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REVIEW ARTICLE

A Revisit on FDA Approved Anticancer Drugs (1949 – 2024)

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Abstract: The U.S Food and Drug Administration (FDA) has approved and expanded indications for many drugs related to the treatment of different types of cancer and adverse events. One of the first drugs used clinically in modern medicine for cancer treatment was the alkylating agent mechlorethamine, a nitrogen mustard found effective in treating lymphomas in the 1940s. FDA-approved cancer drugs undergo rigorous testing to ensure safety and efficacy in treating various types of cancer. These drugs go through extensive clinical trials to demonstrate their benefits and potential side effects. Once approved by the FDA, they are available for use by healthcare providers to treat cancer patients. These drugs have significantly improved treatment outcomes for cancer patients and continue to be a vital part of cancer therapy. Healthcare providers must stay updated on FDA-approved cancer drugs to provide the best care for their patients.

Keywords: Cancer; Food and Drug Administration; Oncology; Drug approval; Mechanism of Action.

1. Introduction

Cancer is a group of diseases characterized by uncontrolled growth and spread of abnormal cells [1]. It is a major health problem worldwide, caused by various factors such as environmental influences, internal stress, and heredity [2]. Breast cancer is the most prevalent type in women, while lung and prostate cancers have the highest incidence in males [3]. Radiotherapy, an integral part of the oncology treatment paradigm, aims to kill cancer cells or slow their growth by damaging their DNA [4]. The development of chemotherapy in the 1950s and 1960s revolutionized cancer treatment [5]. Anti-cancer drugs, also known as anticancer agents or cytotoxic drugs, are designed to treat cancer by killing or inhibiting the growth of cancer cells [6]. These drugs are often used in combination with surgery, radiation therapy, and immunotherapy [7]. The first chemotherapy drug, cyclophosphamide, was introduced in the 1940s and is still in use today [8]. Since then, numerous classes of anti-cancer drugs have been developed, targeting various aspects of cancer cell biology [9].

Anti-cancer drugs play a crucial role in the treatment and management of cancer, improving survival rates and quality of life for many patients [10]. Significant advancements in medical research have led to the development of a wide range of anti-cancer drugs, each with specific mechanisms of action and targets [11]. However, conventional anticancer drugs have disadvantages, such as systemic toxicity and adverse side effects, which necessitate the development of alternative treatments with reduced adverse effects and improved therapeutic efficacy [12].

One effective strategy to increase the selectivity of chemotherapeutics involves the use of prodrugs, which are inactive compounds that are chemically or enzymatically metabolized into the active drug, reducing the systemic toxicity of conventional therapies [13]. Prodrugs can also be useful in reducing drug toxicity, such as in the case of transition metals, which are generally not included in drug therapies due to their intrinsic toxicity [14]. Prodrug therapy provides an alternative approach to designing less reactive and less cytotoxic drugs, helping to overcome pharmaceutical, pharmacokinetic, and pharmacodynamic hindrances [15]. Before a new anti-cancer drug can be marketed and prescribed to patients, it needs to go through a rigorous evaluation process by regulatory agencies like the U.S. Food and Drug Administration (FDA) [16]. The FDA approval process involves several stages, including preclinical testing, investigational new drug (IND) application, clinical trials, new drug application (NDA), FDA review, and approval or rejection [17]. FDA approval signifies that the benefits of the anti-cancer drug outweigh its risks and that it is safe and effective for its intended use [18].

