# Atypical Face Process in Autism Spectrum Disorder and Relevant Interventions

**Hongyue Li[1,a,*]**

[1]*College of Art and Science, New York University, New York, United States*
*a. hl3733@nyu.edu*
*\*corresponding author*

***Abstract:*** The current study overviewed previous research on abnormalities in face processing in individuals with autism spectrum disorder (ASD) and concluded implications on development of relevant social skill interventions. Overall, autistic individuals exhibited deviated neural activity patterns during face processing across different ages, suggesting that atypical development of neural structures might be accountable for deficits in social functioning. Similarly, familial studies found no significant variations in neural activity patterns for high-risk family members, providing insufficient evidence for the inheritance of autism. Although neural activity indices might not be effective as biomarkers for early life diagnosis, researchers could use these indicators to predict the extent of social impairment for patients in later life stages. Social functioning programs based on facial processing schemes had a significant training effect on young patients, but further testing trials are required to estimate their impact on patients' social behaviors in real life. This review can provide some guidance for the design of intervention programs at schools for children with ASD.

***Keywords:*** facial processing, neural imaging, Autism Spectrum Disorder.

## 1. Introduction

Autism was first described in 1943 by Leo Kanner. Plus, it was initially recognized by DSM-III in 1980. In DSM-IV, Autistic Disorder was categorized under the broad term of Pervasive Developmental Disorders (PDDs) along with four other disorders. DSM-5 removed the category of PDD and reintegrated all specified diagnoses into a broader category called autism spectrum disorder (ASD) [1]. According to DSM-5 definition, two key characteristics of autism included both reduced capabilities of social interaction and communication, and repetitive, restricted interests or behavioral patterns. Specifically, social deficits could be characterized by reduced social-emotional understandings, atypical usage of nonverbal behaviors during social situations, and difficulties in understanding, establishing, and maintaining relationships. The increasing prevalence of ASD gave rise to emerging research interests in finding biological basis for the disorder to improve current diagnostic approaches and develop effective treatments. Overall, the underlying mechanisms of this disorder were examined from biological and neurological perspectives.

Past studies had approached autism prognosis by exploring environmental, genetic, and epigenetic risk factors [1]. Genetic studies had identified multiple gene mutations that could contribute to the heritability of ASD, including Fragile X, SHANK3, Caspr2, CHRNA7, and TPH2.

Essential genetic processes, including Histone acetylation, DNA methylation, MECP2 mutations, Folate-methionine, and Chromosome remodeling could impact the expression of genes in patients. For environmental factors, researchers recognized that advanced parental age, prenatal exposure to valproate, and exposure to environmental pollutants were associated with the development of autism. Progresses in genetic research had wide implications on the development of novel therapeutic and diagnostic approaches, including animal models, such as the CNTNAP2, SHANK3 knockout mice, autoantibodies against folate receptor alpha and Caspr2, and dietary interventions based on the impact of folate-dependent pathways [1]. However, previous genetic research had limited explanatory power on the disorder, and future research was expected in estimating the effect of environmental and epigenetic factors in gene expressions. Moreover, genetic mutations in autistic individuals were found to be associated with other mental disorders such as ADHD and OCD, thus lacking the specificity for being used as predictive biomarkers for the disorder [2]. Recent studies on brain structures and neural activities reported early overgrowth in brain volumes within autistic toddlers, which diminished at age 6-8. Such enlargement had been attributed to the increase in cortical surface areas and was suspected to be related to alternated connection patterns of cortical white matters. Well-established studies on neural systems suggested that core regions associated with structural abnormalities including amygdala–hippocampal complex, frontotemporal and frontoparietal regions, anterior and posterior cingulate regions, cerebellum, and basal ganglia were mediating specific symptoms of ASD [2]. Like genetic biomarkers, neural biomarkers have limited generalizability, and further refinements were required for existing models to reach the level of clinical validity and specificity for application in clinical settings.

Face processing, the ability to identify emotions from human faces, is essential to social interactions. Such ability was highly associated with social capabilities and was consisted of several core capabilities, including visual processing ability (in general or for social stimuli), holistic facial processing strategies, and mentalization of emotions conveyed in faces. Researchers monitored individuals' neural activities during implicit facial processing tasks to study the cognitive mechanisms of social processing and to find atypicality in brain structures and neural activity patterns that can be used as biomarkers for diagnosis. It is assumed that autistic individuals have deficits in social emotional functioning which can lead to poor performances in face processing tests. In hypothesis, atypical functional connectivity patterns in patients compared to typically developing individuals found in neuroimaging studies might imply structural abnormalities in brain regions and deficits in social functions. From developmental perspective, longitudinal studies had revealed atypical developmental trajectories in brain structures for ASD patients. The current paper reviewed past studies on face processing of autistic patients and their family members and thus estimated potential directions for future studies in development of biomarkers and social interventions. This paper can provide some guidance to the design of social skill interventions for children with ASD at schools.

