

Leptin-controlled gene can reverse diabetes

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Researchers have found that even a very little bit of the fat hormone leptin goes a long way when it comes to correcting diabetes. The hormone controls the activity of a gene known as IGFBP2 in the liver, which has antidiabetic effects in animals and could have similar therapeutic effect in humans, according to a report published in the January issue of *Cell Metabolism*.

The new findings confirm what some at least had already suspected: that leptin's antidiabetic effects are independent of the hormone's well-known ability to reduce body weight.

"It was surprising to me how potent leptin was in treating <u>diabetes</u>," said Jeffrey Friedman of Rockefeller University. "It had a highly significant impact at plasma levels that were undetectable."

Earlier studies had shown that leptin treatment effectively corrects high blood sugar and insulin levels in leptin-deficient mice and humans. Leptin's usefulness as a therapy has also been shown in some clinical settings, in people with rare metabolic disorders. But it wasn't clear exactly how the hormone produced in fat tissue acts to improve diabetes.

Studies to address that question had been complicated by the fact that leptin also causes marked weight loss, which by itself can improve diabetes, the researchers explained. To get around that issue in the new study, Friedman and his colleagues first identified the lowest dose of leptin that could correct <u>insulin resistance</u> and diabetes without leading animals to eat less or lose weight.



They then looked to see how that very low-level infusion of leptin changes the activity of genes in the animals' livers. That survey led them to IGFBP2.

Treatments designed to increase IGFBP2 expression in obese and diabetic mice reversed their diabetes. Further study showed that animals treated with the protein responded to insulin three times better than untreated ones.

They also found that leptin-deficient patients do indeed have lower blood levels of IGFBP2 at baseline and that those levels can be raised with low-dose leptin treatment.

Friedman said that future experiments in mice lacking IGFBP2 altogether are needed to confirm that the protein is required for leptin's antidiabetic influence. Now that they know that very high levels of IGFBP2 can act to improve diabetes, they'll also need to explore the effects of normal physiologic levels.

"In summary," the researchers concluded, "we have developed a set of conditions in which leptin treatment potently improves diabetes independent of its ability to correct weight and food intake. This protocol was used to identify IGFBP2 as a leptin-regulated gene whose expression is correlated with leptin's antidiabetic effect...Further studies will reveal whether IGFBP2 shows similar antidiabetic effects in clinical settings."

Provided by Cell Press

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