

The social motivation theory of autism

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The idea that social motivation deficits play a central role in Autism Spectrum Disorders (ASD) has recently gained increased interest. This constitutes a shift in autism research, which has traditionally focused more intensely on cognitive impairments, such as theory-of-mind deficits or executive dysfunction, and has granted comparatively less attention to motivational factors. This review delineates the concept of social motivation and capitalizes on recent findings in several research areas to provide an integrated account of social motivation at the behavioral, biological and evolutionary levels. We conclude that ASD can be construed as an extreme case of diminished social motivation and, as such, provides a powerful model to understand humans' intrinsic drive to seek acceptance and avoid rejection.

Social motivation and social cognition: two competing accounts of autism

Over the past three decades, a number of theories have been put forward to account for the pervasive social impairments found in Autism Spectrum Disorders (ASD). Among the various attempts, the idea of a core deficit in social cognition (theory of mind, or ToM, in particular; see [Glossary](#)) has become one of the most prominent accounts of ASD. Concomitantly, the impact of motivational factors on the development of social skills and social cognition has received little attention. Recently, however, social motivation has emerged as a promising research domain at the intersection of social psychology, behavioral economics, social neuroscience and evolutionary biology. In this review, we integrate these diverse strands of research and defend the idea that social motivation is a powerful force guiding human behavior and that disruption of social motivational mechanisms may constitute a primary deficit in autism. In this framework, motivational deficits are thought to have downstream effects on the development of social cognition, and deficits in social cognition are therefore construed as a consequence, rather than a cause, of disrupted social interest.

Providing a complete account of social motivation requires both proximate and ultimate explanations. Proximate explanations pertain to how a behavior functions and ultimate explanations to why it was selected by evolution. At the proximal level, social motivation can be described as a

set of psychological dispositions and biological mechanisms biasing the individual to preferentially orient to the social world (social orienting), to seek and take pleasure in social interactions (social reward), and to work to foster and maintain social bonds (social maintaining). At the ultimate level, social motivation constitutes an evolutionary adaptation geared towards enhancing the individual's fitness in collaborative environments (see [Figure 1](#)).

We first present evidence supporting this integrated model of social motivation in healthy individuals, and proceed to review behavioral manifestations of diminished social orienting, social reward and social maintaining in ASD and the associated disruptions in the neural circuitry that typically underlie these behaviors. We then demonstrate that, as predicted by the evolutionary framework, some areas of social functioning are preserved in ASD. We conclude by arguing that deficits in social cognition are better explained within a social motivation framework, and acknowledge the limits of both socio-cognitive and social motivation theories in accounting for non-social deficits in ASD.

Glossary

Audience effect: refers to the influence of the presence of a spectator on a subject's performance or decisions. This classic effect in social psychology has received robust experimental support. Behavioral economists have demonstrated that the presence of others enhances participants' generosity in a range of games, such as the dictator game, the ultimatum game, and the public good game.

Overjustification effect: refers to the fact that extrinsic incentives, such as money, can undermine intrinsically motivated behaviors, such as altruistic behaviors.

Theory of mind (ToM): the capacity to attribute mental states to others and oneself in order to explain and predict behavior. ToM is an evolved psychological ability – most highly developed in humans – specialized in the rapid attribution of beliefs, intentions, desires or knowledge to others and oneself, and in the spontaneous understanding that others have mental states that may differ from one's own.

'Wanting' and 'liking': reward has two dissociable psychological components: a 'liking' component, which refers to the hedonic value of rewards; and a 'wanting' component, which refers to the incentive salience of the reward (i.e. an incentive motivation promoting approach seeking and consumption of the reward) [10]. Because of the paucity of objective behavioral markers of 'liking' in humans, 'liking' and 'wanting' are typically confounded in behavioral studies of reward (e.g. lip licking after the consumption of a sweet beverage is often used as a behavioral marker of 'liking' in the animal literature but this overt expression of pleasure fades out after infancy in humans). In this respect, neuroimaging is especially useful because it enables researchers to disentangle neural mechanisms that are associated with the anticipation of a reward cue and mechanisms that are associated with the consumption of that reward.

