How cancer is triggered by inflammation.

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Abstract

A recent study demonstrates how cancer might be aided by inflammation. The research discovered that microRNA-155 levels rose in response to inflammation (miR-155). A decrease in DNA repair proteins as a result of this rise leads to a rise in spontaneous gene mutations, which can aid in the development of cancer. The results imply that medications intended to lower miR-155 levels might enhance the treatment of malignancies connected to inflammation.

Keywords: Inflammation, Cancer, Oncology, Breast cancer.

Introduction

Inflammation prompts a rise in levels of a molecule called microRNA-155, according to research from the Ohio State University Comprehensive Cancer Center Arthur G. James Cancer Hospital and Richard J. Solove Research Institute (OSUCCC - James) (miR-155). As a result, the amounts of proteins involved in DNA repair decrease, increasing the likelihood of spontaneous gene mutations that might result in cancer. Research demonstrates that miR-155 is elevated in response to inflammatory stimuli and that miR-155 overexpression enhances the rate of spontaneous mutation, which can aid in carcinogenesis Dr. Esmerina Tili, the study's first author and a postdoctoral researcher, adds. Research offers a biological explanation that clarifies the long-held belief that inflammation plays a significant role in cancer. Findings also imply that tumours associated with inflammation would respond better to medications made to lower miR-155 levels [1].

A wide family of non-coding genes called microRNAs is involved in numerous crucial cellular functions. They do this job by reducing the quantity of specific proteins in cells, with each kind of microRNA frequently having an impact on a wide range of proteins. High levels of MiR-155 have been directly connected to the emergence of leukemia, as well as breast, lung, and stomach malignancies. This molecule is also known to affect immunological responses, autoimmune disorders, and blood-cell maturation. Tili and her coworkers looked at how miR-155 expression and the frequency of spontaneous mutations were affected by inflammatory agents like tumour necrosis factor and lipopolysaccharide (present in bacterial cell walls) in a number of breast cancer cell lines [2].

Breast cancer cells exposed to the two inflammatory stimuli had abnormally high levels of miR-155 and a two- to three-fold rise in mutation rates. The WEE1 gene, which prevents cell division to enable damaged DNA to be repaired, was the focus of the investigators' efforts to determine why. The researchers discovered that miR-155 targets WEE1 as well, and they demonstrated how high miR-155 concentrations result in low WEE1 concentrations. Low levels of WEE1 allowed cell division to proceed even in the presence of DNA damage, they reasoned, increasing the number of mutations [3].

Inflammation is frequently linked to the onset and spread of cancer. Targeting inflammation is an appealing technique for both cancer prevention and cancer therapy because the cells that cause cancer-associated inflammation are genetically stable and do not quickly develop drug resistance. Numerous conditions, such as bacterial and viral infections, autoimmune disorders, obesity, tobacco use, asbestos exposure, and excessive alcohol use all contribute to tumor-extrinsic inflammation, which in turn promotes the growth of cancer cells. By attracting and activating inflammatory cells, cancer-intrinsic or cancer-elicited inflammation, in contrast, can be brought on by cancer-initiating mutations. It can also aid in the development of malignancies. Both intrinsic and extrinsic inflammations may lead to immunosuppression, which creates a favorable environment for the growth of tumours [4].

The body's reaction to tissue damage brought on by physical trauma, ischemia injury (produced by insufficient blood flow to an organ), infection, toxin exposure, or other types of stress is inflammation. The inflammatory response of the body results in immunological reactions and cellular changes that lead to tissue repair and cellular proliferation (growth) at the site of the wounded tissue. If the underlying cause of the inflammation doesn't go away or certain control systems meant to stop the process from continuing, the inflammation may become chronic. Chronic inflammatory responses can

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lead to cell proliferation and mutation, frequently fostering the conditions for the growth of cancer. Cancer patients face a severe struggle known as the perfect storm [5].

Conclusion

Overall, this review offers proof that there is a direct connection between persistent inflammation and cancer. As a result, the development of the disease can be tracked using the inflammatory biomarkers discussed above. These biomarkers can also be used to create fresh anti-inflammatory medicines for the treatment and prevention of cancer. Additionally, as radiotherapy and chemotherapy by themselves activate NF-B and mediate resistance, these medications can be utilized as an adjuvant to these treatments. It has been demonstrated that many anti-inflammatory drugs, including those found in natural sources, possess chemo preventive properties.

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