## 2. Face Processing Deficits in Children with ASD

### 2.1. The Underlying Neural Mechanism of Atypical Face Processing in ASD

Researchers had spotted atypical patterns of neural activities for autistic individuals during facial processing. Such abnormalities were usually specific to certain experimental conditions and were often discovered in terms of developmental trends across time. In one study, Magnetoencephalography (MEG) data was collected for 190 autistic and typical participants (age 6-39 years) during implicit emotional facial processing in eight regions of interest (ROIs) and on four frequency bands with different facial emotions (happy or angry). Controlling for age, no main effect of group differences on functional connectivity level across emotions were found, but the

interaction between age and neural connectivity strength were opposite in ASD and in Typical Development (TD) group. Moreover, the two groups also had opposite trends of age-group interaction for happy or angry faces: within the ASD group, for higher ages, higher strengths for network connectivity to happy faces and lower strength to angry faces were expected, and opposite trends were presented in TD group. The results suggested that alterations in neurophysiological measurements might reflect alternated neural developmental pathways in autistic patients [3].

A similar longitudinal study revealed that the impact of brain structure alterations might only be detectable after patients reached a certain age. Researchers analyzed magnetoencephalography (MEG) data in ASD and TD preadolescent individuals (age 7-13) during processing of a mixed series of stimuli and observed no abnormalities in measurements of long-range and local functional connectivity for autistic participants. In the precedent study, when identical procedures were applied to young adults (age 14-21), reduction in both the local functional connectivity of fusiform face areas (FFA) and the long-range functional connectivity between three cortical areas in higher orders and the FFA were detected. The combined data presented that, while the TD group exhibited positive correlations between age and the two functional connectivity patterns, the opposite trends were observed in the ASD group. Researchers also found that only within ASD group, local functional connectivity had significant interaction effect with age in the ASD group and was related to severity of symptoms in the young adult group. The observation of alternated developmental trends of functional connectivity for face processing implied abnormal developmental trajectories that emerged during the transition from children to young adults [4].

Finally, statistical analysis of neural activities suggested the atypical development patterns might have a quantitative rather than qualitative impact on social deficits of autistic children. In other words, the patients might have key capabilities intact, with the structurally altered processing system operating in reduced efficiency. One study explored the strategies applied by autistic adolescents for facial processing, using electroencephalography (EEG) and event-related potential (ERP) to process the behavioral indexes of autistic subjects and neurotypical controls when asked to recognize faces from Mooney stimuli. Given the low-information nature of stimuli, holistic face processing strategies were required for the detection of facial patterns. A quantitative difference in performance was observed between groups: autistic participants exhibited capabilities of face detection with lower detection rate, longer response times, slowed temporal processing for P1 latency, and prolonged temporal elevation in gamma-band activity. Researchers concluded that, instead of deficit in development of facial processing, autistic individuals possessed maturation in various aspects of the facial processing system. The structures of such systems might be quantitatively different from neurotypical individuals, resulting in reduced efficiency of facial processing [5].

## 2.2. Abnormal Face Processing in High-Risk Siblings of Children with ASD

Aside from autistic individuals, studies were also conducted in families of ASD patients to assess familial risk factors and to establish potential biomarkers for early diagnosis. One study examined ERP responses for 49 verbal autistic participants, 18 unaffected fathers (UF), 36 unaffected siblings (US), and 53 typical controls (UC) under separated EEG reference schemes. Overall, differences in facial processing between ASD and controls were only observed under a particular reference scheme. Reduction in face superiority over objects (marked by N170 latency differences between house and face stimuli) for ASD objects was only observed under average reference scheme. Similarly, the attenuated face inversion effect (marked by differences in peak values between inverted and upright faces in N170 and P1 amplitude) for ASD was recorded only in the vertex reference scheme. Given that ASD subjects also present reduced inversion effects for non-face stimuli, the attenuation could be attributed to diminished general visual processing function instead

of social deficits. Despite that the unaffected siblings show a limited similarity in variation of P170 non-specific inversion effect under vertex scheme, no similarities in neural patterns between ASD and their first-degree relatives. The result corroborated that variations in parameters were more likely the reflection of clinical characteristics of the disorder than the indication of shared genetic/familial backgrounds. Moreover, as the ERP biomarkers observed in the present findings were highly specific to reference schemes, future research should account for the impact of scheme differences in exploring characteristics for ASD [6].

