Targeting molecular pathways in cancer therapy.

Santarpia Naggar*

Department of Oncology, Istituto Toscano Tumori, Prato and Florence, Italy

Received: *27-Apr-2023, Manuscript No. AAMOR-23-97144;* ***Editor assigned:*** *01-May-2023, AAMOR-23-97144 (PQ);* ***Reviewed:*** *15-May-2023, QC No. AAMOR-23-97144;* ***Revised:*** *27-Jun-2023, Manuscript No. AAMOR-23-97144 (R);* ***Published:*** *04-Jul-2023, DOI:10.35841/aamor.7.5.194*

Introduction

Cancer is a complex and heterogeneous group of diseases that arise due to the accumulation of genetic and epigenetic alterations that lead to uncontrolled cell growth and proliferation [1]. In the last few decades, the understanding of the molecular mechanisms underlying cancer development and progression has greatly advanced, leading to the identification of several molecular pathways that are frequently dysregulated in different types of cancer. This knowledge has opened up new avenues for the development of targeted cancer therapies that aim to selectively inhibit these aberrant pathways.

Description

Targeted therapy is a type of cancer treatment that uses drugs or other substances to identify and attack specific cancer cells, while sparing the healthy ones. Unlike traditional chemotherapy, which indiscriminately kills both cancerous and non-cancerous cells, targeted therapy is designed to act selectively on molecular pathways that are altered in cancer cells [2]. The goal of targeted therapy is to disrupt the signaling pathways that are essential for cancer cell survival and proliferation, while minimizing the side effects associated with traditional chemotherapy.

There are several molecular pathways that have been targeted in cancer therapy. One of the most well-known examples is the inhibition of the Epidermal Growth Factor Receptor (EGFR) pathway in non-small cell lung cancer. EGFR is a receptor tyrosine kinase that is frequently overexpressed or mutated in lung cancer, leading to constitutive activation of downstream signaling pathways that promote cell growth and survival [3]. Targeted inhibitors of EGFR, such as erlotinib and gefitinib, have shown significant clinical activity in patients with advanced lung cancer, particularly those with EGFR mutations.

Another example of a molecular pathway that has been targeted in cancer therapy is the B-cell lymphoma-2 (Bcl-2) pathway in Chronic Lymphocytic Leukemia (CLL). Bcl-2 is an anti-apoptotic protein that is overexpressed in CLL, promoting cell survival and resistance to chemotherapy. Venetoclax, a selective inhibitor of Bcl-2, has shown promising results in patients with CLL, leading to high response rates and durable remissions [4].

In addition to these examples, several other molecular pathways have been targeted in cancer therapy, including the PI3K-AKT-mTOR pathway, the Ras-MAPK pathway, the Wnt/β-catenin pathway and the Hedgehog pathway, among others. The development of targeted therapies requires a deep understanding of the molecular mechanisms underlying cancer development and progression, as well as the identification of specific biomarkers that can be used to select patients who are most likely to benefit from these therapies [5].

Despite the promise of targeted therapies, there are several challenges that need to be overcome for their widespread adoption. One of the main challenges is the development of resistance to these therapies, which can occur through the activation of alternative signaling pathways or the emergence of new mutations in the target pathway. Resistance can also arise due to the heterogeneity of cancer cells within a tumor, leading to the emergence of subpopulations of cells that are resistant to the targeted therapy.

Another challenge is the identification of biomarkers that can reliably predict response to targeted therapies. Biomarkers can be used to select patients who are most likely to benefit from these therapies, avoiding unnecessary treatment and minimizing the risk of toxicity. However, the identification of biomarkers is often complicated by the heterogeneity of cancer cells within a tumor, as well as by the dynamic nature of the tumor microenvironment.

Conclusion

Targeted therapy has revolutionized cancer treatment by providing a more selective and less toxic approach to cancer therapy. The identification of molecular pathways that are frequently dysregulated in cancer has paved the way for the development of targeted therapies that aim to selectively inhibit these pathways. However, the development of resistance and the identification of reliable biomarkers remain significant challenges in the field of targeted therapy. Future research efforts should focus on overcoming these challenges and developing more effective and personalized.

References

1. Lapidot T, Sirard C, Vormoor J, et al. A cell initiating human acute myeloid leukaemia after transplantation into SCID mice. Nature. 1994;367(6464):645-8.
2. Fang D, Nguyen TK, Leishear K, et al. A tumorigenic subpopulation with stem cell properties in melanomas. Cancer Res. 2005;65(20):9328-37.

3. Ma S, Chan KW, Hu L, et al. Identification and characterization of tumorigenic liver cancer stem/progenitor cells. Gastroenterology. 2007;132(7):2542-56.
4. Chen J, Li Y, Yu TS, et al. A restricted cell population propagates glioblastoma growth after chemotherapy. Nature. 2012;488(7412):522-6.
5. Schwitalla S. Tumor cell plasticity: The challenge to catch a moving target. J Gastroenterol. 2014;49(4):618-27.

***Correspondence to**

Santarpia Naggar

Department of Oncology,

Istituto Toscano Tumori,

Prato and Florence,

Italy

E-mail: *naggar.samtarpia@ucd.edu.org*