

Cold sore virus may contribute to cognitive and brain abnormalities in schizophrenia

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Exposure to the common virus that causes cold sores may be partially responsible for shrinking regions of the brain and the loss of concentration skills, memory, coordinated movement and dexterity widely seen in patients with schizophrenia, according to research led by Johns Hopkins scientists.

"We're finding that some portion of cognitive impairment usually blamed solely on the disease of [schizophrenia](#) might actually be a combination of schizophrenia and prior exposure to [herpes simplex virus 1](#) infection, which reproduces in the brain," says study leader David J. Schretlen, Ph.D., an associate professor in the Department of Psychiatry at Johns Hopkins University School of Medicine.

The research, described in the May *Schizophrenia Research*, could lead to new ways to treat or prevent the cognitive impairment that typically accompanies this mental illness, including with [antiviral drugs](#), the scientists say.

Doctors have long known that [cognitive impairment](#), including problems with psychomotor speed, concentration, learning, and memory, are prevalent features of schizophrenia, which affects an estimated one percent of the U.S. population. Cognitive deficits often surface months to years before symptoms that are traditionally used to diagnose this disease, such as delusions or hallucinations.

Some previous studies have shown that schizophrenic patients with

antibodies to herpes simplex virus 1 (HSV-1), the virus that causes cold sores, often have more severe cognitive deficits than patients without these antibodies. Other studies have shown that patients with HSV-1 antibodies have decreased brain volumes compared to patients without the antibodies. However, it has been unclear whether the cognitive deficits are directly related to the decreased brain volume.

To investigate, Schretlen and his colleagues recruited 40 schizophrenic patients from outpatient clinics at the Johns Hopkins and Sheppard Enoch Pratt hospitals in Baltimore, Md. Blood tests showed that 25 of the patients had antibodies for HSV-1 and 15 didn't. The researchers gave all of the patients tests to measure speed of coordination, organizational skills and verbal memory. The patients then underwent MRI brain scans to measure the volume of particular regions of their brains.

As in previous studies, results showed that patients with antibodies to HSV-1 performed significantly worse on the cognitive tests than patients without the antibodies. But expanding on those earlier studies, analysis of the brain scans showed that the same patients who performed poorly on the tests also had reduced brain volume in the anterior cingulate, which controls processing speed and the ability to switch tasks. There was also shrinkage in the cerebellum, which controls motor function.

These results suggest that HSV-1 might be directly causing the cognitive deficits by attacking these [brain](#) regions, Schretlen says.

Though the researchers aren't sure why schizophrenia might make brains more vulnerable to a viral assault, Schretlen says the results already suggest new ways of treating the disorder. Data from other studies has shown that antiviral medications can reduce psychiatric symptoms in some patients with schizophrenia. "If we can identify schizophrenic patients with HSV-1 [antibodies](#) early on, it might be possible to reduce

the risk or the extent of cognitive deficits," he adds.

Provided by Johns Hopkins Medical Institutions

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