



King's Research Portal

DOI:

[10.1097/MOP.0000000000000417](https://doi.org/10.1097/MOP.0000000000000417)

Document Version

Peer reviewed version

[Link to publication record in King's Research Portal](#)

Citation for published version (APA):

Happé, F. G. E., & Conway, J. R. (2016). Recent progress in understanding skills and impairments in social cognition. *Current Opinion in Pediatrics*, 28(6), 736-742. <https://doi.org/10.1097/MOP.0000000000000417>

Citing this paper

Please note that where the full-text provided on King's Research Portal is the Author Accepted Manuscript or Post-Print version this may differ from the final Published version. If citing, it is advised that you check and use the publisher's definitive version for pagination, volume/issue, and date of publication details. And where the final published version is provided on the Research Portal, if citing you are again advised to check the publisher's website for any subsequent corrections.

General rights

Copyright and moral rights for the publications made accessible in the Research Portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognize and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the Research Portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the Research Portal

Take down policy

If you believe that this document breaches copyright please contact librarypure@kcl.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.

REVIEW



Recent progress in understanding skills and impairments in social cognition

Francesca Happé and Jane R. Conway

Purpose of review

Social interaction is affected in many different developmental disorders; indeed, the new Diagnostic and Statistical Manual of Mental Disorders has introduced social cognition as one of six core components of neurocognitive functioning. Social cognition is not one thing, but a wide range of putative processes, which may be differentially affected in different clinical groups. This review focuses on recent advances in one aspect of social cognition, 'theory of mind' (ToM, representing what people think), and one core clinical group, autism spectrum disorder (ASD).

Recent findings

It is 30 years since impaired ToM was proposed as an explanation for ASD social difficulties, and recently there has been a widening of interest to other clinical groups. ToM has been found to be distinct from emotion recognition and empathy. Recent research on ASD has focused increasingly on atypical sensory responses and commonly comorbid conditions. Interventions for social deficits, including ToM training and oxytocin, have shown mixed results to date.

Summary

Heterogeneity poses a major obstacle to current research. Theoretical and empirical refinements are needed to elucidate neurocognitive and aetiological underpinnings of sociocognitive processes and inform clinical advances.

Keywords

autism spectrum disorder, heterogeneity, social cognition, theory of mind

INTRODUCTION

Social cognition, broadly speaking the processing of information about and from agents, is a potentially vast field of study. Considering the range of relevant processes, it is clear that recognizing and understanding conspecifics could call on the full panoply of domain-general cognitive capacities; perception, memory, attention, and so forth. Even if one restricted oneself to those processes that may have a special or possibly dedicated role in social cognition, there is a wide array. This might include empathy, emotion recognition and expression, imitation, biological motion and agent detection, attachment, social identity and in-group/out-group judgements, tracking social hierarchy, inference of traits and stereotyping, identity recognition (from face, voice, gait) and memory, and the attribution of mental states often referred to as 'theory of mind' (ToM). To further complicate matters, the interrelations or independence of these different putative processes is as yet unclear (see [1] for discussion and review of relevant data); we lack anything like the empirically

derived and replicated factor structure established in, for example, research on intelligence or personality.

Social cognition is also a vast topic because impairments of social interaction are notable in so many different clinical groups. Within disorders of childhood alone, social impairments have been documented in autism spectrum disorder (ASD), attention-deficit hyperactivity disorder (ADHD), agenesis of the corpus callosum, anxiety and especially social anxiety disorder (SAD), conduct disorder

MRC Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

Correspondence to Francesca Happé, Social, Genetic and Developmental Psychiatry Centre (MRC), Institute of Psychiatry, Psychology and Neuroscience - PO80, De Crespigny Park, Denmark Hill, London, SE5 8AF, UK. Tel: +44 (0)20 7848 0873; fax: + 44 (0)20 7848 0866; e-mail: francesca.happe@kcl.ac.uk

Curr Opin Pediatr 2016, 28:000–000

DOI:10.1097/MOP.0000000000000417

Neurology

KEY POINTS

- The new DSM-5 has introduced social cognition as one of six core components of neurocognitive functioning.
- 'ToM' (representing others' mental states) has been found to be distinct from emotion recognition and empathy.
- Recent research on ASD has focused increasingly on atypical sensory responses and commonly comorbid conditions.
- Interventions for social deficits, including ToM training and oxytocin, have shown mixed results to date.
- Heterogeneity poses a major obstacle to current research.

