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# **Review Of Eye Gaze And Emotion Recognition Deficits And Their Impact In Autism Spectrum Disorder**

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## Abstract

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition characterized by a range of social communication and behavioral challenges. One significant aspect of social communication that has garnered substantial research attention is the eye gaze behavior of individuals with ASD. Eye gaze is a fundamental component of non-verbal communication, serving as a critical means to establish social connections, convey emotions, and share attention. Research consistently shows that individuals with ASD often exhibit atypical patterns of eye gaze behavior. Specifically, they tend to avoid or exhibit reduced eye contact during social interactions, which can hinder their ability to form and maintain social relationships. These eye gaze deficits can be observed from infancy and persist across the lifespan.

Understanding the implications of eye gaze deficits in individuals with ASD is crucial for developing effective interventions and support strategies. Improved knowledge in this area can help tailor interventions that target specific social communication challenges. Interventions such as social skills training, eye gaze-based therapies, and assistive technologies have shown promise in addressing these deficits and enhancing social functioning in individuals with ASD.

In conclusion, eye gaze deficits in individuals with ASD are a significant aspect of the social communication difficulties that characterize the disorder. A comprehensive understanding of the underlying mechanisms and implications of these deficits is essential for the development of targeted interventions aimed at improving the social and communicative well-being of individuals with ASD. Future research in this field will likely provide

further insights and innovative solutions for addressing the unique challenges associated with eye gaze in individuals with autism spectrum disorder.

# Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by persistent deficits in social communication and social interaction across multiple contexts, restricted, repetitive patterns of behavior, interests or activities (DSM V American Psychiatric Association, 2013). The DSM-IV referred to the basic triad problems underlying autism as impaired social interaction, impaired social communication and restricted behaviour pattern (Wing et al., 2007). The new criteria under DSM V have clubbed together social interaction and communication into one criterion and other remains restricted behaviour patterns.

Wing, Gould &Gillberg, 2011 reviewed the DSM V criteria and pointed that the DSM-V criteria do not mention the lack of imagination leading to the inability to foresee the consequences of one's actions for oneself or for others. The DSM instead introduced repetitive behaviour patterns, not the impaired social imagination, as the last leg of the triad of social impairments. Thus the triad has been reduced to dyad of criteria i.e. social interaction & communication and repetitive behavior patterns.

Under DSM V criteria individuals with ASD must show symptoms from early childhood, even if those symptoms are not recognized until later. This is a significant change from DSM IV as earlier people could be diagnosed with four separate disorders: autistic disorder, Asperger's disorder, childhood disintegrative disorder, or the catch-all diagnosis of pervasive developmental disorder not otherwise specified. Now in DSM V all these disorders are clubbed together into a single category of Autism Spectrum Disorder. This will help in reducing the apparent biases in diagnostic labelling with rich, white males receiving (less-stigmatizing) diagnosis of Asperger disorder, while poor, non-white males and females receive PDD-NOS (or autism)

The new criteria are helpful in improving the diagnosis of ASD without limiting the sensitivity of the criteria which in turn is useful for intervention purposes.

## Prevalence

The current global prevalence estimates of ASD suggest a median of 62/10000 (Elsabbagh et al. 2012). In 2002 the US Center for Disease Control estimated that autism affected about 1 in 150 children. By 2012 the CDC estimate had increased to 1 in 88. Now, according to the latest revision of the estimate recently released, autism affects 1 in 50 children. Though the actual incidence is not known the current estimates of the prevalence of autism in India are 1 in 250. This suggests that there are approximately 4 million individuals with an Autistic Spectrum Disorder in India. Eighty per cent of those with autism are males. A survey conducted by International Clinical Epidemiology Network Trust (INCLEN) found about 1 to 1.5 per cent autistic children between ages two and nine in India which means prevalence rate of one in 66. The survey was conducted on 4,000 households in Andhra

Pradesh, Odisha, Himachal Pradesh, Haryana and Goa. These data indicate that ASD is increasing with an alarming rate in India and worldwide.

The major deficits in ASD will be discussed in detail.

## **Theoretical Framework**

The extensive research in the field of autism has traditionally focused more on the cognitive impairments such as deficits in theory of mind. Currently, the focus is now moving towards a concept of diminished social motivation in the individuals. Both the traditional and current frameworks will be discussed.

## Theory of mind

Theory of Mind (ToM) is defined in psychology as the ability to impute mental states to others and to oneself (e.g. Premack, 1976). It is also referred to as 'mindreading'. By theory of mind we mean being able to infer the full range of mental states (beliefs, desires, intentions, imagination, emotions, etc.) that cause action. In brief, having a theory of mind is to be able to reflect on the contents of one's own and other's minds. Difficulty in understanding other minds is a core cognitive feature of autism spectrum conditions.

For individuals with ASD the immediate social environment seems unpredictable and incomprehensible. They are said to 'treat people and objects alike'. Their difficulties with their social environment could be attributed to low intelligence, as majority of autistic children are also mentally retarded but this in itself can't be the only explanation for their social impairment. It becomes necessary to understand underlying cognitive mechanisms. Baron-Cohen et al. (1985) hypothesized that autistic children lack second-order representations which impacts their ability of pretend play and also theory of mind. *Children with ASD show a significant lack of pretend play*.

