2010

An Examination of Autism Spectrum Disorders in Relation to Human Evolution and Life History Theory

Daniel E. Lomelin
delomelin@gmail.com

Follow this and additional works at: http://digitalcommons.unl.edu/nebanthro

Part of the Anthropology Commons

An Examination of Autism Spectrum Disorders in Relation to Human Evolution and Life History Theory

Daniel E. Lomelin

Abstract: Autism and Autism Spectrum Disorders are multifaceted conditions that are being diagnosed increasingly in Western nations. Current research suggests genetic and epigenetic effects as well as social and environmental conditions all playing direct roles in the expression and development of these disorders. Evaluating the development of the human brain's increased intelligence and plasticity in terms of human life history and evolutionary trade-offs allows for new hypotheses to be formed regarding the development of the disorders. Aspects of the broad autism phenotype may have given ancient humans advantages in tool making and mechanical thinking, thus preserving the trait in various cultures to the modern day.

Introduction

Autism is a multifaceted disorder, the study of which has gained considerable momentum in medical research, but its root cause is still unknown. There is a substantial lack of cross-cultural research into the prevalence of autism as well as Autism Spectrum Disorders (ASD) that is necessary to establish an environmental cause of the disorder. Additionally, the rise in cases of autism recorded seems to counter a classical genetic inheritance mode. Given human life history, milestones in human brain development are important in the context of achieving reproductive success, and as such ASDs are likely under great negative selective pressure. Extended brain growth and time spent in juvenile form, along with general neoteny or the prevalence of immature traits, while allowing for longer accumulation of knowledge and social habits, also leaves a larger time frame for damage to occur during key phases of development. Evolutionarily, the advantages of a well established knowledge base and social network would have strongly outweighed a relatively uncommon vulnerability. Additionally, it seems possible that the prevalence of ASDs in Western nations may be linked to factors contributing to other human-specific shifts in
developmental timing. The diversity present in autism, as in all human cultural and biological variation, lends itself well to an anthropologic analysis and style of research.

Autism and related disorders are characterized by an individual’s lack of ability to relate to others, stunted communication skills, and a decreased ability to comprehend common symbolistic information. Language as a whole is often hampered as well. Physiologically, large brain structure changes as well as more subtle brain alterations occur around the age of disorder onset, which is typically around two to four years old. Some studies have found abnormal growth in total cranial volume during these ages; however, the initial growth in volume becomes normalized compared to the rest of the body from four to five years of age. Increased growth in white matter, particularly towards the anterior of the brain, has been reported to be the cause of the observed larger cranial volume. Nevertheless, more white matter does not necessarily imply more connections within the brain; a lack of effective communication between brain sections is known to be present in patients with autism (Williams 2007).

The evolution of the brain in relation to human neoteny is important for understanding and evaluating autism. Many of the traits associated with childhood in humans, such as social dependence and curiosity, are lacking in ASD individuals (Brune 2000). It has been suggested that due to the extended juvenile period, as well as due to delayed maturation in humans, that our minds are much more vulnerable to social and environmental stress. Studies looking at monozygotic twins with autism found that the trait was strongly associated with these more closely related individuals than in fraternal twins. Additionally, the Broad Autism Phenotype (BAP) of traits that seem to resemble very mild versions of ASDs are seen in higher frequency among related individuals, parents as well as siblings (Losh et al. 2009). Mothers, in particular, with BAP traits are most closely associated with having children who are autistic. Due to the lack of clearly defined genetic evidence, the importance of parental behavior as a social and learning environment may be a factor here. It seems probable that, given the historical lead role mothers have played in tending young children, women who exhibit the BAP would, by convention, be less sociable and sterner, perhaps influencing the development of a child in that trajectory.

