

Media Contact: Kelli Hanley
E-mail: kelli.hanley@cshs.org
Telephone: (310) 423-3674

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LABORATORY STUDIES AT CEDARS-SINAI FIRST TO SHOW THAT A NATURAL PROTEIN ANALOG MAY FIX INSULIN-MAKING CELLS ISOLATED FROM THE HUMAN PANCREAS

LOS ANGELES, (November 20, 2003) - For decades, doctors have known that patients who develop higher than normal blood sugar eventually require medication and ultimately need to take insulin, having progressed to what is known as Type 2, or adult-onset diabetes. So when a natural protein analog known as GLP-1 was found to lower blood sugar levels in laboratory mice, researchers began investigating its effectiveness in diabetes patients in clinical trials. Once again, they found that GLP-1 lowered blood sugar and increased insulin production in patients with Type 2 diabetes. But just how GLP-1 pushes cells to produce more insulin has only been partially understood.

Now, laboratory research conducted at Cedars-Sinai Medical Center has shown that GLP-1 not only stimulates the insulin-making capacity of islet cells in the pancreas, but that the compound actually makes new insulin, increases the growth of new islet cells and prevents overworked islets from dying prematurely. The study, reported in the December issue of the journal, *Endocrinology*, (available on-line at <http://endo.endojournals.org>) is the first lab study to apply GLP-1 directly to freshly isolated human islet cells and suggests that GLP-1 may be useful to delay or prevent the onset of Type 2 diabetes.

“Our study shows that GLP-1 is the first compound to actually generate new insulin,” said Riccardo Perfetti, M.D., Ph.D., and Director of the outpatient Diabetes Program at Cedars-Sinai Medical Center. “In other words, it doesn’t just deplete the islet cell by making it work harder to produce more insulin, but it actually fixes the cell’s engine.”

Insulin, a hormone that controls blood glucose levels, is made by islet cells in the pancreas. But when the islet cells begin to fail, not enough insulin is produced, causing blood sugar levels to get too high. This in turn, causes the islet cells to work harder to produce more insulin, ultimately stressing the cells and causing them to die.

But earlier research had shown that GLP-1 increased insulin production and slowed the rate that islet cells died in laboratory mice, which prompted the researchers at Cedars-Sinai to find out whether GLP-1 could actually preserve the function and viability of actual human islets. They found that GLP-1 worked by delaying damage to the human islets’ structure and that the life-span of the cells were significantly increased.

“We found that the islet cells were more efficient when treated with GLP-1, because it prompted them to make insulin only when it was needed,” said Dr. Perfetti.

In the study, two groups of islets were isolated from the human pancreas and cultured in the laboratory for five days. One group was treated with GLP-1 every 12 hours, while the other group served as a control and was not treated with GLP-1. Glucose was added to the islet cultures at the end of the first, third and fifth day of the study and a test was performed to measure the amount of insulin secreted by the cells.

During the five days that the cells were studied, the investigators found that the islets in both groups maintained their shape and structural integrity for one day. However, a progressive loss to the structure of the cell and the numbers of actual cells was observed among islets not treated with GLP-1, with the number of viable cells reduced by 45 percent by day five of the study. Alternatively, the islets treated with GLP-1, were able to maintain their shape and structural integrity for a longer period of time, with only a 15 percent reduction in viable cells by the end of day five.

“This shows that the addition of GLP-1 had a significant effect on cell viability and inhibited the structural deterioration that is characteristic of cells that are dying,” said Dr. Perfetti.

To find out whether GLP-1 was effective to slow down the rate that the islet cells died, the investigators used a specialized staining technique to see how many viable cells remained by day five – or the last day of the study. They found that time was a major factor in both the treated and untreated islets, but that GLP-1 treated cells lived longer, with about 15.5 percent of the untreated cells having died at day 3 as compared to 6.1 percent in the GLP-1 treated cell cultures. On day five of the study, 18.9% of the untreated cells had died, while cell death occurred in only 8.9% of the GLP-1 treated cells.

In addition, when the investigators added glucose to the islet cell cultures to determine whether GLP-1 would stimulate the cells to secrete insulin, they found that GLP-1 treated cells were more sensitive to glucose and secreted more insulin than the untreated islet cells.

“All together, this study shows that GLP-1’s ability to prevent islet cells from dying could possibly be used to prevent Type 2 diabetes,” said Dr. Perfetti.

This study was sponsored by the Max Factor Family Foundation.

Cedars-Sinai Medical Center is one of the largest non-profit academic medical centers in the Western United States. For the fifth straight two-year period, Cedars-Sinai has been named Southern California’s gold standard in health care in an independent survey. Cedars-Sinai is internationally renowned for its diagnostic and treatment capabilities and its broad spectrum of programs and services, as well as breakthrough in biomedical research and superlative medical education. Named one of the 100 “Most Wired” hospitals in health care in 2001, the Medical Center ranks among the top 10 non-university hospitals in the nation for its research activities.

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