



Homozygous Mutant DISC1 Mice, Lines 1001 and 1302

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Technology description

Invention Novelty:

The disclosed technologies are two tangible materials that are improved homozygous mouse strains with inducible expression of mutant human Disrupted-In-Schizophrenia-1 (hDISC1) using Tet-off system under the control of CAMKII.

Technical Details:

Johns Hopkins researchers have generated homozygous mouse strains carrying mutant hDISC1 without gross developmental defects. In these strains, the expression of mutant hDISC1 is restricted to forebrain regions using TET-off system under the control of CAMKII. In addition, the expression can be suppressed by feeding the mice with doxycycline (DOX).

Linkage and association of DISC1 or the region of the DISC1 locus to schizophrenia and psychiatric illnesses have been implicated in a number of genetic analyses and family pedigrees. DISC1 protein is expressed mainly in forebrain regions and is thought to act as a scaffold protein to mediate protein-protein binding. Many *in vitro* and *in vivo* studies have indicated that DISC1 affects neurotransmission, localization, and density of dopamine (DA) neurons via AKT/GSK-3 β signaling.

These transgenic animals allow selective control of mutant hDISC1 expression in forebrain neurons and, herein provides a valuable research tool to study pathogenesis of schizophrenia and to conduct experimental therapeutics for the disease.

Advantages

The disclosed materials are improved mouse strains of inducible expression of hDISC1 to facilitate the research in pathogenesis and preclinical therapeutic experiments of schizophrenia.

Institution

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