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EMERGING ANTICANCER MEDICINES: NOVEL MECHANISMS, THERAPEUTIC TARGETS, AND CLINICAL DEVELOPMENT PATHWAYS

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Abstract

Cancer remains one of the leading causes of death throughout the world, challenging scientists to continue coming up with improved drugs. Chemotherapeutic agents inhibit cancer cell growth through prevention of various processes, such as DNA replication, cell division, and signaling to nearby cells. Conventional chemotherapy, targeted therapies that can selectively inhibit molecules, immunotherapies based on the application of the human body's immune system, and newer approaches like gene therapy and nanomedicine are some of the current treatment methods. While much has been developed, issues still exist, such as resistance of the cancer cells, toxic side effects, and the fact that cancer looks different in every person. Gene study advances of breakthroughs, discovery of the human genome, and drug delivery advances have led to more targeted and personalized treatment approaches. In this essay, new cancer drugs are critically examined with respect to their mechanisms of action, their uses in the real world, and the potential for treating cancer more effectively.

INTRODUCTION

Cancer is a complex disease, characterized by uncontrolled cell proliferation, prolonged cell survival, and the ability of cells to migrate to other locations within the body. It is the leading global cause of death, accounting for approximately 10 million deaths in 2020(WHO, 2021). In 2022, there were 9.7 million cancer deaths (Sun et al., 2025). In 2025 nearly 12 million deaths is increasing in male cases and lowering in women (Kim et al,2025).The global cancer burden continues to escalate due to aging populations, environmental exposures, and lifestyle factors, establishing oncology as a critical healthcare priority (Bray et al., 2018). Anticancer therapeutics encompass diverse drug classes designed to inhibit malignant growth or eliminate cancer cells, including conventional chemotherapy, targeted therapies, immunotherapy, and emerging modalities such as gene therapy and nanotechnology platforms. Traditional cytotoxic agents, including alkylating compounds and antimetabolites, disrupt cellular division processes but lack specificity, resulting in significant normal tissue toxicity alongside their antitumor effects (Chabner & Roberts, 2005; Longley & Johnston, 2005). The emergence of targeted therapy has transformed cancer management by addressing specific molecular aberrations driving malignant behavior. These agents, exemplified by tyrosine kinase inhibitor imatinib and monoclonal antibody trastuzumab, demonstrate enhanced efficacy when matched to appropriate tumor biomarkers while exhibiting improved safety profiles compared to conventional chemotherapy (Zhang et al., 2009; Dienstmann et al., 2013). Immunotherapeutic approaches represent another paradigm shift, harnessing endogenous immune mechanisms to combat malignancy. Immune checkpoint inhibitors targeting PD-1 and CTLA-4 pathways have achieved remarkable outcomes in melanoma, lung, and renal cancers; though responses remain limited to

patient subsets and can produce severe immune-related toxicities (Pardoll, 2012; Weber et al., 2015). Despite therapeutic advances, significant challenges persist, including intrinsic and acquired drug resistance, tumor heterogeneity complicating treatment selection, and prohibitive costs limiting global access, particularly in resource-limited settings (Al-Lazikani et al., 2012; Knaul et al., 2018). Contemporary research focuses on addressing these limitations through improved drug delivery systems, novel target identification, and precision medicine approaches. Nanomedicine platforms enhance therapeutic indices by optimizing drug solubility, stability, and tumor selectivity while minimizing off-target effects (Rosenblum et al., 2018). Pharmacogenomic profiling enables treatment personalization based on individual tumor genetics, maximizing efficacy while reducing toxicity (Dienstmann et al., 2013). Cancer therapy has evolved from broad-spectrum cytotoxic approaches to increasingly sophisticated, personalized interventions, yet substantial scientific, clinical, and access challenges remain. Future progress will depend on interdisciplinary collaboration integrating medical, genetic, immunological, and nanotechnological expertise to develop more effective, tolerable, and accessible cancer treatments. (Hanahan, 2022).

