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## NEWS RELEASE

THE UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL AT DALLAS



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NEW ORLEANS -- Dallas scientist Dr. Roger H. Unger of The University of Texas (Southwestern) Medical School at Dallas today (Jan. 28) received the Veterans Administration's highest award for medical research for his pioneering studies of an obscure hormone now believed to be an important factor in the body's bloodsugar regulation process.

Dr. Unger was presented the prestigious Middleton Award "for outstanding achievement in medical research," for his work in shedding important new light on the function of glucagon, a long identified but little understood hormone. As a result of Dr. Unger's Dallas research, glucagon is thought to play a possibly key role, along with insulin, in metabolic function.

Presentation of the annual award, named for Dr. William S.

Middleton, former chief medical director for the VA, was made at a regional research conference at the New Orleans Veterans Hospital.

It was Dr. Unger's fifth citation since 1964 for research into the mysteries of the metabolic malfunction that causes diabetes.

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Previously he has received the Lilly Award of the American Diabetes Association in 1964 and the Tinsley R. Harrison Award of the Southern Section of the American Federation for Clinical Research in 1966. He was the winner in 1967, and co-winner in 1968, of the Texas Diabetes Association Award for work most significant in the field of diabetes.

Dr. Unger is associate professor of internal medicine at The University of Texas (Southwestern) Medical School at Dallas, and director of research and chief of the metabolic section at the Dallas Veterans Administration Hospital.

Glucagon and its relationship to diabetes and carbohydrate (blood sugar) metabolism has been the subject of intensive research in Dr. Unger's laboratories at the Dallas VA Hospital since 1958. Although glucagon was discovered in 1923, at the same time as insulin, its status as a hormone and the scope of its function has been unrecognized until recently.

"Until two years ago," Dr. Unger explained in an interview,

"we thought glucagon was a hormone which was a relatively minor

participant in the metabolic processes compared to insulin.

"We now think it is a very important hormone which with insulin forms a 'hormonal team' that controls the moment-to-moment glucose level in the blood."

An important research milestone leading to this finding came two years ago when Dr. Unger and his associates developed for the first time a technique to measure minute quantities of glucagon in the blood of humans.

Using radioisotopes, the scientists succeeded in measuring microscopic amounts of the substance, which is secreted by one group of cells in the pancreas. Another set of pancreas cells secretes insulin.

This laboratory method, known as radioimmuno-assay, made possible the measurement of as little as forty million millionths of a gram.

Previously, no means existed to make such minute measurements of the elusive substance, Dr. Unger said. He credited Miss Anna M. Eisentraut, a former research associate, with playing a key role in development of the radioisotopic procedure.

With this new method Dr. Unger and his associates tracked glucagon levels in simple blood tests of both normal and diabetic patients. They found that diabetics appear to have, in addition to inadequate amounts of insulin, an excess of glucagon as well.

Dr. Unger said this discovery suggests that insulin lack "is not the whole picture" in diabetes and that glucagon excess may exaggerate the consequences of insulin deficiency.

"Glucagon-producing cells may be overactive in diabetes," he said, "either due to a primary defect or as a result of the insulin lack, depriving the cell of glucose which normally would reduce its secretion of glucagon.

"Whatever the cause of the glucagon excess, the net effect on the blood sugar control would be the same--namely, unfavorable," he said.

Dr. Unger emphasized that there is at present only far-ranging speculation as to its cause, but it is apparent that the nature of the metabolic breakdown in diabetes is more complex than has long been thought, with the coordination of two hormones involved.

The scientist explained that the human brain uses 120 grams of glucose per day--whether food is eaten or not. Previous research by other scientists established that the function of glucagon is to stimulate the liver to replace the expended glucose if a shortage from food sources develops.

When a person normally eats sufficient amounts of food containing glucose, he said, the natural secretion of glucagon is turned off.

But in a diabetic, the glucagon mechanism goes haywire.

"Diabetics continue to secrete glucagon despite the intake of glucose, so the blood sugar goes even higher. Excess glucagon, in our opinion, exaggerates certain of the consequences of insufficient insulin and may be an important factor determining the severity of the metabolic disorder."

Future control of diabetes could involve treatment of both the lack of insulin and the excess of glucagon, Dr. Unger indicated. In future research scientists will try to determine if the glucagon secretion can be controlled clinically, as insufficient insulin is now overcome through medication.

"Conceivably, if we are able to reduce the amount of glucagon secretion to normal, this would favorably influence control of the disease," he said.

Working with Dr. Unger in continuing glucagon research are Dr. Walter A. Muller and Dr. Jose Marco, research fellows.

Dr. Unger has been a member of the Dallas medical school faculty since 1952. He is a graduate of Yale University and received his doctor of medicine degree from the College of Physicians and Surgeons of Columbia University in 1947. A native of New York City, he was an instructor at New York University Medical School before coming to Dallas.

The scientist holds memberships in numerous professional societies, and served as chairman of session at the sixth congress of the International Diabetes Federation in Stockholm in 1967. He was president of the Dallas Diabetes Association, 1961-62.

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