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THE PSEUDO-MEDIATOR-ANTIOXIDANT EFFECT OF ALCOHOL

M. Osain Welcome, V. Alekseevich Pereverzev

Belarusian State Medical University, Minsk, Belarus

This is a hypothesis that has come a long way to explaining some peculiarities of alcohol effect in the central nervous system. An important postulate in this hypothesis is the role of some alcohol-adducts in neurotransmission, especially in the early stages of intoxication and the degree of thermo-dynamical balance during adducts formation provide important clues to its effect, and to find better treatment options for addiction. Another postulate is that adducts at its initial formation, might act as antioxidants. Adduct formation in relation to the quantity and frequency of alcohol use, its role in behavior, dependence and tolerance is still not known. Majority of data shows that adducts of magnesium halide/alcohol; lipoprotein; 4-mthyl-1,4-dihydropyridine-3,5-dicarbaldehyde, MDHDC; FAAB, MAA are responsible for the carcinogenic/damaging effects of alcohol. The degree of hematological derangements caused by acetaldehyde adducts might have serious implications in brain functions. Recent studies have shown that adducts can induce the release of proinflammatory cytokine TNF-α in heart endothelial cell. Biological responses to adduct-modified proteins are responsible for the development and/or progression of alcoholic liver disease. Researches have consistently shown that any liver dysfunction, even without apparent damage might lead to significantly low performance in neuropsychological tests.

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