Article: 0492
Topic: EPW24 - e-Poster Walk Session 24: Addictive Behaviours part 2

Role of Benzodiazepine Receptor Plasticity Associated with Neuroactive Steroid's Levels in Patients with Compulsive Craving for Alcohol, New Approaches to Therapy

T.V. Shushpanova[1]

[1]Biological Psychiatry, Mental Health Research Institute SB RAMSci, Tomsk, Russia

Background Substances that act on $GABA_A$/benzodiazepine receptors (ethanol, benzodiazepines, barbiturates) can adversely affect the both the short- and long-term effects in the CNS. Our study was to investigate density of platelet's benzodiazepine receptors (BDR) and certain neuroactive steroids: progesterone (PG) and cortisol (CS) in blood serum of patients with compulsive craving for alcohol during chronic exposure to ethanol and evaluate their role in the development of preference for alcohol and alcohol addiction.

Methods Platelet's benzodiazepine receptors (BDR) of patients with alcohol addiction and control persons were explored using radioreceptor analysis with selective ligand [^{3}H]PK-11195. RIA kits were used for assay of CS and PG from blood serum in examined persons.

Results The kinetic binding parameters of the specific BDR ligand [^{3}H]PK11195 have been evaluated in platelets from 36 male alcoholic patients in relation to 19 healthy sex-matched controls. A significant increase of mean value of platelet BDR density was observed in patients as compared to the controls (4733±379 and 3358±242 fmol/mg proteins, $p<0,005$). Comparative study of NS levels in blood serum of alcoholic patients showed significant decrease the level of PG, compared with healthy donors; investigations levels of CS showed significant increase of alcoholic patients.

Conclusion Alterations levels of NS in alcoholic patients suggest the abruption regulation of NS on platelet's BDR density as one of the mechanisms of development of compulsive craving for alcohol. Compulsive behavior links with altered levels of neuroactive steroid hormones that can modulate $GABA_A$/BD receptor's plasticity and controls inhibitory and excitatory processes in the brain.

https://doi.org/10.1016/S0924-9338(15)30388-6 Published online by Cambridge University Press