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Review

Drug Repurposing in Cancer Therapy: A Systematic Review

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Abstract

Cancer is the second leading cause of death globally. Despite the intensive research, the success rate for new anticancer drug development remains below 10%, rendering conventional therapies alone insufficient to reduce cancer burden and mortality. Drug re-purposing offers an alternative way of identifying new therapeutic uses for approved drugs. This approach offers a shorter development timeline, lower costs, and a higher likelihood of clinical success. This systematic review aims to summarize all drugs repurposed against cancer to date comprehensively. Following PRISMA (Preferred Reporting Items for Systematic Review and Meta-Analysis) guidelines, we reviewed 45 publications indexed in PubMed from January 2015 to December 2024. Studies focusing on drug re-purposing in oncology were included. Data were extracted on drug name, original therapeutic class, responsive cancer types, and mechanisms of action. Additionally, annual publication trends were analyzed, and findings were presented in an accessible tabular format. A total of 181 repurposed drugs, belonging to approximately 35 distinct pharmacological classes, were identified as having anticancer activity against 28 different cancer types. With few exceptions, the annual number of publications on cancer drug re-purposing increased over the decade. Each repurposed drug is summarized by its original use, responsive cancers, and the underlying mechanism of action. This study also analyzes the distribution of cancers addressed by this approach. Finally, the study discusses current challenges and prospects for re-purposing drugs as standard cancer therapy.

Keywords: Anticancer Drugs, Cancer, Drug Repurposing, PRISMA

1. Introduction

Drug repurposing, also referred to as drug repositioning, is not a new concept. Several well-known drugs have been repurposed for different diseases in the last few decades. For example, thalidomide, once infamous for causing congenital disabilities, was repurposed and approved for the treatment of multiple myeloma [1]. Similarly, sildenafil, initially developed as a cardiovascular drug, was later approved for treating erectile dysfunction under the name Viagra [2]. These successes have paved the way for an overwhelmingly growing interest in repurposing 'non-oncology' drugs for cancer treatment. In response to these challenges, repurposing drugs has become a viable and economical way to accelerate the development of novel cancer therapeutics. Drug repurposing involves finding new therapeutic uses for existing drugs initially developed to treat conditions unrelated to cancer [3]. In oncology, where time is critical and the failure rate of new drugs in clinical trials is high, repurposing offers a valuable shortcut to potentially effective treatments [4]. Cancer is a significant threat to human health globally, accounting for the leading cause of death. According to the World Health Organization, cancer caused approximately 10 million deaths in 2020, making it the second largest cause of death worldwide [5]. Many patients eventually develop resistance to the conventional treatments, experience severe side effects, or face exorbitant costs associated with treatment using these novel drugs [6]. Drug repurposing is appealing because it might take over a decade and cost up to $2.6 billion to develop a new medication from discovery to market [7]. On the other hand, repurposing drugs can significantly shorten the period by allowing researchers to skip early-stage preclinical testing and move directly to advanced-stage clinical trials. Since the safety profiles of these drugs are already well-established, the focus shifts to determining their efficacy for the new indication, i.e., cancer [4]. Probable mechanisms by which repurposed drugs exert their anticancer effects are varied and depend on the biological pathways they target. One significant advantage of drug repurposing is that many non-oncology drugs target pathways involved in processes like cell proliferation, apoptosis, angiogenesis, and immune response—key factors in cancer development and progression [8]. For instance, the widely used antidiabetic drug metformin has shown potential in cancer treatment due to its ability to inhibit the mammalian target of rapamycin (mTOR) and activate AMP-activated protein kinase (AMPK), pathways involved in cancer cell metabolism [9].

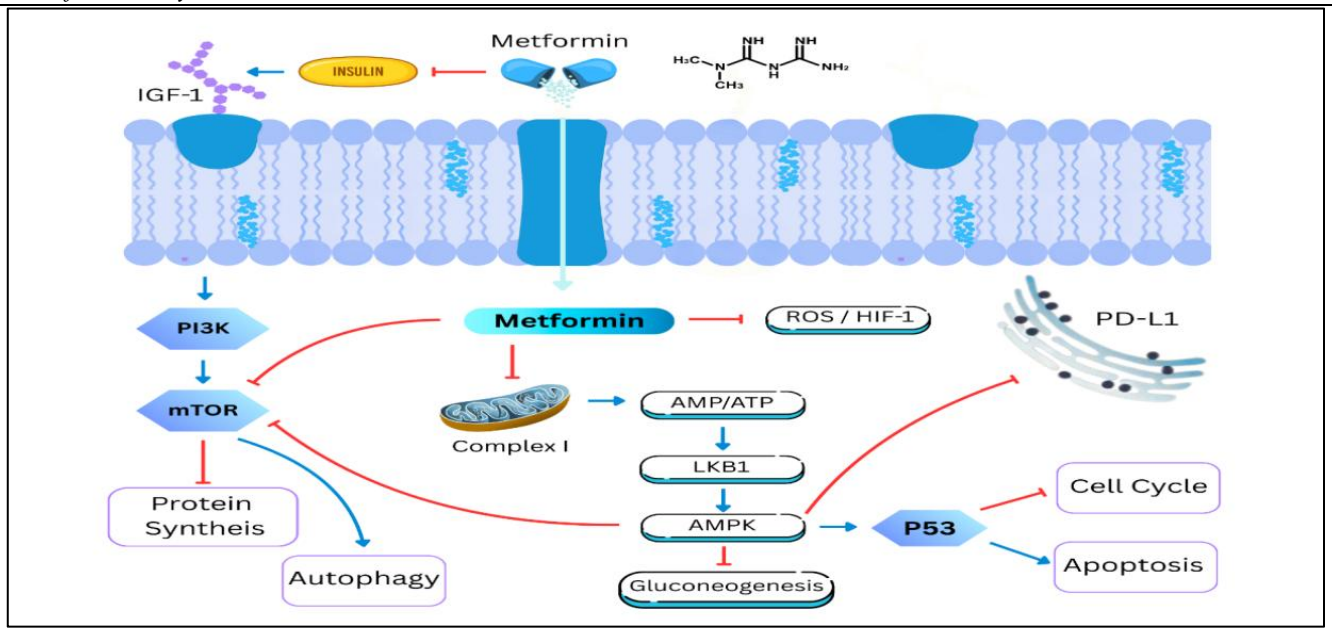

Figure 1. Overview of cellular mechanisms of metformin in cancer therapy.

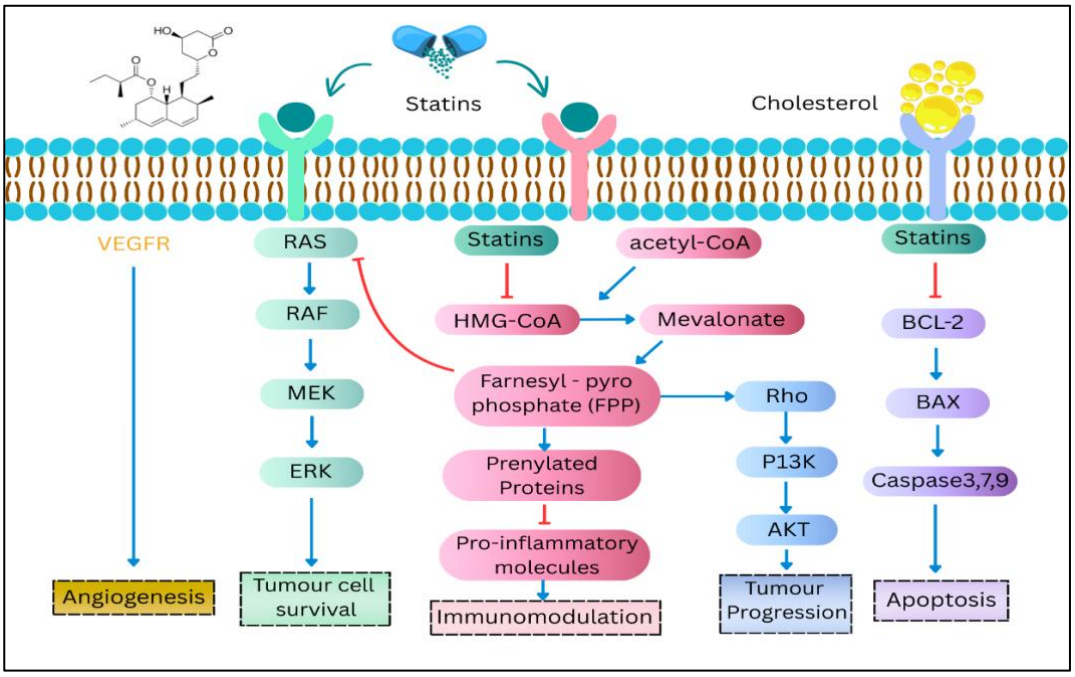

Figure 2. Overview of cellular mechanisms of statins in cancer treatment.

Metformin is one of the most studied examples of drug repurposing in oncology. Originally prescribed to manage type 2 diabetes, metformin has anticancer properties, particularly in breast, colon, and pancreatic cancers [9].

