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PHARMACOLOGICAL ANTICANCER DRUGS: CURRENT ADVANCES AND FUTURE PERSPECTIVES

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ABSTRACT

Cancer treatment has changed considerably over the years, moving from conventional chemotherapy to more selective and mechanism-based therapies. This review focuses on the pharmacological mechanisms of major anticancer drugs and the recent developments that have contributed to the progress of modern cancer treatment. Conventional drugs, including DNA-interacting agents, antimetabolites, and anti-tubulin agents, work by damaging genetic material, interfering with DNA synthesis, or preventing normal cell division. Hormonal therapies and targeted drugs act on specific receptors, enzymes, and signaling pathways that are involved in the growth and survival of cancer cells. Recent developments in monoclonal antibodies, antibody-drug conjugates, immune checkpoint inhibitors, and CAR T-cell therapy have further broadened the available treatment options. The review also discusses newer approaches such as nanomedicine, personalized oncology, genomic profiling, and artificial intelligence, which may help improve drug delivery and make treatment more precise.

However, drug resistance, treatment-related toxicity, and limited access to advanced therapies continue to be important challenges. Overall, a clear understanding of how anticancer drugs work is essential for developing safer, more effective, and patient-specific approaches to cancer treatment.

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1. INTRODUCTION

Cancer treatment has undergone a major transformation over the years, particularly in the field of pharmacological therapy. Earlier approaches mainly relied on conventional chemotherapy, which was designed to destroy rapidly dividing cells. Although this method could effectively reduce tumor growth, it often affected normal cells as well, resulting in significant side effects and toxicity.[1] Along with surgery and radiation therapy,

chemotherapy became an important component of cancer treatment and was widely recognized as the “third pillar” of cancer therapy.[2]

One of the major limitations of traditional chemotherapy is its lack of selectivity. Since the drugs can affect both cancerous and healthy cells, patients may experience various unwanted effects during treatment.[3] In addition, cancer cells can gradually develop resistance to multiple anticancer drugs, making treatment more difficult and reducing therapeutic effectiveness.[4]

With advances in cancer biology, researchers have gained a much clearer understanding of the mechanisms involved in tumor development. Features such as uncontrolled proliferation, resistance to normal growth-regulating signals, and the ability of cancer cells to escape immune surveillance have provided important insights into cancer progression.[5] This knowledge has supported the development of targeted therapies and immunotherapies, which are designed to interfere with specific molecular pathways or strengthen the body's immune response against cancer cells.[6,7]

Looking ahead, emerging technologies such as nanotechnology and personalized medicine are expected to further improve cancer treatment. Nanotechnology can help enhance drug delivery and improve the bioavailability of anticancer agents, while personalized medicine allows treatment strategies to be selected according to the genetic and molecular characteristics of individual patients[8,9] Together, these developments are gradually shifting cancer therapy from a general approach toward more precise, effective, and patient-specific treatment strategies.

2. Molecular Mechanisms of Anticancer Drugs

Anticancer drugs are classified according to the way they act on cancer cells. These drugs may target specific stages of the cell cycle or interfere with important metabolic pathways that cancer cells depend on for their survival and continuous growth. By acting on these essential processes, anticancer drugs can slow down or stop the proliferation of tumor cells.[10]

Cytotoxic Agents and DNA Interaction

DNA-interacting agents are one of the oldest and most widely used groups of chemotherapy drugs. This group includes **alkylating agents**, which damage DNA by forming strong

chemical bonds with DNA bases. As a result, important processes such as DNA replication and transcription are disrupted, preventing cancer cells from growing and dividing normally.[8]

Another important group is the **anthracyclines**, which include drugs such as **doxorubicin and epirubicin**. These drugs work by inserting themselves between DNA base pairs and interfering with the activity of **topoisomerase II**, an enzyme required for proper DNA replication and repair. This can cause severe and often irreversible double-stranded DNA breaks, leading to significant damage to the cancer cell's genetic material and chromatin structure.[11,5]

