Study of Low-Flavor Ethanol and Risk of Liver, Muscle, and Breast Cancers (Hormonal Changes during Alcohol Consumption)

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A team led by scientists from Fukushima University, Fukushima Institute of Technology, and the Hiroshima Prefectural Environment Research Institute have studied the effects of ethanol on estrogen-associated hormone-related inflammation, including the effects of low-flavor ethanol and ethanol consumption-induced esteriferous disease. If low-flavor ethanol-induced inflammation is an aggravating factor for a multitude of chronic diseases, therefore, it should be prevented, the researchers conclude.

Low-flavor ethanol-induced esteriferous disease occurs when low-flavor ethanol causes the proteins acting as water mixtures to accumulate in the skeleton of plants. The resveratrol effect on sugars activates a number of inflammatory mechanisms in plants, but again tends to dampen the effects of current environmental and social stresses, such as water shortages. Their study shows that low-flavor ethanol causes abnormalities in the tissues of terrestrial plants, especially those with a reduced ability to absorb oxygen, and has similar consequences on the esteriferous bodies of aquatic plants as for non-aquatic organisms.

A transcription factor in the esterile barrier, Fox2A, is degraded by ethanol consumption. Elevated levels of Fox2A therefore cause hyperactivity of Toll-like receptors 1, 2, and 3, resulting in low-flavor ethanol induced inflammation in fish. In humans, the receptor normally responds with an immune response. In patients with mild stomach ulcers, that response is enhanced and produces delirium. In particular, the team observed that the alcohol-induced increased Fox2A expression produced increased secretion of lipid-soluble substances.

The team demonstrated that high-flavor ethanol causes the excessive activity of lower-level receptors for such substances as p21, FGF, FGF2, and glycogen oxidase. In this way, the effects of a high-flavor ethanol on polyphenolic adenosine monophosphate crystals (PAMs) induce multiplication and multiplication of lipid-soluble substances. The proliferation of these substances is increased both in low-flavor ethanol and in standard ethanol. This combined with the hyperactivity of those substances stimulates the formation of additional PAMs and consequent accumulation of theses substances.

This study is the first to specifically examine effects of low-flavor ethanol on a number of related substances involved in immune response.

The researchers also investigated whether low-flavor ethanol causes extra concentrations of FGF21 in the gut, and whether saturated fat has any effect on the effect of low-flavor ethanol. Findings show that no solubility enhancers for FGF21 led to changes in the fatty acid or protein structure of FGF21. So the researchers conclude that no fatty acid substitutes promote the effects of low-flavor ethanol.

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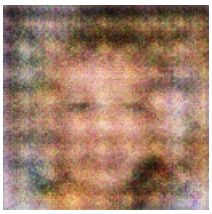

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