

Highlights on Aberrant Face Processing in Autism Spectrum Disorder and Its Percussions on Management of Autistics

Eman Ahmed Zaky¹

Department of Pediatrics, Child Psychiatry Unit, Faculty of Medicine, Ain Shams University, Cairo, Egypt

Corresponding author: Eman Ahmed Zaky, Department of Pediatrics, Professor of Pediatrics and Head of Child Psychiatry Unit, Faculty of Medicine, Ain Shams University, Cairo, Egypt, Tel: 002021062978734; E-mail: emanzaky@med.asu.edu.eg

Received date: July 25, 2017, **Accepted date:** July 29, 2017, **Published date:** August 09, 2017

Copyright: © 2017 Zaky EA. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Highlighting the neurological basis of normal face processing and its abnormalities in ASD seems crucial because of its percussions on symptomatology and the management plan of autistic children. Human face processing that has been proven to be compromised in many autistic individuals is pivotal for proper social interactions. Such spontaneous perceptual task in normal children is carried out by face processing areas of the brain as fusiform gyrus, superior temporal sulcus, and amygdala. Behavioral, electrophysiological, and neuroimaging studies showed evidences of dysfunction of such areas in many autistics who often focus on face periphery and cannot interpret that it tells something about a person's state of mind. Very early targeted intervention can stimulate face processing areas of the brain during the early developmental phases of social brain circuitry which in turn will help autistics to pay attention to faces and learn how to understand emotional expressions. Eventually, prevention or at least significant amelioration of both the spectrum and severity of autistic symptomatology might be possible.

Keywords: Autism spectrum disorder (ASD); Face processing; Fusiform gyrus; Superior temporal sulcus; Amygdala; Functional magnetic resonance imaging

Introduction

Autism spectrum disorder (ASD) is a lifelong neurodevelopmental disorder that is characterized by impaired social and communicative abilities as well as restricted, repetitive, stereotyped pattern of behaviors, interests, and activities. Significant difficulties in social interactions in autistics are manifested mainly by impairment in eye to eye contact, social reciprocity, and response to emotional cues [1-3].

Face perception is an individual's understanding and interpretation of the face, especially the human face, in conjunction with the related information processing in the brain. In general, it is very important in individual's social interaction but it is a complex perceptual function with extensive involvement of different areas in the brain which when damaged can lead to specific impediment in understanding and interpretation of faces; i.e. prosopagnosia [4].

Highlighting the neurological basis of normal face processing and its abnormalities in ASD seems crucial as aberrant face processing has been claimed to be an important neuro-psychopathological mechanism behind social impairment in autistics with subsequent vital percussions on their management plan [5].

Face processing in normal individuals

Naturally, humans have the ability to read others' facial expressions with figuring out the feelings they convey and the state of mind they reflect. Face processing in humans is a cornerstone of most social interactions and orientation to others' people eyes is an innate social programming in humans [6-8].

Neural systems responsible for face processing are present early in life; accordingly its impairment reflects an early dysfunction of such early developed brain circuits. Normal neonates show visual preference for faces and fast face recognition [9-11]. By the age of 6 months, typically developing infants exhibit specific brain responses, which have been documented by event related potentials (ERPs), to different facial expressions such as familiar versus unfamiliar faces or fearful versus unafraid or neutral faces. Such early face processing abilities are vital for interpretation of emotional expressions and sharing attention and interests with others [12-16].

Furthermore, in typically developing infants and children, Positron Emission Tomography (PET) and Functional Magnetic Resonance Imaging (fMRI) revealed significant activation of the right fusiform gyrus of the occipitotemporal cortex (also known as occipitotemporal gyrus) during perception of upright faces compared to non-facial stimuli, inverted or scrambled faces [5,17-19]. Meanwhile, superior temporal sulcus (STS) has been found to be involved in interpretation of facial movements (eyes and mouth) and understanding the meaning of stories and cartoons involving humans, causality, intentionality, and self-perspective [3,19,20]. On the other hand, amygdala; a set of sub-cortical nuclei and a component of the limbic system, has been found to be engaged in understanding familiar faces or those conveying emotional contents. Amygdala is important in both perceiving others and having oneself emotional behaviors and feelings like anger and fear [21,22].

