Review

Disorganisation, thought disorder and socio-cognitive functioning in schizophrenia spectrum disorders

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Background

Poor social cognition is prevalent in schizophrenia spectrum disorders. Some authors argue that these effects are symptom-specific and that socio-cognitive difficulties (e.g. theory of mind) are strongly associated with thought disorder and symptoms of disorganisation.

Aims

The current review tests the strength of this association.

Method

We meta-analysed studies published between 1980 and 2016 that tested the association between social cognition and these symptoms in schizophrenia spectrum disorders.

Results

Our search (PsycINFO, MEDLINE and Web of Science) identified 123 studies ($N = 9107$). Overall effect size as $r = -0.313$, indicating a moderate association between symptoms and social cognition. Subanalyses yielded a moderate association between symptoms and theory of mind ($r = -0.349$) and emotion

recognition ($r = -0.334$), but smaller effect sizes for social perception ($r = -0.188$), emotion regulation ($r = -0.169$) and attributional biases ($r = -0.143$).

Conclusions

The association is interpreted within models of communication that highlight the importance of mentalisation and processing of partner-specific cues in conversational alignment and grounding.

Declaration of interests

None.

Keywords

Schizophrenia; social cognition; theory of mind; emotion recognition; emotion processing; social perception; thought disorder; disorganisation; psychosis.

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'No matter how one may try, one cannot not communicate'
(Watzlawick *et al*, 1967: pp. 48[1])

Researchers in the field of psychosis have long been interested in the role of social cognition in psychotic experiences.[2,3] Consequently, there is now a wealth of meta-analytical evidence showing that deficits in theory of mind (ToM; the ability to infer mental states in others), social perception and emotion recognition are highly prevalent in individuals with schizophrenia spectrum diagnoses.[4,5] Some researchers have suggested that impairments in social cognition play a specific role in disorganised symptoms in schizophrenia spectrum disorders, especially thought disorder.[3,6] Here, we report a statistical synthesis of the evidence on the association between domains of social cognition and thought disorder and other symptoms of disorganisation in participants diagnosed with schizophrenia spectrum disorders.

Socio-cognitive domains

A National Institute of Mental Health workshop defined social cognition as a set of '(the) mental operations that underlie social interactions, including perceiving, interpreting, and generating responses to the intentions, dispositions, and behaviors of others' (Green *et al* 2008: pp.1211[7]). Hence, social cognition is a multifaceted construct, referring to a broad range of higher-level inferential, attributional and regulatory processes, as well as lower-level social cue perception and processing. The consensus is that these processes comprise four core domains, namely: ToM and mental state attribution, social perception, attributional style or biases, and emotion processing.[8] Some have distinguished a fifth domain, referred to as emotion recognition. This encompasses lower-level emotional cue perception and identification (see supplementary table a available at https://doi.org/10.1192/bjp.2018.160 for definition of domains and examples of tasks).

ToM and mental state attribution

ToM (or mental state attribution) refers to the ability of the individual to infer intentions, dispositions and beliefs in others from their speech, actions and/or non-verbal behaviour.[3,9] Relevant assessment tasks may involve reading short passages describing social interactions, where intentions of the characters are inferred from hints or indirect speech acts (e.g. hinting task).[2] Alternatively, participants may be asked to sequence picture-card stories that require the correct inference of false beliefs to understand the story plot (e.g. picture sequencing task).[10]

Social perception

Social perception refers to the ability to decode and interpret social cues (verbal and non-verbal) in an interpersonal situation. This involves both the correct interpretation of cues in a social context and the processing of social knowledge (i.e. the ability to utilise roles, rules and goals in a social situation and the knowledge of how they affect other peoples' behaviours). In some tasks, participants are presented with social situations followed by multiple-choice questions that test their ability to interpret cues about social roles and rules (e.g. Interpersonal Perception Task).[11] Alternatively, tasks may involve the presentation of short audio and video clips that test the accurate interpretation of body postures, gestures, facial expressions or voice cues (e.g. Profile of Nonverbal Sensitivity Test).[12]

Emotion recognition

Emotion recognition refers to the ability to identify human emotion from a range of stimuli and cues, such as facial expressions or tone of voice. Emotion recognition tasks may involve the ability to correctly identify different emotional states from video clips of an actor performing facial, vocal-tonal and upper-body movement

cues (e.g. Bell–Lysaker Emotion Recognition Task)[13] or the identification of different emotional states from the tone of voice of audiotaped speakers reading out loud sentences of neutral content (e.g. Voice Emotion Identification Test).[14]

Attributional bias/style

Attributional bias refers to quick causal inferences that individuals make about positive and negative social events. These inferences (or attributions) are typically classified as external (i.e. the cause is attributed to others) or internal (i.e. cause is attributed to self). Sometimes, external attributions may be classified as personal (i.e. cause is the actions of another person) or situational (i.e. cause is attributed to situational factors). Tasks involve asking the participants to imagine themselves in a positive or negative social situation and to report the most likely causal explanation for an event. Example measures include the Attributional Style Questionnaire[15] and the Internal, Personal, and Situational Attributions Questionnaire.[16]

Emotion processing and regulation

Emotion processing refers to skills that range from the perception of emotion to the understanding and management (regulation) of emotions. Although, some of these skills overlap with the competencies involved in emotion recognition, the construct is broader and encompasses affective regulatory strategies. The assessment of emotional processing can involve questionnaire measures (e.g. Emotion Regulation Questionnaire)[17] or tasks where the participant is asked to rate brief vignettes that tap into the management, regulation or facilitation of emotions (e.g. Mayer–Salovey–Caruso Emotional Intelligence Test).[18]

Thought disorder and cognitive disorganisation

Thought disorder refers to range of thinking, linguistic and communication atypicalities that render the speech and communication of some individuals difficult to follow and apparently unintelligible.[19] These symptoms are a relatively enduring feature in patients with psychosis[20] and have been associated with poorer quality of life,[21] higher rates of readmissions[22] and poorer occupational and social functioning.[23,24] Perhaps more importantly, thought disorder in patients with psychosis has been associated with poor therapeutic alliance,[25] a core process in cognitive behavioural therapy for psychosis.[26] Despite a considerable amount of research in the field, the processes and mechanisms involved in thought disorder are still unclear.[27,28] However, such knowledge may be important for the development of effective psychological treatments for thought disorder.

