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Is Electronic Screen Media Contributing to the Rise in Autism? The Science behind the Theory

Karen Frankel Heffler*

Department of Ophthalmology, Drexel University College of Medicine, USA

***Corresponding author:** Karen Frankel Heffler, Department of Ophthalmology, Drexel University College of Medicine, Philadelphia, USA, Tel: 215-816-5687; Email: karenheffler@gmail.com

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ABSTRACT

This chapter reviews the literature on autism spectrum disorder, early postnatal brain development and neuroplasticity and delineates the scientific basis for a theory of Electronic Screen Media (ESM) viewing in infancy as a likely environmental factor contributing to the rise in prevalence of autism spectrum disorder.

There are many reasons to consider audiovisual screen stimulation in infancy as a potential factor in the development of autism spectrum disorder. Attachment theories suggest that the quality of parent-child interaction is critical for healthy development. Historically, a baby would capture the interest of a family, and the people around the baby would be the most engaging influence in the environment. With technology playing a larger role in society, parents are more distracted from interacting with their young children than ever before, and children are drawn to the light and motion of screen media. Distraction of the screens limiting child-caregiver interaction is only one facet of this theory. Much of the scientific literature suggests that auditory and visual sensory connectivity is atypical in autism spectrum disorder. In autism, there is evidence of auditory and visual superior abilities, while aberrant sensory neural development appears to alter behavior.

A child with autism spectrum disorder does not orient to the social world that surrounds him or her, but instead, is attracted to non-social images, sounds and audiovisual synchrony. Without interest in a social partner, the young child misses out on learning opportunities. Although many researchers have suggested increased sensory neural response in autism, few have considered why this occurs. This chapter reviews many aspects of autism and explains why screen viewing in infancy should be investigated as a potential factor contributing to the rise in autism spectrum disorder prevalence.

AUTISM PREVALENCE

The pattern of Autism Spectrum Disorder (ASD) prevalence over the past several decades is important to consider as a starting point for understanding what may be happening to affected children. Autism rates remained at a steady state for several years, prior to reports of higher incidence and prevalence. Several studies from the 1950s through the early 1980s suggest that autism prevalence was 4 to 5 per 10,000 [1-4] at that time. During the mid 1980s and early 1990s there were indicators that autism rates were no longer stable, but rising. The state of California found a significant rise in autism beginning with birth years in the mid 1980s [5]. Another study found that the incidence of autism rates in a county in Minnesota was 5.5 per 100,000 children in the years 1980-1983, but increased to 44.9 per 100,000 children between 1995 and 1997, and that the incidence of autism was higher starting with cohorts born after 1987 [6].

Blaxill reviewed many studies on autism prevalence in both the United States and the United Kingdom, finding autism rates increased in the United States from less than 3 per 10,000 children in the 1970s to greater than 30 per 10,000 in the 1990s, and in the United Kingdom from less than 10 per 10,000 in the 1980s to 30 per 10,000 in the 1990s. During this period, the broader diagnostic category, autistic spectrum disorder, prevalence rose from 5-10 per 10,000 to 50-80 per 10,000 in both countries [7]. In 2010, the prevalence of ASD among 8-year-olds in the US was reported as 147 per 10,000 (one in 68), almost 30 times the rate of birth cohorts through the early 1980s [8].

Some have suggested that the rise in ASD can be accounted for by broadening diagnostic criteria, more awareness, diagnostic substitution, improved detection, and availability of services [6,9-13]. Further analysis suggests that only a portion of the increase in ASD prevalence can be explained by these factors, suggesting a true increase in the rate of ASD [7,14,15]. If ASD is truly increasing in prevalence, the natural question is what could be contributing to this rise.