Habitat complexity is another potential avenue of research in terms of general childhood mental development that can be applied to autism research. While it is commonly known that an enriched environment is a good way to stimulate a child’s mind, it is due to mental plasticity that human beings can develop a degree of mental variation based on environment. In other animals, such as cichlid fish, there is a clear difference in brain development based on environmental complexity.
More complex environments, measured by number of hiding places, obstacles, and similar objects in the habitat, are correlated to better memory and more developed brains in fish and other, non-aquatic animals. Interestingly, when fish of a less complex environment, in this case a sandy one, are transferred to a more complex one their brains develop additional complexity and are better suited to the enriched environment (Shumway 2008). Similarly, in autism the plasticity of the human brain is evident. When functional scans of the brains of autistic individuals are monitored, alternative pathways are utilized to process information with subject output equaling that of normally developed subjects (Williams 2007). Similar to the fish environment study, placing autistic children in specially enriched environments with high social interaction can take advantage of our longer period of brain growth and the plasticity of the developing mind to achieve new pathways that lead to better developed social skills. Unfortunately identification of faces and relating to them is a trait that develops early on, in infancy, and would be difficult to treat at first onset due to lack of means of diagnosis.

In research conducted on the differing mental abilities of siblings of autistic individuals a statistically significant number of the subjects were found to have lower than average intelligence quotients. Additionally, in social-emotional functioning, the siblings were significantly less adept than average (Toth et al. 2007). The fact that family stress levels and general social environments are controlled for lends credence to autism having a biological origin. Specifically, the fact that siblings without autism share a degree of autism related traits points to either a similar epigenetic effect having occurred during gestation; or perhaps a polygenetic origin, where some alleles that lend to autistic features have been inherited, just not to the same extent as the completely affected individuals. It is also important, as the authors of the research noted, that it is likely a combination of effects; quality of interaction may be altered and put predisposed children at higher risk for impairment (Toth et al. 2007). It is in such observation that non-monozygotic siblings and their degree of affectedness by the autism phenotype indicate the potential for both cultural and evolutionary forces shaping the disorder’s expression.

An additional, environmental, aspect of research in autism is in diet and nutrition. It has been advocated by some that a shift in diet may alleviate some aspects of ASDs but such direct evidence is not common. However, metabolites have been shown to differ in autistic individuals from their normal levels. While these differences may be due to another effect occurring in the body, deficits were investigated and corrected using nutritional supplementation in one study (James et al. 2004). It was also found that there was a lack of antioxidant activity in the same individuals. Being that natural foods, especially berries,
which no doubt were a large part of hunter-gatherer and even prehuman diet, are naturally high in antioxidants there may be an additional nutritional factor in ASDs. While such deficiencies are more likely symptoms of a root issue, looking into the cultural diet of an individual’s ancestors may give clues as to what sorts of foods may aid or exacerbate metabolic deficiencies. Seeing as how ASDs would be strongly deleterious in pair-bonding and other behaviors required for reproduction, classical cultural practices may have lessened the disorders’ impact.