Some authors have argued that no single mechanism will ever be able to explain the full range of symptoms of thought disorder because it is highly heterogeneous cluster of experiences and behaviours.[27] Although, there is no final word regarding the number of factors involved in thought disorder,[29] it is clear that a distinction can be made between an impoverished speech factor, which includes symptoms such as alogia (or poverty of speech), and a disorganisation factor, which includes symptoms such as derailment, tangentiality or incoherence.[30] This dichotomy has also been referred to as negative and positive thought disorder. Thought disorder assessment scales, such as the Scale for the Assessment of Thought, Language and Communication Disorders (TLC)[31] or the Thought Language Index,[32] distinguish between poverty of speech and disorganisation items, and such differentiation has been further supported by factor analytical studies[33] and studies on the psychological mechanisms of both positive and negative thought disorder.[34,35]

Many studies have used measurements obtained by general psychopathology scales (e.g. Positive and Negative Syndrome Scale (PANSS)[36] or the Brief Psychiatric Rating Scale (BPRS)[37]) to test hypotheses about the mechanisms involved in thought disorder. These include single ratings of conceptual disorganisation or symptom factors. The single ratings are highly correlated with more extensive measures of thought disorder[38] and they capture symptoms of disorganisation such as derailment, incoherence or illogicality (i.e. positive thought disorder) but not symptoms of cognitive impoverishment such as alogia or poverty of speech. The symptom factors, which are derived from factor analysis and are typically labelled in the literature as 'disorganisation' or 'cognitive' factors, seem to form an orthogonal cluster of experiences distinct from positive and negative symptoms in schizophrenia spectrum disorders.[39] They are highly associated with positive thought disorder but not alogia or poverty of speech.[40] A further problem is that they tend to encompass variance from PANSS items such as tension, inappropriate affect or mannerisms and posturing, experiences that would not normally fall under the category of thought disorder.[41]

Because of the conceptual and methodological reasons outlined above, we felt that it was important that our analytical strategy distinguished between nuanced constructs, which code different and, at times, distinct phenomena.

Social cognition, thought disorder and cognitive disorganisation

One study has suggested that patients with thought disorder might be aware of their communication difficulties.[42] However, some studies have reported some inconsistency between patient-reported and clinician-rated thought disorder,[43,44] and others have reported that patients seem to be unaware that their verbalisations are idiosyncratic and difficult to follow despite being able to successfully judge the verbalisations of other patients with thought disorder as bizarre and atypical.[45] This apparent inability to shift perspective, repair communication and cooperatively adjust the message to the needs (and level of knowledge) of the listener is crucial when communication goes awry,[46] and has been highlighted by several authors as a crucial feature in thought disorder. For example, Frith[3] suggested that difficulties inferring the state of knowledge, intentions and beliefs of an interlocutor, together with difficulties in interpreting the interlocutor's social signals, could prevent repair when communication fails, thereby leading to speech being perceived by the interlocutor as tangential or derailed. Similarly, Hardy–Baylé et al[6] suggested that symptoms of disorganisation in patients diagnosed with schizophrenia spectrum disorders could be explained by difficulties in representing other peoples' mental states and integrating contextual information during conversations. These hypotheses have been partially supported in a review[47] and a meta-analysis[5] of the literature on ToM in patients diagnosed with schizophrenia spectrum disorders, but difficulties with ToM do not occur in isolation from other kinds of deficits,[48] and it is therefore likely that other domains of social cognition may also be important in thought disorder.

For example, Toomey et al found significant associations between poor social perception and symptoms of disorganisation in patients,[49] and Kee et al found significant associations between disorganisation and poor emotion recognition.[50] It is not difficult to offer interpretations of these findings. For example, stilted speech (pedantic speech that is excessively formal and inappropriate for the context of the conversation)[31] could be partially explained by poor social perception (speaking with excessive formality when the social context requires a more informal style). Although hypotheses such as these are speculative at this time, they highlight the value of exploring a wide range of domains of social cognition in relation to thought disorder and disorganisation.

Study aim

The aim of this review was to quantify the strength of the association between different domains of social cognition and thought disorder, disorganisation and alogia in schizophrenia spectrum disorders.

Method

This review was carried out in adherence to the Meta-Analysis of Observational Studies in Epidemiology guidelines[51] and the general principles of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses statement for reporting systematic reviews and meta-analyses.[52]

Literature search

After initial scoping searches, three electronic databases (PsycINFO, MEDLINE and Web of Science) were searched for papers published between 1980 and 2016, using the following search terms: social cognition OR theory of mind OR theory-of-mind OR mentali$ation OR mental state attribution OR affect* OR emotion* (recognition or identification or regulation or management or processing or perception) social perception OR social knowledge OR attribution* (bias* or style) AND schizophreni* OR psychos* AND formal thought disorder OR thought dis* OR thinking dis* OR disorgani* OR conceptual dis* OR cognitive dis* OR communication dis*. The three searches yielded a total of 3077 records (Fig. 1).

Study selection

The inclusion criteria were: (a) the study was published in English language, (b) the paper was fully accessible, (c) the study was published in a peer-reviewed journal, (d) the sample was composed of patients diagnosed with schizophrenia spectrum disorders, (e) a clear thought disorder or disorganisation measure could be identified, (f) a socio-cognitive measure could be identified and (g) statistical data were available for extraction.

Although thought disorder is a transdiagnostic phenomenon that can be observed in different mental health conditions,[27] we have opted to exclude studies with patients with other diagnoses (e.g. bipolar affective disorder) because there are significant differences across diagnoses on course, quality and temporal stability of these experiences.[53–55]

