Researchers ID Possible Ways to Reverse Type 2 Diabetes and Liver Fibrosis

In a mouse study, they found a pair of proteins that are instrumental in the development of diabetes, one of which helps regulate fibrosis.

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In a pair of new studies, scientists have identified a pair of key proteins that appear to be instrumental in the development of type 2 diabetes; one of them also plays a role in regulating fibrosis (scarring) of the liver. These discoveries in mice may eventually lead investigators to identify treatments that could reverse diabetes and fibrosis in humans.

In one study, published in Nature, scientists conducted their experiments based on the knowledge that fasting causes two proteins, called TET2 and NHF4a, to increase in the liver, which in turn raises the level of blood glucose. In the case of type 2 diabetes, these proteins fail to dissipate after someone eats again as they do in the absence of the disease, thus keeping blood glucose levels abnormally high.

Looking to see whether they could suppress the levels of TET2 and NHF4a and thus prevent diabetes, the study authors injected diabetic mice with viruses that targeted those two proteins and were packaged with small interfering RNAs. This process did indeed interfere with TET2 and NHF4a. Blood glucose and insulin dropped in the animals, stopping diabetes.

In the second study, published in Cell Reports, the study authors found that TET2 played multiple roles in the development of liver fibrosis. This discovery indicated that there may be multiple targets for which to develop drugs that could suppress TET3 in an effort to slow fibrosis or even reverse it.

“Right now, there are no effective drugs for the treatment of fibrosis,” Xuchen Zhang, MD, an associate professor in pathology and coauthor on the fibrosis study, noted in a press release.

To read a press release about the study, click here.

To read the Nature study, click here.

To read the Cell Reports study, click here.