Social motivation hypothesis in ASD

Behavioral, electrophysiological, and neuroimaging studies showed evidences of dysfunction of face processing areas in autistics who often focus on face periphery and cannot interpret that it tells something about a person's state of mind [23-25]. Human face processing that was proved to be compromised in many autistic individuals is crucial for proper social interactions. On the other hand, early experience plays a

crucial role for the normal development of many perceptual and cognitive functions including face perception. Accordingly, aberrant face processing may act as a cause (innate dysfunction of face processing areas of the brain) as well as a consequence of reduced

social interest in autistics. Such concept can add to the understanding of the background of one of the main diagnostic features of ASD and in addition, it proposes vital percussions on its management (Figure 1) [26-29].

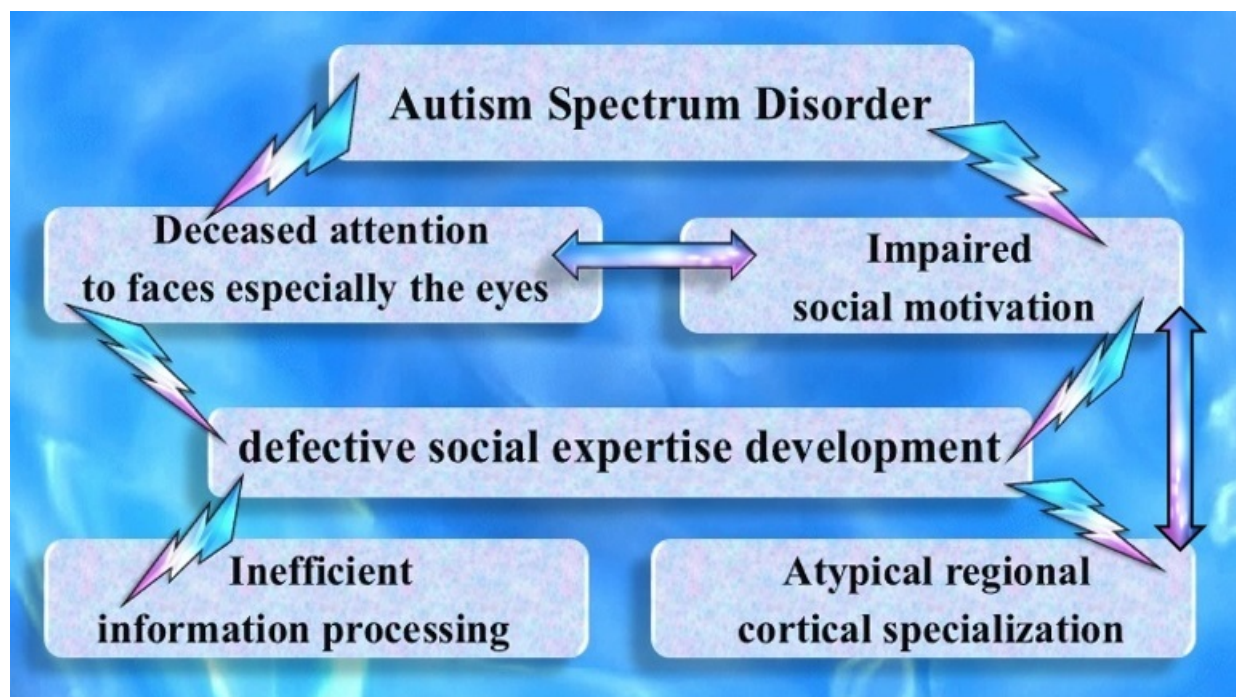


Figure 1: Diagrammatic illustration of social motivation hypothesis of Autism Spectrum Disorder (ASD).

Face processing in autistics

Autistics were found to have significant impairment of social interaction manifested mainly by poor eye to eye contact, impaired social reciprocity, and impediment in response to emotional cues [1-3]. Early failure to attend to others' people faces and speech has been documented in retrospective studies of home videotapes of first birthday parties of infants who were later diagnosed as autistics [30]. By the age of 2 to 3 years autistic children show a wide range of social dysfunction manifested by impediments in joint attention, imitation, and emotional reactions [1,24,25,31].