Although no remarkable differences in neural correlates for autistic patients were observed, neural responses during social tasks might predict patients' social capabilities in later life. In another study on 12-months-old infants, researchers collected EEG data in 12-month-old infants with high-risk for ASD (determined by presence of autistic first or second-degree family members) and low-risk for ASD when presenting them with facial images of a stranger or of their mother. According to their ADOS results administered at 18, 24 and 36 months of age, infants were divided into three outcome groups: high-familial-risk infants without ASD (HRNoASD), high-familial-risk infants who develop ASD (HR-ASD), and low-familial-risk infants (LRC). As behavioral measures, infants were assessed using the Mullen Scales of Early Learning at 6, 9, 12, 18, 24, and 36-month visits. Overall, no statistical difference between outcome groups for the amplitude difference between familiar and unfamiliar social stimuli (Mother-Stranger effect) for each ERP components were recorded. In low-familial-risk infants, greater Mother-Stranger response in Nc predicted higher scores on Mullen Scales for expressive language skills. For infants with high-familial-risks who developed ASD group, larger mother versus stranger response in P400 could predict better communication skills at 12 months and reduced score in social affect at 18 months as measured by Autism Diagnostic Observation Schedule [7].

Consistent with previous studies, Shephard and colleagues' studies on siblings (instead of autistic patients) found no significant between-group differences in both neurophysiological and behavioral estimates and discovered significant association between infancy neural responses to social stimuli and mid-childhood autistic symptoms. Additionally, the researchers proposed that siblings of different genders might have different vulnerability to the negative impact of autistic risk factors. Through a longitudinal and cross-sectional study, the researchers investigated the association between behavioral measurements of face processing, neurophysiological correlates during face viewing, and development of autistic symptoms among siblings with high or low-familial-risk of ASD. To establish the frame of comparison, participants' engagements of visual attention with social stimuli in pop-up tests and neurophysiological indices during passive facial viewing tasks were recorded at age 7 months; in mid-childhood (age 7 years), participants' behavioral performances (measured by Response Time and accuracy) in face recognition tests and neural activity measurements were estimated again, along with their scores on autistic symptoms and Intelligence Quotient test. For mid-childhood face recognition, statistical analysis found no significant between-group differences in behavioral performances, although HR boys were shown to have slower RT for face and non-face stimuli than LR boys (effect not observed in girls). In the HR group, children with faster response time in facial recognition had more N170 lateralization in the right hemisphere, which is correlated with better scores in social communication abilities and less sensory symptoms. During infancy, children's N290 amplitude on passive viewing tests for non-face stimuli were related to lower N170 lateralization and poorer social communication skills in mid-childhood [8].

## 3.    Face Processing Interventions for ASD

With the knowledge in atypical developments of facial processing in autism, behavioral interventions were developed targeting improvements in social functions. The previous study

provided essential insight into the development of treatments regarding social functions through exploring the feasibility of computer-based systematic gaze modification training in 3-years-old autistic children. Gaze enhancement stimuli were generated based on the free-viewing data of TD children for social scheme videos: the areas TD children gazed upon were marked with higher brightness and resolution while other areas were intentionally darkened and blurred out. Autistic participants in the Cue group, who were shown the modified stimuli during the two training phases of experiment, exhibited increased proportion of time gazing at actresses' faces (Face%) after training and no decline in Face% during the experiment phases than the non-Cue group shown with original videos. Such effects were more salient in children with lower nonverbal capabilities. Overall, children after the training trials showed consistent improvements in social directed attention and mitigated diminishing attention, suggesting the potential for new therapeutic interventions to be generalized [9].

A more comprehensive study examined the effects of computer-generated social skill intervention in school-aged autistic children. The computer program named FaceSayTM included three games that separately target three key capabilities essential to social functioning: general attention and eye gaze attention, holistic facial recognition strategies, and the recognition and identification of emotional expressions. Compared to the control group who was assigned with a reading comprehension program, participants trained with FaceSayTM exhibited significant improvements in assessment scores for affect recognition, Theory of Mind, and social skills after training, accompanied by fewer autistic symptoms according to teacher reports. While these results indicated a promising aspect of the implementation of computer-based training programs, no significant between-group differences in both positive and negative social behaviors toward peers were observed. The generalizability of the effect of such intervention in real-life behaviors remained to be questioned [10].

## 4. Conclusions

Rather than specific abnormalities in neural activities, autistic individuals were marked by atypical developmental trajectories in brain structures. Given that no significant alterations in neural activities during face processing were observed for participants in their infancy or early to mid-childhood, the impact of structural abnormalities might not be estimable until the patient reached a certain age. The findings of previous studies suggested that early interventions before the onset age threshold of neural abnormalities might mitigate the effect of such developmental atypicality. Moreover, indices for neural responses might not be significant enough to be used as indicators for early life diagnosis of autism. However, for children diagnosed with autism, neural activity measurements might be applied for predicting later life development in social functions and determining the extent of support needed for the individual. Finally, as autistic individuals could have intact social functions with reduced efficiency or accuracy, the structural alterations observed in autism patients might be the cause of reduced efficiency or compensation for reduced capabilities. Future research is required for exploring the correlation between structural alterations and social capabilities.