A person's ability to make inferences about the beliefs of others helps him/ her to predict their future behavior. This ability starts developing from the second year of life. Baron-Cohen et al (1985) developed 'Sally and Anne test' to assess children's theory of mind. In this test there are two doll protagonists, Sally and Anne. First, it is checked that the children know which doll is which (Naming Question). Sally first places a marble into her basket. Then she leaves the scene, and the marble is transferred by Anne and hidden in her box. Then, when Sally returns, the experimenter asks the critical Belief Question: "Where will Sally look for her marble?". If the children point to the previous location of the marble, then they pass the Belief Question by appreciating the doll's now false belief. If however, they point to the marble's current location, then they fail the question by not taking into account the doll's belief.



An illustration of the 'Sally and Anne test'.

In their study they found that autistic children fail to employ a theory of mind. This failure results from the inability to represent mental states. As a result they are unable to impute beliefs to others and are at great disadvantage when having to predict the behavior of other people. Such deficit can't be attributed to mental retardation as severely retarded Down syndrome children could perform successfully on this task.

This test has also been termed as 'false belief task'. The false belief task is so named because it supposedly tests the child's *belief* about what another person will do, as opposed to the child's prediction about what that person will do. It has been seen that children younger than 4 years of age and those diagnosed with autism don't succeed on false belief tasks. The standard interpretation of the failure on false belief tasks is that children lack a ToM or, cannot read other peoples' minds".

According to Ruffman et al. (2004), "There is now abundant evidence that false belief understanding in children is linked to their language ability." Therefore, many researchers have focused on the role of language in ToM. A certain level of verbal fluency is necessary to make an inference about what another person might be thinking, in addition to predicting what he or she might do. This is because the inferences and predictions themselves are verbal responses evoked by the particulars of the situation, including questions by others. Several studies show a positive correlation between early language development and later ToM abilities (e.g., Astington & Jenkins, 1999; Bretherton&Beeghly, 1982; Shatz, Wellman, & Silber, 1983). Not coincidentally, the verbal behaviors comprising a ToM begin to occur in about the second year of life. Gains in language have been shown to be a predictor of preschoolers' ToM performance (Astington & Jenkins, 1999).

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With significant impairments of ToM in children with autism, interventions are now being aimed improving the ToM. These interventions are aimed directly at children as well as by training teachers. Such researches are tend to hampered by small samples, the absence of randomized controlled trials, and poorly designed outcomemeasures (Smith et al. 2007; Lord et al. 2005). Randomized Controlled Trials (RCT) are rare in autism research that's why most studies have used informants (parents, teachers), who report on children's real life application of ToM skills through questionnaires.

To date, three RCTs specifically focused on the effectiveness of training Theory of Mind skills. Fisher and Happe (2005) selected 6–15 year olds with ASD and varying cognitive abilities, based on their poor Theory of Mind skills. The training,

included up to 10 individual 20–25 min sessions, lasting 5–8 days. Compared to a control group, the trained children, ranging from mentally retarded to normal intelligent, showed marked improvements in their performance on Theory of Mind tasks, which remained stable at follow up, between 6 and 12 weeks later. However, the training did not affect children's emotion recognition skills, nor their daily life Theory of Mind use, as reported by their teachers. They demonstrated that children with ASD could be taught to pass standard ToM tasks.

The second RCT showed the effect of a computer program for training emotion recognition. Six to 18 year olds with ASD and varying cognitive abilities improved compared to a control group on emotion recognition in cartoons and second order Theory of Mindreasoning, but not on their recognition of facial emotional expressions (Silver & Oakes 2001).

In the third RCT Beeger et al. (2011) conducted a randomized controlled trial on the treatment effect of a Theory of Mind training. The study included 40 children with HFASD aged 8–13 years old, conducted 16 one hour weekly sessions where children were trained on precursors of Theory of Mind (perception, imitation, emotion recognition, pretence), elementary Theory of Mind understanding (belief and false belief understanding) and advanced Theory of Mind understanding (second order reasoning and the use of irony and humour). Effects of the treatment were found on the conceptual understanding of Theory of Mind, in particular on

the ability to reason about beliefs and false beliefs, and on the understanding of mixed and complex emotions. Conceptual measures and the awareness of emotions were not affected by the treatment. The Theory of Mind training did not improve children's social skills as reported by their parents. The treatment had a higher impact on conceptual abilities than on daily life skills, no effects were found on the precursors of Theory of Mind and basic emotion understanding.

These studies indicate that treatment may not increase children's objectively measured social skills, but could enhance their quality of life nonetheless, by increasing their self-esteem.

