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An review on recent advances in Anti-Cancer Drugs

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ABSTRACT

Cancer is one of the leading causes of death worldwide. With the increase in life expectancy, the number of cancer cases has reached unprecedented levels. In this scenario, the pharmaceutical industry has made significant investments in this therapeutic area. Despite these efforts, cancer drug research remains a remarkably challenging field, and therapeutic innovations have not yet achieved expected clinical results. However, the physiopathology of the disease is now better understood, and the discovery of novel molecular targets has refreshed the expectations of developing improved treatments. Several noteworthy advances have been made, among which the development of targeted therapies is the most significant. Monoclonal antibodies and antibody-small molecule conjugates have emerged as a worthwhile approach to improve drug selectivity and reduce adverse effects, which are the main challenges in cancer drug discovery. This review will examine the current panorama of drug research and development (R&D) with emphasis on some of the major advances brought to clinical trials and to the market in the past five years. Breakthrough discoveries will be highlighted al ong with the medicinal chemistry strategies used throughout the discovery process. In addition, this review will provide perspectives and updates on the discovery of novel molecular targets as well as drugs with innovative mechanisms of action.

Keywords: Antimicrobial peptides; Cancer therapies; Immunotherapy; Targeted drug delivery; Carcinoma; Cyclophosphamide.

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INTRODUCTION

Cancer is a large group of diseases that can start in almost any organ or tissue of the body when abnormal cells grow uncontrollably, go beyond their usual boundaries to invade adjoining parts of the body and/or spread to other organs. The latter process is called metastasizing and is a major cause of death from cancer.

Cancer is the second leading cause of death globally, accounting for an estimated 9.6 million deaths, or 1 in 6 deaths, in 2018. Lung, prostate, colorectal, stomach and liver cancer are the most common types of cancer in men, while breast, colorectal, lung, cervical and thyroid cancer are the most common among women.

The cancer burden continues to grow globally, exerting tremendous physical, emotional and financial strain on individuals, families, communities and health systems. Many health systems in low- and middle-income countries are least prepared to manage this burden, and large numbers of cancer patients globally do not have access to timely quality diagnosis and treatment.

Cancer is a complex and heterogeneous group of diseases characterized by uncontrolled cell proliferation, invasion into surrounding tissues, and the ability to metastasize to distant organs. It arises

due to the accumulation of genetic and epigenetic alterations that disrupt normal cellular regulatory mechanisms, including cell cycle control, apoptosis, and DNA repair pathways [6]. These abnormalities lead to the transformation of normal cells into malignant phenotypes capable of sustained growth and survival.

Globally, cancer represents one of the leading causes of mortality, accounting for millions of deaths annually. The burden of cancer continues to rise due to increased life expectancy, urbanization, environmental exposure to carcinogens, and lifestyle-related risk factors such as smoking, poor diet, and physical inactivity [17]. In addition to its health impact, cancer imposes a substantial economic and social burden on healthcare systems, particularly in low- and middle-income countries where access to early diagnosis and advanced treatment remains limited.

Conventional cancer treatment strategies, including surgery, chemotherapy, and radiotherapy, have significantly improved patient survival. However, these approaches are often associated with limitations such as non-specific targeting, systemic toxicity, and the development of drug resistance [7]. Chemotherapeutic agents, while effective in killing rapidly dividing cells, also damage normal proliferating cells, leading to adverse effects such as myelosuppression, gastrointestinal toxicity, and immunosuppression.

Recent advances in molecular biology and genomics have transformed the understanding of cancer pathophysiology. The identification of oncogenes, tumor suppressor genes, and signaling pathways involved in tumor progression has paved the way for the development of targeted therapies and personalized medicine [8]. These modern approaches aim to selectively target cancer cells while sparing normal tissues, thereby improving therapeutic outcomes and reducing toxicity. Furthermore, the emergence of immunotherapy and nanotechnology-based drug delivery systems has opened new avenues for more effective and precise cancer treatment [10,12].

Types of Cancer

Cancer can be classified based on tissue origin and cellular characteristics, reflecting its biological diversity and clinical behavior. Understanding these classifications is essential for diagnosis, treatment selection, and prognosis.

