Hyperactivity is associated with decreased numbers of interneurons

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A new study published in *Biological Psychiatry* on May 15th is "another example of how basic science research conducted in animals may help to identify new molecular targets that may be studied for the treatment or even prevention of psychiatric disorders," according to Dr. John Krystal, Editor of Biological Psychiatry and affiliated with both Yale University School of Medicine and the VA Connecticut Healthcare System.

Deficits in gamma-aminobutyric acid (GABA) neuronal populations are being linked to a growing number of psychiatric disorders, including schizophrenia. The researchers in this study have used an animal model to study the role of the neocortex, a part of the brain responsible for motor activity, in hyperactive behavior.

Müller Smith and colleagues demonstrate that mice lacking the fibroblast growth factor receptor 1 (FGFR1) display profound, non-habituating hyperactivity that is correlated with a lack of parvalbumin-positive and somatostatin-positive inhibitory interneurons in the neocortex.

A decreased number of these same interneurons is "one of the most consistent findings in schizophrenia and psychotic disorders," explains Dr. Flora Vaccarino, corresponding author for this article. Dr. Vaccarino adds, "Interestingly, the loss of parvalbumin+ cells was inversely proportional to locomotor hyperactivity in these animals." Although the authors do not know yet know the mechanism by which this occurs, she notes that "these mice can be used a model for developing treatments

that may reverse this deficit."

Source: Elsevier