Shaodong Guo, J Metabolic Synd 2018, Volume 7
DOI: 10.4172/2167-0943-C1-007

JOINT EVENT

3rd International Conference on

ENDOCRINOLOGY AND METABOLIC SYNDROME

&

12th International Conference on

ABDOMINAL IMAGING AND ENDOSCOPY

June 28-29, 2018 Amsterdam, Netherlands

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Insulin signaling, resistance and metabolic syndrome: Insights from mouse models into disease mechanisms

Insulin resistance serves as the major mechanism for the development of obesity, which is pandemic in population worldwide over the past decades, largely owing to over nutrition. Excess energy stores in the adipose tissue and other organs as lipids, promoting lipotoxicity and metabolic inflammation, activating intracellular protein kinases to impair insulin signaling components and resulting in insulin resistance. Insulin resistance is the key etiologic defect that defines "metabolic syndrome", a group of interrelated disorders, including obesity, hyperglycemia, dyslipidemia and hypertension. Following insulin resistance, many of patients with the metabolic syndrome eventually developed pancreatic β-cell failure, which triggers the onset of type 2 diabetes mellitus (T2DM) and its complications. Our cell- and animal-based studies demonstrate that insulin and its signaling cascades normally control cell growth, metabolism and survival through activation of mitogen-activated protein kinases (MAPKs) and phosphatidylinositide-3-kinase (PI3K), of which activation of PI3K-associated with insulin receptor substrate-1 and -2 (IRS1, 2) and subsequent Akt→Foxo1 phosphorylation cascade have a central role in control of nutrient homeostasis and organ survival. Inactivation of Akt and activation of Foxo1, through suppression IRS1 and IRS2 in a variety of organs following over nutrition, lipotoxicity and inflammation may form a fundamental mechanism for insulin resistance in humans. This seminar discusses the basis of insulin signaling, resistance and how excess nutrients and lipid signaling from obesity promotes inflammation and insulin resistance, promoting organ failure with emphasis on the IRS and the forkhead/winged-helix transcription factor Foxo1.

Biography

Shaodong Guo is an Associate Professor in the Department of Nutrition and Food Science at Texas A&M University College. He received his PhD in Physiology from Peking University, China. Then he completed his Post-doctoral research training in Genetics, Biochemistry, and Medicine in the Chinese Academy of Sciences, the University of Illinois at Chicago, and Harvard University, respectively. He was an Instructor in Medicine at Children's Hospital Boston and Harvard Medical School for two years prior to joining the faculty at Texas A&M Health Science Center. Currently, he serves as Senior Editor for the *Journal of Endocrinology* and *Journal of Molecular Endocrinology*, two major official journals of Endocrine Society of Europe, UK, and Australia, and he is the textbook chapter writer for *Metabolic Syndrome* edited by Rexford Ahima and published by Springer in 2016. His lab research focuses on insulin/glucagon and estrogen signal transduction, insulin resistance, gene transcriptional control of nutrient homeostasis, and cardiac dysfunction in diabetes. He has been working on the gene transcriptional regulation of metabolic homeostasis by insulin receptor substrate proteins (IRS) and Forkhead FoxO transcription factors and has been funded by American Diabetes Association (ADA), American Heart Association, and the National Institute of Health of USA. He is a recipient of ADA junior faculty award, career development award, and Richard R Lee Award. His work has been published in a number of journals including the JBC, Endocrinology, Hypertension, Diabetes, Circulation Research, AJP, MCB, and Nature Medicine, receiving more than 5,000 citations from the Google Scholar.

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