Carcinomas are the most prevalent form of cancer and originate from epithelial cells that line the surfaces of organs and tissues. These cancers commonly affect organs such as the breast, lung, prostate, and colon. Due to their epithelial origin, carcinomas often exhibit high metastatic potential and are responsible for the majority of cancer-related deaths worldwide [17].

Sarcomas arise from connective tissues, including bone, cartilage, fat, and muscle. Although relatively rare compared to carcinomas, sarcomas are often aggressive and require specialized therapeutic approaches. Examples include osteosarcoma and liposarcoma.

Leukemias are malignancies of the blood-forming tissues, particularly the bone marrow, leading to the excessive production of abnormal white blood cells. These cancers are systemic in nature and interfere with normal hematopoiesis. Common types include acute lymphoblastic leukemia (ALL) and chronic myeloid leukemia (CML).

Lymphomas are cancers of the lymphatic system and are broadly categorized into Hodgkin's lymphoma and non-Hodgkin's lymphoma. These malignancies affect lymphocytes and may involve lymph nodes, spleen, and other lymphoid organs.

Myeloma originates from plasma cells and is characterized by abnormal immunoglobulin production and bone destruction. Melanoma develops from melanocytes and is known for its aggressive nature and high metastatic potential.

Germ cell tumors arise from reproductive cells, while neuroendocrine tumors originate from hormone-producing cells and may occur in various organs.

Despite their diversity, all cancers share fundamental biological characteristics such as uncontrolled proliferation, resistance to apoptosis, angiogenesis, invasion, and metastasis, collectively described as the hallmarks of cancer [6,15].

Key Characteristics of Cancer

Cancer is defined by a set of fundamental biological properties that distinguish malignant cells from normal cells. These characteristics, often referred to as the hallmarks of cancer, underpin tumor initiation, progression, and metastasis.

One of the primary features of cancer is uncontrolled cellular proliferation. Under normal physiological conditions, cell growth and division are tightly regulated by signaling pathways that maintain tissue homeostasis. However, cancer cells acquire the ability to proliferate continuously due to dysregulation of growth signals and loss of cell cycle control mechanisms. This results in excessive cell division even in the absence of external growth stimuli.

Another critical characteristic is invasion, where cancer cells penetrate surrounding tissues by breaching the basement membrane and extracellular matrix. This invasive capability is facilitated by the secretion of proteolytic enzymes such as matrix metalloproteinases (MMPs), which degrade structural barriers and allow tumor cells to infiltrate adjacent normal tissues.

Table 1: Types of Cancer

S. No.	*Cancer Type*	*Origin*	*Key Characteristics*	*Examples*
1	**Carcinoma**	Epithelial cells	Most common type of cancer	Breast cancer, Lung cancer, Colon cancer, Prostate cancer
2	**Sarcoma**	Connective tissues	Arises from bone, muscle, fat, cartilage	Osteosarcoma (bone), Muscle cancer, Liposarcoma (fat tissue)
3	**Leukemia**	Blood-forming tissues (bone marrow)	Abnormal proliferation of white blood cells	Acute lymphoblastic leukemia (ALL), Chronic myeloid leukemia (CML)
4	**Lymphoma**	Lymphatic system	Affects lymphocytes and lymph nodes	Hodgkin's lymphoma, Non-Hodgkin's lymphoma
5	**Myeloma**	Plasma cells	Produces abnormal immunoglobulins; affects bone marrow	Multiple myeloma
6	**Melanoma**	Melanocytes (pigment-producing cells)	Highly aggressive skin cancer	Skin melanoma
7	**Germ Cell Tumors**	Reproductive cells	Typically occur in gonads (testes/ovaries)	Testicular cancer, Ovarian cancer
8	**Neuroendocrine Tumors**	Neuroendocrine cells	Hormone-secreting tumors affecting various organs	Carcinoid tumors, Pancreatic neuroendocrine tumors

Closely associated with invasion is metastasis, the process by which cancer cells disseminate from the primary tumor site to distant organs through the bloodstream or lymphatic system. Metastasis involves a complex multistep cascade including detachment from the primary tumor, intravasation, survival in circulation, extravasation, and colonization at secondary sites. It is the leading cause of cancer-related mortality.