TYPES OF ANTICANCER MEDICINES

TRADITIONAL CHEMOTHERAPY

Conventional chemotherapy remains a fundamental pillar of cancer treatment despite the emergence of novel therapeutic modalities. These systemic agents exploit the rapid proliferation characteristic of malignant cells, though they lack tumor specificity. Chemotherapeutic drugs encompass several mechanistic classes, including alkylating agents, antimetabolites, anthracyclines, and mitotic spindle inhibitors, each with distinct modes of action. Alkylating agents induce DNA cross-linking through covalent bond

formation, preventing proper chromosomal replication and ultimately triggering apoptotic cell death. Antimetabolites function as structural analogs of natural nucleotide precursors, disrupting DNA and RNA synthesis when incorporated into cellular metabolic pathways (Hanahan, 2022). The fundamental limitation of cytotoxic chemotherapy lies in its inability to distinguish between malignant and rapidly dividing normal tissues, particularly affecting bone marrow hematopoiesis, gastrointestinal epithelium, and hair follicles. This non-selective cytotoxicity manifests as characteristic adverse effects including myelosuppression with consequent immunocompromise and anemia, gastrointestinal toxicity presenting as nausea and vomiting, and alopecia. The compromised immune function secondary to bone marrow suppression significantly increases infection susceptibility, requiring careful monitoring and supportive care interventions. Despite these substantial toxicities, chemotherapy maintains clinical relevance due to demonstrated efficacy across numerous malignancies. Contemporary practice increasingly employs multimodal approaches combining chemotherapy with surgical resection and radiation therapy to optimize treatment outcomes. Advances in dosing strategies, supportive care protocols, and toxicity management have improved patient tolerance, though the inherent lack of selectivity continues to limit therapeutic indices and necessitates careful risk-benefit assessment in treatment planning.One of the key drawbacks of conventional chemotherapy and radiotherapy is their inability to selectively target malignant cells. As a result, normal tissues are frequently affected, producing adverse outcomes such as immunosuppression, nausea, and fatigue. These nonspecific effects not only impair quality of life but also increase vulnerability to secondary infections and complications. Emerging approaches, including the use of nanoparticles for drug delivery and antibody–drug conjugates, have been developed to

enhance tumor-specific targeting, thereby minimizing off-target toxicity and reducing treatment-related side effects (Rituraj et al., 2025).

TARGETED THERAPY

Targeted therapy represents a paradigmatic advancement in oncological treatment, selectively engaging specific molecular targets aberrantly expressed in malignant cells while sparing normal tissue. This precision approach demonstrates superior therapeutic specificity and reduced toxicity compared to conventional cytotoxic regimens. Unlike broad-spectrum chemotherapy that indiscriminately affects rapidly proliferating cells; targeted agents disrupt critical pathways essential for cancer cell survival, proliferation, and metastatic dissemination. The targeted therapeutic arsenal encompasses diverse drug classes including tyrosine kinase inhibitors, monoclonal antibodies, proteasome inhibitors, and angiogenesis inhibitors, each addressing distinct oncogenic mechanisms. Tyrosine kinase inhibitors such as imatinib selectively block enzymatic signaling cascades that drive malignant cell growth and division (Sawyers, 2004). Monoclonal antibodies like trastuzumab bind to tumor-associated surface antigens such as HER2, either neutralizing protein function or facilitating immune-mediated tumor destruction. Angiogenesis inhibitors disrupt neovascularization processes essential for tumor nutrient supply and oxygenation, thereby limiting malignant progression. The molecular specificity of targeted agents typically results in more favorable safety profiles compared to traditional chemotherapy. However, therapeutic resistance frequently emerges through various mechanisms including target mutation, pathway redundancy, and alternative signaling activation, limiting long-term efficacy. Successful targeted therapy implementation requires comprehensive biomarker analysis to identify appropriate patient populations, emphasizing the critical role of precision medicine in contemporary

oncology. The clinical success of targeted interventions has accelerated pharmaceutical development and fundamentally transformed cancer treatment paradigms toward molecularly-informed therapeutic strategies. (Yonesaka et al., 2015). Prevention of drug resistance of cancer from target drugs can be done by following pathways: bypassing activation of signals by inhibiting PI3K/AKT, tumor cells, long-term immunotherapy for avoiding hindering T-cell activity by secreting immunosuppressive factors like TGF-β and IL-10 (Huang et al., 2025).

