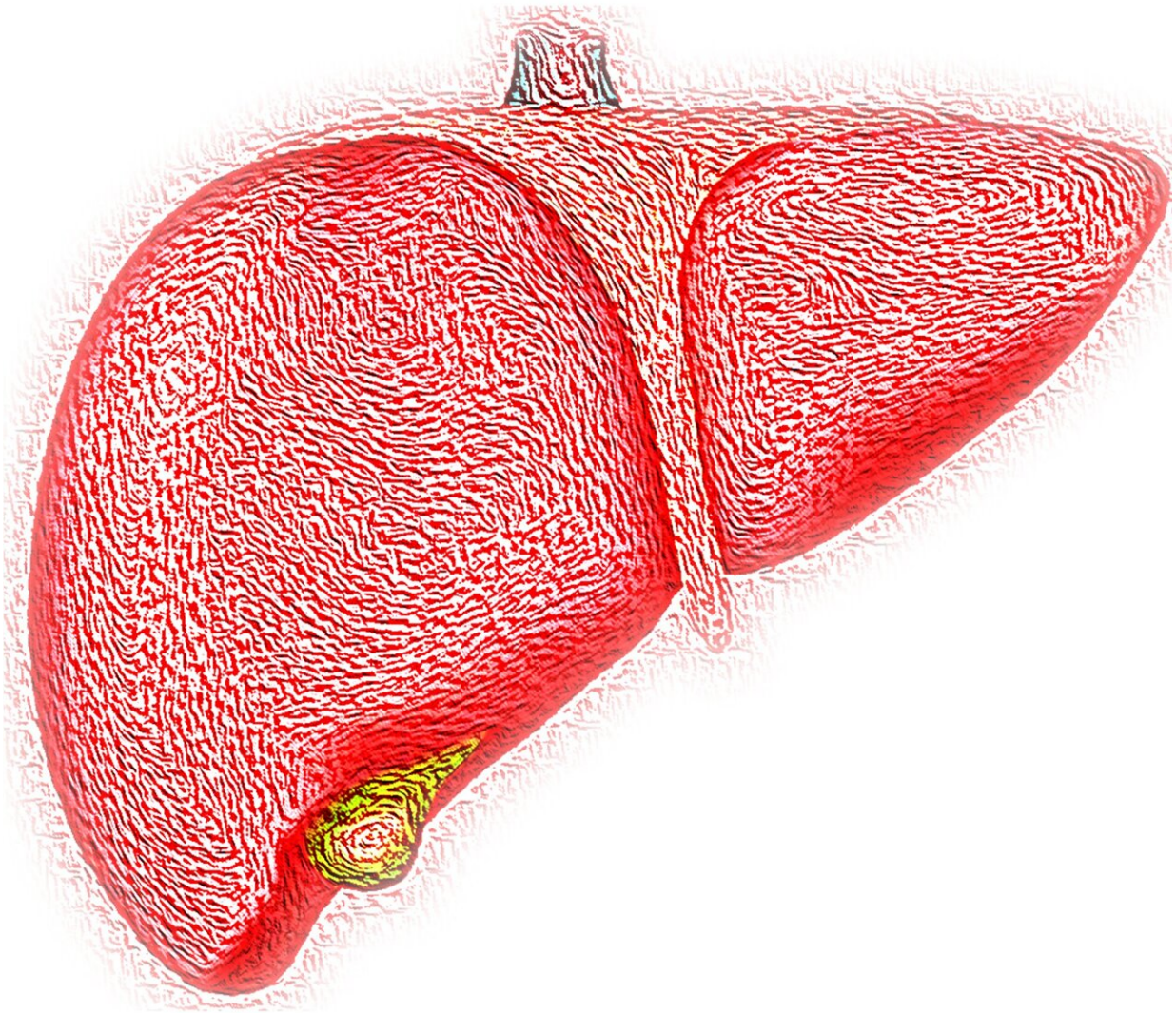


Resistant starch supplement found to reduce liver triglycerides in people with fatty liver disease

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Resistant starch is a non-digestible fiber that ferments in the large intestine, and consumption of it has previously been shown to have a positive effect on metabolism in animal studies.

Now, a four-month randomized controlled trial in people with [non-alcoholic fatty liver disease](#) (NAFLD) indicates that daily intake of resistant starch can alter gut bacteria composition and lower liver triglycerides and liver enzymes associated with liver injury and inflammation. This research appears in the journal *Cell Metabolism*.

NAFLD, caused by a buildup of fat in the liver, affects about 30% of the population worldwide. It can lead to severe liver diseases and contribute to other conditions, such as type 2 diabetes and cardiovascular disease. Currently, there is no approved medicine available to treat NAFLD. Doctors usually recommend dietary changes and exercise to alleviate the conditions.

"We think it would be very meaningful if we can find an effective approach, maybe through identifying new therapeutic targets, to manage NAFLD," says Huating Li, the paper's co-corresponding author at Shanghai Sixth People's Hospital.

Previous research has suggested that NAFLD is associated with perturbed [gut microbiota](#). For example, people with early-stage NAFLD already have an altered gut bacteria profile. So, Li and her team wanted to investigate if resistant starch—a type of fiber known to encourage the growth of beneficial gut bacteria—can help treat NAFLD.

The team recruited 200 NAFLD patients and provided them with a balanced dietary plan designed by a nutritionist. Among them, 100 patients also received a resistant starch powder derived from maize while the other 100 received calorie-matched non-resistant corn starch as a control. They were instructed to drink 20 grams of the starch mixed with 300 mL water (1 ¼ cups) before meals twice a day for four months.

After the four-month experiment, participants who received the resistant starch treatment had nearly 40% lower liver triglyceride levels compared to patients in the control group. In addition, patients who had the resistant starch treatment also saw reductions in liver enzymes and inflammatory factors associated with NAFLD. Importantly, these benefits were still apparent even when statistically adjusted for weight loss.

"Our study shows resistant starch's impact in improving patients' liver conditions is independent of body weight changes," says Yueqiong Ni, the paper's co-first author at Shanghai Sixth People's Hospital and Leibniz Institute for Natural Product Research and Infection Biology—Hans-Knöll-Institute (HKI) in Germany.

By analyzing patients' fecal samples, the team found the resistant starch group had a different microbiota composition and functionality compared with the control. In particular, the treatment-group patients had a lower level of *Bacteroides stercoris*, a key bacterial species that can affect fat metabolism in the liver through its metabolites. The reduction in *B. stercoris* is strongly linked to the decrease in liver triglyceride content, [liver enzymes](#), and metabolites observed.

When the team transplanted fecal microbiota from resistant [starch](#)-treatment patients to mice fed with a high-fat high-cholesterol diet, the mice saw a significant reduction in liver weight and liver triglyceride levels and improved [liver](#) tissue grading compared with mice that

received microbiota from the control group.

"We are able to identify a new intervention for NAFLD, and the approach is effective, affordable and sustainable. Compared with strenuous exercise or [weight loss](#) treatment, adding [resistant starch](#) to a normal and balanced diet is much easier for people to follow through," Li says.

More information: Weiping Jia & colleagues, Resistant starch decreases intrahepatic triglycerides in patients with NAFLD via gut microbiome alterations, *Cell Metabolism* (2023). DOI: [10.1016/j.cmet.2023.08.002](https://doi.org/10.1016/j.cmet.2023.08.002). [www.cell.com/cell-metabolism/f ... 1550-4131\(23\)00297-8](https://www.cell.com/cell-metabolism/fulltext/S1550-4131(23)00297-8)

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