Its anticancer effects stem from its role in reducing insulin levels and improving insulin sensitivity, thereby decreasing the availability of insulin, which can promote cancer growth. Such effect of insulin, as shown in figure 1, is mediated via its binding with both the insulin receptor (IR) and insulin-like growth factor-1 receptor (IGF-1R) in cancer cells [10]. Additionally, metformin can directly target cancer cells by inhibiting mitochondrial respiration and reducing energy production, leading to reduced tumor growth [11]. Researchers are conducting numerous clinical trials to assess the effectiveness of metformin in combination treatments for different types of cancer [12].

Another class of drugs that has sought attention in cancer repurposing is statins, which lower the plasma cholesterol levels. Statins inhibit the enzyme HMG-CoA reductase, a key player in the mevalonate pathway, which is critical for the biosynthesis of cholesterol and other important molecules involved in cell membrane integrity and cell signaling. Cancer cells often exploit this pathway to support their rapid growth and division [13]. By inhibiting this

pathway, statins can reduce the proliferation of cancer cells and induce apoptosis (Figure 2). Studies have found that statins may be particularly effective in cancers with dysregulated lipid metabolism, such as breast and prostate cancers[14]. One of the major advantages of drug repurposing in cancer is the ability to expedite the clinical trial process. As a result, they can often proceed directly to Phase II or III clinical trials in their new indication, saving both time and resources [15]. Moreover, repurposed drugs often come with well-understood mechanisms of action and well-documented side effect profiles which can help oncologists better anticipate how the drug molecule will interact with cancer cells and predict any potential adverse effects when combined with existing cancer therapies. For instance, repurposing drugs like metformin and statins, provide a level of confidence in their safety that is not always present with novel oncology drugs [8].

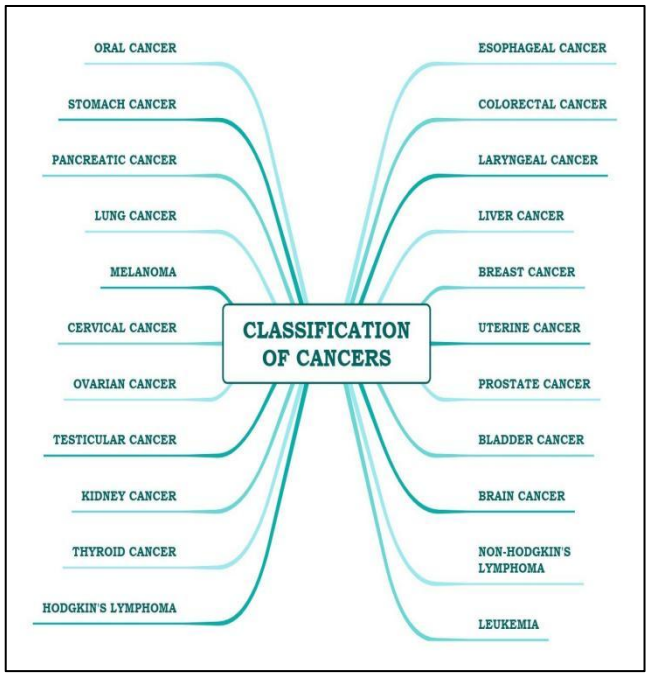

Figure 3. Classification of cancers.

Cancer is a highly heterogeneous disease, and drugs that work well in one cancer type may not be effective in others. Furthermore, the optimal dosing and scheduling of repurposed drugs combined with standard cancer treatments must be carefully evaluated in clinical trials [15]. Of all human diseases, cancer has the most significant clinical, social, and financial cost in terms of cause-specific Disability-Adjusted Life Years (DALYs). This review shows the analysis of 22 types of cancers illustrated in figure 3. Of the 18 million new cases reported in 2018, the most common malignancies are lung (2.09 million cases), breast (2.09 million cases), and prostate (1.28 million cases). It will likely become the number one leading (~18.63 million deaths) cause of death in 2060 [16].

Oral cancer, a squamous cell carcinoma (OSCC) that arises on the lip or oral cavity, poses different levels of differentiation and a propensity for lymph node metastasis [16]. Esophageal cancer can be broadly categorized into two types: squamous cell carcinomas, which usually start in the epithelial lining of the upper and middle section of the esophagus, while adenocarcinomas develop in the cells of the lower section [17].

Approximately 90% of all stomach cancers are adenocarcinomas, while other types, including lymphoma, sarcoma, and neuroendocrine tumors, are rare [18]. Determinants like a western lifestyle, consumption of red meat, alcohol, obesity, and inflammatory bowel disease are all associated with colorectal cancer. The two main hereditary disorders that can lead to cancer are FAP and HNPCC [19]. Adenocarcinoma is the most common type of pancreatic cancer. Important risk factors like smoking, positive family history, genetics, diabetes mellitus, obesity, dietary factors, and alcohol use are associated with pancreatic cancer, the most lethal malignant neoplasm across the world [20]. Cancer of the larynx usually tends to be contained within the primary structures of the larynx. Several pathways allow an extrinsic tumor to spread without invading the thyroid cartilage. Moreover, the tumors can spread directly through the cartilage of the larynx [21]. Another type of cancer that is heavily linked to smoking is lung cancer, especially in men. It is the most common type of cancer, hence leading in terms of mortality rates worldwide. Non-small cell lung cancer (including adenocarcinoma and squamous carcinoma) comprises ~85% of cases, and targeted mutations (EGFR, ALK, etc.) are important in subsets [22]. There are two types of liver cancer: primary tumors and secondary metastatic malignancies. The main kinds of liver cancer are carcinoma, cholangiocarcinoma, and sarcoma [23]. Unlike other cancers, melanoma largely depends on distinct genetic alterations and UV exposure. Mutations influence melanoma development in the CDKN2A and MAPK pathways [24]. For women, the second leading cause of cancer death is breast cancer, which involves a multi-step tumorigenesis process involving multiple cell types. Its prevention remains challenging across the world and can commonly spread to distant organs such as the bone, liver, lung, and brain due to its metastatic nature. However, early detection (mammography) yields high survival; advanced disease can still often be managed chronically [25]. Research shows that the primary cause behind cervical cancer is persistent HPV infections, where approximately 20 percent of cervical cancer cases are adenocarcinomas, and the remaining 80 percent are carcinomas [26]. Conversely, most of the uterine cancer is endometrial cancer. This type of cancer is linked to variables. Endometrial tumors caused by an imbalance in estrogen make up around 90 percent of cancers [27]. There are different kinds of ovarian cancer. The disease is often discovered after it has spread. The common sub-type of ovarian cancer is high-grade serous carcinoma [28]. Currently, uterine cancer is the most common type of invasive gynecological malignancy affecting females in the United States, where most of the cancer is endometrial cancer. Endometrial tumors caused by an imbalance in oestrogen make up around 90 percent of cancers [27]. Ovarian cancer is another type of cancer that can be differentiated into five subtypes based on their unique identifiable risk factors: cellular origins, molecular

compositions, clinical manifestations, and therapeutic approaches. The disease is often discovered after it has spread. The common sub type of ovarian cancer is high-grade serous carcinoma [28]. Prostate carcinoma is a common malignancy in men and a major cause of cancer-related mortality worldwide, often remaining asymptomatic in the early stage and progressing indolently, although advanced disease may present with urinary symptoms and skeletal metastasis, and PSA elevation is frequently used as a diagnostic indicator [29]. Testicular cancer primarily affects young adult males, especially in high-income countries, where the primary source of cancer is germ cells. Age, cellular lineage, and histology are used to categorize germ cell cancers [30]. Exposure to carcinogens, such as smoking, can increase the risk of bladder cancer. Most cases of bladder cancer are initially non-invasive. However, it can manifest as either muscle-invasive or non-muscle-invasive [31]. Kidney cancer is a type of cancer that begins in the kidney. There are types of kidney cancer, like Wilms tumor and renal pelvic tumors, but they are not as common as kidney cancer [32]. Brain cancer affects the nervous system severely as it can cause neurological symptoms, such as headaches, convulsions, altered cognition, and issues with coordination [33, 34]. Thyroid cancer comprises several histological forms arising from follicular or parafollicular cells, with papillary and follicular types being more common and anaplastic and poorly differentiated variants being notably aggressive [35, 36]. Hodgkin's lymphoma (HL) is a B-cell lymphoma characterized by a few malignant cells alongside numerous immune effector cells within the tumor microenvironment. This neoplasm is relatively rare, predominantly affecting young adults, although it can also occur less frequently in older people. The diagnostic process depends on histological and immunohistochemical evaluations of specimens obtained from lymph node biopsies [37]. Compared with Hodgkin's disease, non-Hodgkin lymphomas (NHLs) comprise approximately 5% of head and neck malignancies and display a wide range of appearances, with lymphoid malignant neoplasms showing diverse biological and clinical behaviors. Typically, as the initial course of treatment, patients are administered chemoimmunotherapy, with radiation therapy potentially being incorporated, provided the patients present with early-stage malignancy [38]. Multiplication of blood cells results in leukemia. It comprises ALL, AML, both CLL and CML. Both radiation and genetic factors can increase the risk of leukemia. Diagnosis can be confirmed through analysis of bone marrow specimens or peripheral blood [39].

