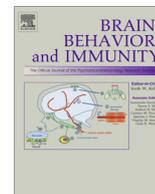




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Short Communication

Reduced microglial immunoreactivity for endogenous NMDA receptor agonist quinolinic acid in the hippocampus of schizophrenia patients

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ABSTRACT

Postmortem and positron emission tomography studies have indicated the pathophysiological involvement of microglial cells in schizophrenia. We hypothesized that the microglial production of quinolinic acid (QUIN), an endogenous N-methyl-D-aspartate receptor (NMDAR) agonist, may be linked to the previously described glutamatergic deficits in the hippocampus of schizophrenia patients.

We performed a semi-quantitative assessment of QUIN-immunoreactive microglial cells in schizophrenia patients and matched controls in the CA1, CA2/3, and dentate gyrus (DG) area of the posterior hippocampal formation. Complementary immunostaining of the commonly used microglial surface marker HLA-DR was performed in adjacent histological sections.

Fewer QUIN-immunoreactive microglial cells were observed in the CA1 hippocampal subregion of schizophrenia patients compared to controls (left $p = 0.028$, right $p = 0.018$). No significant diagnosis-dependent changes were observed in the CA2/3 and DG regions. These results were controlled for potential confounds by age, duration of disease, autolysis time, psychotropic medication, and hippocampal volume. No diagnosis-related differences were observed for the overall density of microglial cells (HLA-DR expression).

Our findings suggest that reduced microglial QUIN content in the hippocampal CA1 region is associated with schizophrenia. We hypothesize that this association may contribute to impaired glutamatergic neurotransmission in the hippocampus of schizophrenia patients.

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