Symptom grouping strategy

To test the effect of different symptoms on social cognition, we organised the effect sizes into three different symptom groups: disorganisation (factor), alogia (poverty of speech) and thought disorder. The first group included effect sizes from studies where researchers calculated the association between social cognition and a symptom factor (e.g. 'disorganisation factor' or 'cognitive factor') derived from clinical symptom scale (e.g. PANSS or BPRS). These factors were likely to include variance from symptoms that, despite being statistically associated with thought disorder, do not represent what would normally be assumed to fall under the remit of the construct (e.g. tension, mannerisms and posturing).[56] The second group (alogia) included effect sizes from studies where extractable data for the association between social cognition and a single item for alogia or poverty of speech was provided. These were almost always clinical symptom scales such as the SANS (Scale for the Assessment of Negative Symptoms).[57] Finally, our third group (thought disorder) included data from studies where effect sizes were calculated from a thought disorder-specific scale score (e.g. TLC[58] or Bizarre Idiosyncratic
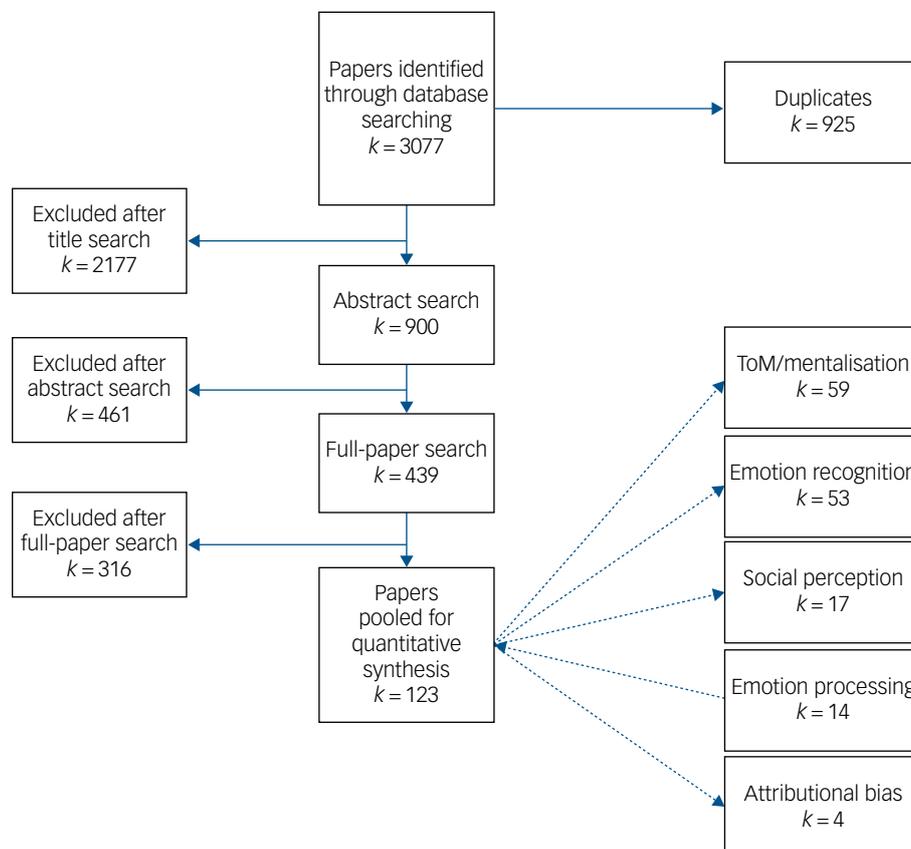

Fig. 1 Flowchart of the different stages of the systematic search. ToM, theory of mind.

Thinking Scale[59]) or from a single item (other than alogia or poverty of speech) from a clinical rating scale (e.g. PANSS items stereotyped thinking or conceptual disorganisation[60,61]). In these cases, we opted to maintain the original designation used by the authors (see supplementary materials 6). Also included in this symptom group were effect sizes that had been estimated from clinical symptom scales that have specific thought disorder subscales (e.g. SAPS (Scale for the Assessment of Positive Symptoms)[62]). The analyses of this group included an effect size for the group as whole and then a second estimate for studies that have used only thought disorder-specific measures (without the scores from single-item clinical rating scales). The reason for this is to understand the strength of the estimate when thought disorder is measured with robust (multi-item) and purposely designed measures.

Statistical analyses

Statistical analyses were carried out with CMA (Comprehensive Meta-Analysis). Overall effect size was estimated by Pearson's correlation coefficient (r) and random effects analysis, given the likelihood that our analysis would carry a substantial amount of variation across studies. In studies with multiple socio-cognitive scores within the same domain, effect size was computed from the average across tasks so that overall effect size could be computed from a single estimate by study.

Heterogeneity was measured with τ^2, Q and I^2, and sensitivity analysis was carried out with group comparisons and meta-regression. Publication bias was tested by the visual inspection of the funnel plot, Begg and Mazumdar's rank order correlation, Egger's regression intercept and Duval and Tweedie's 'trim and fill' procedure.

Results

Study and sample characteristics

Our search identified 123 studies with extractable data. The demographic and clinical characteristics of the studies can be found in

Table 1 Demographic and clinical variables

Study characteristics		$k = 123$
Design	Cross-sectional (%)	114 (92.68%)
	Longitudinal (%)	9 (7.32%)
Sample size	Total	9107
Gender	Male (%)	6338 (69.59%)
	Female (%)	2573 (28.25%)
Age	Mean (s.d.)	36.61 (6.27)
Status	Out-patient (%)	56 (45.16%)
	In-patient (%)	31 (25.00%)
	Mixed (%)	37 (29.84%)
Diagnostic label	Schizophrenia (%)	63 (51.22%)
	Spectrum (%)	60 (48.78%)
Diagnostic criteria	DSM-III-R or above (%)	118 (95.93%)
Socio-cognitive domain	Theory of mind (%)	59 (40.14%)
	Social perception (%)	17 (11.56%)
	Emotion recognition (%)	53 (36.05%)
	Attributional biases (%)	4 (2.72%)
	Emotion processing (%)	14 (9.52%)
Symptom	Disorganisation factor (%)	76 (53.15%)
	Alogia (%)	26 (18.18%)
	Thought disorder (%)	23 (16.08%)
	Other (%)	18 (12.59%)
Scale	Positive and Negative Syndrome Scale/ Scale for the Assessment of Negative Symptoms/ Scale for the Assessment of Positive Symptoms/ Brief Psychiatric Rating Scale	106 (86.18%)
	Other (%)	17 (13.82%)

Table 1 and the methodological characteristics can be found in supplementary table d.

Overall effect size

The pooled effect size for all the studies combined was $r = -0.313$ ($k = 123$; 95% CI −0.346 to −0.279; $z = -17.226$; $P < 0.001$), which indicates a negative correlation of moderate strength. Not surprisingly, there was a significant amount of heterogeneity ($Q[122] = 306.702$; $P < 0.001$; $I^2 = 60.222$; $\tau^2 = 0.022$; s.e. = 0.006; variance = 0.000; $\tau = 0.147$), likely because of both the clinical and methodological diversity across studies (Fig. 2).

Covariates

To test the stability of effect sizes across time, we ran a meta-regression with year of publication as the predicting variable and individual effect size as the outcome variable. Overall, year of publication was found to be a significant predictor of the relationship between symptoms and socio-cognitive performance ($\beta = 0.010$; s.e. = 0.003; 95% CI 0.004–0.016; $z = 3.34$; $P = 0.0008$), suggesting that effect sizes increased over time.

To test if the association between symptoms and social cognition was specific to phase of illness (i.e. state-dependent), we compared the strength of the effect size across different patient groups. The analysis of studies that have tested in-patients yielded a correlation of −0.359 ($k = 31$; 95% CI −0.419 to −0.297; $z = -10.514$; $P < 0.001$), with a significant level of heterogeneity ($Q[30] = 44.344$; $P = 0.044$; $I^2 = 32.347$; $\tau^2 = 0.012$; s.e. = 0.010; variance = 0.000; $\tau = 0.109$). The analysis for studies that tested out-patients yielded a smaller but nevertheless significant correlation of −0.260 ($k = 55$; 95% CI −0.307 to −0.213; $z = -10.350$; $P < 0.001$), with a significant level of heterogeneity ($Q[54] = 120.950$; $P < 0.001$; $I^2 = 55.354$; $\tau^2 = 0.017$; s.e. = 0.007; variance = 0.000; $\tau = 0.132$). Finally, the analysis of studies that have tested mixed samples yielded a correlation of −0.353 ($k = 37$; 95% CI −0.414 to −0.289; $z = -10.121$; $P < 0.001$), with a significant level of heterogeneity ($Q[36] = 122.079$; $P < 0.001$; $I^2 = 70.511$; $\tau^2 = 0.028$; s.e. = 0.014; variance = 0.000; $\tau = 0.168$). Comparison between effect sizes revealed that differences were statistically significant ($Q[2] = 8.563$; $P = 0.014$), with the effect sizes for studies with both in-patients and mixed samples being significantly higher than effect sizes for studies with out-patients.