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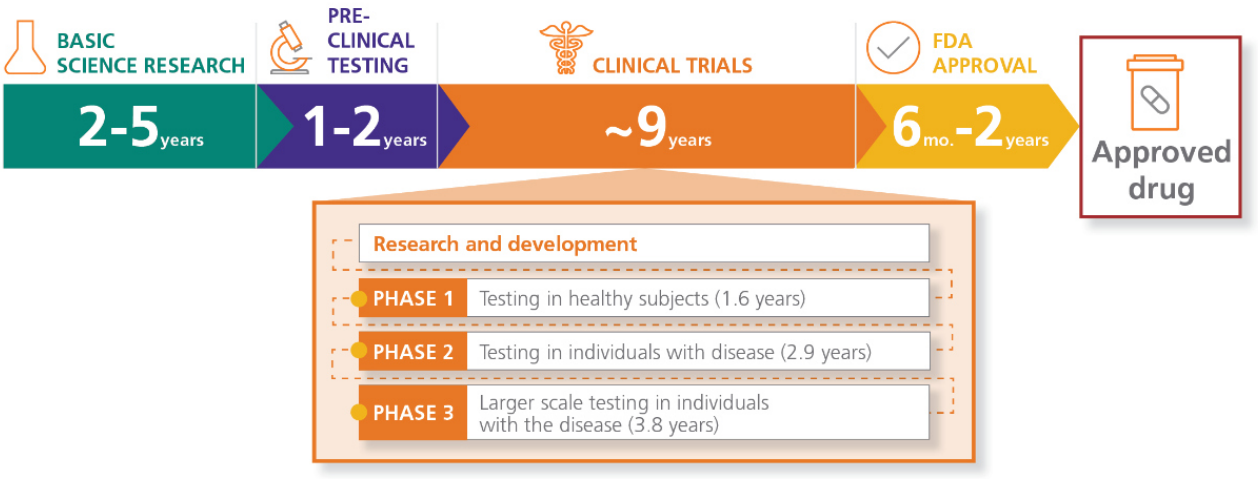

Figure 1. FDA Drug approval process

Table 1. Comparison of Traditional Chemotherapy and Targeted Therapy

Characteristic	Traditional Chemotherapy	Targeted Therapy
Mechanism of Action	Kills rapidly dividing cells, including cancer cells and normal cells	Specifically targets cancer cells by interfering with specific molecules involved in tumor growth and progression
Specificity	Non-specific, affects both cancer cells and normal cells	Highly specific, minimizing damage to normal cells
Side Effects	More severe side effects, including hair loss, nausea, vomiting, fatigue, and increased risk of infection	Generally milder side effects compared to traditional chemotherapy
Administration	Usually administered intravenously	Can be administered orally or intravenously
Efficacy	Effective for many types of cancer, but may not be curative	Highly effective for cancers with specific molecular targets, potentially leading to improved survival and quality of life
Resistance	Cancer cells may develop resistance to chemotherapy over time	Cancer cells may also develop resistance to targeted therapies, requiring the development of new targeted agents
Cost	Generally less expensive than targeted therapies	Often more expensive than traditional chemotherapy

2. FDA-Approved Anticancer Drugs (1949-2024)

2.1. Initial era (1949 to 1975)

Mechlorethamine, approved in 1949, was one of the first drugs used clinically in modern medicine for cancer treatment [19, 20]. It is an alkylating agent that attaches alkyl groups to DNA bases, resulting in DNA fragmentation by repair enzymes attempting to replace the alkylated bases, preventing DNA synthesis and RNA transcription [21].

Leucovorin, approved in 1952, is an active metabolite of folic acid and an essential coenzyme for nucleic acid synthesis [22]. It can be used to selectively "rescue" cells from the adverse effects of methotrexate or to increase the efficacy of fluorouracil [23]. Methotrexate, approved in 1953, competitively inhibits dihydrofolate reductase (DHFR), an enzyme that participates in

tetrahydrofolate synthesis [24]. Methotrexate inhibits the synthesis of DNA, RNA, thymidylates, and proteins by preventing the conversion of dihydrofolate to the active tetrahydrofolate [25].

Mercaptopurine, approved in 1953, inhibits several pathways in nucleic acid biosynthesis, preventing the proliferation of cells involved in the determination and amplification of the immune response [26]. Busulfan, approved in 1954, is an alkylating agent that forms DNA-DNA interstrand crosslinks between the DNA bases guanine and adenine and between guanine and guanine [27].

