Commentary

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The Interplay between Inflammation and Neoplasm Development

Marco Kim*

Department of Pathology, Duke University, Durham, NC, 27710, USA

Corresponding author: Marco Kim, Department of Pathology, Duke University, Durham, NC, 27710, USA, E-mail: marco@kim.edu

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Introduction

The relationship between inflammation and neoplasm development has gained increasing attention in cancer biology, as chronic inflammation is now recognized as a critical factor in tumor initiation, progression, and metastasis. While acute inflammation is a protective physiological response aimed at tissue repair and pathogen clearance, persistent or deregulated inflammation creates a tumor-promoting microenvironment. This interplay between inflammatory pathways and oncogenic processes underlines the complexity of cancer development and offers important insights into prevention and therapeutic strategies.

Description

At the molecular level, chronic inflammation drives neoplasm development by sustaining proliferative signaling, inducing genetic mutations, and creating oxidative stress. Pro-inflammatory mediators such as cytokines, chemokine and growth factors contribute to DNA damage, epigenetic alterations, and activation of oncogenic pathways including NF-κB and STAT3. Inflammatory cells within the tumor microenvironment, particularly macrophages and neutrophils, secrete reactive oxygen species and proteolytic enzymes that not only damage host tissue but also promote mutagenesis, supporting malignant transformation of normal cells [1-3].

Clinically, many cancers are linked to chronic inflammatory conditions, such as colorectal cancer arising from inflammatory bowel disease, hepatocellular carcinoma associated with chronic hepatitis, and gastric cancer linked to Helicobacter pylori infection. In these contexts, persistent immune responses continuously stimulate cell turnover, enhancing the likelihood of malignant transformation.

This dual role of inflammation as both a driver of cancer initiation and a facilitator of its progression underscores its significance in oncology therapeutically, understanding the inflammation–neoplasm connection has paved the way for novel treatment strategies. Anti-inflammatory drugs, cytokine inhibitors, and immune checkpoint therapies are being investigated for their potential to disrupt the tumor-promoting effects of inflammation [4,5].

Conclusion

In summary, the interplay between inflammation and neoplasm development illustrates how a protective biological response can, under chronic conditions, evolve into a driver of malignancy. By elucidating the mechanisms through which inflammatory processes influence cancer initiation and progression, researchers and clinicians can design targeted strategies to mitigate these effects. Integrating anti-inflammatory approaches with conventional cancer therapies holds promise for reducing cancer incidence, preventing recurrence, and improving patient outcomes.

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