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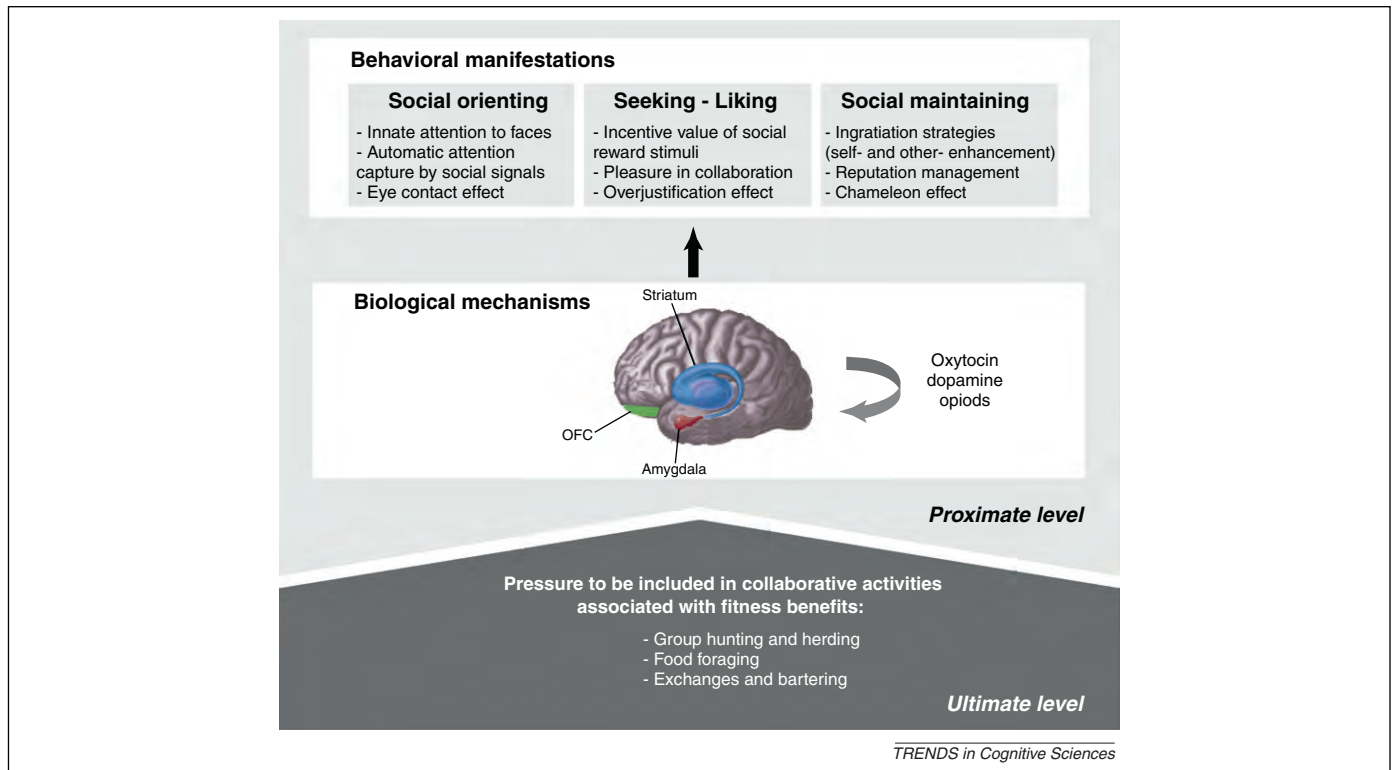


Figure 1. Social motivation constitutes an evolutionary adaptation geared to enhance the individual's suitability for collaborative environments (ultimate level). The orbitofrontal–striatal–amygdala circuit, influenced by specific neuropeptides, underlies a range of behaviors including social orienting, social seeking and liking, and social maintaining (proximate level).

An integrated model of social motivation in typical development

Behavioral level

Behavioral manifestations of humans' social interest are of at least three kinds: (i) objects with social importance are prioritized by attention; (ii) social interactions are rewarding; (iii) interpersonal behaviors are influenced by the desire to maintain and enhance relationships. We now review interdisciplinary evidence supportive of this three-tiered disposition.

