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Mini Review

GABA Benzodiazepine Receptors and GABA-Ergic Neurotransmission Changes in Alcohol and Nicotine Dependency

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GABA-A receptors have an important role in modulation of the effects of ethanol in the brain. GABA receptors as ion channels which are basically ligand gated ones, modulate the inhibitory synaptic transmission in the central nervous system. Alcohol addiction phases such as withdrawal, dependency and tolerance are associated with the GABA-ergic neurotransmission changes in the brain [1,2]. The neuroimaging studies with PET and SPECT have demonstrated that there are lower GABA-A benzodiazepine receptors availability in alcohol dependent subjects. In tobacco smoking status, higher availabilities of GABA-A benzodiazepine receptors have been demonstrated [3,4]. These findings suggest that tobacco smoking may suppress increasing in the availability of GABA-A benzodiazepine receptors in alcohol addiction. Moreover, such higher availability is related with alcohol withdrawal symptoms which are more pronounced in alcohol dependent nonsmokers than smokers. Such findings suggest that by suppression of the increased GABA-A benzodiazepine receptors availability during alcohol withdrawal, tobacco smoking may block symptoms of alcohol withdrawal. Overall, availability of GABA-A benzodiazepine receptors would increase in the early stages of withdrawal and then decrease during time. Such changes are mediated by tobacco smoking. The clinical implications of such findings suggest that nicotine replacement therapy can be helpful to tolerate the nicotine and alcohol withdrawal symptoms in smokers with alcohol dependency [3,4].

Acknowledgement

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Conflict of Interest

No conflict of Interest.

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