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[Alcohol Controls Brain DNA Activity](#)

This explains withdrawal

Alcohol acts like a drug when alcoholism is installed. Alcohol withdrawal symptoms, like anxiety, represent a reality. A new research carried out at the University of Illinois at Chicago and the Jesse Brown VA Medical Center, and published in the "Journal of Neuroscience," has found the reason behind this: the gene expression in the brain cells is impacted. DNA activity is modified by "epigenetic" modifications, minor chemical changes of chromatin, dense bundles of DNA and histone proteins. "This is the first time anyone has looked for epigenetic changes related to chromatin remodeling in the brain during alcohol addiction," said lead author Dr. Subhash C. Pandey, professor and director of neuroscience alcoholism research at the UIC College of Medicine and the Jesse Brown VA Medical Center, in Chicago. The histones control DNA activity. The enzymes histone acetyltransferases (HATs) attach acetyl groups to histones and loosen their packing, letting the genes to produce their encoded proteins. The enzymes histone deacetylases (HDACs) detach acetyl groups from histones, making them to cover tightly the DNA, deactivating the genes. The same team had previously revealed in animal-made tests that levels of neuropeptide Y (NPY) in the amygdala controlled anxiety and alcohol-drinking behavior. Now, they have focused on the HDAC activity, acetylation of histones, and expression of the genes for NPY in the amygdala in cases of anxiety behavior connected to alcohol withdrawal. Acute exposure to alcohol was found to lower HDAC activity and to boost the activity of HATs and levels of NPY, all translated into decreased anxiety in the animals. Oppositely, anxiety-like behaviors related to withdrawal in animals with chronic alcohol exposure was linked to a higher HDAC activity and a lower one in HATs and NPY levels. Blocking the rise of HDAC levels via an HDAC inhibitor during alcohol withdrawal brought HATs and NPY levels in the amygdala to normal values, removing anxiety behaviors. "Our findings suggest that HDAC inhibitors may have potential as therapeutic agents in treating alcoholism. Anxiety associated with withdrawal from alcohol abuse is a key factor in the maintenance of alcohol addiction," said Pandey. The new research also detected that the levels of CREB binding protein, known to have HAT enzymatic activity, were boosted by acute alcohol intake and lowered during alcohol withdrawal. It appears that all enzymes connected to chromatin activity are involved in the anxiety behavior produced by alcohol withdrawal or acute alcohol intake.