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REVIEW

Visual function in autism spectrum disorders: a critical review

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Studies have shown considerable evidence of visual dysfunction in autism spectrum disorders. Anomalies in visual information processing can have a major effect on the life quality of individuals with autism spectrum disorders. We summarise the hypotheses and theories underlying neural aetiologies and genetic factors that cause these disorders, as well as the possible influences of unusual sensory processing on the communications and behaviour characterised by the autistics. In particular, we review the impact of these dysfunctions on visual performance.

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Autism spectrum disorder (ASD) is a developmental disability syndrome characterised by impairments in social communication and interaction defects. When ASD children start to interact socially, a number of features appear in daily activities, for example, learning difficulties, repetitive behaviour, social and communication parries and abnormal interests. These represent the first symptoms of autism spectrum disorder.[1] According to the estimate made in March 2014 by the US Center for Diseases Control (CDC), one out of 68 children is born with an autism spectrum disorder; males are more likely to have autism than females. The number with ASD in the population increased by 2.8 per cent from 2002 to 2012.[2] Research from the Autism and Developmental Disabilities Monitoring Network, US showed an increase from one per 165 in 2002 to one per 68 children in 2012 diagnosed with autism spectrum disorder.[3] Both improved clinical diagnoses of developmental conditions and heightened awareness of the symptoms among parents and public are posited as contributors to the reported increase in ASD prevalence.[3] The new (DSM-5) diagnostic criteria include all subgroups defined by DSM-IV and intellectual disability (ID) disorders under one umbrella, which may serve to facilitate access to appropriate services and supports for individuals who have ASD in addition to intellectual disability.[4] There is a great debate in the scientific community as to how much of the increase is real and how much is reclassification. Therefore, the numbers of the current prevalence of ASD might include individuals, who previously would have been identified as having intellectual disability or being quirky or eccentric.

Symptoms of ASD can be diagnosed as early as two to four years and could vary throughout a child's life.[5] In some cases, the signs of ASD might start as early as six months old.[6] Anomalous visual disorders are found to be associated with this condition. Several studies in ASD reported impairments in visual perception, facial recognition and movement gestures that are reflected on their social, behavioural and communication skills.[7–9] Vision research has linked disturbance performance in visual tasks seen by autistic individuals to specific dorsal dysfunction and disturbance in connectivity between brain regions in visual cortex; however, the main reasons are still unknown. In this review, we will summarise the findings and discuss areas where visual impairments are linked.

DIAGNOSIS OF ASD

Various diagnostic protocols have been used to diagnose ASD. The purpose of this section is to clarify the subgroups of DSM-IV; however, this review will not distinguish between these groups but we will constantly refer to ASD or autism according to DSM-5 to avoid confusion or misunderstanding.

In 1910, Eugen Bleuler,[10] a Swiss psychiatrist was the first to introduce the word autism. It came from the Greek word autos (meaning self); however, his term, defined syndromes of schizophrenia. The real terminology of 'autistic' was first used in 1939 by Hans Asperger,[11] who was working at Vienna University Hospital at that time. He described what has been later defined as Asperger's syndromes and he used the phrase 'autistic psychopathy' to describe the syndromes of four children that he explained as having 'a lack of empathy, little ability to form friendships, one-sided conversation, intense absorption in a special interest and clumsy movements.' Alternatively, he called it 'little professors' syndromes'.[12] Meanwhile, Leo Kanner[13] reported 11 cases, all of whom shared the same unusual behaviour. His first paper 'Autistic aloneness' described the modern sense of autism. A new book by Silberman[14] discusses the history of this disorder.

Since Kanner[13] and Asperger,[11] the definition of autism has evolved. In 1967, the International Classification of Diseases, Eighth Revision (ICD-8) listed what they called 'infantile autism' under schizophrenia, whereas the Diagnostic and Statistical Manual of

Mental Disorders, Second Edition (DSM-II), published around the same year, specified 'schizophrenia, childhood type' without any reference to autism. Later, the DSM-III[15] published what is called the 'pervasive developmental disorder' that includes 'childhood onset pervasive developmental disorders' and 'infantile autism'. In the edition of the DSM-IIIR the subgroups named differently to 'autistic disorder' and 'pervasive developmental disorder – not otherwise specified (PDD-NOS)'. By the release of DSM-IV,[16] there were three subgroups 'Asperger's disorder,' 'childhood disintegrative disorder' and 'pervasive developmental disorder – not otherwise specified (PDD-NOS)', which was also recognised by the International Classification of Diseases, Tenth Revision (ICD-10). In May 2013, the new version of DSM-5 eliminated the subgroups and replaced them by 'Autism spectrum disorder'.[4] No diagnostic subtypes (for example, Asperger's disorder and PDD-NOS) are listed; the idea was to measure the core feature of autism spectrum disorder' by a severity scales:

1. Social communication (SC).
2. Fixed interest and repetitive behaviour (FIRB).

Each scale ranged from 1 to 3; the higher scores will indicate that an individual suffers from several core deficits and/or greater severity of impairment. The severity and range of symptoms for a child diagnosed with ASD may fall anywhere on the scale between 'high functioning' and 'severe developmental delay'. Both IQ and chronological age are usually associated to scales, which categorise ASD.[17] Visual function of patients with autism spectrum disorder are often reported from individuals, who are able to complete the communication, attentional and sensory demands of the testing. Therefore, less is known about individuals with ASD, who have more limited communication or functional skills.[7,18]

Reszka et al[19] showed that most of the individuals classified with the DSM-IV: autism, Asperger syndrome or PDD-NOS also meet the DSM-5 diagnostic criteria of ASD; however, there has been much discussion of the new criteria that have affected diagnosis and treatment of ASD, practically in identifying high-functioning ASD.[20] These arguments suggest that DSM-5 is required to identify subgroups for autism, which could help with the diagnosis, detection of cause factor, and treatment planning.[21] For more details about the diagnostic criteria and subgroups of ASD, the reader is advised to look into the reviews by Ousley and Cermak[4] and Bryant[22] for more information.

