

# SCIENTIFIC METHOD / SCIENCE & EXPLORATION

## Scientists find cure for type 2 diabetes in rodents, don't know how it works

Despite unexplained mechanism, the new treatment will be easy to try in humans.

by Beth Mole (US) - May 25, 2016 10:40pm UTC

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The cure for type 2 diabetes may be all in your head, a new study in rats and mice suggests.

With a single shot to the brain, researchers can rid rodents of all symptoms of the disease for months. The injection, a relatively low dose of a tissue growth factor protein called fibroblast growth factor 1 (FGF1), appears to reset powerful neural networks that can control the amount of sugar in the blood.

So far, it's not completely clear how exactly FGF1 does that, researchers report in Nature Medicine. Early experiments found that FGF1 didn't appear to lower blood sugar levels in some of the most obvious ways, such as curbing the rodents' appetite and spurring sustained weight loss. Nevertheless, because FGF1 is naturally present in human brains, as well as those of rodents, researchers are hopeful that the lucky shot may translate into a useful treatment.

If nothing else, the FGF1 finding "unmasks the brain's inherent capacity to induce sustained diabetes remission," the authors conclude. But because scientists already have the protocols and know-how to safely deliver FGF1 to human noggins via intranasal routes, moving toward clinical trials seems like a no brainer, they argue.

The authors, led by researchers at the University of Washington, gave FGF1 a crack in animal brain experiments after other studies had seen encouraging results with FGF1's cousins. Those related growth factors can activate some of the same brain signals as FGF1 and lowered blood sugar levels after brain injections in animals. Yet FGF1 may be a more powerful player in the brain, the authors reasoned, because it can trigger an even greater number of brain cell signals. And earlier experiments found that large systemic doses of FGF1 can lower blood sugar levels in mice.

Researchers injected just a tenth of that intravenous dose directly into the brains of mice that were genetically engineered to have a moderate case of type 2 diabetes—which is marked by abnormally high levels of glucose sugar in the blood that damage the body's cells and overall health over time. After a week, blood sugar levels in the mice had completely normalized. And they stayed that way for more than four months—as long as the researchers tracked them. Such dramatic results have only been seen in diabetic patients that have undergone certain bariatric surgeries. When the researchers tested out the FGF1 brain shot in rats, they found the same results.

Amid the treatment's success, the researchers were puzzled by what was going on. Right after the injection, the rats and mice curbed their food intake a little and lost a tiny bit of weight. But that effect vanished after the first week. The rodents then went right back to eating and weighing the same amount as control diabetic rodents. This means that the drop in glucose wasn't due to the rodents eating less or having a healthier weight and metabolism—the sugar drop was due to something else.

Next, the researchers looked for changes in insulin, the hormone that prompts cells to take up glucose and use it for energy. Broken insulin production in the pancreas is the root of type 1 diabetes, and type 2 is spurred by an imbalance in insulin relative to the amount of sugar in the blood (generally caused by poor diet, lack of exercise, and genetics). Injections of insulin, which can treat both types of diabetes, clear out excess amounts of blood glucose. But, an insulin boost wasn't clearing out the rodents' sugar. Insulin levels and the effectiveness of its sugar clearing ways weren't altered by the FGF1 shot in the rodents, the researchers found.

With further digging, the researchers discovered that the liver and skeletal muscles were behind the cut in blood sweetness. The liver had cranked up both glucose breakdown machinery and a system that packs up the sugar for long-term storage. Skeletal muscles were also taking in more glucose. But no other tissue or organs seemed to be involved in sopping up the sweet molecule. To the authors' knowledge, it's the first time scientists have found such a sugar-clearing method.

It's still unclear how exactly FGF1 spurs those changes. Interestingly, the authors noted that rodents with severe type 2 diabetes, which have broken insulin signaling, were not cured by the FGF1 shot. The authors speculate that working insulin signals may be a critical link between FGF1's brain activity and the novel un-sweetening system in the liver and muscles.

The researchers will need to do more work to connect all the dots. And with data from just an initial set of rodent experiments, the findings need to be validated and tested further in more animal and clinical studies. But, for now, the researchers are optimistic that the brain, rather than the blood or the stomach, may be the sweet spot for finding a diabetes cure.

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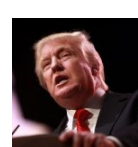
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