

Can one model the social deficits of autism and schizophrenia in animals?

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5 May 2011 - The use of animal models to study human disease is essential to help advance our understanding of disease and to develop new therapeutic treatments.

Social deficits are common in several psychiatric disorders, including [autism spectrum disorders](#) and schizophrenia. Individuals with severe social dysfunction can experience significant difficulties with everyday functioning.

[Oxytocin](#) and vasopressin are hormones that play key roles in emotional and social behaviors and bonding. Oxytocin has been suggested as a treatment to improve social behavior in individuals with autism, and initial studies in humans appear promising.

Now, scientists have further characterized a mouse model that provides some insights into biological factors related to social deficits, by comparing mice that had their oxytocin receptor gene made inactive, using a specialized technique called genetic knockout, with unaltered mice.

The [knockout mice](#) (OTR^{-/-}) displayed impaired social behavior, increased aggression and reduced [cognitive flexibility](#) leading to resistance to change. These behaviors returned to normal when the OTR^{-/-} mice were given oxytocin or vasopressin treatment.

"These findings confirm and highlight the importance of oxytocin for

social behaviors. This animal model also may be useful in evaluating the effectiveness of drugs, including vasopressin agonists, that may help improve social behavior in autism, schizophrenia, and other disorders," said Dr. John Krystal, Editor of [Biological Psychiatry](#), the journal publishing these results.

"While no animal model can be expected to replicate the full complexity of the human behavioral autistic phenotype, the OTR^{-/-} mouse may really help to understand the co-occurrence of these symptoms as a syndrome," explained Dr. Bice Chini, author and senior researcher of CNR - Institute of Neuroscience, Milano.

One important goal now is to fully characterize the neurodevelopmental processes modulated by oxytocin and [vasopressin](#) in order to fully understand their ability to reverse autistic symptoms.

More information: The article is "Pharmacologic Rescue of Impaired Cognitive Flexibility, Social Deficits, Increased Aggression, and Seizure Susceptibility in Oxytocin Receptor Null Mice: A Neurobehavioral Model of Autism" by Mariaelvina Sala et al. The article appears in *Biological Psychiatry*, Volume 69, Number 9 (May 1, 2011)

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