THE BIOLOGY AND THE NEUROSCIENCE OF ASD

From a neurobiological perspective view, autism spectrum disorders are disturbers in the connectivity between brain regions. This could include a weakening of already formed connections or a failure of certain connections to establish correct organisation *de novo*.[23] Research into genetic and biological aspects of autisms found that both the environmental and genetic factors increase the risk of ASD.[24] A disturbed connection may occur *in utero* or during the developmental stage.[25–27] At the developing stages, the influences between genes and environmental factors can vary between individuals and between functional areas, which provide opportunities for differential disruptions that depend on timing of the environmental insult. For example, zinc (Zn^{2+}) deficiency severely affects brain function and neural maturation during the early developmental stage, leading to severe brain impairment in learning and memory in autism spectrum disorders.[28]

Based on family and twin studies, results have shown higher rates of ASD within the monozygotic twins (92 per cent) than dizygotic (10 per cent).[29] Therefore, the risk to having a sibling born with autism to families with an ASD child is high. The disturbance of severity among individuals with ASD could vary; however, research shows that autistic siblings within one family may share the same severity and associated features as evidence of heritability. On the other hand, Hallmayer et al[30] suggested that the consideration of monozygotic twins causing autism is incomplete where environment is a contributing factor. The results point to a possible aetiological heterogeneity of autism, which explains the different aetiologies between individuals with autism spectrum disorders. Therefore, current genetic research is working on differentiation between individuals in order to distinguish relevant genes.

According to the Genome-Wide Association Studies (GWAS), genetic variants in ASD can be either inherited or caused (which is often the case) by *de novo* mutations.[31] So far fewer genes are known to cause autism. Based on genetic studies, autism has a 'complex' inheritance.[32] The disorder does not follow the same predicted patterns of inheritance seen in monogenetic disorders, such as X-linked disorders.[33] The possible genetic mutation can be combined with other environmental factors, which cause the differentiation in the autism spectrum.[24,30] Studies in genetic variants have reported single nucleotide polymorphisms (SNPs) to have a major role in causing autism.[33] Genomic studies have identified and revealed replication and *de novo* variations in several gene mutations, which affect protein formation and functioning that have been found to be linked to ASD.[23,33,34]

The PAGES (Population-Based Autism Genetics and Environment Study) study in Sweden is the largest in this field.[35] This study examined the genetic variants spread across the genomes in more than 1.6 million families with more than 14,000 cases of autism. Specifically, they reported that an inherited common variant accounts for the bulk of the genetic risk for strictly defined autism. They found also that this inherited variant, when compounded with other genes, even with a small part, would increase the risk for autism with family members whose genome is already filled with high-risk common variants. Therefore, although genetic variances accounted for the 60 per cent of the risk of developing ASD, their complex behavioural phenotypes are thought to be due to other factors, such as the environmental and the epigenetics factor as a variation risk for these disorders.[35]

Epigenetic factors

Epigenetic factors refer to the heritable changes in gene activity that are not caused by changes in the DNA sequences but rather by one of the following factors: changing the chromosomal histone modifications, chromatin remodelling, transcriptional feedback loops[36] and RNA silencing.[37] These are endocrine-disrupting chemicals believed to interact with the neurodevelopment of autism. In fact, Qiu[38] has reported that epigenetic factors have more influence than alternation of the DNA sequences in autism, as the covalent modifications of DNA tend to create an interface between the changing environment and the fixed genome.

Studies have linked gene-environmental factors that are likely to contain susceptibility loci for autism on human chromosomes to several environmental causes such as: parental ethanol exposure,[39] paternal age,[40] changes in the digestive tract or new diet,[41] oxidative stress, brain inflammation[42] and / or early brain injury.[43] The reader can refer

to Grabrucker[28] for more details. This altered modification in DNA is linked to various neurodevelopmental alterations in the CNS formation in autism, such as disturbed cortical and subcortical cytoarchitectonics, abnormal cell differentiation with reduced neural size and altered synaptogenesis.[44] Studies on vision have related these anomalies to the differences in local versus global visual motion perception[45] and to the excitatory-inhibitory disturbance[46] that is likely to underline altered visual information processing as well as the social characteristics in ASD.

Brain development in ASD

Early brain overgrowth with a subsequent reduction or plateau, in the first few years of life, followed by an abnormal growth pattern during adolescence is the most common indicator in ASD.[47] Enlargement coinciding with exaggerated cortical thinning seems to be more localised in the frontal region of the brain with an abnormal volume of both grey and white matter compared to a normal population of similar age.[48] As a result, deficits in local connectivity with increased long-range connectivity have been proposed after 24 months of age, suggesting abnormal neural growth trajectories.[47] Although autism may not account for specificity of deficits and they might vary in severity and overlap with other syndromes, they are not synonymous with global intellectual disability or mental retardation. Therefore, research suggests that the key disconnect involves higher-order processing of information between frontal lobe and temporal lobe. For example, reduced activity in the superior partial loci and abnormal related events (for example, cytoarchitectonic abnormalities) could be related to impairments in the visuo-spatial attention in autism.[49]

Studies have shown that abnormalities in the cerebellum can also affect cognition, verbal abilities and communication higher-order executive functions.[50,51] The main defect of the cerebellum in autism was found in the postero-lateral hemispheric region including decreased numbers of Purkinje cells (PC) in autistic conditions.[52] The Whitney et al[52] study compared six autism cases with five-matched controls and used stereological techniques to count the density of Purkinje cells in the postero-lateral cerebellar hemisphere. In the autistic cases, two had mild Purkinje cell decrease and one showed severe Purkinje cell decrease and three were normal. The author suggested that decreased Purkinje cells in the ASD brain may be linked to high intrauterine testosterone in the mother's womb, which results in neural developmental abnormalities;[53] however, the reduced level of Purkinje cells in autistic brains remains unclear. For a good review of cerebellar defects in autism, see Fatemi et al.[54] MRI studies showed significant differences between ASD children and typical developing children (TD) in the trajectories connectivity between the posterior-lateral cerebellar cortex with both the ventral dentate nucleus (VDN) and dorsal dentate nucleus (DDN) due to the decreased number and size of Purkinje cells (Figure 1).[55] Studies identified the posterior cerebellum to control the adaptation of saccadic eye movements by monitoring the difference between expected and observed movement outcomes.[56] Mosconi et al[57] showed reduced vermal activation during saccadic eye movements that reflects on the reduced rate of adaption during gaze shifts, which proves that cerebellar vermis is disrupted in this disorder.