Finally, we ran a meta-regression to test the effect of patient's age on the size of the effect size between socio-cognitive performance and thought disorder. Overall, age was not found to be a significant predictor of the effect size ($\beta = 0.005$; s.e. = 0.003; 95% CI −0.001 to 0.011; $z = 1.80$; $P = 0.072$).

Subgroup analyses by symptom

To calculate the effect size for different symptom groups, we ran a subgroup analysis with a mixed-effects model. The analysis of studies that used disorganisation or cognitive factors derived from scales such as the PANSS and the BPRS yielded a correlation of −0.323 ($k = 76$; 95% CI −0.362 to −0.282; $z = -14.638$; $P < 0.001$), again with a significant level of heterogeneity ($Q[75] = 205.002$; $P < 0.001$; $I^2 = 63.415$; $\tau^2 = 0.021$; s.e. = 0.008; variance = 0.000; $\tau = 0.143$).

A subsample of studies considered alogia (or poverty of speech). For these studies, the calculation yielded a significant correlation of −0.300 ($k = 26$; 95% CI −0.395 to −0.198; $z = -5.584$; $P < 0.001$), again with a significant level of heterogeneity ($Q[25] = 72.995$; $P < 0.001$; $I^2 = 65.751$; $\tau^2 = 0.048$; s.e. = 0.023; variance = 0.001; $\tau = 0.219$).

Studies that calculated the effect sizes for thought disorder (including single items such as stereotyped thinking, difficulties with abstract thinking or incoherence of speech) yielded a correlation of −0.292 ($k = 33$; 95% CI −0.350 to −0.232; $z = -9.115$; $P <$

