# Clinical assessment of social cognitive function in neurological disorders

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Abstract | Social cognition broadly refers to the processing of social information in the brain that underlies abilities such as the detection of others' emotions and responding appropriately to these emotions. Social cognitive skills are critical for successful communication and, consequently, mental health and wellbeing. Disturbances of social cognition are early and salient features of many neuropsychiatric, neurodevelopmental and neurodegenerative disorders, and often occur after acute brain injury. Its assessment in the clinic is, therefore, of paramount importance. Indeed, the most recent edition of the American Psychiatric Association's Diagnostic and Statistical Manual for Mental Disorders (DSM-5) introduced social cognition as one of six core components of neurocognitive function, alongside memory and executive control. Failures of social cognition most often present as poor theory of mind, reduced affective empathy, impaired social perception or abnormal social behaviour. Standard neuropsychological assessments lack the precision and sensitivity needed to adequately inform treatment of these failures. In this Review, we present appropriate methods of assessment for each of the four domains, using an example disorder to illustrate the value of these approaches. We discuss the clinical applications of testing for social cognitive function, and finally suggest a five-step algorithm for the evaluation and treatment of impairments, providing quantitative evidence to guide the selection of social cognitive measures in clinical practice.

Humans are inherently social creatures. Social behaviours emerge in the early stages of infancy[1] and remain critical throughout the lifespan[2,3]. Much of our everyday behaviour is motivated by social and emotional goals; indeed, the disproportionately large size of the human brain might be the result of evolutionary pressures to negotiate complex social systems[4]. For this reason, social cognition — the means by which we perceive, process and interpret social information — is a fundamental neurocognitive capacity. A critical role for social cognition in functional disability is now well established: social cognitive impairment has been linked to poor quality of life, mental health problems, unemployment and loneliness[5–7].

Nearly all neurological disorders that affect the brain have the potential to disrupt social cognitive function. Social cognitive impairment can be a prominent clinical symptom after acute brain damage, such as traumatic brain injury or stroke, and can be a core feature of the early stages of some chronic neurological disorders, such as behavioural-variant frontotemporal dementia (bvFTD)[8]. However, in the early stages of many neurological disorders, such as Alzheimer disease (AD), Parkinson disease and multiple sclerosis, social cognitive disturbances might be relatively subtle and harder to detect informally. Structured social cognitive assessment is, therefore, useful in a wide range of neurological conditions. In patients with acute brain trauma, or if a patient's history or diagnosis could indicate social cognitive dysfunction, social cognitive assessment should be part of the initial standard neurological examination. Even if no impairment is identified, such assessment should be included in routine follow-up in neurological disorders that are associated with social cognitive impairment.

Failures of social cognition most often present clinically in one or more of four ways: impaired theory of mind (ToM), reduced emotional empathy, poor social perception, and abnormal social behaviour. ToM refers to our ability to understand the mental states of others, and to appreciate that these mental states might differ from our own. Affective ToM requires an understanding of others' emotions, affective states or feelings (and overlaps with the construct of cognitive empathy),

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## Key points

- Social cognitive deficits are prominent in many conditions and are critical predictors of functional outcomes because they affect the ability to form and sustain interpersonal relationships

- Assessments of social cognitive impairments typically focus on theory of mind, affective empathy, social perception and social behaviour, four domains that all influence the management of a patient

- Many social cognitive assessment measures that are appropriate for clinical use are now available and should form part of a broader neurocognitive battery

- Common disorders that manifest with prominent social cognitive deficits include schizophrenia, autism spectrum disorders, Alzheimer disease, and behavioural-variant frontotemporal dementia

- A range of effective treatment strategies are currently available, so the nature, magnitude and specificity of social cognitive impairments each have important implications for therapeutic decision-making

whereas cognitive ToM requires an understanding of others' cognitive states, beliefs, thoughts or intentions. Affective empathy is one's emotional response to the perceived situations of others. These responses can be experienced as the same emotions that the other person feels (an empathic response that is often referred to as affective resonance or experience sharing), or can be distinct from the experience of others, for example if we feel embarrassed for someone who is overconfident[9]. Emotional responses that are primarily self-oriented rather than other-oriented, such as personal distress, are not empathic responses. By allowing us to understand others' mental states and experience their emotions, ToM and affective empathy have an important role in prosocial behaviour, inhibition of aggression and moral reasoning. Failures of social perception typically manifest as problems with recognizing and responding to basic social and emotional cues, such as interpreting facial expressions, body language or voices, or responding to social cues, such as eye gaze. Social perceptual

## Box 1 | Indications of social cognitive impairment

- Social withdrawal or avoidance of social contact
- Loss of social graces
- Limited eye contact
- Rude or offensive comments without regard for the feelings of others
- Loss of etiquette in relation to eating or other bodily functions
- Extended speech that generally lacks focus and coherence
- Neglect of personal appearance (in the absence of depression)
- Disregard of the distress or loss of others
- Inability to share in the joy or celebrations of others when expected or invited
- Failure to reciprocate socially, even when obvious social cues are given
- Poor conversational turn-taking
- Overtly prejudicial or racist behaviour
- Increased or inappropriate interpersonal boundary infringements
- Failing to understand jokes or puns that are clear to most people
- Failure to detect clear social cues, such as boredom or anger, in conversational partners
- Lack of adherence to social standards of dress or conversational topics
- Excessive focus on particular activities to the exclusion of important social or occupational demands

deficits fundamentally disrupt the ability to make sense of social interactions and respond appropriately. Indeed, impairments of social behaviour often arise as a direct consequence of social perceptual failures, such as when social cues have been missed or misinterpreted. Social behavioural abnormalities include poor social tact, a lack of manners, interpersonal boundary infringements, reduced use of communicative gestures and unsolicited affiliative contact with strangers (BOX 1).

