Study offers insight to how fructose causes obesity, metabolic syndrome

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A group of scientists from across the world have come together in a just-published study that provides new insights into how fructose causes obesity and metabolic syndrome, more commonly known as diabetes.

In this study which was performed in lab animals, researchers found that fructose can be metabolized by an enzyme that exists in two forms. One form appears to be responsible for causing how fructose causes fatty liver, obesity, and insulin resistance. The other form may actually protect animals from developing these features in response to sugar. These studies may provide important insights into the cause of the prediabetic condition known as "metabolic syndrome", which currently affects more than one-quarter of adults in the United States.

The study, "Opposing effects of fructokinase C and A isoforms on fructose-induced metabolic syndrome in mice" was published today in the journal Proceedings of the National Academy of Sciences. Richard Johnson, MD, the senior author of the study and Chief of the Division of Renal Diseases and Hypertension at the University of Colorado School of Medicine said the findings are significant because we now have a better understanding of how fructose causes obesity and other illnesses.

"These studies provide new insights into how fructose may contribute to the development of obesity and diabetes. In particular, the identification of contrasting roles for two enzymes that are involved in fructose metabolism was surprising and could be important in understanding why some individuals may be more sensitive to the metabolic effects of fructose than others."

Previous research has shown that fructose intake in added sugars such as sucrose and high fructose corn syrup is strongly linked to the epidemic rise in obesity and nonalcoholic fatty liver disease.

Fructose intake also causes features of metabolic syndrome in laboratory animals and humans. It is known to cause visceral (organ) fat accumulation and insulin resistance compared to starch based diets even when calories are kept even.

Provided by University of Colorado Denver