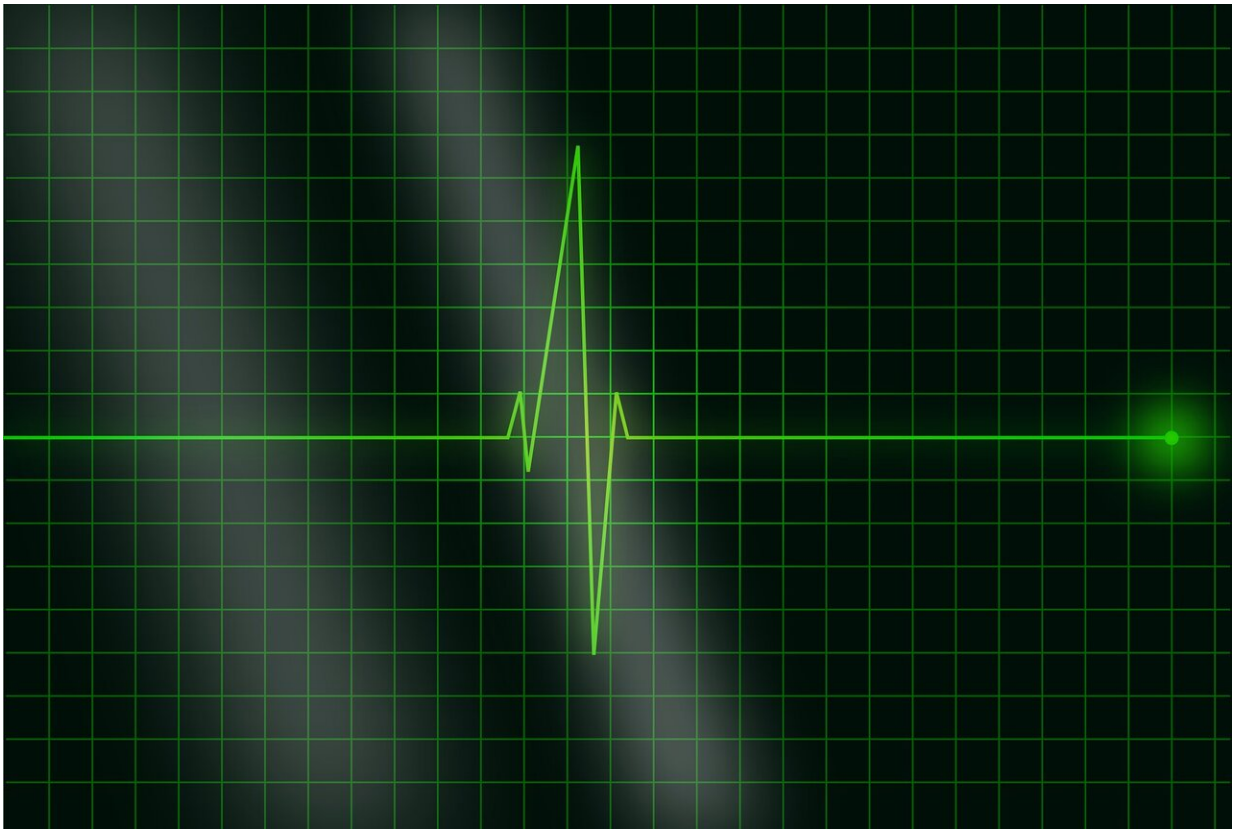


A potential protector against a mild heart attack's aftereffects on metabolism

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A new study in mice shows transplanted brown fat can reduce type 2 diabetes risk factors after a heart attack, an encouraging finding for scientists who hope to apply the so-called "good" fat's beneficial

properties to drugs that can help prevent health problems.

In the study, transplanting [brown fat tissue](#) into the abdomens of obese [mice](#) protected the animals from developing glucose intolerance, a hallmark of type 2 diabetes, after a mild [heart attack](#).