Study (year)	Outcome	Correlation	Lower limit	Upper limit	Z–Value	p–Value
Abdel-Hamid (2009)	Picture sequencing	−0.377	−0.593	−0.110	−2.719	0.007
Abram (2014)	FAP	0.010	−0.247	0.265	0.075	0.940
Allen (2007)	WAIS-R	−0.190	−0.331	−0.040	−2.478	0.013
Altamura (2015)	Mind in the eyes test	−0.401	−0.648	−0.078	−2.403	0.016
Ayesa-Arriola (2016)	Mind in the eyes test	−0.063	−0.216	0.093	−0.790	0.429
Barkhof (2015)	IFE	−0.383	−0.540	−0.200	−3.932	0.000
Bellack (1992)	SPT	−0.420	−0.664	−0.095	−2.493	0.013
Bell (2009)	BLERT	−0.186	−0.365	0.006	−1.901	0.057
Bell (2010)	SAT-MC	0.070	−0.175	0.307	0.557	0.578
Bell (2013)	BLERT	−0.040	−0.262	0.186	−0.344	0.731
Bo (2005)	MAS-A	−0.410	−0.579	−0.208	−3.798	0.000
Bozikas (2004)	APT	−0.700	−0.838	−0.478	−4.906	0.000
Bryson (1997)	BLERT	−0.029	−0.275	0.220	−0.228	0.820
Brüne (2011)	MSAT	−0.490	−0.651	−0.287	−4.355	0.000
Brüne (2012)	MSAT	−0.394	−0.581	−0.167	−3.299	0.001
Castagna (2013)	CATS	−0.233	−0.416	−0.032	−2.264	0.024
Chambon (2006)	Facial affect	0.050	−0.344	0.429	0.240	0.810
Cohen (2006)	FEIT	−0.350	−0.640	0.027	−1.827	0.068
Cohen (2009)	FEIT	−0.370	−0.560	−0.142	−3.107	0.002
Comparelli (2014)	FER	−0.454	−0.614	−0.259	−4.269	0.000
Comparelli (2012)	FAR	−0.299	−0.488	−0.083	−2.689	0.007
Corcoran (1997)	Hinting task	−0.249	−0.473	0.006	−1.916	0.055
Corcoran (2005)	Hinting task	−0.303	−0.509	−0.063	−2.460	0.014
Corrigan (1994a)	SCRT	−0.450	−0.713	−0.076	−2.325	0.020
Corrigan (1994b)	SCRT	−0.440	−0.721	−0.034	−2.112	0.035
Corrigan (1995)	SCRT	−0.150	−0.441	0.169	−0.919	0.358
Corrigan (1996)	SCRT	−0.340	−0.660	0.084	−1.584	0.113
Docherty (2012)	BLERT	−0.320	−0.526	−0.078	−2.569	0.010
Donohoe (2012)	Hinting task	−0.346	−0.422	−0.265	−7.940	0.000
Fett (2013)	DFAR	−0.210	−0.268	−0.151	−6.838	0.000
Fiszdon (2013)	BLERT	−0.130	−0.303	0.051	−1.408	0.159
Fraguas (2008)	ASQ	−0.212	−0.479	0.091	−1.378	0.168
Fretland (2015)	MASC	−0.270	−0.506	0.003	−1.938	0.053
Frith (1996)	6 stories (1st, 2nd order)	−0.656	−0.785	−0.474	−5.672	0.000
Fullam (2006)	AFFECT	−0.270	−0.502	−0.002	−1.977	0.048
Gaebel (1992)	Ekman	−0.510	−0.762	−0.124	−2.517	0.012
Gold (2012)	AER	−0.330	−0.501	−0.134	−3.234	0.001
Greig (2004)	Hinting task	−0.370	−0.511	−0.210	−4.343	0.000
Hamm (2012)	BLERT	−0.350	−0.575	−0.076	−2.479	0.013
Harrington (2005)	First order (picture sequencing)	0.000	−0.395	0.395	0.000	1.000
Henry (2007)	Film clips	−0.090	−0.442	0.286	−0.460	0.645
Henry (2008)	ERQ	0.140	−0.175	0.429	0.869	0.385
Hoschel (2001)	Emotional priming	−0.390	−0.691	0.026	−1.842	0.066
Ihnen (1998)	SCRT	−0.510	−0.749	−0.153	−2.699	0.007
Ito (1998)	Video vignettes	0.067	−0.228	0.350	0.440	0.660
Janssen (2006)	IPSAQ	0.060	−0.361	0.461	0.269	0.788
Johnston (2006)	Ekman	−0.510	−0.789	−0.057	−2.179	0.029
Kee (2003)	FEIT/VEIT/VAPT	−0.380	−0.541	−0.192	−3.816	0.000
Kee (2009)	MSCEIT	−0.180	−0.436	0.104	−1.248	0.212
Kern (2009)	TASIT	−0.070	−0.344	0.215	−0.476	0.634
Kim (2005)	VR	−0.100	−0.554	0.400	−0.375	0.707
Kim (2007)	VR	−0.322	−0.611	0.043	−1.735	0.083
Kohler (2003)	PERT	−0.720	−0.862	−0.474	−4.538	0.000
Kohler (2000)	ERT	−0.460	−0.688	−0.150	−2.813	0.005
Kosmidis (2007)	EDT	−0.547	−0.740	−0.271	−3.581	0.000
Kother (2012)	Mind in the eyes test	−0.266	−0.464	−0.044	−2.333	0.020
Langdon (2001)	Picture sequencing	−0.490	−0.716	−0.170	−2.887	0.004
Langdon (2002)	Non-literal speech	−0.490	−0.742	−0.118	−2.514	0.012
Larøi (2010)	KDEF	−0.536	−0.791	−0.123	−2.468	0.014
Lehmann (2014)	MET	−0.176	−0.422	0.094	−1.283	0.200
Leitman (2005)	VEIT/VEDT/FEIT/FEDT	−0.480	−0.682	−0.210	−3.308	0.001
Lysaker (2011)	Mind in the eyes test/Hinting task/BLERT	−0.440	−0.671	−0.130	−2.713	0.007
Lysaker (2013)	MAS-A	−0.380	−0.540	−0.193	−3.837	0.000
Loughland (2002)	Visual scanpaths	−0.300	−0.507	−0.061	−2.437	0.015
Mancuso (2011)	AIHQ	−0.180	−0.379	0.034	−1.648	0.099
Marjoram (2005)	Cartoon jokes	−0.186	−0.581	0.280	−0.776	0.438
Majorek (2009)	Picture sequencing	−0.527	−0.725	−0.249	−3.467	0.001
Mazza (2001)	Burglar story	−0.385	−0.637	−0.059	−2.296	0.022
McCleery (2016)	RAD/MSCEIT	−0.240	−0.510	0.073	−1.509	0.131
Minor (2015)	BLERT	−0.340	−0.536	−0.109	−2.833	0.005
Minor (2014)	BLERT	−0.360	−0.551	−0.133	−3.039	0.002
Nelson (2007)	FEIT	−0.160	−0.346	0.038	−1.589	0.112
Ng (2015)	Hinting task	−0.320	−0.441	−0.187	−4.571	0.000
Nienow (2006)	AIPSS	−0.220	−0.456	0.046	−1.628	0.103
Ntouros (2014)	PESIT	−0.282	−0.492	−0.041	−2.282	0.022
Pentaraki (2012)	6 stories (1st, 2nd order)/Mind in the eyes test	−0.550	−0.793	−0.155	−2.624	0.009
Peyroux (2014)	IbT	−0.410	−0.645	−0.104	−2.577	0.010
Pickup (2001)	3 stories (1st, 2nd order)	−0.721	−0.825	−0.569	−6.767	0.000
Pijnenborg (2006)	Faux pas	−0.310	−0.551	−0.022	−2.102	0.036
Piskulic (2011)	FEIT/FEDT	−0.170	−0.352	0.024	−1.717	0.086
Poole (2000)	Facial and vocal affect recognition	−0.400	−0.633	−0.101	−2.577	0.010
Popolo (2016)	Hinting task	−0.290	−0.561	0.038	−1.741	0.082
Rassovsky (2010)	BLERT	−0.104	−0.249	0.045	−1.365	0.172
Renard (2012)	4 stories (1st and 2nd order)	−0.546	−0.717	−0.313	−4.157	0.000
Rocca (2016)	FEIT/TASIT/MSCEIT	−0.115	−0.183	−0.047	−3.293	0.001
Romero-Ferreiro (2016)	FAR	−0.461	−0.757	−0.009	−1.994	0.046
Roncone (2002)	Animations	−0.284	−0.536	0.014	−1.870	0.061
Russell (2006)	Animations	−0.205	−0.348	−0.052	−2.622	0.009
Sachs (2004)	CPF/CPFD/EMODIFF/PEAT	−0.350	−0.596	−0.043	−2.223	0.026
Sarfati (1997a)	Cartoon-S	−0.819	−0.924	−0.599	−4.901	0.000
Sarfati (1997b)	FDT	−0.417	−0.679	−0.062	−2.277	0.023
Sarfati (1999a)	Comic strips	−0.490	−0.718	−0.166	−2.854	0.004
Sarfati (1999b)	Comic strips	−0.359	−0.634	−0.004	−1.982	0.048
Schneider (1995)	FDT	−0.310	−0.567	0.002	−1.950	0.051
Schenkel (2005)	FEIT	−0.450	−0.663	−0.169	−3.027	0.002
Sergi (2007)	Computerised task	−0.110	−0.300	0.088	−1.088	0.277
Shamay-Tsoory (2007)	Computerised task	−0.356	−0.676	0.077	−1.623	0.105
Shean (2005)	Faux pax	−0.300	−0.496	−0.075	−2.590	0.010
Shean (2009)	PA card sort (WAIS-R)	−0.360	−0.573	−0.102	−2.692	0.007
Shur (2008)	Faux pax	−0.732	−0.872	−0.482	−4.479	0.000
Silver (2001)	AR	−0.430	−0.665	−0.118	−2.642	0.008
Smith (2012)	IRI	−0.030	−0.318	0.263	−0.197	0.844
Smith (2014)	ERT	−0.150	−0.389	0.108	−1.141	0.254
Sparks (2010)	Cartoon jokes	−0.530	−0.748	−0.210	−3.066	0.002
Stratta (2007)	Cartoon jokes1	−0.330	−0.674	0.132	−1.414	0.158
Subotnik (2006)	MSCEIT	−0.260	−0.509	0.029	−1.765	0.078
Tan (2013)	PONS	−0.330	−0.532	−0.093	−2.689	0.007
Tang (2016)	FERT	−0.198	−0.385	0.005	−1.914	0.056
Toomey (2002)	MSCEIT	−0.250	−0.570	0.136	−1.277	0.202
Tseng (2013)	DANVA	−0.150	−0.338	0.050	−1.473	0.141
Tschacher (2006)	CAUSE	−0.510	−0.732	−0.190	−2.978	0.003
Tso (2012)	FET	−0.010	−0.396	0.379	−0.048	0.962
Tsotsi (2015)	FAR	−0.200	−0.488	0.128	−1.199	0.230
Turetsky (2007)	Hinting task	−0.860	−0.951	−0.635	−4.663	0.000
Uhlhas (2006)	Mind in the eyes test	−0.510	−0.694	−0.264	−3.775	0.000
Urbach (2013)	SAT	−0.013	−0.130	0.104	−0.217	0.828
Vaskinn (2009)	IPT-15	−0.220	−0.430	0.012	−1.858	0.063
Ventura (2015)	BLERT	−0.110	−0.326	0.117	−0.950	0.342
Vohs (2014)	Facial affect	−0.660	−0.834	−0.366	−3.802	0.000
Weniger (2004)	Facial affect	−0.440	−0.650	−0.168	−3.060	0.002
Wolfkuhler (2012)	Ekman	−0.407	−0.669	−0.055	−2.245	0.025
Woodward (2009)	Picture sequencing	−0.410	−0.626	−0.136	−2.856	0.004
Zalla (2006)	Picture sequencing	−0.545	−0.715	−0.315	−4.194	0.000
		−0.313	−0.346	−0.279	−17.226	0.000

