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# Social reward processing in individuals with autism spectrum disorder: A systematic review of the social motivation hypothesis

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ABSTRACT

*Background:* There is increasing empirical research examining the social motivation hypothesis, which posits that social deficits in autism spectrum disorder (ASD) are caused by underlying neural abnormalities in processing the reward value of social stimuli.
*Method:* The present systematic review examines social reward processing in individuals with ASD. This review focuses on the experimental procedures employed across studies and their potential impact on findings regarding the social motivation hypothesis. Twenty-seven studies met the specified inclusion criteria.
*Results:* Fifteen studies found evidence supporting the social motivation hypothesis; whereas, 12 studies found contradictory evidence. Most studies used pictures of faces as social stimuli and money as comparison nonsocial stimuli. Studies examining certain reward subtypes (e.g., reward learning) consistently supported the hypothesis; whereas, studies examining other reward subtypes (e.g., effort valuation) consistently did not support the hypothesis. Otherwise, methodological approaches varied considerably across studies.
*Conclusion:* The current empirical literature on the social motivation hypothesis is mixed, but findings suggest that examining specific sub-dimensions of reward processing may be important to clarify deficits in ASD. It is unclear whether inconsistency in findings is due to methodological limitations. As the literature stands, findings across studies support deficits in reward processing for both social and nonsocial rewards or individual differences in social reward processing. Interestingly, included articles cite few previously published studies on this topic, likely contributing to inconsistency in experimental designs and findings. Comparison across methodological approaches is warranted to help account for contrasting findings and determine the utility of the social motivation hypothesis given mixed evidence.

## 1. Introduction

The DSM-5 specifies that a diagnosis of Autism Spectrum Disorder (ASD) requires (a) persistent deficits in social communication and social interaction, (b) restricted, repetitive patterns of behavior, interests, or activities, (c) symptoms are present in early childhood, and that (d) symptoms limit and impair everyday functioning (American Psychiatric Association, 2013). Since autism was first described, it has been observed that "the outstanding 'pathognomonic,' fundamental disorder is children's *inability to relate themselves* in the ordinary way to people and situations from the beginning of life" (Kanner, 1943, pg. 242). Correspondingly, many researchers and theorists have focused on examining the underlying cause of social impairment in ASD.

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### 1.1. Theories of social impairment in ASD

Early behavioral researchers proposed that the social environment is not reinforcing for individuals with ASD, leading to social deficits (Ferster, 1961). Contemporary theories of social impairment in ASD focus on neurobiological and neuropsychological explanations. Simon Baron-Cohen (1995) proposed that "mindblindness," or a core deficit in theory of mind, causes social impairment. Specifically, individuals with ASD have difficulty understanding mental states, interfering with their ability to interact with others. Despite a large body of research, evidence is mixed (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012), resulting in the development of other more recent theories, such as the social motivation hypothesis.

Within the past decade, there has been increased attention on motivational factors that may impact social functioning in individuals with ASD, leading to the development of the social motivation hypothesis (Chevallier et al., 2012; Dawson & Bernier, 2007). Proponents suggest that individuals with ASD have "deficits in representing the reward value of social stimuli" (Abrams, Lynch, Cheng, & Phillips, 2013;). Deficits in social reward processing cause diminished social orienting, seeking and liking of social interactions, and social maintaining behaviors (Chevallier et al., 2012), ultimately manifesting as global deficits in social functioning. Although the social motivation hypothesis is consistent with early behavioral theories (i.e., social environment is not a reinforcer), it differs by focusing on neural or neuropsychological markers of reward processing rather than overt behavior.

Further, the social motivation hypothesis and mindblindness theory are similar in that they both: (1) are social accounts, (2) do not directly explain non-social deficits in ASD (e.g., repetitive behaviors, intellectual disability), and (3) do not explain social deficits in other disorders (e.g., schizophrenia; Chevallier et al., 2012). However, the two theories diverge in the causal direction of variables. Within the mindblindness framework, inherent deficits in social cognition (i.e., theory of mind) lead to reduced interest in social interactions. Within the social motivation framework, inherent decreased interest in social interactions leads to deficits in social cognition (Chevallier et al., 2012). Beyond accounting for atypical social behavior in individuals with ASD, this motivational framework may also explain why typically developing individuals engage in social behaviors (Mundy, 1995).

