Cambridge Healthtech Institute’s Inaugural

Targeting Diabetes with Novel Therapeutics

New Drug Targets Expand Treatment Options

October 22-23, 2008 • Boston, MA

SESSIONS

Promising Drug Targets for Diabetes
Novel Approaches for New Therapies
A Closer Look at Incretins

PANEL DISCUSSION

Topic: The Future of Incretins as a Target for Diabetes

Participants:
- Richard E. Pratley, M.D., Professor of Medicine and Director, Diabetes and Metabolism Translational Medicine Unit, University of Vermont College of Medicine
- Rachele Berria, M.D., M.S.C.I., U.S. Medical Director, Diabetes Franchise, Roche Laboratories
- Michael R. Hanley, Ph.D., Chief Scientific Officer, Amylin Pharmaceuticals, Inc.

DISTINGUISHED FACULTY

Rachele Berria, M.D., Roche Laboratories
Elaine Chiquette, Amylin Pharmaceuticals Inc.
Max Dang, Ph.D., Metabasis Therapeutics, Inc.
Joseph Grimsby, Ph.D., F. Hoffmann-La Roche Inc.
Jesper Gramada, Ph.D., Novartis Institutes for BioMedical Research, Inc.
Michael R. Hanley, Ph.D., Amylin Pharmaceuticals, Inc.
Richard Ho, M.D., Ph.D., Rosa & Co. LLC.
Reid Huber, Ph.D., Metabolic Endocrine Drug Development, Incyte Corporation
Rob Jones, Ph.D., Arena Pharmaceuticals
Julia Lamenzo Ph.D., Harvard University
Daniel Lin, Ph.D., Amgen Inc.
Peter J. Oates, Ph.D., Pfizer Global Research and Development
Norbert Perrimon, Ph.D., Harvard Medical School
Richard E. Pratley, M.D., University of Vermont College of Medicine
Jean Whaley, Ph.D., Bristol-Myers Squibb

Part of

Discovery on TARGET

October 20-23, 2008
World Trade Center • Boston, MA
Exhibits: October 21-23, 2008

Organized By:
Cambridge Healthtech Institute, 250 First Avenue, Suite 300 • Needham, Massachusetts 02494
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Targeting Diabetes with Novel Therapeutics
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WEDNESDAY, OCTOBER 22

7:00am – 6:00pm Registration Open

PROMISING DRUG TARGETS FOR DIABETES

2:00pm Chairperson’s Remarks
Jesper Gromada, Ph.D., Head of Discovery Biology, Cardiovascular and Metabolism Disease Area, Novartis Institutes for BioMedical Research, Inc.

2:10 FGF-21 for the Treatment of Type 2 Diabetes
Jesper Gromada, Ph.D., Head of Discovery Biology, Cardiovascular and Metabolism Disease Area, Novartis Institutes for BioMedical Research, Inc.

FGF-21 is a recently discovered metabolic regulator. FGF21 activity depends on beta-klotho, a single-pass transmembrane protein. Beta-klotho physically interacts with FGF receptors 1c and 4. FGF-21 stimulates glucose uptake in adipocytes, reduces glucagon secretion and lowers blood glucose and triglyceride levels when administered to diabetic rodents and monkeys. FGF-21 also preserves pancreatic insulin content and beta cell mass when administered to diabetic mice. Finally, FGF-21 protects animals from diet-induced obesity when overexpressed in transgenic mice. These data suggest that FGF-21 might offer a promising new therapeutic approach to treat type 2 diabetes.

2:40 11beta-HSD1 Inhibition as an Entrée to Cardio-Metabolic Benefit in Type 2 Diabetes
Reid Huber, Ph.D., Senior Director, Metabolic Endocrine Drug Development, Incyte Corporation

Excess cortisol activity has been proposed to underlie several cardiovascular risk factors observed in type 2 diabetes, namely insulin resistance, hyperlipidemia, hypertension, and abdominal obesity. The enzyme 11beta-HSD1 catalyzes the tissue-specific, intracellular conversion of biologically inactive cortisone to biologically active cortisol. Emerging preclinical and clinical data indicate that 11beta-HSD1 inhibitors may provide a means to achieve both microvascular and macrovascular benefit in patients with type 2 diabetes.

