

Field:

Bioscience/Life Science

Session Topic:

Body fat : hidden organ, new source of hormonal regulation

Chair:

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Efficient collection and processing of food to deposit fat in adipose tissue would have been advantageous for hunter-gatherer populations, especially child-bearing women allowing them to fatten more quickly during times of food abundance. Fatter individuals carrying 'thrifty' genes would thus better survive times of food scarcity; however, in modern societies with a constant abundance of food, this genotype efficiently prepares individuals for a famine that never comes. The result is widespread chronic obesity and related health problems such as metabolic syndrome. Nearly 1 in 4 adults in the United States is obese. The increasing prevalence of each of these diseases has become a growing concern for the medical community.

Recent research has shown that adipose tissue is not merely an energy storage organ but also an important endocrine organ that secretes many biologically active substances and hormones, such as leptin, free fatty acids (FFAs), tumor necrosis factor- α (TNF- α), and adiponectin, which are collectively termed adipocytokines. Dysregulation of function and the production of pro- and anti-inflammatory adipocytokines seen in visceral fat obesity is associated with metabolic syndrome, suggesting that inflammation may critically contribute to the development of many aspects of metabolic syndrome, resulting in atherosclerosis. Adiponectin is a collagen-like plasma protein secreted by adipocytes that has been suggested to play a causal role in the development of insulin resistance and cardiovascular disease. This protein has been found to be decreased in cases of insulin resistance, diabetes, atherosclerosis, and coronary artery disease.

Meanwhile, recent epidemiological and experimental evidence suggest that metabolic syndrome may be acquired through nutritional effects in early life: intrauterine undernutrition is closely associated with adulthood obesity related to detrimental metabolic sequelae, giving rise to the concept of "developmental origins of health and disease". These observations suggest the epigenetic regulation of metabolic syndrome following intrauterine growth retardation. DNA methylation is a key epigenetic contributor to the maintenance of gene silencing that relies on a dietary supply of methyl groups. DNA methylation may play roles in fetal epigenetic modification, which later affect metabolic syndrome.

In this session, we will discuss the role of adipose tissue as an organ for secreting adipokines and the involvement of epigenetics in the pathogenesis of metabolic syndrome.