Social orienting. In very much the same way that negative signals (e.g. threats) capture attention, potentially beneficial or rewarding information is prioritized. Given their relevance for humans, social signals are therefore granted attentional priority: attention is rapidly captured by human faces and bodies [1], changes in faces are detected better than in other objects [2,3], and masked faces are detected faster and more accurately than masked objects [4]. This preference is expressed early in life, with infants preferentially attending to face-like stimuli rather than to scrambled or inverted faces [5,6]. Highly relevant social signals, such as direct gaze, are particularly powerful in capturing attention both in adults and newborns; they facilitate face-related tasks, such as gender discrimination or encoding of identities [7]; and, when artificially suppressed from conscious perception, they become conscious faster than less salient social stimuli (such as inverted faces or averted gaze) [8,9].

Seeking and liking. Not only do people orient to the social world, they also find it rewarding. There are two components of reward – 'wanting' and 'liking' [10] – both of which apply to social signals. Behavioral economic studies

have shown that adults exert effort to obtain social rewards [11], which highlights their incentive value, and that players in economic games report taking pleasure in mutual cooperation [12]. Similarly, when given the choice to access a reward collaboratively or individually, toddlers strongly prefer collaboration [13]. Importantly, social interactions have intrinsic motivational value. As the 'overjustification effect' illustrates, people typically engage in prosocial behaviors not because they expect some kind of direct benefit to offset their efforts but because they find it inherently rewarding. Paying donors for giving blood, for example, actually decreases willingness to donate [14] and young toddlers are less prosocial after material incentives have been offered in exchange for a helping behavior [15]. Social psychologists have thus argued that the overjustification effect provides evidence that prosociality constitutes its own reward and is intrinsically motivated.

Social maintaining. Another important aspect of social motivation is individuals' desire to engage with others over sustained periods of time. Maintaining strategies, which encompass behaviors by which people establish, maintain, and enhance their relationships with others, are therefore key manifestations of social motivation: people try to be viewed as likeable rather than unlikeable, as competent rather than incompetent, as more rather than less physically attractive, etc. [16]. Concern for others' acceptance is mostly expressed through ingratiating behaviors, such as flattery, which elicit positive attitudes in the recipient and thereby enhance the reputation of the ingratiator [17]. These behaviors emerge early in development, with preschoolers spontaneously engaging in positive self-presentation, prosocial lies, and negative emotion concealment

for politeness purposes [18–20]. Maintaining behaviors, far from being cold-hearted manipulations, often occur outside the individual's conscious awareness. For instance, there is evidence that people unconsciously mimic others' nonverbal manners and that they do so because perceived similarity is an important predictor of likeability, which can be exploited to enhance integration [21]. Consistent with this idea, more empathic individuals [22] and people scoring high in measures of social motivation [23] exhibit stronger mimicry (Box 1).

Box 1. Social exclusion and isolation

The adverse effects of social isolation on well-being are a natural consequence of the strength of social motivation. Economists and social psychologists have long emphasized that social bonds are indispensable for achieving happiness and epidemiologists have confirmed that lack of social support constitutes a major health risk, comparable in magnitude to well-established risk factors such as smoking and alcohol consumption [93]. People who lack positive relationships are likely to experience a range of negative psychological states ranging from loneliness to depression [94]. Social isolation or rejection can lead to a psychological state that is similar to physical pain and activates similar brain circuits [95]. It is thought that this aversive social pain signal evolved by co-opting physical pain circuits to alert the excluded individual that her connections are weakening and to motivate her to repair them [94]. In line with this idea, the impact of social exclusion is manifest in every aspect of social motivation (orienting, seeking and liking, and maintaining). Chronic or induced loneliness enhances attention to social cues [96], sometimes to the extent of inventing humanlike agents (e.g. seeing faces in the clouds, or anthropomorphizing pets and objects; Figure 1) [97]; participants who have experienced social exclusion seek social interactions more and perceive others as more friendly [98]; and social exclusion leads to enhanced social maintaining, e.g. in the form of non-conscious mimicry [99].

Social motivation thus appears to function like other basic homeostatic systems: relative deprivation gives rise to negative feelings that signal to the individual that her needs are not met, and a sophisticated psychological machinery is then triggered in an attempt to restore balance in the system (by increasing orientating, seeking, and maintaining behaviors).