In addition, studies reported abnormalities in the neuronal migration of the anterior cingulated cortex (ACC).[58] This area, in particular, participates in a variety of functions and emotional information processing including the frontal visual field. The anterior cingulated cortex induces early learning, emotional responses and social interaction. This is related to the theory of 'mind' through the connection between the adjacent frontal cortex and temporo-parietal junction.[59] The theory explains the defect of children with autism to detect errors, tasks and motivation that lead to social and communication difficulties as well as difficulty in interrupting facial expression.[60]

The analysis of functional neuroimaging data has revealed perturbations of task-related brain activity for both social and non-social tasks in ASD. Brain responses of individuals with autism to visual stimuli are highly variable in comparison with brain responses of matched controls. This suggests that ASDs are not only dysfunctional in the integration of information across distributed brain networks but also in the basic function of primary cortices. The increased neural variability in autism was specifically associated with alterations occurring in regions implicated in high and low visual perception and

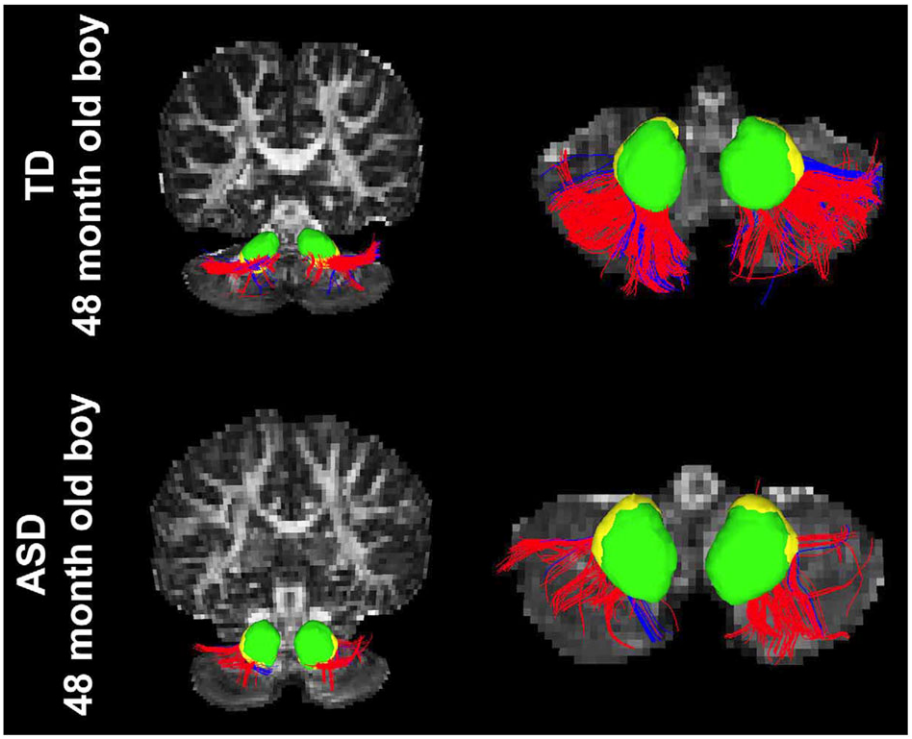

Figure 1. Reduced connectivity between the posterior-lateral cerebellar cortex with the dorsal dentate nucleus (red) and the ventral dentate nucleus (blue) in a boy with autism spectrum disorder, (bottom) compared with a typical developing boy (top). (Reproduced with permission from the author, Jeong et al.[55])

neural connectivity fluctuations, which create unstable visual processing. There is a variety of hypotheses about the neural basis of autism that is way beyond the scope of this review; however, the reader is advised to look at Carlström et al[32] and Baribeau and Anagnostou.[47]

Magnocellular and parvocellular pathways in ASD

Electrophysiological research suggests that specific neurological differences exist in ASD and contribute to the functional differences observed and measured in autism.[61] Research on the magnocellular pathway showed significant defects in children with autism in image processing.[62] A study by Greenaway et al[63] on autistic children, showed a high threshold in the contrast sensitivity in response to three steady pedestal parameters that measured the magnocellular and parvocellular functions. The results showed typical impairments in the visual attention performance in the autistic compared to the healthy control group. This can be attributed directly to a typical lateral visual connectivity and high levels of endogenous noise that account for the defect in the magnocellular area.[63] Research on adults with ASD did not show the same abnormalities functioning in the magnocellular area.[64] This raises the question, whether such abnormalities are overcome in teenagers and adults and/or if they might have lasting effects on the cortical area in autism. Grinter, Maybery and Badcock[65] conducted studies that evaluated the relationship between dorsal and ventral stream in individuals with developmental disorders by studying pattern performance on visual tasks. These were psychophysical experiments using visual stimuli, such as, Glass patterns, motion coherence with random dots and luminance-modulated noise patterns. The findings indicated that dorsal stream is affected in autism;[65] however, evidence of impairment in the higher-level integration and global processing in the ventral stream was also found that might be consistent with the hypothesis of dysfunction in the mirror-neuron system in autism.[66,67]

Visual problems and image processing defects in ASD might vary in onset, severity and behaviour patterns. Bogdashina[68] pointed out unusual behaviour that is linked to visual sensory impairment in autism. She grouped them into hypersensitivity and hyposensitivity. Hypersensitivity, on the one hand, is characterised by focusing on small details, fear of dark and bright lights, avoiding eye contact and tending to look down most of the time, while those on the hyposensitive group, on the other hand, tend to be attracted to bright light and moving objects, standing for a long time gazing at people and using hands to define small details or edges. Here, we aim to evaluate visual functions in ASD, such as visual acuity and colour vision and other common measurement approaches. The reader is referred to other literature reviews[9,69,70] for more details of vision in autism.