3:10 Antidiabetic Effects of Glucokinase Activators: From Benchside to Bedside
Joseph Grimsby, Ph.D., Senior Research Leader, Metabolic Diseases, F. Hoffmann-La Roche Inc.

Glucokinase (GK) plays a key role in glucose homeostasis by controlling the rate of glucose metabolism in many tissues. In pancreatic b-cells GK acts as a molecular sensor for glucose stimulated insulin release (GSIR) and in hepatocytes it catalyzes the first step of glucose metabolism. The relationship between changes in GK activity and fasting plasma glucose in humans with GK diseases has been well established for both loss and gain of function mutations. Such strong biological rationale for targeting GK as a potential antidiabetic therapy led to the discovery of small molecule allosteric GK activators (GKAs). GKAs bind to an allosteric site 20 Å away from the bound glucose site and act as non-essential mixed-type enzyme activators. GKAs that increase Vmax and decrease S0.5 augment glucose metabolism and lower the threshold of GSIR in normal and T2D human islets. Animals treated acutely and chronically with GKAs show improvements in basal and post-prandial glucose levels. Evidence suggests that the glucose lowering effects in rodents are mediated by dual effects on increasing plasma insulin levels and suppression of hepatic glucose levels as assessed by a pancreatic clamp. Early data in humans will be presented. GKAs offer a promising new therapeutic approach to treat T2D.

3:40 Networking Refreshment Break in the Exhibit Hall

4:20 Discovery of a Second Generation FBPase Inhibitor, MB07803, with Reduced Metabolism, Improved Oral Bioavailability, and Clinical POC in Type 2 Diabetic Patients
Max Dang, Ph.D., Director, Medicinal Chemistry, Metabasis Therapeutics, Inc.

MB07803 is the first FBPase inhibitor achieving clinical POC in type 2 diabetic patients. Inhibition of FBPase led to direct control of the overactive gluconeogenesis in type 2 diabetic patients, therefore representing a promising new treatment.

4:50 Technology Watch (Sponsorship Available)

5:20 SGLT2 Inhibition: A Novel Approach to the Treatment of Type 2 Diabetes
Jean Whaley, Ph.D., Director, Diabetes Drug Discovery, Bristol-Myers Squibb Co.

THURSDAY, OCTOBER 23

7:30am Morning Coffee

8:35 Chairperson’s Remarks
Peter J. Oates, Ph.D., Research Fellow, CVMED Translational Pharmacology, Diabetes and Diabetic Complications, Pfizer Global Research and Development

8:40 A Systems Biology Approach to Dissecting the Topology of the Insulin Signaling Network
Norbert Perrimon, Ph.D., Howard Hughes Medical Institute, Department of Genetics, Harvard Medical School

Our studies illustrate how we are combining RNA-interference, Mass-Spectrometry, time course gene expression and phosphorylation profiling, and computational analyses to generate a comprehensive network of the Drosophila insulin signaling network.

9:10 Chemical Screening for Modulators of Beta Cell Differentiation and Proliferation
Julia Lamenzo Ph.D., Research Associate, Stem Cell and Regenerative Biology, Harvard University

There are two potential sources of insulin producing beta cells which could potentially be used in transplant therapies similar to the Edmonton protocol: beta cells derived through the differentiation of embryonic stem cells, and beta cells derived through expansion of an existing beta cell population. We have discovered small molecule modulators of beta cell differentiation and proliferation in high-throughput screens with embryonic stem cells and primary beta cells.