Cross cultural research into Nigerian hospitals and primary to tertiary care health workers reveal that only more specialized and experienced workers have knowledge about autism at a proficient level. A biomedical culture approach to autism in these nations is seen by diversification of health professionals into subfields that deal with issues such as speech and special education (Bakare et al. 2009). The subdivision of medical fields and the expending of large sums of money needed to afford specialists, their training, and facilities is counter intuitive in a part of the world where the exact rate of these disorders is unknown and where most health care workers lack experience outside of what they have been taught. The biomedical approach to autism treatment, and subsequent diagnosis by said professionals does not fit with practicality in these cases. Part of the challenge in giving out help, stated the health workers in Nigeria, is that money, facilities, and a variety of cultural groups make it difficult to manage all health needs in a unified manner. Research into ASD is strongly needed in developing nations, but the current conditions do not lend themselves to doing so effectively. While culturally relative treatment would be useful when combined with the scientific knowledge of the biomedical system, autism is recognized as being present in at least 80 countries, with many groups having recognized a difference and given names for children who fit the autistic profile (Daley 2002). Perhaps normalization of the disorder as a common variation in humans and its acceptance by these cultures falls in line with current research showing that special attention can increase functionality of ASD individuals. In some cultures high-function is not desired, as people with the disorder are believed to be closer to the spiritual world (Daley 2002). It is interesting to note that in India a wide variety of beliefs about psychology, biology, religion, and medical systems create an extensive gamut of treatment options when families have determined that their child is exhibiting less than optimal social skills. Since cross-culturally, autism is recognized in one form or another, there is definite evidence of biological processes at work. However, true prevalence rates of the disorder are hard to come by and make cross comparison difficult when looking at how ASDs are treated, as well as what environment they may manifest most frequently in.
Environmental interaction with the development of these affected children is a particularly intensive area of research in autism. The effect of human-utilized chemicals in the environment of developed nations is also hypothesized to be causing an increase in autism cases. In a study done in California, data from pesticide concentrations in homes near farms were compared to rates of autism (Roberts et al. 2007). Timing of the pesticide treatments and of the pregnancies were also available for analysis. The results were substantial. Organochlorines were found to have a significant effect on the probability of a child having autism. The strength of the effect was based on timing, as well as on distance from the spraying. The association was also particularly strong when exposure lined up with central nervous system embryogenesis; both cord blood and placenta are known to contain the remnants of organochlorines that have been somewhat broken down in the body (Roberts et al. 2007). The implications of this information are tremendous. Being that autism has been shown likely to contain a genetic component, the effect of such environmental toxins may have multigenerational epigenetic effects. As other epigenetic effects concerning fetal programming of nutritionally related health have evidence for an intergenerational effect, we may be seeing something similar with ASDs. Substantiating the environmental toxin results can be accomplished by further study of similar data in other agricultural communities, but in establishing the role of environmental pollution as a substantial culprit, cross cultural research must be done in groups that have very little to no exposure to the chemicals.

Other environmental pollutants such as estradiol, an estrogen hormone, directly impact neurologic development; even early gene expression has been linked to processes directed by the hormone (McCarthy 2008). Estradiol is known to play an important part in determining sex differences in the brain, including how neurons are interconnected as well as how they signal. Immature neurons are somewhat buffered from the effects of estradiol, potentially to shield them until pubertal effects in the rest of the body have been achieved. Interestingly, as autism affects males disproportionately more and increased white matter is an aspect of the male brain, it would seem there is a potential link to the noted increased white matter volume in the brain of young autistic individuals and such compounds. It seems possible that some of the environmental pollutants that are, or mimic, hormones in the human body may be interacting with susceptible genotypes to account for some aspects of autism. Conceivably, the mechanisms by which estradiol, among others, are buffered from affecting the immature brain are unable to prevent an environmental source from causing harm due to early exposure. Also, since the female
human body plan is the default if the *SRY* gene responsible for male fetal development is somehow impaired, in conjunction with the differential prevalence of cases of ASD in males, it seems that perhaps there is an epigenetic effect that affects the Y chromosome or the *SRY* gene's expression. Following an epigenetic root cause of autism, a study of *MECP2* gene promoter methylation found that in males, protein expression of that particular gene was significantly reduced. The effect was also seen in female individuals but to a lesser extent. As *MECP2* is located on an X chromosome, the differential effects of autism on males in comparison to females may fit into the sex-linked model of autism-inherited susceptibility (Nagarajan et al. 2008).

Discussion

With all the potential causes or exacerbating factors of autism, is there an adaptive benefit to autism and ASD? Part of the autism phenotype is that individuals commonly desire repetition and predictability in their daily routine and activities (Baron-Cohen 2004). Additionally, the objects that are often most fixed upon are mechanical in nature. Similarly, cognition tests of autistic individuals have revealed an increased attention to detail. In visual tests, such as the embedded figure task where an individual must find a shape immersed in a mixture of other forms, autistic individuals and their family members perform exceptionally well. Interestingly, it has also been shown that autistic individuals often have family members that are disproportionately involved in engineering and similar academic fields. This observation is valid not only among parents but extends up to grandparents as well; in all, it was found that 28.4% of autistic individuals had an engineer as a father or grandfather in comparison to the control group, with only 15% (Baron-Cohen et al. 1998).

