
After a child is diagnosed with autism spectrum disorder (ASD), parents often wonder if any future children they might have would be at risk of developing autism.

If the cause of autism were a singular gene variant, this question would be easy to answer. Unfortunately, the etiology of autism is more complex. Research has shown that hundreds of genes are involved with ASD, and that the cause is likely a complex combination of environmental and genetic factors.\(^1\)\(^2\)

With this in mind, researchers have sought to uncover indications that might help parents know if they have an increased risk of having a child with autism. For example, if ADHD, ASD, Tourette syndrome, bipolar disorder and/or depression are conditions commonly seen in your immediate family, the chances are greater of your child having autism.\(^3\)

Researchers have also shown that the age of the father plays a part in autism risk. ASD risk in a child increases by 28 percent for fathers aged 40 to 49 and climbs to 70 percent for fathers over the age of 50.\(^4\)

A group of researchers from the Health Research Institute La Fe (Valencia, Spain) sought to investigate this dramatic increase in risk with paternal age.\(^5\)

As our body ages, environmental and lifestyle influences can cause certain genes to be turned on or off. This process is known as DNA methylation and occurs when a methyl group attaches to the DNA strand, preventing or altering its function. While these changes do not alter a person’s actual DNA, they can alter how the gene is expressed. These changes, known as epigenetic changes, can be detected with methylation markers.

Past research has shown that as men get older, there is an increase of epigenetic DNA methylation alterations in their sperm.\(^4\) In addition, the children of older men are at a higher risk of developing autism. The researchers sought to determine if there is a relationship between the two phenomenons. **Can the DNA methylation alterations in a man’s sperm indicate a likelihood that his offspring will develop autism?**

**Study**

The study, directed by Dr. Michael Skinner (Washington State University), involved collecting sperm samples from 13 fathers with children with autism and 13 fathers of children without autism. The sperm was assessed for alterations in DNA methylation and then assessed for differential DNA methylation regions (DMRs). The researchers compared the alterations and the DMRs between the two groups.
Results
The analysis on the sperm from fathers with ASD children revealed 805 specific regions of DNA methylation (DMRs) that were different from the fathers without ASD children.

Following the analysis, a group of men volunteered for a series of blind tests. By analyzing the 805 DMRs, the researchers correctly identified if fathers had a child with autism with 90 percent accuracy.

The genes associated with many of the 805 DMRs were known to be associated with previously identified genes that pose an autism risk.

Conclusions
This study has important implications. Although the sample size was small, the results show the value of investigating sperm to determine the susceptibility to autism of offspring. In the blind tests, the researchers could identify most fathers who had offspring with autism. Could such testing be put in place in doctor’s offices for potential fathers? If doctors are able to identify high-risk children through paternal sperm sampling, interventions could be initiated earlier in a child’s life. Consideration should also be given to the ethical implications of such testing.

Just as important is knowing why these epigenetic changes are occurring. Is it possible to identify the environmental factors causing these changes? Could identifying and eliminating the environmental triggers affect the incidence of autism? It has been suggested that such factors could include exposure to toxic substances, unhealthy nutrition, and even stress. As with most research, many questions remain. However, this is potentially the first step in a global change in the way we assess autism risk for children.

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References

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