Ming-Sheng Zhou, Endocrinol Metab Synd 2014, 3:2
http://dx.doi.org/10.4172/2161-1017.S1.007

OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference on
Endocrinology
October 20-22, 2014 DoubleTree by Hilton Hotel Chicago-North Shore, USA

# Hypertension and insulin resistance: Partners in crime

**Ming-Sheng Zhou**
Liaoning Medical University, China

Insulin activation of the phosphatidylinositol 3-kinase (PI3K )pathway stimulates glucose uptake in peripheral tissues and NOsynthesis in the endothelium. Up to 50% of hypertensive have Insulin resistance (IR) particularly among individuals with the metabolic syndrome and those with salt-sensitive (SS) hypertension. The mechanisms underlying this association are poorly understood. We investigated these mechanisms in a model ofSS hypertension. Dahl SS rats were fed for 6 weeks a normal (NS) or high salt (HS) diet, HS plus the angiotensin II type 1 receptor (AT1R) blocker candesartan, or HS plus the antioxidant Tempol. Hypertensive HS rats manifested increased aortic AT1R mRNA (210%) and protein (101%) expression , O2- production (104%), and impaired endothelium-dependent relaxation (EDR) to acetylcholine. ARB or Tempol normalized O2- and EDR despite minimal reduction in SBP. Hypertensive HS rats manifested metabolic IR (36% reduction in glucose infusion rate), impaired insulin-mediated EDR and PI3K/eNOS phosphorylation, which were significantly improved by either ARB or Tempol treatment. It has been shown that Ang II and ROS activate the proinflammatory transcription factor NF′ B. In a separate group ofhypertensive HS rats, we found that Pyrrolidinedithiocarbamat(PTDC ) inhibited activation of NFkB , improved IR and prevented the upregulation of the proinflammatory cytokines MCP 1 and TNF without significant reduction in SBP. These studies provide insight into the mechanisms that underlie the association between metabolic and hypertensive cardiovascular diseases and support the notion that O2- overproduction linked to tissue angiotensin II concomitantly interferes with insulin signaling pathways and fosters pathways associated with inflammation .

## Biography

Ming-Sheng Zhou received his Ph D from Kagawa Medical University, Japan, and his post-doc training from University of Minnesota and University of Miami. He was faculty member in University of Miami Miller School of Medicine from 2004-2012. He has been professor and chair in Department of Physiology Liaoning Medical University, China. His research interesting focuses on the cellular and molecular mechanism of vascular injury in hypertensive and metabolic diseases. He published over 50 articles in peer-reviewed journals in cardiovascular and renal medicine. He is member for APS, AHA, ADA and ASH and reviewer for numbers of international journals.

zhoums1963@163.com