

Researchers target gut bacteria to reduce weight gain

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A new therapy that involves engineered gut bacteria may one day help reduce the health problems that come with obesity. Incorporating the engineered bacteria into the guts of mice both kept them from gaining weight and protected them against some of the negative health effects of obesity. Researchers will present their findings today at the American Physiological Society's Inflammation, Immunity and Cardiovascular Disease conference.

More than one-third of adults in the U.S. are obese, putting them at greater risk for conditions such as [fatty liver disease](#)—caused by fatty deposits building up in the liver—and atherosclerosis, the hardening and narrowing of the arteries. Scientists have recently discovered that the microorganisms living in our gut, known as the [gut microbiota](#), play an important role in obesity and may offer a new therapeutic target.

Researchers led by Sean Davies, PhD, associate professor of pharmacology at Vanderbilt University, are studying whether obesity-related diseases might be treated or even prevented by altering the gut microbiota. To find out, they engineered gut bacteria that produce a small lipid that helps suppress appetite and reduce inflammation. People who are obese typically produce less of this lipid, which is made by the small intestine.

"We have previously shown that this approach with engineered bacteria could inhibit obesity when standard mice were fed a high-fat diet," Davies said. "Our new studies focused on mice highly prone to develop

atherosclerosis and fatty liver disease, and we showed that the engineered bacteria were beneficial not only in inhibiting obesity, but also in protecting against fatty liver disease and somewhat against atherosclerosis."

The researchers found that standard mice fed a [high-fat diet](#) while also receiving the engineered bacteria via drinking water gained less body weight and body fat than mice given standard drinking water or control bacteria. They also gave the engineered bacteria to mice with increased susceptibility to atherosclerosis and fatty liver disease. These mice accumulated less fat in the liver and showed reduced expression of markers of liver fibrosis, compared to mice that did not receive the treatment. The treated mice also exhibited a modest trend toward reduced atherosclerotic plaques.

"Some day in the future, it might be possible to treat the worst effects of obesity simply by administering these bacteria," Davies said. "Because of the sustainability of [gut bacteria](#), this treatment would not need to be every day."

More information: Davies will present "Altering the microbiota for weight control" at the Inflammation and Hypertension during Pregnancy and Gender Differences symposium on Friday, Aug. 26, from 8:30 to 9 p.m. in the Westminster III room of the Westin Westminster Hotel.

Provided by American Physiological Society

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