Histone deacetylase (HDACS) expression profiling in the amygdala during chronic ethanol treatment and withdrawal

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Histone deacetylase (HDAC) related histone modifications in the amygdala has been shown to be an important mechanism underlying the anxiolytic and anxiogenic behaviors of acute alcohol exposure and chronic ethanol induced withdrawal, respectively. It was shown that the anxiety-like behaviors during chronic ethanol withdrawal were associated with an increase in class I and II HDAC activity in the amygdala of rats. However, the mechanism by which chronic ethanol treatment and withdrawal regulates the expression of different HDAC isoforms remain unclear. In this study, the expression profiling of various HDAC isoforms in the amygdala of rats after chronic ethanol exposure and withdrawal was examined. Male adult Sprague Dawley rats were pair fed with Lieber-Decarli control or ethanol diets. Ethanol diet-fed rats were gradually introduced to the diet containing ethanol and then maintained on the ethanol diet at 9% (v/v) for 15 days. Ethanol fed rats were withdrawn for 0 or 24 hr. Control rats were pair fed with control diet. The rats were perfused after behavioral testing and the brains were used for the measurement of protein and mRNA of each HDAC isoform by using gold immunolabeling and in-situ RT-PCR, respectively. In this model of withdrawal-induced anxiety, it was found that protein and mRNA levels of HDAC2 and HDAC3 isoforms were significantly upregulated in central (CeA) and medial (MeA), but not basolateral nucleus of the amygdala (BLA) during ethanol withdrawal. The chronic ethanol exposure and withdrawal had no effects on protein and mRNA levels of other HDACs (HDAC1, HDAC4, HDAC5 and HDAC6) in CeA, MeA, or BLA. The findings suggested that only HDAC2 and HDAC3 among the HDAC family may be responsible for the increased HDAC activity in the amygdala during ethanol withdrawal, as well as decreased histone acetylation (H3 & H4) in the CeA and MeA but not in BLA and these two isoforms may be crucial in the pathophysiology of alcoholism.

Biography
Huaibo Zhang obtained his MD from Zhengzhou University and his PhD in Neuroscience from the Xi’an Jiaotong University in China. He completed Postdoctoral training from Sun Yet-sen University and University of Illinois at Chicago. He currently works as a research Assistant Professor in the Department of Psychiatry, University of Illinois at Chicago. He is actively involved in investigating epigenetic and molecular mechanisms of the alcohol use disorders.

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