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Figure 1. Wilson, Tom Hank's anthropomorphized companion in the film 'Cast Away' (Twentieth Century Fox Film Corporation, 2000).

Biological level

Social motivation is subserved by a network of brain regions including the amygdala, the ventral striatum, and orbital and ventromedial regions of the prefrontal cortex. Each region plays a greater role in specific aspects of motivation, but no region operates in isolation. Subcortical structures are most involved in the generation of reward utilities, but require cortical involvement for conscious hedonic representations [10]. More specifically, the amygdala plays an important role in guiding attention to biologically relevant stimuli, such as social information conveyed by eyes, faces, or biological motion [24], and in calculating and updating social orienting value [25]. Computing the salience value of social stimuli rests on strong interactions with the ventral striatum and orbitofrontal cortex (OFC) with which the amygdala shares dense connections [26] and which both respond to socially reinforcing stimuli. The ventral striatum, on the one hand, plays a specific role in representing rewards as a 'decision utility' and in computing incentive salience and reward wanting for both non-social and social rewards (e.g. smiling faces [27], cooperation [28], or social approval [29]). Together with the OFC, it is also engaged when participants cooperate with a human partner versus a computer partner, even when monetary gain is identical [30]. Additionally, the OFC plays a key role in transforming reward information into a common currency of subjective hedonic value that then informs executive systems and guides goal-directed action [25].

Interestingly, functional differences in the orbitofrontal–striatum–amygdala network correlate with individual differences in social motivation: higher social orienting is associated with enhanced amygdala and OFC activity in response to emotionally relevant stimuli [31], whereas anti-social traits are associated with weaker activations in these areas in response to uncooperative outcomes [32]. Socially anxious adolescents also show greater amygdala activation when anticipating evaluation from undesired peers [33]; amygdala damage affects subtle social skills, such as people's sense of personal space [34] or their use of eye contact during conversations [35]; and OFC lesions disrupt emotion recognition and interpersonal maintaining behaviors [36].

Both human and animal research further suggests that these social motivational mechanisms are mediated, in part, by neuropeptide signaling. In particular, oxytocin (OXT), through interactions with dopamine, is thought to impact social orienting by modulating social salience and perceptual selectivity via the amygdala, and social reward via the nucleus accumbens [37]. In line with this idea, OXT-receptor knockout mice exhibit a range of social deficits including fewer vocalizations in response to social isolation and impaired social discrimination [38]. In addition to OXT signaling, endogenous opioid, cannabinoid, dopaminergic, glutamatergic, and cholinergic mechanisms are thought to play important roles in mediating social affiliative behaviors, including the rewarding aspects of social play [39,40].

Evolutionary level

That nature selected and conserved mechanisms for orienting, rewarding and maintaining social interactions indicates that these behaviors ultimately have important

fitness benefits for the individual. Indeed, collaborative activities, such as exchanging information or helping one another, allow access to a range of benefits that would remain inaccessible were it not possible to engage in social relationships with others [41]. While many non-human animals live in groups, humans are indeed exceptional in the variety of collaborative activities that they pursue and in the benefits these bring about. In traditional societies, for example, important volumes of foods are pooled and shared, thereby making up for high variance in foraging luck [42]. To take one example, Aché hunter-gatherers return with no game on approximately 40% of their hunts, and the Hadza on over 90% of their hunts. In such contexts, individuals rely on others' resources in times of need, and the value of cooperation far outweighs solitary alternatives [42]. Therefore, appearing as a good partner in the social group is, quite literally, vital.

In other apes, by contrast, food sharing either does not occur (i.e. food is foraged individually) or is not the result of a collaborative process (i.e. once the prey is killed, each hunter tries to secure as much meat as possible) [41]. In a study directly comparing chimpanzees and human children in their motivation to collaborate, it was recently found that, unlike chimpanzees, human children strongly prefer to engage in collaboration to forage food [13].

