Autism cure may finally be possible

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Scientists have found new evidence to show that manipulating a single brain protein can under symptoms of a certain type of autism. A group of scientists from Bangalore and Massachusetts have genetically wired the brain circuits in mice models to manipulate the neurotransmitter glutamate, which plays a role in brain function and communication.

Dr. B.S. Shankaranarayana Rao,

The study tightens the prospects of curing autism, a brain development disorder that impairs social interaction and communication, and causes restricted and repetitive behavior.

Hyamanta Chattoraj, professor in the National Centre for Biological Sciences (NCBS), Dr. B.S. Shankaranarayana Rao of the National Institute of Mental Health and Neurosciences (NIMHANS) and scientists from the Brown Medical School and Massachusetts Institute of Technology in the US collaborated for this path-breaking search work. The group's findings appear in Thursday's issue of journal Neuron.

It is a brain disorder that affects social interaction

The study dealt with fragile X syndrome (FXS), the leading known inherited cause of mental retardation. The condition, tied to a mutated gene on the Y chromosome, has effects ranging from mild learning disabilities to severe autism, high anxiety and epileptic seizures.

"There is no current study in India for FXS, but we do come across cases in the clinic," said Satish Chandra Girish, a child psychiatry expert at NIMHANS. There is no cure available for autism, though interventions can make the condition better, he said.

FXS is caused by loss of the gene for fragile X mental retardation protein (FMRP). The loss of this protein, in turn, has a debilitating impact on brain connectivity—especially on the synapses, which are connections between brain cells. These patients have higher numbers of synapses in their brains, but with an abnormal structure.

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Glutamate is the main neurotransmitter that mediates communication between neurons by activating a variety of receptors. One such receptor is called metabotropic glutamate receptor 5 (mGluR5). The current study investigated if overactive signaling by mGluR5 contributes to many of the symptoms of FXS.

To test this possibility, MIT scientists led by Mark F. Baer made use of mice that, due to a loss of the FMRP gene, exhibit many of the same symptoms commonly seen in FXS patients. These mice were then genetically manipulated to have a 60 per cent reduction in mGluR5. Strikingly, these mice display significantly alleviated symptoms related to FXS by preventing abnormalities in structure and function of synapses, synthesis of brain proteins, memory, epilepsy, and body growth.

"The experiment involved rewiring and remodeling of brain circuitry in mentally retarded mice models," said Rao. "This work raises the hope that a drug blocking mGluR5 might effectively treat FXS and related autism spectrum disorders," said Chattoraj. "This finding is particularly significant in light of recent initiatives involving an mGluR5-blocker called fenobam, a drug being developed for the treatment of FXS."

In collaboration with the Fragile X Research Foundation (FRAXA), the UK-based Neuropharm is developing fenobam for the treatment of FXS. Earlier this year the same Bangalore scientists teamed up with another MIT group and reported positive results from a different approach to treat FXS.

Glimmer of hope: A drug is being developed to treat FXS that causes autism.