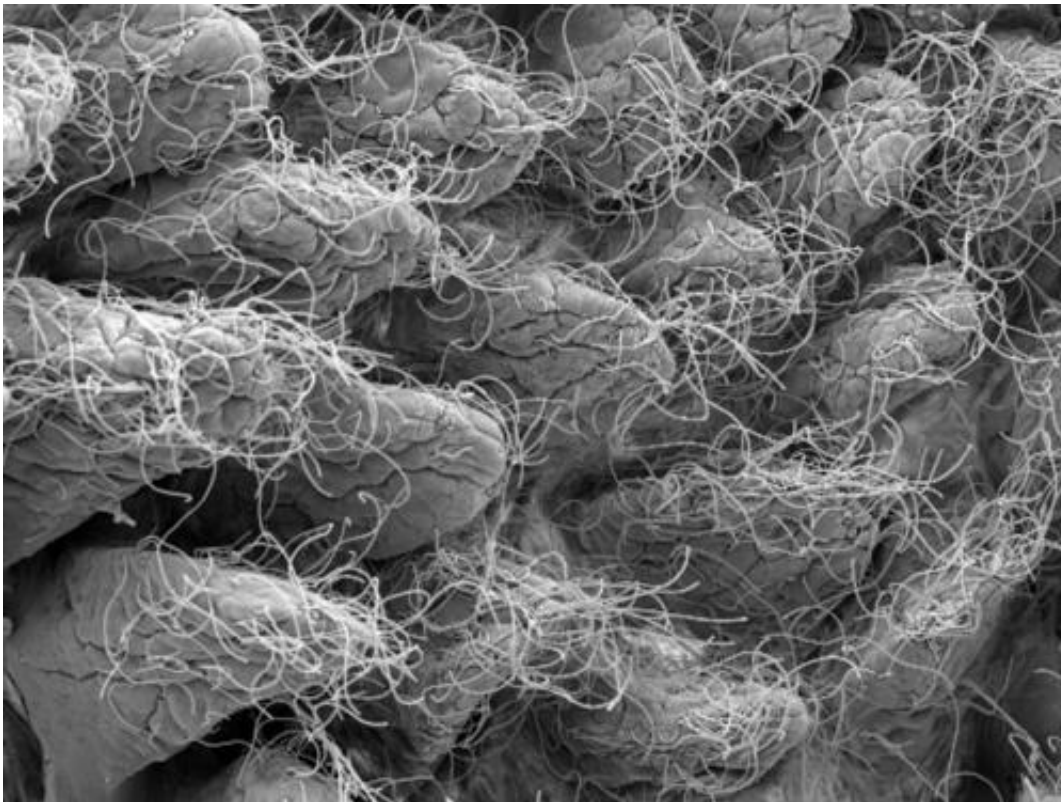


# Artificial sweeteners linked to abnormal glucose metabolism

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Gut microbiota. Credit: Weizmann Institute of Science

Artificial sweeteners, promoted as aids to weight loss and diabetes prevention, could actually hasten the development of glucose intolerance and metabolic disease; and they do it in a surprising way: by changing the composition and function of the gut microbiota – the substantial population of bacteria residing in our intestines. These findings, the

results of experiments in mice and humans, were published today in *Nature*. Among other things, says Dr. Eran Elinav of the Weizmann Institute's Immunology Department, who led this research together with Prof. Eran Segal of Computer Science and Applied Mathematics Department, the widespread use of artificial sweeteners in drinks and food may be contributing to the obesity and diabetes epidemic that is sweeping much of the world.

For years researchers have been puzzling over the fact that non-caloric [artificial sweeteners](#) do not seem to assist in weight loss, and some studies have suggested they may even have an opposite effect. Graduate student Jotham Suez in Elinav's lab, who led the study, collaborated with graduate students Tal Korem and David Zeevi in Segal's lab and Gili Zilberman-Shapira in Elinav's lab in discovering that artificial sweeteners, even though they do not contain sugar, nonetheless have a direct effect on the body's ability to utilize glucose. Glucose intolerance – generally thought to occur when the body cannot cope with large amounts of sugar in the diet – is the first step on the path to metabolic syndrome and adult-onset diabetes.