GENETIC AND ENVIRONMENTAL FACTORS RELATED TO AUTISM CAUSATION

Many have pondered whether changes in genetic patterns may be the primary cause of the increase in ASD. While direct genetic link has been made to syndromes in which individuals display autistic-like behavior, such as fragile X syndrome, Rett syndrome and tuberous sclerosis [16], it is unclear to what degree genetic mechanisms contribute to non-syndromic, idiopathic

ASD. Genes associated with activity-dependent signaling pathways at the synapse have been associated with ASD [17]. Studies have identified a large number of genes, in the hundreds, or possibly thousands, that contribute to ASD phenotypes [18]. Genetic factors currently account for only 20% of cases, with no specific genetic defect known to account for more than 1 to 2% of cases [19]. Despite many genes involved in contributing risk for ASD, many individuals have a predisposing genotype, but do not develop ASD [20]. An environmental trigger could possibly influence gene expression in the brain through epigenetic mechanisms [21,22].

Older studies involving twins have suggested a very high contribution of genetics to ASD causation [23,24]. Recent research has implicated environmental factors in additional to genetic factors, with greater than 50% of ASD risk attributed to environmental factors [25,26]. When considering environmental factors, we should consider what has changed in the environment of young children during the time frame of increasing ASD prevalence.

ENVIRONMENTAL RISK FACTORS

Various environmental factors have been proposed to contribute to autism, but many do not show the significant rise in exposure that would correlate to the rising rates of ASD. Factors such as the Measles-Mumps-Rubella vaccine (MMR) and mercury-thimerosal in vaccinations [27] had been suggested but not substantiated [28], and removal of thimerosal from vaccinations did not decrease prevalence rates of ASD [29]. Parental age [30] and obstetric complications [31] have been associated with ASD [32]. Maternal history of psychiatric disorder and maternal birth outside of Europe and North America were also associated with increased risk of ASD in studies performed in Denmark [33] and Sweden [34]. Other potential risks that have received some attention include air pollution [35], infection and immune dysregulation [36], gastrointestinal pathology [37], environmental toxicants [38] and oxidative stress [39]. Many of these proposed environmental factors are not associated with a vast difference in occurrence or exposure to explain the significant rise in ASD.

Electronic Screen Media Viewing: An Overlooked Environmental Exposure

Viewing of ESM during the first 3 years of life is a risk factor that has been associated with ASD [40,41]. The availability of new technology has changed the way in which families and children spend their time. Viewing ESM in the home is a relatively new experience for mankind, with commercial television broadcasting having been introduced a mere 70 years ago. The opportunity for babies and young children to view ESM has changed significantly over the same time period as we have seen an increase in ASD. ASD began to rise in the 1980s, coinciding with the growing popularity of VCRs, allowing both repetitive viewing and on demand viewing of selected programs [42]. During the same time period, there was significant growth in the cable television industry. Between 1980 and 1990, the percentage of US households owning a VCR increased from 1 to 69 percent, while the percentage of households with cable television increased from 19.9 to 56 percent [43]. Cable television channels fully dedicated to children's programming were

developed around the same time [44]. With the availability of DVRs, DVDs, and online viewing with computers, tablets, smart TVs and smart phones, the opportunities for extensive screen viewing in early childhood are extensive.

SCREEN MEDIA ENVIRONMENTAL EXPOSURE IN YOUNG CHILDREN

As would be expected with more access to screen viewing opportunities, actual viewing at a young age has significantly increased. In the early 1990s Certain and Kahn found that 17% of 0-to 1-year-olds and 58% of 1-to 2-year-olds watch television [45], while in 2006, Zimmerman et al. [46] found by 3 months of age, 40% of infants were viewing DVDs/videos or television, and by 24 months, 90% of children were regularly viewing. According to the Kaiser Family Foundation, in 2003, 68% of children under two-years of age used screen media for a daily average of just over 2 hours [47]. The same group reported that 33% of households had the TV on almost all the time [47]. A recent study shows that screen viewing among children from birth to age 2 years varies widely, with a range from no exposure to over 68 hours per week [48].