Face processing which is a spontaneous perceptual task seems to be significantly difficult in autistics who spend remarkably short times engaged in face to face eye contact or social interactions in general. Such impairment in face processing in autistics represents a pivotal ring in a chain of general dysfunction in the complicated social circuitry of the brain as individuals with ASD need to learn emotional expressions and cues that others understand and imitate spontaneously [24,25,32].

Interestingly, elementary school age autistics have shown worse performance on tests of face discrimination and recognition as well as emotional perception and recognition compared to their mental and chronological age matched counterparts. From the age 4 years to adulthood, an event related potential component (N170) is activated preferentially to faces in typically developing individuals while autistics as young as 3 years of age have been found to show atypical ERPs to faces and facial expressions but not to objects. For instance, when an

autistic scans unfamiliar faces, the fusiform gyrus fails to activate but rather object processing areas activate or eccentric patterns of activation are detected which are variable from one autistic to another [33-36]. In addition, neuroimaging studies indicated that the autistic brains fail to recruit face processing areas of the brain (e.g. fusiform gyrus, STS, and amygdala) with remarkably reduced activity down to none [33-36] but structurally only the mean volume of amygdala was found to be lower compared to controls [37].

Genetics of face processing

Lawrence et al. [38] reported impaired facial recognition abilities in Turner syndrome due to impaired amygdala functioning. Recently, apart from genetic syndromes, facial recognition abilities have been proven to be heritable in twin studies with independent genetic basis from other cognitive abilities [39,40].

Evidence for face processing impairment among first degree relatives of autistics

Many studies have demonstrated higher than normal prevalence of autism related functional neurological impairment (dysfunction) among relatives of autistics (parents, siblings). Determination of autism genetically related quantitative traits (endophenotypes) is a methodology of discovery of its susceptibility genes; face processing impairment is one of these traits. Such discovery might lead to the identification of neonates at high risk to develop autism [41,42]. Implementation of very early targeted intervention for those neonates

could provide a very effective stimulation during the period of early developing social brain circuitry. Activities which are suitable for the developmental stage of infants at risk are highly advisable to be planned with doing all efforts to engage them in. Such strategy could prevent or at least ameliorate the spectrum and severity of autistic symptomatology [14,43].

Impact of social motivation hypothesis on management of ASD

Social motivation hypothesis paved the way for the policy of implementation of early targeted intervention for autistic infants to prevent or at least ameliorate face processing impairment (face recognition, discrimination, and interpretation of emotional cues). Early intervention techniques depend on directing the child's attention to faces and speech during different everyday activities (e.g. biting, combing, coloring, drinking, clapping, hugging, smiling, laughing, and crying) and positively reinforcing them for doing so. Accordingly, social interactions become meaningful and rewarding to the child. Such very early targeted intervention can stimulate face processing areas of the brain during the early developmental phases of social brain circuitry which in turn will help autistics to pay attention to faces and learn how to understand emotional expressions [26-28,43,44]. Although ASD is still a mysterious disorder in many aspects, it is worthy to do our best to give the autistics the chance to see our world and to be engaged in. Believing that whenever there is help, there is hope can make a huge difference for autistics and their caregivers.

Conclusion

Significant difficulties in social interactions in autistics are manifested mainly by impairment in eye to eye contact, social reciprocity, and response to emotional cues. Highlighting the neurological basis of normal face processing and its abnormalities in ASD seems crucial as aberrant face processing has been claimed to be an important neuro-psychopathological mechanism behind social impairment in autistics with subsequent vital percussions on their management plan. Human face processing; a spontaneous perceptual task in normal children, is carried out by many face processing areas of the brain such as fusiform gyrus, superior temporal sulcus, and amygdala. Behavioral, electrophysiological, and neuroimaging studies showed evidences of dysfunction of such areas in autistics who often focus on face periphery and cannot interpret that it tells something about a person's state of mind. Very early targeted intervention can stimulate face processing areas of the brain during the early developmental phases of social brain circuitry which in turn will help autistics to pay attention to faces and learn how to understand emotional expressions. Eventually, prevention or at least significant amelioration of both the spectrum and severity of autistic symptomatology might be possible.

