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TARGETED CANCER THERAPY: A NEW ERA OF MOLECULAR ONCOLOGY

Gera Preethi Sowjanya | Karri Monika Bhagya Lakshmi | Dr.Kiran Manda | Alla Uma Sri
Department of pharmaceutical chemistry, Shri Vishnu college of Pharmacy (Autonomous), Affiliated to Andhra University, Bhimavaram, West Godavari District, Andhra Pradesh, India.
Corresponding author mail id: umasrialla238@gmail.com

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ABSTRACT
Cancer continues to be a major cause of sickness and death worldwide, and because of its toxicity, resistance, and recurrence, it frequently eludes conventional treatments. In oncology, molecular targeted therapy has become a game-changer, allowing for precision treatment by taking advantage of particular genetic and molecular defects in cancer cells. Targeted treatments, such as immune checkpoint inhibitors, monoclonal antibodies, antibody-drug conjugates, and tyrosine kinase inhibitors, provide better therapeutic results, decreased systemic toxicity, and increased selectivity in contrast to traditional chemotherapy. Important molecular targets like EGFR, HER2, VEGFR, BRAF, and PI3K are highlighted in this study along with their significance for the clinical efficacy of treatments for colorectal, breast, and non-small cell lung cancer. Notwithstanding these developments, problems such medication resistance, tumour heterogeneity, and accessibility constraints still exist. Evolving technologies that have the potential to improve precision oncology and optimize therapeutic approaches include CRISPR-Cas9, AI-driven biomarker identification, and multi-omics integration. To overcome existing obstacles and maximize the worldwide impact of molecular targeted cancer therapy, ongoing research, innovation, and equitable healthcare implementation are essential. Drug resistance, tumour heterogeneity, and restricted accessibility are still issues even though these medicines have transformed the management of a number of malignancies, including breast and lung cancer. Targeted treatment will be a key component of cancer care in the future as a result of developments in genomics, CRISPR gene editing, and biomarker identification that are opening the door to more individualized, efficient treatment.

1.INTRODUCTION

Cancer remains one of the world's top causes of illness and mortality, posing significant challenges to global healthcare systems. Despite improvements in conventional therapies such as radiation, chemotherapy, and surgery, many cancer

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types continue to exhibit resistance, recurrence, and severe patient toxicity [1]. In recent decades, molecular targeted therapy has emerged as a promising strategy to improve the outcomes of cancer treatment by utilizing the unique molecular aberrations of cancer cells while protecting healthy tissues. This method improves treatment efficacy while lowering systemic toxicity [1].

1.1The Rationale for Molecular Targeted Therapy

The understanding that cancer is essentially a hereditary illness with dysregulated signalling pathways that encourage unregulated proliferation, survival, angiogenesis, and metastasis is the foundation for the reasoning behind molecular targeted therapy. These abnormal molecular processes arise from oncogenes, tumour suppressor genes, and other regulatory molecules undergoing genetic mutations, amplifications, deletions, or epigenetic changes. In contrast to conventional cytotoxic medicines, researchers have created therapeutics that directly inhibit cancer-promoting proteins or pathways by identifying and targeting these particular molecular drivers [2,3]

Treatment is adapted to the genetic profile of the tumour and its surrounding environment thanks to the paradigm shift from non-specific chemotherapeutic drugs to molecular targeted therapy. Because medications are made to disrupt the molecular aberrations unique to cancer rather than indiscriminately attacking all rapidly dividing cells, this strategy may result in increased efficacy and fewer side effects [4].

1.2Key Molecular Targets and Therapeutic Agents

Numerous molecular target classes have been discovered and used in cancer treatment. Apoptosis-regulating proteins like BCL-2, intracellular signalling molecules like BRAF, MEK, and PI3K, and receptor tyrosine kinases (RTKs) like vascular endothelial growth factor receptor (VEGFR), human epidermal growth factor receptor 2 (HER2), and epidermal growth factor receptor (EGFR) are among them . Tyrosine kinase inhibitors (TKIs) in small molecules, monoclonal antibodies, antibody-drug conjugates, and, more recently, immune checkpoint inhibitors that alter the tumour immunological milieu are examples of targeted agents [5].

By selectively blocking aberrant EGFR signalling in tumours containing EGFR mutations, for example, EGFR inhibitors have revolutionized the treatment of non-small cell lung cancer (NSCLC) [6]. Similar to this, patients with HER2-positive breast cancer now have significantly better results thanks to HER2-targeted treatments like trastuzumab. In order to prevent tumour angiogenesis and deprive tumours of their blood supply, VEGF inhibitors have been utilized extensively.