Statistics for each study · Correlation and 95% CI

worse social cognition   better social cognition

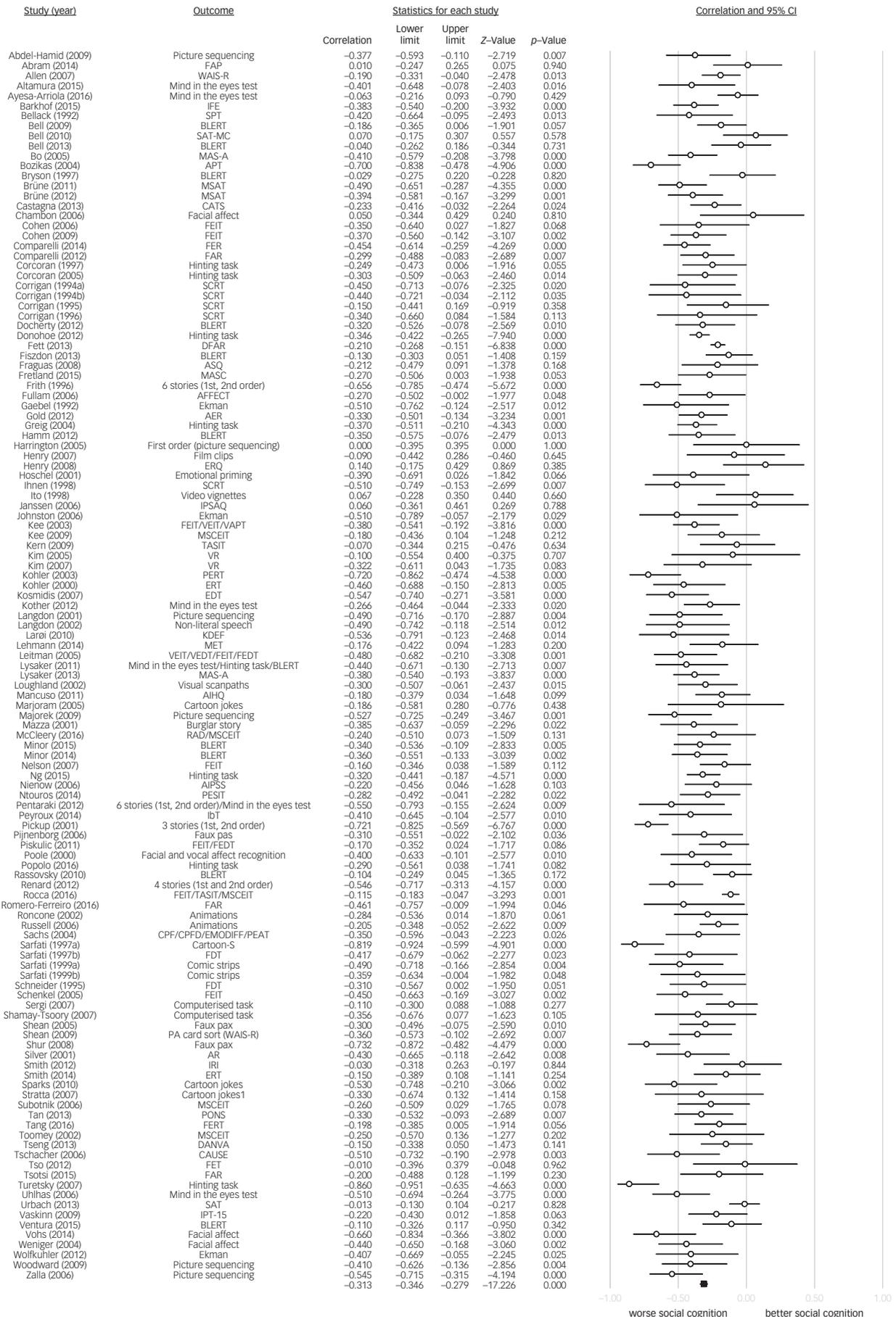−1.00   −0.50   0.00   0.50   1.00

Fig. 2 Forest plot. See supplementary material 2 for full references. Picture sequencing, Picture sequencing task; FAP, Facial Affect Perception Task; WAIS-R, Picture Arrangement subtest and/or Picture Completion subtest; Mind in the eyes test, Reading the mind in the eyes test; IFE, The

0.001), also with a significant level of statistical heterogeneity ($Q[32] = 47.530$; $P = 0.038$; $I^2 = 32.675$; $\tau^2 = 0.011$; s.e. = 0.009; variance = 0.000; $\tau = 0.105$).

To compare the effect sizes for the different symptom groups (i.e. disorganisation factor, alogia and thought disorder), we ran a mixed-effect analysis that revealed that differences between groups were not statistically significant ($Q[2] = 0.758$; $P = 0.684$).

Finally, we calculated the effect sizes just for studies that had used thought disorder-specific measures (e.g. TLC). These studies yielded a correlation of -0.351 ($k = 9$; 95% CI -0.479 to -0.208; $z = -4.623$; $P < 0.001$), this analysis revealed a non-significant level of statistical heterogeneity ($Q[8] = 21.924$; $P = 0.005$; $I^2 = 63.511$; $\tau^2 = 0.033$; s.e. = 0.028; variance = 0.001; $\tau = 0.183$).

ToM

The pooled effect size for the association between ToM and all symptoms combined was of moderate strength (-0.349; $k = 59$; 95% CI -0.396 to -0.301; $z = -13.269$; $P < 0.001$). This association revealed a considerable amount of statistical heterogeneity ($Q[58] = 174.594$; $P < 0.001$; $I^2 = 66.780$; $\tau^2 = 0.025$; s.e. = 0.010; variance = 0.000; $\tau = 0.158$). We also analysed the data across symptom groups (see supplementary materials 5). Effect sizes for disorganisation, thought disorder and alogia were all significant and of moderate strength, with no significant difference across symptom group. The analysis for studies that used thought disorder-specific measures revealed a larger effect size with a non-significant level of heterogeneity (see supplementary materials 5).