References

1. Gillberg C (2009) Autism and autistic-like conditions. In: Aicardi (ed.) *Diseases of the Nervous System in Childhood*, Mackeith Press, London, UK, pp: 902-921.
2. DSM 5 (2013) American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 5th version, Arlington, USA.

3. Zaky EA (2016) When hopes and great expectations have gone with the wind!!!!!! Living with an autistic child, is it a tragedy or a blessing? None can tell but the expert. *J Child Adolesc Behav* 4: 108.
4. Barton JJS, Press DZ, Keenan JP, O'Connor M (2002) Lesions of the fusiform area impair perception of facial configuration in prosopagnosia. *Neurology* 58: 71-78.
5. Marcus DJ, Nelson CA (2001) Neural basis and development of face processing in autism. *CNS Spectrums* 6: 36-59.
6. Brothers L (2000) The social brain: A project for integrating primate behavior and neurophysiology in a new domain. *Concepts in Neuroscience* 1: 27-51.
7. Baldwin DA, Moses LJ (1996) The ontogeny of social information gathering. *Child Development*. 67: 1915-1939.
8. Carey S (1996) Perceptual classification and expertise. In: Gelman R, Au T Eds. *Perceptual and cognitive development*. San Diego: Academic Press, pp: 49-69.
9. Goren CC, Sary M, Wu PY (1975) Visual following and pattern discrimination of face like stimuli by newborn infants. *Pediatrics* 56: 544-549.
10. Bushnell IWR, Sai F, Mullin JT (1989) Neonatal recognition of the mother's face. *British J Develop Psy* 7: 3-15.
11. Johnson MS, Dziurawic H, Ellis M, Morton J (1991) Newborn's preferential tracking of face like stimuli and its subsequent decline. *Cognition* 40: 1-19.
12. Maurer D, Salapatek P (1976) Developmental changes in the scanning of faces by young infants. *Child Development* 47: 523-527.
13. Haith MM, Bergman T, Moore MJ (1977) Eye contact and face scanning in early infancy. *Science* 198: 853-855.
14. Diamond R, Carey S (1986) 41: 1-22. Why faces are and are not special: An effect of expertise. *J Experimental Child Psy*.
15. Farah MJ, Rabinowitz C, Quinn GE, Liu GT (2000) Early commitment of neural substrates for face recognition. *Cognitive Neuropsychology* 17: 117-223.
16. Tzourio-Mazoyer N, De Schonen S, Crivello F, Reutter B, Aujard Y, et al. (2002) Neural correlates of women face processing by 2-month-old infants. *Neuro Image* 15: 454-461.
17. McCarthy G, Puce A, Gore JC, Allison T (1997) Face specific processing in the human fusiform gyrus. *J Cognitive Neuroscience* 9: 604-609.
18. Tong F, Nakayama K, Moscovitch M, Weinrib O, Kanwisher N (2000) Response properties of the human fusiform area. *Cognitive Neuropsychology* 17: 257-279.
19. Nelson CA (2001) The development and neural basis of face recognition. *Infant and Child Development* 10: 3-18.
20. Bachevalier J (1994) Medial temporal structures and autism. A review of clinical and experimental findings. *Neuropsychologia* 32: 627-648.
21. Howard MA, Cowell PE, Boucher J, Brooks P, Mayes A (2000) Convergent neuroanatomical and behavioral evidence of an amygdala hypothesis of autism. *Brain Imaging* 11: 2931-2935.
22. Baron-Cohen S, Ring HA, Bullmore ET, Wheelwright S, Ashwin C, et al. (2000) The amygdala theory of autism. *Neuroscience and Bio Behavioral Reviews* 24: 355-364.
23. Boucher J, Lewis V (1992) Unfamiliar face recognition in relatively able autistic children. *J Child Psychology and Psychiatry and Allied Disciplines* 33: 843-859.
24. Dawson G, Meltzoff AN, Osterling J, Rinaldi J, Brown E (1998) Children with autism fail to orient to naturally occurring social stimuli. *J Autism and Developmental Disorders* 28: 479-485.
25. Adolphs R, Sears L, Piven J (2001) Abnormal processing of social information from faces in autism. *J Cognitive Neuroscience* 13: 232-240.
26. Grelotti DJ, Gauthier I, Schultz RT (2002) Social interest and the development of cortical face specialization: What autism teaches us about face processing. *Developmental Psychobiology* 40: 213-225.
27. Pascalis O, Scott LS, Kelly DJ, Shannon RW, Nicholson M, et al. (2005) Plasticity of face processing in infancy. *PNAS* 102: 52297-5300.

