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Review article

Social cognitive dysfunction as a clinical marker: A systematic review of meta-analyses across 30 clinical conditions

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ABSTRACT

Social cognition includes a range of cognitive processes that help individuals to understand how others think and feel. There is emerging evidence that social cognitive deficits may represent a transdiagnostic issue, potentially serving as a marker of neurological abnormality. We performed an electronic database search in order to identify published, peer-reviewed meta-analyses that compared facial emotion recognition or theory of mind task performance between individuals meeting clinical criteria for a psychiatric, neurological or developmental condition against healthy controls. We identified 31 meta-analyses eligible for inclusion that examined performance across relevant tasks among 30 different clinical populations. The results suggest that social cognitive deficits appear to be a core cognitive phenotype of many clinical conditions. Across the clinical groups, deficits in social cognitive domains were broadly similar in magnitude to those previously reported for more established aspects of cognition, such as memory and executive function. There is a need to clarify the 'real world' impact of these deficits, and to develop effective transdiagnostic interventions for those individuals that are adversely affected.

1. Introduction

Social cognition refers to the 'mental operations that underlie social interactions' and includes a range of cognitive processes that help individuals to understand how others think and feel (Frith, 2008; Green et al., 2008). The most heavily researched aspects of social cognition are emotion recognition and theory of mind (ToM); two partially overlapping but distinct cognitive domains (Mitchell and Phillips, 2015). Emotion recognition refers to an individual's ability to identify and discriminate between the basic emotional states of others, typically based on their facial or vocal expressions. Theory of mind refers to the ability to infer more complex mental states, including the intentions, dispositions, and beliefs of others, and is thought to comprise distinguishable but overlapping cognitive and affective components (Green et al., 2015; Poletti et al., 2012). Collectively these drive interpersonal skills such as empathy, and are thought to be important for fluid communication and social interaction.

The neural basis of social cognitive processing is complex, involving a range of cortical and subcortical regions and connective pathways (Van Overwalle, 2009). This 'social brain' network varies depending on task demands, but is broadly thought to include limbic regions (such as the amygdala), the prefrontal cortex and temporoparietal junction, as well as the anterior cingulate and insular cortex (Forbes and Grafman, 2010; Lavin et al., 2013; Lindquist et al., 2012; Molenberghs et al., 2016). Many of these regions are adversely affected in people with neurological or psychiatric conditions (Batista et al., 2017a, 2017b; Cusi et al., 2012; Patriquin et al., 2016; Seeley et al., 2009), suggesting social cognitive dysfunction may be common across these populations.

Pioneering early work in this field focused on the central role of ToM impairments as a hallmark feature of autism spectrum disorders (Baron-Cohen et al., 1985; Happé and Frith, 1996). Subsequently, deficits in both emotion recognition and ToM have been identified as core cognitive deficits in schizophrenia (Savla et al., 2013; Kohler et al., 2010), and have been reported to be among the strongest predictors of impaired social functioning in this population (Fett et al., 2011; Galderisi et al., 2014; Green, 2016). There is also preliminary evidence that deficits in social cognitive processes may contribute to deterioration in psychosocial functioning in other clinical groups (Aydemir et al., 2013; Phillips et al., 2011; Trevisan and Birmingham, 2016). More recently, some aspects of emotion recognition have also been proposed to play a causal role in the onset and maintenance of a range of mental health conditions (Penton-Voak et al., 2017). As a result, investigation

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of social cognitive dysfunction has become an emerging area of interest across a range of developmental, psychiatric and neurological disorders (Bora and Pantelis, 2016; Cotter et al., 2016; Plana et al., 2014).

Despite recent calls emphasising its importance (Henry et al., 2016), social cognitive assessment has been largely overlooked clinically to date. This could have the potential to serve as a marker of neural deterioration and disease progression, particularly for neurodegenerative conditions, as well as treatment response. There has also been little consideration of these deficits as a transdiagnostic issue. Social cognitive deficits potentially represent an under recognised domain of impairment across disorders, with both functional and clinical relevance. In this review, we sought to collate existing meta-analytic data on social cognitive performance among individuals with a range of clinical conditions.

2. Method

This review was conducted in line with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009).

2.1. Study inclusion criteria

Eligible articles were meta-analyses published in peer-reviewed journals that examined the difference in performance on facial emotion recognition or ToM tasks between individuals with a clinically diagnosed psychiatric, neurological or developmental condition and healthy controls. Facial emotion recognition effect size estimates were required to have been derived from tasks in which participants had to identify, label or match images of faces consisting of all or any combination of the six basic emotions (happiness, sadness, anger, fear, surprise or disgust) (Ekman and Friesen, 1976). Where possible, a 'total' score was used, comprising performance across multiple emotions. ToM or 'mentalizing' tasks required the identification or interpretation of more complex mental states and/or beliefs (such as lies, jokes, sarcasm or faux pas).

