

Running head: AUTISM IN CHILDREN

1

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Autism in Children

Autism rates among children appear to be shooting on an upward scale in recent years. Autism spectrum disorders (ASD) are a range of related conditions grouped in the category of neurodevelopmental disorders. The disorder is characterized by delayed communication, challenges in social interactions and sensory problems. Many findings have suggested that children that have autism often have cognitive challenges, and about 20 – 30 percent acquiring attacks or epilepsy (Christensen et al., 2018). There exist a number of closely related disorders that share the same fundamental characteristics with autism, but only different of certain symptoms and age of occurrence. Such disorders with similar elements as ASD are atypical autism, Asperger syndrome, as well as disintegrative disorders. The present projections of prevalence of autism are at 16 for every 10,000 children (Boukhris, Sheehy, Mottron and Berard, 2016). However, these prevalence levels rise to 63 in every 10,000 when every type of autism spectrum disorders are incorporated (Jain et al., 2015). The precise causes of autism have not yet been established compressively. Some experts believe that changes in the environment such as mercury poisoning, dietary habits, and vaccine sensitivity can cause autism in children. However, none of these theories has been well proven.

Developmental delays, epilepsy, dysmorphic characteristics, obstetric difficulties, an uneven sex ration, and big head size shows different signs that autism is a neuropsychiatric condition. Possibly, the most significant advance in shifting the present understanding of what really causes autism was the finding that genetic elements play a fundamental part in causing autism. Advances in diagnostics or swift genetic changes cannot comprehensively determine the rise of ASD occurrence among children (Boukhris et al., 2016). There is increasing evidence among scientists and physicians that ASDs among children is a result of engagement between

biological susceptible factors and environmental or iatrogenic abuses (Jain et al., 2015). These zeros into aspects of environment and increases chances of an etiological contribution for toxic contacts. This could be either prenatal, postnatal or in some way an aggregated arrangement, that integrates the impact of maternal, gestational as well as infant vulnerabilities.

There is enough evidence that there could be a likelihood of genetic link as far as what causes autism in children is concerned. Autism and related disorders can be traced in families. For instance, in identical twins, if one of them has an autism condition, then the other one has traces of the same disorder in their system (Bahmani, Sarrafchi, Shirzad, and Rafieian, 2016). Children that have been prematurely are highly susceptible to having the autistic disorder. In addition, those children born to fathers who had advanced ages have a greater chance to contract autistic disorders.

Genetically, children that have traces of autism in them may not have the ability to detoxify poisonous environmental elements that their systems are exposed to. This aspect makes them more susceptible to incur neural impairments that could be related to autistic behavioral characteristics (Oberlander & Zwaigenbaum, 2017). When this category of children is exposed to metal exposure, for instance, taking paint chips or those pica, they may ingest toxic metals into their systems. Lacking the ability to detoxify these environmental elements in their systems makes them have serious causes of autistic disorder.

A lesser number of children diagnosed with autism have evidenced a comorbid disorder of the central nervous system, which apparently leads to the ASD. In short, these comorbid disorders possibly account for about 10 -15 percent of ASD scenarios, but physicians should be aware of them because their existence will have clinical implications (Christensen et al., 2018). Considering comorbid medical disorders, enough evidence that indicates that troubles of the

gastrointestinal system are not rampant in children with autism than in the other children that do not have the disorder has been determined. No causative factors have been determined to contrast children with autism from those with other conditions like Asperger syndrome (Boukhris et al., 2016). Much has been determined that such related disorders emanated from a common familial, supposedly, genetic mechanism.

Other studies have suggested that some environmental contacts in the womb may cause the child born to be highly susceptible to these disorders. Heavy metals poisoning comprising of chemical products, pesticides, industrial paints, silver dental fillings, and many others have long been supposed to be causing autism. Even taking fish is a possible source of metals such as mercury. Anticipating mothers may be exposed to dirt near pathways that are a possible source of lead or paints in their house (Christensen et al., 2018). The true position currently is that these metals are linked to triggering or exacerbating other health challenges in a child, autism being included (Bahmani et al., 2016). Essentially, chemical exposures during child development in the womb can pose a great danger to the unborn that it would to adults. Women that have chronic environmental exposure may transfer possible these toxic metals to their fetuses. Even if the children survive, these toxic elements could be passed during nursing, which will intoxicate the infants as they develop. This could be believed to the extent that testing the hairs of children diagnosed with ASD reveal the presence of greater levels of heavy metals in their bodies.

More studies have confirmed the presence of high levels of heavy metals such as mercury, lead, and aluminum in the hairs of children diagnosed with autism. A study done by Bahmani and colleagues (2016), compared the levels of the presence of heavy metals in their subjects with that of the controls and had similar findings. Children with autism had high levels of heavy metals in their hair. These increased levels are positively associated with certain risk

factors such as heavy fish being consumed when the mothers are pregnant, smoking of pregnant mothers, and using anti-D and aluminum utensils. Nonetheless, these higher levels could not be related to autism seriousness. Heavy metals that find their way in the system of the mother, and eventually to the fetus or the infant cause biological damage that play a causal role in aggravating autism (Jain et al., 2015). The genetic vulnerability of children developing the condition of autism implies that these victims have a low ability to eliminate heavy metals or higher environmental exposures at fundamental periods of their development. Therefore, the traces of autism disorders in their system aggravate to higher levels.