28. Sasson NJ (2006) The development of face processing in Autism. *J Autism and Developmental Disorders* 36: 381-394.
29. Weigelt S, Koldewyn K, Kanwisher N (2012) Face identity recognition in autism spectrum disorders: A review of behavioral studies. *Neuroscience & Bio behavioral Reviews* 36: 1060-1084.
30. Baranek G (1999) Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9-12 months of age. *J Autism and Developmental Disorders* 29: 213-224.
31. Dawson G, Osterling J, Meltzoff A, Kuhl P (2000) A case study of the development of an infant with autism from birth to two years of age. *J Applied Developmental Psychology* 21: 299-313.
32. Celani G, Battacchi MW, Arcidiacono L (1999) The understanding of the emotional meaning of facial expressions in people with autism. *J Autism and Developmental Disorders* 29: 57-66.
33. Dawson G, Carver L, Meltzoff AN, Paganiotides H, McPartland J (2002) Neural correlates of faces and object recognition in young children with autism spectrum disorder, developmental delay and typical development. *Child Development* 73: 700-717.
34. De Haan M, Nelson CA (1999) Brain activity differentiates face and object processing in 6 month old infants. *Developmental Psychology* 35: 1113-1121.
35. Pierce K, Miller RA, Ambrose J, Allen G, Courchesne E (2001) Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain* 124: 2059-2073.
36. Schultz RT, Grelotti DJ, Klin A, Kleinman J, Van der Gaag C, et al. (2003) The role of the fusiform face area in social cognition: Implications for the pathobiology of autism. *Philosophical Transactions of the Royal Society, Series B* 358: 415-427.
37. Sparks BF, Friedman SD, Shaw DW, Aylward EH, Artru AA, et al. (2002) Brain structural abnormalities in young children with autism spectrum disorder. *Neurology* 59: 184-192.
38. Lawrence K, Kuntsi J, Coleman M, Campbell R, Skuse D (2003) Face and emotion recognition deficits in Turner syndrome: a possible role for X-linked genes in amygdala development. *Neuropsychology* 17: 39-49.
39. Zhu Q, Song Y, Hu S, Li X, Tian M (2010) Heritability of the specific cognitive ability of face perception. *Current Biology* 20: 137-142.
40. Shakeshaft NG, Plomin R (2015) Genetic specificity of face recognition. *PNAS* 112: 12887-12892.
41. Baron-Cohen S, Cox A, Baird G, Swettenham J, Nightingale N (1996) Psychological markers in the detection of autism in infancy in a large population. *British J Psychiatry* 168: 158-163.
42. Dawson G, Webb S, Schellenberg GD, Dager S, Fried S, et al. (2002) Defining the broader phenotype of autism: Genetic, brain, and behavioral perspectives. *Development and Psychopathology* 14: 581-611.
43. Gauthier I, Tarr M (1997) Becoming a 'greeble' expert: Exploring mechanisms for face recognition. *Vision Research* 37: 1673-1682.
44. Gauthier I, Tarr MJ, Anderson AW, Skudlarski P, Gore JC (1999) Activation of the middle fusiform "face area" increases with expertise in recognizing novel objects. *Nature Neuroscience* 2: 568-573.
- 45.

This article was originally published in a special issue, entitled: "**Pediatric Neurological Disorder**", Edited by Dominic – Gabriel Iliescu