Cancer also has a strong genetic basis, arising from mutations in critical genes such as oncogenes, tumor suppressor genes, and DNA repair genes. These genetic alterations lead to abnormal cellular behaviour, including resistance to apoptosis, sustained proliferative signaling, and genomic instability. Both inherited (germline) and acquired (somatic) mutations contribute to cancer development.

Finally, it is important to distinguish between benign and malignant tumors. Benign tumors are non-cancerous growths that remain localized and do not invade surrounding tissues or metastasize. In contrast, malignant tumors exhibit aggressive behavior, including invasion and metastasis, and have the potential to be life-threatening.

Uncontrolled Growth: Cancer cells multiply excessively when they should die or stop.

- **Invasion:** They break through normal boundaries to invade adjacent tissues.
- **Metastasis:** Cancer cells can travel via blood or lymph to form new tumors elsewhere.
- **Genetic Basis:** It's a disease of the genes, caused by mutations leading to faulty cell instructions.
- **Malignant vs. Benign:** Malignant tumors are cancerous; benign tumors (non-cancerous) grow but don't spread.

Classification of Anti- Cancer Drugs

Anticancer drugs are broadly classified according to their mechanism of action, reflecting their interaction with cellular targets and their ability to inhibit tumor growth.

Alkylating Agents

Alkylating agents are among the earliest classes of chemotherapeutic drugs and function by forming covalent bonds with DNA, leading to cross-linking and strand breakage. This ultimately inhibits DNA replication and transcription, resulting in cell death. Cyclophosphamide and busulfan are widely used examples. **Eg:** Cyclophosphamide, Ifosfamide, Busulfan, Melphalan

Mechanism of Action: Alkylating agents form covalent bonds with DNA, leading to cross-linking of DNA strands (intrastrand and interstrand). This prevents DNA replication and transcription, ultimately causing cell cycle arrest and apoptosis. These drugs are cell cycle–nonspecific but are more effective in rapidly dividing cells.

Antimetabolites

Antimetabolites interfere with nucleotide synthesis and DNA replication by mimicking endogenous metabolites. Methotrexate inhibits dihydrofolate reductase, thereby blocking folate metabolism, while agents such as 5-fluorouracil disrupt pyrimidine synthesis. Eg: Methotrexate, 5-Fluorouracil, Cytarabine, 6-Mercaptopurine.

Mechanism of Action: Antimetabolites mimic endogenous substrates involved in nucleotide synthesis. They interfere with DNA and RNA synthesis by inhibiting key enzymes: Methotrexate → inhibits dihydrofolate reductase (DHFR), 5-FU → inhibits thymidylate synthase, Cytarabine → inhibits DNA polymerase. These agents are S-phase specific.

Antitumor Antibiotics

Antitumor antibiotics, including doxorubicin and actinomycin-D, exert their cytotoxic effects by intercalating into DNA and generating free radicals, leading to DNA damage and inhibition of transcription. **Eg:** Doxorubicin, Daunorubicin, Actinomycin-D, Bleomycin

Mechanism of Action: These drugs act by Intercalating into DNA, disrupting replication and transcription, Generating free radicals, causing DNA strand breaks, Inhibiting topoisomerase II (e.g., doxorubicin), They result in DNA damage and apoptosis.

Plant Alkaloids (Mitotic Inhibitors)

Plant-derived alkaloids, also known as mitotic inhibitors, disrupt microtubule dynamics and prevent cell division. Vinca alkaloids inhibit microtubule assembly, whereas taxanes stabilize microtubules, both leading to mitotic arrest. **Eg:** Vinca alkaloids: Vincristine, Vinblastine, Taxanes: Paclitaxel, Docetaxel, Camptothecins: Irinotecan, Topotecan

Mechanism of Action: Vinca alkaloids: Inhibit microtubule polymerization → prevent spindle formation, Taxanes: Stabilize microtubules → prevent depolymerization, Camptothecins: Inhibit topoisomerase I. These drugs arrest cells in the M phase (mitosis).