9:40 Modeling Complex Mechanisms of Action in Diabetes Therapy
Richard Ho, M.D., Ph.D., Principal, Rosa & Co. LLC

Many therapies in development for diabetes, such as glucokinase modulators, exhibit multiple intrinsic properties of action, complex feedback loops in response to therapy, and strong drug interactions within the disease networks. In this talk, we will present an example of how a systems pharmacology species-specific metabolic constraints also make extrapolation from animal studies misleading and risky, thus it can be extremely difficult for researchers to understand the dynamic response to such therapies sufficiently well for successful drug development. Quantitative physiological modeling and simulation can be a much cheaper and faster method to explore the key dynamics involved in the mechanism of action for these therapies. Such dynamics play a crucial role in determining dose ranges, responder patient populations, and selection of backup compounds. We find that there are more than enough public data available, especially in metabolism, to develop quantitative models capable of quickly, inexpensively, and reliably yielding valuable insights for diabetes drug development projects.

10:10 Networking Coffee Break in the Exhibit Hall

10:55 Diabetic Complications and the Polyol Pathway: Lessons Learned and a New Paradigm
Peter J. Oates, Ph.D., Research Fellow, CVMED Translational Pharmacology, Diabetes and Diabetic Complications, Pfizer Global Research and Development

Driven by a simple paradigm, the “Osmotic Hypothesis,” and armed with positive pre-clinical results on prototype aldose reductase (AR) inhibitors (ARIs), researchers worldwide have targeted diabetic complications with ARIs for four decades. However, the outcomes of most double-blind placebo-controlled AR clinical trials have been disappointing. Ironically, the scientific evidence including new human genetic data that AR plays a key pathogenic role in diabetic complications has continued to mount. Evidence is now strong that the Osmotic Hypothesis and the widely employed sorbitol biomarker were misleading and caused underestimation of doses needed for clinical efficacy and overestimation of drug safety margins. Moreover, current recognition of the pathogenic importance of oxidative stress and its strong link to metabolic flux through AR has led to the “Metabolic Flux Hypothesis,” which emphasizes the importance of cofactor turnover rather than polyol accumulation. Hopefully, these new insights will lead to novel ARIs that will effectively and safely slow the progression of diabetic complications.
11:25  GPR40 Agonists for the Treatment of Type 2 Diabetes
Daniel Lin, Ph.D., Principal Scientist, Metabolic Disorders, Amgen Inc.
GPR40 is a G-protein coupled receptor that has been shown to be expressed in pancreatic β-cells and enteroeccrine cells. Small molecule agonists of GPR40 will be described that improve insulin secretion and post-prandial glucose control and may be useful in the treatment of type 2 diabetes.

11:55  GPR119 Agonists Mediate Glycemic Control via a Glucose Dependent Insulinotropic and Incretinotropic Action
Rob Jones Ph.D., Director, Medicinal Chemistry, Arena Pharmaceuticals
Pancreatic β-cell dysfunction is a hallmark event in the pathogenesis of type 2 diabetes. Injectable peptide agonists of the GLP-1 receptor have shown significant promise as anti-diabetic agents by virtue of their ability to amplify glucose-dependent insulin release and preserve pancreatic β-cell mass. These effects are mediated via stimulation of cyclic AMP through cAMP-dependent protein kinases. Here we report that the GLP-1 agonist GPR119 is largely restricted to insulin-producing β-cells of pancreatic islets together with L-cells of the GI tract, stimulation of GPR119 under hyperglycemic conditions leads to a dual nutrient dependent incretinotropic and incretinotropic effect to maintain glucose homeostasis. Unlike receptors for GLP-1 and other peptides that mediate enhanced glucose-dependent insulin release, GPR119 has proven amenable to the development of potent, orally active, small molecule agonists. Specific orally active GPR119 agonists may offer significant promise as novel anti-diabetics acting in a glucose-dependent fashion.

12:25pm  Lunch on Your Own
12:55  Session Break
A CLOSER LOOK AT INCRETI N S

1:55  Chairperson’s Remarks
Richard E. Pratley, M.D., Professor of Medicine and Director, Diabetes and Metabolism Translational Medicine Unit, University of Vermont College of Medicine

2:00  Incretins
Kevin Tomaselli, Ph.D., Executive Director Discovery Biology, Amylin Pharmaceuticals, Inc.Incretins are glucose-dependent insulinotropes that have emerged as potentially the most powerful class of Type II diabetes therapeutics. Three structural families of incretins will be compared and contrasted: GLP-1, exendin-4, and GIP. Each class has a distinctive pharmacological profile, with varying degrees of additional health benefits, such as weight loss. The future promise of a portfolio of incretin drugs, used as monotherapies or in combination, is compelling.

