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Advancements in Genetic Engineering

Commentary

Significance of Epigenome Editing in Repairing of the Genetic Brain Disorder

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DESCRIPTION

Epigenetic editing processes are important in all kingdoms of life, and they include Deoxyribo nucleic acid (DNA), histone modifications, histone variants, and some non-coding Ribonucleic acid (RNAs). Gene mutations that cause brain disorders can be reversed by using a highly targeted epigenome editing method. This type of gene editing does not alter the DNA sequence of the gene but only corrects epigenome changes. The WAGR syndrome, which was corrected in the research, is linked with obesity and intellectual disability in people. Many brain development disorders, including autism spectrum disorder, manifest themselves in early infant life or later as a consequence of a poorly formed communication pathway between the two brain hemispheres. This is due to chromatin associated mechanism imperfections.

The gene which was corrected is known as *C11orf46*, and it is important for regulating brain development. It is in charge of producing a nuclear protein that regulates certain essential proteins capable of directing the newly formed long nerve fibres growing out of developing nerve cells in the correct orientation. These direction sensing proteins assist white matter fibres in bundling together to create the large nerve trunk that connects the two regions which is called corpus callosum, and if it is not correctly formed, the individual may become disabled and acquire autism, or another brain developmental disorder. Defects in the *C11orf46* gene have been related to corpus callosum hypoplasia or poor development.

When this gene was silenced, axon projections from one cerebral hemisphere to the other were disrupted. Multiple genes like *Semaphorin 6a* (SEMA6A) gene that code for certain essential events regulates the nerve fibre development and are overexpressed in these cells at the same time. This is normalized through epigenetic editing, resulting in normalization of gene expression and restoration of normal connectivity in the corpus callosum. The short, nerve fibres connecting the hemispheres respond to specific remodeling of neuronal genetic matter at specific sites.

The WAGR syndrome, also known as the chromosome IIp13 deletion syndrome, happens when part or all of the gene that forms the *C11orf46* in the eponymous chromosome region is accidentally left out during cell division. The absence of this protein product results in the severe disability that characterizes this condition. A gene-altering tool known as short hairpin RNA is used to reduce protein production in the mouse brain. As a consequence, nerve fibres in the developing brain were unable to form the corpus callosal bundle of white matter, resulting in a condition similar to WAGR. This demonstrates that *C11orf46* is involved in white matter connectivity, mediating the process through genes that help connect axons on either side of the corpus callosum.

In this condition, the brain overexpresses another gene that produces *Semaphorin 6a*, a direction-sensing protein, as well as many other proteins such as Doublecortin-like kinase 1 (Dclk1) and SLIT-ROBO Rho GTPase activating protein 3. (Srgap3). In a variety of aspects, this is a transcriptional regulator during the brain's development. *C11orf46* interacts with a different region known as the KMT-RC. Mutations in *C11orf46* prevent this complex from forming and thus prevent proper brain development. The researchers tested their hypothesis that *C11orf46's* affinity for this complex could be used to alter gene expression patterns.

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