Fructose type and non-alcoholic fatty liver disease

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Thank you for your interest in our paper. As highlighted, the association of added fructose and sugar-sweetened beverages with non-alcoholic fatty liver disease (NAFLD) is well-established. The National Health and Nutrition Examination Survey (NHANES) data have demonstrated a link between increased added sugar consumption and a higher prevalence of NAFLD and obesity. Numerous epidemiological studies have also indicated a dose-response relationship between sugar-sweetened beverage consumption and both NAFLD prevalence and incidence. Consumption of fructose causes harmful changes to the composition of the gut microbiome.

The Framingham Heart Study reconfirmed that frequent consumers of sweetened beverages face a significantly elevated risk of developing NAFLD compared to non-consumers. Data from experimental studies have also consistently aligned with these findings. Notably, research involving the administration of sugar-sweetened beverages to healthy subjects resulted in increased liver fat over time measured by magnetic resonance spectroscopy. Conversely, studies restricting added fructose intake in children with high baseline fructose consumption demonstrated a reduction in liver fat on magnetic resonance spectroscopy. Both experimental and clinical studies have suggested an association between fructose intake and NAFLD. There is a clear association between fructose intake of added sugars and increases in obesity and NAFLD. Therefore, it is recommended to reduce the amount of refined carbohydrates, especially fructose in patients with NAFLD.

However, the results of studies on the effect of fructose beyond added sugars, including fructose contained in fruit, on the development of NAFLD have been more diverse. The role of fructose in fruits and added fructose (industrial corn syrup) is thought to be very different. Epidemiological data from Finland revealed an intriguing inverse relationship between fructose
intake from fruits and NAFLD. This contradictory observation might be attributed to higher levels of fruit consumption and the potential mitigating components against fructose's adverse effects. Although fruit also contains fructose, it is less likely to cause metabolic syndrome because it is low in fructose and contains components that can combat the effects of fructose. In the Rotterdam Study of 3,882 participants, monosaccharide and disaccharide intake showed an inverse correlation with NAFLD prevalence, but this effect disappeared after adjusting for metabolic covariates and BMI. Considering the complexity of this relationship, recent systematic reviews have underscored the insufficiency of data on the overall impact of fructose, including natural sources, on NAFLD.
References