VISUAL IMPAIRMENTS IN ASD

Refractive errors

Incidents of refractive errors have been found among population with ASD; however, there are no general conclusions that autism spectrum disorders are associated with refractive errors. The few studies that have covered this area suffer from small samples of the population. In addition, subjects included in these studies were autistic according to the DSM-IIIR criteria. As these diagnostic criteria were narrower, these individuals had more severe levels of autism and may not be comparable to current subject cohorts. Another limitation for several cited reports is that they used a retrospective study design. Thereby, the findings are vulnerable to selection bias and gaps in recall and data. Without a large scale and/or prospective study, there are too many variables to draw an accurate conclusion that might affect the degree to which these results can be generalised. In addition, running a full refractive examination on children with autism sometimes can be very difficult if not impossible to achieve.

One of the early studies that managed to perform full vision test on 98 per cent of the participants (34 children with ASD) used the Teller Acuity test.[71] They reported a 44 per cent incidence of refractive errors with astigmatism and hypermetropia, which was the highest among all (17.6 per cent for both). In 1997, Denis et al[72] completed a full ophthalmic examination for six girls and four boys with autism. Around 70 per cent of the cases were hypermetropic over 1.00 D and 60 per cent had astigmatism. No cases of myopia were reported in this study, which might be attributed to the small sample. Ikeda et al[73] followed 154 children with ASD from 1998 to 2006. The majority of the cases were males (79 per cent). Refractive errors were found in 29 per cent of the cases and hyperopia was also the most common; however, the report did not include if the children had corrections to their refraction errors and if there were any improvements in vision. On the other hand, Black et al[74] found that with correction, 32 per cent of the autistic sample (44 child with 29 per cent of the cases having refractive errors) reached the visual acuity of 6/6. Mixed astigmatism and anisometropia were the most common among these cases.

Study	Year	Number and gender with ASD	Astigmatism (%)	Hypermetropia (%)	Myopia (%)	Other findings (%)	Study type
Scharre and Creedon[71]	1992	32 M 2 F	17.6	17.6	8.8	5.8% anisometropia	Prospective
Denis et al.[72]	1997	4 M 6 F	60	70	_	60% strabismus	Prospective
Ikeda et al.[73]	2013	122 M 32 F	3.89	16.88	5.8	1.95% anisometropia	Retrospective
Black et al.[74]	2013	44 3:1 M/F	18.2	9.09	11.36	6.81% anisometropia	Retrospective
Ezegwui et al.[75]	2014	13 M 5 F	22.2	11.1	-	-	Retrospective

Table 1. Refractive error incidence in individuals with autism spectrum disorder (ASD)

A study from a developing country (Nigeria)[75] also reported refraction errors in a group of 18 children with ASD (13 male). Results showed that 22.2 per cent of the children had astigmatism, while 11.1 per cent had hypermetropia, while mixed astigmatism and anisometropia were also found among the cases. The data of this and other studies are summarised in Table 1.

The incidence of refractive errors among ASD from previous research is comparable to the incidence within the normal population;[76,77] however, there are many challenges in testing visual acuity and refractive errors in ASD due to several factors such as:

1. Children with ASD are not fully co-operative most of the time and they may not perform a full visual test.[182]
2. Charts usually used to test visual acuity (Snellen chart, HOTV test, E chart, et cetera) are insufficient and could give poor judgment on results due to misunderstanding of the task and/ or visual disorder related to ASD specific defect.[77]
3. Issues related to social and communication difficulties should be considered, as they can easily mislead diagnosis and correction of refractive errors and other ophthalmic disorders at an early stage of life.[78]

With regard to these factors, Singman et al[79] conducted vision examinations using the PlusoptiX photoscreener (a vision screener founded 2001 in Nuremberg, Germany)[80] on 25 children, who reported autism. Vision screening using the PlusoptiX uses an examination distance of one metre, no flashlight is required and it measures both eyes simultaneously. The PlusoptiX was 88 per cent more sensitive in reporting refractive errors and identifying risks of amblyopia according to the results compared to regular refraction; however, it is uncertain if patients were really gazing at the PlusoptiX or were attracted by the sound it released. Kancherla and Braun[81] suggested that the difficulties in diagnosing children with visual impairment associated with ASD can delay the diagnosis after the age five. Therefore, it is important to examine vision in ASD using the most reliable methods.

Eye movement defects

Impairment of eye movements is one of the significant clinical features associated with ASD. Rosenhall, Johansson and Gillberg[82] compared 11 autistic children with a control group of the same IQ, age and sex. The study examined binocular vision using auditory brainstem response audiometry and a non-predictive saccade task. They recorded three angles (20°, 40° and 60°) of voluntary horizontal saccades. Although six of the autistic children were found to have abnormal eye movements, the majority had hypometric saccadic movements and difficulties in performing smooth pursuit eye movements and low velocity movements. Rosenhall, Johansson and Gillberg[82] suggested that saccadic movement disorders might be due to brainstem dysfunction in autism. No further explanation has been given for the smooth pursuit movement disorder in this experiment because of the small sample; however, the results were consistent with the findings of Takarae et al,[83] who studied pursuit eye movement in 60 individuals with ASD (mean age of 20 years) and compared them to an age- and gender-matched control group. The test used neuropsychological tasks and an eye monitor. The results showed no differences in saccadic latencies between the two groups but a significant defect was reported in the autistic group in the right saccadic movements and in gaining smooth pursuit of moving objects. An overall reduction was more pronounced in older individuals with autism than young subjects. Results suggested that a functional disturbance in the cerebellar vermis in autism can affect the final visual motor pathway that causes pursuit disturbances. On the other hand, early studies have found no abnormalities in the saccadic and eye movements in autism.[84,85] Controversially, outcomes can be explained in terms of impairment in spatial working to defects in pre-frontal cortex and posterior cingulate connectivity.

