Abstract

High-fat diet promotes the development of diet-induced obesity, which leads to further complications such as insulin resistance and type II diabetes. The underlying cause for development of obesity associated pathologies is disruption of adipose tissue homeostasis. Excessive lipid accumulation and rapid white adipose tissue expansion stimulate infiltration, proliferation, and activation of immune cells involved in inflammation propagation. Immune cells within white adipose tissue have the ability to modulate adipocyte function as well as whole-body metabolism. These interactions and function modulations are the core topics of immunometabolism, a rapidly developing field of research focused on interpreting how immune system modulates metabolism on cellular as well as systemic level.

In obesity, pro-inflammatory immune cells, for example M1 macrophages and neutrophils, outnumber homeostasis-promoting anti-inflammatory immune cells in white adipose tissue and alter the tissue environment. As a result, pro-inflammatory cytokines prevent adipocytes from adequately responding to extracellular stimuli. The resulting interactions between immune cells and adipocytes maintain inflammation and promote ectopic lipid accumulation.

Experimental studies suggest that white adipose tissue inflammation can be resolved by dietary omega-3 polyunsaturated fatty acid supplementation. This counteracts the effects of omega-6 fatty acids most commonly found in high-fat Western diets and promotes tissue homeostasis.