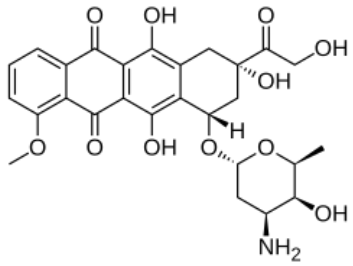
Other DNA-damaging drugs, such as **mitomycin C**, also play an important role in cancer treatment. These agents can be particularly useful in certain treatment strategies aimed at managing cancers that have developed **multidrug resistance**, where cancer cells become less responsive to several commonly used anticancer drugs.[12]

Drugs definition

1. Doxorubicin

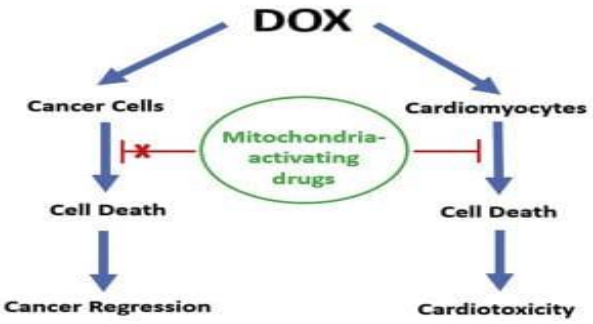
Doxorubicin is an anthracyclinecytotoxic anticancer drugs that inhibit cell growth by damaging DNA.

Structure



Molecular formulas:C27H29NO11
Current use: Mainly used as a **breast cancer,** lymphomas, Leukemias, sarcomas,ovarian cancer.
dosage form: Intravenous (IV) injection solution, liposomal doxorubicin injection

MOA



Doxorubicin

Antimetabolites and Nucleic Acid Synthesis

Antimetabolites are a group of anticancer drugs that resemble naturally occurring substances such as vitamins, amino acids, and nucleotides. Because of this structural similarity, they can interfere with the normal metabolic processes required for cancer cell growth and division, particularly by disrupting the synthesis of DNA and other nucleic acids.[8,5]

For example, **methotrexate** and **fluorouracil (5-FU)** interfere with DNA synthesis and thereby prevent cancer cells from multiplying effectively[10.] **5-FU**, along with its prodrug **capecitabine**, mainly acts by inhibiting the enzyme **thymidylate synthase** and by becoming incorporated into abnormal DNA and RNA components. These effects interfere with normal genetic processes and can ultimately lead to cancer cell death.[11]

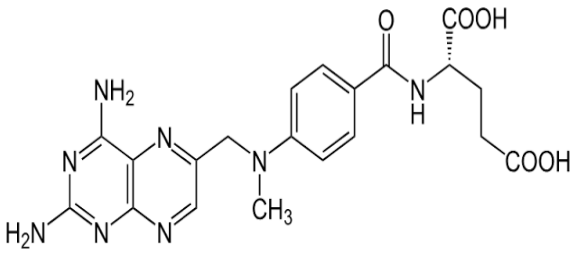
Antimetabolites are generally considered **cell-cycle-specific drugs**, with their strongest effects occurring during the **S-phase**, which is the stage in which DNA replication takes place.[14]

Drugs

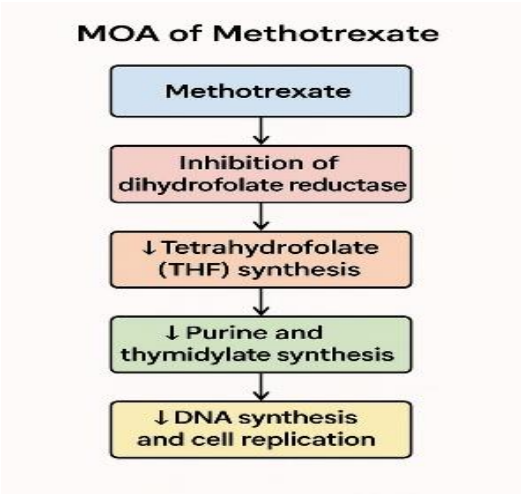
1. Methotrexate

Methotrexate is an **antimetabolite and antifolate anticancer drug** that works by interfering with folic acid metabolism. By blocking this pathway, it reduces the availability of essential components needed for DNA synthesis, thereby preventing cancer cells from growing and multiplying effectively.

