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Commentary

Inflammation and Immunity Control in Obese Cancer Patients

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DESCRIPTION

Obesity is a global health crisis, significantly increasing the risk of various chronic diseases, including cancer. Recent research has highlighted the complex relationship between obesity, inflammation and anti-tumor immunity. Understanding the mechanisms by which obesity influences these processes is essential for developing effective cancer prevention and treatment strategies. This article describes the complex mechanisms through which obesity regulates inflammation and anti-tumor immunity in cancer.

Obesity is characterized by excessive accumulation of adipose tissue, which functions not only as a storage site for fat but also as an active endocrine organ. Adipose tissue secretes a variety of bioactive molecules, known as adipokines and inflammatory mediators, which play a significant role in modulating systemic inflammation and immune responses. In the context of obesity, the balance of these secretions is disrupted, leading to a chronic low-grade inflammatory state known as meta-inflammation.

Meta-inflammation is a important association between obesity and cancer. In obese individuals, enlarged adipocytes and increased fat mass lead to hypoxia, or reduced oxygen levels, within adipose tissue. Hypoxia induces the production of pro-inflammatory cytokines such as Tumor Necrosis Factor-Alpha (TNF-α), Interleukin-6 (IL-6), and Monocyte Chemoattractant Protein-1 (MCP-1). These cytokines attract immune cells, including macrophages, to the adipose tissue, where they adopt a pro-inflammatory phenotype. The resulting chronic inflammation contributes to a tumor-promoting environment.

Inflammatory cytokines produced in adipose tissue can enter the important and affect distant organs, including potential tumor sites. IL-6, for example, can promote cancer cell survival, proliferation and metastasis by activating signaling pathways such as STAT3 (Signal Transducer and Activator of Transcription 3). Elevated levels of IL-6 in obese individuals have been associated with poor prognosis in various cancers, including breast, colorectal and prostate cancers. TNF-α, another key inflammatory mediator, can enhance tumor growth by promoting angiogenesis, the formation of new blood vessels that supply nutrients to tumors.

The chronic inflammatory state in obesity also impairs anti-tumor immunity. Normally, the immune system can recognize and eliminate cancer cells through the actions of Cytotoxic T Lymphocytes (CTLs) and Natural Killer (NK) cells. However, in obese individuals, the function of these immune cells is compromised. Adipose tissue inflammation leads to the production of immunosuppressive molecules such as Transforming Growth Factor-Beta (TGF-β) and Interleukin-10 (IL-10). These molecules inhibit the activity of CTLs and NK cells, reducing their ability to target and kill cancer cells.

Furthermore, obesity is associated with an increase in Myeloid-Derived Suppressor Cells (MDSCs) and Regulatory T Cells (Tregs), both of which contribute to an immunosuppressive tumor microenvironment. MDSCs are a heterogeneous population of immune cells that inhibit T cell activation and promote tumor growth. They are recruited to tumor sites by chemokines produced in the inflammatory adipose tissue. Once in the tumor microenvironment, MDSCs suppress anti-tumor immunity by producing Reactive Oxygen Species (ROS), Nitric Oxide (NO), and arginase, which interfere with T cell function.

Tregs, on the other hand, are a subset of T cells that maintain immune tolerance and prevent autoimmune responses. In the context of cancer, Tregs are often co-opted to suppress anti-tumor immune responses. Obesity promotes the expansion and accumulation of Tregs in adipose tissue and tumors through the action of adipokines such as leptin. Elevated leptin levels in obesity enhance Treg proliferation and function, further inhibiting the activity of CTLs and NK cells.

CONCLUSION

In conclusion, obesity exerts a extreme influence on inflammation and anti-tumor immunity through multiple interconnected mechanisms. The chronic low-grade inflammation associated with obesity creates a tumor-promoting environment by enhancing pro-inflammatory cytokine production, impairing the function of

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cytotoxic immune cells and promoting the expansion of immunosuppressive cell populations. The imbalance of adipokines, metabolic dysregulation and alterations in gut microbiota further contribute to this complex interplay. Addressing obesity and its associated metabolic and inflammatory disturbances is crucial for improving cancer prevention and treatment outcomes. Understanding these mechanisms provides valuable insights for developing targeted interventions to modulate inflammation and enhance anti-tumor immunity in obese cancer patients.