SCREEN VIEWING ASSOCIATED WITH AUTISM

In 2006, Waldman et al. reported a positive correlation between screen media viewing and autism, by analyzing county data on precipitation, time use and autism rates [40,49]. They also looked at county cable subscription rates and found that autism rates in a birth cohort were positively correlated with increasing cable subscription rates when the cohort was younger than 3-years-old. Chonchaiya et al. studied screen viewing history and interactive socialization history in three groups of children, one with ASD, a second group with delayed language development and a third typically developing control group, and published their findings in 2011 [41]. On average, the group with ASD was the youngest to begin screen viewing (6 months of age) and had the most hours of media watching (4.6 hours/day), while the group with typical development was the oldest to begin screen viewing (12 months of age) and had the least hours of viewing (2 hours/day). The group with language delay was in the middle for both age to begin screen viewing and hours per day of screen exposure. Social parameters including hours of social interaction and hours of interactive speech per day had the opposite correlation. The typically developing group had the greatest exposure to these social interactions, the language delay group had an intermediate exposure and the group with ASD had the least hours of interactive social and speech stimulation [41].

SCREEN VIEWING ASSOCIATED WITH LANGUAGE DELAY, ATTENTION DEFICIT AND DECREASED SOCIAL INTERACTION

Other studies have supported the correlation between language delay and increased screen media viewing in young children [50,51]. Audible television has been found to diminish both adult spoken words and infant vocalizations in children aged 2-48-months compared to when

the television is off [52]. Attention problems are also associated with early screen viewing, with each additional hour of viewing/day at ages 1 and 3 years associated with a 9% increase in risk of ADHD diagnosis by age 7 [53]. Both Background and direct screen viewing in children 1-3-years of age has a negative impact on quantity and quality of parent-child interaction during free play [54,55]. Background television also has a negative impact on toy play in toddlers [56], and children 30 months and younger have more difficulty learning from a video demonstration than from real-life experiences [57,58]. In summary, the many associated findings with screen viewing in children 2-years and younger include ASD, language delay, attention problems, negative impact on parent-child interaction, diminished toy play and less learning than from real-life experiences.

While there are many detrimental effects associated with audiovisual screen consumption in children under 2 years, and no known benefits to development, screen viewing in infants and toddlers remains a common practice. Organizations worldwide have publicized recommendations urging parents to avoid ESM exposure in their young children. The American Academy of Pediatrics policy statement issued in 1999 recommends that children under the age of 2 years avoid screen viewing [59]. Countries including Australia [60] and Canada [61] have similar policies, while France has banned screen media targeted to children under the age of 3 years [62].

When do the atypical findings in ASD begin?

Studies show significant changes between the postmortem brain findings of children with autism compared to adults with autism, suggesting an on-going pathologic process [63]. The brains of 6-month-old infants who develop ASD show slightly increased anisotropy of white matter tracts compared to typically developing peers, and undergo a more significant aberrant trajectory of brain development between the ages of 6 months and 2 years [64]. In addition, the brain volume is normal early on in individuals who develop ASD, but the brains become larger than typical by 1 year of age [65,66]. The atypical social behaviors that develop in ASD do not seem to be present in the earliest postnatal months. Gaze to eyes in those who develop ASD is similar to those with typical development at 2 months of age, but declines from 2 to 6 months [67]. Other signs of socialization such as gaze to faces, social smiles and vocalizations are normal at 6 months of age, but become decreased by 12 months of age in children who later manifest symptoms of ASD [68]. These findings suggest a window of typical development prior to the onset of the aberrant brain and social findings, consistent with the concept of an environmental factor impacting both brain and behavior.

What is so interesting about electronic screen media to a baby?

Infants prefer to look at light and motion over looking at faces for the first several months of life [69]. As early as 2 months of age, infants prefer to look at a novel target over a familiar one [70], likely drawing the child to animated screen media, with rapid scene changes. Also, 12-month-old children later diagnosed with ASD were found to take longer to disengage from a visual target than typically developing children [71], possibly giving them a greater risk for

sustained media engagement. However, unlike older children and adults, babies do not have the cognitive development to understand any social meaning in images and sounds on screens.

