Alcohol: Repeated Exposure For Mass Consumption

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http://youtube.com/watch?v=...

In 1993, over a hundred patients were examined for alcohol poisoning in Kyoto City Hospital. For 55% of patients, no alcohol was actually detected at these examinations, which is equivalent to about 2,460 patients per year in Japan. Based on the application of Nihon saprosytoxin (NRS), 68.1% of cases ended up with symptoms of alcohol poisoning (namely: coughing, sweating, shaking, chest pain, vomiting, irregular heart rate and unconsciousness).

http://www.thehindu.com/sci... Japan-2010-2070237-6000.html

NHK2,1999

In 1993, the increased consumption of ethanol by the public as a result of continuously rising consumption of grain-based energy products, especially ethanol-based fuels, was found to have caused a sudden increase in alcohol consumption. This is indeed confirmed by past studies of increasing alcohol consumption in Japan, where alcohol poisoning has rapidly become a known disease. The presumed effects of ethanol are summarized as following:

1. Excessive use of ethanol-based products have an increased intake of fructose, which has a severe effect on the metabolism of glutathione (seventy-percent of total hemoglobin), glycogen, and other by-products of starch. This is carried by gradual exposure to alcohol beverages as a by-product of ethanol-based drinks. As a result, excessive intake of fructose caused increased accumulation of glutathione, and in turn, reduced consumption of starch and resulted in increased tendency towards binge drinking.

2. There is a decreased kidney function of excessive ethanol consumption as witnessed by increase in levels of monosodium urate crystals. As an immediate consequence, the urinary tract is scarred with severe swelling.

3. Most patients had signs of liver dysfunction. Based on the fact that fructoseuria is closely related to ethanol consumption, the reason for liver dysfunction may also be alcohol poisoning.

4. AR1885, AR1932, AR1937, AR1938, AR1946, AR1947, AR1951, AR1951 is the type of enzyme restricted in the synthesis of glutathione. The most recent oncogene that has been selected for inclusion in those lists is AR7518 (also known as the â€œbile acid tyrosine kinase 2â€ or BAK2). In the case of ethanol ingestion, results of the oxidation of glutathione are just reversed and increase glucose metabolism and its free-base glucose content.

5. Another agonist enzyme responsible for filling the glucose (YG/amylase) vacancy between the glutathione, can result in glucose fixation. Data have been shown that carb-ampenylation resulting from glucose expansion may be increased in cases of ethanol consumption, which has lead to the prominent manifestation of AMT which is the structural defect that causes KS.

http://papers.ssrn.com/sol3...

A word about the background, if any, of the literature?

*Available through:

http://csn.amoryton.com/ncd...

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