Importantly, under this specific evolutionary definition, the motivation for social affiliative interactions is distinct from other types of social motivations, such as those associated with sex, parenting, or dominance, that result from more ancient pressures and evolved into functionally and psychologically different systems [43]. Sexual arousal, for instance, is specifically geared to romantic relationships and is obviously inadequate to deal with family members; similarly, grossly uneven sharing may appear perfectly fine in a family context but be frowned upon among non-kins [43]. Thus, there are distinct motivations to deal with conspecifics and each of these can vary across individuals or be selectively impaired (see, e.g. [44] for hyposexuality; or [45] for disorders of mother-infant bonding). In what follows, we argue that ASD is characterized by a fairly specific disruption of motivation for social affiliation.

Social motivation in ASD

Behavioral level

Social motivation models of ASD posit that early-onset impairments in social attention set in motion developmental processes that ultimately deprive the child of adequate social learning experiences, and that the resulting imbalance in attending to social and non-social stimuli further disrupts social skill and social cognitive development [46–48]. As discussed in detail below, recent evidence demonstrates that social orienting, social seeking and liking, and social maintaining are all disrupted in ASD.

Social orienting. Core diagnostic criteria for ASD, as well as descriptions of the first year of life, include infrequent orienting to one's own name, diminished eye contact, and social aloofness [49]. In line with clinical descriptions, eye-tracking experiments have demonstrated impaired orienting to social stimuli: children with ASD look more at the background than at the characters while watching static social photographs (e.g. friends chatting) [50], and

adolescents and young adults freely viewing movie clips fixate less on people, faces and eyes than on other regions of interest [51,52]. Similarly, in the auditory modality, children with ASD do not exhibit a preference for socially salient sounds over non-social control noise [53,54], and display attention deficits for speech but not for non-speech sounds [55,56]. These differences in social attention are among the first manifestations of ASD [57], and preference for non-social patterns in toddlers has recently been identified as a robust predictor of ASD [58].

Seeking and liking. Half the adult population with ASD reports having no particular friends [59]. Yet, despite lower overall acceptance, greater loneliness is either not reported [60] or bears little relation to the individual's actual degree of social involvement [61]. More generally, individuals with ASD score lower on the friendship questionnaire (which tests constructs such as pleasure in close friendships or enjoyment in interaction for its own sake) [62]. Experimental evidence also suggests that the preference for collaborative activities is diminished in ASD. Tasks assessing spontaneous collaborative engagement (e.g. helping an adult who accidentally dropped an object or bouncing a ball with two people moving each end of a trampoline synchronously) indeed reveal that children with ASD are less likely to spontaneously help the experimenter [63] or to re-engage her when she interrupts the game. More generally, children with ASD lack declarative pointing [64], are impaired at initiating [65] and responding to others' bids for joint attention [66], and are less responsive to social rewards, such as verbal praise [67]. Self-reported pleasure in social and non-social situations also reveals selective social anhedonia in adolescents with ASD and a correlation between degree of social anhedonia and ASD severity [68].

Social maintaining. Compared to typically developing (TD) populations, individuals with ASD display fewer maintaining strategies and appear to place less emphasis on preserving their reputation and managing their self image. They are less likely to offer spontaneous gestures of greeting and farewell [69], and to adequately resort to maintaining strategies such as hiding affect [70], presenting themselves strategically to convince a specific audience [71], or displaying social laughter [72] and social emotions (e.g. embarrassment, or coyness) [73]. In a recent study testing reputation management more directly, the experimenter's presence had little influence on the way children with ASD rated the quality of the experimenter's drawing and this flattery index correlated negatively with levels of social anhedonia [74]. Similarly, a study on adults with ASD reported no 'audience effect' on charitable donations [75]. Anecdotally, these experimental findings echo reports of parents and caregivers who have long noted that individuals with ASD appear to be less influenced by considerations of impression management.

Biological level

The orbitofrontal–striatum–amygdala circuit has been repeatedly highlighted as abnormal in ASD [76], in particular in response to social stimuli such as faces [77], social approval [78], or social rejection [79]. One prominent hypothesis has been that social impairments result from a

deficit in representing the reward value of social stimuli [48]. However, only few neuroimaging studies have directly addressed the basis of social versus non-social reward processing in ASD and findings to date have not been entirely consistent (perhaps, in part, due to the lack of potent social reward paradigms) [78,80]. It therefore remains unclear whether aberrant reward processing in ASD is confined to social stimuli or reflects a more general deficit in stimulus-reward association (G. Kohls *et al.*, unpublished). Finally, neuroimaging studies have yet to examine whether both components of social reward, i.e. 'wanting' and 'liking', are equally affected.