In this Review, we consider the clinical contexts in which social cognitive dysfunction arises, and the neurobiological basis for this dysfunction. We then consider how this dysfunction is best assessed, presenting example disorders in which a specific social cognitive domain should be tested, and the tests that can be used to meet the clinical needs of patients with such a dysfunction. Finally, we consider the broader use of these tests in the clinic, and future directions in this area.

## Social cognition in clinical contexts

Arguably, most psychiatric and neurological illnesses are associated with some level of social cognitive impairment that has the potential to disrupt interpersonal relationships[8]. Illnesses that are known to involve social cognitive impairment range from disorders in which social cognitive deficits are core diagnostic criteria, such as bvFTD and autism spectrum disorders (henceforth referred to as autism), to disorders in which social difficulties are often a prominent concern, such as Parkinson disease and AD (BOX 2).

## Neurobiological basis
### Neuroanatomical disturbances
Distinct disturbances of social cognition and function have been linked to abnormalities in specific neural regions. For example, lesions in the orbitofrontal cortex (OFC) are associated with disinhibited behaviours, such as social inappropriateness, hypersexuality and compulsive gambling[10]. This association is believed to reflect the critical role of the OFC in reinforcement-guided decision-making[11]. Lesions in the anterior cingulate cortex (ACC) are associated with behavioural disturbances that include abulia, or its more severe form akinetic mutism, reflecting the ACC's involvement in regulating motivational and emotional behaviour[12]. Damage to the temporoparietal junction has been shown to disrupt the ability to view a situation from an another person's perspective, and has also been linked to abnormal moral reasoning[13,14]. These disturbances are believed to arise because the temporoparietal junction has a central role in integrating social, attentional, memory and language processing streams to construct a social context for behaviour[15].

The ubiquity of social cognitive difficulties among clinical populations is unsurprising given that substantial overlapping and interacting functions exist across brain regions and that evidence shows that social cognition imposes demands on a large number of different brain structures and their connectivity[8]. Specific brain regions are consistently implicated in each of the four social cognition networks (FIG. 1), but overlap

exists between the areas involved in the four networks. Furthermore, many other brain areas are also implicated, and these areas are involved in other functions in addition to social cognition.

Deficits of social cognition can result from damage to the brain regions involved in such cognition or their connections, and should be understood as a disruption of the interactions within and between large-scale social cognition networks. The functional integrity of these networks can be disrupted by relatively mild dysfunction in one structure that is involved, diffuse dysfunction or white-matter damage[8]. Alternative sources provide excellent, detailed descriptions of the specific brain regions involved in ToM[16], empathy[17], social perception[18], and social behaviour[12].

### Neurotransmitter disturbances

Functional abnormalities in neurotransmitters, such as serotonin, γ-aminobutyric acid (GABA) and dopamine, have also been linked to social cognitive dysfunction[19]. The relationship between neurotransmitter levels and cognitive functioning generally follows the Yerkes–Dodson law, and is best described by an inverted U: optimal function requires neurotransmitter levels to be neither too low nor too high. Experimental manipulation of neurotransmitter levels with, for example, acute tryptophan depletion (which decreases CNS levels of serotonin) or drugs such as sulpiride (a dopamine antagonist) or diazepam (a $GABA_A$ receptor modulator that increases the effects of GABA) influences social cognitive function[20–22].

The neuropeptides oxytocin and vasopressin, both of which exert widespread neuromodulatory effects, have a particularly critical role in social cognition and behaviour[19,23]. Behavioural studies have shown that higher peripheral levels of oxytocin correlate with more positive social behaviour[24,25]. In addition, variations in the genes that encode these 'social neuropeptides' have been linked to individual differences in aspects of social behaviour, such as empathy[26], prosociality[27] and autistic-like traits[28], and to heritable disorders, including autism[29]. Furthermore, intranasal administration of oxytocin and vasopressin has been shown to influence socioemotional function[30], a finding that underlies considerable interest in the potential therapeutic use of these agents[31].

### Clinical assessment of social cognition

The clinical assessment of social cognitive function is important in many neurological disorders, including acute brain trauma, such as stroke and traumatic brain damage, and chronic neurological disorders, such as AD and Parkinson disease. This importance is now formally recognized in the fifth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5)[32], which includes social cognition as one of six core neurocognitive domains. Standardized tests are essential for objective quantification of the extent and severity of impairment and for the identification of the strongest residual abilities that can be used to compensate for deficits, yet the DSM-5 does not name any proprietary tests.

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**Box 2 | Disorders with social cognitive impairment**

**Psychiatric disorders**
- Schizophrenia
- Bipolar disorder
- Antisocial personality disorder
- Major depressive disorder
- Post-traumatic stress disorder
- Social phobia
- Anorexia nervosa
- Personality disorders (for example, borderline, antisocial, narcissistic, schizoid, avoidant)

**Developmental disorders**
- Autism spectrum disorder
- Fragile X syndrome
- Williams syndrome
- Angelman syndrome
- Prader–Willi syndrome
- Turner syndrome
- Rett syndrome
- Attention deficit hyperactivity disorder
- Severe conduct disorder
- Fetal alcohol syndrome

**Neurodegenerative disorders**
- Frontotemporal dementia
- Alzheimer disease
- Amyotrophic lateral sclerosis
- Parkinson disease
- Huntington disease
- Progressive supranuclear palsy
- Corticobasal degeneration
- Multiple sclerosis

**Acute brain damage**
- Traumatic brain injury
- Stroke

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In order to facilitate clinical decision-making, in the following sections we present four example disorders to illustrate the appropriate methods of assessment for impairment of each domain of social cognition: poor ToM, reduced affective empathy, impaired social perception and abnormal social behaviour. Important to keep in mind, however, is the fact that many clinical disorders — including all four examples presented in this Review — involve impairment in multiple social cognitive domains.