(especially of the callous/unemotional subtype), depression, developmental coordination disorder, fragile X syndrome, prematurity [2], reactive attachment disorder [3], specific language impairment [4], traumatic brain injury (TBI), Williams syndrome, and more. Indeed, Happé and Frith [5], reflecting on the many groups in whom social impairment is seen, asked whether this points to social cognition being particularly fragile and vulnerable to neurodevelopmental/acquired insult? Or is it rather that evolutionary pressure has made humans particularly sensitive to perturbations of social interaction, noticing even small derailments or mistakes that we could never pick up elsewhere, such as in motor or executive functions. The wide array of clinical groups in which social skills are affected probably also reflects the first point that there are many different aspects of social processing involved in everyday functioning. Disrupting any of these component social processes will lead to some decrement in social adaptation, quite apart from 'downstream' effects on social skills from deficits in domain-general processes (e.g. early social difficulties in congenitally blind children).

In light of this vast range of component processes and affected clinical groups, we have chosen to focus our review of the latest developments in social cognition research on one process and one clinical group. We have chosen 'ToM' as one of the most studied aspects of social cognition and ASD as the neurodevelopmental condition defined by impairments of social and communicative ability.

THEORY OF MIND

The ability to ascribe mental states to others, such as beliefs and intentions, plays a crucial role in social interactions and relationships. Since its

introduction in 1978 [6], the concept of 'ToM' has generated hundreds of studies exploring how humans represent other minds. Although the term is used diversely [7^{*}], much focus is placed on its development, impairments, and neural basis. The emergence of ToM abilities in typically and atypically developing children was first investigated 30 years ago [8]. The finding that children with ASD tended to fail ToM tasks, when those of the same mental age either typically developing or with Down's syndrome passed, has refined our understanding of ASD as characterized by specific sociocognitive impairments rather than simple lack of sociability [8].

Typically developing children usually pass explicit ToM tasks, like the Sally–Anne false-belief test (Fig. 1), around age 3–4 years [8–12]. Although some ASD individuals pass such tests, they tend to do so at much older ages and higher verbal abilities [13]. Eye tracking (e.g. anticipatory looking or longer gaze to events violating expectations) has been proposed to show that 'implicit' ToM, a suggested automatic ability to attribute mental states, is present in typically developing children from 15 months but absent in ASD adults [14]. However, measuring implicit ToM is challenging; current tests have questionable validity [15] and may not differentiate between alternative explanations [16].

Reliable activation in the medial prefrontal cortex and bilateral temporoparietal junction across various ToM measures suggests a specialized neural network for 'mentalizing' [17]. Further investigations of the right temporoparietal junction using brain stimulation methods reveal its relevance to several sociocognitive processes, although ToM-specific findings are inconsistent [18–21]. A recent, carefully controlled neuroimaging study showed that explanations of both social and nonsocial scenes activated the same brain regions, suggesting domain-general, however domain-specific recruitment of the dorsomedial prefrontal cortex in response to social stimuli was predicted by individual differences in social expertise [22^{*}].

REPRESENTING MENTAL STATES VS. EMOTIONS

Of particular significance is the need to reduce the heterogeneity of ToM in both definition and measurement [7^{*}]. The 'Reading the Mind in the Eyes' test (RMET; Fig. 2) asks participants to select emotional or mental state words that match photographs of the eye region of faces [23]. This test has been used extensively as a measure of ToM, however, its reliance on emotional words and facial expressions confounds the distinct abilities to

