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Commentary

CRISPR and the Brain: Current Challenges

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DESCRIPTION

From erasing the horrifying memories of rats to engineering, making decaffeinated coffee beans, the feats of CRISPR gene editing can often sound like science fiction novels. But this method does more than just produce delicious tomatoes and more eco-friendly fuels. CRISPR also offers potential applications in neuroscience-as a research tool and hopefully as a treatment for neurological disorders and disorders. The CRISPR- associated protein (CRISPR-Cas) gadget gives the capacity to substantially enhance primary and translational neuroscience studies. But, in phrases of making personalized brain remedy the usage of those gene enhancing tools, are we there yet? The molecular mechanisms of many brain functions are not well understood, and advances in next-generation sequencing (NGS) and CRISPR applications have filled some of these gaps. Recent research successes such as CRISPR-Gold's intracranial injection for the study of fragile X syndrome may contribute to the development of genetic condition-individualized, brain-targeted therapies that cause mild to moderate intellectual disability.

Current challenges for CRISPR research and neuroscience

Despite the promising research trajectory, scientists have found that the CRISPR system does not need to be easily translated when applied simultaneously to the DNA-cutting machinery, Cas enzyme, brain and psychiatric disorders. This is partly because stem cells (and the neurons that emerge from them for research) have a very active response to DNA damage; A DNA cut from Cas9 can also lead to toxicity, resulting in cell death. This indicates the importance of genetic engineering of complexes that can access the central nervous system cellular or otherwise with minimal potential damage.

Genetic control mechanisms are crucial for the successful coordination and functioning of the nervous system. This carefully initiated control system ensures all the necessary structural, physical and functional features of the neuron circuitry. The question of attempting brain-based therapy arises with any regulation of these carefully integrated processes. A major component of such cellular regulatory mechanisms is hereditary phenotypes that do not involve changes in epigenetics or DNA sequence. These affect how cells read genes and the mechanism by which they are turned on or off.

Whether the problem is from epigenetic patterns or transcriptional dysregulation, the resulting damage can affect neural plasticity (the brain's ability to change and adapt over a lifetime), changes in memory function and behavior. With this in mind, it is not difficult to predict what a misdiagnosed genetic expression would be for a variety of psychological and neurodegenerative conditions. Using CRISPR activation (CRISPRa), they were able to demonstrate the ability to control the endogenous ion channel *in vitro* and *in vivo* in mice. In other words, CRISPRa was able to drastically change the firing rate of neurons, revealing important aspects of neural network behavior in relation to epilepsy patterns. Such a treatment option would be realistic as a safe modification to neuron activity, without initiating the accumulation of harmful protein, toxicity, and cell death. Presumably, this type of genetic expression control can be used to treat other neurological diseases with altered transcription.

CRISPR reagents and the blood-brain barrier

The main problem that arises when designing personalized treatment for brain disorders is the ability to successfully pass through the blood-brain barrier (BBB). BBB works to isolate and protect nerve tissue and regulate the entry of molecules from the blood into the brain. By default, these functions interfere with nuclear-based treatment efforts. Blood-brain barrier (BBB) is related to a genetic modification that makes it difficult to obtain CRISPR reagents into the brain. So far, current research efforts for BBB methods include *in vitro* (static and dynamic), *in vivo* (microdiolysis) and *in situ* (brain perfusion). Nanoparticle delivery is an option currently being explored.

Parkinson's disease is one of the most well-documented treatment problems with the BBB. Patients with Parkinson's are usually treated with the dopamine replacement agent levodopa (L-DOPA), which can alleviate some of the symptoms of

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neurodegeneration for several years. Unfortunately, continuous treatment with synthetic dopamine, which is capable of crossing BBB, leads to changes in cellular transcriptional behavior over time. Because of this, and thanks to DNA methylation, prolonged use of L-DOPA can eventually lead to dyskinesias (involuntary, uncontrolled muscle movements) and adversely affect personality. These less-than-ideal results have been established by establishing the need for treatments that overcome the BBB problem without compromising the integrity of the nervous system, and by making constant changes in control.

Identifying the genes causing brain disorders such as schizophrenia, autism, and depression

For many common neurodegenerative diseases, sporadic factors are the most common cause. Most patients with Parkinson's do not have rare familial genetic mutations; instead, they have extreme or familial manifestations of the disease. However, they still have significant loss of extra incorrectly folded alpha-synclein protein and dopaminergic neurons, leading to the same manifestation of the disease with familial genetic bonds. This unsatisfactory reality has made it a constant challenge to successfully find a biomarker to test a person's risk. CRISPR is more useful for hereditary conditions derived from a dominant gene, such as Huntington's disease (HD), than for hereditary diseases. In theory, if the dominant gene of HD is successfully knocked out or does not work, the healthy gene will be read instead for reference.

CONCLUSION

With CRISPR technology, genetic modification is an excellent choice for the treatment of neurodegenerative, mental illnesses and possibly mental disorders. Perfection of this style of biological engineering would open the door to the elimination of abnormal genes, if only we could finally determine the specificity and accuracy required for complex nervous systems.

REFERENCES

1. Hadshiew IM. Burden of hair loss: Stress and the underestimated psychosocial impact of telogen effluvium and androgenetic alopecia. J Invest Dermatol. 2004; 123(3):455-457.
2. Prado RBR, Neme CMB. Emotive family-related experiences in women with alopecia areata. Estudos em psicologia. 2008; 25(4):487-497.
3. Taheri R, Behnam B, Tousi JA, Azizzade M, Sheikhvatan MRM. Triggering role of stressful life events in patients with alopecia areata. Acta Dermatovenereol Croat. 2012; 20(4):246-250.
4. Díaz-Atienza F, Gurpegui M. Environmental stress but not subjetive distress in children or adolescents. J Psychosom Res. 2011; 71:102-107.
5. Jung CG. Estudos experimentais. Petrópolis Vozes. 201;3.
6. Ito N. Human hair follicles display a functional equivalent of the hypothalamic-pituitary-adrenal axis and synthesize cortisol. FASEB J. 2005; 19(10):1332-1334.