Fluoxymesterone, approved in 1956, produces retention of nitrogen, sodium, potassium, and phosphorus, increases protein anabolism, decreases amino acid catabolism, and decreases urinary excretion of calcium [28]. Its antitumor activity appears related to the reduction or competitive inhibition of prolactin receptors or estrogen receptors or production [29]. Chlorambucil, approved in 1957, interferes with DNA replication and damages the DNA in a cell [30]. The DNA damage induces cell cycle arrest and cellular apoptosis via the accumulation of cytosolic p53 and subsequent activation of Bcl-2-associated X protein, an apoptosis promoter [31]. Cyclophosphamide, approved in 1959, is an alkylating agent of the nitrogen mustard type [32]. Its activated form, phosphoramide mustard, alkylates or binds to DNA, causing cross-linking of DNA and RNA strands and inhibition of protein synthesis [33]. These actions do not appear to be cell-cycle specific [34].

Vincristine sulfate, approved in 1963, interferes with nucleic acid and protein synthesis by blocking glutamic acid utilization [35]. Dactinomycin, approved in 1964, generates DNA strand breaks via interaction with topoisomerase II [36]. Vinblastine sulfate, approved in 1965, blocks cell growth by stopping mitosis (cell division) [37]. Thioguanine, approved in 1966, inhibits the conversion of inosinic acid (IMP) to xanthylic acid (XMP) by competition for the enzyme IMP dehydrogenase [38]. Thioguanine nucleotides are incorporated into both DNA and RNA by phosphodiester bonds [39]. Procarbazine hydrochloride, approved in 1969, inhibits DNA, RNA, and protein synthesis by inhibiting transmethylation of methionine into transfer RNA and may also damage DNA directly through alkylation [40]. Floxuridine, approved in 1970, inhibits the synthesis of DNA and RNA [41]. Fluorouracil, approved in 1970, is converted to fluorodeoxyuridine monophosphate (FdUMP), which forms a stable complex with thymidylate synthase (TS) and inhibits deoxythymidine mono-phosphate (dTMP) production [42]. Mitomycin, approved in 1974, generates DNA damage by producing cross-links between the complementary strands of DNA [43]. Dacarbazine, approved in 1975, alkylates and cross-links DNA during all phases of the cell cycle, resulting in disruption of DNA function, cell cycle arrest, and apoptosis [44].

2.2. Golden era (1976 to 2000)

Lomustine, approved in 1976, forms interstrand crosslinks in DNA, inhibiting DNA replication and RNA and protein synthesis [45]. Carmustine, approved in 1977, generates interstrand crosslinkages in DNA, inhibiting DNA replication and RNA synthesis [46]. Tamoxifen citrate, approved in 1977, is a selective estrogen receptor modulator (SERM) that competitively binds to estrogen receptors on tumors and other tissue targets, producing a nuclear complex that decreases DNA synthesis and inhibits estrogen effects [47]. Cisplatin, approved in 1978, binds to and cross-links DNA, ultimately triggering apoptosis [48]. Doxorubicin hydrochloride, approved in 1979, intercalates between base pairs in the DNA helix, inhibiting DNA replication and RNA synthesis [49]. Idarubicin hydrochloride, approved in 1990, intercalates into DNA and interferes with the activity of topoisomerase II, leading to DNA damage and apoptosis [50]. Paclitaxel, approved in 1992, promotes microtubule assembly and stabilizes tubulin polymers by preventing their depolarization, resulting in the formation of stable, nonfunctional microtubules and consequently inhibiting cell replication [51]. Gemcitabine hydrochloride, approved in 1996, inhibits DNA synthesis, leading to cell death [52]. Irinotecan hydrochloride, approved in 1996, inhibits topoisomerase I activity by stabilizing the cleavable complex between topoisomerase I and DNA, resulting in DNA breaks that inhibit DNA replication and trigger apoptotic cell death [53]. Capecitabine, approved in 1998, is converted to 5-fluorouracil (5-FU) by thymidine phosphorylase, which is found in higher concentrations in many tumors compared to normal tissues [54]. Temozolomide, approved in 1999, is an alkylating agent that can cross the blood-brain barrier and induce cell cycle arrest at G2/M, leading to apoptosis [55]. Anastrozole, approved in 2000, is a potent and selective non-steroidal aromatase inhibitor that significantly lowers serum estradiol concentrations by inhibiting aromatase, the enzyme responsible for the conversion of androgens to estrogens [56].

