Mouse model for the development of more effective schizophrenia drugs

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Technology description

Summary

Treatments for schizophrenia are currently lacking, even though it affects 1% of the population globally. This technology is a mouse model of the human ZDHHC8 gene. The deletion of this gene is known to be the leading cause of nonfamilial schizophrenia. Genetic variance in this gene causes a defect in neuron structure and brain function that most commonly manifests as schizophrenia. This technology may provide a route towards effective schizophrenia therapies.

An accurate genetic model of schizophrenia leads to more successful treatment strategies

Currently, there is a lack of drug development for schizophrenia; previous attempts ended in failure because of the lack of an evidence-based approach. This technology is a well-defined mouse model of nonfamilial schizophrenia. The ZDHHC8 gene encodes a palmitoyltransferase that is essential for proper neuron growth. Deficiency in this protein causes disruption of neuronal axon growth, and manifests in schizophrenia-like behavior in mice and humans. Replacement of this enzyme was shown to normalize behavior and may lead to a potential therapeutic approach.

Tech Ventures Reference: IR CU15276

Publications

Mukai J, Liu H, Burt RA, Swor D, Lai W-S, Karayiorgou M & Gogos JA. Evidence that the gene encoding ZDHHC8 contributes to the risk of the 22q11-associated schizophrenia. Nature Genetics 36, 725-31 (2004).

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Mukai J, Tamura M, Fénelon K, Rosen AM, Spellman TJ, Kang R, MacDermott AB, Karayiorgou M, Gordon JA, Gogos JA. Molecular substrates of altered axonal growth and brain connectivity in a mouse model of schizophrenia. Neuron 86, 680-95 (2015).

Application area

Drug development for schizophrenia medication
Tool to study disease mechanism and pathophysiology

Advantages

Causative genetic association with disease
Clear correlation between molecular mechanism and pathological outcome

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