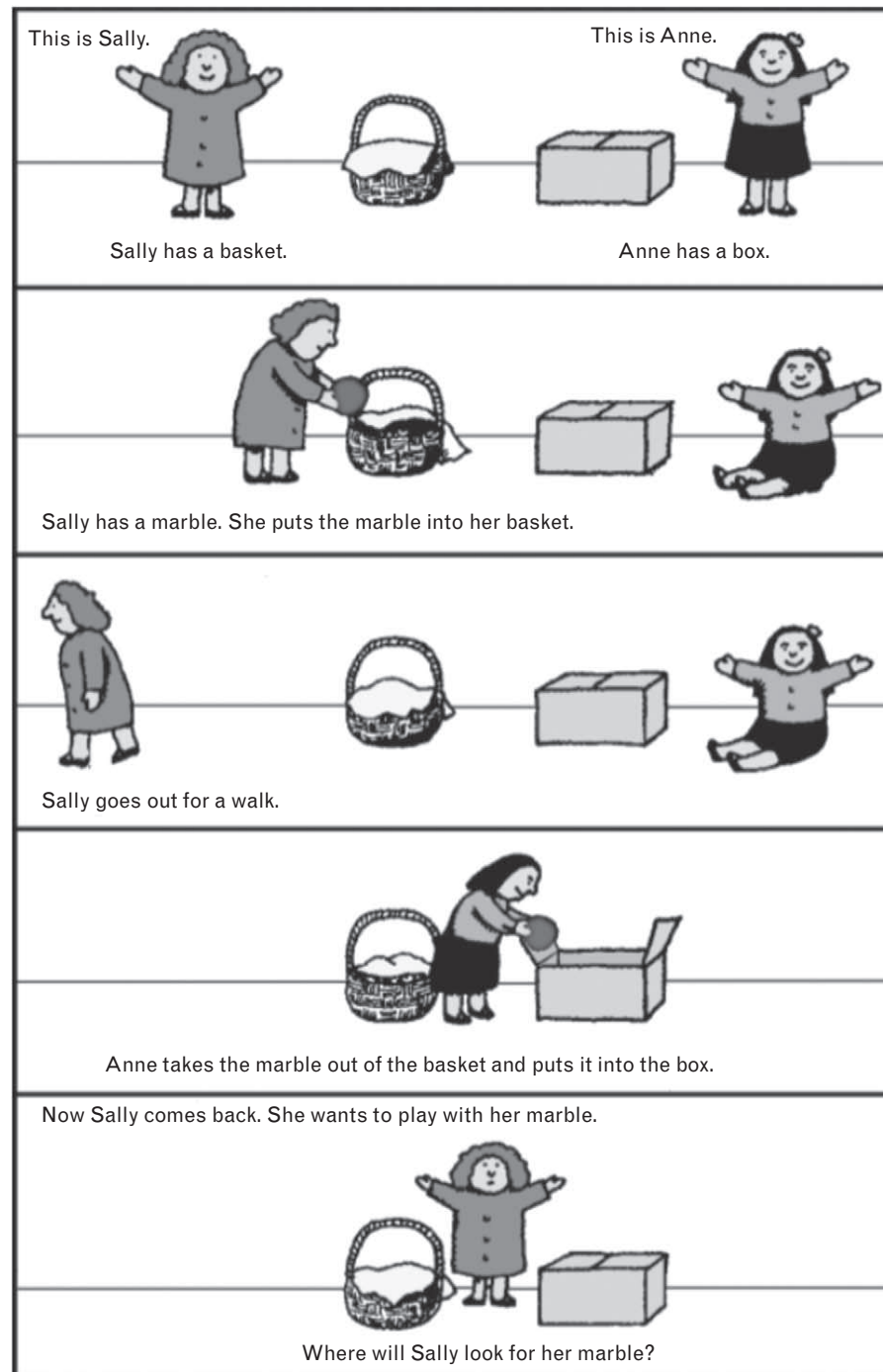


FIGURE 1. The Sally–Anne test. The Sally–Anne test is a false-belief measure of explicit ‘theory of mind’ in which children must verbally report their response. Adapted with permission from [9,10].

recognize emotions and represent mental states [24²²]. In ASD, emotion recognition impairments are only sometimes found, and recent findings suggest these in fact result from co-occurring alexithymia, a subclinical condition comprising difficulties recognizing one’s own emotions (and perhaps other interoceptive bodily states) [25–27]. Performance on the RMET is predicted by

alexithymia, not ASD, whereas performance on ToM tests is predicted by ASD, not alexithymia [24²²,28]. Thus, ToM is distinct from emotion recognition. ToM also appears to be distinct from affective empathy; whereas ASD involves difficulties knowing what others think; most people with ASD care what others feel. Indeed, a double dissociation can be seen contrasting ASD and psychopathy;

Neurology



FIGURE 2. The Reading the Mind in the Eyes test. Depicted is a sample question from the RMET in which participants must choose the word that best describes what the person in the picture is thinking or feeling. Adapted with permission from (http://docs.autismresearchcentre.com/tests/adult_part1.pdf) [23].

psychopathic adults and children who are callous and unemotional perform well on ToM tests (indeed, they may be Machiavellian and manipulate minds) but do not care about the emotional distress of others [29]. Indeed, twin modelling of trait data from over 5000 (nonclinical) twin pairs (aged 7–8 years) suggests that ASD-like social impairments and psychopathy-like callous-unemotional traits show little correlation and have distinct genetic and environmental aetiological influences [30].

