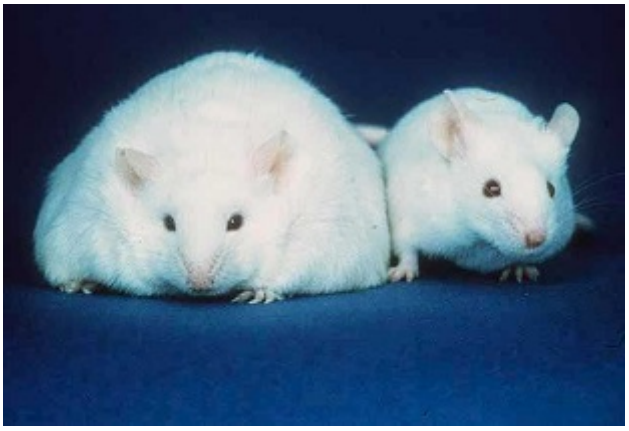
Gut Microbiota–Obesity Link Clarified

Changes in diet can cause gut microbes to produce acetate, which in turn stimulates insulin secretion and obesity in rodents, scientists show.

By Catherine Offord | June 10, 2016



WIKIMEDIA, ORNL

Several studies have linked changes in the gut microbiome to obesity, insulin resistance, and metabolic syndrome, but the details of the link have been unclear. Now, a team led by researchers at Yale University has uncovered one pathway leading from gut microbes to increased food intake and insulin secretion in rodents, pointing to potential therapeutic targets for obesity in humans. The findings were published earlier this week (June 8) in *Nature*.

"Alterations in the gut microbiota are associated with obesity and the metabolic syndrome in both humans and rodents," study coauthor Gerald Shulman of Yale said in a statement. "In this study we provide a novel mechanism to explain this biological phenomenon in rodents, and we are now examining whether this mechanism translates to humans."

The researchers had previously noticed that high-fat diets stimulated increased levels of acetate in rodents' blood streams, and that this increase triggered insulin secretion—but they didn't know where the acetate was coming from. They further explored the link in the current study.

Antibiotic-treated rats and germ-free mice produced relatively low levels of acetate, the team showed, but restoring the animals' normal gut microbiota led to increased acetate production; feeding the rodents a high-fat diet raised acetate levels even further. "Taken together, these experiments demonstrate a causal link between alterations in the gut microbiota in response to changes in the diet and increased acetate production," Shulman said in the statement.

The team also showed that rather than directly stimulating pancreatic cells to secrete insulin, acetate triggers a signal from the brain to the pancreas via the vagus nerve—part of the parasympathetic nervous system—providing a potential target for therapy. "Acetate stimulates beta cells to secrete more insulin in response to glucose through a centrally mediated mechanism," Shulman added. "It also stimulates

secretion of the hormones gastrin and ghrelin, which lead to increased food intake."

Speaking with *Science News,* Jonathan Schertzer of McMaster University in Canada who was not involved in the research called the paper a "tour-de-force," noting that it builds upon other studies on the microbiota-obesity link. "When you find a factor that actually influences metabolism, that's important," he said.

Tags

vagus nerve, rodent, rat, obesity, mouse models, insulin, gut microbiota, ghrelin and acetate

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These studies are fascinating but sadly misleading. if you go to the nature paper then go into extended data figure #4 and also into the supplementary data, you see there was the most acetate in the high fat + high carbohydrate diet. This means that it isn't the high fat that causes obesity but high fat and high carbohydrates together that change acetate levels most significantly.

If you eat a low carb diet, high fat and eat lots of fiber then you will not get this effect. If you reduce your fiber and replace with carbs then your gut microbiota and acetate content with cause insulin resistance, obesity etc...

Everytime a paper says "high fat" unfortunately they mean high fat and high carbohyrdate together. Need to read the find print and that fine print doesn't make it to press. There is an agenda unforunately consciously or subconsciously against those who eat high fat, low carb healthy diets.

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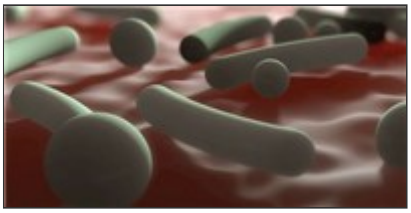

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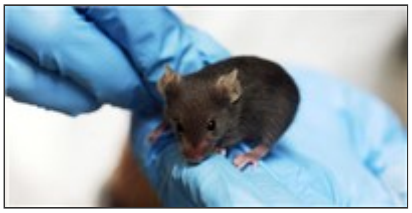

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