Risto Kaaja, J Obes Wt Loss Ther 2012, 2:9
http://dx.doi.org/10.4172/2165-7904.S1.008

OMICS Group Conferences
Accelerating Scientific Discovery

International Conference and Exhibition on
Obesity & Weight Management

December 3-5, 2012 DoubleTree by Hilton Philadelphia, USA

Cross-talk between sympathetic overactivity and insulin resistance

Risto Kaaja
Turku University, Finland

The two major factors related to obesity and metabolic syndrome, sympathetic overactivity and insulin resistance, are closely interrelated. Obesity is accompanied by an increase in sympathetic activity , particularly in the kidney, which could lead to an increase in renin release and contribute to hypertension. Reactive hyperinsulinemia due to overeating, primary tissue insulin resistance, hyperleptinemia and high free fatty acid levels all may promote enhanced sympathic outflow and metabolic syndrome. Although acute administration of insulin in healthy subjects induces vasodilatation and is mediated by the release of nitric oxide (NO), in insulin resistance the NO release is impaired, vasomotor tone increased due to enhanced calcium flux into the vascular smooth muscle cells increasing the sympathetic nervous system activity. Anyhow the exact mechanisms through which insulin resistance and hyperinsulinemia predispose to sympathtic overactivity are not well established. b-adrenergic stimulation has also been shown to inhibit insulin signalling and decrease insulin induced glucose uptake in brown adipocytes. As skletal muscle quantitatively account for the largest portion of whole body insulin resistance it not a surprise that factors affecting muscular blood flow and nutrient delivery to the muscle are those of most importance in inducing insulin resistance. Increased sympathetic drive induces small vessel rarefication or loss of capillary density causes vasoconstriction (via a-receptor) and facilitate the shunting of glucose and insulin to less metabolically active skletal muscle beds. The metabolic properties of antihypertensive agents give further credence to the hemodynamic model of insulin resistance. Vasoconstriction mediated by b-blockers and vasodilatation by a-blockers parallel by their metabolic properties. Thus b-blockers that do vasoconstrict induce insulin resistance, but those with a-blocking properties are not causing insulin resistance.

Biography

Risto Kaaja has completed his Ph.D at the age of 32 years from Helsinki University and postdoctoral studies from Helsinki University. He was an obstetric physician for 23 years in the Women´s Hospital of the Helsinki University Hospital and nowadays professor of Medicine in Turku University. He has published more than 150 papers in reputed journals and the main topics of his publications have been related to manifestations of chronic diseases in pregnancy. Of particular interest has been pre-eclampsia as a state of increased insulin resistance and sympathetic overactivity and of increased risk for cardiovascular diseases later in life.

Risto.Kaaja@hus.fi