THEORY OF MIND IN OTHER CLINICAL GROUPS

There has been a snowballing of interest in examining ToM in other clinical groups [31]. Recent studies of note include Ryan *et al.*'s [32] prospective longitudinal neuroimaging investigation of 112 children with TBI. They examined age at insult effects on social cognition 6 and 24 months after TBI occurring in middle childhood (5–9 years), late childhood (10–11 years), or adolescence (12–15 years), and the relevance of haemorrhaging lesion location, size, and number. Impairments in middle childhood were related to more diffuse neuropathology and, in adolescents, the number of lesions was also linked to performance on social tests. Additional control tasks would have been informative; executive functions such as inhibition and working memory show a strong relationship with ToM and are often tapped by ToM tests. Impairments on tasks purporting to measure ToM have also been reported in individuals with ADHD [33] but appear to be accounted for by executive function impairments rather than deficits in mental state representation *per se*. Language ability, too, shows an important relationship with ToM; late-signing deaf children show delayed ToM

development [34], and maltreated adolescents entering out-of-home care have also recently been reported to show ToM task deficits related to language ability [35].

AUTISM SPECTRUM DISORDER

ASD is a neurodevelopmental condition diagnosed on the basis of social and communicative impairments, accompanied by rigid and repetitive behaviour/ interests. The latest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) collapsed the previous categories of Asperger Disorder, autistic disorder, and pervasive developmental disorder – not otherwise specified/atypical autism into one spectrum, with clinicians instructed to specify the child's level of accompanying intellectual and language abilities. How this change will affect prevalence estimates is still unclear; a recent study using Danish population-based data suggests that 60% of the apparent rise in ASD over the last decades can be attributed to changes in reporting practices and diagnostic criteria [36]. The supposedly rising rates of ASD have raised concerns about environmental causes but the most recent twin studies confirm that genetic factors play the biggest role in aetiology [37,38].

Autism is increasingly conceptualized as a dimensional condition (at least at the behavioural level) and much research now examines ASD traits in the subclinical range [39]. The boundary between autistic traits and ASD depends on level of functional impairment and as such reflects the 'fit' between a person's characteristics and the demands of their environment. It has been suggested that the 4:1 men:women ratio may reflect differences in social adaptation or 'camouflaging', and that current diagnostic processes may under/misdiagnose women and girls with ASD [40–42,43].

SENSORY ISSUES

DSM-5 gives new prominence to abnormal sensory responses and behaviours in ASD [44]; hypo or hyper responsivity or unusual interest in sensory stimuli now appear as one of four types of 'restricted, repetitive patterns of behaviour, interest, or activities' (of which two are needed for diagnosis). An emerging focus is on how low-level sensory sensitivities might contribute to the atypical development of higher order sociocognitive processes [44]. Robertson and colleagues [45] used a binocular rivalry task to measure switching between two visual percepts in ASD and typical controls ($N=41$). Binocular rivalry dynamics reflect the activity of excitatory and inhibitory

neurotransmitters, and atypical binocular rivalry is observed in ASD [45[■],46]. Concentrations of the neurotransmitters γ -aminobutyric acid (GABA) and glutamate in the visual cortex were measured using magnetic resonance spectroscopy. No differences between groups were found for glutamate but GABA concentrations significantly predicted perceptual suppression in controls but not ASD. As the overall concentrations of both neurotransmitters were similar in both groups, the authors concluded that atypical GABAergic signalling pathways might underpin sensory symptomology in ASD.