Social perception

The pooled effect size for the association between social perception and symptoms was weaker (-0.188; $k = 17$; 95% CI -0.256 to -0.117; $z = -5.158$; $P < 0.001$). However, the analysis carried a non-significant amount of heterogeneity ($Q[16] = 18.219$; $P = 0.311$; $I^2 = 12.178$; $\tau^2 = 0.003$; s.e. = 0.008; variance = 0.000; $\tau = 0.052$). The analyses across symptom groups revealed a significant association between social perception and thought disorder ($r = -0.259$), a marginally significant and weak association with alogia and a non-significant effect size for the association between social perception and disorganisation (see supplementary materials 5).

Emotion recognition

The relationship between emotion recognition and symptoms was of moderate strength (-0.334; $k = 53$; 95% CI -0.380 to -0.286; $z = -12.842$; $P < 0.001$). Again, this analysis revealed that there was a significant amount of statistical heterogeneity across studies ($Q[52] = 112.138$; $P < 0.001$; $I^2 = 53.629$; $\tau^2 = 0.018$; s.e. = 0.008; variance = 0.000; $\tau = 0.132$). The analyses by symptom group revealed significant and sizable effect sizes for the individual association between emotion recognition and disorganisation, thought disorder and alogia, especially with the latter ($r = -0.397$), although differences across the three effect sizes were not significant (see supplementary materials 5).

Attributional biases

Only a small number of studies looked at attributional biases and the pooled effect size was non-significant (-0.143; $k = 4$; 95% CI -0.347 to 0.073; $z = -1.298$; $P = 0.194$). Not surprisingly, this analysis revealed a very low amount of heterogeneity ($Q[3] = 5.890$; $P = 0.117$; $I^2 = 49.067$; $\tau^2 = 0.024$; s.e. = 0.040; variance = 0.002; $\tau = 0.154$). The analyses by symptom group revealed a significant association only between attributional biases and disorganisation, but there were no significant associations for thought disorder or alogia (see supplementary materials 5).

Emotion processing and regulation

The analysis of the strength of association between emotion processing and regulation and symptoms was significant but weak (-0.169; $k = 14$; 95% CI -0.243 to -0.092; $z = -4.287$; $P < 0.001$), with a non-significant level of heterogeneity ($Q[13] = 14.532$; $P = 0.337$; $I^2 = 10.540$; $\tau^2 = 0.002$; s.e. = 0.009; variance = 0.000; $\tau = 0.048$). The analyses by symptom group revealed significant associations between emotion processing difficulties and both thought disorder and disorganisation, but not alogia (see supplementary materials 5).

Publication bias

Visual inspection of the scatterplot for the analysis including all of the studies (see supplementary figure 4) revealed some degree of asymmetry suggestive of publication bias. To test the data-set, we used the following tests: (a) Begg and Mazumdar's rank order correlation, (b) Egger's regression intercept and (c) Duval and Tweedie's trim and fill procedure.

Begg and Mazumdar's rank correlation[63] yielded a significant Kendall's τ of -0.235 ($z = 3.854$; $P < 0.001$), suggestive of publication bias. Consistent with this, the Egger's test[64] also yielded a significant intercept of -1.498 (s.e. = 0.275; 95% CI -2.042

to −0.955; $t[121] = 5.458$; $P < 0.001$), supporting the existence of bias. Finally, Duval and Tweedie's trim and fill procedure[65] identified 35 potential missing studies (to the right of the mean). The recomputed point estimate, using a random-effects model, was −0.228 (95% CI −0.265 to −0.191), suggesting that even after adjustment, the estimate was significant and sizable.

Discussion

The overall pooled effect size suggests a significant and moderate association between poor performance on socio-cognitive tasks and severity of disorganised symptoms in patients diagnosed with schizophrenia spectrum disorders. More importantly, subanalyses by symptom groups showed that correlations were sizable and significant for thought disorder, alogia and disorganised symptoms, with no significant differences between the three symptom groups. However, it is important to point out that we found a considerable amount of statistical heterogeneity. In part, this is not unexpected, given the methodological diversity in the assessments of both social cognition (e.g. emotion recognition tasks that tap into different sensory modalities or ToM tasks with different levels of complexity) and symptoms (some studies measured disorganisation with an assessment of general psychopathology, e.g. PANSS, and others measured thought disorder with specific scales, e.g. TLC). Moreover, there are considerable discrepancies across the conceptual frameworks that underlie the different thought disorder measures.[66–68] Different measures rely on different ratings, scoring systems or methodologies to elicit speech samples (e.g. proverb interpretation, clinical interview, etc.),[31,69] and have different clinical, cognitive and neuroanatomical correlates.[59,70–73] Hence, caution is required when interpreting these findings.

One of the few analyses that did not reveal significant heterogeneity was the relationship between thought disorder and social cognition, especially in the case of the effect size calculated for studies that used thought disorder-specific measures. A possible explanation is that these studies used specific symptom measures instead of general psychopathology scales, which often only have limited items to measure cognitive disorganisation or thought disorder (e.g. PANSS or the SAPS) and may also include non-thought disorder-related items. Given that thought disorder is a heterogeneous construct,[29] it is not surprising that heterogeneity was greater when more general psychopathology measures were used. In other words, the more robust the thought disorder measure, the stronger and clearer the overall effect.

Another finding that might speak to the issue of statistical heterogeneity is the association between year of publication and effect size. Our meta-regression suggested a linear and significant relationship between these two variables, with effect size increasing with time. It is possible that the emergence of dominant theories about the role of social cognition in schizophrenia spectrum disorders has inadvertently led to a publication bias toward 'positive' findings in the field. This explanation is consistent with the results of our Begg and Mazumdar's rank correlation and the Egger's test, which were consistent with the presence of publication bias, and with the trim and fill' procedure, which identified 35 potentially missing studies. However, recalculation of the point estimate after adjustment for missing studies revealed an effect size that was sizable and significant, so it seems unlikely that missing data would be sufficient to nullify the main findings.

Interestingly, the analysis by age of participants turned out to be non-significant, suggesting that the relationship between social cognition and thought disorder is relatively stable across different age groups. In contrast, the subgroup analyses by patient status revealed that effect sizes were significantly greater in studies that have tested in-patient samples. Although there is evidence suggesting that both socio-cognitive difficulties[74] and thought disorder[20] are not specifically characteristic of patients diagnosed with schizophrenia spectrum disorders (they can be found in other diagnostic groups), it is likely that both thought disorder and poor social cognition become more salient during periods of psychotic crisis, when patients are highly distressed. For example, it is a well-established finding that thought disorder worsens when patients are asked to talk about personally and emotionally salient topics, a phenomenon known as the affective reactivity of speech effect.[75,76] It follows that if social cognition is important in thought disorder, then the relationship may well be more evident during an acute in-patient admission.