It has also been suggested that some instances of ASD can be linked to maternal exposure to some viruses and bacteria during the process of vaccination. During vaccination against measles, rubella, herpes, syphilis, toxoplasmosis and chemicals like thalidomide as well as valproic acid, the mother and the unborn inside her is exposed to the causal agents of ASD (Oberlander & Zwaigenbaum, 2017). Nonetheless, these could be attributed to a small percentage of all the scenarios. Evidence reveals that perseverative thimerosal used in childhood vaccines could be associated with acquiring this causal agent to ASD (Christensen et al., 2018). Again, these have not been widely researched and are subject to variation as other findings may bring up new insights into the disorder.

It is broadly acknowledged that atypical brain development motivates the growth of the symptoms of ASD seen, that is, the initial signs of autism. There is much debate on exactly how genes and the environment engage to trigger the start of awkward brain development. However, it is confirmed that such brain development disparities could be linked to the birth of autism or very soon following birth, although the behavioral and social indications of autism are not seen until after 1.5 years of life (Bahmani et al., 2016). The precise composition of the brain

variations is not evident either. Scholars have displayed distinctive growth in several brain areas such as the frontal and temporal lobes, the cerebellum, and the sub-cortical amygdala as well as the hippocampus. Less evidence, methodological trials, and contradicting discoveries have not given chance for accurate inferences concerning either the particular brain parts affected or the framework of development that amounts to observed brain disparities to be made (Christensen et al., 2018). There are also suggestions on the paradigms of connectivity between and within brain regions as the main problem instead of the particular loci whereas other researchers concentrate on particular kinds of neurons or paradigms of neuronal activation.

It is significant to recognize that from the initial years of life through the lifetime, children with ASD depict a great scenario of heterogeneity as far as its severity, beginning, progress, and pattern of ASD symptoms are demonstrated: cognitive, linguistic as well as diminished communications; social-emotional, behavioral, and regulatory challenges; and sensory feelings. Matching with their heterogeneity, there is variety in the early development of the disease (Oberlander & Zwaigenbaum, 2017). In early development, it is usually the nonexistence or shortfall of typical social development indications that reveal the start of the ASD diagnosis instead of the existence of atypical actions (Bahmani et al., 2016). Nonetheless, some children do display atypical actions like repetitive and inflexible play, strange body poses, and motor movements such as rocking, hand flapping, flipping the fingers, or visually checking objects at uncommon angles, out of the edge of their eyes, and for an extended time.

It is significant to utilize assessments that advocate for good social behaviors like social smiling, taking pleasure in simple reciprocating activities such as throwing a ball up and down, and shared attention behaviors like following a guardian or any speaker's gaze. They could also be involved in proto-declarative pointing in which the attention of the child is taken to an object

that is at some distances. This gaze can be combined with producing a voice or showing a gesture to request something (Oberlander & Zwaigenbaum, 2017). The child could also be shown an object that he might be interested in by raising it up, vocalizing and examining the parent or the physician examining the child. The point is creating a platform that will direct diverse sets of facial expressions to the child suffering from autism.

In conclusion, many studies carried out have suspected several aspects to the causes of ASD. Maybe, the scholars that allude to genetic links may be close to determining the source of ASDs. The observation of an identical twin with one of them being diagnosed with autism, and the other having traces and higher chances of developing the disorder is largely valid. It shows some genetic elements play a significant role in terms of causing ASD in children. The supposition that environmental interactions cause pregnant mothers to consume heavy metals into their systems cannot be neglected. These heavy metals in some way are transferred to unborn babies so that they are born with autism disorder. Tests on the hairs of these children diagnosed with ASD have revealed a high presence of these heavy metals. Such suppositions still leave us with a doubt that the precise causes of autism have not yet been established in a comprehensive manner.

References

Bahmani, M., Sarrafchi, A., Shirzad, H., & Rafieian-Kopaei, M. (2016). Autism:

Pathophysiology and promising herbal remedies. *Current pharmaceutical design*, 22(3), 277-285.

Boukhris, T., Sheehy, O., Mottron, L., & Bérard, A. (2016). Antidepressant use during pregnancy and the risk of autism spectrum disorder in children. *JAMA pediatrics*, 170(2), 117-124.

Christensen, D. L., Braun, K. V. N., Baio, J., Bilder, D., Charles, J., Constantino, J. N., & Lee, L. C. (2018). Prevalence and characteristics of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *MMWR Surveillance Summaries*, 65(13), 1.

Jain, A., Marshall, J., Buikema, A., Bancroft, T., Kelly, J. P., & Newschaffer, C. J. (2015).

Autism occurrence by MMR vaccine status among US children with older siblings with and without autism. *Jama*, 313(15), 1534-1540.

Oberlander, T. F., & Zwaigenbaum, L. (2017). Disentangling maternal depression and antidepressant use during pregnancy as risks for autism in children. *Jama*, 317(15), 1533-1534.