Standardised mean difference effect size estimates (Cohen's d or Hedges' g) must have been derived from two or more independent studies in order for the meta-analysis to be included. Where data was reported for different states of a given condition (e.g. remitted/acute symptoms), we included the overall 'pooled' estimates from across those states when available. Where there were multiple publications for a given medical condition that met our inclusion criteria, we included the most recently published paper. Meta-analyses of interventions were ineligible. No language restrictions were placed on studies for inclusion.

2.2. Search strategy

On 1st May 2017, we conducted an electronic database search of Ovid MEDLINE, PsycINFO, Embase and the Cochrane Database of Systematic Reviews (from inception) using the following keyword search terms: "social cogniti*" or "theory of mind" or "mentalizing" or "emotion" and "meta-analysis". In addition; a search of Google Scholar was conducted and the reference lists of retrieved articles were also reviewed to identify any additional relevant publications. An additional eligible meta-analysis that was published following the formal search date was also later identified and included.

2.3. Study selection and data extraction

Three of the authors (J.C., R.B. and M.H.) independently screened articles for eligibility. A standardised data extraction spreadsheet was used for all eligible studies to record: (1) study characteristics (authors, year of publication, clinical condition(s) examined); (2) social cognitive domains assessed (facial emotion recognition and/or ToM); (3) sample characteristics (the number of studies and the sample sizes for the clinical and control groups included for each of the summary estimates); (4) social cognitive performance in the clinical relative to control group (effect size, effect size metric and associated 95% confidence intervals).

Effect sizes generated using either Cohen's d or Hedges' g were included. These represent the difference between the clinical and control group means, divided by the pooled standard deviation and weighted for sample size. Hedges' g includes an additional correction for small sample size bias, but is broadly comparable to Cohen's d (Lakens, 2013). Effect sizes generated using either approach are typically interpreted as representing small (≤ 0.2), medium (0.5), or large (≥ 0.8) group differences based on these methods (Cohen, 1988). Scores were re-coded where necessary so that negative effect size estimates always reflected poorer performance in the clinical group relative to controls. Where data was reported from both fixed and random effects models, we included the results from the random effects analyses. These models provide more conservative estimates by accounting for observed heterogeneity. Where reported, we also recorded the degree of heterogeneity within effect size estimates (I^2 statistic), as well as any tests that were performed to detect evidence of potential publication bias (Egger's test, Begg's test and/or the Fail-safe *N*). Effect size estimates were classified as having a 'low risk' or 'possible risk' of publication bias based on the results of these analyses.

3. Results

The study selection process is summarised in Fig. 1. We identified 31 meta-analyses eligible for inclusion that examined performance on facial emotion recognition (24 papers) and/or ToM tasks (24 papers) among 30 different clinical populations relative to controls (Table 1). Across clinical conditions, the facial emotion recognition and ToM effect size estimates each collectively included data acquired from around 20,000 clinical and control participants. Though the effect sizes are not directly comparable across clinical conditions (due to methodological differences between studies and in meta-analytic procedures), they demonstrate consistent and statistically significant deficits in facial emotion recognition and ToM across almost all of the clinical groups included in this review (Figs. 2 and 3). These also provide an indication of the magnitude of these difficulties. Within specific conditions, the severity of deficits in facial emotion recognition and ToM task performance were broadly comparable with one another, suggesting patients with particular disorders have similar levels of difficulty across each of these social cognitive domains. Among individuals with neurological or developmental disorders, deficits across both task types were in the medium-to-large range (-0.41 to -1.81). Individuals with psychiatric disorders exhibited wider variation in the severity of these deficits, potentially due to greater variability in disease state and severity. Perhaps unsurprisingly, the greatest impairments were observed in patients with neurodegenerative disorders, though large deficits were also observed in people with psychotic disorders.

Heterogeneity was evident in the effect size estimates and ranged from low to high. There was a possible risk of publication bias associated with 7 of the 34 effect size estimates that included tests for publication bias, suggesting overall that these results provide a robust indication that social cognitive deficits are common across many of the included clinical populations.