#### **Social Motivation Theory of Autism**

Deviating from the traditional paradigm of ToM, Chevallier, et al. (2012) have propounded a social motivation theory of autism. Recently social motivation has emerged as a promising research domain at the intersection of social psychology, behavioral economics, social neuroscience and evolutionary biology. The authors suggest that social motivation is a powerful force guiding human behavior and that disruption of social motivational mechanisms may constitute a primary deficit in autism. Social motivation models of ASD posit that early-onset impairments in social attention set in motion developmental processes that ultimately deprive the child of adequate social learning experiences and that the resulting imbalance in attending to social and non-social stimuli further disrupts social skill and social cognition development. Social motivation is subserved by dedicated biological mechanisms and can be seen as an evolutionary adaptation to humans' highly collaborative environment: by enhancing attention to social information, by rewarding social interactions, and by promoting the desire to effectively maintain social bonds, social motivation smoothes relationships, promotes coordination and ultimately fosters collaboration. In ASD, by contrast, there appears to be an overall decrease in the attentional weight assigned to social information. Diminished social orienting, social reward and social maintaining, are all found in autism and can account for a range of behaviors, including cascading effects on the development of mature social cognitive skills. These deficits appear to be rooted in biological disruptions of the orbitofrontal-striatal-amygdala circuitry as well as in dysregulation of certain neuropeptides and neurotransmitters. ASD can thus be seen as an extreme case of early-onset diminished social motivation and provides a powerful model for understanding humans' intrinsic drive to seek acceptance and avoid rejection.

However, both the Theory of Mind and Social motivation paradigms are unable to explain non-cognitive deficits such as repetitive behaviours and restricted interests along with intellectual disabilities in ASD.

# Eye gaze

The eye gaze of an individual is one of the important cues in social communication and interaction. It indicates a person's interest in things around her/him including human beings and other things. It shows a person's direction of attention and the ability to share that attention with others. Eye gaze has been found to be key factor in language development (Baron-Cohen et al. 1997). It has been seen that typically developing children use Speaker's Direction of Gaze (SDG) strategy to learn various nuances of language. For example, if a speaker says "Tree" while looking at it, the child would infer that since the speaker's gaze is directed toward it, it would most probably be "Tree". For this strategy to work, however, *joint attention* ability needs to be present in the child. Joint attention refers to two people attending to the same object at any given time. Toddlers have the ability to produce joint attention behaviours from 9-14 months of age (Butterworth, 1991) which include not only gaze-monitoring but pointing and showing gestures. Joint attention abilities play crucial role in autism. Impairments in joint attention are one of the earliest signs of the disorder.

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Human eyes have the ability to attract attention of others. Eye tracking studies have revealed that adults and even infants fixate to the eyes than any other region of the face when looking at others' faces (Yarbus, 1967). Therefore, information obtained from eye region is crucial for determining various aspects of an individual's identity such as race, age, emotional expression. Neuropsychological studies have suggested the core role of amygdala in preferential orienting to eyes. For example, a patient with bilateral amygdala legions makes fewer spontaneous fixations on the eyes in the context of face-to-face communication (Spezio et al. 2007c). Neuroimaging has shown structural and functional impairments in ASD in 'social' brain regions involved in processing goal-directed actions and biological motion (superior temporal sulcus, STS), theory of mind (medial prefrontal cortex; mPFC and temporo-parietal junction; TPJ), and emotion (amygdala).

Eye contact modulates concurrent and/or immediately following cognitive processing and/or behavioural responses, a phenomenon termed as "eye contact effect" (Senju & Johnson, 2009). Results from neuroimaging studies also indicate that perceived eye contact modulates the activation of social brain network (defined as the cortical and subcortical structures specialized for the processing of social information, such as fusiform gyrus, superior temporal sulcus, medial prefrontal and orbitofrontal cortex and amygdala (Senju & Johnson, 2009).

## Models: Eye contact processing

#### 1) Affective Arousal Model

This model proposes that eye contact directly activates brain arousal systems and thus directly elicits an emotional response. Two distinctive models, the hyperarousal model and the hypoarousal model, have been developed based on the affective arousal model.

The "hyperarousal model" states that the face and eyes of others are strongly aversive stimuli to individuals with ASD, and thus gaze avoidance is an adaptive response (Corden et al., 2008, Joseph et al, 2008). It is hypothesized that individuals with ASD are in a state of physiological hyperarousal which causes them to avoid eye contact. In typical development, this model hypothesizes that the repeated co-occurrences of eye contact and a wide variety of positive experiences through social interaction attaches the positive reward value to eye contact. Based on this model, the development of ASD can be hypothesized as the failure to form such an association, possibly due to the predominant withdrawal (or anxiety) motivation and/or sustained states of overarousal. This hypoarousal model predicts that individuals with ASD should not show any preference for eye contact from early in development, because of the lack of any attached positive reward value.

## 2) The communicative intention detector model

This model proposes that eye contact directly activates theory of- mind computation, because it signals the intent to communicate to the perceiver. This model is consistent with the claims that the atypical eye contact observed in individuals with ASD is based on their difficulty in reading others' mental states from their eyes (Baron-Cohen,

1995; Baron-Cohen et al., 1997b, 2001a) as impairment in theory of mind computation is among the most prominent characteristics of ASD (Baron-Cohen et al., 1985; Frith & Frith, 1999).

The communicative intention detector model often involves an assumption about the innate capacity to detect and react to eye contact. For example, Baron-Cohen (1995) proposed the existence of an innate eye direction detector (EDD) module. The function of EDD is to detect eyes and then input to another module that then calculates others mental states, called the theory of mind mechanism (ToMM). Such mechanisms are usually claimed not to require postnatal experience because their function is to guide subsequent learning. Based on this model, individuals with ASD are hypothesized to lack one or both modules. This modular impairment leads to the failure to infer "mentaristic significance of the eyes", even though the capacity for the decoding of gaze direction (hypothesized to be computed by EDD) may be spared.

