AMERICAN FEDERATION FOR MEDICAL RESEARCH

TRANSLATIONAL SYMPOSIA PRESENTED AT EXPERIMENTAL BIOLOGY 2006

A COMPREHENSIVE STEM CELL RESEARCH UPDATE

Sunday April 2, 2006 – 8:00 to 10:00 am

Room 135 – Moscone Convention Center

Organizer Name: Meredith Hawkins, MD
Albert Einstein College of Medicine, Bronx, New York

Richard Burt, MD
Professor of Medicine
Director, Division of Immunotherapy
Northwestern University

Sanjeev Gupta, MD
Professor of Medicine
Albert Einstein College of Medicine

David A. Prentice, PhD
Professor of Life Sciences and Adjunct Professor of Medical Genetics
Indiana State University

Nigel M. de S. Cameron, PhD
Strategic Futures Group, LLC

Abstract: The pluripotent nature of "stem cells" offers tremendous promise for cell therapy approaches to many debilitating diseases. Notwithstanding, this area of science is in its infancy. Embryonic, fetal, and adult stem cells all carry unique possibilities alongside sizeable risks and limitations. Furthermore, the potential use of human fetal and embryonic cells opens vast bioethical minefields, which must be treaded cautiously. This symposium will combine overviews of the scientific and bioethical issues with up-to-the-minute scientific presentations demonstrating significant advances in using both fetal/embryonic and adult stem cells to treat important clinical conditions. Drs. Prentice and Cameron, in reviewing the relevant science and bioethics, will draw upon considerable experience in presenting these challenging topics to high-profile audiences, including Congress and many parliaments, as well as diverse scientific audiences. Drs. Gupta and Burt will present specific applications of fetal/embryonic and adult stem cell research in the treatment of such debilitating diseases as liver failure, diabetes mellitus, multiple sclerosis, and systemic lupus erythematosus. They are both highly productive in this area and have contributed substantially to the literature. Given the active nature of stem cell research, we anticipate that by the time of the symposium there will be many scientific advances, even relative to what is state-of-the-art at the time of submission!

HIV LIPODYSTROPHY: LESSONS FROM A NOVEL METABOLIC SYNDROME

Sunday, April 2, 2006 – 10:30 am to 12:30 pm

Room 250/262 – Moscone Convention Center

Organizer Name: Steven Grinspoon, MD
Massachusetts General Hospital, Boston, Massachusetts

Ashok Balusubramanyam, MD
Associate Professor of Medicine and Cell Biology
Baylor College of Medicine

Steven Grinspoon, MD
Director, Massachusetts General Hospital Program in Nutritional Metabolism
Massachusetts General Hospital
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DOI 10.2310/6650.2005.X0022

Abstract: Recent evidence demonstrates a novel metabolic syndrome among HIV-infected patients, including altered lipid metabolism, substrate flux, fat distribution, and insulin resistance. Increased visceral adiposity and subcutaneous fat atrophy are most prominent. Significant progress has been made in recent years to understand this syndrome. Increased lipolysis and flux of fatty acids to the liver and muscle contribute to insulin resistance. Protease inhibitors have been shown to affect PPAR signaling, adipocyte differentiation, and apoptosis. At the same time, NRTIs, through inhibition of mitochondrial DNA polymerase gamma, impair fatty acid oxidation, resulting in lipid accumulation in both the liver and muscle and repartitioning away from the subcutaneous compartment. Furthermore, reduction in critical cytokines, including adiponectin, impairs fatty acid oxidation.

Recent data suggest significant clinical consequences of insulin resistance in HIV-infected patients, including increased cardiovascular disease. Recent data also suggest potentially beneficial effects of insulin sensitizing agents, such as the thiazolidinediones, to increase adiponectin and subcutaneous adipogenesis while inhibiting lipolysis and reducing hepatic and intramuscular fat accumulation. The HIV lipodystrophy syndrome is a novel metabolic syndrome in which to understand the mechanisms of insulin resistance and fat redistribution. The proposed symposium will cover critical pathophysiologic mechanisms of altered lipid metabolism and nutrient trafficking, highlighting the most recent clinical and molecular data on the effects of protease inhibitors and nucleoside reverse transcriptase inhibitors on these processes.

PATHOLOGICAL CALCIFICATION: CRYSTALLIZATION, INFECTION, OR CELL TRANSDIFFERENTIATION

Sunday, April 2, 2006 – 3:15 to 5:15 pm

Room 274/276 – Moscone Convention Center

Organizer Name: Virginia M. Miller, PhD
Mayo Clinic College of Medicine, Rochester, Minnesota

Karim Benzerara, PhD
Stanford University

Neva Ciftcioglu, PhD
National Aeronautics and Space Administration
Johnson Space Center

Linda Demer, MD, PhD
School of Medicine
University of California, Los Angeles

John C. Lieske, MD
Mayo Clinic College of Medicine

Neal Chen, MD
Indiana University School of Medicine

Discussant: Howard H.T. Hsu, PhD
Department of Pathology
University of Kansas

Abstract: It is becoming increasingly clear that the pathophysiology of calcification within diseased human tissues is complex. Inflammation appears to be associated with many calcific processes, including advanced atherosclerosis, nephrolithiasis, and calciphylaxis of end-stage renal disease. In all three instances characteristic cellular responses could potentially either mediate or ameliorate the calcific response. Increasingly, microorganisms are being identified as an unexpected cause of disease, with a recent well-known example being the association of *H. pylori* and peptic ulcer. The possibility that microorganisms contribute to pathologic calcification and the associated inflammation is, however, controversial. This symposium will examine evidence for three possible but not mutually exclusive mechanisms of pathologic calcification: (1) processes that drive and influence *inorganic* crystallization; (2) the mechanisms by which cells mediate crystallization, including cell transdifferentiation; and (3) evidence for the presence of calcifying microorganisms within diseased human kidneys and arteries. The program will conclude with a panel discussion led by a moderator who will comment on divergent points from the perspective of a pathologist.