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Role of inflammatory mediators in obesity-induced insulin resistance

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The prevalence of obesity has increased exponentially worldwide (in 2014, 10.8% of men and 14.9% of women were obese). Obesity is related with several health disturb on society because it associates strongly with many diseases, including cardiovascular disease and immunological disorders such as rheumatoid arthritis and multiple sclerosis. The knowledge that obesity-induced inflammation mediates the development of insulin resistance in animal models and humans has been raising strong support. It was shown that immune cells in visceral adipose tissue play a major role in the regulation of obesity-induced inflammation. Furthermore, obesity increases the numbers and activation of proinflammatory immune cells, including M1 macrophages, neutrophils, Th1 CD4 T cells and CD8 T cells, while simultaneously suppressing anti-inflammatory cells such as CD4 regulatory T cells, regulatory B cells and eosinophils. Recently, some studies have been made in the attempt to understand obesity and insulin resistance linked to immunology mediators. We focused on the roles that these relatively new players in the metabolism field play in obesity-induced insulin resistance and the regulation of obesity.

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