UTNews
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NOTE TO EDITOR: THERE WILL BE A PRESS CONFERENCE TUESDAY, JULY 15 AT 10:00 A.M. IN E6.200 ON THE UTHSCD CAMPUS TO ANNOUNCE THE PROGRAM.

****Diabetes Press Conference

DALLAS--"The parents of a diabetic child have a special kind of hell," said one adult diabetic hospitalized for complications. She remembers her parents' concern. Not only do these parents live with their fears about the quality of life--or even life itself--for their stricken child, but they realize that one or more of their other children, too, could develop the same symptoms at any time.

In order to find the answer to the question of who is at risk for juvenile-onset diabetes mellitus, or Type I diabetes, researchers at The University of Texas Health Science Center at Dallas are launching a major research effort into the genetics of this disease. The first step is the establishment of a diabetes registry of patients with Type I diabetes in the North Texas area.

"The idea is to identify families who have one child with diabetes and to register their brothers and sisters who are not yet affected by this genetic disease," says Dr. Daniel Foster, professor of internal medicine at UTHSCD. Foster and Dr. Roger Unger, also professor of internal medicine at the Dallas health science center, are directing this project along with other major work that they hope will ultimately lead to cure and prevention of the disease. The two physicians are internationally known researchers in diabetes. Both have received the Banting Medal, the world's highest award for research in diabetes. Unger, especially, has received acclaim for his discovery of the role of glucagon along with insulin in diabetes.

Diabetes is the most common metabolic disease of Western civilization. Type I diabetes affects at least one or two out of every 100 Americans. This type begins early in life and can cause diabetic coma and death. Fortunately, the discovery of insulin in the 1920s has made death from diabetic coma a rare event. However, diabetes is associated with a devastating array of long-term complications involving the eyes and kidneys as well as nerves and blood vessels supplying the heart and brain. Insulin therapy, which in most cases prevents the diabetic patient from going into coma, does not prevent these complications. Consequently, diabetes is a leading cause in this country of adult blindness, kidney failure and amputations to limbs because of gangrene. Type I diabetes is also a major cause of heart attacks and strokes.

The first phase of the research program is to enlist the help of 4,000 North Texans with diabetes and their immediate relatives who do not have the disease. These volunteers will become part of a Type I diabetes registry to help researchers identify brothers and sisters of diabetics who do not have the disease but who are at increased risk of developing it. Dr. James Marks, associate professor of pediatrics at UTHSCD, and director of the diabetes clinic at Children's Medical Center spent a sabbatical year in Pittsburgh, Pa., in 1984 working with a prototype registry in preparation for starting the Dallas project.

The identification of 200 of these brothers and sisters who are willing to participate in long-term studies will allow researchers to look for patterns in the way the disease develops in these volunteers who are currently free of diabetes.

(more)

All participants, including the unaffected brothers and sisters and, whenever possible, parents and children of the volunteer patients with Type I diabetes, will have HLA- tissue typing and tests for the newly discovered T-cell receptor genetic markers.

The development of Type I diabetes requires a genetic susceptibility to the disease, Unger explains. This susceptibility is associated with certain protein markers that can be identified in the laboratory by analyzing blood cells. These markers are designated human leukocyte antigens (HLA) and T-cell receptor antigens.

At this time, four major sites are recognized in the HLA region; they are designated A,B,C,and DR, and each has variations (alleles) designated by numbers. If tests show that an individual has HLA DR3 or DR4, his or her risk of developing diabetes three to four times higher than that of the general population. If the volunteer's test shows that he or she carries both of these markers, then the chance for developing the disease is very high indeed, he said.

However, the genetic susceptibility to diabetes alone doesn't cause the disease, pointed out Foster. It is thought that something else triggers the process. That something else seems to be what researchers refer to as an "environmental event."

The environmental event may well turn out to be a viral infection, said the UTHSCD researchers. The theory is that a virus sets up an inflammatory response which causes an alteration in the surface structure of the insulin-producing beta cells in the pancreas, causing them to be destroyed by the immune defense mechanisms of the body. "The idea is that the virus causes the beta cell to be perceived as an enemy," said Unger. Then "killer" cells from the immune system and circulating proteins in the blood, called islet-cell antibodies, destroy the insulin-producing cells.

Dr. Donald Capra, professor of microbiology and internal medicine, and his research associate, Dr. Marie Hoover, are conducting the genetic aspects of the research effort. They believe that new analytical techniques, which allow identification of differences in genes that seem identical by straight HLA typing, will greatly enhance the predictability rate. Secondly, Capra believes that further splitting of the T-cell genes will give clues as to how the immune system is activated.

The researchers involved believe the establishment of the diabetes registry to be of utmost importance. "However, without volunteers from the North Texas area it cannot be done. And without the registry, there will be no way to take a further look into the genetics of diabetes," said Unger.

For information about the program, contact Marilyn Alford, coordinator, at Metro 263-2088 or (214) 879-6121.

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