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******UT Southwestern diabetes researchers publish key study.

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DALLAS--The lead article in the Aug. 31 issue of the prestigious NEW ENGLAND JOURNAL OF MEDICINE is a report by two Dallas researchers that may resolve a major medical controversy--one with implications for millions of diabetics.

In that article, Drs. Roger Unger and Philip Raskin of The University of Texas Southwestern Medical School and the Dallas Veterans Administration Hospital present new and convincing evidence that the controversial hormone "glucagon" is important in human diabetes.

Although glucagon was discovered in 1923, it was not until Dr. Unger's pioneering research that the potential significance of glucagon in diabetes began to be recognized. Until then, diabetes was considered a "unihormonal" disorder caused by a lack of insulin alone. But Dr. Unger's work indicated that an excess of glucagon also is involved.

As is often the case when a new idea is introduced into science, at first there was a period of "skeptical silence," recalls Dr. Unger. Then a few researchers produced experimental evidence that seemed to contradict his theory and criticism began to grow.

"It was kind of like attacking motherhood to say something other than insulin is involved in diabetes," he says.

But with this newly published study, Dr. Unger feels he has answered the criticism by proving conclusively that glucagon does play an important role in diabetes.

"Nobody argues about the fact that every diabetic has an abnormally high level of glucagon," he explains. "They just argue about how important that abnormality is.

"I think this study convincingly demonstrates that it is important and that correcting the excess of glucagon brings the diabetic's blood sugar levels back to or towards normal. The results suggest a new approach for improving blood sugar regulation in diabetics."

It's high blood sugar levels that are believed to cause the serious and potentially life-threatening complications of diabetes. According to the National Institutes of Health, diabetes currently is the fifth leading cause of death in the United States, and diabetic retinopathy is a leading cause of blindness. Approximately one million Americans have the most severe form of the disease--called "juvenile onset" diabetes--while about nine million more have milder forms.

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In their journal article, the Dallas researchers describe how they placed four juvenile onset-type diabetics on a constant infusion of insulin for seven days. "We gave them a relatively low but constant dose of insulin so their blood sugar was kept high and they were excreting large amounts of sugar in their urine," explains Dr. Unger.

At this point in the experiment, excessive amounts of glucagon were present in the blood, which is the case in all diabetics. After two days of closely monitoring levels of blood sugar and glucagon, the investigators began infusing a constant amount of a brain hormone called "somatostatin," which suppresses the secretion of glucagon.

Almost immediately the levels of glucagon and--more importantly--the levels of blood sugar returned to the normal range. The excess sugar in the urine also disappeared. Only when the researchers began replacing the missing glucagon 48 hours later did the blood sugar levels rise again.

These findings thus confirm that glucagon and insulin have opposite effects--glucagon raises blood sugar, insulin lowers it. "It's kind of like a traffic cop who uses one hand to halt traffic and the other to make it flow," he explains.

Consequently, Dr. Unger regards diabetes as "double trouble":

"In the diabetic there is improper regulation of blood sugar concentrations because the glucagon levels are too high and insulin is much too low. Our work indicates that unless you have both abnormalities, you don't get diabetes in its complete form."

This leads to the conclusion that both abnormalities must be corrected in order to completely control the diabetic's blood sugar levels--a finding that may result in significant improvements in treatment of this common disease.

Conventional insulin therapy has proven inadequate in controlling high blood sugar, Dr. Unger says. "You get very erratic patterns; the blood sugar may go up too high after meals and down too low between meals."

Diabetes researchers have been working for years on development of methods that would deliver the right amount of insulin at the right time, but have had only limited success.

Dr. Unger says this study raises the possibility that a variable rate insulin delivery may not be necessary if glucagon levels are suppressed.

"In this study we had good blood sugar control with a constant rate of delivery. So it may be possible to achieve improved control of blood sugar in diabetics by adding a glucagon suppressor to a constant rate insulin delivery."

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Dr. Unger emphasizes that this has not been proven yet. "The fact that it was done for 48 hours in our study isn't sufficient proof that it could be done for months or years. Moreover, these patients were in a hospital setting, which is a highly controlled environment. They weren't running around outside, so exercise was not a factor.

"I wouldn't want to overstate the potential here, but to me it looks very promising."

For his pioneering work, Dr. Unger has received numerous awards including the Banting Medal, the highest scientific honor given by the American Diabetes Association. Both he and Dr. Raskin are affiliated with the Dallas V.A. Hospital. Dr. Unger is professor of internal medicine at UT Southwestern and Dr. Raskin is associate professor of internal medicine at the Dallas medical school. Dr. Unger joined the Southwestern faculty in 1956.

Support for their study was provided by grants from the Veterans Administration, the National Institutes of Health, the Texas Research Foundation of the Salk Institute, Pfizer Laboratories, Bristol Myers Company, Ciba-Geigy Corporation, Upjohn Company, Eli Lilly and Company and the North Texas Affiliate of the American Diabetes Association.

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