

The Role of Epigenetic Mechanisms in the Pathogenesis of Metabolic Syndrome

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The Metabolic Syndrome (MetS) represents a cluster of cardiometabolic risk factors, including central obesity, insulin resistance, glucose intolerance, dyslipidemia, hypertension, hyperinsulinemia and microalbuminuria. Although the definition of the MetS as well as the single criterion for identifying this condition is still being debated due to the lack of a unifying underlying mechanism, the prevalence of a metabolic syndrome phenotype is rapidly increasing worldwide [1]. Several studies have provided evidence of epigenetic modifications resulting from nutrition during early development, which mediate future and persistent changes in the expression of key metabolic genes contributing to determine an adult metabolic syndrome phenotype [1].

During the past decade, the role of epigenetic mechanisms in the pathogenesis of diseases has been increasingly recognized. Epigenetic modifications, typically including microRNAs (miRNAs, miRs), DNA methylation, histone modification and ubiquitination, refer to phenotypic changes induced by mechanisms unrelated to changes in the underlying DNA sequence.

The term epigenetics refers to heritable changes in gene expression (active versus inactive genes) that do not involve modifications in the underlying DNA sequence, i.e. a change in phenotype without a change in genotype. An epigenetic change is a regular and natural occurrence but can also be influenced by several factors, including age, environment/lifestyle, and diseases. Epigenetic modifications usually manifest as the manner in which cells eventually differentiate to lead to hepatocytes or myocytes. On the other hand, epigenetic changes have also harmful effects that may end up in neoplasms. At least three systems including DNA methylation, histone modification and non-coding RNA (ncRNA)-associated gene silencing are currently considered to initiate and sustain epigenetic changes [2]. New and ongoing research is continuously unraveling the role of epigenetics in a variety of human disorders and fatal diseases.

The main long-term goals in this field are the identification and understanding of the role of epigenetic marks that could be used as early predictors of metabolic risk and the development of drugs or diet-related treatments able to delay these changes, and eventually reverse them [3]. However, weight gain and insulin resistance/diabetes are influenced by factors other than epigenetics [3]. The characterization of all the factors able to modify the epigenetic signatures and the determination of their real importance are hampered by three findings, i.e. the extent of the changes produced by dietary and environmental factors is relatively small and mostly cumulative; the cell types studied vary greatly; many factors with multiple interactions between themselves, such as age, are involved [3].

Therefore, a continued and greater understanding of these mechanisms will eventually help identify subjects at high risk of Mets, and lead to the development of therapeutic interventions, in accordance with current global government strategy [1], utilizing also food constituents as PUFAs. In fact, there is recent evidence that PUFAs may modulate the promotor epigenetic marks in several adipogenic genes and regulate the expression of several miRNAs [4].

Diet play a role in normal biological processes and is involved in the regulation of pathological progression over a lifetime. Evidence has emerged indicating that dietary factor-dependent epigenetic modifications can significantly affect genome stability and the expres-

sion of mRNA and proteins, which are involved in metabolic dysfunction [5]. The intake of dietary components that regulate epigenetic modifications can provide significant health effects and, as an epigenetic diet, may prevent various pathological processes in the development of metabolic syndrome [5].

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