4. Discussion

4.1. Overview of findings

In summary, we identified significant deficits among individuals with a wide range of clinical conditions in their ability to identify emotions from facial expressions and to successfully complete ToM tasks, compared to healthy controls. Though these results do not provide directly comparable estimates between clinical conditions, they

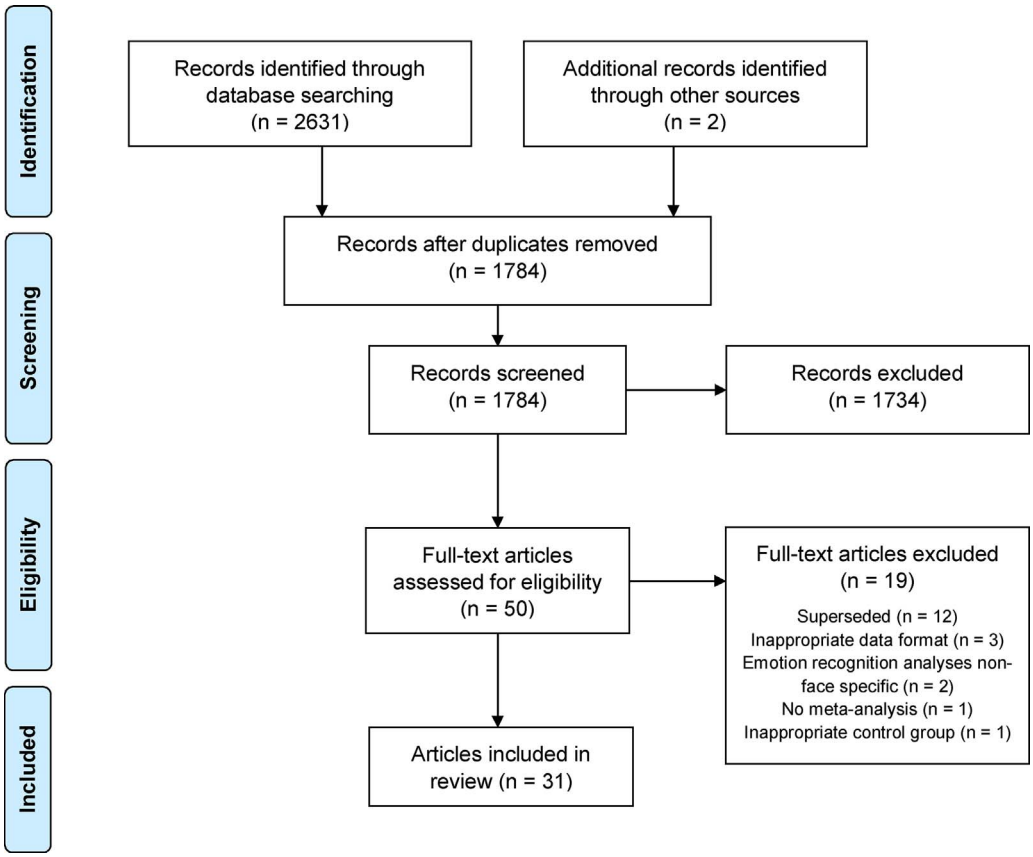

Fig. 1. PRISMA flow diagram.

provide a robust indication that social cognitive deficits appear to be a core cognitive phenotype of many developmental, neurological and psychiatric disorders.

4.2. Social cognition as a clinical marker

Across many conditions, deficits in social cognitive domains were broadly similar in magnitude to those previously reported on more established aspects of cognition, such as memory, processing speed and executive function (e.g. Beeldman et al., 2016; Demetriou et al., 2017; Fioravanti et al., 2012; Prakash et al., 2008). The distinction between neurocognitive and social cognitive processing remains an area of ongoing investigation. Though social cognitive deficits are likely to be exacerbated as a secondary deficit to more general cognitive impairments (for example, on tasks requiring rapid processing or a memory component), there is evidence that these are dissociable cognitive constructs. For example, neuroimaging studies suggest that these domains rely on different (though partially overlapping) neural systems, while analysis of performance on social and neurocognitive tasks demonstrate that these also form different factors (Bertoux et al., 2016; Mehta et al., 2013; Van Overwalle, 2009). Studies have also indicated that social cognitive dysfunction is exhibited by patients with otherwise intact cognitive and perceptual performance (Cotter et al., 2016; Fanning et al., 2012; Lagravinese et al., 2017). Given that these neurocognitive deficits are a target for therapeutic intervention in many disorders, the magnitude of social cognitive deficits should also be considered clinically meaningful.

Social cognitive impairment has been posited as a potential marker for autism and schizophrenia (Derntl and Habel, 2011). However, the findings of this review suggest it may serve as a general biomarker indicative of neurological abnormality across a range of clinical conditions and warrants further attention. The results from this review suggest that social cognitive measures should be integrated into large-scale, longitudinal projects (alongside existing neurocognitive measures) using repeated assessments in order to examine their potential to serve as longitudinal predictors of clinical and functional outcomes.