The communicative intention detector model predicts that either fixation on the eyes or perceived eye contact should have no effect on concurrent behavioural performance, physiological arousal or neural response in the social brain network, because of defects in the module(s) required to attribute and infer 'mentalistic significance', including communicative intention, to eye contact. As this model claims that the eye contact effect depends of the inference of communicative intention from eye contact, lack of such inference should lead to the lack of behavioural, physiological or neural response that follows it. In addition, such lack of modulation by eye contact should not change throughout development, as it is generally hypothesized to not be due to learning.

3) The fast-track modulator model (Senju & Johnson, 2009)

This model proposes that eye contact processing is mediated by the subcortical face detection pathway hypothesized to include superior colliculus (SC), pulvinar and amygdale (Johnson, 2005b). It has been proposed that the subcortical route is also responsible for face preferences in newborn infants in whom the cortical visual pathways are only poorly functioning (Johnson,

2005b). The fast-track modulator model also assumes that infants are born with widespread connections between the subcortical route and cortical structures (Johnson, 2005b). As a consequence, input from eye contact initially activates widespread structures, which combines with architectural bias in cortex (Elman, 1996; Johnson, 2005a) to form specialized connections between a subcortical route activated by eye contact and relevant cortical and subcortical structures during the course of development.

According to this model, atypical eye contact in ASD could be caused by either impairment in the subcortical face and eye contact detecting route, or in the cortical architectural biases that interacts with subcortical input to form specialized connections. As a result, individuals with ASD fail to develop the social brain, a network of cortical and subcortical structures specialized for the processing of social information.

#### Neural basis of eye gaze deficits

The core disability is thought to revolve around social functioning. Coordinating visual attention with others and understanding their intentions and mental states are the most prominent features of the disability. It has been found that gaze processing deficits appear early in life.

Baron-Cohen (1995) suggested that the deficit is not based in lack of gaze discrimination but to infer the meaning and intentions based on others' gaze.

Pelphry et al. (2005) suggest that the right posterior Superior Temporal Sulcus (STS) region is an important component of the neural architecture supporting social cognition and social perception in neurologically normal subjects. This region of the STS is sensitive to the intentionality and appropriateness of biological motion. They also suggest that individuals with autism fail to link the perceptual representation of eyes moving and the concurrent representation regarding a character's goals, motives and desires (i.e. the contents of the actor's mind) to determine the intentions of another person. Thus, additional processing does not occur in the STS region in subjects with autism, either because an initial expectation regarding what the subject ought to do is never formed (i.e. they do not spontaneously adopt an intentional stance towards the virtual actor) or because information concerning violation of this expectation never reaches the STS region and thus no demand is made for additional processing (i.e. the STS is not re-engaged when the intentional stance is violated). Both interpretations of the present findings point to a disconnection between the perceptual processing of eye movements in the STS region and its connection with the mentalistic significance of these motions. Their findings support previous studies linking deficits in aspects of social cognition and theory of mind in autism to functional abnormalities in prefrontal cortex, the amygdala and the STS.

The findings of Pelphrey et al., 2005 revealed that the STS region and other brain structures that have previously been implicated in social cognition and theory of mind were active during observation of gaze shifts in subjects with autism, but these brain regions did not differentiate congruent and incongruent gaze shifts. Recent work has demonstrated that gaze processing involves a large network of brain regions encompassing anterior and posterior sections of STS, lateral parietal cortex and mPFC. These regions have all been reported to distinguish different gaze directions which imply that the functional roles in this network are not mutually exclusive. aSTS may be involved in perceptual processing of gaze because it distinguishes different averted gaze directions in a head view-invariant manner ; lateral parietal cortex may contribute to gaze-cued attentional orienting because in both human and macaque this region shows a functional overlap between gaze-cued and non-gaze cued attentional shifts; finally, mPFC may be particularly engaged when gaze following is used to establish joint attention.

Baron-Cohen et al. (1999b) used fMRI to measure brain activity during a task requiring participants to infer the mental state of another individual from the expression conveyed by that person's eyes alone. The superior temporal gyri, left amygdala and the insula were activated in neurologically normal subjects performing this 'Eyes Task'. Relative to controls, subjects with autism activated frontal components less extensively than did neurologically

normal subjects, and showed decreased activation in the amygdala and increased activity in the superior temporal gyri.

# How eye gaze is related to autism

We have seen that eye gaze of a person is important for social interaction and communication.

Zwaigenbaum et al.(2005) studied 150 infant siblings of clinically diagnosed children with autism and suggested that prolonged latency to disengage in visual attention can be seen in the first year of life of children who are later diagnosed with autism along with several specific behavioral markers, including atypicalities in eye contact, visual tracking, disengagement of visual attention, orienting to name, imitation, social smiling, reactivity, social interest and affect, and sensory-oriented behaviors.

To understand whether difficulties in eye gaze can be predictive of later emerging autism Elsabbagh et al. (2013) measured the efficiency of disengaging from a central visual stimulus to orient to a peripheral one in a cohort of 104 infants with and without familial risk for autism by virtue of having an older sibling with autism. Their findings indicate that at 7 months of age, disengagement is not robustly associated with later diagnostic outcomes. However, by 14 months, longer latencies to disengage in the risk group later diagnosed with autism was observed relative to other infants at risk and the low-risk control group. Moreover, between 7 months and 14 months, infants who were later diagnosed with autism at 36 months showed no consistent increases in the speed and flexibility of visual orienting.

