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BENZODIAZEPINE RECEPTOR SYSTEM OF HUMAN BRAIN IN ALCOHOL ADDICTION

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Background: alcohol abuse induce neuroadaptive alters of benzodiazepine receptors (BDR) that modulate $GABA_AR$, mediation of GABA and activity of dopaminergic neurons in brain regions, associated with reward function in the brain that serve alcohol addictions.

Method: properties of BDR 'central' (CBR) and 'peripheral' (PBR) types were studied in synaptosomal and mitochondrial membranes from different brain areas of alcohol abused patients and non-alcoholic persons (early postmortem material, less 5 hours after death) by radioreceptor assay with using selective ligands: [^{3}H]flunitrazepam and [^{3}H]PK-11195.

Results: comparative study of kinetic parameters (affinity and capacity) of [^{3}H]flunitrazepam and [^{3}H]PK-11195 binding with synaptosomal and mitochondrial membranes in autopsy samples from different areas of human brain was showed that affinity of BDR was decreased: CBR – in 1,2 times in prefrontal cortex and n.caudatus, less – in cerebella cortex; PBR – in 2,1 times in n.caudatus, 1,3 times – in prefrontal cortex and cerebella cortex in patients with alcohol addiction.

Capacity of BDR was increased in different areas of human's brain under influence of alcohol abuse. More alters of CBR appeared in prefrontal cortex (179%) and cerebella cortex (176%), less – in n.caudatus (145%); PBR – in n.caudatus (262%); and cerebella cortex (205%), and less in prefrontal cortex (123%).

These results showed that alcohol addiction induce more alterations in PBR (mitochondrial) than CBR (synaptic) that agree with maintaining function of glial cells in CNS under influence of different toxic factors.

Conclusion: our findings suggest hypothesis of difference in response BDR from various brain areas on alcohol influence.

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