In familial studies, similarities in neural activity parameters between siblings and autistic individuals were either limited to certain experimental conditions or not statistically significant. Neural studies provided no strong evidence for genetic background found in studies involving high-risk family members, suggesting that neural parameters are only attributable to development of specific symptoms. Similar association between neural activity indices and later life social functions were observed for high-risk siblings. The facial processing based intervention trials generated by computer programs yielded encouraging results on children in their early to mid-childhood. However, improvements in training scores might not directly translate into better social

performances in real-life. As past studies had also found mediating effects of gender and severity on social performances, specific interventions targeting patients with different genders and severity of disorder might be developed in the future.

In terms of the construction of future studies, abnormalities in neural activity patterns should still be estimated under multivariate designs and across age. Furthermore, comparisons should be made between patients of different disorders across age to uncover unique trends of development for autism and identify potential biomarkers for particular disorders. It should be noted that previous research mainly relied on the estimation of facial processing as assessment for social functioning. While atypical developments in facial processing might be attributed to reduced emotional reciprocity in autistic individuals, it might not be sufficient in explaining other aspects of deficit in social functions. Future studies are required for understanding the specific role of facial processing abnormalities in determining social abilities. Moreover, other than social deficits, there was little research targeting the neurological mechanisms underlying the emergence of repetitive interests in autism patients, which is another definitive characteristic of the disorder. From an application perspective, previous interventions on autistic disorders involving facial processing solely focus on the early developmental stage. Intervention methodologies should be developed for children and adolescents separately. This review can provide some guidance to the social skill curriculum for children with ASD at schools.

## References

[1] Waye, M.M.Y. and Cheng, H.Y. (2018). Genetics and epigenetics of autism: A review. Psychiatry and Clinical Neurosciences, 72(4), 228-244.

[2] Ecker, C., Bookheimer, S.Y. and Murphy, D.G.M. (2015). Neuroimaging in autism spectrum disorder: Brain structure and function across the lifespan. The Lancet Neurology, 14(11), 1121-1134.

[3] Safar, K., Vandewouw, M.M. and Taylor, M.J. (2021). Atypical development of emotional face processing networks in autism spectrum disorder from childhood through to adulthood. Developmental Cognitive Neuroscience, 51, 101003.

[4] Mamashli, F., Khan, S., Bharadwaj, H., Losh, A., Pawlyszyn, S.M., Hämäläinen, M.S. and Kenet, T. (2018). Maturational trajectories of local and long‐range functional connectivity in autism during face processing. Human brain mapping, 39(10), 4094-4104.

[5] Naumann, S., Senftleben, U., Santhosh, M., McPartland, J. and Webb, S.J. (2018). Neurophysiological correlates of holistic face processing in adolescents with and without autism spectrum disorder. Journal of Neurodevelopmental Disorders, 10(1), 27.

[6] Sysoeva, O.V., Constantino, J.N. and Anokhin, A.P. (2018). Event-related potential (ERP) correlates of face processing in verbal children with autism spectrum disorders (ASD) and their first-degree relatives: A family study. Molecular autism, 9(1), 1-16.

[7] Glauser, J., Wilkinson, C.L., Gabard-Durnam, L.J., Choi, B., Tager-Flusberg, H. and Nelson, C.A. (2022). Neural correlates of face processing associated with development of social communication in 12-month infants with familial risk of autism spectrum disorder. Journal of neurodevelopmental disorders, 14(1), 1-14.

[8] Shephard, E., Milosavljevic, B., Mason, L., Elsabbagh, M., Tye, C., Gliga, T., Jones, E.J.H., Charman, T. and Johnson, M.H. (2020). Neural and behavioural indices of face processing in siblings of children with autism spectrum disorder (ASD): A longitudinal study from infancy to mid-childhood. cortex, 127, 162-179.

[9] Wang, Q., Wall, C.A., Barney, E.C., Bradshaw, J.L., Macari, S.L., Chawarska, K. and Shic, F. (2020). Promoting social attention in 3‐year‐olds with ASD through gaze‐contingent eye tracking. Autism Research, 13(1), 61-73.

[10] Rice, L.M., Wall, C.A., Fogel, A. and Shic, F. (2015). Computer-assisted face processing instruction improves emotion recognition, mentalizing, and social skills in students with ASD. Journal of Autism and Developmental Disorders, 45(7), 2176-2186.