1.3 Advantages and Challenges

Compared to traditional chemotherapy, molecular targeted therapies provide a number of benefits, such as increased specificity, less toxicity, and the ability to be combined with other modalities to overcome resistance. The creation and clinical application of targeted medicines are not without difficulties, though, in spite of these advantages.

One significant drawback is the emergence of drug resistance, which can be caused by

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a number of different processes, including tumour heterogeneity, alternative signalling pathway activation, and secondary mutations in target genes. Both intra tumoral and inter-tumour heterogeneity make treatment more difficult because certain cancer cell subpopulations may not have the target mutation or may become resistant over time, which might cause the disease to persist even after an initial response.

Additionally, advanced genomic profiling techniques are necessary for identifying appropriate molecular targets, and they may not be easily available in all clinical settings, particularly in countries with limited resources. Furthermore, the high expense of targeted medicines presents major obstacles to their broad use, bringing up crucial issues regarding healthcare equity [7].

1.4 Emerging Technologies and Future Directions

Recent developments in genome editing technologies, especially CRISPR-Cas9, provide fresh chances to get past some of these obstacles by allowing for accurate genetic modification of cancer cells, which may reverse medication resistance or increase sensitivity to certain medicines. Furthermore, the creation of more efficient and individualized treatment plans is being made easier by the rapid identification of new targets and predictive biomarkers brought about by the combination of multi-omics data, artificial intelligence, and machine learning. Combination therapies are becoming more popular as a means of addressing tumour heterogeneity and resistance mechanisms. They employ molecular targeted medicines in conjunction with immunotherapies or chemotherapies, and they have the potential to improve clinical outcomes through synergistic effects [8,9,10].

2.MAIN SECTIONS

2.1 Targets in Molecular Medicine for Cancer

The knowledge that certain genetic and molecular abnormalities cause cancer and propel tumour initiation and growth is capitalized upon by molecular targeted therapy. The major categories of these molecular targets include intracellular signalling molecules, cell surface receptors, and molecules involved in the regulation of apoptosis.

Tyrosine Kinases (RTKs) in Receptors.

Some of the most researched targets include RTKs, including EGFR, VEGFR, and HER2. These receptors modulate growth factor signals that control angiogenesis, cell survival, and proliferation. Cell growth is out of control when there is aberrant activation due to mutations or overexpression. In NSCLC patients with EGFR mutations, EGFR therapies have demonstrated impressive efficacy. Similarly, the prognosis for HER2-positive breast cancer has improved using HER2-targeted treatments such as trastuzumab [11].

2.2 Intracellular Signalling Pathways

Proliferative and survival signals are transmitted by downstream signalling molecules like BRAF, MEK, and PI3K. Numerous malignancies frequently exhibit mutations in these pathways, which small molecule inhibitors can target therapeutically. By focusing on these molecules, tumour growth is inhibited by breaking oncogenic signalling cascades[12,13] .

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2.3 Apoptosis Regulators

Proteins including those in the BCL-2 family that regulate programmed cell death are also targeted to induce apoptosis in cancer cells [14]. Treatment efficacy is increased by modifying these regulators, which promotes the death of cancer cells.

3. CLASSES OF MOLECULAR TARGETED THERAPIES

There are several types of targeted therapies, all of which are intended to selectively disrupt the chemicals that cause cancer.

3.1 Small Molecule Inhibitors

These tiny molecular weight medications have the ability to enter cells and stop intracellular targets' kinase activity. TKIs targeting EGFR, BRAF, and MEK are a few examples. Although they are convenient due to their oral absorption, resistance is still a problem [15].

3.2 Monoclonal Antibodies

In order to prevent ligand binding or receptor dimerization, these biologics engage extracellular targets such RTKs. Examples are bevacizumab (VEGF) and trastuzumab (HER2) [16].

3.3 Antibody-Drug Conjugates (ADCs)

ADCs combine selectivity and cytotoxicity by binding cytotoxic chemicals to antibodies that target cancer cells. They offer a viable strategy for raising the therapeutic index [16].

3.4 Immune Checkpoint Inhibitors

By altering the immune response to malignancies, checkpoint inhibitors (such as anti-PD-1 and anti-CTLA-4) support molecular targeted treatments, despite their primary use in immunotherapy.