The scientists gave [mice](#) water laced with the three most commonly used artificial sweeteners – in the equivalent amounts to those permitted by the FDA. These mice developed [glucose intolerance](#), as compared to mice that drank water, or even sugar water. Repeating the experiment with different types of mice and different doses of the sweeteners produced the same results – these substances were somehow inducing glucose intolerance.

Next, the researchers investigated a hypothesis that the gut microbiota are involved in this phenomenon. They thought the bacteria might do this by reacting to new substances like artificial sweeteners, which the body itself may not recognize as "food." Indeed, artificial sweeteners are not absorbed in the gastrointestinal tract, but in passing through they

encounter trillions of the bacteria in the gut microbiota.



Weizmann Institute research shows that artificial sweeteners promote glucose intolerance in a surprising way: by changing the composition and function of the gut microbiota. Credit: Weizmann Institute of Science

The researchers treated mice with antibiotics to eradicate many of their gut bacteria; this resulted in a full reversal of the artificial sweeteners' effects on [glucose metabolism](#). Next, they transferred the microbiota from mice that consumed artificial sweeteners to 'germ-free' mice – resulting in a complete transmission of the glucose intolerance into the recipient mice. This, in itself, was conclusive proof that changes to the gut bacteria are directly responsible for the harmful effects to their host's metabolism. The group even found that incubating the microbiota

outside the body, together with artificial sweeteners, was sufficient to induce glucose intolerance in the sterile mice. A detailed characterization of the microbiota in these mice revealed profound changes to their bacterial populations, including new microbial functions that are known to infer a propensity to obesity, diabetes and complications of these problems in both mice and humans.

Does the human microbiome function in the same way? Elinav and Segal had a means to test this as well. As a first step, they looked at data collected from their [Personalized Nutrition Project](#), the largest human trial to date to look at the connection between nutrition and microbiota. Here, they uncovered a significant association between self-reported consumption of artificial sweeteners, personal configurations of gut bacteria and the propensity for glucose intolerance. They next conducted a controlled experiment, asking a group of volunteers who did not generally eat or drink artificially sweetened foods to consume them for a week and then undergo tests of their [glucose levels](#) as well as their gut microbiota compositions.

The findings showed that many – but not all – of the volunteers had begun to develop glucose intolerance after just one week of artificial [sweetener](#) consumption. The composition of their [gut microbiota](#) explained the difference: The researchers discovered two different populations of human gut bacteria – one that induced glucose intolerance when exposed to the sweeteners, the second that had no effect either way. Elinav believes that certain bacteria in the guts of those who developed glucose intolerance reacted to the chemical sweeteners by secreting substances that then provoked an inflammatory response similar to sugar overdose, promoting changes in the body's ability to utilize sugar.

Segal: "The results of our experiments highlight the importance of personalized medicine and nutrition to our overall health. We believe

that an integrated analysis of individualized 'big data' from our genome, microbiome and dietary habits could transform our ability to understand how foods and nutritional supplements affect a person's health and risk of disease."

Elinav: "Our relationship with our own individual mix of [gut bacteria](#) is a huge factor in determining how the food we eat affects us. Especially intriguing is the link between use of artificial sweeteners – through the bacteria in our guts – to a tendency to develop the very disorders they were designed to prevent; this calls for reassessment of today's massive, unsupervised consumption of these substances."

**More information:** Artificial sweeteners induce glucose intolerance by altering the gut microbiota, *Nature*, [dx.doi.org/10.1038/nature13793](https://doi.org/10.1038/nature13793)

Provided by Weizmann Institute of Science

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