

Researchers demonstrate link between brain chemical, cognitive decline in schizophrenia

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In one of the first such studies involving human patients with schizophrenia, researchers at UC Davis have provided evidence that deficits in a brain chemical may be responsible for some of the debilitating cognitive deficits — poor attention, memory and problem-solving abilities — that accompany the delusions and hallucinations that are the hallmarks of the disorder.

The study, published online today in the [Journal of Neuroscience](#), suggests an important avenue of inquiry for improving cognitive function in the more than 2 million Americans who suffer from schizophrenia, according to Jong H. Yoon, an assistant professor of psychiatry and behavioral sciences at UC Davis Health System and the study's lead author.

"We still know very little about the neurobiology of schizophrenia, particularly at the level of specific circuits and molecules and how their impairments affect behavior and cognition in the disease," said Yoon, a researcher at the UC Davis Imaging Research Center. "We need this level of specificity to guide targeted treatment development. This is one of the first studies to show that there is a strong association between cognitive deficits and a decrease in a particular neurotransmitter."

Schizophrenia is characterized by psychosis — abnormalities in the perception or expression of reality. Sufferers may experience visual or auditory hallucinations and have paranoia, delusions and disorganized speech and thinking. But they also experience profound cognitive

difficulties that interfere with daily functioning.

Psychosis is treated with a variety of antipsychotic medications that dampen overactivity of the [neurotransmitter dopamine](#), an acknowledged cause of psychotic behavior. But no medications are available to address cognitive deficits in schizophrenia because the source of the deficits has not been determined. Deficits in one [brain chemical](#), the neurotransmitter gamma-aminobutyric acid, or GABA, have been implicated as playing a causal role in cognitive difficulties in people with schizophrenia in research involving animal models and post-mortem analyses of GABA concentrations in human schizophrenic brains.

"People think of schizophrenia as being related to psychosis. But patients' cognitive limitations can be even more debilitating for them," said Cameron Carter, professor of psychiatry and behavioral sciences, director of the Imaging Research Center and the study's senior author. "This study actually looked at brain chemistry in live patients in relation to cognitive performance to determine the underlying neurobiology of the cognitive deficits. Our ultimate goal is discovering ways to help patients lead more productive lives."

Yoon and his colleagues measured the levels of GABA in the visual cortexes of the brains of 13 study subjects with schizophrenia and 13 control subjects without the disorder. The measurements were conducted with high-field magnetic resonance spectroscopy, a technique that involves using a magnetic resonance imaging scanner to examine neurotransmitter activity. The schizophrenic patients were found to have a deficit in GABA of about 10 percent when compared with their non-schizophrenic counterparts.

The second half of the study involved demonstrating the significance of the neurochemical deficit on cognition and behavior. To do this the researchers measured the visual perception of the subjects for whom

GABA levels were assessed by showing them a well-known illusion in which the presence of a high-contrast surrounding region inhibits the ability to perceive information in the center of the visual field.

The researchers showed that this surround-suppression illusion had less of an effect on patients with schizophrenia, resulting in a highly unusual situation in which they outperformed healthy subjects when baseline differences in generalized task performance were accounted for. The researchers then found that the lower levels of GABA in patients were responsible for this behavioral abnormality.

"The link between changes in patients' brain chemistry and the cognitive impairments they experience never has been shown before in this way," Carter said. "This work provides tremendous support for targeting the GABA system for treatment of cognitive decline in [schizophrenia](#)."

Provided by University of California - Davis

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