Drug repurposing represents a promising and innovative strategy in the fight against cancer. By identifying new uses for existing drugs, researchers can capitalize on established safety profiles and reduce the time and cost associated with developing novel cancer therapeutics. Initially developed for non-cancerous conditions, drugs like metformin and statins have demonstrated significant potential in preclinical and clinical studies, offering new hope for patients with difficult-to-treat cancers. As the understanding of cancer biology continues to grow, so will the opportunities to repurpose existing drugs to target critical pathways in cancer cells. With the right support and collaboration, drug repurposing could be crucial in expanding the therapeutic arsenal available to cancer patients and improving outcomes globally. The aim of the study is to summarize all the drugs that have been repurposed against various cancers comprehensively. We also tried to find out their mechanism of action along with the therapeutic classes. And finally, we also analyze the percentages of cancers we addressed by this approach.

2. Materials and methods

A systematic review was done according to the PRISMA guidelines to look at recent studies about using existing drugs for cancer treatment. A systematic review needs a clear research question, finding and assessing relevant studies, and clearly defined results [40].

In this study, 45 research articles from PubMed, published between 2015 and 2024, were chosen to look at how far drug repurposing has come in cancer treatment and what might happen next. The trend showing how many selected studies were published each year is shown in figure 4.

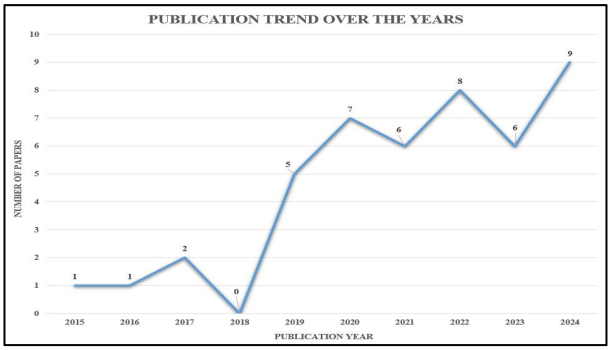

Figure 4. Publication trend over the years.

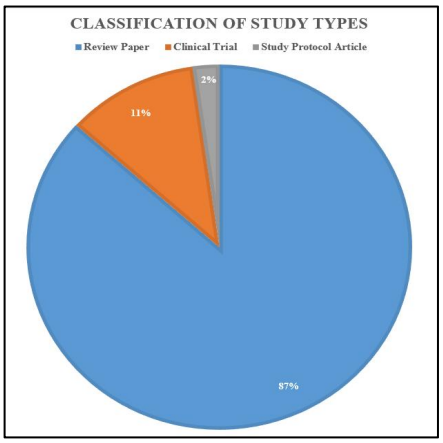

Figure 5. Classification of study types.

The studies that were included were then grouped according to the type of study and the level of the journal they were published in. Most of the studies were review articles, then came clinical trials and study protocols, showing that there was more focus on analyzing existing literature. The different types of studies are shown in figure 5. Additionally, the quality distribution of the selected studies, based on their journal quartile rankings, is shown in Figure 6. In this figure, most of the included publications came from Q1-ranked

journals.

Only articles written in English that were published in journals listed in PubMed were counted as eligible. Both types of studies—qualitative and quantitative—were included to give a full picture of research on repurposing anticancer drugs. The literature search was done using a specific plan in PubMed, as shown in table 1.

Table 1. Advanced search string strategy.

Database	Search strings
PubMed	("Drug repurposing" OR "drug repositioning") AND (cancer* OR chemotherapy* OR "cancer treatment*" OR "cancer therapy*")

The process of choosing the studies included managing the data, checking each study, and collecting the necessary information. Research questions were created to help guide the study's goals and to carefully review the chosen articles [41].

The research questions looked at in this review are listed in table 2. The studies were checked using specific rules for including and excluding them, and the search was done on PubMed using Boolean terms, as shown in table 3.

During the screening process, articles that were duplicates, studies that were still in progress, and studies that did not meet the required criteria were excluded. The detailed process for selecting studies, based on PRISMA guidelines, is shown in figure 7.

First, 1,159 articles were removed during the initial review. Then, the abstracts were checked, and after that, the full texts were examined. This process led to the selection of 45 studies for the final analysis.

A standard way of gathering data was used to reduce bias and keep the results from the chosen studies consistent. Important details about how the study was set up, the methods used, and the drugs that were originally meant for cancer treatment were gathered and examined. The number of studies chosen at various stages of the screening process is shown in table 4.

Table 2. Preferred research questions investigated in this study.

Q. No.	Research question	Description
Rq1	What is the overall status of drug repurposing in cancer therapy?	This research will lead to identifying the overall progression in drug repositioning in cancer therapy.
Rq2	How many drugs have been repurposed so far?	This question aims to quantify the total amount of drugs that have been repurposed for various cancers.
Rq3	Which cancers have been addressed, and which cancers have not?	This research question aims to identify the cancers that have been addressed as well as those that have not been addressed for drug repurposing.
Rq4	What kinds of studies are used for drug repositioning?	This question will explain the methodologies of the research being carried out for the drug repurposing.
Rq5	In which countries the research is going on mostly?	The goal of this query is to understand the geographical status for drug repurposing.

Table 3. Lists of inclusion and exclusion criteria.

Area specification	Inclusion criteria	Exclusion criteria
Database	IC1. PubMed	EC1. Other journals except predatory journals and journals mentioned “inclusion Criteria”
Journal/Conference type	IC2. Research article	EC2. Book, Chapter, etc.
Keywords on search engine	IC3. Drug repurposing, anticancer drug, chemotherapy	EC3. keywords excluding “Inclusive Criteria”
Area of Interest	IC4. Cancer	EC4. Area excluding “Inclusive Criteria”
Language	IC5. English	EC5. Language except English
Selected time period	IC6. 2015-2025	EC6. Before 2015

Table 4. Selected studies according to different digital libraries in different stages.

Digital library	Selected research articles	Screening	Included research articles
PubMed	3756	2597	45

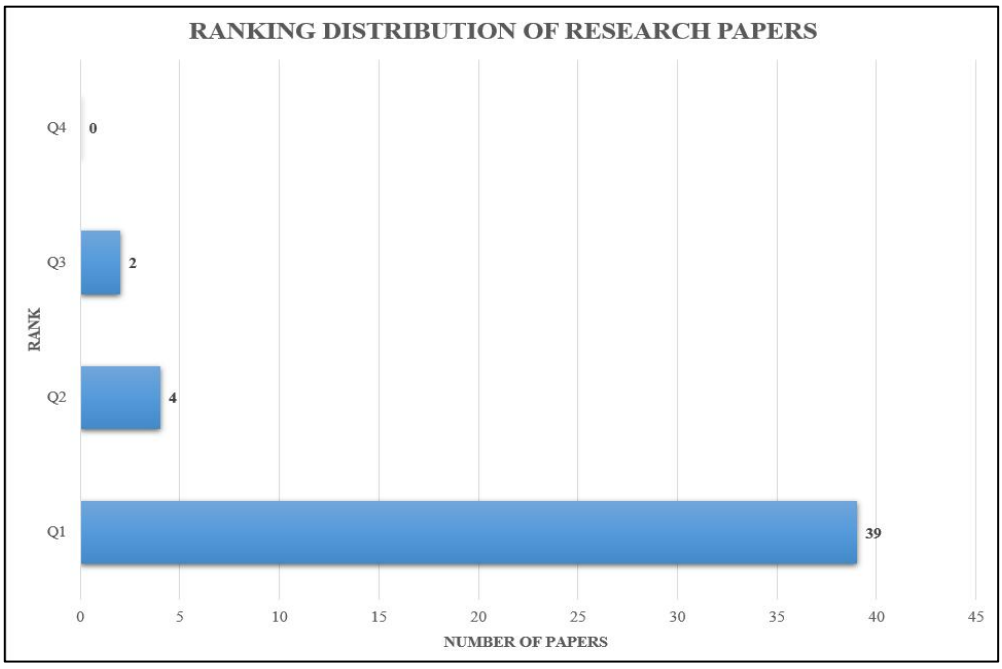

Figure 6. Ranking distribution of research papers.

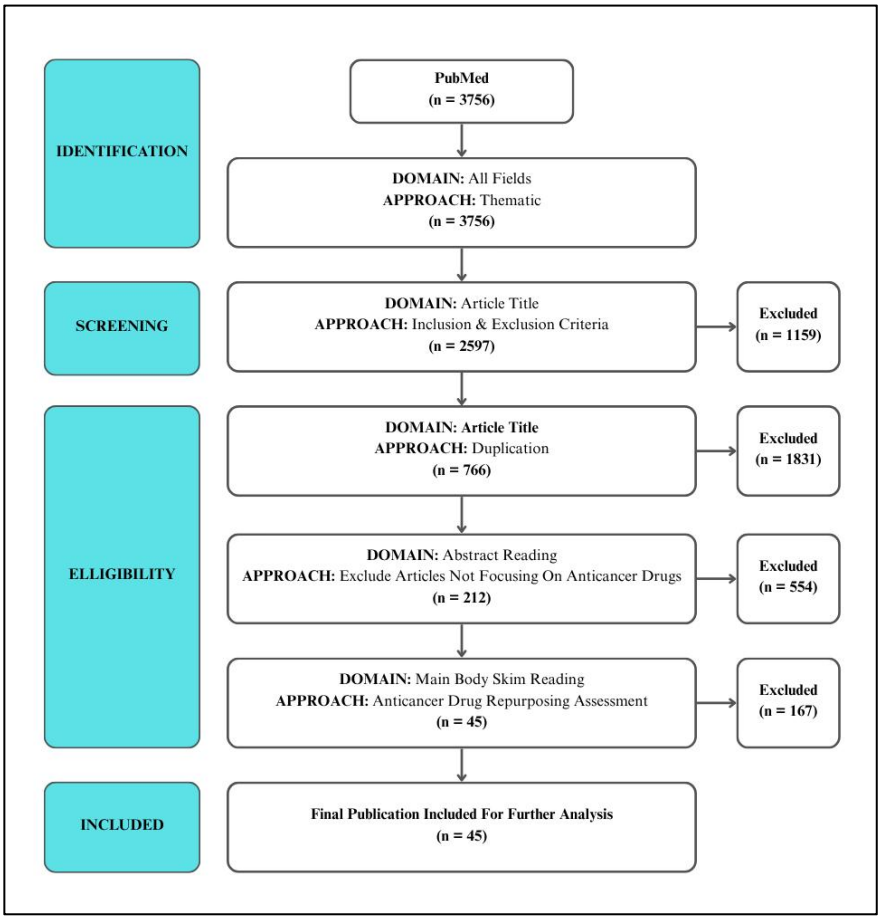

Figure 7. Flow diagram of the literature search process using PRISMA guidelines.