Structure



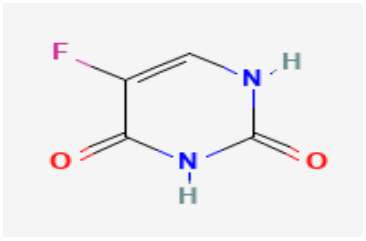
Chemical formula:C20H20N8O5
Current use: acute lymphoblastic leukemia, breast cancer, osteosarcoma, choriocarcinoma, some other cancers
Dosage form: tablets, injection (IM,IV) other depending uopon the formulation



2. Flurouracil(5-FU)

5-Fluorouracil (5-FU) is an **antimetabolite anticancer drug** that closely resembles the natural pyrimidine bases involved in DNA synthesis. By interfering with the normal DNA synthesis process, 5-FU prevents cancer cells from growing and multiplying, ultimately contributing to their death.

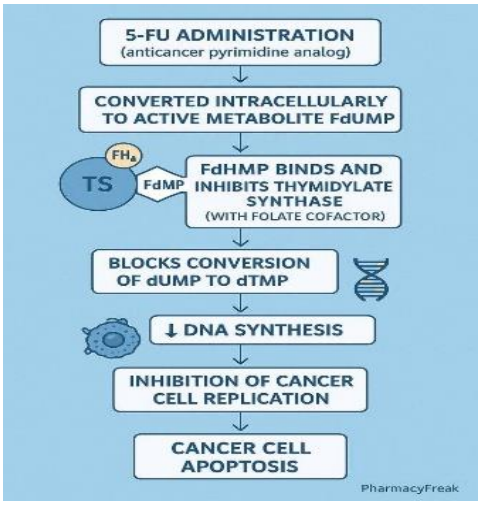
Structure



Chemical formula:C4H3FN2O2.

Current use: colorectal cancer, breast cancer, gastric cancer, pancreatic cancer, head and neck cancer Dosage form: injection (IV) infusion, topical cream for certain skin cancers.

Flurouracil (5FU)



Anti-tubulin Agents and Mitotic Arrest

Anti-tubulin drugs are an important group of anticancer agents that work by interfering with **microtubules**, which are essential for proper cell division. By disrupting the microtubule network, these drugs prevent cancer cells from completing mitosis and therefore stop their growth and multiplication.

Vinca alkaloids, such as **vincristine and vinorelbine**, act by destabilizing the α/β-tubulin structures that form microtubules. This causes the microtubules to break down and prevents the cell from progressing normally through mitosis, eventually leading to **mitotic arrest**.[11,5,10]

In contrast, **taxanes**, such as **paclitaxel**, work in the opposite way. They stabilize microtubules and prevent them from being properly brokendown during cell division. As a result, the cancer cell becomes unable to complete mitosis and is arrested mainly at the **G2/M phase** of the cell cycle.[8,5]

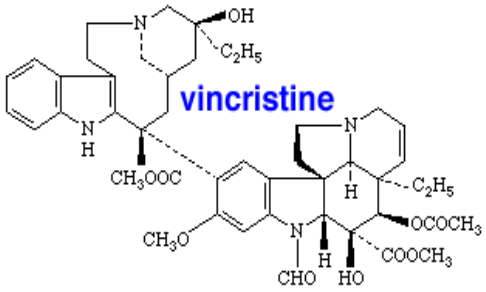
Drugs

Vinka alkaloids

1. vincristine

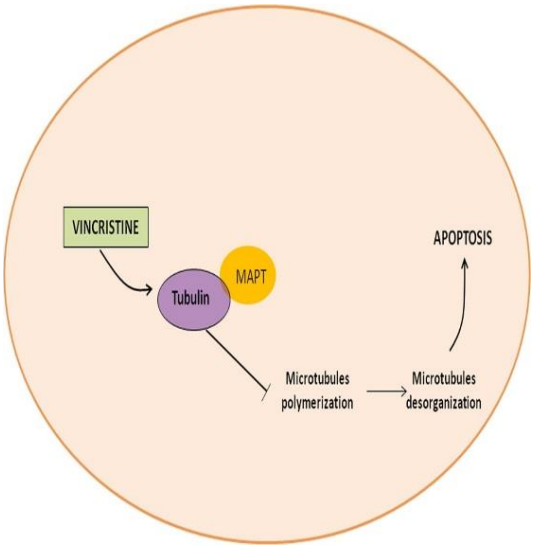
Vincristine is a **vinca alkaloid anticancer drug** that interferes with the formation of microtubules, which are essential for normal cell division. By disrupting microtubule formation, vincristine prevents cancer cells from completing mitosis, leading to **mitotic arrest** and ultimately slowing down their growth and proliferation.