Their findings are supportive of other researches which indicate that infants who receive a diagnosis of autism as toddlers show few differences in their behavior at 6 months but begin to show observable differences by about 12 months of age, characterized by the presence of atypical social and nonsocial behaviors documented by observational as well as experimental studies, including unusual eye contact, lack of orientation to name, and poor motor control (Landa & Garrett-Mayer, 2006; Bryson et al., 2007). Thus it can be stated that reduced flexibility in the control of visual attention is among the first early emerging features of autism, evident by at least 14 months of age.

Nummenmaa et al., (2012) studied how autism spectrum traits can predict the neural response to eye gaze in typical individuals. Their study shows that in typical individuals, the neural response to eye gaze across the social attention network (pSTS, TPJ, amygdala, IPS, SPL, and SMG) is closely related to the number of autism spectrum characteristics they display. Pelphrey et al. (2005) showed that relative to typical controls, individuals with ASD show less activation to eye gaze cues in areas such as the pSTS. Understanding intentions of others from social cues such as gaze is often impaired in autism (Baron- Cohen et al., 1995), and imaging studies have found that when compared to controls, individuals with ASD show an abnormal pSTS response to the perceived intentionality of gaze shifts (Pelphrey et al., 2005). Their results demonstrate a relationship between neurophysiology and autism

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spectrum traits in the typical (non-ASD) population and suggest that changes in the functioning of the neural circuit for social attention

perception is associated with an extended autism spectrum in the typical population.

Akechi et al. (2011) demonstrated how children with ASD use referential gaze to learn the name of an object. They conducted two eye-tracking experiments to investigate this difficulty by controlling and recording children's gaze fixation. Typically developing (TD) children (age: 6-11 years) mapped the novel word to the novel object in the speaker's focus more frequently than children with ASD (age: 6-11 years). Additionally, the former looked at the object in the speaker's focus longer than the object in their own focus, while the latter looked at these objects for the same duration. When the saliency of the object in the speaker's focus as well as TD children (age: 6-12 years). The findings suggest that the duration of gaze at an object in the speaker's focus is related to difficulty in referential word learning in children with ASD.

Thus eye gaze deficits in early childhood can predict later development of ASD and can be seen as one of the earliest signs of ASD.

# **Emotion Recognition**

Understanding emotions is an important element in social interactions because it enables individuals to recognize intentions of others and fosters appropriate responses. Their understanding is also useful for establishing relationships which in turn is necessary for developing social skills. Detecting the emotions of others is also essential in establishing relationships and in developing emotional reciprocity. Accurate recognition and interpretation of facial expressions help individuals decide when to make socially acceptable statements and provide guidance in determining approach or withdrawal strategies in interpretations (Izard et al. 2001).

Understanding emotions usually requires multi-sensory processing (e.g., Klucharev and Sams 2004). Primarily, information about others' emotions and mental states is derived from their speech prosody, faces, eyes and body gestures.

Understanding emotions during early childhood relates positively to the development of adaptive social behavior and negatively to internalizing behaviors (Izard et al. 2001) and behavioral problems (Blair and Coles 2000). Further, the ability to recognize and label emotions predicts children's social competence. These findings suggest that consistent misconception and misinterpretation of emotion cues or frequent failure to perceive emotions may impede the development of social competence and adjustment (Izard et al. 2001). Ability to recognize emotions is present in infants since 10 months of age (Haviland and Lelwica 1987) and continues to develop through childhood (Herba et al. 2006) and achieve adult level interpretation by the age of 10-11 years (Tonks et al., 2007).

Information received from faces is socially relevant and fundamental in maintaining reciprocal social interactions and interpersonal communication. Faces are highly salient to individuals even in early postnatal development. Facial expressions have a communicatory function and convey specific information to the observer (Blair 2003). Newborn infants spend more time fixating on face like stimuli than scrambled patterns. This face-directed behaviors of the newborn mark the beginning of a protracted developmental pathway by which a small repertoire of basic abilities is transformed into a highly sophisticated and mature face processing system (Pelphrey et al., 2002). Face perception is usually holistic or configural rather than elemental or piecemeal. To identify a particular face or a facial expression, normally developing individuals generally rely on the spatial configuration of the major features of the face, including the eyes, nose, and mouth.. Neuropsychological studies indicate that some subjects with damage to particular regions of the brain show impaired face recognition but intact object recognition (prosopagnosia). The results of neuroimaging studies demonstrate that occipito-temporal cortical areas, particularly parts of the fusiform gyrus, are activated more in response to faces than to other stimuli. Studies have demonstrated that emotions are expressed differently on different regions of the face. Dimberg & Petterson (2000) suggest that positive emotions are expressed on the lower part of the face whereas negative emotions are expressed on the upper part. Such findings suggest that the right hemisphere of the brain may be relatively more involved than the left hemisphere in processing facial information such as identity and emotion. Pelphry et al. (2002) in their study demonstrated that participants with autism visual behavior was erratic, undirected and disorganized whereas controls focused more on eyes, nose and mouth tracing a triangle between them. Participants with autism fixated for a significantly less time on the core features of the face. These results suggest that autistic individuals examine faces differently than do normally developing individuals and that these differences may underlie deficits in face perception and facial affect recognition among autistic individuals.

