

Binge drinking may cause Alzheimer's disease—and it might strike younger and in a severe form

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Binge drinking may be linked to both the onset and severity of Alzheimer's disease, but scientists have only now embarked on a path to decipher each molecular step involved in how excessive alcohol consumption leads to the most common form of dementia.

The research, underway at the Feinstein Institutes for Medical Research in New York, builds on a deceptively simple premise: Excessive alcohol consumption is toxic to the brain. Binge drinking likely plays an insidious role in the alteration of a normal brain [protein](#) into a biological rogue that is highly prevalent in Alzheimer's disease. The protein is identified by a simplistic monosyllabic name—tau.

In its normal conformation, tau is found in neurons modulating the stability of axonal microtubules. But in its abnormal conformation, tau has long been considered one of the leading hallmarks of Alzheimer's, and makes up the tangles in the

notorious "plaques and tangles" pathology. The plaques are deposits of the protein beta amyloid. The Feinstein Institutes research involving binge drinking and Alzheimer's dementia is riveted, however, on tau.

A potential breakthrough in the investigation would be a definitive explanation of how tau transforms from a normal protein into a neuron-annihilating cause of Alzheimer's under the influence of excessive alcohol. The New York researchers think they're on the right path to make that discovery.

Already, the scientists are delving into how tau can become phosphorylated, which means its structural conformation changes and its role in the brain becomes chemically altered under the influence of binge drinking.

"Studies have shown that frequent and heavy alcohol drinking is linked to earlier onset and increased severity of Alzheimer's disease," Dr. Max Brenner, assistant professor at the Feinstein Institutes told Medical Xpress. "It has been reported that alcohol consumption correlates with Alzheimer's-like cortical atrophy in individuals at high risk of developing the disease as well as younger age of onset.

"In addition, chronic alcohol exposure caused neural tau phosphorylation in the hippocampus and memory-impairment in Alzheimer's-predisposed mice," Brenner said.

The goal of the research is to shed light on specific proteins that apparently play key roles in the proliferation of tau. Brenner and colleagues want to understand the activities of cold-inducible RNA-binding protein (CIRP) and its associated form, extracellular cold-inducible RNA-binding protein (eCIRP).

"CIRP is normally present in the [cell nucleus](#) where it helps to regulate which proteins each cell produces," Brenner explained. "When cells detect potentially harmful conditions, such as alcohol exposure, they release molecules like eCIRP to alert other cells nearby to start preparing their defenses to overcome the stress conditions. The cells being alerted recognize eCIRP outside the cell when it binds to specific protein receptors in the cell membrane. The cascade of eCIRP proteins is triggered when alcohol diffuses throughout the brain, and while alcohol is a major influence, the eCIRP cascade can occur under other deleterious conditions. "A number of potentially harmful conditions trigger the release of eCIRP, including low oxygen, low temperature and radiation exposure," Brenner said.

Alzheimer's disease is the sixth-leading cause of death in the United States, and is the most common form of neurodegenerative dementia. It afflicts 5.8 million people nationwide. Globally, the disease is inexorably on the rise. An estimated 50 million people are believed to be living with Alzheimer's and other forms of dementia. According to the United Nations, the number of affected people could reach 152 million worldwide by 2050 unless therapeutics are discovered to stop the escalating number of cases. Brenner and colleagues theorize that blocking eCIRP might prove to be a viable treatment for alcohol-related Alzheimer's disease.

Binge drinking, meanwhile, is a major societal and public health concern. In the United States, the Centers for Disease Control and Prevention has found an array of collateral problems associated with the habit: car crashes, falls, burns, and alcohol poisoning. The agency also has traced [domestic violence](#), homicide and intimate partner violence, sexual assault, sexually transmitted diseases and suicides to binge drinking. It is estimated that roughly half of all alcohol-related deaths in the U.S. are directly related to binge drinking.

Brenner and colleagues say these drinkers can have a blood alcohol concentration ranging anywhere from 0.08% to amounts that are substantially higher.

Despite the tantalizing clues of their preliminary research, the Feinstein team is uncertain about precise sequence of mechanisms involved in how [excessive alcohol consumption](#) leads to Alzheimer's. However, they're keenly aware that eCIRP is a critical mediator of memory impairment induced by exposure to binge-drinking levels of alcohol.

The National Institutes of Health has found the research by Brenner and his team so intriguing that it has awarded the Long Island-based Feinstein Institutes a \$2.1 million grant to further investigate the role of alcohol in the development of Alzheimer's disease.

Brenner said there are also early suggestions that beta amyloid, the cause of Alzheimer's plaques, may also be linked to [binge drinking](#).

"Early-stage studies suggest that alcohol aggravates beta amyloid deposition by increasing the levels of amyloid precursor protein (APP), which increases the enzyme that changes the precursor into beta amyloid and decreases the cellular disposal of beta amyloid. We have decided to focus our research on the effects of [alcohol](#) on tau, however, because tau deposition correlates better with the cognitive decline in Alzheimer's [disease](#) than beta amyloid," Brenner said.

More information: Archana Sharma et al. Potential Role of Extracellular CIRP in Alcohol-Induced Alzheimer's Disease, *Molecular Neurobiology* (2020). [DOI: 10.1007/s12035-020-02075-1](https://doi.org/10.1007/s12035-020-02075-1)

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