4.Strategies in Targeted Cancer Treatments and measures for precision medicine.

Strategy Category	Strategy	How it Works	Advantages / Challenges	Precision Medicine Measures
Enzyme/small molecule-based	**Protac (proteolysis targeting chimeras)**	Using the ubiquitin-proteasome system, bifunctional molecules enlist e3 ligase to break down target proteins.	Improves tissue distribution, stability, and solubility; yet ligand and linker modification presents limitations.	1. Look for mutations that affect ligand binding. 2. Verify suitable expression of the target protein.
	Antibody-drug conjugates (adcs)	Locally, an enzyme that is given to the tumour transforms a non-toxic prodrug into a cytotoxic drug.	Despite the existence of fda-approved medications, problems include	1. Determine whether the target antigen is expressed. 2. Check for

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Strategy Category	Strategy	How it Works	Advantages / Challenges	Precision Medicine Measures
			drug resistance and target antigen downregulation. .	mutations that could affect antibody binding. 3. The process of choosing patients
	Enzyme/Prodrug & ADEPT	Locally, a tumour-delivered enzyme converts a non-toxic prodrug into a cytotoxic drug.	Restricted by problems with the immune system, quick clearance, and the absence of tumour-specific enzymes	Target expression and genetic stability are identical to those of ADCs.
Immunotherapy-Based	**Immune Checkpoint Inhibitors (ICIs)**	Inhibit PD-1, PD-L1, and CTLA-4 pathways to system's defences strengthen the immune against cancer.	Resistance and toxicities that are effective yet immune-related can happen .	1. Examine the expression of PD-L1. 2. Check for alterations that impact the binding of inhibitors.
	Immune Cytokines & Antibody–Cytokine Fusions	Locally provide cytokines to boost the immune system's defences against tumours.	Provide cytokines locally to strengthen the immune system's anti-tumour responses.	1. Find the biomarkers that predict outcomes. 2. Establish safety profiles tailored to individual patients .
Combination Therapy	**Cancer Combination Therapy**	Use a variety of treatments with various mechanisms to increase effectiveness and lower resistance.	This can improve results, but it also raises toxicity and causes problems with patient tolerance.	Careful tracking and patient selection ensure a balance between side effects and effectiveness.

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4. TARGETED THERAPIES FOR SPECIFIC CANCER TYPES

1. Breast Cancer: Targeted treatments for breast cancer concentrate on particular molecular changes that fuel the tumour.
   Advancement for instance, HER2-targeted treatments such as trastuzumab, pertuzumab, and ado-trastuzumab emtansine can be used to treat breast cancer that is positive for the human epidermal growth factor receptor 2 (HER2). Tamoxifen and aromatase inhibitors are examples of hormonal treatments that block estrogen or progesterone receptors and can be used to treat hormone receptor-positive breast cancer.

2. Lung Cancer: To stop certain genetic abnormalities that fuel tumour growth, targeted medicines are used in lung cancer.
   For patients with EGFR mutations who have non-small cell lung cancer (NSCLC), EGFR inhibitors such as gefitinib and osimertinib are beneficial. ALK gene rearrangements are treated with ALK inhibitors, such as crizotinib and alectinib. Additionally, BRAF inhibitors and ROS1 inhibitors are becoming targeted treatments for particular groups of lung cancer patients.

3. Specifically, targeted medicines for colorectal cancer aim to suppress the vascular endothelial growth factor.
   (VEGF) receptor and the epidermal growth factor pathway. The anti-VEGF antibody bevacizumab prevents the growth of blood vessels within the tumour. EGFR inhibitors, like as cetuximab and panitumumab, target the KRAS wild-type coloured EGFR pathway.

4. Melanoma: The BRAF mutation is the target of targeted therapy for melanoma, which affects approximately
   of all melanomas, 50%. Vemurafenib and dabrafenib are examples of BRAF inhibitors that selectively target the mutant BRAF protein and prevent its activity. However, combined treatments including MEK inhibitors, such as trametinib, have demonstrated greater efficacy because resistance can develop.

