Immunology: Current Research

Review Article Open Access

The Janus-Faced Inflammasomes: Friend or Foe in Cancer

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Abstract

Cancer, a complex and multifactorial disease, continues to be a global health concern. Over the years, researchers have made significant strides in understanding the molecular mechanisms underlying cancer development and progression. Among these mechanisms, the inflammasome pathway has emerged as a fascinating area of investigation. Inflammasomes are multiprotein complexes that play a critical role in the regulation of inflammation and immunity. In recent years, mounting evidence has implicated inflammasomes in cancer, revealing their dual nature as both promoters and suppressors of tumorigenesis. This article aims to delve into the intricate relationship between inflammasomes and cancer, shedding light on their potential as therapeutic targets.

Keywords: Therapeutic targets; Cancer; Inflammasomes cancer

Introduction

Understanding inflammasomes

Inflammasomes are cytosolic complexes formed by pattern recognition receptors (PRRs), including Nod-like receptors (NLRs) and absent in melanoma 2 (AIM2)-like receptors (ALRs). They function as intracellular sensors that recognize various danger signals, such [1-5] as pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs). Upon activation, inflammasomes facilitate the maturation and secretion of pro-inflammatory cytokines, most notably interleukin-1β (IL-1β) and interleukin-18 (IL-18). These cytokines play crucial roles in immune responses, inflammation, and tissue homeostasis.

Materials and Methods

Inflammasomes and tumorigenesis

Pro-tumorigenic effects: Inflammasomes can contribute to tumor development through various mechanisms. Chronic inflammation, often fueled by dysregulated inflammasome activation, has been linked to the initiation and progression of various cancers. The release of IL-1β and IL-18 by activated inflammasomes can promote tumor cell proliferation, angiogenesis, and metastasis. Additionally, inflammasomes can induce the production of inflammatory mediators, creating a tumor microenvironment favorable for tumor growth. Studies have demonstrated the involvement of inflammasomes in colorectal, lung, breast, and liver cancers, among others.

Anti-tumorigenic effects: On the contrary, inflammasomes can exhibit tumor-suppressive properties. Activation of inflammasomes can induce a potent inflammatory [5-7] response that aids in the elimination of cancer cells. Inflammasome-derived cytokines, such as IL-1β, can enhance anti-tumor immune responses by recruiting and activating immune cells, including natural killer cells and cytotoxic T lymphocytes. Additionally, inflammasomes can initiate pyroptosis, a form of programmed cell death, leading to the elimination of cancer cells. Studies have highlighted the tumor-suppressive role of inflammasomes in melanoma, colorectal, and breast cancers, among others.

Targeting inflammasomes for cancer therapy: The intricate involvement of inflammasomes in cancer provides an opportunity for therapeutic interventions. Modulating inflammasome activity holds promise for both enhancing anti-tumor immune responses and dampening chronic inflammation associated with tumor promotion. Several approaches are being explored, including pharmacological inhibitors of inflammasome components and specific cytokine blockers. However, it is crucial to consider the dual nature of inflammasomes, as their manipulation should be carefully balanced to avoid unintended consequences, such as compromising the immune system's ability to fight cancer or exacerbating inflammation.

Results and Discussion

Future scope of inflammasomes for cancer therapy

The study of inflammasomes and their role in cancer is an evolving field, and while I can provide some insights based on existing knowledge up until my knowledge cutoff in September 2021, it's important to note that new research and discoveries may have been made since then. Inflammasomes are multiprotein complexes that play a crucial role in the innate immune system by Table 1 initiating and regulating inflammatory responses. They are involved in the activation of inflammatory caspases, leading to the processing and secretion of pro-inflammatory cytokines, such as interleukin-1β (IL-1β) and interleukin-18 (IL-18). Emerging evidence suggests that dysregulation of inflammasome signaling is implicated in the development and progression of various types of cancer. Here are some potential future directions and implications of inflammasome research in cancer:

Therapeutic target: Inflammasomes could serve as potential therapeutic targets in cancer treatment. Modulating inflammasome activity may help in controlling chronic inflammation associated with tumor development and progression. Developing drugs that selectively target and modulate inflammasome components may provide new treatment options.