Structure



Chemical formula: C46H56N4O10
Current use: acute lymphoblastic leukemia, Hodgkin lymphoma, non Hodgkin lymphoma, euroblastoma, wilms tumor, chemotherapy regimens
Dosage form: intravenous (IV) injection solution.

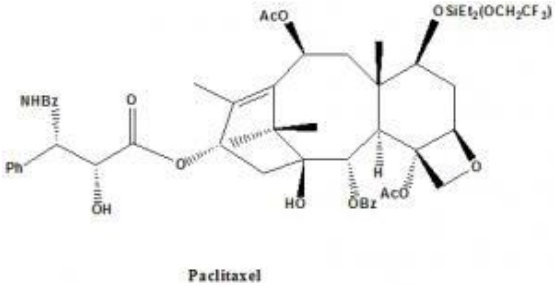
MOA



2. Taxens: paclitaxel

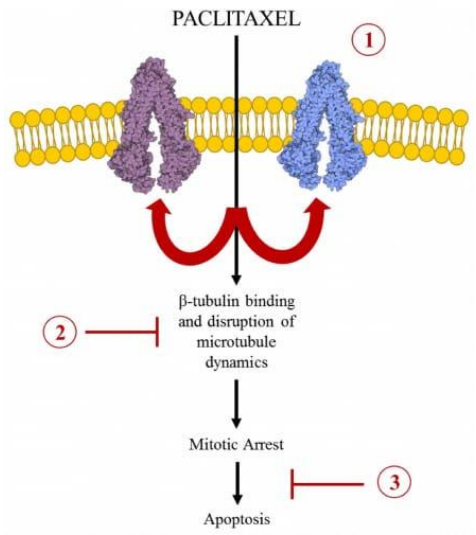
Paclitaxel is a **taxane-based anticancer drug** that works by disrupting the normal function of microtubules, which are essential for cell division. By interfering with this process, paclitaxel prevents cancer cells from completing cell division, thereby slowing their growth and proliferation.

Structure



Chemical formula:C47H51NO14
Current use: breast cancer, ovarian cancer, non-cell lung cancer, pancreatic cancer, kaposi's sarcoma
Dosage form: intravenous (IV) infusion
Conventional paclitaxel injection
Albumin-bound paclitaxel is another formulation.

MOA



Hormonal and Signaling Pathway Inhibitors

Hormonal therapies are an important treatment option for cancers that depend on hormones for their growth. Drugs such as **tamoxifen** and aromatase inhibitors like **anastrozole** work by reducing the effects or availability of estrogen. Tamoxifen mainly prevents estrogen from binding to its receptor, while aromatase inhibitors reduce estrogen production in the body. By limiting estrogen signaling, these drugs can slow down or prevent the growth of hormone-sensitive cancer cells.[11,8]

In addition to hormonal therapies, advances in modern pharmacology have led to the development of drugs that specifically target important molecular signaling pathways involved in cancer development and survival. Some of the major examples include.

Proteasome Inhibitors (PIs): These drugs interfere with the ubiquitin–proteasome system, which is responsible for breaking down unwanted proteins within cells. By disrupting this process, proteasome inhibitors can interfere with cell-cycle regulation and promote cancer cell death.[15] **mTOR Inhibitors:** These agents block the **mammalian target of rapamycin (mTOR)** pathway, an important signaling pathway that regulates cell growth, metabolism, and survival. Since this pathway is often abnormally activated in different types of cancer, its inhibition can help limit tumor growth.[15] **BCL-2 Inhibitors:** Drugs such as **venetoclax** act as BH3 mimetics and specifically target the BCL-2 protein. By blocking the protective effect of BCL-2, these drugs promote **apoptosis**, or programmed cell death, in cancer cells.[15] **Notch and Wnt Pathway Inhibitors:** These are newer and emerging therapeutic approaches that target the Notch and Wnt signaling pathways. Since these pathways play important roles

