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Bacteria from Lean Humans Can Slim Obese Mice

A new study shows how interactions between diet, body mass, and gut microbiota underlie metabolic disturbances more than previously thought.

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Graduate student Vanessa Ridaura and colleagues at the Center for Genome Sciences and Systems Biology, University of Washington School of Medicine reported in the September 6 issue of Science that mice lacking bacterial colonies of their own that received gut bacteria from obese humans put on more weight and accumulated more fat than mice that were given bacteria from the guts of lean humans.

To directly test the influence of the human gut microbiome on obesity, the investigators sampled microbes living in the guts of human fraternal and identical twins, one of whom was lean while the other, obese. They introduced these microbes into germ-free mice fed low-fat mouse chow, as well as diets representing different levels of saturated fat and fruit and vegetable consumption typical of the U.S. diet. Increased total body and fat mass, as well as obesity-associated metabolic phenotypes, were transmissible with uncultured fecal communities and with their corresponding fecal bacterial culture collections.

“The first thing that Vanessa identified in these mice, which were consuming a standard mouse diet, was that the recipients of the obese twins' microbiota gained more fat than the recipients of the lean twins' microbiota,” Jeffrey Gordon, M.D., director of the Center and a co-author of the Science report, explained. Since, he said, the differences could not be attributed to the amount of food the mice consumed, “there was something in the microbiota that was able to transmit this trait. Our question became: What were the components responsible?"

To perform what Dr. Gordon called “The Battle of the Microbiota,” the investigators housed mice that had received microbes from a lean twin (Ln mice) with mice that were given microbes from an obese twin (Ob mice). “Mice—delicately put—exchange their microbes readily,” said Dr. Gordon, referring to coprophagia, or the consumption of feces.

When Ridaura and her colleagues housed Ln mice with Ob mice for 10 days, they discovered that the Ob mice—affected by their cage mates’ microbes—slimmed down, adopting the “leaner” metabolism of the Ln mice. Ln mice, on the other hand, appeared unaffected and maintained their own metabolism, they say. The “rescue” of mice from obesity was correlated with colonization of specific members of *Bacteroidetes* bacteria that were part of the Ln biota, and, importantly, was diet-dependent. Only those mice eating a low-saturated fat, high fruit and vegetable diet became colonized with the Ln–associated bacteria. These animals did not become obese.

These findings suggest that more complex interactions between diet, body mass, and gut microbiota underlie human metabolic disturbances than previously understood. The mouse models employed by investigators could be used to identify other aspects of how the human gut microbiota and our diets influence human health.

“We now have a way of identifying such interactions, dependent on diet, and thinking about what features of our unhealthy

diets we could transform in ways that would encourage bacteria to establish themselves in our guts, and do the jobs needed to improve our well-being,” said Dr. Gordon. “In the future, the nutritional value and the effects of food will involve significant consideration of our microbiota—and developing healthy, nutritious foods will be done from the inside-out, not just the outside-in.”

The study appears in the September 6 issue of Science with the title, “Gut Microbiota from Twins Discordant for Obesity Modulate Metabolism in Mice”.

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