3. Results and Discussion

According to global statistics, more than 20 million people will be diagnosed with cancer in 2025. Malignancies with advanced stages, such as colorectal, prostate, and breast cancer, that are often incurable with current treatments, will contribute significantly to this increase [8]. The clinical efficacy of pharmacotherapy is frequently limited by drug resistance and severe toxicity or adverse effects; therefore, there is still a crucial need to create innovative cancer medicines. In recent years, one potential method that has gotten extensive attention is medication repurposing: identifying new applications for existing, clinically authorized pharmaceuticals [42]. This systematic review will represent data on drug repurposing in cancers.

3.1. Search results

A detailed search flow and process diagram has been illustrated in the figure 7. From 1 database, we selected a total of 3756 articles. After excluding 1159 articles based on inclusion and exclusion criteria, we screened 2597 articles for the next phase. After excluding the duplicates, and via reading the abstract and full-text skimming, we finally included 45 articles for further analysis. Based on the first author's country, we can conclude that most of the research has been conducted on China, the USA, India, Australia, England etc., as represented in figure 8.

3.2. Conventional treatment of cancer

Cancer is not a single disease. Instead, it is a group of diseases. Table 5 represents the types of cancers and their conventional treatment and medication. The information has been retrieved based on the National Cancer Institute (2025).

During the review, we have retrieved approximately 181 drugs which were repurposed against 28 types of cancer in total. In the case of the drugs classification, we found around 35 classes of drugs which have been repurposed. The following figures (Figure 9 and Figure 10) show the result at a glance.

With 23 documented cases, the Antidiabetic pharmacological class exhibits the highest frequency of pharmacological repurposing, as seen in Figure 9. This indicates a strong desire to investigate novel uses for currently available diabetic drugs. Antihyperlipidemic medications and antibiotics are two more well-known pharmacological families for repurpose, with 20 and 19 cases, respectively. Together, these top three classes represent a significant share of all drug repurposing initiatives, according to the cumulative percentage line

Figure 10 shows that, with 28 citations, breast cancer is the most commonly mentioned cancer type in medication repurposing research. This suggests a considerable emphasis on developing novel pharmacological applications for treating breast cancer. With 24 and 17 mentions, respectively, colorectal and prostate cancer are also mentioned frequently. The graph shows various repurpose initiatives for different cancer types, including at least one mention for a few less prevalent cancers.

In this systematic review, we tried to identify all the repurposed drugs that have been used to treat various cancers. We have arranged 181 drugs in total based on the cancers they have been used against. For example, Table 6 and Table 7 will represent drugs that have been repurposed for breast and colorectal cancer, respectively. The remaining data (Tables S1-S18) are provided in the supplementary files section. In the Table, we listed their therapeutic classes and key mechanisms of action, including how they control cancer. We have also summarized the general mechanisms by which repurposed drugs act to control cancer.

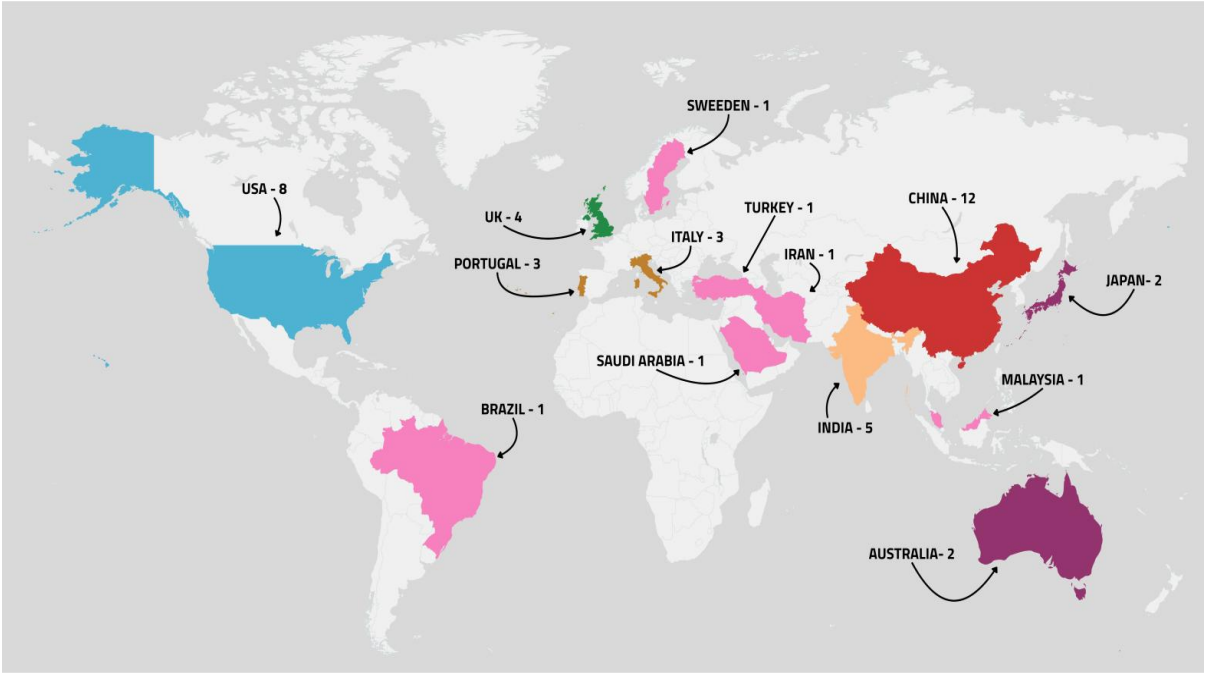

Figure 8. Geographically colored mapping of 45 research articles in the review.

Table 5. Various cancers and their conventional treatment.

Types of cancer	Conventional treatment	Anticancer drugs
Breast cancer	chemotherapy, surgery, radiotherapy	doxorubicin, cyclophosphamide
Lung cancer	targeted therapy, surgery, chemotherapy	etoposide, cisplatin
Prostate cancer	hormone therapy, radiotherapy, surgery	docetaxel, leuprolide
Colorectal cancer	surgery, radiotherapy, chemotherapy	5-fluorouracil, capecitabine, oxaliplatin
Pancreatic cancer	surgery, chemotherapy	5-fluorouracil, folfirinox, gemcitabine
Liver cancer	surgery, chemotherapy, liver transplant	regorafenib, sorafenib, atezolizumab
Leukemia	stem cell transplant, chemotherapy	methotrexate, cytarabine, imatinib
Ovarian cancer	targeted therapy, surgery, chemotherapy	paclitaxel, carboplatin, bevacizumab
Cervical cancer	chemotherapy, surgery, radiotherapy	cisplatin, paclitaxel, bevacizumab
Esophageal cancer	chemotherapy, surgery, radiotherapy	5-fluorouracil, cisplatin, paxlitaxel
Bladder cancer	surgery, chemotherapy, immunotherapy	cyclophosphamide, gemcitabine
Head and neck cancer	chemotherapy, surgery, radiotherapy	cisplatin, docetaxel, cetuximab
Thyroid cancer	chemotherapy, surgery, radiotherapy	levothyroxine, doxorubicin, sorafenib
Kidney cancer	targeted therapy, surgery, immunotherapy	sorafenib, nivolumab, sunitinib
Multiple myeloma	stem cell transplant, chemotherapy	lenalidomide,bortezomib, dexamethasone
Melanoma	surgery, immunotherapy, chemotherapy	nivolumab, dabrafenib, pembrolizumab
Non-hodgkin lymphoma	immunotherapy, chemotherapy	rituximab, vincristine, cyclophosphamide
Hodgkin lymphoma	radiotherapy, chemotherapy	bleomycin, doxorubicin, vinblastine
Testicular cancer	surgery, chemotherapy	bleomycin, etoposide, cisplatin
Sarcoma	chemotherapy, surgery, radiotherapy	doxorubicin, methotrexate, ifosfamide
Brain cancer	chemotherapy, surgery, radiotherapy	carmustine, bevacizumab, temozolomide
Gallbladder cancer	chemotherapy, surgery, radiotherapy	cisplatin, gemcitabine, fluorouracil
Bile duct cancer	surgery, chemotherapy	cisplatin, oxaliplatin, gemcitabine
Skin cancer (basal cell)	chemotherapy, surgery, radiotherapy	imiquimod, fluorouracil
Skin cancer (squamous cell)	chemotherapy, surgery, radiotherapy	5-fluorouracil, methotrexate, cisplatin
Chronic myelogenous leukemia	targeted therapy, chemotherapy	imatinib, nilotinib, dasatinib
Acute lymphocytic leukemia	stem cell transplant, chemotherapy	methotrexate, asparaginase, vincristine
Chronic lymphocytic leukemia	targeted therapy, chemotherapy	cyclophosphamide,rituximab, fludarabine