There is emerging evidence that social cognitive performance may serve as a useful screening tool among individuals with neurodegenerative conditions. The presence of social cognitive deficits in pre-manifest Huntington's disease have been reported to indicate a significantly higher risk of developing motor symptoms in the following 5 years (Bora et al., 2016b). Social cognitive screening also has the potential to help improve early diagnosis between different forms of cortical dementias (Bertoux and Hornberger, 2015). Individuals with behavioural-variant frontotemporal dementia exhibited significantly poorer performance across both facial emotion recognition and ToM tasks compared to those with Alzheimer's disease, despite worse Mini–Mental State Examination performance in the Alzheimer's group (Bora et al., 2016a, 2015a). Effect size differences across these measures were much larger than those previously reported across more traditional neurocognitive tasks (Hutchinson and Mathias, 2007). This suggests that social cognitive measures may be useful for identifying which individuals are in the early stages of, or at risk of developing behavioural-variant frontotemporal dementia compared to Alzheimer's disease, particularly among patients with mild cognitive impairment.

As well as being a potential marker for disease onset, social cognitive assessment also appears to offer the potential to examine disease progression. There was evidence that social cognitive deficits were evident even among individuals in the early stages of a number of conditions, with only mild levels of disease severity (Bora, 2017; Bora et al., 2015b; Cotter et al., 2016; van Donkersgoed et al., 2015), but also that they were more severe in those individuals with neurological conditions with a longer disease duration (Bora et al., 2016b, 2015a), and those with psychiatric disorders experiencing an exacerbation in symptom severity (Bora et al., 2016c; Bora and Berk, 2016; Bora and Köse, 2016).

Table 1
Overview of included meta-analyses.