Measures were selected on the basis of either their wide use in clinical practice, or their potential advantages over more commonly used measures. Measures that are known to have good reliability and clinical sensitivity were prioritized. The inclusion of an appropriate control was also an important consideration in test selection because deficits of social cognition can be secondary to other cognitive deficits rather than the result of a primary disturbance; fully understanding the origin and specificity of the deficit is necessary for making appropriate therapeutic decisions. Most clinical
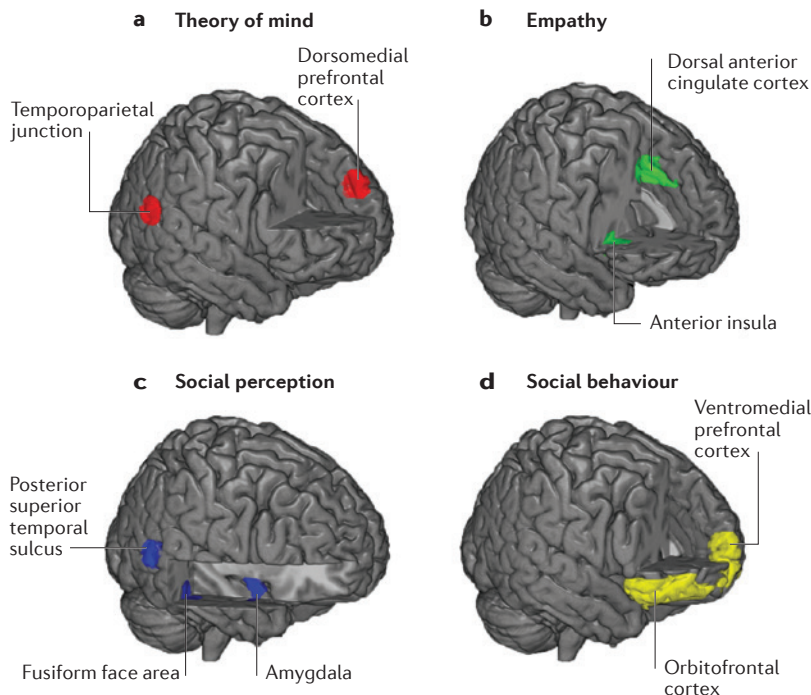
**a | Theory of mind**

Dorsomedial prefrontal cortex

Temporoparietal junction

**b | Empathy**

Dorsal anterior cingulate cortex

Anterior insula

**c | Social perception**

Posterior superior temporal sulcus

Fusiform face area

Amygdala

**d | Social behaviour**

Ventromedial prefrontal cortex

Orbitofrontal cortex

Figure 1 | **Brain regions that are consistently involved in the four social cognition networks. a |** The dorsomedial prefrontal cortex integrates social information across time and enables reflection and cognitive representation of traits and norms, whereas the temporoparietal junction represents temporary goals and intentions[113]. **b |** The dorsal anterior cingulate cortex is often involved in the cognitive aspects of empathy, whereas the anterior insula is more often involved in the affective aspects of empathy[114]. Both areas are often active when watching others in pain[115]. **c |** The posterior superior temporal sulcus is often activated in response to real or implied biological motion, specifically in relation to social cues[116]. The fusiform face area is critical in the face perception network[117]. The amygdala is often associated with the social perception network because it attributes either a positive or negative emotional valence to stimuli. **d |** Activation in the orbitofrontal cortex and ventromedial prefrontal cortex is not essential for affective responses, but is critical for the attribution of meaning to an affective stimulus[118]. Activation in the lateral part of this prefrontal region is often associated with a feeling of displeasure and inhibits behaviour, whereas activation in more medial parts typically reinforces behaviour through feelings of pleasure[119]. Damage to these areas often leads to inappropriate social behaviour.

studies of social cognition have focused on adults[33], so this Review focuses primarily on measures that are suitable for use in adult populations. The false-belief understanding task, however, was developed to characterize developmental changes in preschool children and in middle childhood, so is appropriate for use in young cohorts. Child-appropriate versions of several other of the tasks discussed are available, and we provide details where this is the case.

### Theory of mind: schizophrenia
Schizophrenia is a clinically heterogeneous disorder that is characterized by positive symptoms (such as delusions and hallucinations), negative symptoms (such as anhedonia, avolition and affective flattening) and disorganized speech and behaviour. Severe impairments of social interaction and associated abnormalities are also key features: evidence of social or occupational dysfunction is a prerequisite for diagnosis[32]. Specific impairments of social function include difficulty in maintaining

relationships with family and friends, disengagement from socially important activities, such as work and study, and poor self-care. These clinical symptoms and behavioural abnormalities are believed to reflect a disorder of brain network organization, or functional dysconnectivity[34].

Impairment of social functioning is not only critical to the initial diagnosis of schizophrenia, but is also one of the most important predictors of long-term prognosis[35]. However, social functioning is only moderately related to clinical symptomatology and the outcomes of standard neurocognitive assessment. Instead, a patient's ability to infer what others are thinking and feeling, and to reason about how their thoughts and feelings will influence their behaviour, seems to be central to understanding the poor occupational and social functioning evident in many people with schizophrenia[36]. Assessing a patient's capacity for ToM is, therefore, critical for treatment and rehabilitation.

