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COMMENTARY

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Cancer Risk in Obesity Individuals and Polarization of Adipose Tissue Macrophages

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Description

One of the most significant worldwide health issues, obesity is increasing annually and co-occurs with the emergence of cancer. In this situation, adipose tissue macrophages (ATMs) play a key role in bridging obesity-related inflammation and tumour development. The roles of ATMs in the development of cancer linked to obesity are yet unknown. We discuss the history, phenotypes, and purposes of ATMs in this review. The potential mechanisms for reprogramming ATMs in the microenvironment linked to obesity are then briefly discussed. These include changes in the gut microbiota and its metabolites, excessive cytokines and other signalling mediators, the transfer of extracellular vesicle cargo, and excess cytokines and other signalling mediators. New treatment strategies for obesity-related malignancies will be developed as a result of a better knowledge of the characteristics and operations of ATMs in situations of obesity.

Many adipose tissue depots, including subcutaneous and visceral white adipose tissue, brown adipose tissue, inter- and intramuscular adipose tissue, marrow adipose tissue, and dermal adipose tissue, are found throughout the body. Adipocytes and a variety of non-adipocyte cells, such as pericytes, endothelial cells, monocytes, macrophages, and adipose-derived stromal/stem cells, are assumed to make up adipose tissue. The macrophage hybrid known as adipose tissue macrophages (ATMs) is made up of myeloid monocytes and tissue-resident macrophages. Crown-Like Structures (CLSs), in which macrophages encircle and phagocytose a dead or dying adipocyte, are a typical structure of macrophages in adipose tissues.

It is noteworthy that various cancer types and intratumor areas might have diverse macrophage phenotypes. The variety of macrophages is first partially explained by the reprogramming of macrophages from one phenotype into another. ATMs alter their transcriptional programmes to 'polarise' from a homeostatic state into an inflammatory one or vice versa in response to various environmental signals. Thus, the changing of ATM phenotypes is a result of the interaction between macrophages and other cells, including fibroblasts, endothelial cells, and adipocytes. For instance, exosomes from stromal/stem cells coming from adipose tissue encourage macrophage polarisation to an alternatively polarised phenotype, which aids in angiogenesis.

Polarisation of ATMs

Many polarisation patterns and the subsequent formation of several functional phenotypes in response to varied environmental signals demonstrate the high adaptability of macrophages. The conventionally polarised (M1) macrophage and the alternatively polarised (M2) macrophage are two unique polarisation types for macrophages. In response to bacterial moieties like lipopolysaccharide or Inter Freon-Gamma (IFN-), macrophages are activated towards the M1 phenotype. On the other hand, IL-4 activation causes macrophages to polarise towards the M2 subtype. The release of proinflammatory cytokines, higher cytotoxic action against bacteria and viruses, and promotion of antitumor immunity are the key characteristics of M1 macrophages.

The flipping nature of ATMs, which secretes both proinflammatory and anti-inflammatory cytokines and is also present in malignancies such malignant mesothelioma, is crucial because it may be triggered. These macrophages could be self-reprogramming or going through a polarisation shift, which would cause the alternative phenotype to inhibit each other's immune systems. Macrophages in overlapping or intermediate states have been observed in vivo under pathological circumstances with a variety of activating cues that change over time.

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For instance, the overlapping profile of M1- and M2-associated genes is enhanced in CD11c+ ATMs generated from fat mice.

Macrophages are well recognised for their plasticity and variety. In the development of malignancies linked to obesity, ATMs are crucial. Definitions of the surface phenotype, activating signals, and molecular pathways connected to various types of ATM activation have advanced. The effectiveness of therapies that target ATMs according to various activation pathways has not been adequately studied, despite the fact that several studies have shown that ATMs significantly alter their roles in malignancies linked to obesity.

In addition to ssRNA-seq, novel experimental methods including as microfluidics, 3D spheroids, and organs-on-chips have been used in the research of microenvironments. This system has been utilised in lymphoma research and consists of cancer cells, fibroblasts, and lymphocytes for the dynamic investigation of cellular interactions, proliferation, and treatment effectiveness. Spheroids are filled with a novel hydrogel based on droplet microfluidics to promote cell adhesion and aggregation.