



Apoptosis as a Target for Cancer Therapy: Exploiting Cell Death Pathways

Sanjeev kaur*

Department of Biotechnology, Panjab University, Chandigarh, India

*Corresponding author: Sanjeev kaur, Department of Biotechnology, Panjab University, Chandigarh, India; E-mail: sanjeevkaur@puc22.in

Received date: 22 March, 2023, Manuscript No. JCEOG-23-99206;

Editor assigned date: 24 March, 2023, PreQC No. JCEOG-23-99206 (PQ);

Reviewed date: 07 April, 2023, QC No. JCEOG-23-99206;

Revised date: 14 April, 2023, Manuscript No. JCEOG-23-99206 (R);

Published date: 21 April, 2023, DOI: 10.4172/2324-9110.1000348

Description

Cancer continues to be a significant global health challenge, driving the search for effective therapeutic strategies. Apoptosis, a tightly regulated process of programmed cell death, plays a vital role in maintaining tissue homeostasis. Dysregulation of apoptosis is a hallmark of cancer, as cancer cells evade cell death and proliferate uncontrollably. Exploiting the cell death pathways and targeting apoptosis has emerged as a promising approach in cancer therapy. Apoptosis is mediated through two primary signaling pathways they are intrinsic (mitochondrial) pathway and the extrinsic (death receptor) pathway. The intrinsic pathway is initiated by various intracellular stress signals, such as DNA damage or cellular stress. These signals lead to the activation of pro-apoptotic proteins, including members of the B-Cell Lymphoma-2 (BCL-2) family, which regulate the permeability of the mitochondrial membrane. Disruption of mitochondrial integrity results in the release of cytochrome c and subsequent activation of caspases, leading to cell death.

The extrinsic pathway, on the other hand, is triggered by the engagement of death receptors on the cell surface by specific ligands. This interaction leads to the formation of a Death-Inducing Signaling Complex (DISC) and subsequent activation of caspases, ultimately resulting in apoptosis. Both pathways converge at the activation of caspases, which execute the dismantling of the cell. Exploiting apoptosis as a therapeutic target in cancer involves different strategies that aim to either reactivate apoptosis in cancer cells or enhance the sensitivity of cancer cells to apoptotic stimuli. Several approaches have been developed to achieve these goals. One approach involves

the use of small-molecule inhibitors or targeted therapies that directly target components of the apoptotic pathways. For instance, targeting anti-apoptotic proteins of the BCL-2 family, such as B-cell lymphoma 2 (BCL-2), B-Cell Lymphoma-Extra Large (BCL-XL), or Myeloid Leukemia-1 (MCL-1), with specific inhibitors can restore apoptotic signaling in cancer cells. Additionally, agents that mimic the actions of death ligands, such as TNF-Related Apoptosis-Inducing Ligand (TRAIL) have shown promise in selectively inducing apoptosis in cancer cells.

Another strategy involves combination therapies that aim to sensitize cancer cells to apoptotic signals. This approach often involves combining conventional cancer therapies, such as chemotherapy or radiation, with agents that modulate apoptotic pathways. For example, combining chemotherapy agents with Inhibitors of Apoptosis Proteins (IAPs) can enhance the sensitivity of cancer cells to apoptosis. Immunotherapy has also emerged as a promising avenue in utilizing apoptosis for cancer treatment. Strategies such as immune Checkpoint Inhibitors or Chimeric Antigen Receptor (CAR) T-cell therapy can reinvigorate the immune system and promote cancer cell apoptosis through immune-mediated mechanisms.

While targeting apoptosis in cancer therapy holds significant promise, several challenges need to be addressed. Resistance to apoptosis is a common feature in cancer, often attributed to dysregulation of apoptotic regulators or activation of survival pathways. Overcoming these resistance mechanisms is essential to maximize the therapeutic potential of apoptosis-targeting strategies. Moreover, understanding the context-specific regulation of apoptosis is essential. Apoptosis signaling can differ among different cancer types or even within the same tumor, necessitating personalized approaches. Furthermore, identifying predictive biomarkers of response to apoptosis-targeting therapies will aid in patient stratification and treatment selection.

Conclusion

Apoptosis, as a target for cancer therapy, provides a promising avenue in the fight against cancer. Exploiting the cell death pathways through the reactivation of apoptosis or sensitisation of cancer cells to apoptotic stimuli holds tremendous potential for improved treatment outcomes. However, addressing resistance mechanisms, understanding context-specific regulation, and identifying predictive biomarkers are important steps to maximize the therapeutic benefits of apoptosis-targeting strategies. With continued studies and development, apoptosis-based therapies may significantly impact the future of cancer treatment.

Citation: Kaur S (2023) Apoptosis as a Target for Cancer Therapy: Exploiting Cell Death Pathways. *J Clin Exp Oncol* 12:2.