Gene activation linked to negative effects after the [heart](#) attack was dampened in the transplanted mice, suggesting that brown fat—or adipose—tissue "talks" to other tissue in the body in ways that affect a variety of metabolism-related processes. The research team is continuing to tease out the substances and mechanisms behind that cross talk and how it affects whole-body physiology.

"In this study, the mice transplanted with brown adipose tissue were still obese but more metabolically healthy. The heart attack-induced glucose intolerance was negated by brown adipose tissue. The findings make a pretty powerful statement," said senior study author Kristin Stanford, associate professor of physiology and cell biology in The Ohio State University College of Medicine.

"We think brown fat is secreting something, and if we can identify what's being released we can target that as a therapeutic."

The research is published online in the *International Journal of Obesity*.

Clinical research has shown that after a mild heart attack, people are more likely to develop [insulin resistance](#) and glucose intolerance, and are consequently more susceptible to having a second heart attack. Stanford said that what remains unclear is the cause for those increased risks: Does the first cardiac event itself make people more insulin resistant, or does the condition develop because people tend to be more sedentary after a heart attack?

"Our primary thought process was, if we could improve glucose metabolism and reduce insulin resistance, would that have a protective effect later?" said Stanford, whose lab is based in Ohio State's Davis Heart and Lung Research Institute.

All mice in the study were fed a [high-fat diet](#) for eight weeks before being divided into experimental or control groups. Researchers transplanted brown fat from donor mice into the abdomens of the experimental group. Sixteen weeks later, half of all of the mice underwent a surgery in which one coronary artery was obstructed, inducing a mild heart attack.

The mice, all males, were kept on the high-fat diet and monitored for 24 weeks after the heart attack. At this point, the mice that had a heart attack but did not receive brown adipose tissue transplants had developed type 2 diabetes. The mice that had received brown adipose tissue transplants, while still obese, maintained normal glucose tolerance.

"These results showed brown adipose tissue was protective against glucose intolerance even throughout the duration of the heart attack and the massive high-fat diet these mice were on for 40 or so weeks," Stanford said.

The transplanted tissue had additional long-term protective effects against problems seen in other mice after the heart attack, staving off an increase in the size of the heart's left ventricular chamber—a sign of scarring that can lead to heart failure—and preventing a drop in exercise tolerance.

Brown fat is known for its heat-generating properties—it helps keep babies warm, for example—but is hard to come by in the adult human body, with small amounts interspersed between the shoulder blades.

Stanford's lab had previously shown that exercise can drive up a beneficial lipid that comes from brown fat, a finding that helped explain how exercise boosts metabolism at the cellular level.

"We didn't know whether brown adipose tissue would increase duration of exercise, and it did, suggesting that it improves whole-body health, which is an important marker," she said. "We still need to figure out whether the protection comes from something secreted from the brown fat or from just increasing its mass."

The transplantation method could help the researchers in their pursuit of the tissue cross talk theory. Brown fat tissue was lodged in the animals' abdomens among folds of visceral white adipose tissue—the far more abundant type of fat in mammals' bodies.

The team analyzed post-heart attack changes in expression of almost 100 genes linked to inflammation, scarring, insulin signaling, glucose metabolism and specific cell functions in the brown and white fat, the liver, heart and muscles of all of the mice. The increased presence of brown fat negated a host of damaging post-heart attack gene activations, leading the researchers to suggest that brown fat could be a key to preventing metabolic changes that harm the health of obese patients with cardiovascular disease.

"Our hope is that we could eventually translate that to see how increasing [brown adipose tissue](#) could be a potential therapeutic in humans to protect them against insulin resistance or subsequent heart attacks," Stanford said.

"Our data show that brown fat is affecting other tissues—we're just not exactly sure how. There could be several subtle changes working together as opposed to one direct tissue being modified," she said. "Brown fat is such a small tissue, but it is so active."

More information: Carmem Peres Valgas da Silva et al, Brown adipose tissue prevents glucose intolerance and cardiac remodeling in high-fat-fed mice after a mild myocardial infarction, *International Journal of Obesity* (2021). [DOI: 10.1038/s41366-021-00999-9](https://doi.org/10.1038/s41366-021-00999-9)

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