Heart sends out signals to fatty tissue

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Overweight patients are often told that extra pounds are bad for their hearts.

It turns out the heart itself may be "talking" to fat.

That is the thrust of ongoing research by cardiologist Walter J. Koch, a professor at Temple University's Katz School of Medicine.

Koch was awarded a $1 million grant from the American Heart Association this week to explore the heart's apparent ability to regulate the production of fat. His lab found preliminary evidence for this odd phenomenon in mice two years ago, yet the exact mechanism remains unclear.

The key seems to be a gene that carries the recipe for an enzyme called GRK2, which is elevated in patients suffering from heart failure and has been the subject of Koch's research for more than two decades.

When the Temple researchers inhibited the gene in mouse hearts, resulting in lower levels of that enzyme, the animals gained weight when fed a high-fat diet. But when the researchers ramped up the gene's activity, resulting in higher levels of the enzyme, mice given the same high-fat diet were somehow able to stay thin, Koch said.

"We found that the heart can talk to fat" in the lab mice, he said. "We know the heart is secreting something."

The communication signal may be in the form of a hormone or some other type of chemical secretion, he said. Koch and his colleagues plan to use the grant to identify this signal from the heart, and then determine if it is present in people.

What that might mean for human health is unclear, but if the signal turns out to be a hormone, it would not be the only such agent secreted by the heart. The heart also releases a hormone called BNP, which has been used to help diagnose heart failure—when the heart's pumping action is too weak to keep up with the body's demands, resulting in shortness of breath.

In previous research on the GRK2 enzyme, Koch and his colleagues found that when they inhibited its production, mice with heart failure seemed to recover somewhat. Mice with higher levels of the enzyme fared worse.

So on the one hand, GRK2 seems to play a role in helping mice stay thin, but also it contributes to heart failure. Could this have something to do with the "obesity paradox," the controversial finding that heavier patients may fare better after a heart attack?

Koch is not ready to go that far, and cardiologists stress that this apparent contradiction is not a reason to gain weight. On the contrary, extra pounds raise the risk of heart problems in the first place.

Koch is eager to find more answers. The $1 million award is to be distributed over five years.