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Commentary

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Obesity and Its Connection with the Immune System – Enhanced Cytokine Production

Jesmine Khan*

The interactions between nutrition and nutritional status and thus the immune function are the object of research for some time now, and it is known that under nutrition, or more generally, malnutrition, results in inadequate immune function and impaired immune competence, in order that the body is more susceptible to illness and diseases. Obesity, which could be a state of malnutrition by overabundance, has been related with resistant dysfunction as well, after observations of higher rates of diseases and impeded wound recuperating in corpulent subjects. Excess body fat is accompanied by changes in leucocyte counts, with elevated leucocyte, neutrophil, monocyte and lymphocyte checks, but lower T- and B-cell mitogen-induced proliferation. In addition, other ponders have appeared that the generation of antibodies after immunization is decreased in obese patients. Indeed in children prove was gotten of impaired cell-mediated resistant responses with obesity. Considering all this, it appears clear that obesity, like other circumstances of malnutrition, impairs immune function.

The Immune system-obesity

The link seems to be fat itself. There are several associations indeed between adipose tissue and the immune system. For a start, macrophages and lymphocytes can be normally found within the non-adipose fraction of the tissue. Additionally, white adipocytes have been proposed to share embryonic origin with immune cells, whereas characterisation of adipose tissue-resident lymphocytes driven to the notion that this tissue was an ancestral immune organ. And more as of late, immature haematopoietic cells have been found in adipose tissue, thus it has been proposed as a location for formation and maturation of immune cell precursors. In the early 2000s, histological ponders in mice showed that macrophage invasion in fat was more prominent in obese than in lean animals. The macrophages appeared as crown-shaped aggregates, almost like those observed in other known inflammatory conditions, like rheumatoid arthritis, and grew larger with increasing degrees of obesity.

This finding led to the idea that macrophage aggregates might partially explain the obesity-related incendiary state. In support of this speculation, two diverse phenotypes for adipose tissue-resident macrophages were later depicted: one that acts as pro-inflammatory (known as M1 or 'classically activated'), and another that acts as anti-inflammatory (M2 or 'alternatively activated'). Interestingly, obesity has been related with a switch from the M2 to the M1 phenotype; that's, to a more pro-inflammatory profile. Besides, the absence of the M2 phenotype has been associated with a better vulnerability to obesity, inflammation and insulin resistance. The enhanced macrophage invasion within the obese adipose tissue clarifies in part, but not completely, the increased production of cytokines and chemokines. These molecules are mainly produced by immune cells, e.g. monocytes, macrophages and T-lymphocytes, and also by other cells such as mast cells, fibroblasts, endothelial cells, neurons, or adipocytes themselves. Fat cells secrete, among others, TNFα, IL-6, monocyte chemotactic protein, transforming development factor β, or acute phase proteins. Consequently, alteration of adipocyte function plays an important role within the development of obesity-associated inflammation.

Why does an inflammatory response initiate within the adipose tissue?

This is still a sophisticated question to address. Cytokines are chemical messengers that are released to direct gene expression in a particular organ or cell type, and which interact in a complex way, mutually inducing or repressing their production and effects.

What is the reason or use of the enhanced cytokine production in obesity?

We can as it were speculate in this respect so far, but the answers can be searched in the enlarged, lipid-loaded adipocyte. During a situation of excessive nutrient influx and storage, there must be mechanisms operating within and from the cell in arrange to maintain or restore energy homoeostasis. Certain cytokines, e.g. IL-6 and TNFα, are known to induce insulin resistance, so their local production could constitute a regulatory mechanism to prevent the hypertrophied adipocyte from storing lipids. Or, macrophage infiltration in response to adipocyte-derived chemokines, such as monocyte chemotactic protein or macrophage migration inhibitory factor, may respond to the need for clearing the adipose tissue of dysfunctional and necrotic fat cells, such as phagocytes normally do, for instance, during an acute inflammatory response in the context of an infection. The problem arises when, as a result of sustained obesity, the inflammatory response does not achieve its goal and it's not resolved, thus turning from a local reaction to a systemic chronic state.

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***Corresponding author:** Jesmine Khan, Faculty of Medicine, Universiti Teknologi MARA, Malaysia, E-mail: jesminek454@gmail.com

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Author Affiliations Top

Faculty of Medicine, Universiti Teknologi MARA, Malaysia