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******Dallas Research Scientist to receive American Diabetes Association's highest honor, the Banting Medal, for major research achievement.*

DALLAS--A noted medical scientist here whose pioneering studies of a little-known hormone called glucagon have turned diabetes research in a promising new direction has been named winner of the prestigious Banting Medal, highest scientific honor bestowed by the American Diabetes Association.

Dr. Roger H. Unger, professor of internal medicine at The University of Texas Southwestern Medical School and chief of medical research at Dallas' Veterans Administration Hospital, will receive the award and deliver the Banting Memorial Lecture during the diabetes association's annual meeting in New York City June 14-17.

Announcement of Dr. Unger's selection for the honor by a scientific committee of the American Diabetes Association was made by Dr. Max Ellenberg, ADA president. Notifying Dr. Unger of his selection by a scientific committee of the association, Dr. Ellenberg told the Dallas scientist "Your pioneer work with glucagon--has established you and the subject at the very forefront of all diabetic investigation at this time."

The Banting Medal is regarded as one of the most prestigious honors among researchers in metabolic disease. The award is named for the late Sir Frederick Banting of Toronto, who with Dr. Charles H. Best discovered the function of insulin in 1922, paving the way for the first successful treatment of the metabolic disease.

Previous winners of the medal, first awarded upon the ADA's founding in 1941, include Dr. Best and Dr. Elliott P. Joslin, for whom Boston's Joslin Clinic is named.

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Dr. Unger, a member of the Southwestern Medical School faculty since 1956, has won numerous awards including the VA's Middleton Award for outstanding research achievement. He also has received the Lilly Award of the American Diabetes Association and has been cited twice by its Texas affiliate "most significant work in his field." He has published scores of scientific papers in the area of carbohydrate metabolism.

Dr. Unger's own research has played a major role in the recent broadened concept of diabetes--that not just one, but two pancreatic hormones malfunction to cause the disorder in which the body loses its normal ability to maintain blood-sugar balance.

During two decades of painstaking investigation, Dr. Unger has compiled striking medical evidence implicating glucagon, a lesser known hormone, in the disease process along with insulin.

Two major scientific publications, the British medical journal Lancet and Science magazine, have just published reports by Dr. Unger and his associates on their glucagon research.

In Lancet, Dr. Unger and Dr. Lelio Orci of the Institute of Histology and Embryology in Geneva outline the "bi-hormonal abnormality hypothesis," which in essence says that diabetes is twice as complicated a disease as has been recognized, and existing insulin therapy deals with only half the problem.

Drs. Unger, Orci and others, writing in Science (Feb. 14), describe animal experiments which "suggest that the development of diabetic hyperglycemia (high blood-sugar) does, indeed, require the presence of glucagon." When both insulin and glucagon are suppressed to unmeasurable concentrations by a new experimental hormone called somatostatin, the excessive blood-sugar disappears--unless glucagon concentrations are restored.

This suggests, they report, "a potentially valuable new approach to the treatment of diabetes."

Somatostatin, still in experimental development, has been used by West Coast scientists in tests of human diabetics with similar results, they note. Somatostatin was discovered last year by Dr. Roger Guillemin at California's Salk Institute.

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Dr. Unger's trailblazing work has laid the basis for wide experimentation into the "other side" of the diabetic syndrome. A key factor in much of the increasing research interest was the development in his Dallas VA hospital laboratories of a complex technique for measuring minute quantities of glucagon in the blood. The radioimmunoassay process, utilizing radioactive iodine to "tag" glucagon molecules, has enabled others to join in the study of glucagon.

Helped greatly by a cooperative rabbit which developed large numbers of glucagon antibodies, the Dallas lab has supplied the rabbit's serum containing these antibodies to dozens of laboratories in many nations.

Although rabbit "30K" has now died, enough of his serum is retained in freezers to supply researchers at the present rate for several more years, Dr. Unger says. Each small vial is sufficient for 1,000 diagnostic tests. In tribute to the animal's contribution, Dr. Unger's assistants have had Rabbit "30K" stuffed and he continues to occupy a special niche in the lab where he made his contribution to science.

Finding a means of glucagon suppression is now a major goal of many diabetes researchers, including Dr. Unger. But, he says, glucagon has become an area of major scientific interest in only the last four years. And hard evidence as to its function was unearthed only recently.

"Until last year, nobody could say that it would have been possible to suppress glucagon," the Dallas scientist notes. "Secondly, nobody could say that if you could suppress glucagon, that it would ameliorate diabetes.

"But now both those questions have been answered. We can suppress glucagon and it does permit a remarkable amelioration. Blood sugar drops dramatically."

Up until only a few months ago, he explained, there was no alternative but to attribute diabetes totally to a lack of insulin. That was before experiments dramatically demonstrated that all glucagon was not coming from pancreatic cells.

Researchers removed the pancreas in animals and glucagon levels remained high--an "impossibility" according to prevailing scientific thought, because the pancreas contained all cells then known to produce both insulin and glucagon.

This sent Dr. Unger and other scientists back to the drawing board. Further search turned up "alpha" cells indentical in structure to those which secrete glucagon ("beta" cells turn out insulin) in the pancreas.

"There are alpha cells in the top of the stomach and the upper small intestine." Dr. Unger says. These new-found cells are believed to provide a backup mechanism for producing glucagon. And their discovery helped prove convincingly that glucagon's presence is required for diabetes to occur--turning upside down the conceptual picture of diabetes, which heretofore had focused solely on the pancreas.

"The minute we found that you are removing only half the glucagon-producing tissue when you remove the pancreas, then it became possible to entertain the notion that glucagon was essestial to diabetes," Dr. Unger said.

Dr. Unger's group has found excessive glucagon levels in more than a dozen forms of diabetes--further evidence, he says, that the overlooked hormone is necessary to the disease process.

The effect of recent studies, in Dallas and elsewhere, is to show that a lack of insulin alone, in the absence of glucagon, does not produce the elevated blood-sugar characteristic of diabetes, Dr. Unger said. Actually, two things are happening in glucose metabolism:

"First, there is a reduced ability to use sugar that comes in your diet, and that's due to insulin lack. That can be corrected only by giving insulin or something similar.

"The second abnormality is that even if you are not eating, your liver overproduces glucose at a rate which exceeds the needs of the body, so that the blood sugar rises. That is due to glucagon. And that should be corrected by suppressing glucagon."

What does all this mean to the diabetic patient?

"Recognition of this premise opens a whole new avenue for investigation of the control of diabetes by an entirely new approach." Dr. Unger said. "I think it really does raise tremendous hope for a new form of therapy--the development of a chemical agent, either a form of somatostatin or some other substance, which will suppress or block glucagon, so that the balance of the effects of insulin and glucagon are closer to normal."

He stresses that only after long years of further study, once glucagon is controlled and a better blood-sugar balance achieved in the diabetic patient, can it be determined whether the physiological problems believed associated with the disease--such as blood vessel, eye and kidney changes--will be helped by improved diabetes therapy.

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