Neuroplasticity

Studies in the field of neuroplasticity have shown that a young animal's brain connections are altered by sensory experiences. The brain connectivity determines what attracts the animal's attention and how that animal responds. For example, an animal that has visual deprivation in one eye from birth will have a change in the connectivity of the brain so that the sighted eye drives a much greater proportion of the visual cortical cells than the non-sighted eye [72]. However, the cortical neural connections are not significantly altered if the visual deprivation is experienced when the animal is an adult [72]. This suggests a critical time period early in life, when sensory exposure has the greatest effect on brain development. Reversal of the atypical brain connectivity can occur, also during a critical time period early in life, if the sensory experiences of the animal are again altered [73]. According to Pascual-Leone et al., plasticity is an intrinsic property of the brain that allows adaptation to environment and experiences, but can also be a cause of pathology and lead to abnormal behavior [74].

At birth, the human brain is less developed compared to other mammals, and undergoes significant growth in volume of 1% per day during the early postnatal period [75]. This allows an even greater effect of experience on brain development in human infants compared to other species [76]. Several studies have shown that early sensory exposure in infants affects the brain and behavior. One to two minutes a day of exposure to monkey faces in 6-month-old infants over a 3 month period, leads to the ability to discriminate this type of non-human face at 9 months of age [77]. Acoustic training for 20 minutes a week during ages 4 to 7 months, affects the neurologic response measured by event-related potentials at 7 months compared to infants not exposed to the training [78]. These studies demonstrate that the brain is altered by sensory exposure and this drives the behavioral response of the infant. The occipital (visual) cortex is the area of the brain that is most rapidly developing connections during the first 3 months of life, while the higher-order frontal regions of the brain are later to mature [79], allowing the possibility that early visual experience impacts subsequent brain development.

The Brain in Autism: Auditory and Visual Brain Development

When an infant or very young child experiences screen media with images and sounds, he or she does not derive any meaning. The brain connectivity that allows an older typically developing child or adult to understand language, social meaning, and context from a screen has not yet occurred in an infant. The screen images do not interact with the baby in a social sense. The faces on a screen do not respond to the smiles of the infant and the actors do not return eye gaze or engage in joint attention. There is no change in the screen image if the baby is giggling, screaming or crying. Although ESM is not contributing to social learning in an infant, it is reasonable to consider that the audiovisual screen stimuli does affect brain connectivity based on we know about

neuroplasticity. Pascalis et al. revealed that an environmental sensory exposure experienced as minimally as one or two minutes a day in infancy, changes the brain connectivity and behavioral response of a baby [77]. The potential affect on early brain development of viewing screens an average of 2 hours a day [47] and in some cases much more [48], is very concerning. As suggested by Pascual-Leone et al., the potential for abnormal behavior as a result of plasticity-related neural activation [74] must be considered.

Through neuroplastic mechanisms, we would expect that exposure to audiovisual screen media in infancy would affect the sensory areas of vision, hearing and motion detection. Children and adults with ASD have superior abilities in visual discrimination [80-82], and visual acuity [83]. Children with ASD are able to search faster with briefer fixation times than those with typical development, and the superior perception of stimulus features is positively associated with autism symptom severity [84]. Gliga et al. found that superior visual search ability in 9-month-old infants is predictive of worse autism symptoms at 15 months and 2 years of age [85]. In toddlers, visual preference for geometric patterns over social scenes correlates with more severe ASD symptoms, more language deficit and lower IQ [86].

