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Hope for Autism

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Introduction

We all love nothing more than our children, and the thought of having an autistic child is frightening. Autism's behavioral, social, verbal and non-verbal disturbances affect children in major ways [1].

Autism is a mysterious disorder; there is little understanding of its cause [2].

What causes Autism? A short time ago, the only sentence that we can say "we have no idea." Fortunately, the researchers are recently starting to deliver the answers. We now understand that there isn't a single cause for autism, we also know that there are multiple types of autism [2].

Recently, researchers have reported a scale of gene polymorphisms linked to autism. However, only some of these polymorphisms are capable of causing autism directly. Both of gene polymorphisms and environmental factors affecting brain growth appear to be the cause of major cases of autism [3].

If the genetic risk factor for autism is present, a number of non-genetic, or "environmental," factors are also having a role in increasing a child's chance for becoming autistic. The risk factors for autism include what happened before and through birth, for example old parental age at time of pregnancy - both mother and father, not just the mother; maternal diseases; and some complications during birth, especially fetus' brain oxygen deprivation [3,4].

It is very important note that autism risk factors do not cause autism by themselves. But, in addition to genetic factors, they can increase the risk [4].

Recent growing research reports that a mother can decrease her risk of delivering an autistic baby by taking folic acid tablets and/or eating meals rich in folic acid (500 mcg per day) during the months before and after pregnancy [5].

Recently, researchers have reported greater than thousand different gene polymorphisms in children with autism, but they did not reach how these polymorphisms can increase the risk of autism [6-9]. Researchers of UNC School of Medicine demonstrated how one of these polymorphisms affects a molecular regulation in one of these genes and so causes autism [10].

The research reported that an enzyme named UBE3A can be switched off when a phosphate is added onto it. During normal brain growth and in neurons in general, this switch can be turned on and off, prompting tight control of UBE3A. Ubiquitin ligase activity of UBE3A must be tightly regulated to promote normal brain growth. But the team of Dr. Zylka, PhD, associate professor of cell biology and physiology, reported that the UBE3A polymorphism cut down this regulatory switch. Cutting down of the switch, in turn, makes an enzyme without tight control. So, UBE3A becomes overactive leading to abnormal brain growth and autism [10].

In our work in the Clinical Pathology Department at Assiut University School of Medicine, Egypt, we have discovered that one specific type of UBE3A mutation - T485A - was found in 70% of the fifty studied Egyptian autistic children (of which 51.5% with Homozygous mutation and 48.5% with Heterozygous mutation).

Since this one single genetic mutation alone is linked to a majority of the autism cases studied, targeting it for treatment gives hope for a possible cure to multiple types of autism in children.

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