Review

Autism, genetics, and inbreeding: An evolutionary view

Alex S. Prayson
National Council on Rehabilitation Education

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Recently there have been increased reports of autism, yet the disease is not contagious. Since it is not catching, there must be other forces at work that somehow create or pass on the autistic symptoms. DNA reports show that deviations in the genetic code due to ancient inbreeding can follow a human line for generations. Studies show that inbreeding was widespread until a few hundred years ago and is continued today, but to a lesser degree. After millions of inbreeds, the world population has become so numerous that it is globally sharing ancestors which is producing genetic abnormalities. In other words, autism may be the result of the widespread inbreeding of ancient generations. We are all touched by autism to one degree or another through common ancestors. The DNA of modern Homo sapiens of European and/or Asian descent will show 1 to 4% Neanderthal from 40,000 years ago. With that in mind, today’s outbreaks may be due to descendants of ancient inbreeding times surfacing at the same time.

Key words: Autism, genetics, inbreeding, DNA, ancient generations, consanguineous marriage, incest

INTRODUCTION

The world might be smaller than you think

Currently there are increased reports of autism, yet the disease is not contagious. Since it is not catching, other forces must somehow create or transmit the autistic symptoms. Studies indicate that inbreeding will eventually produce autistic symptoms. The one commonality is our ancestors and after millions of inbreeds, the world population has become so numerous that it is now globally sharing ancestors which is producing genetic abnormalities. Autism may be the result of worldwide inbreeding of ancient generations. DNA reports show that once a human’s DNA is altered, it will stay altered for generations. A consanguineous marriage does not produce birth defects (Leavitt, 2003), but it increases the chances of inheriting a bad DNA fit which results in a birth defect. Inbred disorders may cause other abnormalities and autism can also be brought on by other conditions, but the focus of this paper is how autism might be related to huge generations of past world inbreeding.

An estimated number of people on earth by the U.S. Census Bureau, U.S. and World Population Clock (Ross, 2015):

- 1AD – 300 million
- 1250 – 400 million
- 1500 – 500 million
- 1804 – 1 billion
- 1927 – 2 billion

E-mail: aprayson@ymail.com.

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A BRIEF HISTORY OF CONTEMPORARY AUTISM

Earliest autism diagnosis

From the early 1940s until the 1960s, Dr. Leo Kanner's premise, although incorrect, was that autism is caused by child neglect, withdrawal of affection, and in general, poor parenting. In his 1943 paper, (Kanner, 1943) Kanner called attention to what he saw as a lack of parental warmth and attachment to their autistic children. In his 1949 paper, he attributed autism to a "genuine lack of maternal warmth" which gave birth to the "Refrigerator Mother" theory. In a 1960 Time Magazine interview with Kanner who described mothers of autistic children as "just happening to defrost enough to produce a child."

In 1964, Bernard Rimland, a psychologist and the father of an autistic child, wrote a book debunking the refrigerator mother hypothesis. He became the spokesman for parents of autistic children and helped found the Autism Society of America (Laidler, 2004).

In the 1970s, researchers determined that autism is not the result of emotional abuse. Abused or neglected children may display similar behavioral problems, but these problems are distinct from autism disorders.

Between 1993 and 2003, American school children diagnosed with autism increased over 800% by some researchers. The medical world is uncertain if this rise is an actual increase in autistic children or the number of children diagnosed with autism. In 2006, the Center of Disease Control and Prevention (CDC) noted a slight leveling in the number of cases reported indicating autism may have been over diagnosed (Rudy, 2013).

Changes in diagnostic criteria

In 1991, an Autism Diagnostic Interview (ADI) first published an accepted way to identify autism. By 1992, the American Psychiatric Association (APA) printed the Diagnostic and Statistical Manual, DSM-IV. In 2014, the APA's DSM-5 revised edition, the committee redefined autism to more accurately reflect clinical cases of autism and help doctors make more accurate diagnoses (Sifferlin, 2014).

Fraudulent autism study

In 1998, Andrew Wakefield, MD published his infamous study linking autism to childhood measles, mumps, and rubella (MMR) vaccines. His study misrepresented the records used by altering the medical histories of all 12 patients to make it appear as though vaccines were the source of their autism (CNN, 2011).

