



A Literature Review on Studies Supported Social Motivation Theory Account of ASD

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Abstract. Social Motivation Theory has become a leading theory and model to explain patients with Autism Spectrum Disorders (ASD)'s behaviors and several studies using neuroimaging methods have provided biological and neural evidence to support the theory. Based on the existing literature research, this paper overviews the theoretical research on ASD social motivation. This literature review investigated some of the most recent studies related to and integrated the results and the findings. After research, the results show that most of the findings were in line with each other and showed the hypoactivation in ASD children's NAc.

Keywords: ASD · Social Motivation Theory · fMRI · NAc · Incentives

1 Introduction

According to the definition and diagnostic criteria proposed in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), common deficits and symptoms shown among Autism Spectrum Disorder (ASD) patients include “Deficits in social-emotional reciprocity,” “Deficits in nonverbal communicative behaviors used for social interaction,” and “Deficits in developing, maintaining, and understanding relationships” [1]. Over the past few decades, researchers have been trying to explain the deficits shown among ASD patients, and several accounts have emerged.

One of the leading theories was proposed by Chevallier et al. in 2012, referring to the framework of social motivation and the social motivation model. Chevallier et al. demonstrated the social motivation model on three levels-behavioral level, the biological level, and the evolutionary level [2]. Chevallier et al. argued that behaviorally, ASD patients showed disrupted social orienting, social seeking and liking, and social maintaining; biologically, based on Bachevalier & Loveland's work in 2006, the orbitofrontal–striatum–amygdala circuit showed extreme abnormality in ASD [3]. Evolutionarily, Chevallier et al. referred to the evolutionary framework that helped account for why affiliative drives were impaired while sexual/romantic or familial drives were spared in ASD. To test the social motivation model, several researchers applied imaging techniques like fMRI and EEG and provided biological evidence corresponding to the biological level of Chevallier et al.'s demonstration of the social motivation model account of ASD.

This literature review, compares and concludes several related studies in the field, strongly supports the Social Motivation Theory, and provides an organized way of understanding the neural activation of incentives in ASD children.

2 Scott-Van Zeeland et al., 2010's Work

Since little was known regarding the social reward processing mechanisms in ASD children, Scott-Van Zeeland et al. used functional magnetic resonance imaging technique (fMRI) to examine the neural activations and brain mechanisms when children with ASD were shown for monetary and social rewards. As compared with typically developing children, ASD children showed diminished neural responses to both monetary and social rewards and strong hypoactivation in frontostriatal areas.

In the study done by Scott-Van Zeeland et al., researchers designed two tasks based on the weather prediction task that was originally designed by Knowlton et al. in 1994 [4, 5]. Unlike the original stimuli used in Knowlton et al.'s 1994 work, Scott-Van Zeeland et al. used abstract images and asked subjects to categorize into "Group 1" and "Group 2" instead of images with geometric symbols and the groups of "Sunshine" and "Rainy". The tasks required the participants to classify pictures into "Group 1" and "Group 2" by pressing the buttons "1" and "2". The feedback can mainly be categorized into four versions—monetary reward, monetary neutral, social reward, and social neutral.

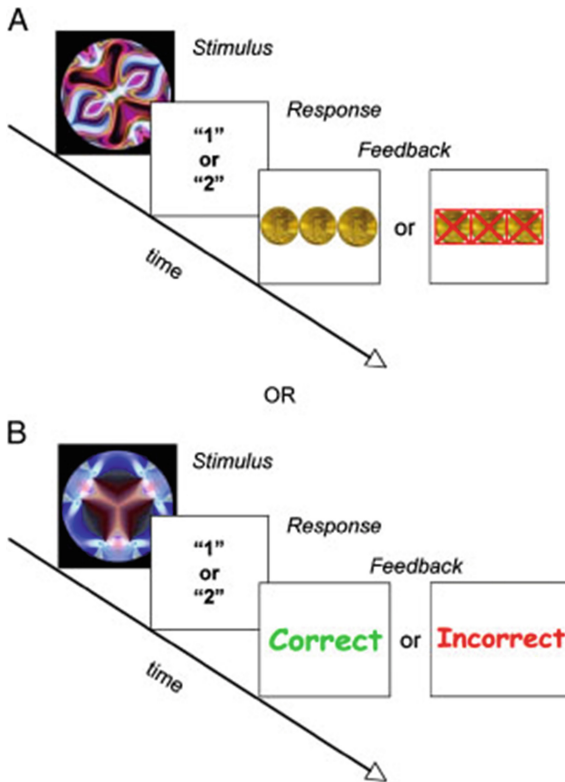


Fig. 1. Paradigm design from Scott-Van Zeeland et al., 2010's work [4] (Scott-Van Zeeland, Dapretto, M., Ghahremani, D. G., Poldrack, R. A., & Bookheimer, S. Y. Reward processing in autism. *Autism Research*, 3(2) (2010) pp.53–67, Licensed by the author)