Condition	Reference	Domains assessed	Studies included	Sample	*n*	Effect size (95% CI)	Effect size metric	Heterogeneity (I^2)	Publication bias
Psychiatric disorders									
At-risk mental state	van Donkersgoed et al. (2015)	FER	10	Clinical Controls	444 375	−0.48 (−0.69, −0.27)	Cohen's d	N/R	N/R
		ToM	7	Clinical Controls	348 267	−0.44 (−0.68, −0.19)	Cohen's d	N/R	N/R
First-episode psychosis	Barkl et al. (2014)	FER	11	Clinical Controls	378 369	−0.88 (−1.42, −0.32)	Cohen's d	N/R	N/R
	Bora and Pantelis (2013)	ToM	8	Clinical Controls	285 228	−1.00 (−1.18, −0.81)	Cohen's d	0%	Low risk
Schizophrenia	Kohler et al. (2010)	FER	53	Clinical Controls	3822	−0.91 (−0.97, −0.84)	Cohen's d	N/R	Possible risk
	Savla et al. (2013)	ToM	50	Clinical Controls	1760 1536	−0.96 (−1.09, −0.83)	Hedges' g	66.50%	Possible risk
Bipolar disorder	Kohler et al. (2011)	FER	31	Clinical Controls	N/R N/R	−0.46 (−0.63, −0.29)	Cohen's d	N/R	N/R
	Bora et al. (2016c)	ToM	34	Clinical Controls	1214 1097	−0.63 (−0.74, −0.52)	Cohen's d	36.50%	Possible risk
Major depressive disorder	Dalili et al. (2015)	FER	22	Clinical Controls	977 843	−0.16 (−0.25, −0.07)	Hedges' g	0%	Possible risk
	Bora and Berk (2016)	ToM	18	Clinical Controls	613 529	−0.58 (−0.84, −0.33)	Cohen's d	74.80%	Low risk
Borderline personality disorder	Daros et al. (2013)	FER	10	Clinical Controls	266 255	−0.45 (−0.80, −0.09)	Cohen's d	N/R	Low risk
	Richman and Unoka (2015)	ToM	5	Clinical Controls	224 186	−0.06 (−0.26, 0.13)	Cohen's d	N/R	N/R
Generalised anxiety disorder	Plana et al. (2014)	FER	2	Clinical Controls	58 48	−0.12 (N/R)	Cohen's d	N/R	N/R
Obsessive-compulsive disorder	Plana et al. (2014)	FER	12	Clinical Controls	313 357	−0.16 (N/R)	Cohen's d	N/R	N/R
		ToM	2	Clinical Controls	55 55	−0.30 (N/R)	Cohen's d	N/R	N/R
Panic disorder	Plana et al. (2014)	FER	2	Clinical Controls	73 79	−0.25 (N/R)	Cohen's d	N/R	N/R
Social phobia	Plana et al. (2014)	FER	10	Clinical Controls	217 239	−0.20 (N/R)	Cohen's d	N/R	N/R
Post-traumatic stress disorder	Plana et al. (2014)	FER	4	Clinical Controls	68 63	−1.60 (N/R)	Cohen's d	N/R	N/R
Anorexia Nervosa	Caglar-Nazali et al. (2014)	FER	5	Clinical Controls	185 188	−0.32 (−0.55, −0.09)	Cohen's d	N/R	Low risk
	Bora and Köse (2016)	ToM	14	Clinical Controls	450 441	−0.59 (−0.81, −0.37)	Cohen's d	N/R	N/R
Bulimia Nervosa	Caglar-Nazali et al. (2014)	FER	2	Clinical Controls	51 98	0.01 (−0.33, 0.36)	Cohen's d	N/R	Low risk
	Bora and Köse (2016)	ToM	8	Clinical Controls	227 267	−0.34 (−0.58, −0.09)	Cohen's d	45.31%	N/R
Alcohol use disorder	Bora and Zorlu (2017)	FER	12	Clinical Controls	410 352	−0.65 (−0.89, −0.42)	Cohen's d	60.18%	Low risk
		ToM	12	Clinical Controls	317 298	−0.58 (−0.81, −0.36)	Cohen's d	44.55%	Low risk
Substance use disorder (non-alcohol)	Castellano et al. (2015)	FER	10	Clinical Controls	438 422	−0.65 (−0.93, −0.37)	Cohen's d	72.50%	Low risk
Neurological disorders									
Alzheimer's disease	Bora et al. (2015a)	ToM	20	Clinical Controls	402 421	−1.15 (−1.52, −0.79)	Cohen's d	N/R	Low risk
Amyotrophic lateral sclerosis	Bora (2017)	FER	5	Clinical Controls	92 152	−0.69 (−0.97, −0.42)	Cohen's d	0%	Low risk
		ToM	11	Clinical Controls	311 339	−0.65 (−0.84, −0.47)	Cohen's d	21%	Possible risk
Behavioural variant frontotemporal dementia	Bora et al. (2016a)	FER	13	Clinical Controls	237 339	−1.81 (−2.28, −1.35)	Cohen's d	80.7%	Low risk
	Bora et al. (2015a)	ToM	20	Clinical Controls	334 391	−1.79 (−2.18, −1.40)	Cohen's d	N/R	Low risk
Frontal lobe epilepsy	Stewart et al. (2016)	ToM	3	Clinical Controls	55 125	−1.03 (−1.33, −0.72)	Hedges' g	N/R	Low risk
Idiopathic generalised epilepsy	Stewart et al. (2016)	ToM	2	Clinical Controls	62 104	−0.59 (−0.87, −0.31)	Hedges' g	N/R	Low risk
Temporal lobe epilepsy	Bora and Meletti (2016)	FER	16	Clinical Controls	580 425	−0.87 (−1.05, −0.69)	Cohen's d	46.55%	Low risk
		ToM	11	Clinical Controls	569 353	−0.86 (−1.07, −0.64)	Cohen's d	49.95%	Low risk

(*continued on next page*)

Table 1 (*continued*)

Condition	Reference	Domains assessed	Studies included	Sample	*n*	Effect size (95% CI)	Effect size metric	Heterogeneity (I^2)	Publication bias
Huntington's disease	Bora et al. (2016b)	FER[a]	18	Clinical Controls	413 446	−1.33 (−1.52, −1.14)	Cohen's d	N/R	Possible risk
		ToM	12	Clinical Controls	238 199	−1.72 (−2.09, −1.35)	Cohen's d	70%	Low risk
Mild cognitive impairment	Bora and Yener (2017)	FER	13	Clinical Controls	370 434	−0.58 (−0.73, −0.43)	Cohen's d	0%	Low risk
		ToM	6	Clinical Controls	143 259	−0.63 (−0.91, −0.35)	Cohen's d	34%	Low risk
Multiple sclerosis	Cotter et al. (2016)	FER	13	Clinical Controls	473 423	−0.64 (−0.81, −0.47)	Hedges' g	36%	Low risk
		ToM	12	Clinical Controls	429 345	−0.71 (−0.88, −0.55)	Hedges' g	23%	Low risk
Parkinson's disease	Gray and Tickle-Degnen (2010)	FER	28	Clinical Controls	1110	−0.41 (−0.64, −0.19)	Hedges' g	N/R	Low risk
	Bora et al. (2015b)	ToM	18	Clinical Controls	487 459	−0.83 (−1.09, −0.57)	Cohen's d	N/R	Low risk
Traumatic brain injury	Babbage et al. (2011)	FER	13	Clinical Controls	296 296	−1.11 (−1.25, −0.97)	Hedges' g	N/R	Low risk
Developmental disorders									
Attention deficit hyperactivity disorder	Bora and Pantelis (2016)	FER	25	Clinical Controls	1021 764	−0.44 (−0.59, −0.30)	Cohen's d	N/R	Low risk
		ToM	24	Clinical Controls	1010 1024	−0.45 (−0.62, −0.29)	Cohen's d	N/R	Low risk
Autism spectrum disorder	Chung et al. (2014)	ToM	11	Clinical Controls	264 243	−0.81 (−1.14, −0.48)	Hedges' g	N/R	Possible risk
Intellectual disability	Yirmiya et al. (1998)	ToM	17	Clinical Controls	N/R N/R	−0.45 (−0.61, −0.29)	Cohen's d	N/R	N/R
Specific language impairment (children)	Nilsson and de López (2016)	ToM	17	Clinical Controls	329 416	−0.98 (−1.23, −0.74)	Cohen's d	55.23%	Low risk