2.3. Modern era (2001 to till date)

Fulvestrant, approved in 2002, is an estrogen receptor antagonist that binds to the estrogen receptor and downregulates its expression, leading to reduced estrogen signaling [57]. Gefitinib, approved in 2003, inhibits the intracellular phosphorylation of tyrosine kinase associated with the epidermal growth factor receptor (EGFR) [58]. Erlotinib hydrochloride, approved in 2004, inhibits the intracellular phosphorylation of tyrosine kinase associated with the epidermal growth factor receptor (EGFR) [59]. Sorafenib tosylate, approved in 2005, is a kinase inhibitor that decreases tumor cell proliferation by targeting the RAF/MEK/ERK signaling pathway at the level of RAF kinase and by exerting an antiangiogenic effect through inhibition of the vascular endothelial growth factor receptor (VEGFR) and platelet-derived growth factor receptor (PDGFR) signaling pathways [60]. Sunitinib malate, approved in 2006, inhibits multiple receptor tyrosine kinases, including platelet-derived growth factor receptors (PDGFRα and PDGFRβ), vascular endothelial growth factor receptors (VEGFR1, VEGFR2, and VEGFR3), stem cell factor receptor (KIT), Fms-like tyrosine kinase-3 (FLT3), colony-stimulating factor receptor Type 1 (CSF-1R), and the glial cell-line derived neurotrophic factor receptor (RET) [61].

Lapatinib ditosylate, approved in 2007, inhibits the tyrosine kinase activity of the epidermal growth factor receptor (EGFR) and human epidermal growth factor receptor 2 (HER2), leading to inhibition of tumor cell growth [62]. Pazopanib hydrochloride,

approved in 2009, is a multi-tyrosine kinase inhibitor of vascular endothelial growth factor receptor (VEGFR)-1, VEGFR-2, VEGFR-3, platelet-derived growth factor receptor (PDGFR)-α and -β, fibroblast growth factor receptor (FGFR)-1 and -3, cytokine receptor (Kit), interleukin-2 receptor inducible T-cell kinase (Itk), leukocyte-specific protein tyrosine kinase (Lck), and transmembrane glycoprotein receptor tyrosine kinase (c-Fms) [63]. Cabazitaxel, approved in 2010, binds to and stabilizes tubulin, resulting in the inhibition of microtubule depolymerization and cell division, cell cycle arrest in G2/M, and the inhibition of tumor cell proliferation [64]. Vemurafenib, approved in 2011, is a kinase inhibitor that decreases tumor cell proliferation by inhibiting the serine-threonine kinase BRAF [65]. Crizotinib, approved in 2011, inhibits receptor tyrosine kinases, including anaplastic lymphoma kinase (ALK), Hepatocyte Growth Factor Receptor (HGFR, c-Met), ROS1 (c-ros), and Recepteur d'Origine Nantais (RON) [66]. Axitinib, approved in 2012, inhibits receptor tyrosine kinases, including vascular endothelial growth factor receptors (VEGFR)-1, VEGFR-2, and VEGFR-3, resulting in inhibition of endothelial cell proliferation, migration, and survival [67]. Regorafenib, approved in 2012, inhibits multiple membrane-bound and intracellular kinases involved in normal cellular functions and pathologic processes, including those in the RET, VEGFR1, VEGFR2, VEGFR3, KIT, PDGFR-alpha, PDGFR-beta, FGFR1, FGFR2, TIE2, DDR2, Trk2A, Eph2A, RAF-1, BRAF, BRAF(V600E), SAPK2, PTK5, and Abl pathways [68]. Dabrafenib mesylate, approved in 2013, inhibits the activity of BRAF V600 mutant kinase, leading to inhibition of tumor cell proliferation [69]. Afatinib dimaleate, approved in 2013, irreversibly binds to and inhibits the kinase activity of the epidermal growth factor receptor (EGFR), human epidermal growth factor receptor 2 (HER2), and HER4, resulting in inhibition of tumor cell proliferation [70]. Ibrutinib, approved in 2013, inhibits Bruton's tyrosine kinase (BTK), a signaling molecule of the B-cell antigen receptor (BCR) and cytokine receptor pathways, leading to inhibition of B-cell proliferation [71].