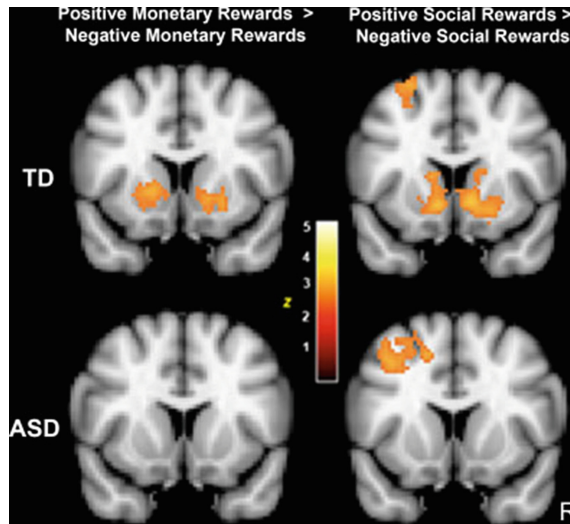


Fig. 2. Brain activation of reward response within groups for positive vs. negative reward feedback between ASD and TD children from Scott-Van Zeeland et al.'s work [4] (Scott-Van Zeeland, Dapretto, M., Ghahremani, D. G., Poldrack, R. A., & Bookheimer, S. Y. Reward processing in autism. *Autism Research*, 3(2) (2010) pp.53–67, Licensed by the author)

The reward would be given in the form of either a monetary version or a social version (image of three gold coins for correct feedback and the same image with three red “X” through the coins for monetary rewarded feedback; image of a smiling woman with the green text “That’s right!”). As for the neutral version, the words “Correct!” and “Incorrect” for monetary neutral feedback; and the image of the same woman with a sad face and the red text “That’s Wrong” for socially rewarded feedback (Fig. 1).

The fMRI results revealed a pronounced deficit in ventral striatal response to social rewards and a decreased frontostriatal activity during social reward learning among ASD children compared to typically developing children (Fig. 2).

3 Dichter et al., 2011’s Work

To better understand ASD children’s neural activations and neural responses when they anticipated possible rewards and received feedback, the fMRI technique was used by Dichter et al. to examine ASD children and controlled group (typically developing children). In a study performed by Dichter et al. in 2011, researchers modified the Monetary Incentives Delay Tasks (MID) that were originally designed by Knutson et al. in 2000 and redesigned the task so that the participants could complete “money runs” and “face runs”. Each trial in the task consisted of a 2,000 ms cue that indicates the possible results after responses—a triangle for “win” and/or a circle for “not win”; a crosshair fixation that lasts for 2,000 to 2,500 ms; a target bulls-eye that lasts for 500 ms and requires a quick button-press response; a feedback of whether the trial was a “win” or “not win” that lasts for 3,000 ms; and a variable length ITI crosshair [6]. If participants responded

quickly enough during money runs, they would win \$1, and they would be presented with a face image if they responded quickly enough during face runs (Fig. 3).

The fMRI results showed that during monetary anticipation, individuals with ASDs showed hypoactivation in the right NAc and decreased activation in the right OFC and the ACC. During face anticipation, individuals with ASDs showed greater activation in the bilateral amygdala and the left frontal pole but no remarkable differences in NAc or VMPFC activation as compared to the control groups (Fig. 4).

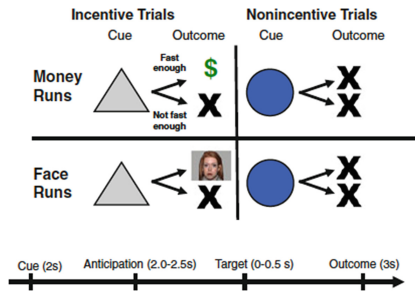


Fig. 3. Paradigm design from Dichter et al., 2011’s work [6] (Dichter, Richey, J. A., Rittenberg, A. M., Sabatino, A., & Bodfish, J. W. Reward Circuitry Function in Autism During Face Anticipation and Outcomes. *Journal of Autism and Developmental Disorders*, 42(2) (2011) pp.147–160, Licensed by the author)