Abbreviations: CI: Confidence Interval; FER: Facial emotion recognition; N/R: Not reported; ToM: Theory of Mind.
[a] Negative emotions subscale score; comprising total score for anger, disgust, sadness and fear facial affect recognition tasks only.

4.3. Strengths and limitations

This is the first review to our knowledge to examine social cognition across such a broad range of clinical disorders. As a result, we concentrated on facial emotion recognition and ToM, which have been the overwhelming focus of research in this field to date. Currently, ToM tasks are typically grouped together, though these include a relatively heterogeneous array of measures, requiring participants to make inferences regarding sarcasm, hints, faux pas and/or complex affective states (for example see Baron-Cohen et al., 2001; Corcoran et al., 1995; Gregory et al., 2002). In future, differentiation of tasks assessing cognitive and affective components of ToM may provide greater sensitivity to domain-specific impairments. Similarly, though 'global' scores indicated a deficit among clinical groups in the identification of facial emotions, there was variation in the emotion-specific patterns of deficits among the different clinical conditions. A greater understanding of

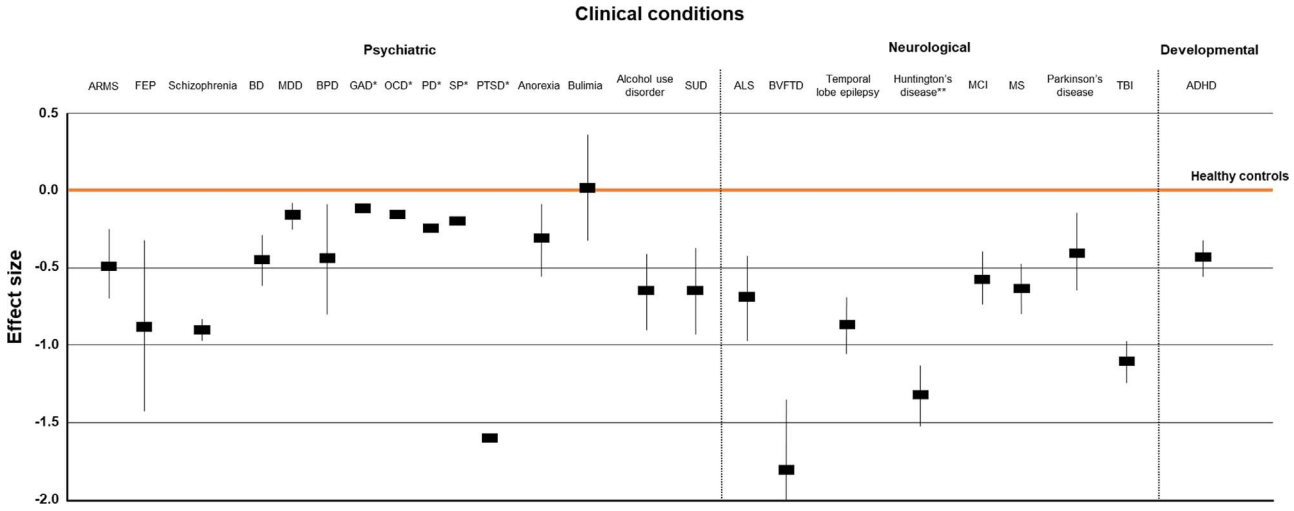

Fig. 2. Facial emotion recognition effect size estimates and corresponding 95% confidence intervals across clinical conditions relative to healthy controls.
*95% CI data not available.
**Negative emotions subscale score, comprising total score for anger, disgust, sadness and fear facial affect recognition tasks only.
Abbreviations: ADHD: Attention deficit hyperactivity disorder; ALS: Amyotrophic lateral sclerosis; ARMS: At-risk mental state; BD: Bipolar disorder; BPD: Borderline personality disorder; BVFTD: Behavioural variant frontotemporal dementia; FEP: First-episode psychosis; GAD: Generalised anxiety disorder; MCI: Mild cognitive impairment; MDD: Major depressive disorder; MS: Multiple sclerosis; OCD: Obsessive-compulsive disorder; PD: Panic disorder; PTSD: Post-traumatic stress disorder; SP: Social phobia; SUD: Substance use disorder (non-alcohol); TBI: Traumatic brain injury.