False-belief tasks[37] are extensively validated measures of ToM that assess the ability to disregard one's own knowledge about the world and consider that someone else might have a different, erroneous belief. Relative to healthy controls, people with schizophrenia often exhibit a reduced capacity for false-belief understanding[38,39]. When engaged in false-belief reasoning, these patients also exhibit less recruitment of neural circuitry that has been related to ToM than do healthy controls; these neural abnormalities seem to relate to a patient's level of social adjustment[40]. Most evidence indicates that difficulties with false-belief understanding in schizophrenia are not simply related to secondary cognitive task demands, such as working memory[38,39], but instead reflect more fundamental problems with mental state reasoning.

Measures that assess social inference, such as the ability to detect *faux pas* and to interpret speech with hidden meanings, such as sarcasm, also provide insight into when and why ToM difficulties are expected to disrupt social interaction. For instance, Parts 2 and 3 of The Awareness of Social Inference Test (TASIT)[41] — a video-based measure that depicts actors in different social scenarios — assess the understanding of sincere and sarcastic interpersonal exchanges. Studies that used the TASIT have shown that people with schizophrenia can understand sincere social exchanges but struggle to comprehend sarcasm[42,43]. Social inference can also be assessed with the Strange Stories test[44], which involves patients reading a series of written stories. People with schizophrenia are impaired in their ability to understand stories in which a character's behaviour is best explained by assuming that they have knowledge of another character's underlying mental state[45]. The Faux-Pas Test[46] also involves a series of written stories but assesses a more specific aspect of social inference: the ability to identify social gaffes. As in the Strange Stories test, people with schizophrenia can understand the factual content of the stories, but their ability to identify social *faux pas* is impaired[47].

In addition to profound impairments of 'high-level' social inferences and mental state reasoning, people with schizophrenia often exhibit deficits in more-basic

mental-state decoding — the ability to make inferences on the basis of observable features, such as facial expression and eye gaze, for example. The Reading the Mind in the Eyes Test (RMET)[48] is the most commonly used and extensively validated method of assessing this behavioural deficit. The test involves asking participants to infer the mental state of a person on the basis of a photograph of their eyes and the surrounding area. People with schizophrenia have greater difficulty than do healthy controls in using these eye gaze cues to determine what another person is thinking or feeling[45]. An important clinical strength of the RMET is that it imposes minimal demands on 'higher level' cognitive control operations, such as working memory and abstract reasoning, that are commonly required in other measures of ToM. However, visual and verbal demands of the RMET mean that apparent deficits can, in fact, be a consequence of broader visuoperceptual impairment or aphasia.

As already noted, ToM deficits are not unique to schizophrenia: a large body of literature on the topic demonstrates that ToM is disrupted in a wide range of neuropsychiatric, neurodegenerative and neurodevelopmental disorders[8], and is often impaired after acute brain damage[49]. In many of these disorders, as in schizophrenia, ToM deficits are not simply attributable to secondary task demands, and have been linked to important functional outcomes. However, the literature also shows that considerable heterogeneity exists between and within different clinical populations with respect to the nature, severity and specificity of ToM impairments, an observation that reinforces the need for objective measures to inform therapeutic decision-making on a case-by-case basis (TABLE 1).

### Affective empathy: autism

Autism is one of the most common neurodevelopmental disorders: the estimated prevalence is 1 in 132 people[50]. The condition is characterized by a restricted repertoire of interests and activities, and deficits in communication skills and social interaction[32]. It has been linked to a range of neural abnormalities, including aberrant functional connectivity[51]. Maladaptive emotional reactions are common among people with autism, and include a reduced affective empathic response[52]. Reduced distress–response measures in infants as young as 12 months are predictive of a later autism diagnosis[53]. However, some studies indicate that emotional empathic responses can be intact[54], or even heightened, in people with autism, indicating a more general affective imbalance[55]. This heterogeneity could indicate that some atypical emotional reactions in people with autism reflect problems with understanding the perspectives of others rather than a lack of care or concern per se[56].

Valuable clinical insight into the social cognition of people with autism can be gained from self-report measures of affective empathy (BOX 3). Such measures typically involve a series of simple statements that directly enquire about the degree to which a person experiences warm, concerned or compassionate feelings for others.

One of the most extensively validated of these measures is the Empathic Concern subscale of the Interpersonal Reactivity Inventory (IRI-EC)[57]. When administered alongside the Perspective-Taking subscale of the IRI (IRI-PT), the IRI-EC can distinguish between affective abnormalities that reflect a lack of caring and those that reflect a lack of understanding. For a broader understanding of empathic difficulties, the Empathy Quotient (EQ)[58] should also be considered, as this measure provides insight into both affective and cognitive empathy. Most, but not all, studies that have used the measures described above have found self-rated empathy to be lower in people with autism than in controls[54,59,60].

Nevertheless, self-report requires emotional insight and a willingness to self-disclose personal information, so in most clinical groups, self-report measures should be supplemented with other assessments of affective empathy. These other assessments are particularly important for specific clinical groups, such as people with autism, which is highly comorbid with alexithymia[61], a personality construct characterized by difficulties in identifying and describing emotions and in distinguishing feelings from the physical sensations of emotional arousal. Many individuals with autism also have intellectual impairments[32].

Although observation during a clinical interview can provide potentially valuable insights into affective empathic disturbances, in highly structured situations, patients might behave similarly to controls[62], meaning that empathic deficits might not be evident during brief observations. For these reasons, informant-rated and emotion-relevant performance tasks might provide the clearest clinical insights into the affective empathic disturbances associated with autism, particularly when combined with self-report.