Research on neuropeptide signaling and ASD, although still at early stages, suggests that disrupted oxytocin regulation might also play an important role in social reward dysfunctions in ASD [81] by impeding the accurate association of social stimuli with motivation values [37]. Consistent with this idea, associations between the OXT receptor gene and autism have been reported [82]. Furthermore, emerging animal models of ASD, with mutations in ASD-relevant neural cell adhesion molecules, have shown deficits in both the development of social affiliative behaviors and in glutamatergic synaptic structure in various brain regions, including circuits that may involve reward pathways [83,84].

Evolutionary level

Viewing the social motivation deficit in an evolutionary framework helps to explain the specificity of social affiliative

impairments in ASD and why other interpersonal dispositions such as attachment or sexual drive are spared. Indeed, despite their unarguably social nature, these latter dispositions result from different pressures and are therefore distinct from the motivation for social affiliation. Consistent with this idea, researchers have long noted that attachment to parents and offspring, and levels of sexual drive are spared in ASD. Children with ASD indeed show similar responses after separation from and reunion with their primary caregiver and have similar attachment styles compared to TD controls [85]. Similarly, interest in love and sexual relationships is spared in ASDs: autobiographies and parental journals indicate that people on the spectrum wish to develop intimate relationships and controlled surveys involving both parental and self-reports have confirmed that, although the social skills needed to approach potential partners may be impaired (i.e. courtship skills), the desire for romantic and sexual partnership is present [86,87]. An evolutionary framework thus helps account for why affiliative but not sexual/romantic or familial drives are impaired in ASD.

What is the scope of the social motivation theory?

Although many questions remain (Box 3), the research reviewed here suggests that the social motivation theory provides a credible framework to account for social impairments in ASD. However, by concentrating on social deficits, the social motivation account faces similar shortcomings as the ToM account. Unlike non-social accounts (e.g. execu-

Box 2. Boosting social motivation to enhance social cognition?

In the social motivation framework, impaired social cognition is seen as the consequence, rather than the cause, of impaired social attention. This predicts that boosting social attention in various ways (e.g. by providing explicit instructions to attend the social stimulus, increasing the relevance of the social stimulus to solve the task, or increasing the participant's intrinsic interest for the stimulus) should lead to enhanced social cognitive performance.

Instructions: Although high functioning adults with ASD do not spontaneously attribute mental states (as assessed in their looking times), they display control-like performances in verbally instructed versions of the FBT [89]. Similar results are observed with ironical utterances [100], speech sounds [101] and gaze following [102].

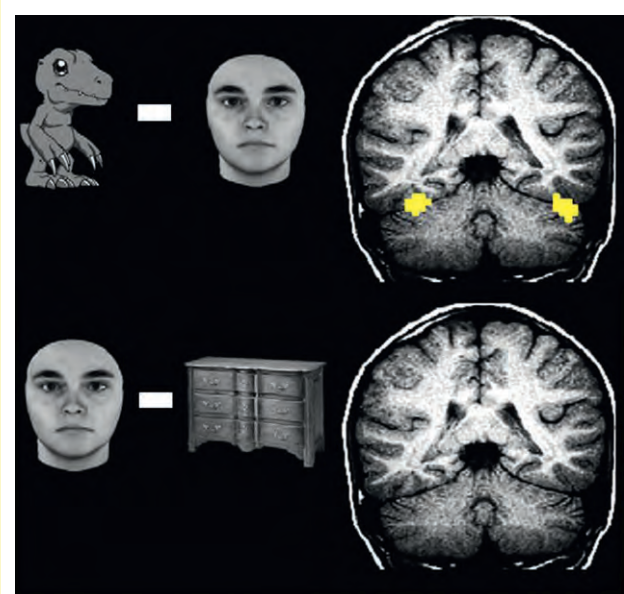
Relevance: There is robust evidence showing that gaze following is impaired in ASD. However, when gaze direction has a predictive value and is useful to solve the task, children with ASD do follow other people's gaze [103]. This suggests that, despite a spontaneous disinterest in mutual gaze, they are not blind to eye direction.