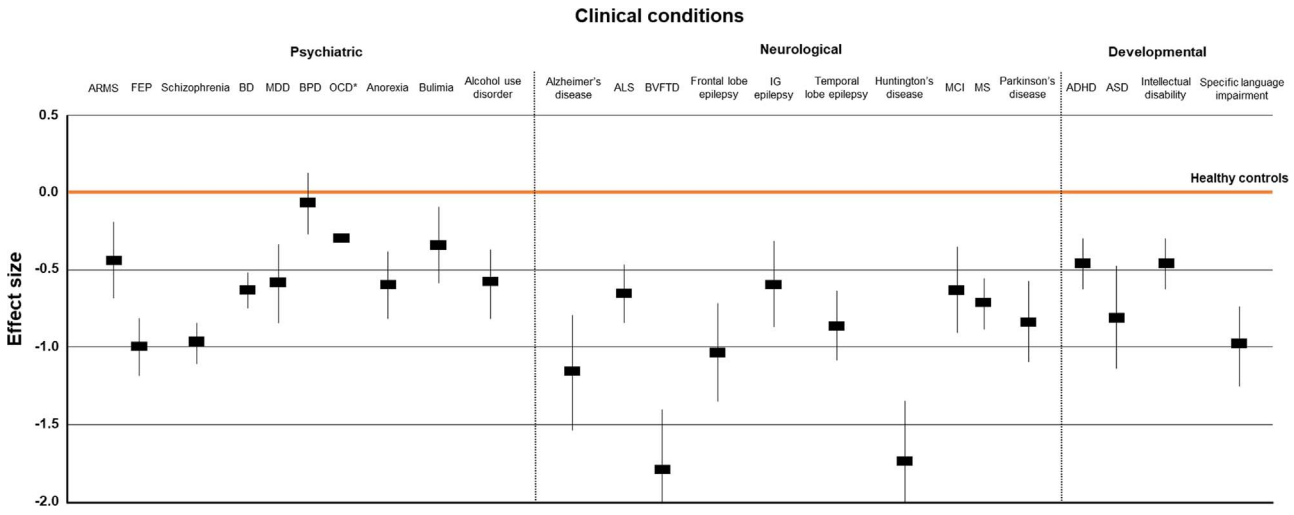

Fig. 3. Theory of mind effect size estimates and corresponding 95% confidence intervals across clinical conditions relative to healthy controls.
*95% CI data not available
Abbreviations: ADHD: Attention deficit hyperactivity disorder; ALS: Amyotrophic lateral sclerosis; ARMS: At-risk mental state; ASD: Autism spectrum disorder; BD: Bipolar disorder; BPD: Borderline personality disorder; BVFTD: Behavioural variant frontotemporal dementia; FEP: First-episode psychosis; IG: Idiopathic generalised epilepsy; MCI: Mild cognitive impairment; MDD: Major depressive disorder; MS: Multiple sclerosis; OCD: Obsessive-compulsive disorder.

these condition-specific emotion recognition profiles could provide further insight into underlying disease pathology. For example, the recognition of fear and sadness has been reported to be strongly associated with the integrity of medial temporal lobe structures, whereas disgust is more closely associated with the insular cortex, though there is evidence of considerable overlap across emotions (Adolphs, 2002). There are also other aspects of social cognition which have received comparatively little attention. Vocal tasks, for example, are potentially more sensitive than faces for identifying emotion recognition deficits in Parkinson's disease (Gray and Tickle-Degnen, 2010), and can also be used to assess emotion recognition among individuals with visual impairments. Bodily affect recognition is also an emerging area of interest across disorders, providing an additional approach for social cognitive assessment (Cecchetto et al., 2014; Vaskinn et al., 2016).

