

Searching for causes of neural disconnection in schizophrenia

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Dendritic spines act as hubs for communication between nerve cells. Reductions in spines may contribute to a lack of coordination in activity between brain regions. This structural abnormality is particularly relevant in schizophrenia, where pyramidal neurons located in layer 3, the principal cell type receiving communication from other brain regions, have fewer dendritic spines.

Studying this phenomenon further, researchers at the University of Pittsburgh now provide new insights into the dendritic spine deficits in schizophrenia. Drs. Masayuki Ide and David Lewis studied postmortem brain tissue of individuals diagnosed with schizophrenia and compared it to tissue from healthy individuals.

They found that the expression levels of two gene products, CDC42EP3 and septin 7, were higher and lower, respectively, in tissue from those with schizophrenia. CDC42EP3 and septin 7 play important roles in regulating spine plasticity. Since CDC42EP3 is preferentially expressed in layer 3, the findings suggest that the altered expression of these transcripts may contribute to the layer-specific deficits in dendritic spines associated with schizophrenia.

"We still have a long way to go to fully understand the neurobiology of schizophrenia. An important step in this process will be to begin to learn what we can about neural structure and chemistry from postmortem [brain tissue](#) from individuals diagnosed with schizophrenia," commented Dr. John Krystal, Editor of [Biological Psychiatry](#). "These studies may

help, ultimately, to develop new treatments that attempt to prevent or reverse these disturbances in brain structure associated with schizophrenia."

Dr. Lewis agreed, remarking that "These findings provide a potential basis for novel treatments for [schizophrenia](#), and for preemptive interventions for individuals who are at high risk for the illness."

More information: The article is "Altered Cortical CDC42 Signaling Pathways in Schizophrenia: Implications for Dendritic Spine Deficits" by Masayuki Ide and David A. Lewis. The authors are affiliated with the University of Pittsburgh in Pennsylvania. The article appears in *Biological Psychiatry*, Volume 68, Issue 1 (July 1, 2010) www.sobp.org/journal

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