EARLIEST INDICATORS OF AUTISM SPECTRUM DISORDER

An important goal in current ASD research is earlier identification of sociocognitive impairments and effective interventions to optimize outcome. Given the high heritability of ASD [38[■]], approximately 20% of siblings of ASD probands will also have ASD. Thus, prospective 'infant sib' follow-up studies offer a window into ASD's earliest presentations. Differences in social response before 12 months have not proven to reliably predict which infants later receive an ASD diagnosis [47]; however, delays in attentional and visuomotor skills from 6 months have been shown to have predictive value [48,49].

COMORBIDITIES

DSM-5 allows, for the first time, multiple disorders to be diagnosed alongside ASD; indeed, recent research suggests 'pure' ASD is rare and additional psychiatric problems (notably anxiety, depression, ADHD, and also sleep and eating problems) are common [50] and often amenable to treatment [51]. Siblings of those with ASD also show elevated rates of many disorders as shown in a recent study of three huge national registries in Finland [52[■]].

Anxiety, especially SAD, is very common in ASD and may exacerbate sociocognitive difficulties. Maddox and White [53] compared SAD symptoms in comorbid ASD+SAD ($N=14$) vs. ASD-SAD ($N=14$) and SAD alone ($N=26$). Half the ASD sample met criteria for SAD, with significantly more ASD women (69%) than men affected (33%). The ASD+SAD group reported significantly more social communication impairments and less social motivation than the ASD-SAD group, and significantly higher anxiety about social interactions than the SAD group. Recent typically developing adult studies suggest anxiety moderates everyday ToM ability; incidental experiences of uncertainty (e.g. when anxious or surprised) increased egocentric bias in perspective-taking tasks [54]. Anxiety may therefore

be a particularly important treatment target in those with ASD.

INTERVENTIONS

Interventions to improve sociocognitive abilities are clearly needed but have shown mixed effects to date. A recent review of 22 randomized controlled ToM interventions in ASD samples ($N=695$) showed little evidence of their efficacy; the only positive effect was improvement in emotion recognition in those of average intellectual ability [55]. A crucial, yet understudied, aspect of interventions is how children with ASD might differ from typically developing controls in their understanding of pedagogical communication [56[■]]. The first study to do so found that relative to younger typically developing controls ($N=35$), high-functioning children with ASD ($N=35$) were impaired in their understanding of teaching as an intentional activity requiring a knowledge gap between teacher and learner [56[■]]. The authors suggest that factoring this difference into future intervention design may increase efficacy. Greater understanding of pedagogical understanding in ASD could also help elucidate how their developmental environments differ from those of typically developing children or other clinical groups, and how this might contribute to a developmental cascade of impairments [9].

The neuropeptide oxytocin has been much investigated for its potential as a drug treatment for improving social cognition in ASD and other groups. One recent randomized controlled trial [57[■]] found no significant effect of oxytocin on parent/clinician-rated behaviour in teenagers with ASD but a significant placebo effect on caregiver reports of improvement. Indeed, meta-analyses show mixed findings, and there have been calls for increased theoretical and empirical rigor concerning study of oxytocin treatments [58,59].

CONCLUSION AND FUTURE DIRECTIONS

Autism is increasingly being referred to as the 'autisms' to capture the heterogeneity in presentation and likely neurocognitive and aetiological underpinnings. Heterogeneity is a stumbling block in research and also complicates clinical approaches. For example, infant sib studies of earliest ASD indicators may benefit from closer neurocognitive phenotyping of probands to understand variable outcome. Optimal outcome deserves more study; does good social outcome in a minority of those with ASD reflect late developing ToM or compensation, and can we teach either robustly? Lastly, although we have focused on ToM and ASD, better

Neurology

understanding of the wide range of sociocognitive processes and transdiagnostic studies will be important to allow clinicians to address the diverse ways in which social development can go awry.

Acknowledgements

None.

