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Creation of AD-relevant isogenic lines with the gene editing method CasMasTREE

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Since 1901, Alzheimer's disease (AD) claims at least 5.5 million individuals each year, making it the sixth leading cause of death in the United States1. Alzheimer's disease is an age-related neurodegenerative disorder denoted by severe memory deterioration due to amyloid beta and tau2. These proteins are known for creating knots and tangles within the brain, ultimately causing neuronal death and memory loss. AD has two forms: familial AD (FAD) and sporadic AD (SAD)3. About 70% of the population is affected by SAD, meaning that AD can develop because of the interplay of many genes2. Currently, there is no known cure or treatment for patients suffering with FAD and SAD. However, research has found that some genes, such as Apolipoprotein E (APOE) isoforms 2, 3, and 4, play a role in genetic risk of developing SAD. Specifically, the isoform, APOE 4, has been linked to the increased risk of developing AD, while the isoform, APOE 2, has been linked to decreased risk of developing AD4. How these genes increase the risk of AD is not well understood, but every gene found linked to Alzheimer's can increase the understanding of the disease forms. Since most AD cases are sporadic, it makes this disease difficult to study in vitro. Thus, there is a need for the ability to create isogenic lines to study single genes and their roles in neurodegenerative diseases.

Biography

David Brafman is an expert in pluripotent stem cell research and a deep passion for contributing to the scientific community. His current research is focused on gene editing systems and the affects they have on neurodegenerative diseases. These genomic editing techniques have been created over years of research and innovation to help positively impact the scientific community. Technologies created by Dr. Brafman's lab will significantly enhance gene editing techniques that can be applied to multiple applications including synthetic biology, regenerative medicine, and disease modeling.