POSTER 42

CHRONIC ETHANOL EXPOSURE ALTERS EPIGENETIC MECHANISM IN THE BASAL FOREBRAIN

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One of the most commonly observed characteristics of alcohol withdrawal is insomnia. Importantly, there is a strong association between insomnia and relapse to alcoholism. In a recent study, we have shown that insomnia in ethanol dependent rats may result from reduced gene expression of proteins responsible for adenosine release and transmission in the basal forebrain (BF). Histone acetylation is an epigenetic phenomenon that promotes gene expression. Does ethanol dependence alter histone acetylation in the BF and affect gene expression? To address this issue, adult male Sprague Dawley rats were divided in two groups: an ethanol dependent group and a control group. The ethanol dependent group was intragastrically administered ethanol (35% v/v; ~9 g/kg) whereas the control group was administered sterile water (30 ml/kg) in three divided doses per day for four days. During withdrawal (after 12 hrs of the last dose of ethanol), the rats were euthanized and brains processed for acetylated histone H3 (AcHis; marker for histone acetylation) immunohistochemistry. Our result showed that there was a significant reduction in the number of cells with AcHis in the BF region of ethanol dependent rats (N=5) compared to the controls (N=5). Based on our results, we suggest that ethanol dependence leads to the reduction of histone acetylation in the BF region which may affect gene expression.