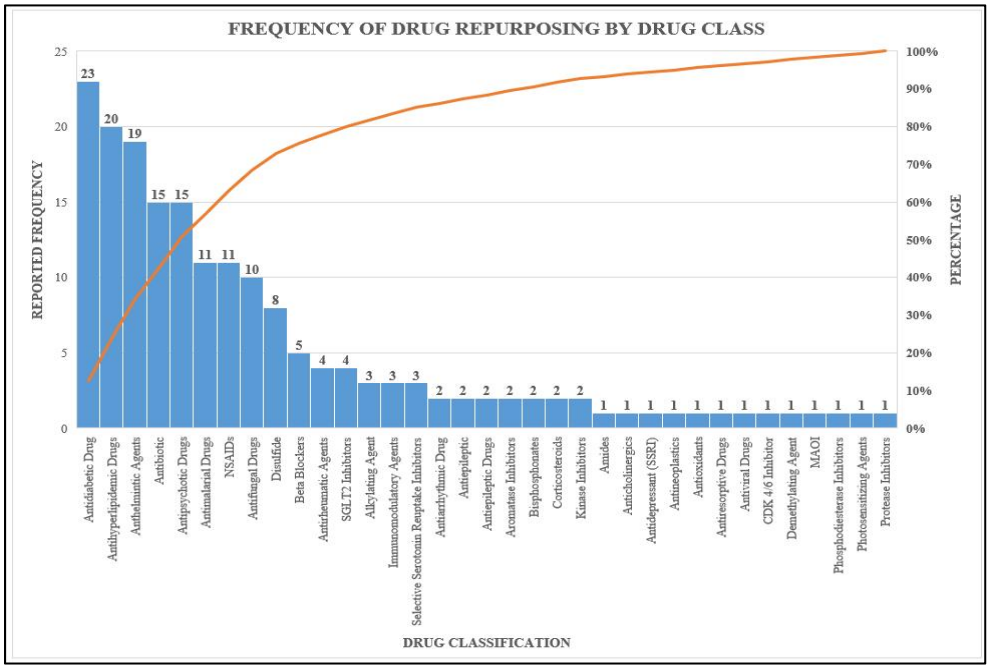

Figure 9. Frequency of drug repurposing by drug class.

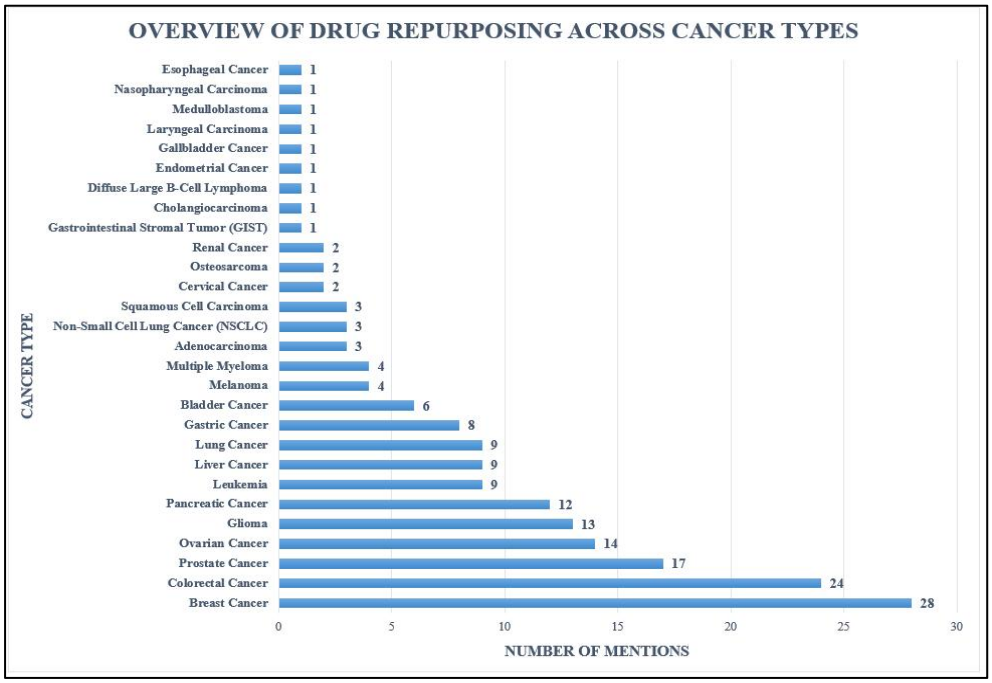

Figure 10. Overview of drug repurposing across cancer types.

4. General mechanisms by which repurposed drugs exert anticancer effects

Research into drug repurposing has uncovered many ways in which non-oncology approved drugs impact tumor growth, cancer cell death, and metastasis. Non- oncology drugs are clinically approved and have mechanisms that are not specific to cancer types. Instead, they work on universal biological mechanisms that are disturbed in many cancer types.

4.1. Modulation of oncogenic signaling pathways

The aberrant activation of intracellular signaling pathways is a defining hallmark of cancer, and many repurposed drugs exert anticancer effects by targeting these pathways at one or more nodes.

4.1.1. PI3K/AKT/mTOR pathway

The phosphatidylinositol-3-kinase (PI3K)/AKT/mammalian target of rapamycin (mTOR) signaling axis is one of the most

Table 6. Repurposed drugs along with their mechanism in breast cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Doxorubicin	antibiotic	DNA intercalation	[43]
Sildenafil	phosphodiesterase inhibitors	increases the level of cyclic guanosine monophosphate (cGMP), which inhibits tumor growth, induces apoptosis, and potentially enhances the effects of other cancer treatments.	[44]
Thalidomide	immunomodulatory agents	regulates cytokine secretion, enhancing the host's immune response and inhibiting the growth of blood vessels that nourish cancer cells.	[44]
Cyclophosphamide	alkylating agent	The active metabolite (phosphoramide) alkylates DNA, leading to intra- and interstrand cross-links, inhibiting DNA replication and transcription.	[45]
Thiotepa	alkylating agent	interference with DNA replication and RNA transcription ultimately lead to the disruption of nucleic acid function.	[45]
Palbociclib	CDK 4/6 inhibitor	blocks the activity of CDK4/6 in cancer cells, thereby preventing them from progressing through the G1 phase of the cell cycle and dividing. Inhibit the binding of estradiol to ER.	[45]
Tamoxifen	aromatase inhibitors	blocks estrogen binding in breast cancer cells, inhibiting their growth and proliferation.	[45]
Exemestane	aromatase inhibitors	blocks aromatase, responsible for converting androgens into estrogen, which helps to inhibit the growth and spread of (ER+) breast cancer cells.	[45]
Docetaxel	antineoplastics	inhibiting microtubule dynamics, Docetaxel leads to cell cycle arrest in the G2/M phase and ultimately, cell death through apoptosis.	[45]
Edaravone	antioxidants	targets and suppresses the NF-κB pathway ,and activates the Nrf2 pathway.	[46]
Fluoxetine	SSRIs	inhibits various pathways involved in cell proliferation, survival, and invasion, including the AKT/mTOR and NF-κB pathways.	[47]
Fluoroquinolones	antibiotic	targets DNA gyrase and topoisomerase-II, leading to DNA damage, cell cycle arrest, and ultimately apoptosis in cancer cells.	[48]
Atovaquone	anthelmintic agents	Inhibits Complex III, leading to loss of mitochondrial membrane potential, impaired ATP synthesis, and inhibition of Pyrimidine biosynthesis.	[49]
Metformin	antidiabetic drug	Inhibits Complex I, which reduces ATP production, and reduces insulin/IGF-1 levels; activates AMPK pathway, and inhibits cell proliferation.	[45,50]
Statins	antihyperlipidemic drugs	inhibits HMG-CoA reductase, leading to decreased mevalonate levels and reduced cholesterol biosynthesis. Also inhibits isoprenoid metabolites necessary for various cellular functions.	[51]
Chloroquine	antibiotic	It prevents the acidification of lysosomes and hinders the process of autophagy.and inhibits autophagy.	[52]
Canagliflozin	SGLT2 inhibitors	Reduces glucose uptake and inhibits PI3K-Akt/β-catenin pathways.	[53]
Propranolol	beta blockers	enhances the function of infiltrated CD8+ T cells through intensifying glycolysis in T Cells.	[54]
Phenelzine	MAOI	constrains the M2 polarization of TAMs through reducing ROS levels and the activation of JAK-STAT6 signaling.	[54]
Statins	antihyperlipidemic drugs	blocks mevalonate pathway, impairs YAP/TAZ activity; reduces GGPP and RhoA activity, decreases cyclin D1 and increases p27 expression, thereby suppressing proliferation, migration, and invasion, promoting apoptosis, and enhancing the efficacy of PD-1 inhibitors and other therapies.	[55]
Artemisinin	antimalarial drugs	inhibits JNK pathway, triggers autophagy, and apoptosis.	[56]
Penfluridol	antipsychotic drugs	induces apoptosis dependent on ROS by down regulating transcription factors Sp1, Sp3, and Sp4, as well as cMyc; suppresses integrin expression, blocking cell migration; inhibits VEGF signaling, impairing angiogenesis.	[57]
Disulfiram	disulfide	DSF with docetaxel enhances autophagy via ROS production; epidemiological data link DSF use to reduce cancer-specific mortality	[58]
Ivermectin	anthelmintic Agents	restores tamoxifen sensitivity in triple-negative breast cancer cells. Inhibits proliferation, though specific mechanisms are less detailed.	[59]

