EDITORIAL

Motivational Impairments in Autism May Be Broader Than Previously Thought

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Social communication impairments have been a defining feature of autism spectrum disorder (ASD) since Leo Kanner's seminal description of 11 children with idiosyncratic social behaviors.[1] In the 75 years since Kanner's pivotal article, ASD research has focused on understanding and ameliorating the social communication and social cognitive impairments that are a defining feature of the disorder. Recently, however, the social motivation hypothesis of autism has been proposed as a novel account of the causal mechanisms of social impairments in ASD. This framework suggests that certain individuals with ASD are less motivated to interact with other people and derive less pleasure from social interactions. This account of ASD was initially proposed by Dawson and colleagues[2] in 2005 to describe impaired electrophysiological responses to faces and the potential developmental outcomes of such impairments on the emergence of face expertise in ASD. Chevalier et al[3] expanded the description of this framework in 2012 by considering the behavioral, neurobiological, and evolutionary features of the social motivation hypothesis of autism and suggested brain systems (orbitofrontal-striatal-amygdala neural circuitry) that are likely to account for social motivational impairments in ASD.

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The social motivation hypothesis of autism is not contradictory to more classic conceptualizations that social communication and social cognitive impairments in ASD derive from disruptions of brain circuits that process social information.[4] Rather, it suggests a complimentary biological mechanism that contributes to the development of social behavioral profiles of individuals with ASD. The social motivation hypothesis of autism is particularly appropriate for a neurodevelopmental disorder such as ASD because it highlights that functional disruptions in the development of the brain circuits that support social motivation may constitute a principal deficit in ASD that has downstream effects on the development of social cognition and social communication. In other words, because young children with ASD may be less motivated to interact with others, they may actively seek out social experiences to a lesser degree than neurotypical peers. This impoverished social environment would diminish opportunities for social experiences that are critical for the development of social cognitive skills. Undoubtedly, decreased social motivation is not the only mechanistic account of the full range of social deficits associated with ASD, given that some individuals with ASD have robust social interests and seek out social interactions. However, the social motivation hypothesis of autism has provided an explanation for seemingly perplexing findings showing that social functioning in ASD may improve under certain motivational contexts.[5]

The social motivation hypothesis of autism has spurred a budding field of research into neurobiological mechanisms that underpin social motivation in ASD, including a growing literature using functional magnetic resonance imaging to study neural responses to social rewards in individuals with ASD. However, most of the studies that comprise this literature have relatively modest sample sizes, and so, unsurprisingly, findings have been inconsistent.

In this issue of *JAMA Psychiatry*, Clements et al[6] used meta-analytic techniques to synthesize findings from 13 studies examining neural responses to social and nonsocial rewards in 259 individuals with ASD and 246 control participants. A crucial feature of this meta-analysis is that it includes studies that used both social and nonsocial reward stimuli to allow for insights about whether different reward-based responses in ASD are specific to responses to social rewards or rather may extend to other classes of rewards. There were 5 main findings: (1) across the 7 studies examining responses to social rewards (typically face stimuli), regions within brain reward circuitry were hypoactive (eg, the bilateral caudate and the anterior cingulate cortex) and hyperactive (eg, the right insula and the putamen); (2) across 10 studies examining responses to nonsocial rewards (typically monetary rewards), regions within brain reward circuitry were hypoactive (eg, the bilateral caudate, bilateral nucleus accumbens, anterior cingulate cortex, and the right insula), and hyperactive (eg, the left insula and the left caudate); (3) an exploratory analysis of 3 studies examining responses to restricted interest rewards revealed some hypoactive brain reward circuitry (eg, the left nucleus accumbens) but more widespread hyperactivation (eg, in the right caudate, left putamen, left insula, anterior cingulate gyrus, and right nucleus accumbens); (4) differences in neural responses during reward wanting and liking epochs for social and nonsocial rewards; and (5) exploratory metaregressions showed that the age of participants affected patterns of neural activation in the group with ASD.

There are a number of implications of this important meta-analysis. First, the consistent finding of functional disruptions in mesocorticolimbic circuitry activation in response to social rewards in ASD solidifies that this is an important avenue for continued research addressing the causal mechanisms and clinical significance of these disruptions. Second, because social motivation is critical for optimal social development and learning, it will be important to develop treat-

ments that address social motivation to improve clinical outcomes in individuals with ASD. When young children with ASD lack the motivation to participate in activities where social skills are typically forged, the resulting impoverished social environment detrimentally alters the emergence of social communication and social cognitive skills. Third, clinical trials of novel ASD treatments designed to affect motivational systems should consider the use of responses to nonsocial rewards as additional clinical endpoints. For example, Greene et al[7] recently reported that a single dose of intranasal oxytocin, a compound that has received interest as a therapeutic agent to improve social functioning in ASD, alters brain reward circuitry responses to nonsocial but not social rewards. This suggests the possibility that a therapeutic agent that has benefits for improving social communicative functioning, when taken over a period of weeks to months, may initially affect responses to nonsocial rewards. Fourth, reward processing is not a unitary construct, with factors such as the chronometry of responses (ie, reward wanting vs liking vs learning) and type of reward being crucial factors. Finally, and perhaps most importantly, this meta-analysis convincingly demonstrates that neural responses to nonsocial rewards are impaired in ASD, suggesting the need for the research community to appreciate that, despite the implications of the term *social motivation hypothesis of autism*, ASD is characterized not only by impaired motivational responses to social rewards, but also by atypical neural responses to a range of social and nonsocial motivational stimuli.

The number of studies available for inclusion in the meta-analysis by Clements et al[6] was relatively limited, and it will be important to continue to examine reward-based processes in ASD. Future research should examine responses to a range of rewarding stimuli to better understand the causal mechanisms of these impairments and to develop interventions that target these impairments. Moderating and mediating factors associated with neural mechanisms of reward processing, including neuroinflammation, psychotropic medications, and reward-based phenotypes, will also need to be explored. Finally, it will be crucial to unravel the developmental trajectories of reward-based responses in ASD using longitudinal studies to understand potential linkages between neural responses to rewards and the clinical presentation of ASD symptoms, as well as the prediction of responses to treatments that affect motivational brain systems.

ARTICLE INFORMATION

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