Ceritinib, approved in 2014, inhibits anaplastic lymphoma kinase (ALK), insulin-like growth factor 1 receptor (IGF-1R), insulin receptor (InsR), and ROS1 [72]. Olaparib, approved in 2014, is a poly(ADP-ribose) polymerase (PARP) inhibitor that inhibits PARP enzymatic activity, leading to inhibition of PARP-dependent DNA repair and increased formation of PARP-DNA complexes resulting in DNA damage, apoptosis, and cell death [73]. Palbociclib, approved in 2015, is an inhibitor of cyclin-dependent kinases (CDK) 4 and 6, leading to reduced retinoblastoma (Rb) protein phosphorylation and inhibition of cell cycle progression [74]. Lenvatinib mesylate, approved in 2015, inhibits vascular endothelial growth factor (VEGF) receptors VEGFR1 (FLT1), VEGFR2 (KDR), and VEGFR3 (FLT4), fibroblast growth factor (FGF) receptors FGFR1, 2, 3, and 4, platelet-derived growth factor receptor alpha (PDGFRα), KIT, and RET, leading to inhibition of tumor angiogenesis and tumor cell proliferation [75] Osimertinib mesylate, approved in 2015, inhibits the epidermal growth factor receptor (EGFR) by irreversibly binding to select EGFR mutations, including T790M, L858R, and exon 19 deletions, leading to inhibition of tumor cell proliferation [76]. Alectinib hydrochloride, approved in 2015, inhibits anaplastic lymphoma kinase (ALK) and RET, leading to inhibition of tumor cell proliferation [77]. Venetoclax, approved in 2016, is a selective inhibitor of the anti-apoptotic protein BCL-2, leading to apoptosis in cells that overexpress BCL-2 [78]. Brigatinib, approved in 2017, inhibits anaplastic lymphoma kinase (ALK), ROS1, insulin-like growth factor-1 receptor (IGF-1R), and FLT-3, leading to inhibition of tumor cell proliferation [79]. Abemaciclib, approved in 2017, is an inhibitor of cyclin-dependent kinases (CDK) 4 and 6, leading to reduced retinoblastoma (Rb) protein phosphorylation and inhibition of cell cycle progression [80]. Ribociclib succinate, approved in 2017, is an inhibitor of cyclin-dependent kinases (CDK) 4 and 6, leading to reduced retinoblastoma (Rb) protein phosphorylation and inhibition of cell cycle progression [81]. Midostaurin, approved in 2017, inhibits multiple receptor tyrosine kinases, including FLT3, KIT, PDGFR-α, PDGFR-β, and VEGFR2, leading to inhibition of tumor cell proliferation [82]. Acalabrutinib, approved in 2017, inhibits Bruton's tyrosine kinase (BTK), leading to inhibition of B-cell proliferation [83]. Lorlatinib, approved in 2018, inhibits anaplastic lymphoma kinase (ALK) and ROS1, leading to inhibition of tumor cell proliferation [84]. Larotrectinib sulfate, approved in 2018, is a selective inhibitor of tropomyosin receptor kinases (TRK) TRKA, TRKB, and TRKC, leading to inhibition of tumor cell proliferation [85]. Talazoparib tosylate, approved in 2018, is a poly(ADP-ribose) polymerase (PARP) inhibitor that inhibits PARP enzymatic activity, leading to inhibition of PARP-dependent DNA repair and increased formation of PARP-DNA complexes resulting in DNA damage, apoptosis, and cell death [86]. Erdafitinib, approved in 2019, is a kinase inhibitor that binds to and inhibits enzymatic activity of fibroblast growth factor receptors (FGFR) 1, 2, 3, and 4, leading to inhibition of tumor cell proliferation [87]. Alpelisib, approved in 2019, is a phosphatidylinositol-3-kinase (PI3K) inhibitor that targets the PI3Kα isoform, leading to inhibition of the PI3K/AKT/mTOR pathway and tumor cell proliferation [88]. Selinexor, approved in 2019, is a selective inhibitor of nuclear export (SINE) that blocks exportin 1 (XPO1), leading to accumulation of tumor suppressor proteins in the cell nucleus and inhibition of tumor cell proliferation [89]. Entrectinib, approved in 2019, is a kinase inhibitor that targets anaplastic lymphoma kinase (ALK), ROS1, and neurotrophic tyrosine receptor kinase (NTRK) gene fusions, leading to inhibition of tumor cell proliferation [90]. Zanubrutinib, approved in 2019, inhibits Bruton's tyrosine kinase (BTK), leading to inhibition of B-cell proliferation [91]. Tazemetostat, approved in 2020, is an EZH2 inhibitor that blocks the enzymatic activity of EZH2, leading to reduced levels of H3K27 trimethylation and altered gene expression, including genes involved in cell cycle regulation, and inhibition of tumor cell proliferation [92].

