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There is a wealth of evidence indicating the involvement of the endocannabinoid (EC) system in the pathophysiology of schizophrenia. Previous studies have described an increase in anandamide levels in the cerebrospinal fluid and in the blood of schizophrenic subjects. However, to our knowledge, there are no published data reporting EC levels in the brain tissue of schizophrenic subjects.

The aim of this study was to evaluate the EC levels in three postmortem brain regions of schizophrenic subjects and matched controls.

Human brain samples were collected from 19 patients with diagnosis of schizophrenia (DSM-IV), and 19 controls matched by age, gender and postmortem delay. After liquid–liquid extraction of the lipid fraction from homogenized brain samples, the ECs were determined by quantitative liquid chromatography with tandem mass spectrometric detection in cerebellum (CB), hippocampus (HC) and prefrontal cortex (PFC). This method was used to measure the levels of four ECs (2-arachidonoylglycerol (2-AG), arachidonoylethanolamide (anandamide, AEA), dihomo-γ-linolenoylethanolamide (LEA), and docosahexaenoylethanolamide (DHEA)) and two other cannabimimetic compounds (palmitoyl ethanolamide (PEA) and oleoyl ethanolamide (OEA)).

A significant increase in 2-AG levels was observed in the PFC of schizophrenic subjects compared to controls (+137±25%; p=0.009). The AEA and DHEA concentrations were significantly reduced in the HC of schizophrenic subjects compared to the control group (-34±8%; p=0.004 and -32±7%; p=0.004, respectively). There was also a significant decrease in DHEA levels in the CB of schizophrenic subjects compared to matched controls (-40±6%; p=0.001). No statistically significant differences were found in the levels of LEA, PEA and OEA between schizophrenic and control subjects in any of the studied brain areas.

The present results demonstrate that some ECs are specifically altered in some areas of the brain of schizophrenic subjects. Moreover, these data provide further evidence that the EC system may be involved in the pathophysiology of schizophrenia.