5. Lymphomas and Leukaemia's: Kinase inhibitors and monoclonal antibodies are examples of targeted therapy for leukaemia's and lymphomas.
   inhibitors. In B-cell lymphomas, rituximab is used in conjunction with chemotherapy, and in chronic lymphocytic leukaemia (CLL), it is utilized as an anti-CD20 antibody.
   Tyrosine kinase inhibitors such as dasatinib, nilotinib, and imatinib target the BCR-ABL fusion gene in individuals with chronic myeloid leukaemia (CML).[17]

5. CLINICAL APPLICATIONS AND THERAPEUTIC IMPACT

A number of cancers have seen a change in the clinical landscape due to molecular targeted therapy:
For mutation-positive non-small cell lung cancer (NSCLC), EGFR and ALK inhibitors are now considered standard of therapy .

• HER2-targeted treatments have significantly increased survival in HER2-

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positive instances of breast cancer .
In cases of metastatic colorectal and other cancers, VEGF and BRAF inhibitors provide treatment alternatives.
Clinical benefits include improved response rates, prolonged progression-free survival, and better tolerability compared to chemotherapy [18,19].

6. MECHANISMS OF DRUG RESISTANCE

Targeted therapy resistance limits long-term efficacy and presents a serious therapeutic concern. Mechanisms include:
• Secondary Mutations: Target gene changes that inhibit drug binding, including the EGFR T790M mutation .
• other route Activation: By upregulating other pathways, including MET amplification, tumours circumvent the blocked route .
• Tumour heterogeneity: Relapse and diverse responses can result from distinct mutations present in various cancer cell groups within the tumour.
Effective drug concentration may be lowered by pharmacokinetic factors, such as drug efflux and metabolism.
For the development of combination treatments and next-generation inhibitors, an understanding of these processes is essential.[20,21]

7. EMERGING TECHNOLOGIES AND FUTURE PERSPECTIVES

7.1 CRISPR-Cas9 and Genome Editing

CRISPR technology may be used to directly target genes that cause cancer or to fix mutations that result in medication resistance. Its accuracy raises the possibility of customized genetic therapies.

7.2 Biomarker Discovery and Multi-Omics Integration

The combination of artificial intelligence and genomes, transcriptomic, proteomics, and metabolomics speeds up the process of finding new targets and predictive biomarkers for treatment selection.

7.3 Combination Therapies

Molecular targeted therapies are used in conjunction with immunotherapy or chemotherapy in an effort to improve clinical outcomes by overcoming resistance and taking advantage of synergistic processes.

7.4 Accessibility and Cost Considerations

Global impact depends on initiatives to lower therapeutic costs and democratize modern diagnostics [22,23].

8.DISCUSSION

Through the use of precision medicine techniques that take advantage of the distinct molecular aberrations causing tumour development and progression, molecular targeted therapy has completely changed the way that cancer is treated, replacing non-specific cytotoxic drugs. These treatments have outperformed traditional chemotherapy in terms of specificity, efficacy, and toxicity by focusing on important molecules such receptor tyrosine kinases, intracellular signalling proteins, and apoptotic regulators
Clinically, molecular targeted drugs have revolutionized the treatment of a number of malignancies, such as colorectal, breast, and non-small cell lung cancer, providing notable advantages in terms of response rates, progression-free survival, and patient quality of life. Numerous obstacles,

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however, limit these treatments' broad clinical effectiveness. A significant barrier that restricts long-term efficacy is drug resistance, which can be caused by secondary mutations, alternative pathway activation, and tumour heterogeneity. Moreover, these treatments are expensive and require sophisticated genetic profiling, which limits their accessibility, especially in low-resource environments.

Through the development of more accurate, individualized, and flexible treatment plans, emerging technologies like CRISPR-Cas9 genome editing, multi-omics integration, and AI-driven biomarker discovery are well-positioned to meet these issues. Combination treatments, which combine immunotherapies or chemotherapy with molecular targeted medicines, also present intriguing ways to overcome resistance and enhance patient outcomes.

To summarize, one significant development in precision oncology is molecular targeted therapy. For the purpose of overcoming current restrictions, increasing access, and improving treatment plans, further research and technical advancement are necessary. These initiatives represent a promising future for the treatment of this complicated illness and show enormous promise to further increase survival and quality of life for cancer patients globally.

9.CONCLUSION

A significant development in precision oncology, molecular targeted therapy provides extremely tailored and efficient treatment choices that enhance patient outcomes and quality of life. As technologies such as genome editing, multi-omics, and AI-driven biomarker identification continue to advance, this strategy keeps changing. With the potential to further customize and enhance cancer treatment, these advancements bode well for oncology and patient care worldwide.

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