ReachMD
Be part of the knowledge.

TRANSCRIPT

Transcript Details
This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/neurofrontiers/understanding-schizophrenia-the-role-of-neurotransmission-and-inflammation/36637/

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

Understanding Schizophrenia: The Role of Neurotransmission and Inflammation

Announcer:
You're listening to *NeuroFrontiers* on ReachMD. On this episode, sponsored by Bristol Myers Squibb, we'll hear from Dr. Jeffrey A. Lieberman, who's the Constance and Stephen Lieber Professor of Psychiatry at Columbia University's Vagelos College of Physicians and Surgeons, as well as the President and CEO of ARETÉSCIENCE. He'll be sharing new insights into the pathophysiology of schizophrenia. Here's Dr. Lieberman now.

Dr. Lieberman:
Schizophrenia is believed to be caused by a disturbance in synaptic neurotransmission. And in this context, the neurotransmitters—of which there are many in the body and hundreds in the brain—that have been implicated for schizophrenia are, first and foremost, dopamine and glutamate. Serotonin creeps in there, and now also, more recently, acetylcholine creeps in there for various reasons.

But there are other mechanisms which could also be involved in underpinning why the disturbance in chemical neurotransmission occurs or occurs as a result of the disturbance in chemical neurotransmission. So in that regard, we found that even though there's no direct evidence of substantial neurodegeneration in patients with schizophrenia by things like loss of neurons, glial cell proliferation, or massive atrophy, we can say that there's evidence that there is progression because there is a loss of gray matter or neuropil, which is the dendrites and the synaptic connections that occur in the brain as the illness wears on and in the context of repeated or recurrent psychotic episodes. So there is this limited process of deterioration or loss of organic tissue that occurs in the course of the illness.

Now, as this occurs, and because of the dysregulation in chemical neurotransmission primarily associated with dopamine and glutamate, it's also very likely that an inflammatory process occurs. Even though inflammation used to be thought to be limited to specific illnesses like rheumatoid arthritis or other types of illnesses, inflammation occurs as a secondary phenomenon to almost everything, including cardiovascular disease and diabetes. So why shouldn't it happen in the brain when you have some type of excitatory pathologic process? So inflammation is probably not a primary pathophysiologic process in the brain or an etiologic one; it's a byproduct of the dysregulation in chemical neurotransmission as is the loss of neuropil and the dendrites—the neural elements that are really the scaffolding for synapses and chemical neurotransmission. This is a byproduct of this also.

The best way to pull together and integrate our understanding of the pathophysiology of schizophrenia and the functional and clinical consequences of that is to identify patients who have the illness as early as possible, institute treatment to a point of achieving maximal improvement and ideally symptom remission, and sustain that with continued maintenance treatment so that there are not further recurrences.

Announcer:
You've been listening to *NeuroFrontiers*, and this episode was sponsored by Bristol Myers Squibb. To access this and other episodes in our series, visit *NeuroFrontiers* on ReachMD.com, where you can Be Part of the Knowledge. Thanks for listening!