

## Action BM0602

2007 - 2011

# Adipose Tissue: A Key Target for Prevention of the Metabolic Syndrome

Participating countries: AT, BE, BG, CH, CY, CZ, DE, ES, FI, FR, HU, IL, IT, NL, NO, PL, RO, SE, SI, SK, SRB, TR, UK

Intention to participate: DK, GR, IE, LV, MK, PT

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[http://www.cost.esf.org/domains\\_actions/bmbs/Actions/Adipose\\_Tissue](http://www.cost.esf.org/domains_actions/bmbs/Actions/Adipose_Tissue)

## Working Group 1

### Analysis of the central regulation of food intake and adipocyte storage function

The main objective of this part of the Action is to identify new molecular targets that may serve to control food intake and to prevent the development of the metabolic syndrome. An additional specific objective is to promote our knowledge on glucose sensing systems and their role in the control of energy homeostasis and body weight. Adipose cell size is critically related to glucose tolerance and insulin sensitivity. Therefore, a major goal is to understand how hypertrophy of adipose cells leads to impaired lipid storage capacity, impaired adipogenesis and systemic insulin resistance.

## Working Group 2

### Elucidation of adipose tissue secretory function

The main objective of this research task is to comprehensively explore the regulation of adipose tissue secretory function, the origin of secretory products within the tissue, the actions of these products at the local and peripheral levels and the pathophysiological relevance for humans. Additional objectives include the identification of novel secretory products and the investigation of the pathophysiological role of the adipose tissue and its secretory products in human or animal models of the metabolic syndrome and type 2 diabetes.

## Working Group 3

### Assessment of the relationship between cytokines, inflammation and vascular dysfunction and skeletal muscle insulin resistance

One main objective is to identify the downstream targets of the crosstalk between adipose tissue-derived proinflammatory cytokines and vascular endothelial and smooth muscle cells. This will be essential to develop new therapeutic strategies for prevention of the secondary complications associated with the metabolic syndrome and type 2 diabetes. Attempts will also be made to uncover potential synergistic interactions between cytokines, hyperglycemia and increased levels of free fatty acids.

## Working Group 4

### Glucolipotoxicity, islet inflammation, beta cell dysfunction and type 2 diabetes

The main objective is to expand the current research on glucose- and free fatty acid-induced damage to beta cells to the role of adipose tissue-derived factors (i.e., proinflammatory cytokines) on the function of beta cells in such adverse environment. Detailed understanding of the molecular mechanisms that lead to beta cell apoptosis and dysfunction is essential for developing new strategies to prevent or delay the onset of type 2 diabetes.

## Objectives:

- To improve our knowledge on the specific role of adipose tissue in the development of the metabolic syndrome
- To analyse the central regulation of food intake
- To investigate adipocyte storage function
- To identify novel adipokines
- To study their impact for inflammation, peripheral insulin resistance and vascular and beta-cell dysfunction
- To define the role of adipose tissue in the manifestation of type 2 diabetes

## Main Achievements:

- Identification of novel adipokines and mediators of peripheral Insulin resistance and dysfunction
- Progress in understanding adipose tissue inflammation, plasticity and crosstalk with other organs
- Advanced knowledge on beta-cell apoptosis