IMMUNOTHERAPY

Immunotherapy employs the body's immune system to combat cancer. In some cases, cancer cells can evade immune detection through a mechanism known as immune checkpoints, which transmit signals to the immune system to cease attacking normal cells. Immunotherapy focuses on blocking these checkpoints, allowing the immune system to identify and attack cancer cells (Topalian et al., 2015). Unlike chemotherapy or targeted therapies, immunotherapy has the potential to provide long-lasting protection by teaching the immune system how to fight cancer again in the future. Various types of immunotherapy exist, such as monoclonal antibodies, cancer vaccines, T-cell therapy, and cytokine therapy. For example, checkpoint inhibitors have been significant in treating cancers like melanoma, non-small cell lung cancer, and kidney cancer. However, not all patients respond to immunotherapy, and researchers are still working to develop more effective ways to predict who will benefit. Another challenge is that immunotherapy may cause the immune system to attack normal tissues, leading to inflammation in organs such as the lungs, liver, or colon. The efficiency of immunotherapy was enhanced by supplying patients with additional gut microbiota (Mahmoudian et al., 2025). Physicians closely monitor patients and may use additional

drugs to manage these side effects. Despite these challenges, immunotherapy offers hope for those previously difficult to treat for cancers, and ongoing studies aim to improve its effectiveness and accessibility. (Pardoll, 2012)

HORMONE THERAPY

Hormonal therapy represents a cornerstone treatment modality for hormone-dependent malignancies, particularly breast and prostate cancers, where tumor growth relies on endogenous hormone stimulation involving estrogen, progesterone, and testosterone. This therapeutic approach functions by either reducing circulating hormone levels or blocking hormone-receptor interactions essential for cancer cell proliferation. In breast cancer management, selective estrogen receptor modulators such as tamoxifen competitively inhibit estrogen binding to tumor cells, effectively suppressing malignant growth (Jordan, 2003). Aromatase inhibitors provide an alternative mechanism by reducing estrogen production in postmenopausal women through enzymatic blockade of peripheral hormone conversion. Prostate cancer treatment employs androgen deprivation therapy achieved through surgical castration or pharmacological interventions that suppress testosterone production or block androgen receptor signaling. The favorable toxicity profile of hormonal agents compared to cytotoxic chemotherapy, combined with convenient oral administration, enables prolonged treatment duration with improved patient tolerance. However, hormonal manipulation frequently produces characteristic adverse effects including vasomotor symptoms, bone mineral density loss, fatigue, and sexual dysfunction resulting from altered endocrine homeostasis. Treatment resistance inevitably develops through various molecular mechanisms, necessitating therapeutic modifications or alternative interventions. Despite these limitations, hormonal therapy remains integral to

hormone-sensitive cancer management, demonstrating significant survival benefits and enhanced quality of life outcomes for appropriately selected patients. (Smith & Saad, 2012). Targeting the HER family of growth factor receptors using monoclonal antibodies such as trastuzumab or tyrosine kinase inhibitors (e.g., gefitinib, lapatinib) has proven effective in breast cancer management (Zafar et al., 2025). In a randomized trial, patients with ER-positive advanced breast cancer who received gefitinib combined with anastrozole achieved longer progression-free survival compared with anastrozole alone. The efficacy of aromatase inhibitors (AIs) may also be influenced by genetic variation. Specific single-nucleotide polymorphisms (SNPs), including rs6493497 and rs7176005, have been implicated in modulating CYP19 (aromatase) activity following AI therapy (Amaral et al., 2025), while SNP rs4646 has likewise been associated with treatment response. Furthermore, a comparative study in patients treated with letrozole alone or with chemotherapy highlighted the contribution of HIF1-α and P44/42 MAPK to therapeutic resistance (Panda et al., 2025).