## Eye gaze and emotion recognition

Studies have demonstrated that emotions are differentially expressed on specific regions of the face. For example, negative emotions are expressed more on the upper part of the face, whereas positive emotions are expressed more on the lower part of the face (Dimberg &Petterson 2000). To understand the meaning of the expressed emotion a person needs to pay visual attention to the cues from the face and gestures. Thus if a person is poor in visual attention, he/she may fail to understand the meaning of emotion being expressed by others which in turn will affect social interaction and relationships.

Therefore, better visual attention to specific face regions may result in better performance in emotion recognition. It has been suggested that the deficits found for face and eye processing may underlie impairments seen in the recognition of emotion by such individuals and subsequently may contribute to their social impairments.

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Schurign et al., 2014 suggest that when emotions are of moderate intensity individuals fixate more on the eyes but when the intensity increases the fixation to eyes decreases. They found that neutral faces are more fixated at than more emotional faces. For example, while viewing faces with the expression of disgust, participants looked less at the eye region and more toward the upper nose and upper lip as the intensity of disgust expression increased. These differences are likely due to the importance of the furrowing of the nose and mouth when making the disgust facial expression, thus making the upper lip more salient. For joy, participants fixated the least at the eyes and spent the most time fixating at the upper lips. This is likely driven by the importance of the upper lip relative to the smile, the most salient facial feature of joy. They explain that when faces become more neutral fixation to other parts of face decreases and instead eyes are fixated more frequently. Their findings suggest that the five facial regions (eyes, upper nose, lower nose, upper lip, nasion) may be the most critical for emotional recognition within faces. Their findings are consistent with the idea focusing attention on certain diagnostic regions is beneficial for emotion processing. Individuals with autism, schizophrenia, social phobia, and Alzheimer's Disease typically focus longer at nondiagnostic regions (such as the brow or cheeks) relative to diagnostic regions of the face (eyes, mouth, nose) during emotional recognition. These abnormal eye movement patterns contribute to social or emotional deficits that accompany abnormal face recognition.

## Importance of emotion recognition in Autism Spectrum Disorder

Because the core deficits in ASD involve impairments in reciprocal social interactions and social behaviors, several studies have investigated emotion recognition. Some studies have identified deficits whereas others have reported no evidence of deficit. These differences could be due to different experimental task demand, experimental stimuli (ambiguous or unambiguous).

Emotional competence in ASD seems to be dependent on many variables such as intelligence, age and context (Begeer et al. 2008). Individuals with ASD and typically developing individuals may perform differently in natural and laboratory settings when experiencing, expressing emotions and interacting with others. As we have seen individuals with ASD display impaired visual behavior, such impairment impacts their social interactions and emotion recognition.

Impairment in individuals with ASD is seen to be more prominent in negative, more subtle or more complex emotions or expressions embedded in a social context. Face identity research has provided evidence for atypical processing strategies in individuals with ASD (Weigelt et al. 2012). People with ASD show an attenuated reliance on the eyes region: they are less attentive towards the upper face part in comparison to neurotypicals, but instead the lower face seems to be more salient.

Loveland et al. (1997) studied four groups of High Functioning Autism (HFA), High functioning Comparison (HFC), Low Functioning Autism (LFA) and Low Functioning Comparison (LFC). They hypothesized that participants with LFA or HFA would correctly identify emotion less often than the comparison groups, less often

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when the information is non verbal and implicit. They found out there was no statistical difference either between tow LF groups or HF groups in indentifying emotion. This could be attributed to the developmental issues rather than presence or absence of ASD. This indicates that developmental level is an important determinant for emotion recognition. Also participants found it easier to identify emotion when both verbal and non-verbal cues were provided. Thus difficulties in emotion recognition in participants LFA or mental retardation were not due to a general inability to make use of information provided but due to developmental issues. Recognizing emotion was more difficult when information was implicit. Thus, according to the authors emotion recognition is more successful when information has both verbal and non verbal cues, is explicit and there are no developmental issues in the participants. Deficits in emotion recognition are not restricted to ASD and can be seen in individuals with Down syndrome and mental retardation as well.

Adolphs et al (2001) suggest that individuals with autism do not show visuoperceptual impairments in discriminating human faces either on the basis of identity or emotional expression. Attentional, perceptual, cognitive and neural processes are altered in individuals with autism which lead to difficulties in emotion recognition. The information provided by eyes and facial expressions may be difficult for persons with ASD to interpret. However, individuals with High Functioning Autism (HFA) show relatively selective impairments in recognizing higher order mental states from faces. Baron-Cohen, Wheelright, & Jolliffe (1997) found that higher-order mental/ social states which were signaled primarily by eyes were not recognized by individuals with HFA. However, they were able to recognize basic emotions such as happiness. It has been argued that social impairments seen in autism result from impaired ability to use a "theory of mind" (Baron-Cohen, 1995).

