



Obesity an inflammation

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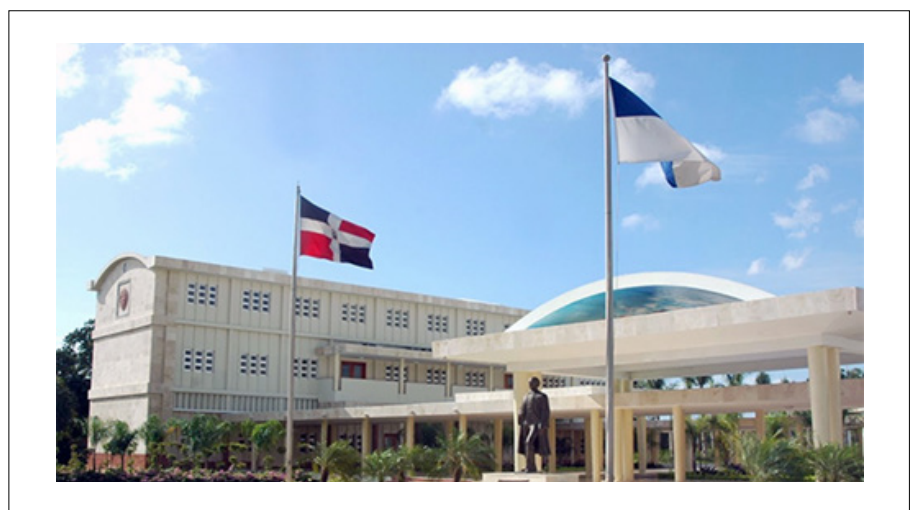
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Abstract

In the last 10-20 years, overweight and obesity have become a true epidemic that is plaguing the world population. The Institute of Health Metrics and Evaluation, estimates that more than 2.2 billion of people in the world suffer from obesity and they forecast data derived from the NHANES, that prevalence among USA adults aged from 20-74 will be 50 % by the 2030. Obesity contributed in 2015 to 4 million deaths globally. Obese people consume 68% more drugs and 20% more in healthcare expenses, due to all the diseases and disorders that lead to overweight and obesity. Obesity and metabolic syndrome are related with the development of cardiovascular diseases. Adipocyte precursors have potent phagocytic capacity and can be transformed into macrophage-like cells in response to appropriate stimuli. Many genes that are critical to adipocytes, including those encoding transcription factors, cytokines, inflammatory molecules, fatty acid transporters, and scavenger receptors are also expressed in macrophages and have an important role in macrophage biology. The excess of saturated fat intake generates a recruitment of macrophages by adipose tissue and a greater release of non-esterified fatty acids (NEFAs), reactive oxygen species, pro-inflammatory cytokines (TNF- α and IL-6), lipoproteins low density (VLDL) rich in cholesterol and oxidized low density lipoproteins (LDL-ox). Also decrease in adiponectin and nitric oxide, favours inflammatory processes at the local and systemic level, macrophage recruitment, endothelial dysfunction and the development of cardiovascular diseases. Metabolic overload cause adipocyte hypertrophy facilitates cell rupture that will evoke an inflammatory reaction. The inability of adipocyte to engulf incoming fat leads to deposition in the liver, with consequences on insulin resistance. Infiltration of macrophages and lymphocytes was also observed in adipose tissue. There is evidence of chronic inflammation occurring in obese adipose tissue, which is a consequence of endoplasmic reticulum stress.

Biography

Maxima Mendez Castillo, Internist Cardiologist and Master in Lipids from The National Lipid Association, Specialist in investigation methodology, actual President of the Lipid and Atherosclerosis Council of the Dominican Society of Cardiology and CEO and Founder of the Cardiovascular Lipid Club Dominican Republic. She is graduated from the Autonomous University of Santo Domingo (UASD), trained in critical post-surgical cardiovascular care and coronary care at the National Institute of Cardiology Ignacio Chavez in Mexico. She is currently assistant physician of the non-invasive cardiology department of the cardiovascular centre Cedimat in the Dominican Republic. She has participated in multiple national and international conferences, as well as several courses and symposiums on cardiovascular medical updating and a diploma in strategic health management.



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