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Alcoholism and Serotonin

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Introduction

Predisposition to alcoholism and chronic drinking consequences would cause the CNS serotonin dysfunction. In the presence of deficiency in the serotonin which is induced by 5,6-dihydroxytryptamine or parachlorophenylalanine, alteration in the behavioral effects of the ethanol would occur and due to this deficiency, alcohol consumption would be increased. Low levels of 5-hydroxyindole acetic acid (5HIAA) is an evidence which shows that in alcoholic subjects, the serotonergic function would be reduced. There would be a shift from pathways which lead to 5-hydroxyindole acetic acid to ones which produce 5-hydroxytryptophol and 5-hydroxyindole acetaldehyde in serotonin metabolism [1-4]. Low responses to serotonin agonists would support the serotonin dysfunction in alcoholic subjects. Fenfluramine in abstinent alcoholic subjects, induced a smaller response with prolactin in comparison with the controls. There would be no effects on consuming ethanol by doing rapid tryptophan depletion studies but doing such studies in subjects with alcoholism and concomitant major depressive disorder, shows that serotonin depletion would increase symptoms of depression and drinking needs [5,6].

5-HT1b, 5-HT3 and 5-HT2c are the receptors which ethanol acts on them. 5-HT3a receptor presence is related to the ethanol consumption reduction. Ondansetron in early-onset alcoholism can reduce the consumption of the alcohol as it is the antagonist of the 5-HT3 receptor. The serotonin transporters expression would be altered by a functional repeat polymorphism in the promoter region of the 5-HTTLPR as the serotonin transporter gene. There would be more serotonin receptors in the homozygous carriers of long alleles than ones with short alleles. Reduction in sensitivity to alcohol and developing the risk for alcohol dependence would be associated with the glutamatergic, serotonergic and GABAergic systems interactions [7,8].

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