GENE THERAPY AND NANOMEDICINE

Gene therapy and nanomedicine are emerging areas of cancer combat, with the goal of more effectively and less harmfully targeting treatments. Gene therapy achieves this through the introduction of genes into a patient's cells to fix or control genes that enable cancer development, such as stopping cancer cell growth, applying RNA interference to knock out risky genes, or reshaping immune cells to enhance their ability to kill cancer, as seen in CAR T-cell therapy (June et al., 2018). Although these approaches hold promise, they are currently in a large experimental phase and face challenges, such as ensuring the genetic material targets the right cells safely and treating possible immune responses. Nanomedicine utilizes extremely small particles

called nanoparticles to deliver cancer medications directly to tumors. These particles can enhance drug solubility, prevent degradation, and initiate drug release when they reach specific conditions in the tumor, such as temperature or pH. This site-specific approach protects healthy cells while concentrating drugs in cancer cells, improving treatment efficacy and reducing side effects. Examples include special drug carriers like liposomes used with chemotherapy agents such as doxorubicin, and recently developed nano-carriers now in clinical trials. Merging nanotechnology with other treatments, such as immunotherapy or gene therapy, holds significant potential for future cancer therapies, but further research and testing are required before large-scale implementation(Peer et al., 2007).Nan et al. (2025) provided preclinical evidence supporting the therapeutic potential of IL-18 mimetics in cancer immunotherapy, underscoring the importance of bioengineering approaches for cytokine-based therapies. They developed a novel bispecific humanized nanobody, A4B2-mdFc, designed to activate IL-18 signaling. This construct demonstrated robust IL-18 activity without inhibition by IL-18BP and enhanced $CD8^+$ T-cell responses. In hu-PBMC CDX models, A4B2-mdFc inhibited tumor growth in a dose-dependent manner, both as monotherapy and in combination with immune checkpoint inhibitors such as anti–PD-1 or anti–CTLA-4. Zhang et al (2024) build a therapeutic platform using molybdenum disulfide (MoS_2) nanozymes and *Chlorella*-derived photosensitizers embedded in a hyaluronic acid–dopamine/2-aminoethyl methacrylate (HA-DA/AMA) matrix. This system combines photodynamic and photothermal therapies to selectively destroy cancer cells while inducing immunogenic cell death (ICD), which in turn promotes an antitumor immune response.

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MECHANISM OF ACTION OF DRUGS

Anticancer therapeutics target essential cellular pathways required for malignant cell survival, proliferation, and growth. Conventional chemotherapeutic agents primarily disrupt cell cycle progression, thereby inhibiting cancer cell division and metastatic spread. Alkylating agents induce DNA damage through the addition of alkyl groups, creating strand breaks and cross-links that prevent proper DNA replication and transcription. Antimetabolites mimic natural nucleotide precursors, causing incorporation errors during DNA and RNA synthesis that result in defective genetic material and impaired cellular division. Targeted therapies focus on specific molecular aberrations that drive oncogenic processes. Tyrosine kinase inhibitors block critical signaling enzymes that promote cancer cell growth and survival (Zhang et al., 2009). Monoclonal antibodies bind to tumor-associated surface proteins, either disrupting their function or marking cancer cells for immune-mediated destruction. Immunotherapeutic approaches enhance the host immune response by removing inhibitory signals that normally prevent T-cell activation against malignant cells, enabling effective tumor recognition and elimination. Hormonal interventions modulate the endocrine environment by reducing circulating hormone levels or blocking hormone receptor signaling, particularly effective against hormone-sensitive malignancies where growth depends on estrogen or androgen stimulation.. More recently, treatments such as gene therapy aim to repair or replace the genetic abnormalities responsible for cancer. Nanomedicine helps in the targeted delivery of drugs to cancer cells, increased drug concentration at the tumor site, and sparing of healthy tissues from damage. The aptamers and magnetites are crucial in targetting the malignant part of bodies (Askari et al., 2025). Though these therapies work through different mechanisms, one of the major