Rump et al. (2009) suggest that due to developmental processes typically developing individuals become more proficient at recognizing subtle facial expressions. There is continuous improvement in the ability to recognize facial expressions as a child grows into adulthood. They also become better at recognizing faces. Individuals with autism are unable to reach the proficiency at emotion recognition of typically developing adults. This inability stems from the tendency to process faces and, hence,

probably facial expressions, based more on featural than configural information. However, it is unclear whether individuals with autism are *unable* to process configural information or whether they are just less efficient at processing this information. Another factor may be that individuals with autism have poor mental representations of the basic emotions. Individuals with autism have difficulty comparing a subtle facial expression to their inadequately formed stored prototypic representations of the different basic expressions.

Tell et al., 2014 investigated eye gaze direction and expression intensity effects on emotion recognition in children with autism disorder and typically developing children. They found out that happy expressions were easily recognized by children with ASD and typically developing children regardless of eye gaze direction. This finding indicates that positive emotion recognition is relatively intact in children with ASD. Anger was also recognized

by children with ASD. All children perceived expressions of anger with *direct* eye gaze as more intense than expressions with averted eye gaze thus indicating that direct gaze is related to more intense emotions.

Jones et al. (2011) found no evidence of a fundamental emotion recognition deficit in the ASD group and analysis of error patterns suggested that the ASD group is vulnerable to the same pattern of confusions between emotions as the non-ASD group. Despite this finding, recognition ability was seen to be significantly impaired in the ASD group for surprise.

Uljarevic & Hamilton (2013) in their meta analytic study of emotion recognition in autism found a large negative effect size (-0.80) indicating that there is indeed a general impairment in emotion recognition in individuals with ASC. They found that ASC individuals had difficulties in the recognition of five basic emotions but did not have difficulties in recognition of happiness and that recognition of fear was worse Therefore, if recognition of happiness is not impaired in autism, this argues against the idea that poor emotion recognition is universal and primary in autism and if recognition of fear is worse than recognition of happiness, this favours theories that link autism to poor eye contact and poor fear processing in the amygdala.

Evers et al. (2014) studied a group of six-to-eight-year-old boys with ASD and an age- and intelligence-matched typically developing (TD) group without intellectual disability performed an emotion labelling task with hybrid facial expressions. Five static expressions were used: one neutral expression and four emotional expressions, namely, anger, fear, happiness, and sadness. Hybrid faces were created, consisting of an emotional face half (upper or lower face region) with the other face half showing a neutral expression. The results indicated no emotion recognition impairment of angry, happy, sad, and fearful and neutral original expressions was found in children with ASD. They found an interaction between emotion and hybrid type, providing evidence for the existence oftopand bottom-emotions in children. Mouth information holds crucial information to correctly identify the emotion happiness. For the emotions anger, fear, and sadness, the most salient information is located in the region of the eyes. Their findings suggest that using hybrid stimuli is valuable for a coarse evaluation of attention bias, without the need for eye-movement registration. The participants recognized original expressions more accurately than hybrid faces with neutral eyes and hybrid faces with neutral mouth. Such a finding could be attributed to the apparent overrepresentation of top emotions in the stimulus selection. In addition, providing the most salient or relevant emotional information (mouth or eyes region, for bottom- and top-emotions, resp.) is not only sufficient for a successful identification of the expression, but moreover, adding more emotional cues (namely, the other face half), does not cause a significant increase in performance level. This trend was seen in both groups suggesting that children did not integrate emotional information across both face parts, depending mostly on amore local analysis of the salient regions. This could be indicative of the fact that they tend to use a more feature-based recognition strategy in daily life too.

Thus this study contributes to the research body which maintains that emotion recognition is not impaired in ASD. However, this applies to basic emotions only and complex emotions such as surprise which require a theory of mind are not recognized by individuals with ASD.

Can emotion recognition abilities be enhanced in those diagnosed with ASD? Golan et al., 2009& Baron-Cohen et al.(2009) believe it can be enhanced. In their study they evaluated The Transporters, an animated series designed to enhance emotion comprehension in children with ASD. According to Baron-Cohen (2008) children with ASC love to watch films about vehicles because they are strong 'systemizers'. They are drawn to predictable, rule-based systems, whether these are repeating mathematical patterns, or repeating electrical patterns (e.g., light switches), or repeating patterns in films. According to the hyper-systemizing theory (Baron-Cohen 2006), vehicles whose motion is determined only by physical rules (such as vehicles that can only go back and forth along linear tracks) would be much preferred by children with autism over vehicles like planes or cars whose motion could be highly variable, moving at the whim of the human driver operating them. With this premise, Baron-Cohen et al. (2009) created a children's animation series, The Transporters (www.thetransporters.com), based around eight characters who are all vehicles that move according to rule-based motion. Onto these vehicles they grafted real-life faces of actors showing emotions. They tested whether creating an autism friendly context of predictable mechanical motion could render facial expressions of emotion more learnable and increase the motivation to learn them. The 15 key emotions depicted on the vehicles are happy, sad, angry, afraid, disgusted, surprised, excited, tired, unfriendly, kind, sorry, proud, jealous, joking and ashamed. They investigated the effects of the animated series over a 4 week period. The results show that use of the DVD led children with ASC to improve significantly in their emotion comprehension and recognition skills on tasks including the emotions presented by The Transporters. The improvement of the intervention group was not limited to tasks that required close generalization; these participants were also able to generalize their knowledge to perform at the level of typical controls on the distant generalization task, which required emotion recognition from naturalistic clips of human characters that were not attached to vehicles. Golan et al. (2009) also investigated the effectiveness of individual use of The Transporters animated series (with parental support) over a 4 weeks period. Our results show that use of the DVD led children with HFA/AS to improve significantly in their emotion comprehension and recognition skills for the 15 key emotions presented by The Transporters which could be generalized to real life situations.

