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EEG power spectra and connectivity changes in animal serotonergic and glutamatergic models of psychosis – implications for treatment

T. Palenicek[1], F. Tyls[1], M. Fujakova[1], A. Kubesova[1], P. Novakova[1], L. Kaderabek[1], M. Brunovsky[2], J. Horacek[3]

[1]Laboratory of Biochemistry and Brain Pathophysiology, Prague Psychiatric Center, Prague, Czech Republic ; [2]Laboratory of EEG and Clinical Neurophysiology, Prague Psychiatric Center, Prague, Czech Republic ; [3]Clinical unit, Prague Psychiatric Center, Prague, Czech Republic

Pharmacological models of psychosis bring a unique tool for studying brain disconnection in humans as well as in animals. Even though several electrophysiological biomarkers have been already described in schizophrenia, little is known about EEG biomarkers in pharmacological models of psychosis and about the translational validity of these data. Studies on EEG brain connectivity in rodents under these circumstances are extremely rare. To elucidate the characteristic patterns of EEG connectivity in freely moving rats we have conducted a series of experiments in serotonergic and glutamatergic models of psychosis. Using multiple cortical electrodes, EEG spectral and connectivity analysis was performed on selected episodes of behavioral inactivity – a model of resting EEG. The analyses showed consistent changes that were specific for each of the models used. Glutamatergic models with ketamine and dizocilpin (MK-801) induced typical global increase in high frequency oscillations. On the contrary, in serotonergic models (psilocin, mescaline, LSD, DOB, 2C-B) a global power decrease was observed. A common denominator in both models was a global decrease in connectivity expressed as decreased coherence in most of the frequency bands. A translational validity of these findings is supported by findings in schizophrenia patients and by recent human studies with ketamine and psilocybin. Recently obtained results with drugs modulating serotonergic, dopaminergic and glutamatergic neurotransmission will be also discussed in the light of potential therapeutic implications.

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