Ethanol may Increase Musa Bloc in the Liver

Authors: Kevin Nguyen Frederick Turner Ryan Lucas Michelle Gill Judy Williams

Published Date: 05-07-2015

University of California-Santa Cruz

School of Environmental Studies

This timely article, published by The European Journal of Gastroenterology (H/1), is a cautionary tale for the use of ethanol in fuel: ethanol might increase specific markers of inflammation, including monosodium urate crystals (MUS). The effects of ethanol on MUS were assessed in a rodent model of â€œcardiovascular diseaseâ€ (CVD). In mice, dose-dependent changes in resistance and tolerance of cardiovascular processes occurred as a result of a decrease in ethanol dosing and/or an increase in MUS production.

Cardiovascular researchers have been increasingly concerned about the effects of ethanol on the cardiovascular system. ETH researchers previously established that ethanol was increasing some forms of parathyroid hormone (the endogenous antagonist of the parathyroid hormone), inducers of serotonin, and mediators of depression and hypocalcemia.

ETH researchers have now also determined that ethanol consumption in humans causes changes in MUS in the liver. In a mouse model of CVD, mice that consumed three grams (kg) per day of ethanol received increased production of MUS. In subjects of the control group that drank one kilo of beer or on September 1st and drank four kg of beer per day, alcoholic cirrhosis (bereavement) occurred in the following months.

The number of MUS crystals in the liver at the beginning of the experiment fell compared to liver assessed at six months. However, at nine months, the number of monosodium urate crystals increased in every single group in mice who consumed ethanol compared to control groups. In study mice treated for 12 weeks with an ethanol compound and untreated mice, the total number of musas increased more than threefold. All mice were given a compound that was equivalent to 21.3 grams of ethanol, (Twenty-four cubes of 42mmÂ² bottled distilled Vodka).

The study found that elevated levels of mono- and thiamine (Mth) found in an initial period of ethanol consumption was associated with alterations in vascular and endothelial functions, irrespective of alcohol utilization. In some case models, genetically selected mice that adhered to the alcohol criteria tested had low amounts of musas and no changes in blood lipids, insulin resistance, or fatty acid oxidation.

The authors conclude that the implications of these results are that the liver contains hepatic distans, or activators of Mth and resistant parathyroid hormone, that induce monosodium urate crystal formation as a result of ethanol consumption. In humans, potential cardiovascular risks associated with ethanol consumption can be more easily assessed and reduced than otherwise possible by ethanol replacement.

The observations made in this article show that ethanol consumption may alter the liver microbiome, leading to increased musa proliferation, no matter the type of alcohol consumed. The authors note that alcohol is a monosodium urate crystal-producing enzyme. Hydrogen peroxide, an oxidant produced by alcohol metabolism, induces an Mth-related decrease in lipids. This suggests that the liver cannot eliminate lipids from the blood efficiently when an alcoholic increases his calorie intake. Conversely, increased alcohol concentration suggests that an increased use of Mth can potentially lead to liver dysfunction. Alcohol consumption, in other words, may raise these inflammatory factors in the liver.

Physicians and researchers will definitely benefit from holding meetings that bring together specialists and various fields of study to explain potential aspects of ethanolâ€™s effect on the liver microbiome, and to explore possible strategies to minimize the presence of fungi, to inhibit or to reduce the growth of the musas.

A Close Up Of A Red And White Fire Hydrant