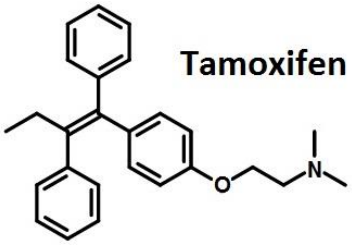
in tumor growth, cell survival, and cancer stem-cell maintenance, inhibiting them may help control tumor progression and reduce the stem-like properties of cancer cells.[15,16]

Drugs

1. Tamoxifen

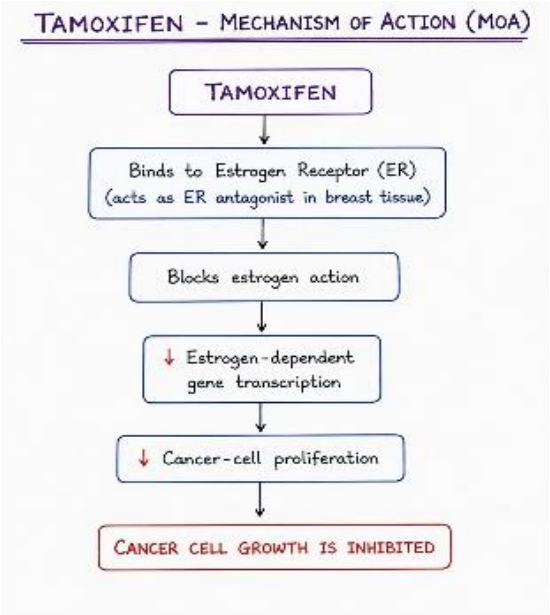
Tamoxifen is a **Selective Estrogen Receptor Modulator (SERM)** that is widely used to treat and help prevent **estrogen receptor-positive breast cancer**. It works by blocking the action of estrogen in breast tissue, thereby reducing the stimulation of cancer cells and helping to slow their growth.

Structure



Current use: estrogen positive action in breast cancer
Prevention the cancer in high risk patients
Dosage formula: oral tablets
chemical formula:C26H29NO.

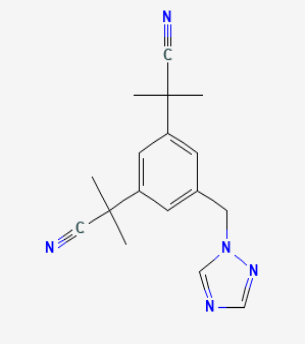
MOA

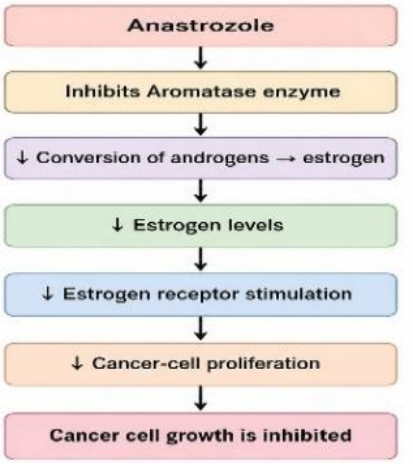


2. Anastrozole

Anastrozole is a **non-steroidal aromatase inhibitor** commonly used to treat **hormone receptor-positive breast cancer**, especially in postmenopausal women. It works by reducing the production of estrogen in the body, which helps limit the growth and progression of estrogen-dependent cancer cells.

Structure





Chemical formula: C17H19N5
Current use: hormone receptor positive breast cancer
Postmenopausal women
Dosage form: oral tablets

3. Current Therapeutic Advances

The growing focus on **precision medicine** has led to the development of more targeted cancer therapies that specifically recognize **tumor-associated antigens** or abnormal signaling proteins involved in cancer growth. This approach helps target cancer cells more selectively while potentially reducing damage to healthy tissues.

Targeted Therapy

Targeted therapy is a more precise approach to cancer treatment that uses **small-molecule inhibitors and monoclonal antibodies** to interfere with specific molecular pathways that are important for tumor growth and survival.[17] Unlike conventional chemotherapy, these treatments are designed to act on particular targets that are more closely associated with cancer cells.