Though many of the original research studies matched the clinical and control groups for various demographic characteristics (such as age, sex and level of education), this was inconsistently reported across the included meta-analyses and may have influenced their results. Variation in the clinical and demographic samples of the included studies, as well as the range of social cognitive assessments that were used, are likely to have contributed to the statistical heterogeneity that was observed among many of the effect size estimates. Comorbidities are also common across many of these disorders and could be considered as a potential confounder. For example, depression is common among people with psychiatric and neurological diagnoses (Fusar-Poli et al., 2014; Siegert and Abernethy, 2005; Ulfvebrand et al., 2015), and may have influenced emotional processing. However, a number of studies included in the meta-analyses explicitly excluded individuals with major depressive disorder or else reported no association between the severity of depressive symptoms with social cognitive task performance, suggesting social cognitive deficits were not secondary to depressive comorbidity (e.g. Cotter et al., 2016; Daros et al., 2013; Gray and Tickle-Degnen, 2010). The impact of other common comorbid neuropsychiatric symptoms such as apathy and anxiety on social cognitive performance is less well understood, though the results presented in this review suggest the latter is unlikely to be a major causal factor for social cognitive decline.

The majority of the meta-analyses included in this review included a large number of studies and participants, providing robust effect size estimates. However, given this is an emerging field, several of the epilepsy, PTSD, anxiety and eating disorder estimates were based on a limited pool of original research studies and should be interpreted with caution. Similarly, tests for evidence of publication bias associated with each of the effect size estimates indicated a low risk of publication bias across the majority of the meta-analyses included in this review. However, some of the meta-analyses included a relatively small number of studies (< 10 for each estimate), and are therefore likely to be underpowered to detect evidence of potential publication bias (Higgins and Green, 2011). Many of the meta-analyses also performed meta-regression analyses to examine potential clinical, cognitive and demographic moderators of these effect sizes. However, these meta-regression analyses would have been underpowered to detect anything but very large study-level effects since, in many cases, they would include only aggregate data from a relatively small number of studies. We therefore did not include the results of these moderator analyses in the current review.

4.4. Future opportunities in social cognitive research

Impairments in social and occupational functioning and reduced quality of life are common across a wide range of psychiatric, developmental and neurological disorders. There is emerging evidence that social cognitive dysfunction may contribute to such difficulties (Aydemir et al., 2013; Bishop-Fitzpatrick et al., 2017; Cotter et al., 2017; Fett et al., 2011; Phillips et al., 2011; Trevisan and Birmingham, 2016). In addition to ongoing psychiatric or neurological symptoms, misperception of others emotional or mental states coupled with difficulties in empathising during social situations may play a causal role in the breakdown of interpersonal relationships (Couture et al., 2006). Though these links would benefit from further (particularly longitudinal) investigation, social cognitive dysfunction does represent a potentially modifiable risk factor for these difficulties (Penton-Voak et al., 2017). Assessment and treatment of social cognitive dysfunction may also offer wider treatment benefits. For example, recent evidence suggests that cognitive training aimed at treating facial emotion recognition biases exhibited by individuals with low mood can also help to alleviate their depressive symptoms (Iacoviello et al., 2014; Penton-Voak et al., 2012).

Despite the evidence that social cognitive dysfunction is common among individuals with a range of medical conditions, targeted treatments for these deficits remain in their relative infancy. The most widely researched pharmacological agent for the treatment of social cognitive impairment has been oxytocin, a neuropeptide hormone thought to play a key role in social behaviours (Meyer-Lindenberg et al.,

2011). Despite some promising trials conducted in patients with psychotic, affective and autism spectrum disorders (Mercedes Perez-Rodriguez et al., 2015; Preti et al., 2014), its benefits appear inconsistent across studies and methodologically problematic (Bradley and Woolley, 2017; Leppanen et al., 2017). At present, more effective approaches have involved the use of social cognitive remediation, though these also appear to offer only modest benefits (García-Casal et al., 2017; Kurtz et al., 2016).

An important limitation in this area is the poor or unknown psychometric properties of many social cognitive tests, though efforts are currently underway to establish how these measures compare (Pinkham et al., 2016). Improved characterisation and operationalization of social cognition and other 'hot' cognitive processes are necessary to facilitate and advance treatment efforts. There are currently a wide range of measures in use (Henry et al., 2015), which also makes direct comparisons between studies difficult. In an effort to combat this, test batteries such as EMOTICOM are currently under development, providing comprehensive computerised assessments for social cognitive and cognitive-affective processes that can be used across both healthy individuals and clinical populations (Bland et al., 2016).

4.5. Conclusion

Social cognitive deficits appear to be a core cognitive phenotype of many developmental, neurological and psychiatric disorders. There is a need to raise awareness of the importance of these difficulties among clinicians, researchers and patients alongside the more established aspects of cognition that may be negatively affected, such as attention and memory. Future studies should seek to address the 'real world' implications of these deficits and to develop effective transdiagnostic interventions for those individuals that are adversely affected.

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