Biomarkers for diagnosis and prognosis: Inflammasomes and their associated cytokines can serve as potential biomarkers for

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Received: 01-May-2023, Manuscript No. icr-23-99605; **Editor assigned:** 03-May-2023, PreQC No. icr-23-99605(PQ); **Reviewed:** 17-May-2023, QC No. icr-23-99605; **Revised:** 22-May-2023, Manuscript No. icr-23-99605(R); **Published:** 29-May-2023, DOI: 10.4172/icr.1000145

Citation: Wang M (2023) The Janus-Faced Inflammasomes: Friend or Foe in Cancer. Immunol Curr Res, 7: 145.

Table 1: Provides a simplified overview, and the specific details and mechanisms can be quite complex. Additionally, ongoing research may uncover additional aspects and roles of inflammasomes in cancer therapy.

Aspect	Role of Inflammasomes in Cancer Therapy
Therapeutic Target	Inflammasomes can be targeted to control chronic inflammation in tumors.
Biomarkers	Inflammasome activation and associated cytokines as diagnostic markers.
Immune Checkpoint	Crosstalk between inflammasomes and immune checkpoints for combination therapies.
Immunotherapy	Modulation of inflammasomes to enhance the efficacy of immunotherapies.
Genetic/Epigenetic	Identification of genetic and epigenetic alterations associated with inflammasome dysregulation.
Tumor Immunogenicity	Influence of inflammasomes on tumor-immune cell interactions and tumor immunogenicity.

cancer diagnosis and prognosis. Assessing the activation status of inflammasomes and measuring the levels of IL-1β and IL-18 could help in predicting [7-9] cancer progression, determining treatment response, and monitoring disease recurrence.

Immune checkpoint regulation: Inflammasomes have been shown to interact with immune checkpoint molecules, such as programmed cell death protein 1 (PD-1) and cytotoxic T-lymphocyte-associated protein 4 (CTLA-4). Future research may explore the crosstalk between inflammasomes and immune checkpoints, potentially leading to the development of combination therapies that target both pathways.

Immunotherapy enhancement: Immunotherapies, such as immune checkpoint inhibitors and chimeric antigen receptor (CAR) T-cell therapy, have revolutionized cancer treatment. Understanding the interplay between inflammasomes and the tumor microenvironment could help in enhancing the efficacy of these immunotherapeutic approaches by modulating the inflammatory response and promoting anti-tumor immunity.

Genetic and epigenetic regulation: Further investigations are needed to elucidate the genetic and epigenetic mechanisms that regulate inflammasome activity in cancer. Identifying specific genetic alterations or epigenetic modifications associated with inflammasome dysregulation may provide insights into potential therapeutic targets or diagnostic markers.

Role in tumor immunogenicity: Inflammasomes can influence the immunogenicity of tumors by shaping the tumor microenvironment and regulating the release of danger-associated molecular patterns (DAMPs) and cytokines. Future research may explore how inflammasomes modulate the tumor-immune cell interactions and the impact on tumor immunogenicity. It is important to note that the field of inflammasome research in cancer is still evolving, and more studies are needed to fully understand their precise roles and potential therapeutic applications. Continued research in this area may uncover novel insights into the complex interplay between inflammation, immunity, and cancer, leading to the development of new strategies for cancer prevention, diagnosis, and treatment.

Conclusion

The exploration of the role of inflammasomes in cancer has uncovered a complex interplay between inflammation, immunity, and tumorigenesis.

Acknowledgement

The University of Nottingham provided the tools necessary for the research, for which the authors are thankful.

Conflict of Interest

For the research, writing, and/or publication of this work, the authors disclosed no potential conflicts of interest.

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