Recent research has explained more of the involvement of cerebellar dysfunction in the visuo-motor and the disturbance of gaze and saccadic movements as well as learning disability and language abnormalities in ASD.[84–86] The study by Mottron et al[86] found that children with autism tend to look at objects using 'lateral gazing', which means that ASD moved their pupil to the edge of the temporal corner eye socket, where the head is turned in the opposite direction. This behaviour attempted to stimulate peripheral vision of moving objects to reduce the amount of information produced by central vision. One suggestion, the delay of the cerebellum to transfer information of moving objects or a saccadic task is consistent with the increase in the response time. This prolonged duration of the saccade in ASD relative to typical developing children is related to the caudal fastigial nucleus and the cerebellar vermal lobules VI and VII, where post-lesion resulted in increased duration of the saccade consistent with cerebellar impairment that altered the oculomotor system.[87] Therefore, Mosconi et al[57] measured defects in adaptation rate and amplitude variability in autism, by evaluating the performance on a traditional neuropsychological test of manual motor control in ASD compared to typical developing children. The results showed that 30 per cent of individuals with ASD have slower adaptation than typical developing children in electing saccadic movements across trials compared to only six per cent of the typical developing children group, who failed to adapt to the saccadic amplitude. The author also related reduction of the neural plasticity within the learning centre area of the oculomotor vermis to abnormality in cerebellar neurons, which is consistent with the previous reports.

Eye contact, gaze abnormalities and facial recognition are types of behaviour that characterise individuals with ASD and have been related to the disturbances in eye movements irrespective of the diagnostic category.[88–90] Several measures and methods for assessing the differences of eye movements in autism have suggested that social impairments are reflected in their vision proceeding to variant visual cues. The implications vary between facial recognition and recognising objects. Other explanations involve the influence of memory on visual processing. It has been suggested that autism confirmed domain-memory general impairments[91] that might affect the incoming visual information and the representation stored in memory. So far, studies highlight the influence impairment of eye movement in ASD related to their disorders of facial recognition and therefore, it is important to find the link between disturbance in neural networks in ASD compared with typical developing children.

Contrast sensitivity

Bertone et al[64] studied contrast sensitivity by using two different grating stimuli, simple (first-order) and complex (second-order) both presented at 90° and 180° randomly to stimulate two different pathways in the ventral stream. The study also measured the contrast threshold using flicker contrast sensitivity that stimulates magnocellular and parvocellular pathways with luminance gratings of 0.5 cpd at 6.0 Hz and 6.0 cpd. at 1.0 Hz, respectively (Table 2). Thirteen

Study	No. A	Age A	No. C	Age C	Stimuli type	Amplitude of the luminance modulation Lmin – Lmax cd/m^2	Grating frequencies cpd	Drift frequency (Hz)	Life-time (ms)	Results = A to C
Bertone, Mottron, et al. 2005[64]	13	22:3	13	20:5	Static	0.0 – 0.5	0.75	0	750	High sensitivity to first-order
					Texture	0.0 – 1.0	0.5	6		Low sensitivity to second-order
					Flicker	0.01 – 35.40	6	1		No difference
Jemel, Mimeault, et al. 2010[92]	16	18 – 31	14	20 - 33	Vertical spatial Frequency	Values of 4 %, 8 %, 32 %, or 90 %	LSF = 0.8 MSF = 2.8 HSF = 8	90	-	LSF = No difference MSF + HSF = low sensitivity
Kéita, et al. 2014[93]	21	13 – 33	15	14-24	Contrast Texture	0.5 – 99.50	0.5, 1, 2, 4, 8	75	500	HSF = Low sensitivity LSF = high sensitivity
Koh, Milne and Dobkins 2010[94]	10	15:1	25	15:7	Static	Mean = 23	0.5, 2, 4, 8, 12, 16, 20	100	250	No difference

Table 2. Study results that covered contrast sensitivity in autism spectrum disorder. cpd: cycles per degree, LSF: low spatial frequency, MSF: medium spatial frequency, HSF: high spatial frequency.

autistic individuals were compared to a number-matched control group. A two alternative forced-choice procedure was used to choose between stimuli. Results showed low threshold in first-order stimuli in autism compared to the control group, contrary to high threshold in second-order stimuli. No significant differences were seen in the flicker sensitivity task. The author discussed the results as a deficit in the magnocellular pathway specified by lateral inhibition in the visual system that affected different levels of visual processing. Jemel et al[92] suggested that clear explanation of the reduced responses to spatial frequency information in autism should be measured with dynamic targets that stimulate the spatial filter channels in the visual system. Therefore, they used early visual-evoked potentials (VEP) to record responses to three speeds of sine-wave grating stimuli (low, medium and high) run at four different contrasts randomly. The author suggested that using VEP is a non-invasive method and more specific in defining processing channels within the visual cortex, so that the results will be specified for the elected spatial frequencies response. The results of 16 with ASD and 14 controls showed no differences between the groups responding to low spatial frequency gratings. Mid and high spatial frequencies responses were reduced in the processing through the cortical visual stream channels in autism. These findings proposed reduced function in processing special frequencies that vary between large and fine range in children with ASD.

These early abnormalities on processing visual perception have the impact of abnormal development in the early visual system; however, Morton et al[93] suggest that there is enhanced activation seen in VI of autistic compared to typical developing children, which showed activation in different locations of visual areas. Koh, Milne and Dobkins[94] found no evidence of high spatial frequency differences between patients with ASD and a normal population. We can argue that there were few participants in the experiment of Koh, Nilne and Dobkins[94] in addition to the absence of an age/gender/number-matched control group that makes these results problematic (Table 2). Kéïta et al[95] measured the thresholds of 21 with ASD and a matched-control group of 15, using vertical grating bar stimuli moving across a display in a range of spatial frequencies (with and without noise) and a texture contrast stimuli. In the static version of the experiment, results showed that autistic subjects are more sensitive to luminance-defined and high spatial frequency stimuli and no group difference was reported for fine grating for either luminance or texture contrasts. The authors suggested abnormal connectivity in early stages of visual processing, with compensatory mechanisms accounted for the deficits in visual processing at later stages. The results might be explained as a weak later inhibition in the visual cortex,[96] which increased the neural noise in ASD.[9] That leads to atypical early peaks and disturbs inputs to simultaneous visual channels. The sequences of activities in visual areas during contrast information processing seem to delay at later stage that has an affect on decreasing contrast detection ability at a range of signal/noise ratio in ASD.[97] Taken together, it is evident that results examining low level visual processing remain inconclusive. The variability in methods used to examine visual processing within the visual cortex, in combination with small samples, makes it difficult to compare results across studies. In addition, impairments between age groups and syndrome severity often decline with age. This suggests further investigation to determine whether such improvements in performance among adults with ASD are the result of compensatory factors or the result of the changes in low-level factors related to neural plasticity.