A second set of analyses concerned the effect sizes across the different socio-cognitive domains. As expected on the basis of socio-cognitive theories of thought disorder and disorganisation,[3,6] a strong association was found between poorer performance on ToM tasks and all symptom groups. We also found an equally sizable and significant association between poor emotion recognition and symptoms. This is not unexpected given that some ToM tasks (e.g. 'reading the mind in the eyes' test) are based on emotion recognition. However, it is interesting to note that most robust association was with alogia. In the case of social perception and emotion processing tasks, although effects were evident, they were much weaker, with former being particularly associated with positive forms of thought disorder as opposed to alogia. Regarding the weak associations with emotion processing, this is somehow unexpected given the well-reported finding that thought disorder worsens with negative affect.[75] Finally, the moderate association between attributional biases and disorganisation should be interpreted with caution given that there were only two studies included in the analysis. We are aware of no theoretical model that predicts these patterns of association, but it is worth noting that some of these domains do not necessarily have absolute and categorical boundaries and may overlap greatly.

There are good theoretical reasons for expecting a relationship between thought disorder and poor social cognition. As mentioned earlier, Frith[3] suggested that communication difficulties in patients (i.e. thought disorder) could be partly explained by their inability to infer the state of knowledge of the listener. This is consistent with studies that have found that, when patients with thought disorder are provided with the opportunity to explain their perspective and contextualise their communications, their verbalisations no longer sound bizarre or 'disordered'.[77] Hence, it seems reasonable to propose that difficulties at the level of social cognition (e.g. delayed activation of the frontotemporal-parietal areas that support mentalisation)[78] may render the patient unable to repair or readjust communication when unprompted, because of difficulties in timely detecting subtle and dynamic emotional and social cues from the interlocutor.

The establishment of conversational alignment,[79] or grounding[80] in communication or dialog is dependent on the early, automatic and timely processing and monitoring of partner-specific information (e.g. verbal and non-verbal paralinguistic cues and signals). This process helps the addressee disambiguate language and the speaker adjust communication to the needs of the addressee, enabling the incremental shared understanding between interlocutors (as dialog unfolds) and leading to more effective and efficient communication over time. According to Brennan et al[80] (pp. 316):

'(…) dialog can be viewed as a highly coordinated hypothesis-testing activity that individuals engage in together, where one partner's presentation (their hypothesis of what their partner will understand) plays a dual role by providing the other

person with evidence of how the previous utterance has been understood.'

A person who cannot disambiguate the question of the interviewer or cannot infer the state of knowledge of the listener is more likely to answer questions in an egocentric or tangential way, by intermingling, interweaving or blending in decontextualised concerns and worries into the context of the conversation,[81] thereby making communications sound idiosyncratic or even bizarre. This account is consistent with findings from studies that have reported that patients who display thought disorder have significant difficulties disambiguating and processing linguistic and conversational context.[82]

One important point to acknowledge at this stage is that the ability to infer other peoples' mental and emotional states may not be independent from the ability to reflect and understand one's own mental state (i.e. self-reflection or meta-awareness). For example, one study showed that gains in self-reflection predicted improvements in social cognition and, more specifically, the patient's ability to infer the mental or emotional states of others.[83] Some authors have hypothesised that patients with thought disorder have difficulties synthesising and making sense of their own cognitive experiences (resulting in 'cacophonous selves')[84] and, consistent with this idea, two studies have reported that patients with disorganised symptoms are significantly impaired in both self-reflexivity and social cognition.[85,86] There is also evidence that patients diagnosed with schizophrenia spectrum disorders have difficulties recalling autobiographical memories,[87] which may be necessary when making sense of others through analogical reasoning.[88,89] So it is plausible that difficulties with self-reflection or meta-awareness may underlie both poor mentalising and thought disorder. However, the relationship between poor self-reflection and other domains of social cognition also associated with thought disorder would be more difficult to explain.

Another possible interpretation is that symptoms of disorganisation may have a detrimental effect on both the patient's ability to reason about their own and other peoples' mental states. For example, Minor and colleagues reported that symptoms of disorganisation moderated the relationship between neurocognition and both social cognition and self-reflexivity in patients diagnosed with schizophrenia spectrum disorders.[90,91] However, such interpretation does not explain why patients with thought disorder fail to see their verbalisation as bizarre and idiosyncratic, but can successfully judge the verbalisation of other patients with thought disorder as anomalous.[45]

One of the limitations of the present meta-analysis is that the calculated strength of the associations between domains of social cognition and symptoms did not account for symptom comorbidity. This is important because difficulties with ToM have been reported to be significantly associated with negative symptoms and persecutory delusions.[5] In future studies, it will be important to establish the strength of the association between domains of social cognition and thought disorder after accounting for other psychotic experiences, especially negative symptoms, given its association with both poor mentalisation and dysfunctional mirror neuron activity.[92] Moreover, it might be suggested that the strength of the effect size could just reflect general 'severity of illness' or more general cognitive difficulties. However, if this was the case, then one would expect the correlations with social perception, emotion regulation and attributional biases to be equally sizable, which they were not. Another limitation of the review is the overrepresentation of men in the study samples. Few studies have attempted to control or account for gender differences, so it is possible that some of these difficulties are, to some extent, gender-specific.

Social cognition is only one piece in the puzzle of thought disorder, and other psychological mechanisms have been shown to be involved in this cluster of experiences. For example, we have reported previously that difficulties in internal source monitoring (ability to correctly discriminate whether self-generated cognitions were verbalised or just thought)[93] coupled with negative affect are important to explain exacerbation of thought disorder during emotional challenge,[75] and that poverty of speech seems to be specifically associated with impoverished inner speech (especially dialogical inner speech).[35] Finally, how these mechanisms relate to important social predictors of thought disorder remains a matter of speculation. Some authors have suggested that difficulties recognising and reasoning about mental states in patients diagnosed with schizophrenia spectrum disorders could be a consequence of early experiences such as poor early attachments, childhood trauma or isolation,[94] factors that have been found to be associated with thought disorder.[38,95–97] For example, a recent study showed that poor ToM mediated the relationship between insecure attachment and emerging psychotic symptoms.[98] In future studies, it will be important to examine the relationships between social predictors and socio-cognitive processes in thought disorder with more complex psychosocial models.

It may also be fruitful to test if existent socio-cognitive training packages have an effect on thought disorder (e.g. social cognition enhancement training).[99] A published meta-analysis of socio-cognitive training in schizophrenia spectrum disorders reported significant and sizable effect sizes on both ToM and facial affect recognition and identification.[100] The effect size for psychotic symptoms for this kind of intervention have been modest, but given the findings of the current meta-analysis, it would be pertinent to trial socio-cognitive packages that focus on both emotion recognition and perspective taking in communication on patients with persistent thought disorder. This is important given the known association between thought disorder and poorer quality of life, relapse and poorer occupational and social functioning.

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Supplementary material

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