

S39-02 Antipsychotic effects of cannabidiol

F.M. Leweke¹, D. Koethe¹, F. Pahlisch^{1,2}, D. Schreiber^{1,2}, C.W. Gerth¹, B.M. Nolden¹, J. Klosterkötter¹,

¹Dept. of Psychiatry and Psychotherapy, University of Cologne, Cologne, Germany

²Depts. of Pharmacology and Biological Chemistry, University of California, Irvine, USA

³Institute for Medical Statistics, Informatics, and Epidemiology, University of Cologne, Cologne, Germany

Available online 13 March 2009.

Background

In contrast to delta-9-tetrahydrocannabinol, the phytocannabinoid cannabidiol does not exert psychotomimetic effects. Cannabidiol was suggested a re-uptake inhibitor of anandamide and potential antipsychotic properties have been hypothesized for it. We therefore performed a clinical trial to investigate thesis hypothesis and to clarify the underlying link to the neurobiology of schizophrenia.

Methods

We performed an explorative, 4-week, double-blind, controlled clinical trial on the effects of purified cannabidiol in acute schizophrenia compared to the antipsychotic amisulpride. The antipsychotic properties of both drugs were the primary target of the study. Furthermore, side-effects and anxiolytic capabilities of both treatments were investigated.

Results

42 patients fulfilling DSM-IV criteria of acute paranoid schizophrenia participated in the study. Both treatments were associated with a significant decrease of psychotic symptoms after 2 and 4 weeks as assessed by BPRS and PANSS. However, there was no statistical difference between both treatment groups. In contrast, cannabidiol induced significantly less side effects (EPS, increase in prolactin, weight gain) when compared to amisulpride.

Conclusions

Cannabidiol revealed substantial antipsychotic properties in acute schizophrenia. This is in line with our suggestion of an adaptive role of the endocannabinoid system in paranoid schizophrenia, and raises further evidence that this adaptive mechanism may represent a valuable target for antipsychotic treatment strategies.

The Stanley Medical Research Institute (00-093 to FML) and the Koeln Fortune Program (107/2000 + 101/2001 to FML) funded this study.

European Psychiatry

Volume 24, Supplement 1, 2009, Page S207

17th EPA Congress - Lisbon, Portugal, January 2009, Abstract book