The most widely used informant-rated measures of affective empathy are simple modifications of self-report measures, such as the IRI-EC or EQ. Several studies that have used these measures have revealed autism-related deficits; in some cases, they identified greater impairments than did the corresponding self-report versions of these scales[63]. In emotion-relevant performance tasks, emotionally arousing videos[64] or photographs[59,65] are presented to participants, who are asked to rate their emotional response. One such measure is the Multifaceted Empathy Test (MET)[59], which differentiates between mental state understanding (cognitive empathy) and subjective emotional response (affective empathy). This test has been used to identify abnormalities of affective empathic responding in people with autism[65].

Affective empathy is impaired in many other disorders that present with relatively diffuse brain damage, including traumatic brain injury and dementia[66], and in many personality disorders. For instance, the affective empathic response is dysfunctional in narcissistic personality disorder[67], and a lack of affective empathy is a defining feature of antisocial personality disorder. Moreover, people with psychopathic personality disorders exhibit a specific breakdown of the neural processes that support the ability to experience others'

Table 1 | Descriptions of stimuli from theory of mind measures

| Measure | Experimental task | Control task(s) |
|---|---|---|
| False-belief Tasks[37] | Participants are told a story that involves two characters, Sally and Anne. In one example, Sally has a basket and Anne has a box. Sally puts a marble in her basket, then goes for a walk. While Sally is away, Anne moves the marble to the box. Sally comes back and wants to play with her marble. At the end of this story, participants are asked where Sally will look for her marble. The task measures whether a participant can understand that Sally holds a belief that is different to their own, and which is contrary to reality (a false belief) | Non-mental reality control questions that assess participants' understanding of the situation and/or a series of true belief scenarios |
| The Awareness of Social Inference Test[41] | Questions focus on the ability to detect sarcasm in a social interaction | Questions focus on the ability to detect sincerity in a social interaction |
| Strange Stories Test[44,120] | Participants are asked to demonstrate their understanding of a written story in which a character's behaviour can be best understood by attributing to them a specific underlying mental state | Identical to the experimental task, except a character's behaviour can be explained without any need for mental inference |
| Faux-Pas Test[46] | Participants are read a story that contains a *faux pas* and subsequently asked questions that focus on their ability to detect the *faux pas*, and to understand beliefs, intentions and inappropriateness | Identical to the experimental task, except participants are asked questions that focus on a protagonist's behaviour and do not require mental inference to answer |
| Reading the Mind in The Eyes Test[48] | Participants are shown photographs of the eye regions of people's faces, and asked to select one of four alternatives describing what the person in the photograph is thinking or feeling | No standard control task exists; in some studies, participants are shown the same photographs as in the experimental task, but are asked to select which age range or gender is correct for each person in the photographs |

emotions[68]. Such difficulty in identifying with the distress of others should be regarded as particularly clinically important, as it has been linked to premeditated and goal-directed acts of aggression[69].

### Social perception: Alzheimer disease

AD is the most common cause of dementia. The disease involves gradual and progressive neurodegeneration that initially affects the hippocampi, entorhinal cortex and posterior cingulate cortex, and subsequently the entire temporal, parietal and frontal cortices[70]. Mild episodic memory impairment is often the earliest cognitive marker of AD[71], but with disease progression, memory deficits become more severe, and impairments in other neurocognitive domains become increasingly evident. Deficits in social cognition have also emerged as an important aspect of the disease. Evidence has shown that such deficits explain aspects of patients' functional dependence that are independent of the effects of deficits in general cognition[72], and are related to problems with managing treatment and behaviour[73], increased agitation,[74] and poor interpersonal relationships[75]. In particular, social difficulties and behavioural abnormalities related to AD have been linked to deficits in interpreting cues to emotional states. Consequently, basic social perceptual functioning should be considered when modelling the effects of AD on important clinical and behavioural outcomes, such as mental health and social functioning.

Social perceptual failures often manifest as difficulties with identifying others' emotions, and many measures are available that assess this ability through the presentation of static photographs of high-intensity facial expressions (TABLE 2). The most extensively validated stimuli are the Ekman Faces[76], which are black and white photographs that depict the six basic emotions (disgust, anger, fear, surprise, sadness and happiness) and neutral faces. Most studies that have used the Ekman Faces to assess patients with AD have identified impairments in patients when compared with healthy controls[77–79]. AD-related deficits have also been identified when different sets of photographs[79], schematic line drawings of faces[73] or 3D virtual actors have been used[80]. However, most standard measures of facial expression

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Box 3 | Measures of affective empathy

**Empathic Concern[57]**
- Self-rated or informant-rated
- Participants are asked about feelings of warmth, compassion and concern for others

**Empathy Quotient[58]**
- Self-rated or informant-rated
- This measure assesses the ability to understand and predict others' behaviour, and the nature of any emotional response to other people

**Multifaceted Empathy Test[59]**
- Performance task
- The empathic responses of participants to emotionally intense photographic images are assessed

