

High fructose diets could cause immune system damage

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New research by Swansea scientists in collaboration with researchers at the University of Bristol and the Francis Crick Institute in London has indicated that consuming a diet high in the sugar fructose might prevent the proper functioning of peoples' immune systems in ways that has, until now, largely been unknown.

Fructose is commonly found in sugary drinks, sweets and processed foods and is used widely in food production. It is associated with obesity, type 2 diabetes and [non-alcoholic fatty liver disease](#) and its intake has increased substantially throughout the developed world in recent years. However, understanding the impact of fructose on the immune system of people who consume it in high levels, has been limited until now.

The new study published in the journal *Nature Communications* shows that fructose causes the [immune system](#) to become inflamed and that process produces more reactive molecules which are associated with inflammation. Inflammation of this kind can go on to damage cells and tissues and contribute to organs and body systems not

working as they should and could lead to disease.

The research also brings a deeper understanding about how fructose could be linked to diabetes and obesity—as low- level inflammation is often associated with obesity. It also builds on the growing body of evidence available to public health policy makers about the [damaging effects](#) of consuming high levels of [fructose](#).

Dr. Nick Jones of Swansea University's Medical School said: "Research into different components of our diet can help us understand what might contribute to inflammation and disease and what could be best harnessed to improve health and wellbeing."

Dr. Emma Vincent in the Bristol Medical School: Populational Health Sciences (PHS) added: "Our study is exciting because it takes us a step further towards understanding why some diets can lead to ill health."

More information: Nicholas Jones et al. Fructose reprogrammes glutamine-dependent oxidative metabolism to support LPS-induced inflammation, *Nature Communications* (2021). [DOI: 10.1038/s41467-021-21461-4](#)

Provided by University of Bristol

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