Beer compound could help fend off Alzheimer's and Parkinson's diseases

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The health-promoting perks of wine have attracted the spotlight recently, leaving beer in the shadows. But scientists are discovering new ways in which the latter could be a more healthful beverage than once thought. They're now reporting in ACS' *Journal of Agricultural and Food Chemistry* that a compound from hops could protect brain cells from damage—and potentially slow the development of disorders such as Alzheimer's and Parkinson's diseases.

Jianguo Fang and colleagues note that mounting evidence suggests that oxidative damage to neuronal cells contributes to the development of diseases that originate in the brain. If scientists could find a way to guard these cells from this type of damage, they might be able to help prevent or slow down Alzheimer's disease, Parkinson's disease and other neurodegenerative conditions. One compound found in hops, called xanthohumol, has gotten the attention of researchers for its potential benefits, including antioxidation, cardiovascular protection and anticancer properties.

Fang's team decided to test xanthohumol's effects on brain cells.

In lab tests, the researchers found that the compound could protect neuronal cells and potentially help slow the development of brain disorders. The scientists conclude xanthohumol could be a good candidate for fighting such conditions.

More information: Xanthohumol, a Polyphenol Chalcone Present in

Hops, Activating Nrf2 Enzymes to Confer Protection against Oxidative Damage in PC12 Cells, *J. Agric. Food Chem.*, Just Accepted Manuscript, DOI: 10.1021/jf505075n

Abstract

Xanthohumol (2', 4', 4-trihydroxy-6'-methoxy-3'-prenylchalcone, Xn), a polyphenol chalcone from hops (Humulus lupulus), has received increasing attention due to its multiple pharmacological activity. As an active component in beers, its presence has been suggested to link to the epidemiological observation of the beneficial effect of regular beer drinking. In this work, we synthesized Xn with the total yield of 5.0 % in seven steps, and studied its neuroprotective function against oxidative stress-induced neuronal cell damage in the neuron-like rat pheochromocytoma cell line, PC12 cells. Xn displays moderate free radical-scavenging capacity in vitro. More importantly, pretreatment of PC12 cells with Xn at submicromolar concentrations significantly upregulates a panel of phase II cytoprotective genes as well as the corresponding gene products, such as glutathione, heme oxygenase, NAD(P)H:quinone oxidoreductase, thioredoxin, and thioredoxin reductase. Mechanistic study indicates that the alpha, beta-unsaturated ketone structure in Xn and activation of the transcription factor Nrf2 are key determinants for the cytoprotection of Xn. Targeting the Nrf2 by Xn discloses a previously unrecognized mechanism underlying the biological action of Xn. Our results demonstrate that Xn is a novel small molecule activator of Nrf2 in neuronal cells, and suggest that Xn might be a potential candidate for the prevention of neurodegenerative disorders.

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