### 1.2. Other motivational explanations of impairment in ASD

Alternative explanations also exist within the motivational framework. First, researchers have observed that some individuals with ASD actively avoid social interaction (e.g., Freitag, 1970). Such observations led to the hypothesis that social stimuli act as unconditioned punishers rather than as neutral or unrewarding stimuli (Bowman, Hinkley, Barnes, & Lindsay, 2004; Tanaka & Sung, 2013). Second, individuals with ASD may experience nonsocial stimuli (i.e., stimuli without a social component, such as objects or pictures of objects) as disproportionately reinforcing relative to social stimuli (Cascio et al., 2012; Kim et al., 2014). Disproportionately high reward value of nonsocial stimuli may also account for restricted and repetitive interests. Third, a general dysfunction in reward processing may be present rather than specific dysfunction for social stimuli (Dichter et al., 2012; Kohls et al., 2014; Lin, Rangel, & Adolphs, 2012). Specifically, deficits in reward processing may exist for rewards in general (i.e., both social and nonsocial rewards) rather than solely social rewards. Fourth, individual differences in reward processing across behavioral presentations (e.g., aloof vs. active but odd; Kim et al., 2014) may exist such that some individuals with ASD have intact social reward processing and other individuals have varying levels of impairment in social reward processing. Unfortunately, few studies directly compare alternative explanations and an in-depth review of each explanation is beyond the scope of the present review.

### 1.3. Mechanisms of the social motivation hypothesis

In typically developing populations, research has demonstrated that social stimuli are processed as rewarding in the brain (Lin et al., 2012; Rademacher et al., 2010; Spreckelmeyer, Krach, & Kohls, 2009) and that social stimuli can improve task performance when used as a reward (e.g., Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). Researchers have proposed particular brain networks that potentially underlie social motivation, such as the "social brain" (e.g., Zilbovicius et al., 2006). Important neural regions within this network include the superior temporal sulcus (STS), fusiform gyrus (FFA), amygdala, prefrontal cortex including both the ventromedial prefrontal cortex (vmPFC) and the orbitofrontal cortex (OFC), and the ventral striatum (see Chevallier et al., 2012 and Neuhaus, Beauchaine, & Bernier, 2010 for more detailed reviews).

The STS is thought to process socially relevant sensory information; whereas, the FFA is selective for responding to human faces (Neuhaus et al., 2010). The remaining structures function collectively to facilitate motivational approach towards social stimuli (Chevallier et al., 2012; Neuhaus et al., 2010). Specifically, the amygdala directs attention to stimuli of interest and updates the value associated with a behavior (Chevallier et al., 2012; Neuhaus et al., 2010). The OFC and vmPFC are associated with determining the salience value of the stimuli and then assigning hedonic reward value to the stimuli (Chevallier et al., 2012; Neuhaus et al., 2010). The ventral striatum "represents rewards as a 'decision utility… and comput[es] incentive salience" (Chevallier et al., 2012, p. 233). Thus, these areas are primarily involved in experiencing motivation and reward. There has also been research on how neuropeptides, such as oxytocin, may interact with dopamine and impact salience value as well as attention towards stimuli (Chevallier et al., 2012; Dawson & Bernier, 2007; Neuhaus et al., 2010).

Research has demonstrated this network is important for appropriate social functioning in typical populations. Interestingly, all regions seem to be functionally, structurally, or both functionally and structurally, abnormal in individuals with ASD compared to typically developing individuals (Chevallier et al., 2012; Neuhaus et al., 2010). Within the social motivation framework, these neural abnormalities potentially underlie social impairments in individuals with ASD. Specifically, neural abnormalities in social reward processing in ASD are thought to reduce attention to social stimuli (due to decreased reward value), consequently decreasing