Financial support and sponsorship

None.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Happé F, Cook J, Bird G. The structure of social cognition: in(ter)dependence of sociocognitive processes. *Annu Rev Psychol* 2017. (in press).
2. Telford EJ, Fletcher-Watson S, Gillespie-Smith K, *et al.* Preterm birth is associated with atypical social orienting in infancy detected using eye tracking. *J Child Psychol Psychiatry* 2016; 57:861–868.
3. Davidson C, O'Hare A, Mactaggart F, *et al.* Social relationship difficulties in autism and reactive attachment disorder: Improving diagnostic validity through structured assessment. *Res Dev Disabil* 2015; 40:63–72.
4. Bakopoulou I, Dockrell JE. The role of social cognition and prosocial behaviour in relation to the socio-emotional functioning of primary aged children with specific language impairment. *Res Dev Disabil* 2016; 49:50:354–370.
5. Happé F, Frith U. Annual research review: towards a developmental neuroscience of atypical social cognition. *J Child Psychol Psychiatry* 2014; 55:553–577.
6. Premack D, Woodruff G. Does the chimpanzee have a theory of mind. *Behav Brain Sci* 1978; 4:515–526.
7. Schaafsma SM, Pfaff DW, Spunt RP, Adolphs R. Deconstructing and ■ reconstructing theory of mind. *Trends Cogn Sci* 2015; 19:65–72.
- The study outlines consequences and solutions to theoretical inconsistency in the 'ToM' concept.
8. Baron-Cohen S, Leslie AM, Frith U. Does the autistic child have a 'theory of mind'? *Cognition* 1985; 21:37–46.
9. Happé F. Autism as a neurodevelopmental disorder of mind-reading. *J Br Acad* 2015; 3:197–209.
10. Frith U. *Autism: Explaining the Enigma*. Oxford, UK: Blackwell.
11. Wimmer H, Perner J. Beliefs about beliefs: representation and constraining function of wrong beliefs in young children's understanding of deception. *Cognition* 1983; 13:103–128.
12. Wellman HM. *Making minds: how theory of mind develops*. New York, NY: Oxford University Press; 2014.
13. Happé F. The role of age and verbal ability in the theory of mind task performance of subjects with autism. *Child Dev* 1995; 66:843–855.
14. Senju A, Southgate V, White S, Frith U. Mindblind eyes: an absence of spontaneous theory of mind in Asperger syndrome. *Science* 2009; 325:883–885.
15. Phillips J, Ong DC, Surtees AD, *et al.* A second look at automatic theory of mind: reconsidering Kovács, Téglás, and Endress (2010). *Psychol Sci* 2015; 26:1353–1367.
16. Heyes C. False belief in infancy: a fresh look. *Dev Sci* 2014; 17:647–659.
17. Schurz M, Radua J, Aichhorn M, *et al.* Fractionating theory of mind: a meta-analysis of functional brain imaging studies. *Neurosci Biobehav Rev* 2014; 42:9–34.
18. Santiesteban I, Banissy MJ, Catmur C, Bird G. Enhancing social ability by stimulating right temporoparietal junction. *Curr Biol* 2012; 22:2274–2277.
19. Santiesteban I, Banissy MJ, Catmur C, Bird G. Functional lateralization of temporoparietal junction: imitation inhibition, visual perspective taking and theory of mind. *Eur J Neurosci* 2015; 42:2527–2533.
20. Mai X, Zhang W, Hu X, *et al.* Using tDCS to explore the role of the right temporo-parietal junction in theory of mind and cognitive empathy. *Front Psychol* 2016; 7:380.
21. Krall SC, Volz LJ, Oberwelland E, *et al.* The right temporoparietal junction in attention and social interaction: a transcranial magnetic stimulation study. *Hum Brain Mapp* 2016; 37:796–807.
22. Spunt RP, Adolphs R. Folk explanations of behavior: a specialized use of a ■ domain-general mechanism. *Psychol Sci* 2015; 26:724–736.
- A neuroimaging 'ToM' study showing that domain-specific recruitment of the dorsomedial prefrontal cortex varies as a function of social skill.
23. Baron-Cohen S, Wheelwright S, Hill J, *et al.* The 'Reading the Mind in the Eyes' Test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism. *J Child Psychol Psychiatry* 2001; 42:241–251.
24. Oakley B, Brewer RJ, Bird G, Catmur C. 'Theory of mind' is not theory of ■ emotion: a cautionary note on the reading the mind in the eyes test. *J Abnorm Psychol* 2016; 125:818–823.
- The study demonstrates that the 'RMET' test measures emotion recognition not 'ToM' (although used extensively as a test of the latter). Findings showed that emotion recognition, not mental state representation, is impaired in alexithymia, but the reverse is found in ASD.
25. Cook R, Brewer R, Shah P, Bird G. Alexithymia, not autism, predicts poor recognition of emotional facial expressions. *Psychol Sci* 2013; 24:723–732.