2:30  Incretin Mimetics: Clinical Perspective and Position in the T2DM Treatment Algorithm
Richard E. Pratley, M.D., Professor of Medicine and Director, Diabetes and Metabolism Translational Medicine Unit, University of Vermont College of Medicine
This talk will review the clinical data for incretin mimetics on the market or in late-stage development and the evolving role for these agents in the management of type 2 diabetes.

3:00  Networking Ice Cream Refreshment Break in the Exhibit Hall (Last Chance for Viewing)

3:40  GLP-1: Physiology and Therapeutic Potential
Rachele Berria, M.D., M.S.C.I., U.S. Medical Director, Diabetes franchise, Roche Laboratories
The prevalence of type 2 diabetes, as well as obesity, is increasing worldwide. The physiology of GLP-1 and its potential as part of a pharmacological intervention have drawn considerable interest over the most recent years. The known glucoregulatory actions of GLP-1 include enhancement of glucose-dependent insulin secretion, suppression of unsuitable elevated glucagon secretion, slowing of gastric emptying and appetite reduction. Furthermore, weight loss has been a consistent finding both in animal and human studies and makes incretin mimetics a valuable option in the treatment of type 2 diabetes and fighting of obesity.

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The Future of Incretins as a Target for Diabetes
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5:10pm  Close of Targeting Diabetes with Novel Therapeutics Conference

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5:10pm  Close of Targeting Diabetes with Novel Therapeutics Conference

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Reasons You Should Present Your Research Poster at Discovery on Target:
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• Receive $50 off your registration fee
• Your poster abstract will be published on our conference CD
• Your research will be seen by leaders from top pharmaceutical, biotech, academic and government institutes
To secure a poster board and inclusion in the conference CD, your abstract must be submitted, accepted and registration paid in full by September 22, 2008.

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Hotel and Travel Information

CONFERENCE VENUE
World Trade Center
200 Seaport Boulevard
Boston, MA 02210
T: 617-385-5049

HOTEL INFORMATION
Seaport Hotel (located directly across the street from venue)
One Seaport Lane
Boston, MA 02210
T: 617-385-4000
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Discounted Room Rate: $235 s/d
Discounted Room Rate Cut-off Date: September 22, 2008

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For reservations via phone, please call the hotel directly to make your room reservation. Identify yourself as a Cambridge Healthtech Institute conference attendee to receive the reduced room rate. Reservations made after the cut-off date or after the group room block has been filled (whichever comes first) will be accepted on a space-and-rate-availability basis. Rooms are limited, so please book early.

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Car Rental Information:
Special discount rentals have been established with AVIS for this conference. Call AVIS direct at 800-331-1600 and reference the Avis Worldwide Discount (AWD) Number J668190.
Targeting Diabetes with Novel Therapeutics
New Drug Targets Expand Treatment Options

October 22-23

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Delivery Preferences: How would you prefer to receive notices from CHI:
COMMERCIAL: ● Yes  ● No
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Single Course:
RNAi for Therapeutics: $695
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Afternoon Short Courses 2pm - 5pm
SC4: Tackling RNAi Delivery
SC5: Ion Channel Assays for Safety Screening
SC6: Screening for Potential Drug Targets - Design Strategies for Novel-Generation Kinase Inhibitors

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I cannot attend but would like to purchase the conference CD for $750 (plus shipping). Massachusetts deliveries will include 5% sales tax.

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Invoice me, but reserve my space with credit card information listed below.
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The latest industry news, commentary and highlights from Bio•IT World

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□ Drug Discovery Chemistry (DDCH)
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Program and speakers are subject to change.

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