Thus emotion recognition research in autism has yielded mixed findings about the impairment. Some studies claim that there is indeed impairment in recognizing emotions in those diagnosed with ASD whereas others claim that this impairment is not generalized and that basic emotions such as happiness and sadness which do not require reading the mind of others can be easily identified by those with ASD. It has also been found that impaired emotion recognition has a significant impact on social interaction abilities as well. The ongoing research has found that emotion recognition may be successfully taught which can be replicated in real life situations. Thus improvement in emotion recognition abilities can result in improved social interaction for the affected individuals.

#### **Social Communication**

Profound deficit in social reciprocity skills is the core, underlying feature of the autism spectrum disorders (ASD), which include autistic disorder, Aspergers disorder, and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS). Socialization deficits are a major source of impairment regardless of cognitive or language ability for individuals with ASD (Carter et al. 2005). Contrary to popular view these deficits don't get over by age. Individuals with ASD suffer direct and indirect consequences related to social interaction deficits. Landa et al. (2007) conducted a prospective study of ASD from 14 to 36 months of age to document distinct patterns of social, communication, and play development associated with early and later diagnoses of ASD. They evaluated communicative, social-affective, and symbolic abilities using communication temptations (e.g, the adult opens a bubble jar, blows bubbles, tightly replaces the lid, then sets the jar in front of the child), probes for response to joint attention, receptive language, and an opportunity to play with a standardized set of toys. The findings these findings support the retrospective findings of significantly disrupted development by the first birthday in some children with autism but not others. The toddlers with an early diagnosis of ASD were clearly differentiated at 14 months of age from all other groups, even from those with a later diagnosis of ASD, in social and communication domains. Their social, communication, and play behavior also differed from that of the non-ASD groups at 24 months of age. In contrast, toddlers having a later diagnosis of ASD exhibited a progressive shift away from typical social and communication development between 14 and 24 months of age. At 14 months, they were essentially indistinguishable from those without ASD on the social and communication variables examined. By 24 months, however, they exhibited less frequent and diverse social and communication behavior than the non-ASD groups. Gains in toddlers with ASD outcomes were minimal, in stark contrast to toddlers without ASD outcomes. Examination of growth trajectories indicated that social gains were even more negligible than gains in play and communication. In fact, a statistically significant decrease in shared positive affect was noted in the later-diagnosis group. Their findings suggest that ASD has a progressive phase involving developmental arrest, slowing, or even regression in social and/or language systems. Children classified as having an early ASD diagnosis showed abnormalities in all aspects of joint attention, in initiation of communication with others, and in the variety of vocal and nonvocal forms used to express communicative initiations by 14 months of age, and these persisted through 24 months of age. Children classified as having a later ASD diagnosis began to exhibit signs of ASD by the second birthday in most cases. Early and persistent impairment in the ASD groups involved joint attention, including coordination of attention with another through triadic gaze shifting (from object/event to person and back to object/event), response to others' bids to share attention to an object, and initiation of bids for others to share attention with the child.

Social communication skills being significantly impaired in ASD, Thiemann and Goldstein (2001) investigated the effects of written text and pictorial cuing with supplemental video feedback on the social communication of 5 students with autism and social deficits. Treatment was implemented twice per week and consisted of 10 min of systematic instruction using visual stimuli, 10 min of social interaction, and 10 min of self-evaluation using video feedback. Results showed increases in targeted social communication skills when the treatment was implemented.

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Sansosti and Powell-Smith (2008) investigated the effects of computer-presented Social Stories and video models on the social communication skills of three children with High-Functioning Autism/Asperger's Syndrome (HFA/AS). Using a multiple- baseline across-participants design, computer-presented Social Stories and video models were implemented and direct observations of the participants' identified target behaviors were collected two times per week during unstructured school activities (e.g., recess). Overall, data demonstrated that the combined treatment package was effective for improving the rates of social communication for the participants. All three participants demonstrated maintenance of skills at a 2-week follow-up. This study provides evidence for intervention presented via computer may be a beneficial method for remediating social skill difficulties for individuals with HFA/AS.

## Conclusion

It has been seen that ASD is characterized by various deficits such as inability to understand others' intentions, ideas; inability to hold joint visual attention; inability to experience, express and recognize emotions and difficulties in communicating with others. These deficits have been well researched. Many studies have shown how signs of ASD can be seen in children below the age of 3 years through their visual patterns. This knowledge can contribute greatly in understanding the disorder better and to plan out intervention modalities. Research in emotion recognition abilities has suggested that emotion recognition abilities can be enhanced in the individuals which can then positively impact their social interaction and communication skills.

# **Future** Implications

Empirical studies evaluating intervention in emotion recognition skill can further add to the existing knowledge as there are few studies in this area. The new paradigm of social motivation theory of autism need to be evaluated and supported by empirical findings.

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