Table 2 | **Descriptions of stimuli from social perception measures**

| Measure | Experimental task | Control task(s) |
|---|---|---|
| Ekman Faces – Emotion Labelling[76] | Participants are shown a picture of a face and asked which emotion is depicted; emotion labels are typically provided, and participants are asked to choose between them | Executive control and language tasks |
| Ekman Faces – Emotion Discrimination[76] | Participants are shown two faces concurrently and asked whether they show the same or different emotions | Facial recognition |
| Facial Expressions of Emotion – Stimuli and Tests[81] | Uses the Ekman Faces; photographs can depict expressions with 100% intensity (as in the standard Ekman Faces), but computerized morphing and caricaturing procedures are also available to modulate emotion intensity | Depends on whether the task involves emotion labelling (in which case executive control and language control tasks should be used) or emotion discrimination (in which case a facial recognition control task should be used) |
| Comprehensive Affect Testing System[82] | • Subtest 1: Ekman 3-faces task<br>• Subtest 3: Affect matching<br>• Subtest 4: Affect discrimination<br>• Subtest 5: Affect naming<br>• Subtest 6: Prosody identification<br>• Subtest 7: Prosody naming<br>• Subtest 9: Emotional prosody discrimination<br>• Subtest 10: Match emotional prosody to face<br>• Subtest 11: Match emotional face to prosody<br>• Subtest 12: Conflicting facial emotion and prosody — respond to face<br>• Subtest 13: Conflicting facial emotion and prosody — respond to prosody | • Subtest 2: Identity matching<br>• Subtest 8: Non-emotional prosody discrimination |
| Florida Affect Battery[83] | • Subtest 2: Facial affect discrimination<br>• Subtest 3: Facial affect naming<br>• Subtest 4: Facial affect selection<br>• Subtest 5: Facial affect matching<br>• Subtest 7: Emotional prosody discrimination<br>• Subtest 8: Name the emotional prosody<br>• Subtest 9: Match emotional prosody to an emotional face<br>• Subtest 10: Match emotional face to the emotional prosody | • Subtest 1: Facial identity discrimination<br>• Subtest 6: Non-emotional prosody discrimination |
| The Awareness of Social Interference Test Part 1: Emotion Evaluation Test[41] | Participants are shown videos in which an actor portrays one of seven basic emotions, sometimes with ambiguous dialogue, sometimes without any dialogue. Participants are asked to identify the emotional expression depicted | Executive control and language tasks |

recognition present extreme emotional intensities, so when assessment of subtle social perceptual impairment is required, measures that present less intense facial expressions should be used. The Facial Expressions of Emotion: Stimuli and Tests[81] includes images that vary in their emotional intensity, enabling clinicians to create tasks that are graded in difficulty. A study that used this measure showed that AD-related deficits in identifying emotions were greater when expressions with an intensity of 75% were presented than when those with an intensity of 100% were presented[5].

The breadth and specificity of difficulties in recognizing emotions can be assessed with batteries of tests such as the Comprehensive Affect Testing System[82] and the Florida Affect Battery[83], which use not only visual stimuli, but also auditory. Both of these batteries incorporate multiple subtasks that assess the ability to process visual (facial expressions), auditory (prosody) and visual–auditory (simultaneous facial expressions and prosody) emotional information. Use of these measures has shown that some subtasks are

impaired in AD but some are not[84,85], indicating that patients have residual strengths with implications for individualized interventions.

Evaluation of the ability to integrate social perceptual cues with contextual information that forms part of normal social encounters can also be clinically useful. One measure that can be used for such an assessment is the Emotion Evaluation Test[41], which forms part of the TASIT and assesses the ability to recognize emotions from dynamic, multimodal stimuli that are embedded into specific social scenarios. AD-related deficits detected by this measure are minimal or absent[77,84]. This finding might be explained by greater redundancy that results from multiple channels of information, helping to attenuate declines in the speed or efficiency of processing social perceptual cues in patients with AD.

As previously noted, an important consideration in the development of any treatment plan for impairments of social cognition is establishing the specificity and potential causes of the impairment. Impairments of perception, language, and executive function often co-occur

Box 4 | **Measures that assess social behavioural abnormalities**

**Frontal Systems Behaviour Scale[93]**
- Self-rated or informant-rated
- Assesses behaviour that is related to frontal lobe dysfunction
- Focuses on apathy, disinhibition and executive dysfunction

**Frontal Behavioural Inventory[96]**
- Informant-rated
- Assesses behaviour that is related to frontal lobe dysfunction
- Assesses behavioural symptoms that include aspontaneity, indifference or emotional flatness, inflexibility, disorganization, inattention, personal neglect, loss of insight, perseveration and stereotypy, inappropriateness, excessive jocularity, poor judgement, impulsivity and hypersexuality

**Socioemotional Dysfunction Scale[100]**
- Informant-rated
- Provides a global score of social competency
- Focuses on a range of social behaviours, including extraversion, warmth, social influence, insight, openness, appropriateness and maladjustment

**Peer-Report Social Functioning Scale[101]**
- Informant-rated
- Assesses socially appropriate and inappropriate behaviour, as well as the tendency to engage in stereotyping or prejudicial behaviour towards others

**Social Impairment Rating Scale[103]**
- Clinician-rated
- Assesses specific domains of social impairment
- Domains are: lack of attention or response to social cues, inappropriate trusting or approach behaviour, lack of adherence to social norms, difficulty with recognizing people, social withdrawal and socioemotional detachment

with impairments of social cognition and contribute to poor social functioning in many clinical groups, including patients with AD. In particular, AD-related deficits that are detected by measures of facial affect labelling (in which explicit choices must be made between different affective labels) are partially explained by difficulties with language[79] and executive control[5]. By contrast, difficulties with facial affect discrimination (which requires participants to decide whether two faces display the same or differing emotions) are predicted by face processing ability[5,79].

The use of similar tests to establish the specificity and cause of impairment is important for many clinical populations that present with social perceptual failures, including patients with common neurodegenerative disorders other than AD, such as Huntington disease[86], and demyelinating disorders, such as multiple sclerosis[87]. Social cognitive difficulties — including broad-based social perceptual failures — are also regarded as core impairments in traumatic brain injury[49], and are common in many psychiatric illnesses[88].