challenges is that cancer cells are capable of becoming drug-resistant. Drug repurposing of nonsteroidal anti-inflammatory drugs (NSAIDs), particularly fenamates, has shown promise in cancer therapy, as these agents may reduce cardiovascular risk when guided by aptamer-based approaches. Fenamates exhibit anticancer activity through both COX-dependent and COX-independent pathways. COX-2 functions not only as a central regulator of inflammation but also as a key driver of malignant transformation, linking chronic inflammation to cancer. Its overexpression leads to elevated prostaglandin levels within the tumor microenvironment, thereby promoting tumor progression (Li et al., 2025). This is usually achieved by gene mutations or by the utilization of alternative routes. Due to the reality of drug resistance, doctors often prefer to use treatments in combination to improve their effectiveness. Additionally, ongoing research is conducted to develop more potent and targeted therapies to combat cancer and other diseases.(Holohan et al., 2013).

CLINICAL CHALLENGES IN CANCER TREATMENT

Cancer therapy continues to overcome several major challenges that limit treatment effectiveness and patient access to care. The primary obstacle is tumor heterogeneity, where individual malignancies contain genetically and behaviorally diverse cancer cell populations, making it difficult to identify therapies that effectively target all cellular variants and often resulting in treatment resistance or disease recurrence. Many cancers are diagnosed at advanced stages due to vague or absent early symptoms, requiring more aggressive and toxic therapeutic approaches once metastatic spread has occurred. Current treatments like chemotherapy and radiation therapy cause significant collateral damage to healthy tissues, leading to immunosuppression, organ dysfunction, fatigue, and increased infection susceptibility (Marusyk et al., 2012). Healthcare disparities

further compound these issues, with geographic barriers limiting access to specialized treatment centers and financial constraints preventing optimal patient management. Low clinical trial participation due to location, cost, and lack of awareness impedes therapeutic advancement and evidence generation. Additionally, the psychological burden on patients and families remains inadequately addressed despite being crucial for comprehensive care. These collective challenges underscore the urgent need for continued therapeutic innovation, enhanced early detection strategies, personalized patient-centered care models, and healthcare system reforms to improve treatment accessibility and clinical outcomes across diverse patient populations. (Sullivan et al., 2011). Other challenges include the development of drugs that, during absorption by patients, do not release radical oxygen species, such as uracil, as it occurs in galactosamine receptors (Jiang et al., 2024) or lutein receptors, without risk of hazardous compounds in the excreted residues (Zhang et al 2025).

DRUG RESISTANCE IN CANCER THERAPY

Z significant issue that renders newly developed cancer medications ineffective over time. While initial improvements may be evident, cancer cells can eventually become resistant, leading to relapse or worsening of the condition. This resistance can occur in two general ways: it may be inherent at the onset, with cancer cells not responding to treatment, or it may develop later as cells adapt to the therapy after prolonged drug use. Various factors contribute to the development of resistance, including genetic mutations in cancer cells, such as mutations in the EGFR gene, which render certain drugs ineffective, particularly in lung cancer. Other mechanisms include cancer cells shedding excess P-glycoprotein, which acts as a pump to remove drugs from the cells, rendering them ineffective (Gottesman et al., 2002). Additionally, cancer cells can become more

