Opinion

Medicine

Contribution of Body Fat to the Pathogenesis of Cancer

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The pathogenesis of cancer has been significantly influenced by body fat, which has the capacity to affect a variety of biological processes. Chronic inflammation and insulin resistance are known to be exacerbated by excess body fat, particularly visceral adipose tissue, which can establish an optimal microenvironment for tumor cell proliferation and progression. Adipocytes generate hormones and cytokines that can induce cancer cell proliferation, angiogenesis, and metastasis. Furthermore, adipose tissue serves as a storage facility for environmental toxins and carcinogens, which may be released into the bloodstream during periods of metabolic duress or weight loss. Additionally, obesity-induced modifications in the metabolism of adipose tissue can result in the dysregulation of lipid signaling pathways, which in turn induces tumorigenesis.

Keywords: Body Fat; Adipose Tissue; Pathogenesis; Cancer; Causal Relationship

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CANCER is the most prevalent cause of mortality worldwide, and it is anticipated that its prevalence will continue to increase in the years ahead. Excess body fat is widely recognized as a significant factor in the pathogenesis of cancer, despite the fact that the precise causes of cancer are not yet entirely understood (Khandekar, Cohen, & Spiegelman, 2011).

Excess body fat is a significant risk factor for a variety of cancers, such as breast, colon, kidney, and endometrial cancers. This is due to the fact that chronic low-grade inflammation, which is induced by excess body fat, impedes the body's capacity to combat cancerous cells and promotes the development of tumor cells (Calle & Kaaks, 2004). Furthermore, the accumulation of excess body fat results in hormonal imbalances, particularly an elevation in insulin and estrogen levels, which have been associated with the development of specific cancers (Sung et al., 2017).

Adipose tissue is identified as the source of a variety of bioactive substances known as adipokines, including leptin and adiponectin. These substances are involved in the regulation of inflammation, immune response, and cancer cell proliferation (Heilbronn & Campbell, 2008). Although adiponectin possesses anti-inflammatory and anti-cancer properties, leptin has been demonstrated to facilitate the growth and invasion of cancer

cells (Liu et al., 2017). Consequently, the pathogenesis of cancer may be influenced by an imbalance in the production of these adipokines as a result of excess body fat (Sivaprakasam et al., 2020).

Insulin resistance and type 2 diabetes are also associated with excess body fat, which have been associated with an elevated risk of specific types of cancer, including pancreatic and colorectal cancers (Giovannucci et al., 2010). Insulin resistance results in an increase in the circulating levels of insulin, which in turn facilitates the growth of cancer cells by providing them with the requisite nutrients and energy to increase their proliferation. Furthermore, insulin resistance has been demonstrated to facilitate the establishment of a pro-inflammatory microenvironment that is favorable to the proliferation and dissemination of cancer cells (Stan & Paul, 2024).

In addition to fostering the growth and proliferation of cancer cells, excess body fat also facilitates the metastasis of cancer cells to distant organs. The formation of new blood vessels, or angiogenesis, is a critical process in the dissemination of cancer cells, and adipose tissue is known to generate a variety of factors that facilitate this process (Luca et al., 2024). Additionally, adipose tissue generates factors that increase the motility and invasiveness of cancer cells, thereby facilitating their dissemination to remote regions of the body (Deng et al., 2016).

Research has demonstrated that body fat accumulation can exacerbate resistance to cancer treatment, particularly chemotherapy and radiation therapy. The reason for this is that adipose tissue functions as a reservoir for chemotherapeutic agents, which prevents them from reaching their target cells and reduces their efficacy (Biskupiak et al., 2024). Furthermore, the pro-inflammatory microenvironment that is generated by increased body fat can also shield cancer cells from the cytotoxic effects of radiation therapy and chemotherapy, thereby increasing their resistance to treatment (Săvulescu-Fiedler et al., 2024).

The development of a pre-cancerous condition known as fatty liver disease, or non-alcoholic fatty liver disease (NAFLD), has been demonstrated to be facilitated by excess body fat. NAFLD is defined by the accumulation of fat in the liver, which can result in liver injury and inflammation (Wang et al., 2018). Additionally, NAFLD has been associated with an elevated propensity to develop liver cancer, as well as other forms of cancer, including breast and colorectal cancer (Dinani & Sanyal, 2024). Consequently, the pathogenesis of cancer is influenced by excess body fat's direct effects on cancer cells and its indirect effects on the liver.

Research has demonstrated that excess body fat can suppress the immune system, particularly the activity of natural killer cells, which are essential for the elimination of cancer cells from the body (Wei et al., 2016). The detection and destruction of abnormal cells, such as cancer cells, prior to their development into tumors is the responsibility of natural killer cells. Nevertheless, the function of natural killer cells has been demonstrated to be impaired by excess body fat, which enables cancer cells to proliferate unabated and evade detection (Levy et al., 2011).

It has been demonstrated that excess body fat is conducive to the growth and dissemination of cancer cells by fostering the development of a pro-inflammatory microenvironment in the body. Adipose tissue produces a variety of pro-inflammatory cytokines, including tumor necrosis factor-alpha and interleukin-6, which facilitate the proliferation and growth of cancer cells (Vasilenko et al., 2019). This is the reason. Furthermore, an increase in C-reactive protein levels is precipitated by excess body fat, which is a marker of inflammation that has been linked to an elevated risk of cancer (Kaptoge et al., 2010).

In sum, the role of body fat in the pathogenesis of cancer is a multifaceted and intricate matter that encompasses a diverse array of biological processes and mechanisms. The development of cancer can be influenced by excess body fat through its effects on inflammation, hormone levels, insulin resistance, lipid metabolism, adipokine secretion, and other pathways. It is crucial to comprehend the function of body fat in the pathogenesis of cancer in order to create effective strategies for the prevention, detection, and treatment of the disease. By addressing obesity and its associated metabolic abnormalities, it may be possible to alleviate the burden of cancer and enhance the outcomes for cancer patients.■

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