26. Brewer R, Happé F, Cook R, Bird G. Commentary on 'Autism, oxytocin and interoception': alexithymia, not autism spectrum disorders, is the consequence of interoceptive failure. *Neurosci Biobehav Rev* 2015; 56:348–353.
27. Bird G, Cook R. Mixed emotions: the contribution of alexithymia to the emotional symptoms of autism. *Transl Psychiatry* 2013; 3:e285.
28. Milosavljevic B, Carter Leno V, Simonoff E, *et al.* Alexithymia in adolescents with autism spectrum disorder: its relationship to internalising difficulties, sensory modulation and social cognition. *J Autism Dev Disord* 2016; 46:1354–1367.
29. Jones AP, Happé F, Gilbert F, *et al.* Feeling, caring, knowing: different types of empathy deficit in boys with psychopathic tendencies and autism spectrum disorder. *J Child Psychol Psychiatry* 2010; 51:1188–1197.
30. O'Nions E, Tick B, Rijdsdijk F, *et al.* Examining the genetic and environmental associations between autistic social and communication deficits and psychopathic callous-unemotional traits. *PLoS One* 2015; 10:1–12.
31. Henry JD, von Hippel W, Molenberghs P, *et al.* Clinical assessment of social cognitive function in neurological disorders. *Nat Rev Neurol* 2015; 12:28–39.
32. Ryan NP, Catroppa C, Cooper JM, *et al.* The emergence of age-dependent social cognitive deficits after generalized insult to the developing brain: a longitudinal prospective analysis using susceptibility-weighted imaging. *Hum Brain Mapp* 2015; 36:1677–1691.
33. Mary A, Slama H, Mousty P, *et al.* Executive and attentional contributions to Theory of Mind deficit in attention deficit/hyperactivity disorder (ADHD). *Child Neuropsychol* 2015; 7049:1–21.
34. Peterson C, Slaughter V, Moore C, Wellman HM. Peer social skills and theory of mind in children with autism, deafness, or typical development. *Dev Psychol* 2016; 52:46–57.
35. Kay C, Green J. Social cognitive deficits and biases in maltreated adolescents in UK out-of-home care: Relation to disinhibited attachment disorder and psychopathology. *Dev Psychopathol* 2016; 28:73–83.
36. Hansen SN, Schendel DE, Parner ET. Explaining the increase in the prevalence of autism spectrum disorders: the proportion attributable to changes in reporting practices. *JAMA Pediatr* 2015; 169:56–62.
37. Colvert E, Tick B, McEwen F, *et al.* Heritability of autism spectrum disorder in a UK population-based twin sample. *JAMA Psychiatry* 2015; 72:415–423.
38. Tick B, Bolton P, Happé F, *et al.* Heritability of autism spectrum disorders: a ■ meta-analysis of twin studies. *J Child Psychol Psychiatry* 2015; 57:585–595.
- A meta-analysis of twin studies demonstrating high heritability of ASD.
39. Blanken LME, Mous SE, Ghassabian A, *et al.* Cortical morphology in 6- to 10-year old children with autistic traits: a population-based neuroimaging study. *Am J Psychiatry* 2015; 172:479–486.
40. Lai MC, Lombardo MV, Auyeung B, *et al.* Sex/gender differences and autism: setting the scene for future research. *J Am Acad Child Adolesc Psychiatry* 2015; 54:11–24.
41. Halladay AK, Bishop S, Constantino JN, *et al.* Sex and gender differences in autism spectrum disorder: summarizing evidence gaps and identifying emerging areas of priority. *Mol Autism* 2015; 6:36.
42. Hiller RM, Young RL, Weber N. Sex differences in prediagnosis concerns for children later diagnosed with autism spectrum disorder. *Autism* 2016; 20:75–84.
43. Wilson CE, Murphy CM, McAlonan G, *et al.* Does sex influence the diagnostic ■ evaluation of autism spectrum disorder in adults? *Autism* 2016; doi: 10.1177/1362361315611381. [Epub ahead of print]
- This study showed sex differences in socio-communicative ASD symptoms vary according to ASD severity; no sex differences observed in comorbidity prevalence.
44. Baum SH, Stevenson RA, Wallace MT. Behavioral, perceptual, and neural ■ alterations in sensory and multisensory function in autism spectrum disorder. *Prog Neurobiol* 2015; 134:140–160.
- The article highlighted the sensory features of ASD.
45. Robertson CE, Ratai EM, Kanwisher N. Reduced GABAergic action in the ■ autistic brain. *Curr Biol* 2016; 26:80–85.
- GABA levels in the visual cortex, measured using magnetic resonance spectroscopy, predicted binocular rivalry dynamics in typical controls but not in participants with ASD. As overall GABA concentrations were similar in both groups, the authors concluded that atypical GABAergic signalling pathways might underpin sensory symptomatology in ASD.

