conferenceseries.com

2nd International Conference and Expo on

Lipids: Metabolism, Nutrition & Health

October 03-05, 2016 Orlando, USA



M Raj Lakshman

George Washington University, USA

Brown fat and metabolic syndrome

urrently, there is world-wide epidemic of obesity, metabolic syndrome, and particularly, childhood obesity. The primary cause for this alarming malady is defective energy expenditure linked to metabolic disease. Metabolic syndrome is a complex disease of the industrialized societies that encompasses obesity, type 2 diabetes, hypertension and hyperlipidemia. Poor dietary habits and sedentary life style lead to impaired adipose tissue fuel handling, and ectopic lipid deposition in vital organs such as liver, pancreas, muscle and heart. Obesity and its metabolic consequences represent a significant clinical problem. Thermodynamically speaking, obesity is due to a gross imbalance in energy intake and expenditure. However, lifestyle interventions by reducing energy intake and/or increasing energy expenditure have proved to be ineffective in the prevention and/or treatment of obesity, primarily because of poor long term adherence to such interventions. Mitochondria, the power house of mammalian cells, play a major role in energy expenditure. Significantly, roughly 80% of mitochondrial membrane potential generated by fuel oxidation is responsible for ATP production, whereas the remaining 20% is dissipated as heat energy due to mitochondrial uncoupling reactions. The uncoupling of mitochondrial phosphorylation with the assistance of the uncoupling protein 1 (UCP1), represents, a viable mechanism for the dissipation of fuel energy essentially as heat, and could potentially lead to weight control. Our body has both UCP1-poor white adipose tissue (WAT) and UCP1-rich brown/ beige adipose tissue (BAT). While BAT was considered to be present only in the neonates, the recent rediscovery of BAT depots in adult humans has revived tremendous promise in the manipulation of mitochondrial uncoupling reactions as a means to transform fuel energy into heat, thereby counteracting obesity and metabolic syndrome. A number of tissues and cell types have been found to secrete factors that regulate brown and beige adipose activity through systemic, autocrine and paracrine mechanisms. In this, I will discuss the current status of the relative roles of WAT versus BAT in the maintenance of lipid homeostasis in humans with special emphasis on potential mechanisms by which the relative proportions of WAT and BAT can be controlled by tissue-specific strategies that could lead to a therapeutic role of UCP1 in the treatment of obesity and metabolic syndrome.

Biography

M Raj Lakshman is currently the Director of Research Laboratories and the Chief of Lipid Research at the VA Medical Center, Washington, D.C. He also has joint appointments as a Professor in the Departments of Biochemistry & Molecular Medicine as well as in the Department of Medicine at the George Washington University, Washington, D.C. He directs studies in the areas of Alcoholism, Alcoholic Liver Disease, Oxidative Stress, Coronary Artery Disease, Lipids & Lipoproteins, Metabolic & Genetic Obesity, Hepatotoxins and Gene Regulation & Expression. He joined the National Institute of Health, to work on Alcoholic Hyperlipidemia under the able guidance of Professors Richard Veech and Nobel Laureate, Hans Krebs. In 1979, he received the prestigious VA Research Career Scientist Award working in the field of Alcohol and Alcoholism at the VA Medical Center, Washington, D.C. He was honored the "Washington Heart Ball" Research Award in 1990 in the field of Hyperlipidemia.

Raj.Lakshman@va.gov