Researchers uncover potential 'cure' for type 1 diabetes

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Type 1 diabetes could be converted to an asymptomatic, non-insulin-dependent disorder by eliminating the actions of a specific hormone, new findings by UT Southwestern Medical Center researchers suggest.

These findings in mice show that insulin becomes completely superfluous and its absence does not cause diabetes or any other abnormality when the actions of glucagon are suppressed. Glucagon, a hormone produced by the pancreas, prevents low blood sugar levels in healthy individuals. It causes high blood sugar in people with type 1 diabetes.

"We've all been brought up to think insulin is the all-powerful hormone without which life is impossible, but that isn't the case," said Dr. Roger Unger, professor of internal medicine and senior author of the study appearing online and in the February issue of *Diabetes*. "If diabetes is defined as restoration of glucose homeostasis to normal, then this treatment can perhaps be considered very close to a 'cure.' "

Insulin treatment has been the gold standard for type 1 diabetes (insulin-dependent diabetes) in humans since its discovery in 1922. But even optimal regulation of type 1 diabetes with insulin alone cannot restore normal glucose tolerance. These new findings demonstrate that the elimination of glucagon action restores glucose tolerance to normal.

Normally, glucagon is released when the glucose, or sugar, level in the blood is low. In insulin deficiency, however, glucagon levels are

inappropriately high and cause the liver to release excessive amounts of glucose into the bloodstream. This action is opposed by insulin, which directs the body's cells to remove sugar from the bloodstream.

Dr. Unger's laboratory research previously found that insulin's benefit resulted from its suppression of glucagon.

In type 1 diabetes, which affects about 1 million people in the U.S., the pancreatic islet cells that produce insulin are destroyed. As a countermeasure to this destruction, type 1 diabetics currently must take insulin multiple times a day to metabolize blood sugar, regulate blood-sugar levels and prevent diabetic coma. They also must adhere to strict dietary restrictions.

In this study, UT Southwestern scientists tested how mice genetically altered to lack working glucagon receptors responded to an oral glucose tolerance test. The test – which can be used to diagnose diabetes, gestational diabetes and prediabetes – measures the body's ability to metabolize, or clear, glucose from the bloodstream.

The researchers found that the mice with normal insulin production but without functioning glucagon receptors responded normally to the test. The mice also responded normally when their insulin-producing beta cells were destroyed. The mice had no insulin or glucagon action, but they did not develop diabetes.

"These findings suggest that if there is no glucagon, it doesn't matter if you don't have insulin," said Dr. Unger, who is also a physician at the Dallas VA Medical Center. "This does not mean insulin is unimportant. It is essential for normal growth and development from neonatal to adulthood. But in adulthood, at least with respect to glucose metabolism, the role of insulin is to control glucagon.

"And if you don't have glucagon, then you don't need insulin."

Dr. Young Lee, assistant professor of internal medicine at UT Southwestern and lead author of the study, said the next step is to determine the mechanism behind this result.

"Hopefully, these findings will someday help those with type 1 diabetes," Dr. Lee said. "If we can find a way to block the actions of glucagon in humans, then maybe we can minimize the need for insulin therapy."

Dr. Unger said anything that reduces the need for injected insulin is a positive.

"Matching the high insulin levels needed to reach glucagon cells with insulin injections is possible only with amounts that are excessive for other tissues," he said. "Peripherally injected insulin cannot accurately duplicate the normal process by which the body produces and distributes insulin. If these latest findings were to work in humans, injected insulin would no longer be necessary for people with type 1 diabetes."

Provided by UT Southwestern Medical Center

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