Transcript Details
This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/clinicians-roundtable/roseburia-hominis-obesity-treatment/39658/

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

From Gut Microbe to Metabolic Modulator: The Potential Role of R. hominis in Obesity

Dr. Blevins:

You're listening to *Clinician's Roundtable* on ReachMD, and this is an *AudioAbstract*. I'm Dr. Hallie Blevins, and today, we'll be discussing how a depleted gut microbe might hold potential as a next-generation biotherapeutic for obesity.

What if a missing microbe in the gut isn't just a bystander in obesity—but an active player in the metabolic story? That question encouraged researchers in Hong Kong to take a closer look at *Roseburia hominis*, a bacterial species that repeatedly shows up as depleted in individuals with obesity. And in this new study, the team went beyond observing patterns—they tested whether reintroducing this microbe could actually shift metabolic outcomes.

Across a cohort of 100 adults, *R. hominis* levels were noticeably lower in people with obesity, and the lower the abundance, the higher the BMI and triglycerides tended to be. That same relationship held true in a larger dataset too. So the question was: was this bacterium simply a biomarker of a leaner metabolic state, or could it meaningfully influence metabolism when brought back into the system?

To explore that possibility, the team turned to a classic high-fat diet mouse model of obesity. Male C57BL/6 mice were randomized to receive either daily oral doses of live *R. hominis* or a placebo while consuming a high-fat diet. A separate group stayed on normal chow as a lean comparison. For 11 weeks, the researchers tracked body weight, glucose and lipid metabolism, liver and adipose function, and the changing ecosystem of the gut microbiome.

By week 11, mice receiving the *R. hominis* gained significantly less weight than their high-fat diet controls. Their BMI was lower, and their glucose handling looked noticeably better, with lower insulin levels during glucose tolerance testing and a reduced insulin resistance index. Serum triglycerides and total cholesterol had also dropped to healthier levels.

One interesting detail was that the treated mice excreted more calories in their stool. That observation suggested that *R. hominis* may reduce energy extraction from food—an effect that, in a high-calorie context, could meaningfully shape body weight over time.

The liver told a similar story of improvement. High-fat-fed mice typically accumulate lipid droplets, show elevated hepatic triglycerides, and develop features of steatohepatitis. But with *R. hominis* supplementation, the livers had far less lipid accumulation, lower inflammation and steatosis scores. Serum markers of liver injury, including ALT and AST, improved as well.

And the story continued into adipose tissue. White fat deposits in treated mice contained smaller adipocytes, suggesting less hypertrophy. And brown adipose tissue retained its healthier multilocular appearance instead of taking on the "whitened," lipid-laden morphology typical of high-fat feeding. Genes supporting thermogenesis were upregulated, which hints that energy expenditure pathways may have been revived.

The gut microbiome itself also shifted in response to *R. hominis*. Beneficial species often associated with improved metabolic profiles became more abundant, while, certain pathogenic species declined. These shifts mirrored improvements in weight, liver enzymes, and lipid levels, reinforcing the idea that *R. hominis* acts not only directly but also by nudging the entire community toward a healthier ecological balance.

But probably the most intriguing discovery emerged from cellular experiments. Researchers found that *R. hominis* secretes nicotinamide riboside, a precursor to NAD plus. This molecule helped prevent lipid accumulation in liver cells, restored NAD plus levels, and activated the SIRT1 mTOR signaling pathway—which is deeply involved in metabolic regulation. And to go one step further, when SIRT1 was chemically inhibited, part of this protective effect disappeared.

So taken together, these findings paint a compelling picture. *R. hominis* appears to do more than just coexist in the gut—it may actively help guard against diet-induced metabolic dysfunction. While these results are preclinical, they position *R. hominis* as a promising next-generation live biotherapeutic candidate for obesity and related metabolic diseases.

References

Huang W, Zhu W, Lin Y, Chan FKL, Xu Z, Ng SC. *Roseburia hominis* improves host metabolism in diet-induced obesity. *Gut Microbes*. 2025;17(1):2467193. doi:10.1080/19490976.2025.2467193