drug-resistant and malignant through the epithelial-to-mesenchymal transition process. The tumor microenvironment, characterized by limited oxygen levels, an immunocompromised nature, and interactions with surrounding tissues, can also help cancer cells survive by shielding them from drugs or the immune system. Some tumors can seek other paths in an effort to grow even if their natural avenues of growing are targeted by treatment, thus they still exists. Cancer stem cells represent a critical factor in treatment resistance, constituting a subpopulation within tumors that exhibits intrinsic resistance to conventional therapies and possesses the capacity for tumor regeneration following treatment cessation. The rapid and multifaceted mechanisms underlying resistance development present substantial challenges for achieving durable disease control. Consequently, clinicians increasingly adopt strategies including combination chemotherapy, treatment protocol modifications, and the development of novel therapeutic agents designed to circumvent resistance pathways. Enhanced understanding of resistance mechanisms and their clinical management remains fundamental to improving patient survival outcomes and optimizing therapeutic efficacy in oncological practice.(Holohan et al., 2013).Single-cell RNA sequencing studies in breast cancer have identified a C3 PCLAF^{+} tumor cell subtype in HR^{+} disease, associated with poor prognosis and resistance to endocrine therapy. myc-associated zinc finger protein (MAZ) has been recognized as a key regulatory factor driving tumor progression, while the tumor microenvironment plays a critical role in facilitating immune evasion (Ni et al., 2025)

PERSONALIZED MEDICINE IN CANCER CARE

Precision cancer employs broad-range tumor profiling to identify the unique genetic mutations, protein levels, and molecular drivers of the neoplastic transformation. With

broad-range molecular profiling, the clinician is able to select for the targeted therapies that are uniquely targeting the pathogenic processes at play with the highest therapeutic value and lowest treatment toxicity. In breast cancer, HER2 overexpression status predicts trastuzumab appropriateness, while lung cancer carriers of EGFR mutation and ALK rearrangement respond well to targeted tyrosine kinase inhibitors rather than to conventional cytotoxic therapy. Biomarker assessment to predict treatment response, and to forecast resistance patterns, makes it possible to modify therapy proactively (Dienstmann et al., 2013). Choice of immunotherapy now increasingly depends on biomarkers such as PD-L1 level of expression and tumor mutational burden to select patients who would receive maximum benefit from immune checkpoint inhibition. Next-generation sequencing technologies have made it possible to analyze the whole tumor genome quickly, which reduces clinical decision-making time while achieving maximum diagnostic accuracy. Artificial intelligence (AI) is a strong tool for large-scale genomic data analysis that can detect new mutations, therapeutic targets, and patient-specific treatment plans (O'Connor, & McVeigh, 2025). Drug behavior is highly associated with genomic alterations of cancer cells, and AI-based models such as Alpha Fold have been set up to predict such behaviors based on genomic data. Aside from that, deep learning methods are increasingly applied for the purposes of enriching patient databases and gene banks in order to identify biomarkers out of pathetic and radiomic information and bring about non-invasive solutions into the traditional diagnosis procedures (Gnanaolivu, R., & Hart, S. N. (2024)). However, some of the implementation issues include high expense, requirement of specialized lab facilities, and lack of professionals in rural areas (Guo et al., 2024). The dynamic nature of cancer is other issues because heterogeneity of the tumor and resistance acquired need

to be monitored constantly with molecular surveillance and therapeutic adjustment. Yet, precision medicine is a new concept in which the therapeutic management is blended with the biology of the disease, which results in improved patient outcomes, fewer side effects, and increased probability of long-term remissions in many cancers (Garraway & Verweij, 2013).

CONCLUSION

Cancer treatment has evolved leaps and bounds in the last ten years with more precise and targeted treatments overshadowing mass-based therapies like chemotherapy. Immunotherapy and targeted drugs are revolutionizing cancer treatment by hitting specific spots in cancer cells and employing the body's own immune system to kill the disease. These new technologies are providing better results and less toxicity to the patients, setting their hopes upon them. Novel technologies like nanomedicine and gene therapy are enhancing the efficiency and safety of drug delivery and potentially making it capable of resolving some of the limitations in current therapies. There are still numerous challenges, however, such as drug resistance, the complexities of cancer development, and the cost and unavailability of new drugs. To fight such challenges, more research and fine-tuning are needed, such as developing more combination therapies and pushing further on personalized medicine so that each patient's unique cancer can be targeted. More coordination across various scientific disciplines and healthcare systems is also required regarding availability and effectiveness of these new therapies. The future holds promise in cancer treatment in converting cancer into a chronic illness, improving survival, and improving the well-being of patients globally.

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