American Journal of Pharmacy and Pharmacology

australiansciencejournals.com/pharmacy

E-ISSN: 2689-0240

VOL 05 ISSUE 03 2024

Mechanisms of Action of Anticancer Agents: A Comprehensive Review

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Abstract: *Anticancer agents exert their therapeutic effects through various mechanisms targeting different cellular pathways essential for cancer cell survival and proliferation. This review categorizes and describes the primary mechanisms of action of conventional chemotherapeutics, targeted therapies, hormone therapies, immunotherapies, and emerging treatment modalities. An in-depth understanding of these mechanisms aids in rational drug design, predicting drug resistance, and optimizing treatment strategies. The article also addresses challenges and future prospects in anticancer drug development.*

Keywords: *Anticancer Agents, Chemotherapy, Targeted Therapy, Immunotherapy, Cancer Mechanisms, Drug Resistance.*

INTRODUCTION

Cancer treatment has evolved significantly with the development of diverse classes of anticancer agents. These agents target specific aspects of tumor biology, including DNA replication, cell division, signaling pathways, and immune evasion. Understanding the mechanisms by which these drugs act is critical for effective therapy selection, combination strategies, and overcoming resistance. This review comprehensively outlines the mechanisms of action of major anticancer drug classes and highlights the ongoing advancements in the field.

Conventional Chemotherapeutic Agents

1. Alkylating Agents

These compounds form covalent bonds with DNA, leading to cross-linking and strand breaks. This disrupts DNA replication and transcription, causing apoptosis. Examples: Cyclophosphamide, Melphalan.

2. Antimetabolites

Antimetabolites mimic natural substrates in DNA and RNA synthesis, inhibiting enzymes involved in nucleotide synthesis. Examples: Methotrexate, 5-Fluorouracil.

3.Topoisomerase Inhibitors

These agents interfere with topoisomerase enzymes, preventing DNA unwinding necessary for replication and transcription. Examples: Doxorubicin, Etoposide.

4. Mitotic Inhibitors

They disrupt microtubule function, inhibiting mitosis and inducing cell cycle arrest. Examples: Paclitaxel, Vincristine.

Targeted Therapy

1. Tyrosine Kinase Inhibitors (TKIs)

TKIs block aberrant signaling pathways driven by mutated or overexpressed kinases. Examples: Imatinib (BCR-ABL), Erlotinib (EGFR).

2. Monoclonal Antibodies

These antibodies bind to specific antigens on cancer cells, promoting immune-mediated destruction or blocking growth signals. Examples: Trastuzumab (HER2), Rituximab (CD20).

3. PARP Inhibitors

Inhibit DNA repair enzymes, particularly in BRCA-mutated cancers, leading to synthetic lethality. Examples: Olaparib, Niraparib.

Hormonal Therapy

Hormone-dependent cancers such as breast and prostate cancer can be treated by disrupting hormonal signaling. Agents include selective estrogen receptor modulators (SERMs), aromatase inhibitors, and androgen deprivation therapies. Examples: Tamoxifen, Letrozole, Leuprolide.

Immunotherapy

1. Immune Checkpoint Inhibitors

These drugs block proteins that inhibit T-cell activation, enhancing the immune response against tumors. Examples: Nivolumab (PD-1), Ipilimumab (CTLA-4).

2. CAR-T Cell Therapy

Engineered T cells expressing chimeric antigen receptors (CARs) target specific tumor antigens, leading to direct cytotoxicity.

3. Cancer Vaccines and Cytokine Therapies

Vaccines prime the immune system against tumor-specific antigens, while cytokines such as IL-2 and IFN-α boost immune activity.

Emerging Therapies and Mechanistic Insights

1. Epigenetic Modifiers

Drugs that alter gene expression without changing DNA sequence, such as HDAC and DNMT inhibitors. Examples: Vorinostat, Azacitidine.

2. Proteasome Inhibitors

They prevent degradation of pro-apoptotic proteins, leading to accumulation of damaged proteins. Example: Bortezomib.

3. Oncolytic Viruses

Viruses that selectively infect and kill tumor cells while stimulating anti-tumor immunity. Example: Talimogene laherparepvec (T-VEC).

Summary

The mechanisms of action of anticancer agents span a wide range of biological targets and cellular processes. From DNA damage to immune activation, these therapies exploit vulnerabilities in cancer cells. A comprehensive understanding of these mechanisms informs clinical decision-making, supports drug development, and helps to overcome treatment resistance. Ongoing research is expected to yield even more precise and effective anticancer strategies.

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