Individuals with ASD also have superior abilities in auditory perception including pitch discrimination [87] and increased ability to detect loudness of a sound [88]. Another area in which children with ASD have a superior ability is in detecting motion [89]. Through neuroplastic mechanisms, one would expect increased brain connectivity relating to those specific tasks in which there has been more sensory experience. Increased abilities in visual, auditory and motion detection tasks seen in those with ASD, mirrors exactly the types of stimuli that a young child experiences while consuming videos and other screen media. These extra-ordinary abilities, however, do not seem to impart higher general functioning in those with ASD, as many of these abilities correlate to more severe ASD disability. The correlation with these abilities in sensory perception and severity of ASD symptoms suggests that the sensory connectivity not only affects local processing, but also interferes with higher levels of cognitive function, affecting the core symptoms of autism.

PREFERENCE FOR AUDIOVISUAL SYNCHRONY OVER BIOLOGICAL MOTION IN ASD

Movement is a strong stimulus for attracting an infant's attention [69]. The ability to attend to the movement of other living beings has been studied extensively. For humans in prehistoric times, the ability to pay attention to the movement of people and animals was important to allow safety from prey, as well as to identify others in the environment from the same species. Animals also recognize the movement of other creatures, and studies have demonstrated this ability in many different animal species [90-92]. In these studies, the movement is represented on a screen by point sources of light depicting the main joints of a moving animal [93]. Newly hatched chicks [94] and 2 day-old babies [95] prefer biological motion over other random patterns of motion, suggesting an inborn preference for biological motion.

Considering the consistency of preference for biological motion over other types of motion throughout the animal kingdom, it is very striking that 2 year-old children with ASD do not orient to biological motion [96]. In this Study by Klin et al., multiple point-light video animations were made from scenes in which actors played children's games. Two animations were shown side by side, with one upright and the other upside down (disrupting perception of biological motion) and played backward. An eye-tracker was used to detect each child's viewing patterns, and the soundtrack coincided with the upright video. The children with ASD were found to preferentially look at the scenes in which the motion change and the sound change occurred at the same time. An example of this is in the pat-a-cake animation, when the point sources of light came together representing the clap at the same time as the sound of the clap was heard in the upright presentation, but not in the inverted backwards segment. When the authors analyzed the soundtrack and videos both forward and backward for audiovisual synchrony, they found that audiovisual synchrony accounted for 90% of the variance in viewing of the autism group [96].

What could possibly affect these children so that their attention is so dramatically altered from an innate preference seen throughout the animal kingdom? Considering environmental experiences and neuroplasticity, these children are likely experiencing powerful audiovisual stimuli to become expert processors in determining where the motion change and sound change are occurring at the same time. Perhaps these children have become superior processors of audiovisual synchrony, and the neural pathways that process these stimuli have altered the attention mechanisms of preferred looking.

SPEECH AND LANGUAGE

Children with autism have greater abilities in pitch perception, but more impairment with attending to speech than non-speech sounds [97]. Social interaction with a live speaker is important for learning language in infants, and the same benefits cannot be gained by audio or video experiences [98]. A more recent study on phonetic learning in a second language, found that infants' eye-gaze behaviors during interactive sessions with a Spanish speaking tutor predicted Spanish phoneme discrimination assessed by Event-Related Potentials (ERP) [99]. These studies suggest that social interaction and joint attention play a significant role in language learning in infancy. The role that ESM plays in interfering with parent-child interaction [54,55], would likely impact the joint attention important in language learning.

A voice sensitive area of the superior temporal cortex shows increased response to the human voice compared to non-vocal sounds at 7 months, but not at 4 months of age [100]. In addition, these researchers found that the right inferior frontal cortex shows increased response to happy prosody. These studies suggest that brain connections related to speech learning are dependent on experience, and emotional speech may play an important role. Preschool children with more severe ASD symptoms prefer computer synthesized non-speech auditory signals to the 'motherese', infant-directed speech [101]. Song et al. found that slow speaking rate and vowel hyper-articulation found in infant-directed speech significantly improved the ability of infants

to recognize word meaning [102]. Early life exposure to audiovisual screen stimuli, which lacks social interaction, infant-directed emotion and the slower, hyper-articulated components of infant-directed speech, would likely affect the neural pathways that are developing in response to these experiences. The difference in how an infant experiences language when consuming ESM compared to real-life, could potentially impact the preference in autism for non-speech sounds.

