cc: McMurtry
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http://www.research.ucla.edu/oipa

August 23, 2002

Dr. Nancy Saxman
Business Manager
Biocontrol Technology, Inc.
625 Kolter Drive
Indiana, PA 15701

Subject: UCLA Case No. 2002-318
"The Hepatic Recycling Glucose Tolerance Test"
UCLA Case No. 1997-561
"Pre-diagnostic and Therapeutics for Diabetes"

Dear Dr. Saxman:

Researchers at UCLA have developed the subject technologies for the detection and treatment for diabetes. Based on our knowledge of your company's line of business and research, we believe that this UCLA technology may be of interest to you.

For your consideration, I have enclosed non-confidential descriptions of the technologies that are also located at the following links:

1. http://www.research.ucla.edu/tech/ucla02-318.htm
2. http://www.research.ucla.edu/tech/ucla97-561.htm

If you would like further information about this UCLA licensing opportunity, or are interested in hearing about the latest research in related areas at UCLA, please email me directly at, Ewaldron@resadmin.ucla.edu, or contact our office at (310) 794-0558.

In addition, I would like to note that our *New Technologies and Research Collaboration Opportunities* website has been updated recently. The site includes a searchable faculty research interests database, news from our labs, and upcoming UCLA research collaboration opportunities. The URL is listed below.

I look forward to hearing from you.

Sincerely,

Emily Waldron
Assistant Director
Office of Intellectual Property

Enclosures: Non-Confidential Descriptions

THE HEPATIC RECYCLING GLUCOSE TOLERANCE TEST (HR-GTT)

UCLA Technology Available For Licensing

Diabetes is a condition of high blood glucose resulting from a number of enzymatic/metabolic disorders involving the muscle, fat, islet cells, and the liver. Current modes of diagnosing these metabolic disorders by the standard glucose tolerance test cannot accurately assess the nature of the dyregulation in hepatic glucose production dynamically. Fasting glucose level may be elevated in Type II diabetes secondary to hepatic insulin resistance, defined as the failure to restrain the flow of glucose carbon (termed flux) during an overnight fast. Elevated post-prandial glucose excursions may also result, in part, from resistance to the action of insulin in the liver to restrain excessive glucose flux. UCLA researchers have developed a hepatic recycling glucose tolerance test (HR-GTT) that assesses the relative rates of glucose flux through glucose metabolic pathways in the liver.

The hepatic recycling glucose tolerance test (HR-GTT) developed by UCLA investigators estimates a hepatic recycling constant (KHR) with the use of specific stable isotope tracers. The hepatic recycling constant (KHR) is a measure of the relative rate of glucose recirculation through hepatic glucokinase, glucose-6-phosphatase and the pentose phosphate pathway. Since glucose-6-phophate is extensively exchanged with glycogen, glucose and intermediates of the hepatic pentose cycle, the hepatic recycling constant KHR reflects the substance fluxes through these pathways, and can be used to assess how those pathways affect liver insulin sensitivity. The disappearance of glucose tracer is a measured of the combined effect of hepatic and peripheral glucose utilization. The time course of the disappearance of the labeled glucose metabolites seen during the HR-GTT also yields important information regarding this combined hepatic and peripheral effect.

The insight that the KHR measurement gives into glucose metabolism reflects its utility as an in vivo screen for the hepatic action of diabetes mellitus drugs in discovery research. Other research applications include correlating hepatic insulin action with the putative candidate genes thought to be associated with Type II DM. In the clinic, the tool may be used to diagnose a subtype of Type II diabetes mellitus by evaluating whether the primary site of dysregulation involves the liver.

Reference: UCLA Case No. 2002-318

For information on licensing this invention, please contact the office below.

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NCD URL: http://www.research.ucla.edu/tech/ucla02-318.htm

Lead Inventor: Irwin J. Kurland

Search! Research Interests

UCLA Technologies Available for Licensing
http://www.research.ucla.edu/tech

keywords: diagnostics uclancd ucla technologies intellectual property patents technology transfer invention business card

PRE-DIAGNOSTIC AND THERAPEUTICS FOR DIABETES

UCLA Technology Available For Licensing

The prevalence of Insulin-Dependent Diabetes Mellitus (IDDM) in the U.S is 300,000 to 500,000 individuals of all ages with 30,000 new cases reported each year. IDDM is an autoimmune disease caused by the destruction of pancreatic beta-cells by the body's own T-lymphocyte. Current measures to diagnose IDDM require that patients manifest clinical symptoms, which become evident only after a vast majority of beta-cells had been destroyed. Treatment then involves the replacement of the body's insulin with an exogenuous source, an approach that may lead to complications such as hypoglycemia, local allergic reactions, insulin resistance, and generalized insulin allergy. Importantly, insulin therapy involves life-style adjustment in which patients need to strictly adhere to therapy regimens. An alternative to insulin therapy is the use of immunosuppressants to control the diverse autoreactive T cell population. This strategy of treatment lacks specificity and thus can also interfere with the normal functions of the immune system.

Researchers at UCLA have identified a strategy to diagnose IDDM prior to onset of symptoms by determining the ratio of T helper 1 cells to T helper 2 cells specific to a pancreatic beta-cell associated antigen. This method is based on the principle that the destruction of beta-cells results from the presence of T helper 1 cells. The concept of differential response between T helper 1 and T helper 2 to the same beta-cell antigen can be extended to disease treatment. Therapy therefore involves the administration of a tissue-associated antigen such as glutamic acid decarboxylase (GAD) to shift the pathogenic Th1 response toward a protective Th2 response. This strategy can also be applied to protect beta cells after transplantation.

Using NOD mice, a murine model of human IDDM, the researchers observed that treatment with GAD inhibits lymphocytic infiltration of the islets with respect to the controls. In addition, 80% of GAD-treated mice showed no signs of hyperglycemia at 40 days of age, compared to 80% of control mice that developed IDDM by 35 weeks of age.

Reference: UCLA Case No. 1997-561 **US Patent Number:** 6,207,159 | 6,022,697

For information on licensing this invention, please contact the office below.

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NCD URL: http://www.research.ucla.edu/tech/ucla97-561.htm

Lead Inventor: Dan Kaufman

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