### Social behaviour: bvFTD
bvFTD is a chronic neurodegenerative disorder that is characterized by changes in personality and interpersonal conduct, loss of empathy, increased stereotypical behaviours, disinhibition, apathy and emotional dysregulation[89]. Sociopathic acts, including unsolicited sexual acts and physical assaults, are also common. These behavioural changes have been associated with progressive degeneration of the prefrontal and anterior temporal neocortex[90]. Individuals with bvFTD often exhibit deficits in executive function that can be detected with neuropsychological tests, but they often perform normally on other standard neurocognitive assessments.

Mood and behavioural disturbances are often the earliest presenting symptoms of bvFTD, so the condition is clinically under-recognized and often misdiagnosed. In the early stages and in young patients in particular, bvFTD is often mistaken for a psychiatric rather than neurodegenerative disease[91]. Misdiagnosis as AD or other types of dementia is also relatively common.

Simple tools that are designed to detect abnormal interpersonal behaviour often provide an effective way to distinguish bvFTD from other psychiatric and neurodegenerative disorders[92]. Patients' self-report data might be distorted owing to a lack of emotional insight, known as frontal anosodiaphoria, that is often present in this condition. As a consequence, informants such as a close confidant, a caregiver, or a spouse are widely regarded as the best source of clinical data in patients with bvFTD. A range of informant-rated measures are now available to gain insight into abnormalities of social behaviour (BOX 4).

The Frontal Systems Behaviour Scale (FrSBe)[93] was developed specifically to quantify the behavioural disturbances associated with frontoexecutive dysfunction. The scale provides a total score and separate scores for three behavioural domains: apathy, disinhibition and executive dysfunction. Scores obtained with the FrSBe are higher for patients with bvFTD than for patients with AD[94], and increase with greater prefrontal and temporal grey matter loss[95]. Similarly, the Frontal Behavioural Inventory (FBI)[96] quantifies changes in personality and behaviour that are associated with frontoexecutive dysfunction. Scores on the FBI can distinguish bvFTD from other dementias[97,98], and are sensitive to disease progression[99].

The FrSBe and FBI each include items that assess social behavioural symptoms, but also assess patients for a broader range of nonsocial behavioural disturbances, such as executive dysfunction and stereotyped movements. If a more focused and nuanced understanding of social impairment is required, the informant-rated Socioemotional Dysfunction Scale (SDS)[100] should be considered. The SDS focuses on interpersonal phenomena, such as social inappropriateness, social disengagement and personal warmth, and can differentiate between early-onset AD and bvFTD[100]. Another promising informant-rated measure that focuses on interpersonal function is the Social Inappropriateness Scale[101], which can identify increased levels of socially insensitive behaviour in people with dementia[102].

A clinician-rated measure, the Social Impairment Rating Scale (SIRS)[103], has been developed to systematically grade the severity of social behavioural symptoms across seven domains, including social withdrawal and inappropriate trusting or approach behaviour. In people with bvFTD, deficits in specific SIRS domains differentially relate to atrophy in distinct corticolimbic networks. Systematic observation of patients during everyday social activities can also provide valuable
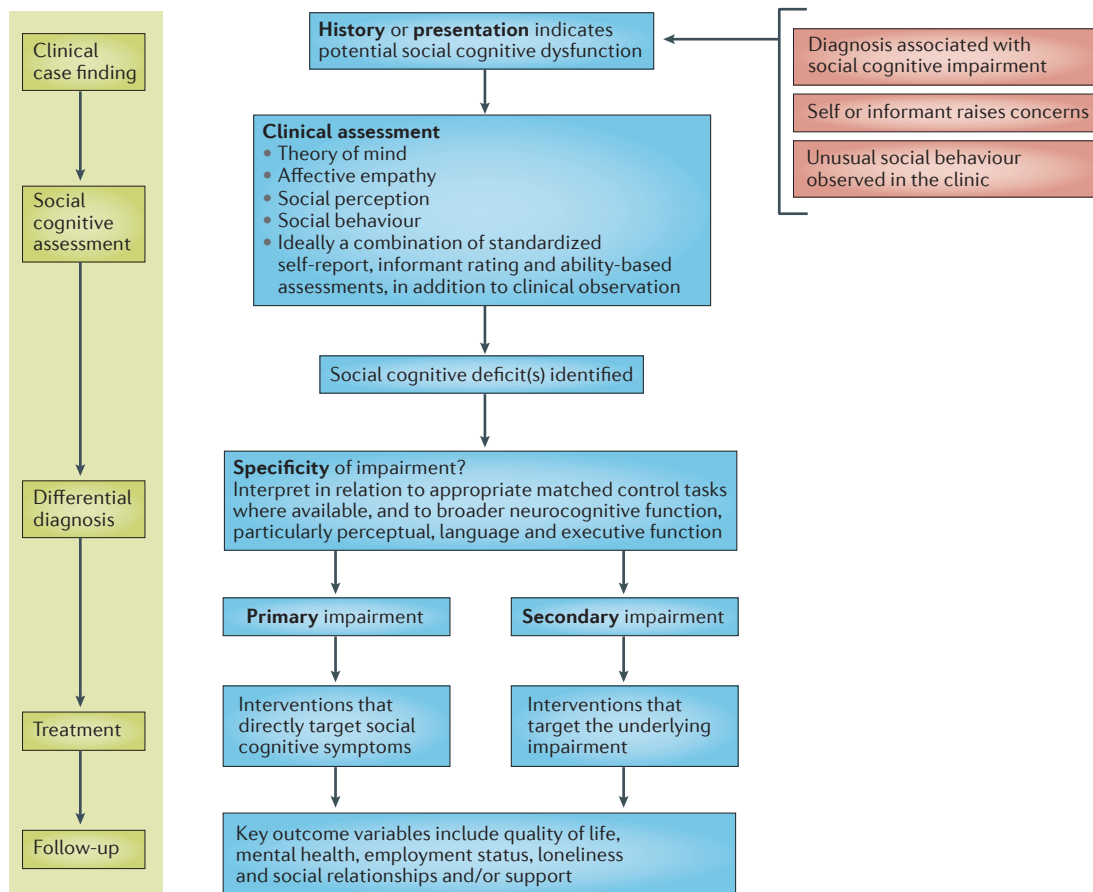
Figure 2 | **Algorithm for the evaluation and treatment of social cognitive impairments.** If patient history or clinical presentation indicates social cognitive dysfunction, each of the four domains should be assessed with at least one measure. Results of these assessments should be supplemented with formal clinical observation. If specific social cognitive deficits are identified, a more comprehensive assessment that focuses on the domain(s) in question should be conducted. Before recommendations for treatment can be made, establishing the specificity of any impairments, particularly whether the difficulties reflect a primary social cognitive deficit or a secondary consequence of broader neurocognitive impairment, is critical. Upon completion of treatment, follow-up should focus on community integration and mental health.