Colour vision

There are few studies, which directly address colour performance in ASD. Based on existing results, it can be said that there is poor colour perception in autism. Franklin, Pilling and Davies[98] and Franklin et al[99,100] carried out a series of colour-detection

experiments on high functioning children with autism using various tasks, such as recognition memory, a search task and a target detection task. The findings found a general reduction in sensitivity to colour detection rather than having a specific colour defect such either tritanopia (blue-yellow) or deuteranopia (red-green). To these findings, Franklin et al[99] worked with 14 high-functioning autistic (HFA) children (mean age of 14 years) attending specialty a school and 14 matched typical developing children as a control group. The first experiment used the Farnsworth-Munsell 100 hue test[101] to measure the accuracy of chromatic discrimination and to identify the nature of any colour deficit in autism. The experiment was done with four trays of different coloured caps and the statistical results reported higher errors in the ASD group than the typical developing children group for colour discrimination. A second experiment of a threshold discrimination task was conducted to investigate colour blindness of the subsystem of colour vision (red-green or blue-yellow). There were 34 high-functioning autistic children compared to 33 typical developing children. The first part of the task was to define a boundary line between the two halves of different coloured circles that varied in colours but had constant luminance for chromatic threshold. The second part was a luminance threshold task, the luminance of the two hemispheres changed along the task, while the colour was constant. All children had been pre-tested with the City colour vision test[102] and they were fully instructed throughout the experiment. Results showed a higher threshold in chromatic discrimination in high-functioning autistics but no significant differences in defining luminance boundaries between the two groups as well as between the age or the non-verbal inelegancy. Both experiments suggested that a true deficit was found in colour perception in ASD and no task difficulty or/and experimental differences can account for the variation of the results. This pattern of findings agrees well with those from previous studies.[100,103] The proposed investigation further explored that those with ASD have reduced sensitivity to colour differences that might arise from impairments in both the retina and visual cortex. Colour processing starts at the retina, where cones with photopigments are sensitive to certain wavelengths. Then, information is processed to the lateral geniculate nucleus at the primary visual areas, where two different pathways will carry chromatic information and luminance to the visual cortex.[104] Several studies have found that other visual areas, mainly in the ventral occipito-temporal cortex as well as the dorsal pathway are involved in colour processing[105,106] As autism spectrum disorders are attributed to changes in visual perception, this might disturb processing of colour information between visual pathways. Another explanation is that it could be similar to the causes of decline in chromatic sensitivity found in the elderly,[107] that neural noise increases or that cone photoreceptors become less sensitive. Therefore, such deficits might account for the reduced chromatic discrimination shown by those with ASD. Alternatively, reduced chromatic discrimination could arise from atypical connectivity in the neural area of the visual cortex with cortical areas that later lead to a general reduction in chromatic perception.[100] Neurophysiological research, such as fMRI of chromatic discrimination in ASD, is essential to test the plausibility of a neural basis to chromatic sensitivity.

Colour processing differences in ASD

The link between colour discrimination efficiency in autism on visual functions has been presented in some studies.[108–111] The findings suggested using colours combined with training methods to improve different levels of visual function in ASD. For example, coloured filters showed improved performance in individuals with ASD on visual perception, social tasks and reading.[112] The proposed mechanism is that coloured filters reduce cortical hyperexcitation, increased by the cortical noise in ASD, especially in primary sensory cortices.

Ludlow, Wilkins and Heaton[111] were the first to use colour overlays, namely, 'a coloured transporting plastic sheet that can be placed over printed text without interfering with clarity' and the results showed an improvement in reading speed in an ASD group of 13 per cent; however, Wilkins, Sihra and Myers[113] explained that there is an overall improvement in reading speed as a result of enhancement of the function of rods and cones to chromatic energy that stimulates the response mechanism of reading. Relatively, autistic responses are not the same for all colours, as overlays work on reducing the contrast and minimise the luminance scattered in the visual pathway due to neurological defects in the visual cortex,[106] which can explain the slow reading speed using white more than darker colours.[113]

Wilkinson and McIlvane[114] showed that children with ASD performed better with the colour-based clustering method in search and match experiments rather than specifying one colour in a pattern. A case has also been reported linking colour-processing differences to obsession and phobia.[115] The explanation for the mechanism of these findings is still unknown; however, further research on colour defects in autism compounded with gaze direction, visual attention and neuroimaging should be considered to define the exact areas of impairment and its relationship to other visual perception deficits in this group.

Visual search

Experiments that used 'embedded figures' and 'block design' tasks for visual attention and visual search have revealed superior performance in individuals with ASD to detect the local details and neglected the global ones compared with control, no matter what the IQ or age.[116] Several studies[117–119] suggest that the ability to detect specific details embedded in an overall picture is the result of overcoming the stimulus of the whole pattern to see specified targets. To this extent, Frith[120] first introduced The Weak Central Coherence theory that was developed further by Frith and Happé.[121] Happé[122] suggested that autistics have the ability to see local information with a relative failure to extract the gist or meaning of events.[122] Her theory was based on the fact that abnormalities in the superior temporal sulcus in the dorsal stream and/ or neurological deficits in the anatomical development of the visual system and image processing areas affected the local and global perception[123] and has been extended by other research.[117–119]