FACIAL PROCESSING IN ASD

Newborn babies exhibit an early preference of looking toward a face pattern over a scrambled similar stimulus, suggesting an inborn facial responsiveness [103]. In typically developing individuals, face processing occurs in an area of the brain known as the fusiform gyrus [104,105]. ERPs show that typically developing 6-month-old infants recognize faces faster than objects and recognize a familiar face [106]. Individuals with autism have impairments in face processing [107-109], and do not display typical activation of the fusiform gyrus on fMRI during a face perception task [110]. Typically developing children are better at recognizing parts of faces in the context of a whole face [111] and are better at recognizing a face from viewing the eyes than the mouth [112]. Children with autism display better recognition of isolated facial components, and can recognize a face better by viewing the mouth rather than the eyes [112]. While face processing is impaired in children with autism, they have no difficulty recognizing objects compared to their typical peers [107,108] suggesting that general object processing is not affected, but rather, the abnormality lies in the recognition of faces as socially important. fMRI testing during face processing in ASD demonstrates a pattern of brain activation similar to the way in which typical individuals process objects [113].

Studies suggest that the neurologic response to faces is a learned phenomenon. An ERP response similar to that typically seen in face processing can be seen when dog and bird experts are viewing their animal of expertise [114]. It appears that the newborn has an innate potential to develop neurologic specialization for face processing in the fusiform gyrus region of the brain, but that the actual neural connections are dependent upon experience. The face of an actor on a video does not share eye contact, joint attention, or emotional response with the baby viewing. Depiction of a face on a screen would not be recognized in a social or emotional way, and would not be likely to elicit a response in the social regions of the brain. The eyes viewed on a video by an infant are also not recognized as socially significant. The young infant would likely process these screen images of eyes and faces in the same way that the infant would process objects. Live interaction with people whose faces react to and interact with a baby would be more likely to stimulate the fusiform gyrus, and contribute to social neural connectivity.

ALTERED BRAIN CONNECTIVITY LIMITS OPPORTUNITIES FOR SOCIAL LEARNING

According to the AV causation model of ASD [115], the attention of the screen exposed infant is determined by experience-induced neural connectivity which directs the baby to orient to non-social objects, sounds and motion, instead of the social factors in the environment. These

children would attend to the mouth instead of the eyes, because of the synchrony between the movement of the mouth and the sound of the spoken words [96]. Children who develop ASD have diminished attention to people and their activities [116,117], limiting opportunities for further social learning and social brain development. Lack of interest in the infant-directed speech of a parent, and more interest in non-speech sounds [101], would negatively impact the opportunities to learn language.

A child, who does not recognize eyes and faces as socially relevant [113], would have no greater interest in attending to faces than objects, affecting the neural components of face processing, consistent with the findings of aberrant and slow face processing seen in those with ASD [118]. Lack of interest in eyes [119], would likely contribute to the difficulty in identifying the direction of one's gaze toward versus away, as found on ERP of infants who were later diagnosed with ASD [120]. Inability to determine eye gaze direction would impact ability to engage in joint attention and limit opportunities for social engagement. Elsabbagh et al. found that infant neural response to eye gaze correlates with both infant affect and the quality of parent-infant interactions [121], while Wan et al. found that quality of parent-infant interaction at 12-15 months in those at-risk for ASD, correlates with 3 year autism outcome [122]. Decreased attention to a social partner would limit interest in imitation, which has also been associated with ASD outcome [123]. Heffler suggests that social brain connectivity is enhanced in infancy through live interactions with people, sharing attention, and attending to faces, eyes, voices and social activity [115]. Audiovisual screen experiences decrease the available time for social interaction and quality of interaction [54,55]. The AV causation model of ASD proposes that non-social audiovisual stimulation in an infant or young child has the potential to alter neural connectivity, and direct attention to similar types of auditory and visual stimuli, in lieu of social stimuli. These specialized audiovisual brain pathways would further affect behavior, social development and ultimately contribute to global developmental delay [115].