Progress in understanding social cognition Happé and Conway

46. Robertson CE, Kravitz DJ, Freyberg J, *et al.* Slower rate of binocular rivalry in autism. *J Neurosci* 2013; 33:16983–16991.
 47. Ozonoff S, Iosif A-M, Baguio F, *et al.* A prospective study of the emergence of early behavioral signs of autism. *J Am Acad Child Adolesc Psychiatry* 2010; 49:256.e1-e2–266.e1-e2.
 48. Bolton PF, Golding J, Emond A, Steer CD. Autism spectrum disorder and autistic traits in the Avon longitudinal study of parents and children: Precursors and early signs. *J Am Acad Child Adolesc Psychiatry* 2012; 51:249.e25–260.e25.
 49. Suttera S, Pandey J, Esser EL, *et al.* Predictors of optimal outcome in toddlers diagnosed with autism spectrum disorders. *J Autism Dev Disord* 2007; 37:98–107.
 50. Tick B, Colvert E, McEwen F, *et al.* Autism spectrum disorders and other mental health problems: exploring etiological overlaps and phenotypic causal associations. *J Am Acad Child Adolesc Psychiatry* 2016; 55:106.e4–113.e4.
 51. Frye RE, Rossignol DA. Identification and treatment of pathophysiological comorbidities of autism spectrum disorder to achieve optimal outcomes. *Clin Med Insights Pediatr* 2016; 10:43–56.
 52. Jokiranta-Olkonien E, Cheslack-Postava K, Sucksdorff D, *et al.* Risk of psychiatric and neurodevelopmental disorders among siblings of probands with autism spectrum disorders. *JAMA Psychiatry* 2016; 73:622–629.
- A large Finnish population-based cohort showed that siblings of those with ASD also show elevated rates of many psychiatric and neurodevelopmental disorders indicating possible common risk factors.
53. Maddox BB, White SW. Comorbid social anxiety disorder in adults with autism spectrum disorder. *J Autism Dev Disord* 2015; 45:3949–3960.
 54. Todd AR, Forstmann M, Burgmer P, *et al.* Anxious and egocentric: how specific emotions influence perspective taking. *J Exp Psychol Gen* 2015; 144:374–391.
 55. Fletcher-Watson S, McConnell F, Manola E, McConachie H. Interventions based on the Theory of Mind cognitive model for autism spectrum disorder (ASD). *Cochrane Database Syst Rev* 2014; Art. No.: CD008785. doi: 10.1002/14651858.CD008785.pub2.
 56. Knutsen J, Mandell DS, Frye D. Children with autism are impaired in the understanding of teaching. *Dev Sci* 2015; doi: 10.1111/desc.12368. [Epub ahead of print]
- The study found that children with ASD are impaired in their understanding of pedagogical communication.
57. Guastella AJ, Gray KM, Rinehart NJ, *et al.* The effects of a course of intranasal oxytocin on social behaviors in youth diagnosed with autism spectrum disorders: a randomized controlled trial. *J Child Psychol Psychiatry Allied Discip* 2015; 56:444–452.
- The double-blind, placebo-controlled trial examined the effects of oxytocin vs. placebo nasal sprays on social behaviour in adolescents with ASD. Results showed no effects of oxytocin but a significant placebo effect on caregiver reports of improvement.
58. Leng G, Ludwig M. Intranasal oxytocin: myths and delusions. *Biol Psychiatry* 2016; 79:243–250.
 59. Guastella AJ, Hickie IB. Oxytocin treatment, circuitry, and autism: a critical review of the literature placing oxytocin into the autism context. *Biol Psychiatry* 2016; 79:234–242.