Pemigatinib, approved in 2020, is a kinase inhibitor that targets fibroblast growth factor receptors (FGFR) 1, 2, and 3, leading to inhibition of tumor cell proliferation [93]. Capmatinib hydrochloride, approved in 2020, is a kinase inhibitor that targets MET, including the mutant variant produced by exon 14 skipping, leading to inhibition of tumor cell proliferation [94]. Selpercatinib, approved in 2020, is a kinase inhibitor that targets RET, leading to inhibition of tumor cell proliferation [95]. Pralsetinib, approved in 2020, is a kinase inhibitor that targets RET, leading to inhibition of tumor cell proliferation [96]. Lurbinectedin, approved in 2020, binds to guanine residues in the minor groove of DNA, leading to inhibition of the nucleotide excision repair pathway, double-strand breaks, and apoptosis [97]. Tucatinib, approved in 2020, is a kinase inhibitor that selectively inhibits human epidermal growth factor receptor 2 (HER2), leading to inhibition of tumor cell proliferation [98]. Avapritinib, approved in 2020, is a kinase inhibitor

that targets platelet-derived growth factor receptor alpha (PDGFRA) and KIT, including wild-type and mutant isoforms, leading to inhibition of tumor cell proliferation [99]. Ripretinib, approved in 2020, is a kinase inhibitor that targets KIT, including wild-type and mutant isoforms, and platelet-derived growth factor receptor alpha (PDGFRA), leading to inhibition of tumor cell proliferation [100]. Abemaciclib, approved in 2021, is an inhibitor of cyclin-dependent kinases (CDK) 4 and 6, leading to reduced retinoblastoma (Rb) protein phosphorylation and inhibition of cell cycle progression [101]. Umbralisib tosylate, approved in 2021, is a kinase inhibitor that targets phosphoinositide 3-kinase delta (PI3Kδ) and casein kinase 1 epsilon (CK1ε), leading to inhibition of tumor cell proliferation [102]. Tepotinib hydrochloride hydrate, approved in 2021, is a kinase inhibitor that targets MET, including the mutant variant produced by exon 14 skipping, leading to inhibition of tumor cell proliferation [103]. Infigratinib phosphate, approved in 2021, is a kinase inhibitor that targets fibroblast growth factor receptors (FGFR) 1, 2, and 3, leading to inhibition of tumor cell proliferation [104]. Sotorasib, approved in 2021, is a KRAS G12C inhibitor that irreversibly binds to KRAS G12C, locking it in an inactive state and inhibiting downstream signaling and tumor cell proliferation [105]. Belzutifan, approved in 2021, is a hypoxia-inducible factor-2α (HIF-2α) inhibitor that binds to and inhibits HIF-2α, leading to reduced transcription of HIF-2α target genes involved in cellular proliferation, angiogenesis, and tumor growth [106]. Mobocertinib succinate, approved in 2021, is a kinase inhibitor that targets epidermal growth factor receptor (EGFR) exon 20 insertion mutations, leading to inhibition of tumor cell proliferation [107]. Asciminib hydrochloride, approved in 2021, is a BCR-ABL1 tyrosine kinase inhibitor that binds to the ABL myristoyl pocket, leading to inhibition of BCR-ABL1 and tumor cell proliferation [108]. Pirtobrutinib, approved in 2023, is a highly selective, non-covalent Bruton's tyrosine kinase (BTK) inhibitor that inhibits wild-type and C481-mutated BTK, leading to inhibition of B-cell proliferation and survival [109]. Adagrasib, approved in 2023, is a KRAS G12C inhibitor that irreversibly binds to KRAS G12C, locking it in an inactive state and inhibiting downstream signaling and tumor cell proliferation [110]. Ziftomenib, approved in 2024, is a menin-KMT2A inhibitor that binds to menin, disrupting the menin-KMT2A interaction and inhibiting KMT2A-dependent gene expression and tumor cell proliferation [111]. Futibatinib, approved in 2024, is a kinase inhibitor that targets fibroblast growth factor receptors (FGFR) 1, 2, 3, and 4, leading to inhibition of tumor cell proliferation [112].

