



## Associational Investigation between Blood Brain Barrier (BBB) Alterations in First-Episode Psychosis (FEP) Patients

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### ABOUT THE STUDY

A wide clinical entity, schizophrenia is currently operationalized by symptoms and course patterns. A growing body of research shows that frequent cerebrospinal fluid (CSF) examinations in patients with psychosis could uncover underlying inflammatory or viral processes as well as a probable diagnosis of autoimmune encephalitis. National schizophrenia guidelines are now recommending lumbar puncture in first-episode psychosis (FEP) patients when an underlying somatic illness is suspected. In light of recent discoveries, it has been recommended that all patients with first-episode psychosis have a more complete CSF-based screening that includes neuronal autoantibodies.

Inflammatory responses result in the development of neurodegenerative processes or processes of unsuccessful neural regeneration, at least in part. In this particular situation, a recent meta-analysis found that schizophrenia had a higher rate of blood-brain-barrier (BBB) disruption than did healthy controls. The part played by BBB issues in the aetiology of schizophrenia is still unclear, though. Though it can be hypothesised that BBB integrity, or lack thereof, may be crucial in the pathogenesis of schizophrenia and possibly psychotic disorders in general given the association between BBB dysfunctions and pathomechanisms implicated in the development of schizophrenia, such as disrupted glutamatergic signalling or inflammation. A secretion called CSF has the potential to reveal important details about the integrity of the BBB and inflammatory processes in the brain.

Psychotic diseases have been associated with elevated Qalb levels, which suggest that BBB is compromised in these illnesses. Prior research including a substantial sample size discovered an elevated Qalb in roughly 16% of all FEP patients. Considering

that vitamins are involved in the manufacture of proteins that support brain growth and repair, they may be particularly important in this situation. Homocysteine (Hcy) and methylmalonic acid (MMA) build up as a result of methionine synthase and mitochondrial methylmalonyl-CoA mutase being inactive due to vitamin B12 deficiency. Hcy, an endogenous amino acid that promotes inflammation, is created when nutritional methionine is demethylated. It is then mainly re-methylated back to methionine, requiring folate as a methyl donor and vitamin B12 as a cofactor.

In conclusion, low levels of folate and vitamin B12 raise Hcy levels, which may lead to changes in the BBB. From a pathophysiological standpoint, there is data that suggests that a number of vitamin and mineral deficiencies may be linked to an increased risk of schizophrenia, which may be partly due to weakened BBB integrity. Notably, the Prenatal Determinants of Schizophrenia (PDS) study examined banked sera from a US cohort of pregnant women and found that a higher Hcy level in the third trimester was linked to a twofold increase in the risk of schizophrenia in offspring.

Additionally, serum markers indicating lower levels of D and B vitamins in schizophrenia patients have been found to significantly correlate with the severity of the illness, particularly with regard to negative symptoms. Additionally, these vitamin deficits are linked to neuropsychological abnormalities seen in schizophrenia, such as cognitive decline and hippocampus volume decrease. A randomised clinical trial found that supplementing with folate and vitamin B12 reduced negative symptoms in people with persistent schizophrenia, despite the fact that genetic diversity in folate absorption affected treatment response.

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