Table 7. Repurposed drugs along with their mechanism in colorectal cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoxetine	SSRIs	Fluoxetine induces apoptosis by inhibiting various cellular proliferation, survival, and invasion pathways, including the AKT/mTOR and NF-κB pathways. It modulates immune responses and impacts cellular stress pathways, contributing to its anticancer effects.	[60]
Benzimidazoles	anthelmintic agents	Benzimidazoles primarily disrupt tubulin polymerization, which is crucial for cell division and microtubule formation. This mechanism leads to cell cycle arrest, apoptosis, and cancer cell proliferation, migration, and invasion inhibition.	[61]
Fluoroquinolones	antibiotic	fluoroquinolones inhibit key enzymes in DNA replication and cell cycle progression. They target DNA gyrase and topoisomerase-II, leading to DNA damage, cell cycle arrest, and ultimately apoptosis in cancer cells.	[62]
Metformin	Anti-diabetic	inhibits Complex I, which reduces ATP production, and reduces insulin/IGF-1 levels	[49]
Statins	anti-hyperlipidemic	inhibits HMG-CoA reductase crucial for mevalonate production and thus decreases cholesterol biosynthesis. Also inhibit isoprenoid metabolites crucial for cellular functions	[51]
Canagliflozin	SGLT2 inhibitors	reduces glucose uptake and inhibits PI3K-Akt/β-catenin pathways.	[53]
Glyburide	anti-diabetic	modulates insulin/IGF pathways and induces ROS-dependent apoptosis.	[53]
Aspirin	NSAIDs	inhibits COX-1/COX-2 and reduces prostaglandin synthesis.	[63]
Celecoxib	NSAIDs	inhibits COX-2, suppresses NF-κB pathway, induces apoptosis, angiogenesis, and tumor cell invasiveness.	[63]
Metformin	anti-diabetic	lowers insulin and inhibits mitochondrial Complex I, activating AMPK to suppress mTOR, inhibit angiogenesis, reduces tumor hypoxia, and induces apoptosis and cell-cycle arrest.	[64]
Chloroquine	anti-malarial	promotes the tumor infiltration of NK cells through activating theTLR3/IFN-β/RIG-1/CCL3 axis of tumor cells. By combining disulfiram and 5-FU, a synergistic apoptotic effect was observed in 5-FU-resistant cell lines	[65]
Disulfiram	disulfide	by combining with 5-FU, Disulfiram decreases chemo resistance.	[65]
Statins	anti-hyperlipidemic	induces immunogenic cell death (ICD) by increasing eukaryotic initiation factor 2 alpha (eIF2α) phosphorylation and triggering the translocation of calreticulin to the cancer cell membrane. Also inhibiting BRAF activity, Statins promote apoptosis.	[65]
Niclosamide	anthelmintic	reverse CRC resistance to molecular targeted therapy by inhibiting the STAT3 pathway, reducing tumor cell survival and proliferation.	[65]
Aspirin	NSAIDs	promotes the repolarization of TAMs to M1-like phenotype and enhance the function of infiltrated CD8+ T cells through inhibiting the activation of platelets in tumor microenvironment (TME);	[66]
Bupivacaine	amides	inhibits the NF-κB.	[67]
Mefloquine	anti-malarial	inhibition of NF-κB activation	[67]
Mebendazole	anthelmintic	inhibition of MYC.	[67]
Artemisinin	anti-malarial	inhibition of PI3K-AKT-mTOR and NF-κB pathways and also induces autophagy.	[56]
Penfluridol	anti-psychotic	suppresses mTOR through AMPK activation; induces damage to mitochondrial and lysosomal membranes, impairing cellular metabolism and survival.	[68]
Ivermectin	anthelmintic	inhibits proliferation and promotes apoptosis in colorectal cancer cell lines (CC14, CC36, DLD1, Ls174T) by blocking the Wnt/β-catenin pathway. Increase caspase-3 expression, enhancing apoptosis. Downregulates downstream Wnt/β-catenin pathway genes (AXIN2, LGR5, ASCL2).	[59]
GP-15	anti-fungal	inhibits centrosomal clustering, a mechanism cancer cells survive mitotic stress, leading to multipolar spindle formation, mitotic failure, and subsequent apoptosis in cancer cells with supernumerary centrosomes.	[69]

common pathways in human cancer owing to its leading role in cancer cell survival and proliferation and in the resistance to the apoptosis . The pathway has been the target of many repurposed drugs.

Cancer research shows that biguanide Metformin, an antidiabetic used to treat type II diabetes, can activate AMP-activated protein kinase (AMPK), which leads to suppression of mTOR complex 1 (mTORC1) and subsequently, halts protein synthesis and proliferation of cancerous cells [49]. It has also been shown that Propranolol, a non-selective beta-adrenergic receptor blocker, antagonizes beta-adrenergic signaling. This subsequently suppresses the PI3K/AKT/mTOR signaling cascade in a number of solid tumors [70, 71].

4.1.2. NF-κB pathway

Nuclear factor kappa B (NF-κB) is known to be continuously active in many tumor types. NF-kB is associated with cancer cell survival, growth, and chemoresistance. Chloroquine causes dysfunction of the 20S/26S proteasome, leading to the accumulation of the NF-kB inhibitor IκB, inhibiting NF-κB activity [72]. Nelfinavir has both anti-proteasome activity and inhibition of the NF-κB signaling pathway [72].

4.1.3. STAT3/STAT5 signaling

Constant activation of signal transducers and activators of transcription (STAT) proteins, especially STAT3 and STAT5, facilitates proliferation and survival while enabling immune evasion in many cancers. Artemisinin targets IL-6-JAK-STAT3 in melanoma and also inhibits the effects of cancer stem cell-mediated metastasis and STAT3 in laryngeal carcinoma while helping the induction of ferroptosis in lung cancer via the same pathway [56].

4.2. Induction of apoptosis

Apoptosis, or programmed cell death, is a significant strategy for killing cancer cells that is employed by anticancer agents. There are many repurposed drugs that are reported to activate apoptosis via intrinsic (mitochondrial) and extrinsic (death receptor-mediated) pathways. Metformin triggers apoptosis in ovarian cancer cells by downregulating the anti-apoptotic proteins Bcl-2 and Bcl-xL and upregulating the pro-apoptotic proteins Bax and Cytochrome c, thus leading to cell death [73, 74]. On the other hand, Fluoxetine activates apoptosis, in the framework of modulating immune and cellular stress responses, in pancreatic and gastric cancer cells by activating caspases, cleaving PARP, and eliciting ER stress, and ultimately enhances the efficacy of other drugs that are administered concurrently [60].

4.3. Cell cycle arrest

Uncontrolled cell cycle progression is a defining feature of malignant cells. Discoveries show the possibility of repurposed drugs, which can reinstate cell cycle checkpoints in various phases, and halt cancer cell proliferation at the G0/G1, S, or G2/M transition. Fluoroquinolone antibiotics react with DNA, target DNA gyrase and topoisomerase-II, inflicting DNA damage, and cause the activation of checkpoint kinases leading to cell cycle arrest and apoptosis in a wide variety of cancers such as liver, lung, bladder, and cervical cancers [48, 70].

4.4. Disruption of cellular metabolism

Cancer cells exhibit profound metabolic reprogramming to sustain rapid growth, and targeting these altered metabolic dependencies is a key strategy in drug repurposing.

4.4.1. Inhibition of mitochondrial function and ATP depletion

Several previously approved drugs target cancer cells by reducing ATP levels via disruption of oxidative phosphorylation and mitochondrial protein synthesis. Metformin is a Complex I inhibitor, and in addition to reducing respiration and ATP synthesis, it also decreases insulin and IGF-1 levels. Therefore, metformin also diminishes signaling pathways upregulated by growth factors [49, 75]. Chloramphenicol, also a Complex I inhibitor, decreases the respiratory chain and ATP synthesis, and targets protein synthesis in mitochondria of multiple myeloma cells [49].

4.4.2. Inhibition of HMG-CoA reductase and the mevalonate pathway

Stanins are able to competitively block HMG-CoA reductase, an important enzyme in the mevalonate pathway. As a result, statins inhibit and remove the production of the small GTPase membrane proteins from mevalonate, cholesterol, farnesyl pyrophosphate, and geranylgeranyl pyrophosphate. This membrane and post translational protein modification remove the small GTPase membrane proteins RAS, RHO, RAC, and several GTPase membrane proteins that have a role in cell functions from migration and cell invasion in all cancer types [53]. In prostate cancer this intervention will also decrease the androgen synthesis coming from cholesterol and will act as another hormone deprivation therapy in prostate cancer which is an androgen remote dependent cancer [72].