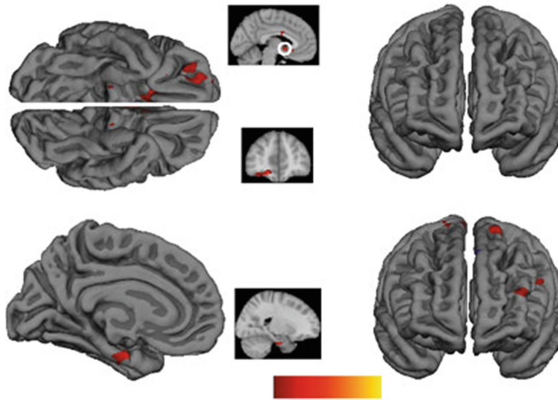


Fig. 4. Brain activation illustration from Dichter et al., 2011’s work [6] (Dichter, Richey, J. A., Rittenberg, A. M., Sabatino, A., & Bodfish, J. W. Reward Circuitry Function in Autism During Face Anticipation and Outcomes. *Journal of Autism and Developmental Disorders*, 42(2) (2011) pp.147–160, Licensed by the author)

4 Delmonte et al., 2012's Work

To examine whether the abnormality in reward processing regions existed among ASD children, Delmonte et al. applied the fMRI imaging technique to monitor ASD children's neural activations and responses when performing both monetary incentive delay tasks and social incentive delay tasks [7].

In the study done by Delmonte et al., researchers studied and compared social and monetary reward processing differences between ASD groups and control groups. Based on the previous studies [8, 9], researchers adapted and used monetary incentives delay tasks (MID) and social incentives delay tasks (SID) in both of which the participants were told to respond to a cue that had three forms (triangle for no reward, a circle with one horizontal line for a small reward, and a circle with two horizontal lines for a large reward), and receive feedback based on whether it was a no-reward trial, a small-reward trial, or a large-reward trial (Fig. 5).

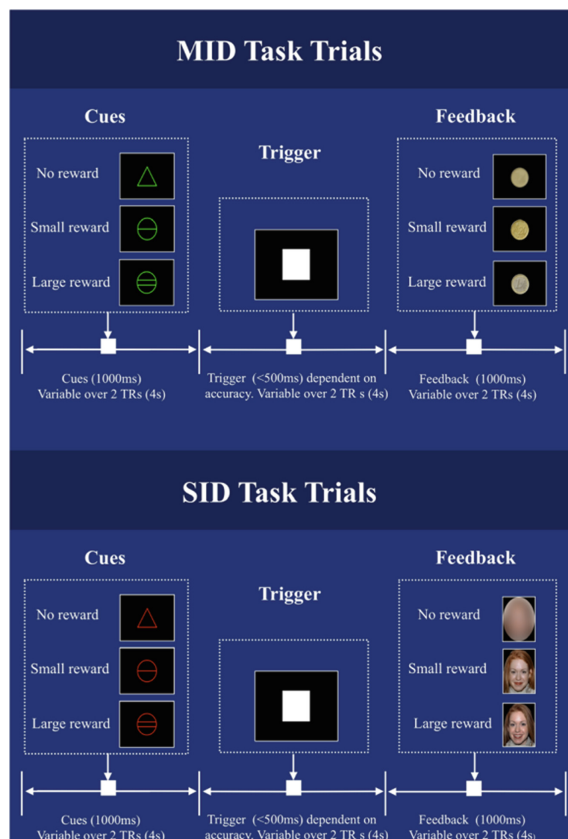


Fig. 5. Paradigm design from Delmonte et al., 2012's work [7] (Delmonte, Balsters, J. H., McGrath, J., Fitzgerald, J., Brennan, S., Fagan, A. J., & Gallagher, L. Social and monetary reward processing in autism spectrum disorders. *Molecular Autism*, 3(1) (2012) p7, Licensed by the author)

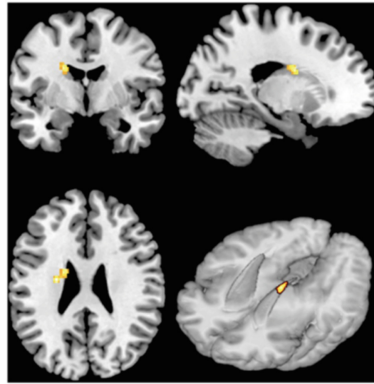


Fig. 6. Brain activation illustration from Delmonte et al., 2012's work [7] (Delmonte, Balsters, J. H., McGrath, J., Fitzgerald, J., Brennan, S., Fagan, A. J., & Gallagher, L. Social and monetary reward processing in autism spectrum disorders. *Molecular Autism*, 3(1) (2012) p7, Licensed by the author)

According to the results of functional magnetic resonance imaging and statistical analysis, the activation of social reward in the left dorsal caudate nucleus in the autistic group decreased, but not monetary reward. The results are consistent with the theory of social motivation, indicating that the characteristic of ASD is the abnormal response of the striatum to social reward (Fig. 6).

