International Journal of Endocrinology

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Special Issue on

Insulin Resistance, Obesity, and Metabolic Syndrome: Common Inflammatory Pathways Leading to Type 2 Diabetes

CALL FOR PAPERS

Modern lifestyles play a major role in facilitating the derailment of normal biological functions of cells and metabolic processes. In particular, an unbalanced intake of nutrients, in combination with environmental factors, gives greater vulnerability to several diseases. At a molecular level, peripheral resistance to insulin in fat, muscle, and liver provokes hyperglycaemia and a consequent increment of insulin secretion by the pancreatic cells. Generally, multiple molecular mechanisms of insulin resistance (IR) have been described, and it is possible to attribute IR pathologic phenotype to a single specific cause, i.e., in some extreme and rare forms of IR, in which the altered expression of insulin receptor (INSR) is genetically determined.

Recent data have shown that dysfunction of proteins involved either in insulin signal transduction or in the regulation of INSR expression provokes IR. In addition, a considerable amount of evidence about the pathogenesis of IR has suggested a strong relationship between nutrient excess and inflammatory molecules. A common immunological factor is the activation of the inflammasome and the elevated levels of circulating inflammatory mediators, including cytokines and chemokines, that promote the initiation and progression of metabolic diseases. In particular, obesity is associated with chronic low-grade inflammation in which the accumulated adipose tissue macrophages (ATMs) mediate the action and secretion of adipose-derived hormones (adipokines) and proinflammatory cytokines (IL-1 , IL-18), leading to progression and maintaining of IR.

This special issue aims to study multiple aspects of insulin resistance and its interactions with metabolic inflammation and diabetes. Many processes underlying IR, metabolic inflammation, and diabetes are not entirely clarified. In addition, there is little data on the cross-talk between adipose tissue dysfunction, the systemic pathological state of the endothelium, and heart failure. There is a lack of information regarding how inflammation can be instrumental in some aspects of metabolic syndrome or in causing reduced energy expenditure and cell dysfunction.

We welcome original manuscripts that are highly innovative, in order to add pieces to the mosaic of knowledge on insulin resistance related disorders in an attempt to get closer to the root of the disease pathogenesis. Special attention will also be paid to review articles. Manuscripts should summarize the current state-of-the-art in this field.

Potential topics include but are not limited to the following:

- Molecular mechanisms of insulin resistance
- Beta cells in the pathogenesis of type 2 diabetes
- Adipocyte dysfunction in the pathogenesis of metabolic syndrome
- Mechanisms linking inflammation to dysmetabolic consequences
- Endothelial dysfunction in the progression of diabetic vascular complications
- Endoplasmic reticulum stress in pancreatic beta cell degeneration
- Type 2 diabetes and cardiovascular disease

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/ije/irop/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

Lead Guest Editor
Arcidiacono Biagio, University of Catanzaro "Magna Graecia", Catanzaro, Italy
arcidiacono@unicz.it

Guest Editors
Jun Nakae, International University of Health and Welfare, Narita, Japan
nakaejun5330@iuhw.ac.jp

Gaia Mannino, University of Catanzaro "Magna Graecia", Catanzaro, Italy
gaiamannino@gmail.com

Jan Skupien, Jagiellonian University, Krakow, Poland
jan.skupien@uj.edu.pl

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