Interest: Young children with ASD are better at matching facial and vocal expressions of emotion when these are portrayed by familiar, compared to unfamiliar, adults [104]. Similarly, activity in the fusiform face area (FFA), which is often diminished in ASD [46], is enhanced when ASD participants are presented with familiar faces [105] or cartoon characters of specific interest to them (e.g. Digimon) [106] (Figure 1).

Taken together, these findings suggest that the underlying competence to process social stimuli may be more spared than previously thought and that atypical performance can, at least in part, be accounted for by differences in spontaneous attentional patterns. This hypothesis is further supported by evidence showing that controlling for social attention has an important impact on observed performances [107].

This has important implications for intervention and suggests that boosting social motivation and attention might be a powerful lever for social learning. The most effective interventions might therefore be

aimed at social motivation rather than at specific social skills. In this respect, OXT – which is known to enhance social salience [37] – can be seen as a promising therapeutic target and has indeed been found to increase performance in a range of social cognitive tasks [81].



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Figure 1. Activation of the FFA to Digimon (top panel), but not to faces (bottom panel) in patient DD. Right and left are reversed by radiological convention. Voxels are colored if the smoothed data have a $t \geq 4$ (which corresponds to $P < .0001$, uncorrected). Adapted from [106].

tive dysfunction or weak central coherence), both of these social theories indeed fall short of explaining non-social deficits in ASD, such as repetitive behaviors and restricted interests, as well as other important features of the disorder, such as its association with intellectual disabilities, comorbidities (e.g. anxiety, depression, attention-deficit/hyperactivity disorder – ADHD), or peaks of abilities (e.g. rote memory, systemizing, savant skills). Arguably, another challenge for social accounts is that both social cognition and social motivation deficits are not specific to ASD and can be found in other conditions (e.g. schizophrenia). It is important to note, however, that these shortcomings are only problematic if one considers that there ought to be a single explanation behind all the symptoms of ASD. On the contrary, if one agrees that ASD should be studied from a multiple-deficit perspective, it becomes more important to decide which of several competing theories provides the best account for a given set of deficits. In the case of social theories then, it is important to compare the explanatory power of social motivation vs. social cognition in accounting for social deficits.

The key difference between social motivation and social cognition accounts is one of causality. In the social motivation framework, diminished social interest is thought to deprive the developing child of social inputs and learning opportunities, which, ultimately, leads to diminished expertise in social cognition. In the ‘mindblindness’ framework, social impairments are explained by the fact that individuals who struggle to understand the intricate workings of the social world are likely to end up losing interest in social interactions. Positing that deficits in social cognition are a consequence, rather than a cause, of diminished social motivation yields a number of predictions.

First, social cognition deficits, which constitute a downstream consequence of diminished exposure to the social world, might only appear in a subgroup of individuals, whereas social motivation deficits, which are primary, ought to appear in all or nearly all individuals with ASD. In line with this, the general consensus has come to be that mentalizing deficits, although widespread in ASD, are by no means universal [88]. In particular, there is evidence that although some may fail to spontaneously use ToM, a significant proportion of children and adults with ASD do demonstrate an ability to represent others’ mental states in standard and advanced false belief tasks (FBTs) [89]. In this subpopulation of individuals with functional ToM skills, however, social motivation deficits remain (and in fact, constitute a fundamental part of the diagnosis). It is also important to highlight that there has been growing concern over the validity of FBTs, which served as a starting point for the ToM account and remain widely used as a test of ToM in autism. Research in developmental psychology has indeed revealed that very young infants succeed in age-appropriate versions of the FBT, hence indicating that failure in standard FBTs should not be taken as evidence for impaired ToM [90]. In the case of ASD then, it is equally unclear that failure to pass FBTs reflects lack of ToM.

Second, social motivation deficits should precede social cognition deficits in ontogeny. Following this second predic-

tion, disrupted social orienting has to date been evidenced much sooner than deficits in social cognition, with recent findings highlighting atypical social orienting from as early as 6 months in infants later diagnosed with ASD [57]. Arguably though, ToM in young ASD infants has yet to be assessed using adequate tools. Indeed, recent tasks designed to test ToM in young infants [90] have not been used in ASD and longitudinal studies assessing the development of social motivation and social cognition are still lacking.