Pediatrician Dr. Laurel Schultz (Howard, 2015) of San Francisco writes, “Children are exposed to more antigens in our day to day environment than are found in all the vaccinations combined.” Vaccine experts at CDC (CDC, 2015) and the American Academy of Pediatrics (AAP), agree that MMR vaccines are not responsible for the recent swell in the number of children diagnosed with autism (Magliaro, 2015).

Scientists have grappled with the sudden increase in autism reports and a recent study now indicates it is not an epidemic at all. In Sweden, over a million children were studied for a period of 10 years, from 1993 to 2002, the number of autism spectrum disorder diagnoses increased significantly, as in the United States, but the number of patients who actually showed symptoms remained fairly constant. It is highly possible that doctors are over misdiagnosing autism (MacDonald, 2015).

CHROMOSOME ANOMALIES MAY ELICIT AUTISM

The genetic blueprint of life

Chromosomes are structures within the nucleus of cells that comprise the genetic blueprint of life. This inherited material is deoxyribonucleic acid (DNA) which contains noticeable sub-units known as genes. A normal cell has 46 chromosomes, 23 from the father's sperm and 23 from the mother's egg. Here is where complications may arise. If the father has the same exact flawed DNA as the mother, they may reproduce a child with issues.

According to the National Institute of Health (NIH), abnormal chromosomes can be divided into two basic groups: (1) numerical abnormalities, when an individual is missing a chromosome from a pair or has more than two of a pair. A numerical abnormality is Down syndrome which has three copies of chromosome 21. (2) Structural abnormalities are when the chromosome itself is altered, such as deletions, duplications, inversions, rings, and translocations. Chromosomes 5, 15, and 16 are suspected participants in autism disorders (NIH, 2014).

ANCESTRAL INBREEDING MAY AFFECT CHROMOSOMES THAT CAUSE AUTISM TODAY

Centuries of inbreeding

Early hominids were not fussy about their sexual partners. Homo sapiens would interbreed with whichever hominid species was nearby, such as the Neanderthals and the mysterious Denisovans (Marshall, 2013). The Neanderthals appeared for a brief time, but eventually
died out from inbreeding causing a reduction in population, thus creating more inbreeding cycle. They did, however, leave a small percent of their DNA in the Homo sapiens’ chromosome history. Of course, if the first Homo sapiens had not interbred, the human race, as we know it, may have become extinct (Estes, 2011).

The mechanisms of evolution may be leading to global inbreeding. After generations of consanguineous marriages, it could be that the world’s population is so large that it is starting to inbreed with itself. It is becoming more and more difficult finding a mate who does not share a common ancestor of some sort in our contemporary chromosomes. We have all probably inherited genetic changes that were not as common a century ago. For instance, the average height for a human has increased about three inches since the 1700s. These are forced general population mutations due to an improved life style, but even good changes require genetic intervention (Inglis-Arkell, 2012).

**Possible inbred mutations causing autism**

Both autism and inbred disorders may have similar abnormalities of the brain structure and/or function. Brain scans of these children show variances in the shape and structure of the brain when compared with the neurotypical or normal brain found in children. Researchers (Wahl, 2014) are exploring a number of theories that led to autism, including links to heredity and genetics.

Inbreeding is considered a problem in humans, because it heightens the chances of receiving a damaged chromosome inherited from a common ancestor (Ochap, 2004). Interbreeding increases the probability of a child being born with a double dosage of one or more recessive genetic problems that can cause congenital birth defects.

**40,000 to 30,000 years ago**

Autism could be the result of a slight gene mutation inherited thousands of years ago. Skeletal remains found in Northern Italy are from 40,000 to 30,000 years old and “believed to be that of a human/Neanderthal hybrid,” according to a paper in PLOS/ONE (Condemi et al., 2013). If this is correct, it is direct evidence that Homo sapiens interbred with Neanderthals. Modern genetic research can determine, after thousands of years, that the DNA of people with European or Asian ancestry are 1 to 4% Neanderthal (Viegas, 2010).

There could be inbreeding disorders found in every human’s DNA. Most have no effect until matched up with the same mutant gene, locus or position on paired chromosomes through inbreeding or happenstance. These abnormal alleles (Alleles are pairs or series of genes on a chromosome that determine the hereditary characteristics, Merriam-Webster's Medical Dictionary) create subtle refined autism symptoms similar to those found in affected consanguineous off springs.