The fMRI study by Boucher et al[124] showed significant differences in the functional distance between certain limbic structures 'amygdala and hippocampus' and other areas in the medial temporal lobe in autism compared to the control group. These differences interfered in the connectivity, which emphasised the role of a rapid and a transient integration and segregation of both local and internal levels of information processing between the studied regions. Boucher et al[124] suggested that these neuropsychological impairments are connected to the deficits in the socioemotional perception and impaired memories in ASD by reducing the spatial

working memory abilities, which underlying the altered search strategy in autism.[125] This area and others in the brain, where abnormalities have been demonstrated in studies of autism, have focused on what is called 'the social brain',[126] which is related to the social and behavioural characteristic abnormalities in ASD. Neuroimaging results showed atypical function in the social brain areas in ASD that affected their visual searching, such as in face recognition, specially for unfamiliar faces.[127] This intentional dysfunction is one of the most reliable early signs of the disorder among affected children;[128] however, Joseph et al[129] compared 21 children with ASD to a similar matched control group to examine memory enhancement and visual perception in target-detecting tasks using dynamic and static search methods. In both tasks, groups were asked to detect the letter 'T' among 'L's in different random selected patterns. In the static method, one frame was used for random a position of the T, while different frames were used for the dynamic search method with interval time of 500 ms between frames. They also used eye tracking to examine spatial attention behaviour throughout the search process. The results showed no difference in the efficiency of searching with the dynamic method between the two groups. The authors argue that autistic children do not memorise the targets. In fact, they moved their eyes searching for the target in the same way as the control group, while in the static searching task, the autistic children's performance was less accurate. The results showed a significant correlation between the severity of ASD (according to the Autism Diagnostic Observation Schedule)[130] and static searching. Joseph et al[129] explained the differences in their findings and previous research by discounting the possibility that memory for rejected distractors augments autistic visual search abilities. They indicated that it was the first time this type of stimulus was used, which does not include other searching triggers, such as linearity and colour with conjunction searches.[131] The findings might be linked to neuro-functional differences that disturb the nature of brain growth, which later characterises the unusual behaviour and sensory interests of ASD. These features seem to be specific to ASD; however, research evidence from other groups on neuro-developmental disorders that have similar learning disabilities or neuropathology, such as Williams syndrome and fragile X syndrome, have shown distinct search deficits compared to control groups.[132,133] The 'enhanced perceptional functioning' theories proposed by Mottron et al[93] and others have found that both low-level (discrimination) and mid-level (pattern detection) perceptual processes are enhanced in ASD.

Following to the hypothesis that linked behaviour and interests of autism to their superior performance on visual search, Blaser et al[134] used task-evoked pupil responses, which measure the involuntary reaction of pupil diameter that happens during visual attention tasks. The idea behind this method is that pupil diameter varies during target detection, and there is a positive correlation between increasing searching task difficulty and pupil diameter. Blaser et al[134] found that autistic children have increased pupil response during the experiment and performed better than the control group. His suggestion was that children with autism might not use the same searching strategy as normal developing children but they are using extra focusing attention that makes them in constant hyperphasic states. Thereby, their performance decreased on tasks that require shifting of attention and increased on tasks that benefit from focused attention and reduced distractibility on fixed objects. In a related review Kaldy et al[135] cover most of the experimental and task methods, which have been used to measure visual attention in ASD in the last 15 years. They concluded that many types of repetitive behaviour of those with ASD came from the unusual visual attention interests, which could be restricted to objects more than people or to the whole environment and later will be reflected by poor social engagement, skills and general attention. Kaldy et al[135] also note that training experiences could improve visual attention that might improve the communication development in autism.

Depth and stereopsis

Children with autism are mostly associated with 'locally oriented' perception and enhanced low-level operation.[136] Their abilities in processing three-dimensional images tend to neglect detailed information from either short-exposure stimuli or long exposure-stimuli in enhanced perceptual function.[93] As we explained previously, several hypotheses have been proposed that this hyper-local orientation might be due to undeveloped (or under-developed) neural perceptual mechanisms in autism, resulting in abnormalities in the magnocellular pathway that enhanced processing defects.[137] For example, Giovannini et al[138] reported that people with ASD underestimate distances in matching tasks compared to a matched control group. Mitchell et al[139] suggested that top-down perception effects are actually developed in ASD. Explaining that individuals with autism are sensitive to visual illusionary tasks, for example, participants with autism have been able to draw the 'devil's fork' and 'penrose triangle' relatively easily, as they are less distracted by the impossibility of the whole image.[140] Mitchell et al[141] used the shaped illusion task in which both groups have to be immune to the distortions induced by 3-D cues. Autistic performance was better than the normal group, and they were less affected by the illusion of the images. On the other hand, Sheppard, Ropar and Mitchell[136] studied the drawing strategies in autism; they concluded that autistics could draw three-dimensional objects with the same accuracy as the control group by using global strategy starting from drawing the figure's outlines first then forming the 3-D inner lines. The author explained that the enhanced perception of the top-down or higher-order might take precedence.

In an experiment that studied the effect of practice on searching strategies in autism, Gonzalez et al[142] used the luggage-screening task with 13 ASD adults and 13 of the normal population. The task usually comes with 3-D screen images of luggage with low and high clutter and participants have to specify the included items. The results revealed similar errors attributed to time and speed reaction between the two groups at the first part of the screening; however, the ASD group showed a great improvement in performance after several trials, stating that the more the ASD group became accustomed to the task, the more they remained focused and the better they inhibited distractor triggers. Accordingly, these results could be very promising especially if practice were to start at a young age. This could give us an indication that autistic people see objects differently or are not influenced by most of the details of the 3-D images when compared to the general population.

Visual field

Most visual research tasks that investigated visual impairment in autism presented stimuli in the central visual field; however, Milne et al[143] were the first to study the visual field in ASD. Eleven participants with ASD (five

with Asperger's disorder and six with autism based on DSM-IV) were matched and compared to 21 controls. They used perimetry to assess the vision field between 30° and 85°. The task was to determine a flashing light with different illumination levels in 12 positions along eight axes. The performance of those with ASD showed impairment in visual field perception, especially at the nasal side more than the temporal side compared to control. Results proposed that these impairments are more likely to be related to a defect of rod-function more than underlying neurocognitive or perceptual problems; however, the test stimuli were presented in the peripheral field and the test was held in a dark room, which was most likely rod-mediated. Therefore, data presented from this study can not provide a direct test for dorsal-ventral stream processing in autism. Rutherford et al[119] tested visual attention in those with ASD using the 'useful field of view'. Their aim was to study if the superiority of autism in advanced visual search tasks is extended to peripheral field tasks. In the experiments, participants underwent three phases, where letter targets were presented between the centre and the peripheral field. The letters were presented at the centre followed by flashing light points in the periphery, followed by a divided-attention task, where letters and light point were both simultaneously presented. The examined area covered 4° to 20° and the findings indicated that ASD performance was the same for all fields of the test points. This may suggest that ASD might have visual field impairments beyond 30°.[143] Accordingly, the small number of participants in Milne et al[143] cannot really reflect all visual field defects in autism. The evidence of visual attention in ASD proved a possible top-down role for the fronto-parietal attentional mechanisms in the integration of spatio-temporal information and specific zoom-out attentional difficulties[144] that might also contribute to the findings of Milne et al.[143] Attempts have been made to explain spatial attention between central and peripheral field in autism using different task properties.