2.4. Targeted therapy

The development of anti-cancer drugs has been a major focus of pharmaceutical research and development efforts over the past several decades. Since the approval of mechlorethamine in 1949, numerous classes of anti-cancer drugs have been developed, targeting various aspects of cancer cell biology [113]. These drugs have significantly improved treatment outcomes and quality of life for cancer patients [114].

Table 2. FDA-Approved Targeted Therapies for Specific Cancer Types

Drug Name	Target	Cancer Type	Year of Approval
Trastuzumab	HER2	Breast Cancer	1998
Imatinib	BCR-ABL, KIT, PDGFR	Chronic Myeloid Leukemia, Gastrointestinal Stromal Tumors	2001
Gefitinib	EGFR	Non-Small Cell Lung Cancer	2003
Bevacizumab	VEGF	Colorectal Cancer, Non-Small Cell Lung Cancer, Glioblastoma, Renal Cell Carcinoma	2004
Erlotinib	EGFR	Non-Small Cell Lung Cancer, Pancreatic Cancer	2004
Sorafenib	VEGFR, PDGFR, RAF	Renal Cell Carcinoma, Hepatocellular Carcinoma, Thyroid Cancer	2005
Sunitinib	VEGFR, PDGFR, KIT, FLT3	Renal Cell Carcinoma, Gastrointestinal Stromal Tumors, Pancreatic Neuroendocrine Tumors	2006
Lapatinib	HER2, EGFR	Breast Cancer	2007
Vemurafenib	BRAF	Melanoma	2011
Crizotinib	ALK, ROS1	Non-Small Cell Lung Cancer	2011
Olaparib	PARP	Ovarian Cancer, Breast Cancer, Prostate Cancer	2014

The FDA approval process for anti-cancer drugs is rigorous and involves multiple stages, including preclinical testing, clinical trials, and regulatory review [115]. This process ensures that approved drugs are safe and effective for their intended use [116]. The approval of new anti-cancer drugs has accelerated in recent years, with numerous approvals in the past decade alone [117].

Despite the significant progress made in the development of anti-cancer drugs, there are still many challenges to be addressed [118]. Cancer is a complex and heterogeneous disease, and the development of effective therapies requires a deep understanding of the underlying biology and mechanisms of drug resistance [119]. Additionally, the high cost of drug development and the need for personalized therapies present significant challenges for the pharmaceutical industry [120]

3. Conclusion

In conclusion, the development of FDA-approved anti-cancer drugs has been a major focus of pharmaceutical research and development efforts over the past several decades. These drugs have significantly improved treatment outcomes and quality of life for cancer patients. However, there are still many challenges to be addressed in the development of effective and affordable therapies for cancer. Continued research and innovation in the field of anti-cancer drug development will be critical for improving patient outcomes and reducing the burden of cancer worldwide.

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