**ACTUAL STUDIES SHOWING CONNECTION BETWEEN INBREEDING AND AUTISM**

Roughly half of the people who live in Arab countries are inbred. A large percentage of the parents who are blood related come from families where intermarriage has been a tradition for generations (Cook, 2013). In ancient generations, “Pharaohs often married their own sister or half-sister and after a handful of generations the off springs were mentally and physically unfit to rule” (Sennels, 2010).

Two researchers, Walsh and Morrow, recently studied 104 families (Walsh, 2010) from the Arab Middle East, Turkey, and Pakistan. They found that of the 104 parents, “in 88, the parents were cousins. The average family had two autistic children. One Kuwaiti and one Pakistani family, however, each had four.”

“Marriage between first cousins doubles the risk of neurological birth defects. Researchers now think that shared ancestry can increase the risk of autism produced by recessive mutations that cause problems only when a child inherits the same defective gene from both parents” (Sennels, 2010). In this analysis of 104 families, approximately 97% had heredity problems.

**From as far back**

It is conceivable that autism symptoms stem from ancient ancestors from as far back as the migration out of Africa and passed down through the ages randomly. These genetic anomalies may combine with an existing abnormal gene that is inactive or dormant. The combination could produce various intellectual and formational issues spread by the increased chances, in an increasing population. Since autism is considered a social learning type of disorder, it seems to confirm that autism depression follows the same depression steps as inbreeding depression. Genetic studies suggest that inbreeding depression is mainly caused by the increased presence of recessive deleterious mutations found in our modern populations (Charlesworth and Willis, 2009).

**Modern human migrations**

There were groups of early humans who traversed Africa and went westward across Europe until they ultimately reached the Atlantic Ocean thousands of years later. Explorers eventually took to the seas and sailed to North America and other land masses. They encountered their distant cousins from Asia who ventured eastward crossing Asia and spanned the Bering Straits when it was still a land bridge about 30,000 to 18,000 years ago.
These sea voyagers sailed across the Atlantic Ocean carrying with them whatever disorders and diseases they had developed in Europe plus any gene changes they may have inherited. As a small example, in 1620, there were 102 English Puritans who landed in Plymouth, MA and many colonized that area by intermarriages because of their small population. Half of these Puritans did not survive the first harsh winter. Today, fifteen generations later, 35 million people claim an ancestral lineage all the way back to the original 24 males. This is about 12 percent of the American population (Galluzzo, 2004).

GENETIC DEFECTS IN CHROMOSOME 16

Researchers find not all mutations are inherited

By applying a very large genome scan, which is a complete set of DNA, researchers have discovered that the genetic abnormalities of a section of chromosome 16, can develop spontaneously while in the embryonic stage of development and not necessarily inherited directly from either parent. A culmination of genetic disorders may enhance or increase the combination so they affect one or more genes. This is an additional method for brain disorders to develop. This shows that some mutations are possible just by the interaction of various other genetic factors inherited from each parent (Amst, 2008).

CONCLUSION AND FUTURE OUTLOOK

Scientists have discovered the strongest link yet between specific chromosomal abnormalities and an increased risk of autism. New studies of families carrying glitches in the region of chromosome 16 have been strongly associated with this condition. Disrupting even a small piece of that region, 16p11.2, can sometimes cause autism. Research has identified numerous reputed areas on specific genes located in this chromosomal region that may play a part in instigating autism (Gusella, 2008). The new chromosome micro-array tests can now predict the probability of recurrence in future pregnancies (Hughes, 2011).

Science has made advances in genetic improvements that will someday reduce or eliminate autism. Progress in discovering which genetic components are involved and the genes that affect environment and learning is a large improvement since these are the most likely genes used when Homo sapiens took their first steps. This also aids in the theory that globally shared ancestors producing genetic abnormalities as a result of widespread inbreeding of ancient generations.

Studies by Guoping Feng, an MIT professor of brain and cognitive sciences, indicates that approximately 1 percent of autistic people are missing a gene called Shank3. Those without the Shank 3 gene develop typical autism symptoms such as repetitive behavior and avoidance of social interactions. Feng claims that some of the brain defects are reversible and treatment for autism will be developed in the near future (Trafton, 2016).

Conflict of Interests

The author has not declared any conflict of interests.

REFERENCES


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