The evolutionary advantages of having autism-like traits are widespread. Given that the hunter-gatherer lifestyle is commonly agreed to have existed for the majority of the time *Homo sapiens* have lived on earth, the establishment of predictable patterns of game movement as well as understanding where to safely go during various times of the day to gather foods or get water would have been useful. More specific to ASD symptoms, an understanding of mechanical systems and attention to fine details in their creation is extremely important. Human-made tools such as the delicately crafted clovis points, with their fine shape and bifacial flaking, are a testament to the understanding our ancestors possessed of the properties of the materials they worked with. Similarly, projectile tools such as the atlatl used mechanical leverage to maximize force and distance in the use of spears. There is no doubt that dwelling and tool engineering done by our ancestors was key to their survival and success.
In terms of the diagnosis of autism and similar phenotypes, identification of these conditions has risen largely in the last two decades. Research points to the current “rise” of ASD being closely linked to changes in the medical literature, specifically the *Diagnostic and Statistical Manual (DSM)* from the *DSM-III* to the *DSM-IV*, as well as changing public awareness and procedures for diagnosis (Nassar et al. 2009). In many instances intellectual disability (ID) is present along with autism, but when comparing rates of ID with autism and ASD the data strongly suggests that affected individuals are shifting into the autism category rather than ID when both symptoms are present. These findings are helpful in establishing the evolutionary background of autism, as the phenotype being stable cross-culturally and over time adds evidence for its existence in an ancient past. As described earlier, maintaining traits of autism likely gave our ancestors an analytical advantage when genetics has allowed partial expression of the ASD traits. Strongly rising rates due to an environmental cause would run counter to this model, but this does not seem to be the case.

Conclusion

In closing, autism is a disorder that has only recently been given an official diagnostic criteria, which in itself has changed drastically and has many currently contributing factors, including social, nutritional, and intrauterine environments, as well as a genetic history. Human evolution may even have allowed for autism as a slight trade off for other traits that are seen as highly advantageous, seen in human behavioral neoteny. Additionally, because the disorder is seen as a spectrum of phenotypes, a multitude of genes as well as the aforementioned factors all contribute to the mix of individuals who have autism. Likewise, treatment of autism must take into account the multifaceted nature of human growth and development. Treatments will need to be tailored to individuals based on their largest contributing factors; even in cases where treatments, behavioral, nutritional or chemical, can be used, the underlying mechanisms by which autism works are still poorly understood. Cross-cultural research in developing nations around the world in different ecologic and social environments will likely shed light into the disorder, and may provide evidence for its biologic advantages. Autism may possibly reveal itself to be the result of a balanced polymorphism, like sickle cell anemia, that is advantageous in a certain mixture of genes and disadvantageous in specific combinations, with the traits selected for helping our ancestors construct their way to environmental dominance. Additionally, it may be that while individuals with ASDs are present in other countries they are much lower in density or perhaps treatment of these individuals is handled by cultural practices that better enable the affected to lead
normal lives. In these instances, investigation into family occupational history, particularly craftsmen and craftswomen, will provide a better understanding of how and why autism exists today.

Acknowledgements
My utmost thanks goes to Dr. Osborne who encouraged the submission of this paper along with having given advice while on such a tight schedule.

References Cited


McCarthy, Margaret M.  

Nassar, Natasha, Glenys Dixon, Jenny Bourke, Carol Bower, Emma Glasson, Nick de Klerk, Helen Leonard.  


Roberts, Eric M., Paul B. English, Judith K. Grether, Gayle C. Windham, Lucia Somberg, Craig Wolff  

Shumway, Caroly A.  

Toth, Karen, Geraldine Dawson, Andrew N. Meltzoff, Jessica Greenson, Deborah Fein  

Williams, Diane  