A study by Ronconi et al[145] used 'coherent dot motion' (CDM) stimuli for a directional discrimination task. The dots were presented in the central view (fovea and para-foveal) then in the peripheral view (16° to 21°). In the peripheral task, the central dots completely disappeared, so that the participants are forced to enlarge their attentional visual field to relevant task information. The study also measured the deficiency in the perception of the visual field in the ASD group and the adaptation time needed to shift focus from central to peripheral field by using an attentional zooming task. The results showed a high threshold in the CDM response in both central and peripheral fields of view and a deficiency in zoom-out attention, which suggested that impairment might be selective to the central view in those with ASD. A positive relationship was seen between the severity of ASD and higher impairment in the CDM and attentional tasks. The authors propose that the magnocellular-dorsal (M-D) stream defect found in ASD can be responsible for the rapid change in the stimuli, such as flicker and motion in the visual system.[146] These results supported other findings that those with ASD are intact in low-level M-D stream information processing and impaired in the high-level perception.[147,148] The superiority in processing low-level information in the central field has been attributed to the performance of high-level attention in the peripheral field stimuli, which induced high threshold in detection of the direction of the motion dots. This abnormality in processing motion perception could be improved by influencing the attention in the peripheral visual field in children with ASD using practising tasks for this demand. In conclusion, given that visual field attention appears to be abnormal in ASD, the reduced sensitivity to peripheral information cannot be generalised for several reasons, for example, the small number of participants in those studies limited the results; only a few researchers have investigated the non-central vision and different paradigms in the previous studies had the impact on disturbed attention and misunderstanding of task requirements.

Motion perception and driving performance in autism.

Motion perception is relatively impaired in ASD (A Bakroon and V Lakshminarayanan, unpublished data). In this part our aim is to link between motion perception defects in ASD and driving for the purpose of further research in this area. Since driving is the means of independence and self-identity, it is important to study the ability of those with ASD to react to the 'big picture' for any given driving situation. Will their visual defects stop them from responding to actions in roads, such as time to collision or time to cross a busy intersection? Driving studies in elderly have linked motion perception with other visual impairments as the main visual defects that affect elders' ability to control the vehicle, to interact with other vehicles on the road and to avoid traffic accidents; however, to apply for a driving license, the major visual area that is covered is visual acuity. It has been reported that there is no link between acuity and safety on roads.[149] In fact, results proved that motion perception is linked to the poor performance in driving among the elderly.[150] There are no studies, which have related such impairment to the driving performance and safety in autism. Furthermore, DeLucia and Tharanathan[151] have shown that brief delays in adequate response to relevant moving targets in a driving environment are likely to have potentially dangerous consequences and a reduced ability to adequately discriminate speed or time-to-contact, which could lead to unsafe and problematic driving behaviour. Cox et al[152] conducted a survey of parents of autistic children who learned or are already driving. The results showed that their children do not have the skills for driving. These include the ability to make quick decisions in the context of sudden environmental demands and skills of notes of environmental warnings on roads, which are all primary to proficiency for a driver. Our hypothesis proposes that individuals with ASD will be distracted by their superiority in processing local details at the expense of the global picture. Thus, their driving performance is reduced.

SUMMARY

In this review, we summarised the research on various aspects of visual perception and performance of individuals with ASD. Studies presented visual impairments as the ultimate cause of some social and communication impairments in ASD.[153] Other research preferred to relate the social problems in autism as the main cause of misinterpretation of receiving or processing visual information. In other words, individuals with ASD receive visual information correctly but they fail to interpret it because of their inadequate social and communicative analysis of the visual scene.[64] Overall, visuo-perceptual processing in this group is characterised by superior performance on static spatial tasks and inferior performance on dynamic tasks.[65,154] The general idea suggests there are deficits in the dorsal stream processing and atypical neural connectivity network of visual cortex. This altered low-level perceptual information reduces lateral inhibition that impaired several

visual areas, such as a decrease in contrast sensitivity and visual attention.

Performance differences between several visual tasks for those with autism spectrum disorders, proposed by a number of studies are attributed to task demands, stimulus paradigms and/ or scale changes in the development of the syndrome, which differentiates performance between children and adults for the same tasks.[155] From our point of view, there is one main question that emerges from this review.

The concerns about the impact of DSM changes should be considered in the context of sweeping changes occurring in vision research. The new criteria DSM-5 tended to have more severe impairments than individuals meeting DSM-IV. Also, it eliminated Asperger's and PPD-NOS from the criteria for autism and encompasses them under related disorders. A lack of consistency in the definition complicated the interpretation of new findings in visual impairments in ASD in relation to previous approaches. Some areas of potential autistic visual disorders were consistent, for example, atypical dorsal stream processing in autism. Research found that DSM-5 offers greater specificity but may result in reduced sensitivity, especially for specific subgroups and from higher-functioning autism. Therefore, we can argue that the controversial performance in processing visual tasks may arise as a result of changes in the inclusion criteria for subjects with ASD for recent vision research rather than those before 2013 (when DSM-5 was first established). It is also worth mentioning that insight into the aetiology of ASD is still limited; however, disorders that are caused by a single gene might share the same social impairments as autism but may vary in onset and severity and were excluded from the criteria at a later stage. For example Rett's disorder was included in DSM-IV, even though it was not thought to be a form of autism. Subsequent to Rett's inclusion, a specific genetic aetiology was found. The removal of the condition from DSM-5 reflects intent to avoid distinctions between medical and psychiatric disorder.[156] Therefore, further investigation for visual impairments in ASD diagnosed under the new criteria should be considered to observe to what extent visual impairments are accurately related.

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