BRAIN DEVELOPMENT IN ASD

The visual occipital cortex is the part of the brain most actively developing connectivity in the first 3 months of life, while the frontal cortex is later to develop [79]. In the ASD brain, the fiber tracts appear to be somewhat more organized at 6 month compared to typical, as measured by anisotropy, but undergo an altered trajectory of development compared to typical from 6 months to 24 months of age [64]. Children who are later diagnosed with ASD undergo a period of early brain overgrowth with significant enlargement of the brain by 2 years of age [65,66]. Researchers have related the increased brain size in infants with ASD to white matter enlargement [124] and accelerated maturation [125]. Some have suggested that the hyper connectivity, thought to be primarily involving short-range connections in the frontal lobe [126], interferes with the normal trajectory of development [127]. Supekar et al. studied children with ASD and typical age-matched controls and found significant whole brain hyper connectivity in the group with ASD, with the degree of hyper connectivity predicting ASD symptom severity [128]. Another study

showed functional hyper connectivity involving frontal and central areas on phase-lagged alpha EEG in 14-month-old infants while viewing videos, which correlated to restricted and repetitive behaviors at age 3 years [129].

The hyper connectivity over the frontal lobe found on EEG during video viewing in infants [129], suggests neural responsiveness to this type of stimuli, consistent with previous exposure. It is possible that extensive viewing of bright animated screen media early in infancy could cause sensory hyper connectivity that subsequently impacts higher order neural development. During peripheral visual task fMRI, the group with ASD showed more occipital activation and lowered prefrontal, parietal and temporal activation than the typical group. These researchers suggest that increased primary sensory processing impairs relevant stimuli selection and overloads higher cognitive processes [130]. Novel auditory detection was studied by fMRI in children with high functioning ASD and typical peers, finding that those with ASD responded more quickly and had atypical activation in the front parietal network, which correlated to Autism Spectrum Quotient [131]. A fMRI visual change detection study by Clery found in ASD, the anterior cingulate cortex has greater functional activation with the occipital (visual) cortex and reduced frontal activation compared to typical [132]. Thakkar et al. found that atypical activation of the ACC during response monitoring was related to repetitive behavior [133]. Khan et al. assessed directionality of connectivity using Magneto Encephalography (MEG), finding evidence of increased feed forward and reduced feedback, suggesting more sensory driven connectivity and less executive control [134]. These studies are consistent with the idea that sensory connectivity in ASD is interfering with higher order processing and cognitive development.

EFFECTIVE TREATMENTS OF ASD

If ASD is a result of neuroplastic mechanisms based on environmental exposure, we would expect reversibility of the condition if the environmental conditions were markedly changed to favor typical development early in life. The AV causation model of ASD suggests that there is a competition between the brain pathways that direct attention to social stimuli and the neural systems that orient to pure auditory and visual sensory stimuli [115]. According to this theory, promoting social interaction and limiting or avoiding audiovisual stimulation, if done early enough, would modify the brain pathways to allow significant improvement. Early intervention is effective in treating ASD if applied intensively at a young age [135,136]. These treatments may involve up to 40 hours a week of 1:1 face to face therapy [137], leaving little time for screen viewing. Other models utilize therapists for fewer hours, but teach the parents strategies for social engagement throughout the day [138]. Some therapeutic programs specifically recommend turning off screens and removing electronic toys which may interfere with the child's social attention [139]. The Denver Early Start Program has been reported not only to improve ASD symptoms, but also to reverse atypical face processing [140]. The AV causation model of ASD [115] explains the success of early intensive intervention, as these therapies promote social brain connectivity, while limiting exposure to electronic screen stimuli and other forms of non-social engagement.