5 Dichter et al., 2012's Work

Similar to the scientific motivation in Dichter et al., 2011's work, Dichter et al. in 2012 also applied fMRI imaging to examine the neural activations among ASD children [10].

In a study performed by Dichter et al. in 2012, researchers designed an incentive delay paradigm while using fMRI to access BOLD activation in individuals. The researchers designed six functional imaging runs, three of which were money and three were objects, mainly including and involving machines, object motion, vehicles, buildings, computers, and physics. Turner Brown et al. demonstrated it in 2011 based on the confirmation of children with and without autism [11]. Like in Dichter et al.'s 2011 work, researchers made each trial in the task consist of a 2,000 ms cue that indicates the possible results after responses—a triangle for “win” and/or a circle for “not win”; a crosshair fixation that lasts for 2,000 to 2,500 ms; a target bulls-eye that lasts for 500 ms and requires a quick button-press response; a feedback of whether the trial was a “win” or “not win” that lasts for 3,000 ms; and a variable length ITI crosshair (Fig. 7).

fMRI results revealed that the reward area of the striatum in the autism group, including the left NAC and the right putamen, showed less activation for money stimulation. For object stimulation, the activation of dorsal anterior cingulate cortex rather than striatum decreased in the ASD group (Fig. 8).

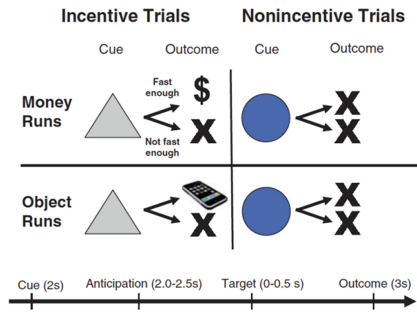


Fig. 7. Paradigm design from Dichter et al., 2012’s work [10] (Dichter, Felder, J. N., Green, S. R., Rittenberg, A. M., Sasson, N. J., & Bodfish, J. W. Reward circuitry function in autism spectrum disorders. *Social Cognitive and Affective Neuroscience*, 7(2), (2012) pp.160–172, Licensed by the author)

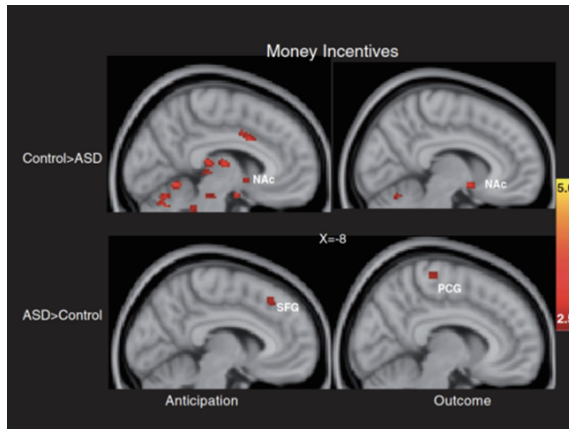


Fig. 8. Brain activation illustration from Dichter et al., 2012’s work [10] (Dichter, Felder, J. N., Green, S. R., Rittenberg, A. M., Sasson, N. J., & Bodfish, J. W. Reward circuitry function in autism spectrum disorders. *Social Cognitive and Affective Neuroscience*, 7(2), (2012) pp.160–172, Licensed by the author)

6 Kohl et al., 2018’s Work

In a study conducted in 2018, researchers designed and performed an adapted “incentive delayed task (IDT)”, the origin of which was based on the experiment designation of Knutson et al. in 2005 [12, 13]. Unlike the studies performed by Delmonte et al. in 2012 and by Dichter et al. in 2011 that used images as feedback, Kohl et al. designed the feedback to be “video clips” that depicted “under the condition of social reward, actors express facial expressions with other nonverbal gestures” and “personalized interests under the condition of reward” [14]. Trials that participants experienced could be divided into “social reward trials (SR)” and “interest reward trials (IR)”, as the former trials either depicted positive expressions when the target was hit or neutral expressions when the