Tyrosine Kinase Inhibitors (TKIs): These drugs block the activity of tyrosine kinases, which are enzymes involved in cell growth and signaling. By interfering with their activity, TKIs can reduce phosphorylation and disrupt downstream signaling pathways that promote cancer cell growth.[11] For example, **imatinib** specifically targets the **BCR-ABL** protein in chronic myeloid leukemia. Similarly, **gefitinib** and **osimertinib** target the **Epidermal Growth Factor Receptor (EGFR)**, particularly in certain types of lung cancer.[10] In soft tissue sarcomas, multikinase inhibitors such as **pazopanib** and **sunitinib** can target receptors including **VEGFR and PDGFR**, thereby reducing the formation of new blood vessels and limiting tumor growth.[15]

Monoclonal Antibodies: These are specially designed antibodies that recognize and bind to specific proteins or antigens associated with cancer cells. For instance, **trastuzumab** targets the **HER2** protein in HER2-positive breast cancer, while **bevacizumab** targets **VEGF**, helping to block the formation of new blood vessels that tumors need for continued growth.[18]

Antibody-Drug Conjugates (ADCs)

Antibody–Drug Conjugates (ADCs) are an advanced form of targeted cancer therapy that combines the precise targeting ability of monoclonal antibodies with the powerful cancer-killing effects of cytotoxic drugs.[19] In this approach, the antibody acts like a **delivery vehicle**, recognizing specific proteins on cancer cells and carrying the cytotoxic drug directly to them.

Once the ADC binds to its target and enters the cancer cell, it can release potent drugs such as **topoisomerase I inhibitors** (e.g., deruxtecan) or **microtubule inhibitors** (e.g., DM1). These drugs then interfere with essential cellular processes, ultimately leading to cancer cell death.[20] Because the cytotoxic drug is delivered more selectively to cancer cells, ADCs can improve treatment precision while potentially reducing the exposure of healthy tissues to the drug and thereby lowering systemic toxicity.[13] Currently, ADCs are showing promising results in the treatment of **non-small cell lung cancer (NSCLC)** by targeting proteins such as

HER2, HER3, and TROP-2[19]

Immunotherapy: Harnessing

- **Immunotherapy** is a modern approach to cancer treatment that uses the body's own immune system to recognize and destroy cancer cells.[17] Instead of directly killing tumor cells with conventional drugs, immunotherapy mainly works by strengthening or redirecting the immune response against cancer.
- **Immune Checkpoint Inhibitors (ICIs):** These drugs, such as **pembrolizumab** and **nivolumab**, target immune checkpoint proteins including **PD-1, PD-L1, and CTLA-4**. Normally, these proteins act as "brakes" on the immune system and prevent excessive immune activity. By blocking these checkpoints, ICIs help restore T-cell activity and allow the immune system to recognize and attack tumor cells more effectively.[18,13]
- **CAR T-Cell Therapy:** This treatment involves collecting a patient's own T-cells and genetically modifying them to produce **chimeric antigen receptors (CARs)** that can

recognize specific antigens present on cancer cells. The modified T-cells are then returned to the patient, where they can identify and selectively destroy the targeted malignant cells.[18,7]

- **Cancer Vaccines: Cancer vaccines** are a promising and developing area of immunotherapy that aim to train the immune system to recognize specific **tumor-associated antigens** and respond more effectively to cancer cells. By stimulating a stronger and longer-lasting immune response, these vaccines may help the body identify and eliminate tumor cells more efficiently.
- Another emerging approach is **microbiome-based therapy**, which focuses on modifying or influencing the gut microbiome to support the immune system. A healthier or appropriately altered microbiome may enhance the body's ability to mount and maintain an effective **anti-tumor immune response**, making it a potential strategy for improving cancer treatment outcomes.[7,20]

4. Future Perspectives in Oncology

Future developments in cancer therapy are increasingly focused on overcoming the physical and biological challenges that limit effective drug delivery. At the same time, the goal is to make treatment more **precise and personalized**, so that therapies can be selected according to the individual characteristics of each patient and their tumor.