MODELS OF ASD

Many different models have been proposed to explain the development of ASD. Two different hypotheses involving face processing have been discussed by Dawson et al. as relating to autism causation [141]. The first, a perceptual/cognitive explanation, suggests that the infant has a primary deficit in a higher order cognitive function or in the fusiform gyrus that is present at birth and prevents prototype formation impacting typical face processing [142]. The second theory referred to as social motivational, states that the deficits in face processing in ASD stem from a primary deficit in social motivation, causing less attention to faces and other social stimuli, impacting social learning [141,143]. Weak central coherence, inability to form a global concept, has been proposed as a possible cause, or alternatively, that the deficit in coherence may be an outcome of local processing [144]. Elsabbagh and Johnson suggest that causation is likely multi-factorial, with genetic risk as an underlying factor, that requires the added risk of environmental factors to determine not only if a specific child develops ASD, but the extent of involvement [145]. Mottron had previously proposed an 'enhanced perceptual functioning' model to explain many of the sensory findings in ASD and the impact on higher cognitive function [146]. More recently proposed, the 'trigger-threshold-target model' suggests genetic mutations trigger brain changes that account for both the sensory abilities and the social impairments [147].

The AV causation model of ASD, while explaining many of the findings in the other models, is based on neuroplasticity, and implicates an environmental trigger [115]. This model proposes that early environmental exposure to ESM in susceptible infants causes specialization of auditory and visual neural processing pathways. These specialized sensory brain pathways interfere with typical social brain development, and impair higher cortical connectivity [115]. Superior sensory processing for auditory [87], visual [81] and motion discrimination [89], alter the attention of the infant or child to orient to audiovisual synchrony [96] and non-speech sounds [101], rather than social stimuli. The infant does not perceive faces and eyes as social due to the non-social experience of screens and the neural pathways that are directing attention to other things instead of people, and their faces and voices. Heffler [115] proposes that the acute sensory exposure causes rapid neural connectivity, explaining the altered trajectory of brain development with increased anisotropy of white fiber tracts at 6 months of age [64]. This heavy sensory connectivity interferes with higher cognitive function. The brain is less able to prune synapses as would occur in typical development as the social learning is not taking place to reinforce meaningful connections. Brain overgrowth occurs, but is disorganized, leading to reduced anisotropy compared to typical development [64].

The child with ASD is not paying attention to social stimuli, further diminishing the opportunities to learn from social interactions such as joint attention, imitation, speech and facial expressions. This is compounded by the negative effects of screens on parent-child interaction [54,55] and spoken language [52]. Areas of innate social potential are not developed because of lack of social orientation. During a critical period of plasticity, this aberrant neurodevelopment

could be reversed with intense social experience and avoiding the pure sensory screen exposure [115]. Some children may be predisposed to develop the sensory aberrant connectivity because of genes that impact experiential learning [17]. The wide spectrum of ASD involvement could be explained by the contributing influences of factors including age of screen viewing onset, extent of screen consumption, depth of social experience and underlying genetic risk [115].

FUTURE DIRECTIONS

There are many reasons to consider electronic screen media as an environmental factor that is potentially contributing to the development of ASD in susceptible infants. While there are studies showing an association [40,41,49], more studies are needed to specifically evaluate causation. Since infant siblings of children with ASD have a much greater risk of developing ASD [148], a large controlled sibling trial removing screen stimuli in one group and promoting social interaction in both study and control groups would help to determine any causative role of ESM viewing in ASD. If studies confirm ESM as contributing to ASD causation, altering exposure could impact both the prevalence of ASD and treatment outcomes. Since ESM exposure has been associated with other negative outcomes and no developmental benefits in the youngest children, limiting exposure to ESM in these young children could potentially have tremendous positive impact and offers little risk. In the meantime, raising awareness about this association and recommending that parents heed the warnings of the American Academy of Pediatrics to avoid screen viewing in children under age 2 is advised.

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