4.4.3. AMPK activation

AMPK is an energy sensor which, when activated, will inhibit anabolic processes and activate catabolic ones. This includes inhibition of mTOR and activation of autophagy. Metformin activates AMPK directly and indirectly. This causes a level of disruption to the metabolism, producing a decrease in proteins and lipids. This slows down proliferation and decreases the tumor-associated inflammatory response to ovarian and pancreatic cancers [49, 73-75]. Artemisinin also activates AMPK and, in multiple cancers, is associated with autophagy and ferroptosis with inhibition of STAT3 [53]. Canagliflozin activates AMPK and also inhibits the PI3K-Akt/β-catenin pathway. This leads to a decrease in glucose uptake, ultimately depriving cancer cells of both fuel and a survival signal [53].

4.4.4. COX inhibition and prostaglandin modulation

NSAIDs such as aspirin, diclofenac, and celecoxib, block the activity of cyclooxygenase-2 (COX-2), which is overexpressed in many tumors. COX-2 catalyzes the production of pro-inflammatory mediators, particularly PGE2, and consequently, NSAIDs inhibit the tumor-promoting inflammation, angiogenesis, and metastasis [71, 76]. Aspirin can also inhibit the COX-2, NF-κB, STAT3, and subsequently, the apoptosis and cell cycle arrest [77].

4.5. DNA damage and topoisomerase inhibition

Originally designed as DNA gyrase inhibitors, fluoroquinolone antibiotics also inhibit eukaryotic topoisomerase-IIα. In cancer cells, fluoroquinolones stabilize the topoisomerase-II/DNA cleavage complex and block the re-ligation of the DNA breaks forming persistent double strand breaks. This activates the DNA damage response and the p53 pathway, resulting in the G2/M cell cycle blockade and apoptosis in multiple cancer types, including liver, lung, bladder, cervical, and prostate cancers [48, 70]. Carboplatin is a traditional alkylating agent that inflicts DNA damage, and is being repurposed with the demethylating agent azacitidine in melanoma. Here, the DNA damage done is hypothesized to synergize with the epigenetic modifications to increase the immune visibility of the tumors [78]. These drugs demonstrate that topoisomerase poisoning and alkylating agents, and etoposide and doxorubicin, can all be used in drug repurposing in oncology.

4.6. Generation of Reactive Oxygen Species (ROS) and induction of oxidative stress

Due to their activated metabolism, reactive oxygen species (ROS) rise in concentration in cancer. Although elevated ROS do not kill cancer cells, it does pose an advanced level of threat. Auranofin is an antirheumatic gold compound that inhibits TrxR causing the redox control system in cells to collapse. It then causes NSCLC, osteosarcoma, and GIST to undergo apoptosis [49]. Disulfiram, when used in conjunction with copper, forms a complex that causes the cells to ROS. In addition to forming toxic ROS, Disulfiram inhibits ALDH and proteasomal activity. Disulfiram also binds the cofactor NPL4 and immobilizes it causing cell death. Disulfiram induces apoptosis and autophagy in several cancers [58].

4.7. Autophagy modulation

Autophagy is a cellular process where damaged organelles and proteins are degraded and recycled through lysosomes. Clarithromycin is a macrolide antibiotic that targets the hERG1 potassium channel to inhibit autophagy in leukemia. This case shows how the blockage of autophagy can act as an anticancer strategy, in contrast to hydroxychloroquine's lysosomal mechanism [67]. Artemisinin induces autophagy and apoptosis in bladder cancer and activates the CaMKK2-AMPK-ULK1 cascade in B-cell lymphoma [56]. Chlorpromazine induces endoplasmic reticulum (ER) stress and the unfolded protein response (UPR) in glioma, which also leads to autophagic cell death [79-81].

4.8. Endoplasmic reticulum (ER) stress and Unfolded Protein Response (UPR)

The ER contains three major functions including lipid synthesis, protein folding, and calcium balance. When the cell experiences stress, proteins that are incorrectly folded build up in the lumen of the ER. This activates the three main sensors of the unfolded protein response (UPR), PERK, IRE1, and ATF6. While the response is pro-survival at first, prolonged activation of the response induces CHOP and other cellular effectors that are pro-apoptotic. This is the reason that many of the drugs used to treat cancer are repurposed drugs. Chlorpromazine is one of the drugs that causes both ER stress and reactive oxygen species build up in glioma cells. This causes UPR activation and cell death through apoptosis [81].

4. Challenges in anti-cancer drug repurposing

While repurposing non-oncology drugs is faster and more cost-effective than de novo drug discovery, transitioning these molecules to oncology clinics faces critical barriers [82]. Anti-tumor efficacy often requires significantly higher dosages than a drug's original indication [83]. This escalation alters its absorption, distribution, metabolism, and excretion (ADME) profile, potentially introducing severe toxicities and transforming a safe medication into a clinical risk [84, 85]. Most viable candidates for repurposing are off-patent generic drugs. As the pharmaceutical companies cannot secure exclusive "composition-of-matter" patents, the potential return on investment (ROI) is negligible compared to novel molecules [86]. Additionally, if an institution funds the necessary clinical trials for a new indication, competing generic manufacturers can immediately market the substance for that same indication without sharing development costs [86]. This market flaw drives widespread, unapproved "off-label" clinical use, which lacks regulatory oversight, triggers insurance reimbursement denials, and restricts clinical adoption [86]. Cancer exhibits vast genetic and phenotypic variation across patient populations and within individual tumors. Single-pathway repurposed agents frequently trigger adaptive resistance or fail across diverse cell subsets within the tumor microenvironment [85]. Advancing candidate therapies requires large-scale randomized controlled trials. These trials routinely face resource deficits, high regulatory burdens, and poor patient recruitment, frequently stalling projects in Phase I or II [87].

5. Conclusion and future work

Drug repurposing can be a powerful, fast, and cost-effective strategy for expanding cancer treatments. This review identified approximately 181 non-oncology drugs with demonstrated anticancer potential. Among these, metformin, propranolol, mebendazole, disulfiram, and statins exhibited the strongest preclinical and clinical evidence. This study also revealed the most common mechanisms: modulation of oncogenic signaling pathways, metabolic reprogramming, and induction of apoptosis. It also highlights the potential of this

strategy, suggesting that repurposing is a practical way to bring new options to patients faster than traditional drug development. While traditional drug development may take over a decade and cost billions, repurposing leverages existing safety and manufacturing data to significantly shorten the timeline. Optimizing priming cycles, combination strategies, and biomarker validation can be options for the future. Sometimes, monotherapy may be relatively ineffective for cancer patients due to the multiple mechanisms of resistance and complex oncogenic signaling pathways of cancers. For such instances, we can explore combination therapies as a potential opportunity, as these target both tumor metabolism and classic oncogenic pathways. Novel drug screening using computational approaches can advance personalized treatment strategies based on molecular profiling of tumors. The identification of the biomarkers and their validation will help to select patients most likely to benefit. We need to focus on developing novel formulations and targets for broader applications. We should define the evidence framework for moving repurposed drugs into large-scale phase III trials. To overcome this barrier, collaborations among academic institutions, non-profit organizations, and government agencies are essential to advance research into drug repurposing for cancer.

Author contributions

Conceptualization and study design, Runa Akter. Muhammad Asaduzzaman., Investigation, Runa Akter. Riya Majumder Moriom. Ankur Biswas. Faisal., Visualization and original figure design, Hasin Eshrak., Writing original draft preparation, Riya Majumdar Moriom. Ankur Biswas. Faisal. Bijoy Chowdhury., Writing review and editing, Runa Akter. Muhammad Asaduzzaman., Supervision, Muhammad Asaduzzaman. Runa Akter., All authors have read and agreed to the published final version of the manuscript.

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Not applicable.

Conflicts of Interest

The authors report no conflicts of interest.