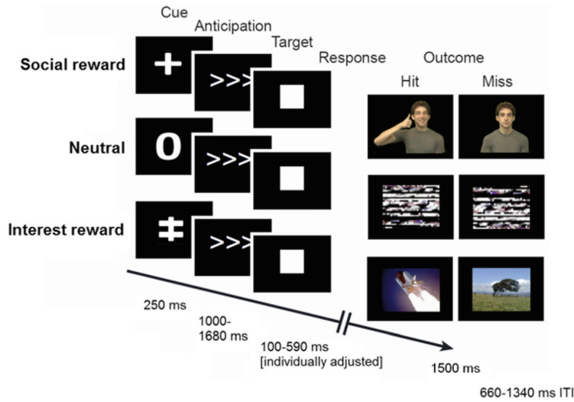


Fig. 9. Paradigm design from Kohls et al., 2018’s work [12] (Kohls, Antezana, L., Mosner, M. G., Schultz, R. T., & Yerys, B. E. Altered reward system reactivity for personalized circumscribed interests in autism. *Molecular Autism*, 9(1), (2018). p.9, Licensed by the author)

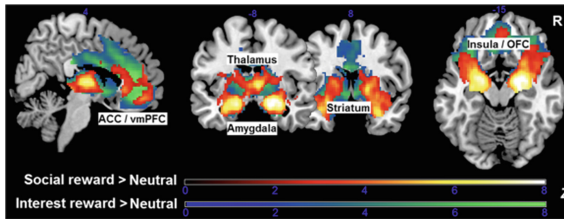


Fig. 10. Brain activation illustration from Kohls et al., 2018’s work [12] (Kohls, Antezana, L., Mosner, M. G., Schultz, R. T., & Yerys, B. E. Altered reward system reactivity for personalized circumscribed interests in autism. *Molecular Autism*, 9(1), (2018). p.9, Licensed by the author)

target was missed, while the latter trials either depicted the participant’s interest when the target was hit or “a tree that with slight natural movement” [14] (Fig. 9).

The fMRI results showed that “the caudate nucleus of autistic patients has abnormal response, and the brain responds more strongly to restrictive interest than social reward, which is also related to social disorder”, and concluded that “adolescents with autism respond more strongly to restrictive interest than social reward, mainly in non social fields (such as video games)” [14] (Fig. 10).

7 Supekar et al., 2018’s Work

Supekar et al.’s 2018 study divided participants into “primary cohort” and “replication cohort,” with data from the former obtained to investigate brain systems and neural mechanisms of social reward processing among ASD children and data from the latter obtained to examine the structural connectivity of large-scale brain networks in patients with autism [14]. By using High Angular Resolution Diffusion Weighted Imaging techniques (HARDI techniques- “a unique noninvasive technique capable of quantifying the

diffusion process of water molecules in living biological tissues like the human brain white matter [15]”) and fMRI, Supekar et al. reported that the density of the NAc-VTA tracts was significantly lower in children with ASD, indicating the aberration of ASD children’s mesolimbic reward pathways. Additionally, researchers have discovered a strong association between the low density of NAc-VTA tracts and the more severe social interaction impairments [“primary cohort: $r_s(22) = 0.50$, $P = 0.02$, Bayes factor = 3.57; replication cohort: $r_s(15) = 0.62$, $P = 0.01$, Bayes factor = 4.24”].

8 Discussion

Studies above provided evidence to support the biological level of Chevallier et al.’s social motivation model, and one of the findings in common was the diminished activation of NAc in ASD patients. However, there was still no consensus reached regarding the within-subject activation results when ASD subjects were asked to respond to monetary and social incentives. In addition, they failed to use negative reinforcement in the task, and failed to provide evidence to support the behavior and evolutionary level of Chevallier et al.’s social motivation model.

9 Conclusion

This review investigated several studies focusing on neural mechanisms of both social and monetary reinforcement in children with ASDs and argued that these studies have provided biological and neural evidence to support Chevallier et al.’s Social Motivation Theory. However, the review only focused on studies’ task designs and neural findings, lacking statistical analysis of the data. Furthermore, the studies cited only focused on positive reinforcement and positive incentives, leaving out negative incentives. In the future, reviews should include statistical analysis and articles studying negative incentives.

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