Nanomedicine and Innovative Drug Delivery Systems

- **Nanotechnology** is becoming increasingly important in cancer treatment because it can improve the **bioavailability, stability, and targeted delivery** of anticancer drugs.[17] Nanocarriers such as **liposomes, nanoparticles** made from materials like porous silicon and silk fibroin, and **micelles** can protect drugs from early degradation and help deliver them more effectively to the tumor site.[21,22]
- **Stimuli-Responsive Systems:** These advanced nanoparticles are designed to release their drug payload only when they encounter specific conditions within the tumor microenvironment. For example, changes in **pH** or increased **oxidative stress** can trigger drug release directly at the tumor site, potentially improving treatment effectiveness while reducing unwanted effects on healthy tissues.[3,12]

- **Lipid-Based Nanoparticles (LBNPs):** LBNPs offer biocompatible and relatively non-toxic platforms for delivering therapeutic drugs and developing cancer vaccines. Their

ability to encapsulate and transport different types of therapeutic molecules makes them promising tools in modern cancer treatment.[23]

- **Nanobots and Engineered Cell Therapies:** Looking further ahead, researchers are exploring the possibility of using **nanobots** for highly precise drug delivery, as well as **genetically or otherwise engineered cells** that can perform specific therapeutic functions. These emerging technologies may provide more accurate and individualized approaches to cancer treatment in the future.[11]
- **Personalized medicine** is an important approach in modern cancer treatment that uses information from a patient's **genomic, transcriptomic, and proteomic profiles** to select therapies that best match their individual biological and genetic characteristics[21,8] Instead of using the same treatment for every patient, this approach helps clinicians choose therapies based on the specific molecular features of the patient's tumor.
- **Biomarkers** also play an increasingly important role in treatment selection. Factors such as **PD-L1 expression** and **tumor mutational burden (TMB)** can provide useful information about how a patient may respond to certain targeted therapies or immunotherapies. This helps doctors make more informed decisions when selecting the most appropriate treatment.[9]

The integration of **artificial intelligence (AI)** is further advancing personalized cancer therapy. AI can support the discovery of new drug candidates, identify potential therapeutic targets, and help match specific drug payloads with suitable targets during the development of **antibody–drug conjugates (ADCs)**. These advances may improve the precision, efficiency, and effectiveness of future cancer treatments.[17,19]

5. Limitations and Challenges

Despite the major advances made in cancer treatment, **drug resistance and treatment-related safety concerns** continue to remain important challenges in oncology.

Pharmacological Mechanisms of Resistance

Resistance to anticancer treatment is a complex problem that can develop through several different mechanisms. Cancer cells may become resistant by increasing the activity of **drug efflux pumps**, changing the molecular targets of drugs, or improving their ability to repair damaged DNA.[24] These changes can allow cancer cells to survive even when exposed to anticancer drugs. The **tumor microenvironment**, **epithelial–mesenchymal transition**

(EMT), and the presence of **cancer stem cells** can further contribute to treatment resistance and failure.[16,24] For example, **BMI-1** and the **Wnt/β-catenin signaling pathway** have been associated with cancer stemness and the development of drug resistance.[16] A better understanding of these resistance mechanisms is important for developing **next-generation anticancer drugs** and designing effective **combination therapies** that can overcome resistance and improve treatment outcomes.[17]

Toxicity and Safety Management

Traditional chemotherapy often has limited selectivity, meaning that it can affect healthy cells along with cancer cells. This lack of specificity can result in significant side effects and may limit the overall clinical use of these treatments.[3] Although **targeted therapies and immunotherapies** are generally more selective and may have a better safety profile, they can still cause treatment-related side effects. In addition, the high cost of these advanced therapies can make them less accessible to many patients.[17,25] Therefore, improving the safety and effectiveness of cancer treatment remains an important goal. The use of **advanced drug-delivery systems** and **personalized dosing strategies** may help deliver medicines more precisely and reduce unwanted toxicity while maintaining therapeutic effectiveness.[19,26]

6. CONCLUSION

Cancer treatment has changed greatly over the years, moving from conventional chemotherapy toward more targeted and personalized approaches. Traditional anticancer drugs still have an important role, while targeted therapies, hormonal treatments, antibody-drug conjugates, and immunotherapies have provided better ways to control cancer-cell growth and survival. Newer approaches such as nanomedicine, genomic profiling, and artificial intelligence may further improve the accuracy and effectiveness of cancer treatment. However, drug resistance, treatment-related toxicity, high costs, and limited accessibility remain important challenges. Therefore, continued research into cancer biology and the mechanisms of anticancer drugs is needed to develop safer, more effective, and patient-specific therapies and to improve overall treatment outcomes.

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