Introduction

The widespread epidemics of obesity and type 2 diabetes mellitus (T_2DM) suggest that both conditions are closely linked. An increasing body of evidence has shifted the view of adipose tissue from a passive energy depot to a dynamic "endocrine organ" that tightly regulates nutritional balance by means of a complex crosstalk of adipocytes with their microenvironment. Dysfunctional adipose tissue, particularly as observed in obesity, is characterized by adipocyte hypertrophy, macrophage infiltration, impaired insulin signaling, and insulin resistance. The result is the release of a host of inflammatory adipokines and excessive amounts of free fatty acids that promote ectopic fat deposition and lipotoxicity in muscle, liver, and pancreatic beta cells, ***(Cusi, 2010)***.

The metabolic syndrome (MS) or syndrome X is the name for a clustering of risk factors for cardiovascular diseases and type II diabetes that are of metabolic origin. This syndrome, first described in the adults, is more and more studied during childhood and adolescence. Metabolic syndrome is now described in youth, particularly in subjects with risk factors as obesity, ***(Gutafson et al., 2009)***.

During the last years, obesity and subsequent metabolic disorders and cardiovascular diseases have tremendously increased. Recent studies have shown that risk factors of cardiovascular diseases appear as soon as in infancy. In many situations, these disorders are programmed in early life during fetal development, ***(Motte et al., 2010)***.

Alterations of intra-uterine environment lead to modified early development and represent short-term adaptations transmitted from one generation to another. This intergeneration effect contributes to the burden of

adult metabolic disorders and cardiovascular diseases, as seen in the last decades. There is considerable evidence for the contribution of epigenetic mechanisms for the lifelong and the intergenerational alteration of gene transcription by variation in the early life environment. One of the major challenges in the following years is to promote public health programs which are aimed at prevention of long-term consequences of fetal programming, ***(Gutafson et al., 2009)***.

The risk of nutritional and metabolic disorders during adulthood was linked to either excess of fetal growth as it is observed during pregnancy with maternal diabetes or poor fetal growth. Substantial evidence now supports the hypothesis that 'accelerated' or too fast infant growth increases the propensity to the major components of the metabolic syndrome (glucose intolerance, obesity, raised blood pressure and dyslipidemia), the clustering of risk factors which predispose to cardiovascular morbidity and mortality. The association between infant growth and these risk factors is strong, consistent, shows a dose-response effect, and is biologically plausible. Moreover, experimental data from prospective randomized controlled trials strongly support a causal link between infant growth and later cardiovascular risk factors. These observations suggest therefore that the primary prevention of cardiovascular disease could begin from as early as the first few months of life, ***(Singhal, 2010)***.