Environmental determinants of obesity and insulin-resistance

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Background: Obesity and insulin-resistance (IR) are growing epidemic problems linked to both genetic and environmental factors. However, genetic predisposing variants only explain a small part of the increased individual risk. Furthermore, the long time required for genetic changes at the population level cannot explain the rapid increment in the rate of obesity and IR observed worldwide, underlying the key role for gene-environment interactions, mainly acting through the epigenome (i.e. DNA methylation, histone acetylation/deacetylation, non-coding mRNA).
Materials and methods: it has been performed a literature review (PubMed) exploring the link between the environmental determinants of obesity, IR and the epigenome.
Results: The maternal nutritional status during pregnancy can influence the metabolic phenotype of the offspring (fetal programming of later obesity), affecting gene expression through epigenetic mechanisms, and this can be passed to the next generations. It has been also shown in monozygotic twin pairs that candidate genes for obesity and type 2 diabetes are differentially expressed in discordant twins mainly due to different DNA methylation patterns, underlying, again, the relevance of environmental and epigenetic factors in the development of these diseases. Recent studies clearly show that similar pathways are also involved in the case of several "obesogenic" toxics (Endocrine Disrupting Chemicals, persistent organic pollutants, air pollution), widely diffused into the environment and food chain and linked with obesity/IR and metabolic homeostasis both in children and adults, also due to prenatal exposure (foetal period of life, preconceptional period).
Conclusions: Further studies are needed, pointing to better assess the influence of modifiable and epigenetic factors on obesity and IR, and to verify the efficacy of primary prevention strategies involving both lifestyle and environmental exposure to toxics.

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