Third, diminished social attention in development should be associated with diminished social cognition regardless of diagnoses (e.g. in non-ASD populations). The rationale is that diminished social motivation and attention starting in early childhood can ultimately deprive the child of crucial social inputs during what may be a sensitive period for the development of social cognition. Because the reward value of social interactions normally leads to a range of experiences that further allow social cognitive skills to develop, social exposure (mediated by social interest) should predict expertise in social cognition. In line with this idea, individual

Box 3. Outstanding questions

- ASDs are notably heterogeneous. Does the social motivation account apply to all subtypes of ASDs (e.g. ‘aloof’ vs. ‘passive’ or ‘active-but-odd’)? In particular, individuals in the ‘active-but-odd’ subtype appear to display genuine signs of social motivation. It is therefore important to further characterize subgroups of ASDs that do or do not have diminished social motivation.
- How can the social reward deficit be further characterized? Is the motivation deficit restricted to the social world or is there a more general stimulus-reward pairing deficit? Is social reward impaired at a general level or is the deficit circumscribed to one reward component only (e.g. ‘liking’ or ‘wanting’)? In addition to decreased prioritization of social signals, are non-social stimuli disproportionately prioritized in ASD?
- What is the role of co-morbidities, such as ADHD, depression or anxiety, which are known to have an impact on motivation and reward processing? In particular, does social anxiety (or social aversion) play a role in social motivation deficits in ASD?
- What is the role of social motivation in learning? Does reduced motivation necessarily lead to impaired social skill learning? Given that humans learn a lot in the context of social interactions, how much might a social motivation deficit impact learning of non-social skills? Are there ways to extrinsically enhance social motivation to boost learning?
- Are there developmental changes in social motivation in ASD, such that motivation to engage in social interactions increases spontaneously during adolescence and adulthood? If that is the case, how much do early deficits in social motivation have a long-lasting impact on social skills? Is there a critical period for the development of social skills and social cognition?
- What are the implications of such a theoretical framework for intervention strategies? It appears that intervention can have a positive effect on social attention behaviors and that this, in turn, can positively affect skills such as joint attention. Future research should determine how malleable social attention in childhood is and which intervention tools are most effective in boosting social attention.
- Answering these outstanding questions will require the development of adequate tools to measure social motivation. At the moment, most research uses indirect measures, such as social attention, and as such relies on approximations to the direct measurement of social motivation. Future research should therefore focus on designing tools that measure social motivation directly, from the youngest age.

differences in social attention in TD infants correlates with preschool ToM abilities [91] and extreme social deprivation has been linked to the development of quasi-autistic symptoms (see, e.g. the case of institutionalized Romanian infants [92]).

Finally, if diminished social motivation and attention cause social cognition deficits, boosting social attention ought to enhance performance in social cognition. This fourth point is associated with the richest set of empirical findings which, as we demonstrate in Box 2, converge to suggest that boosting social attention has a positive impact on social cognitive performance in ASD. This suggests that ASD involves a lack of spontaneous interest in mobilizing social cognitive skills for social purposes but that underlying cognitive skills may be more spared than previously thought.

Concluding remarks

The social world summons our attention like no other domain: social signals are prioritized by attention, interactions are intrinsically rewarding, and social maintaining permeates interpersonal behaviors. Social motivation is subserved by dedicated biological mechanisms and can be seen as an evolutionary adaptation to humans' highly collaborative environment: by enhancing attention to social information, by rewarding social interactions, and by promoting the desire to effectively maintain social bonds, social motivation smoothes relationships, promotes coordination and ultimately fosters collaboration. In ASD, by contrast, there appears to be an overall decrease in the attentional weight assigned to social information. Diminished social orienting, social reward and social maintaining are all found in autism and can account for a range of behaviors, including cascading effects on the development of mature social cognitive skills. These deficits appear to be rooted in biological disruptions of the orbitofrontal–striatal–amygdala circuitry, as well as in dysregulation of certain neuropeptides and neurotransmitters. ASD can thus be seen as an extreme case of early-onset diminished social motivation and provides a powerful model for understanding humans' intrinsic drive to seek acceptance and avoid rejection.

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