Hormones and Hormone Antagonists

Hormonal therapies are effective in hormone-dependent cancers such as breast and prostate cancer. These agents act by blocking hormone receptors or inhibiting hormone synthesis. **Examples:** Tamoxifen, Letrozole, Anastrozole, Flutamide, Leuprolide

Mechanism of Action: These agents act by Blocking hormone receptors (e.g., estrogen receptor by tamoxifen), Inhibiting hormone synthesis (e.g., aromatase inhibitors), Suppressing hormone production (e.g., GnRH analogs). They are effective in hormone-dependent cancers such as breast and prostate cancer.

Platinum Compounds

Platinum-based compounds, such as cisplatin, induce DNA damage by forming cross-links, leading to apoptosis. **Eg:** Cisplatin, Carboplatin, Oxaliplatin

Mechanism of Action: Platinum compounds form DNA cross-links, similar to alkylating agents, leading to: DNA damage, Inhibition of replication, Activation of apoptosis pathways. They are cell cycle–nonspecific.

Targeted Therapy

Targeted therapies represent a major advancement in oncology, selectively inhibiting molecular targets such as tyrosine kinases and growth factor receptors. Imatinib and trastuzumab are notable examples of this class [8]. **Eg:** Imatinib, Erlotinib, Gefitinib, Trastuzumab, Bevacizumab

Mechanism of Action: Targeted therapies selectively inhibit specific molecular targets involved in cancer progression. Tyrosine kinase inhibitors (TKIs) → block signaling pathways, Monoclonal antibodies → target surface receptors (e.g., HER2), Angiogenesis inhibitors → block VEGF signaling. These drugs provide high specificity with reduced toxicity.

Table 2: Classification of Anticancer drugs based on their mechanism of action

Class	*Mechanism*	*Cell Cycle Specificity*
Alkylating agents	DNA cross-linking	Non-specific
Antimetabolites	Inhibit DNA synthesis	S-phase
Antitumor antibiotics	DNA damage/free radicals	Non-specific
Plant alkaloids	Mitotic spindle inhibition	M-phase
Hormonal agents	Block hormone signaling	Variable
Platinum compounds	DNA cross-linking	Non-specific
Targeted therapy	Molecular target inhibition	Specific
Immunotherapy	Immune activation	Not cycle-specific

Immunotherapy

Immunotherapy enhances the body's immune response against cancer cells. Immune checkpoint inhibitors such as nivolumab and pembrolizumab block inhibitory signaling pathways, enabling T-cell-mediated tumor destruction [24]. **Eg:** Nivolumab, Pembrolizumab, Interleukin-2, CAR-T cells

Mechanism of Action: Immune checkpoint inhibitors block PD-1/PD-L1 or CTLA-4 pathways, restoring T-cell activity. Cytokines enhance immune response. CAR-T cells are genetically engineered to target cancer cells. These therapies stimulate the host immune system to destroy tumor cells.

Miscellaneous Agents

Additionally, miscellaneous agents with unique mechanisms further expand the therapeutic landscape of cancer treatment. Eg: L-Asparaginase, Hydroxyurea, Procarbazine

Mechanism of Action: L-Asparaginase → depletes asparagine required for tumor cell survival, Hydroxyurea → inhibits ribonucleotide reductase,

Procarbazine → causes DNA strand damage. These drugs act via unique biochemical pathways.

Recent advances

Cyclophosphamide: Recent advances in cyclophosphamide (CYC) in cancer therapy focus on its immunomodulatory effects at low doses, enhancing delivery via nanotechnology (microemulsions), combining it with immunotherapies (checkpoint inhibitors, vaccines) to overcome immunosuppression, and exploring metronomic dosing for anti-angiogenic and Treg-depleting effects, showing promise in diverse cancers like breast, ovarian, and prostate cancer, though larger trials are needed.

Immunomodulation with Low-Dose (Metronomic) CP

Treg Depletion: Low doses selectively target and deplete regulatory T cells (Tregs), which usually suppress anti-tumor immunity, thereby "releasing the brakes" on the immune system.