insight into social behaviour. For example, in one study, recorded segments of mealtimes revealed consistent differences between the behaviour of patients with different forms of dementia[104]. Patients with bvFTD used fewer phrases that contained the word 'you' than did caregivers or individuals with AD, and they also exhibited less tact and manners. Such data highlight the fact that clinical observation of a patient's spontaneous social behaviour can provide valuable insight into the level and nature of social impairment, even when high-quality informant reports are available.

Abnormal interpersonal behaviour is commonly seen in clinical practice, and it forms part of the core diagnostic criteria for many clinical disorders in addition to bvFTD, including schizophrenia, autism, Williams syndrome and social phobia[32]. Acute brain damage can also precede profound changes in social behaviour. For instance, people with traumatic brain injury often exhibit a range of behaviours that are difficult to deal with and cause distress and burden among family caregivers, thereby directly contributing to poor social relationships[105]. Given the critical role of family and friends in any

rehabilitation plan, treatment efforts should be directed towards managing such difficult behaviour, and ensuring the availability of appropriate education and support for caregivers.

## Clinical application and the future

Social cognitive deficits rarely occur in isolation, so all four domains should routinely be assessed in clinical practice when a patient presents with a neurological disorder and indications of social cognitive impairment (BOX 1). In the context of a broader neurocognitive assessment, such clinical data can be used to clarify the nature, magnitude and specificity of social cognitive impairment, with important implications for therapeutic decision-making. We present here a five-step algorithm for evaluation and treatment (FIG. 2) that includes details of how to approach the assessment of social cognition in clinical practice, starting with data gathering and proceeding through treatment to follow-up.

When social cognitive dysfunction is suspected, we recommend that at least one measure of each of the four domains is administered. Selection of these assessments

should be guided by their reliability, clinical validity and population norms (see Supplementary Tables S1–S4). Clinical validity is judged according to whether a measure has shown appropriate sensitivity and specificity, where these data are available, for disorders that are characterized by social cognitive dysfunction, with particular reference to autism and bvFTD. The variation in these aspects demonstrates the challenge in assessing social cognitive function. In particular, many measures have no formal, or only modest, population norms. The interpretation of clinical data depends on an appropriate match between the individual being assessed and the normative data with which their test performance is compared; a concerted effort is now needed to gather normative data for assessments for which such data are currently unavailable or limited. The availability of norms will become increasingly important as this field of research grows.

Social cognitive intervention is a relatively new area of research, but many promising inroads have already been made. Progress has included the development of targeted training programmes that have been associated with improvements in some functional domains[106] and with changes in the neural systems that support social cognitive processes[107]. Several available interventions focus on individual social cognitive skills, such as facial affect recognition[108]; a common strategy among such interventions is to direct a patient's attention to specific aspects of a facial expression, and to provide verbal descriptions of distinguishing perceptual characteristics. Other interventions target social behaviour and communication skills more broadly, often via role-play or social cognitive training batteries that encompass repeated practice of a range of social cognitive tasks[109]. Considerable interest also exists in the potential benefits of pharmacotherapy. Peripheral administration of exogenous oxytocin has already been shown to augment social cognitive skills training in schizophrenia[110], and might help people with other disorders, such as autism and bvFTD[31,111].

## Conclusion

For neurologists, assessment of social cognitive deficits in many disorders associated with brain dysfunction is now recognized to be as important as traditional neurocognitive assessment. Problems with memory or language might affect a patient's ability to work or live independently, but the negative impact of such disabilities on mental health and wellbeing can be ameliorated by strong social networks. Social cognitive deficits, however, impair the ability to form and sustain interpersonal relationships, thereby eliminating the benefits that social interactions have for patients with other neurocognitive impairments. Indeed, social isolation has long been known to be a major risk factor for morbidity and mortality[7,112]. A comprehensive assessment of social cognitive dysfunction in patients with acute brain trauma, as well as in patients with either a history or diagnosis that points to social cognitive dysfunction, should therefore be central in planning any neurorehabilitation effort. We have detailed the four key domains of social cognitive function that should be assessed in such patients, and some of the best validated assessment tools that can be used to meet the clinical needs of patients with such dysfunction. When used in combination with more standard neurocognitive assessments to inform treatment efforts, these measures have the potential to substantially enhance treatment decision-making and outcomes.

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**Review criteria**
For each social cognitive measure, psychometric properties, validity information and normative data were identified by searching PubMed, Scopus and Web of Science for articles published up to August 2015. Search terms were the names of each social cognitive task in conjunction with each of the following terms: "reliability", "validity", "psychometric", "norms" and "normative data".

**SUPPLEMENTARY INFORMATION**
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