psychopathology. *Annual Review of Clinical Psychology, 8*, 49–76.
Wible, C. G. (2012a). Schizophrenia as a disorder of social communication. *Schizophrenia Research and Treatment, 2012*, 920485.
Wible, C. G. (2012b). Hippocampal temporal-parietal junction interaction in the production of psychotic symptoms: A framework for understanding the schizophrenic syndrome. *Frontiers in Human Neuroscience, 6*.
Winfield, H., & Kamboj, S. K. (2010). Schizotypy and mental time travel. *Consciousness and Cognition, 19*, 321–327.
Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*, 11–29.
Winton-Brown, T. T., Fusar-Poli, P., Ungless, M. A., & Howes, O. D. (2014). Dopaminergic basis of salience dysregulation in psychosis. *Trends in Neurosciences, 37*, 85–94.
Wise, R. J. S., & Braga, R. M. (2014). Default mode network: The seat of literary creativity? *Trends in Cognitive Sciences, 18*, 116–117.
Wolfberg, P. J. (2009). *Play and imagination in children with autism*. Shawnee Mission, KS: AAPC Publishing.
Woodard, C. R., Chung, J., & Korn, M. (2014). A pilot study of the meta-play method: A novel play intervention for toddlers with autism. *Journal of Autism, 1*, 3.
Woodard, C. R., & Van Reet, J. (2011). Object identification and imagination: An alternative to the meta-representational explanation of autism. *Journal of Autism and Developmental Disorders, 41*, 213–226.
Wray, N. R., Lee, S. H., Mehta, D., Vinkhuyzen, A. A., Dudbridge, F., & Middeldorp, C. M. (2014). Research review: Polygenic methods and their application to psychiatric traits. *Journal of Child Psychology and Psychiatry, 55*, 1068–1087.
Young, L. N., Winner, E., & Cordes, S. (2013). Heightened incidence of depressive symptoms in adolescents involved in the arts. *Psychology of Aesthetics, Creativity, 7*, 197.
Zabelina, D. L., O'Leary, D., Pornpattananangkul, N., Nusslock, R., & Beeman, M. (2015). Creativity and sensory gating indexed by the P50: Selective versus leaky sensory gating in divergent thinkers and creative achievers. *Neuropsychologia, 69*, 77–84.
Zahn-Waxler, C., & Van Hulle, C. (2011). Empathy, guilt, and depression: When caring for others becomes costly to children. In B. Oakley, A. Knafo, G. Madhavan, & D. S. Wilson (Eds.), *Pathological altruism* (pp. 321–344). New York: Oxford University Press.
Zvyagintsev, M., Clemens, B., Chechko, N., Mathiak, K. A., Sack, A. T., & Mathiak, K. (2013). Brain networks underlying mental imagery of auditory and visual information. *European Journal of Neuroscience, 37*, 1421–1434.