Combination with Immunotherapy: CP is increasingly used to prime the immune system before or alongside checkpoint inhibitors (like anti-CTLA-4) or tumor vaccines, enhancing their effectiveness.

Anti-Angiogenic Effects: It also shows anti-angiogenic properties, targeting tumor blood supply.

Nanotechnology & Drug Delivery

Novel Formulations: Researchers are developing nanoparticles and microemulsions (like PEGylated ones) to improve CP's poor solubility, stability, and targeted delivery, leading to better tumor penetration and reduced systemic toxicity.

Enhanced Potency: Nanocarriers have shown superior in vitro killing of cancer cells (e.g., MCF-7 breast cancer) and better in vivo tumor regression.

Combination Therapies

Synergy with Immunomodulators: Combining metronomic CP with other immune-modulating drugs (like lenalidomide) and anti-angiogenics shows promise in delaying tumor progression.

Targeting Resistance: CP's ability to trigger immunogenic cell death (ICD) and enhance antigen presentation helps overcome resistance to other treatments.

New Applications & Strategies

Chemo-Switch Regimens: A strategy involves initial high-dose CP followed by a metronomic maintenance phase, potentially reducing toxicity while maintaining efficacy.

Improved Imaging: MRI is being used to evaluate the anti-angiogenic effects of CP-based regimens in real- time for solid tumors like Small Cell Lung Cancer (SCLC**).**

Methotrexate: Recent advances in methotrexate (MTX) (MTX) cancer therapy focus on nanotechnology for targeted delivery, using nan carriers like nanoparticles, liposomes, and MOFs (Metal-Organic Frameworks) to enhance tumor accumulation, reduce side effects, and enable combination therapies. New strategies also explore MTX's immune-activating role in the tumour microenvironment (TME), potentially as an adjuvant to immunotherapy and radiotherapy, showing promise for improved cancer outcomes by harnessing its immunomodulatory effects alongside traditional chemotherapy.

Nanoparticle-Based Delivery Systems:

Targeting & Controlled Release: Nanomaterials (e.g., graphene oxide, magnetic SPIONs, albumin nanocages) are designed to encapsulate MTX, targeting cancer cells via specific receptors and releasing the drug slowly, improving efficacy and lowering toxicity.

pH-Responsiveness: Carriers like MOFs release MTX more effectively in the acidic tumor environment (pH 5.5) than in normal tissues (pH 7.4), preventing premature release.

Combination Therapies: Nanocarriers can load multiple agents (MTX + natural extracts/other drugs) for synergistic effects, as seen with MTX/Que-loaded nanoparticles for ovarian cancer.

Immunomodulation

Immune Activation: MTX has been shown to activate anti-tumor immunity by inducing cGAMP production in cancer cells, inhibiting its breakdown, and improving responses to immunotherapy and radiotherapy in clinical trials.

Advanced Formulations & Synergies

Ferrofluid Hyperthermia: Combining MTX with magnetic nanoparticles and hyperthermia offers dual targeting and enhanced chemotherapy/radiotherapy effects.

Enhanced Efficacy: Nanocochleate-loaded MTX showed superior anti-proliferative effects against breast cancer cells compared to traditional liposomes, demonstrating better drug delivery.

Actinomycin-D: Recent advances in Actinomycin D (ActD) focus on overcoming its toxicity and enhancing efficacy through combination therapies**,** particularly using low-dose ActD to sensitize cancers (like pancreatic, glioma, Wilms tumors, lung) to other drugs, exploiting its role in p53 activation**,** disrupting protein homeostasis**,** and synergizing with agents like immunotoxins (RG7787)**,** BH3 mimetics (ABT-737)**,** or NFκB inhibitors (DMAPT)**,** revealing its potential in targeted approaches and personalized medicine.

Synergistic Combinations

With Immunotoxins: ActD enhances the killing power of immunotoxins like RG7787 in mesothelin-positive cancers (pancreatic, stomach) by activating apoptosis pathways.

With BH3 Mimetics: Combining low-dose ActD with ABT-737 shows synergistic killing in pancreatic and lung cancer cells, potentially by downregulating anti-apoptotic proteins like Mcl-1, while sparing normal cells.

With NFκB Inhibitors: DMAPT and ActD showed synergistic effects in pancreatic cancer by overcoming NFκB's suppression of apoptosis, leading to more cell death.

Targeting Specific Pathways

p53 Activation: Low-dose ActD activates the p53 pathway, inducing apoptosis in wild-type p53 cells, suggesting its use in "cyclotherapy" for aerodigestive cancers, notes this study.

Proteasome Inhibition: In Wilms tumors, ActD disrupts protein balance; inhibiting the proteasome enhances ActD's effects, linking proteasome activity to prognosis.

Sox2 Downregulation: ActD effectively reduces Sox2, a stem cell marker, in glioma models, inhibiting self-renewal, migration, and invasion, improving survival in preclinical studies.

Personalized & Targeted Use

Context-Dependent Efficacy: Researchers are identifying specific molecules and pathways (like RNase L) that influence ActD sensitivity, paving the way for targeted therapies for specific tumors, say Frontiers.

Addressing Resistance: ActD shows promise in treating methotrexate-resistant gestational trophoblastic neoplasia (GTN), demonstrating effectiveness where other drugs fail.

New Delivery & Low-Dose Strategies

Studies focus on low doses to reduce toxicity while enhancing specific anti-tumor mechanisms, leveraging its ability to induce DNA damage and apoptosis.

Etoposide: Recent advances in Etoposide (ETO) therapy focus on overcoming its limitations (poor solubility, resistance, toxicity) through nanomedicine for targeted delivery (liposomes, nanoparticles), combination therapies (immunotherapy, PARP inhibitors, other targeted agents), and optimized dosing schedules, aiming for synergistic effects, improved tumor targeting, and reduced side effects in cancers like SCLC and lymphomas. Key strategies include using nano-carriers for better bioavailability and exploring novel delivery systems like ferroptosis- inducing nanomedicines.

Advanced Drug Delivery Systems (Nanomedicine)

Nanocarriers: Developing liposomes, polymeric nanoparticles (PLGA), dendrimers, and mesoporous silica nanoparticles (MSNs) to encapsulate ETO.

Targeted Delivery: Functionalizing nanocarriers with ligands (like folic acid) for specific tumor targeting, enhancing drug accumulation in cancer cells.

Improved Solubility & Release: Nanoparticles improve ETO's poor solubility, offer sustained or controlled release, and increase its therapeutic window, potentially reducing systemic toxicity.

Novel Combination Therapies & Strategies

Overcoming Resistance: Combining ETO with agents that block drug efflux pumps (like P-glycoprotein inhibitors) or target other survival pathways to combat multidrug resistance (MDR).

Immunotherapy Integration: Pairing ETO with immunotherapies (like ICIs) to enhance anti- tumor immunity.

Precision Medicine: Using biomarker-guided approaches to tailor ETO therapy for individual patients.

Ferroptosis Induction: Creating ETO-loaded nanomedicines that trigger ferroptosis (iron-dependent cell death) in cancer cells, a new cell death mechanism.

Optimized Dosing & Scheduling

Chronic Dosing: Studies suggest prolonged, low-dose oral ETO (e.g., 21 days) can be effective, even in resistant tumors, potentially acting like a "new drug".

Synergistic Schedules: Exploring schedules that maximize synergy with other agents, particularly for small cell lung cancer (SCLC) and germ cell tumors.

Addressing Specific Challenges

Myelosuppression: Using cytokines to mitigate ETO's dose-limiting bone marrow toxicity.

Bacterial Resistance: Recognizing that ETO can promote antibiotic resistance in tumor-associated bacteria, necessitating combined anti-cancer/anti-microbial strategies.

CONCLUSION

The development of effective drug-delivery systems remains a key challenge in cancer therapy. Conventional chemotherapies are associated with systemic toxicities that limit their dosage and lack tumor selectivity. Advanced DDS aim to enhance the therapeutic index by increasing tumor targeting, longer circulation times, enabling controlled release, and increasing intracellular drug uptake. Recent developments demonstrate promising results. The ADCs, such as Kadcyla (ado-trastuzumab emtansine),

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