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Supplementary material

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REFERENCES

1. Singhal S., et al.(1999). Antitumor activity of thalidomide in refractory multiple myeloma. The New England journal of medicine, 341(21), 1565–1571.
2. Goldstein, I., et al. (1998). Oral sildenafil in the treatment of erectile dysfunction. Sildenafil Study Group. The New England journal of medicine, 338(20), 1397–1404.
3. Ashburn, T.T., and Thor, K.B. (2004). Drug repositioning: Identifying and developing new uses for existing drugs. Nature Reviews Drug Discovery, 3(8), pp. 673–683.
4. Pushpakom, S., et al. (2019).Drug repurposing: Progress, challenges, and recommendations. Nature Reviews Drug Discovery, 18(1), pp. 41–58.
5. World Health Organization (2020) Cancer. Available at: https://www.who.int/news-room/fact-sheets/detail/cancer (Accessed: 14 April 2025).
6. Almendro, V., Marusyk, A. and Polyak, K. (2013). Cellular heterogeneity and molecular evolution in cancer. Annual Review of Pathology: Mechanisms of Disease, 8, pp. 277–302.
7. DiMasi, J.A., Grabowski, H.G. and Hansen, R.W. (2016). Innovation in the pharmaceutical industry: New estimates of R&D costs. Journal of Health Economics, 47, pp. 20–33.
8. Sleire, L. et al. (2017). Drug repurposing in cancer. Pharmacological Research, 124, pp. 74–91.
9. Pollak, M. (2012). Investigating metformin for cancer prevention and treatment: The end of the beginning', Cancer Discovery, 2(9), pp. 778–790.
10. Michalis, A., et al. (2023). IGF-1R targeting in cancer–does sub-cellular localization matter? Journal of Experimental & Clinical Cancer Research, 42(1), 192.
11. Foretz, M., et al. (2014). Metformin: from mechanisms of action to therapies. Cell metabolism, 20(6), 953–966.
12. Hirsch, E. C., Jenner, P., & Przedborski, S. (2013). Pathogenesis of Parkinson's disease. Movement disorders : official journal of the Movement Disorder Society, 28(1), 24–30.
13. Demierre, M.F. et al. (2005). Statins and cancer prevention. Nature Reviews Cancer, 5(12), pp. 930–942.
14. Jung, G. W., et al. (2013). Prospective, randomized, open-label trial comparing the safety, efficacy, and tolerability of an acne treatment regimen with and without a probiotic supplement and minocycline in subjects with mild to moderate acne. Journal of cutaneous medicine and surgery, 17(2), 114–122.
15. Pantziarka, P. et al. (2016) The Repurposing Drugs in Oncology (ReDO) project. ecancermedicalscience, 10, p. 682.
16. Mattiuzzi, C. and Lippi, G. (2019). Current cancer epidemiology. Journal of Epidemiology and Global Health, 9(4), pp. 217–222.
17. Zhang, Y. (2013). Epidemiology of esophageal cancer. World Journal of Gastroenterology, 19(34), pp. 5598–5606.
18. Ilic, M. and Ilic, I. (2022). Epidemiology of stomach cancer. World Journal of Gastroenterology, 28(12), pp. 1187–1203.
19. Stintzing, S. (2014) .Management of colorectal cancer. F1000Prime Reports, 6, p. 108.
20. Ilic, M. and Ilic, I. (2016). Epidemiology of pancreatic cancer. World Journal of Gastroenterology, 22(44), pp. 9694–9705.
21. Steuer, et al. (2017). An update on larynx cancer. CA: A Cancer Journal for Clinicians, 67(1), pp. 31–50.
22. Thandra, et al. (2021). Epidemiology of lung cancer. Contemporary Oncology/Współczesna Onkologia, 25(1), pp. 45–52.
23. Mohammadian, et al. (2018). Liver cancer in the world: Epidemiology, incidence, mortality and risk factors. World Cancer Research Journal, 5(2), e1082.

24. Schadendorf, etal. (2015). Melanoma. Nature Reviews Disease Primers, 1(1), pp. 1–20.
25. Sun, Y.S., et al. (2017). Risk factors and preventions of breast cancer. International Journal of Biological Sciences, 13(11), pp. 1387–1397.
26. Koh Koh, W.J., et al. (2013). Cervical cancer. Journal of the National Comprehensive Cancer Network, 11(3), pp. 320–343.
27. Felix, A.S. and Brinton, L.A., 2018. Cancer progress and priorities: uterine cancer. Cancer Epidemiology, Biomarkers & Prevention, 27(9), pp.985-994.
28. Matulonis, U.A., et al. (2016). Ovarian cancer. Nature Reviews Disease Primers, 2(1), pp. 1–22.
29. Rawla, P., 2019. Epidemiology of prostate cancer. World journal of oncology, 10(2), p.63.
30. Cheng, L., et al. (2018).'Testicular cancer. Nature Reviews Disease Primers, 4(1), p. 29.
31. Sanli, O., et al. (2017). Bladder cancer. Nature reviews Disease primers, 3(1), pp.1-19.
32. Scelo, G. and Larose, T.L. (2018). Epidemiology and risk factors for kidney cancer. Journal of Clinical Oncology, 36(36), pp. 3574–3581.
33. Tandel, G.S., et al. (2019). A review on a deep learning perspective in brain cancer classification. Cancers, 11(1), p.111.
34. Seyfried, T.N., et al. (2011). Metabolic management of brain cancer. Biochimica et Biophysica Acta (BBA) - Bioenergetics, 1807(6), pp. 577–594.
35. Boucai, L., Zafereo, M. and Cabanillas, M.E. (2024). Thyroid cancer: A review. JAMA, 331(5), pp. 425–435.
36. Kitahara, C.M. and Sosa, J.A. (2016). The changing incidence of thyroid cancer. Nature Reviews Endocrinology, 12(11), pp. 646–653.
37. Connors, J.M., et al. (2020). Hodgkin lymphoma. Nature Reviews Disease Primers, 6(1), p. 61.
38. Ansell, S.M. (2015). Non-Hodgkin lymphoma: Diagnosis and treatment. Mayo Clinic Proceedings, 90(8), pp. 1152–1163.
39. Davis, A.S., Viera, A.J. and Mead, M.D. (2014). Leukemia: An overview for primary care. American Family Physician, 89(9), pp. 731–738.
40. Khan, K.S., Kunz, R., Kleijnen, J. and Antes, G. (2003). Five steps to conducting a systematic review. Journal of the Royal Society of Medicine, 96(3), pp. 118–121.
41. Shamseer, L., et al. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015: elaboration and explanation. BMJ, 349, g7647.
42. Xia, Y., Sun, M., Huang, H. and Jin, W.L. (2024). Drug repurposing for cancer therapy. Signal Transduction and Targeted Therapy, 9(1), p. 92.
43. Rodrigues, R., Duarte, D., & Vale, N. (2022). Drug repurposing in cancer therapy: influence of patient's genetic background in breast cancer treatment. International journal of molecular sciences, 23(8), 4280.
44. Aggarwal, S., et al. (2021). Drug repurposing for breast cancer therapy: Old weapon for new battle. In Seminars in cancer biology (Vol. 68, pp. 8-20). Academic Press.
45. Malik, J. A., et al. (2022). Drugs repurposed: An advanced step towards the treatment of breast cancer and associated challenges. Biomedicine & Pharmacotherapy, 145, 112375.
46. Duranti, E., Cordani, N., & Villa, C. (2024). Edaravone: A novel possible drug for cancer treatment?. International Journal of Molecular Sciences, 25(3), 1633.
47. Kadasah, S. F., Alqahtani, A. M. S., Alkhammash, A., & Radwan, M. O. (2024). Beyond Psychotropic: Potential Repurposing of Fluoxetine toward Cancer Therapy. International Journal of Molecular Sciences, 25(12), 6314.
48. Yadav, V., & Talwar, P. (2019). Repositioning of fluoroquinolones from antibiotic to anti-cancer agents: An underestimated truth. Biomedicine & Pharmacotherapy, 111, 934-946.
49. Aminzadeh-Gohari, S., et al. (2020). From old to new - Repurposing drugs to target mitochondrial energy metabolism in cancer. Seminars in cell & developmental biology, 98, 211–223.
50. Benjamin, D. J., Haslam, A., & Prasad, V. (2024). Cardiovascular/anti-inflammatory drugs repurposed for treating or preventing cancer: A systematic review and meta-analysis of randomized trials. Cancer medicine, 13(5), e7049.
51. Joharatnam-Hogan, N., et al. (2021). Statins as potential chemoprevention or therapeutic agents in cancer: A model for evaluating repurposed drugs. Current Oncology Reports, 23(3), p. 29. doi:10.1007/s11912-021-01023-z.
52. Varisli, L., Cen, O., & Vlahopoulos, S. (2020). Dissecting pharmacological effects of chloroquine in cancer treatment: interference with inflammatory signaling pathways. Immunology, 159(3), 257–278.
53. Dhas, Y., et al. (2024). Repurposing metabolic regulators: antidiabetic drugs as anticancer agents. Molecular biomedicine, 5(1), 40.
54. Fu, Z., Zhang, X., Gao, Y., Fan, J., & Gao, Q. (2023). Enhancing the anticancer immune response with the assistance of drug repurposing and delivery systems. Clinical and Translational Medicine, 13(7), e1320.
55. Jiang, W., et al. (2021). Statins: a repurposed drug to fight cancer. Journal of Experimental & Clinical Cancer Research, 40, 1-33.
56. Shi, Q., et al. (2022). Discovery and repurposing of artemisinin. Frontiers of medicine, 16(1), 1-9.
57. Ali Ibrahim Mze, A., & Abdul Rahman, A. (2024). Repurposing the antipsychotic drug penfluridol for cancer treatment. Oncology Reports, 52(6), 174.
58. Lu, C., Li, X., Ren, Y., & Zhang, X. (2021). Disulfiram: a novel repurposed drug for cancer therapy. Cancer chemotherapy and pharmacology, 87, 159-172.
59. Tang, M., et al. (2021). Ivermectin, a potential anticancer drug derived from an antiparasitic drug. Pharmacological research, 163, 105207.
60. Kadasah, S. F., et al. (2024). Beyond Psychotropic: Potential Repurposing of Fluoxetine toward Cancer Therapy. International Journal of Molecular Sciences, 25(12), 6314.
61. Laudisi, F., et al. (2020). Repositioning of Anthelmintic Drugs for the Treatment of Cancers of the Digestive System. International Journal of Molecular Sciences, 21(14), 4957.
62. Yadav, V., & Talwar, P. (2019). Repositioning of fluoroquinolones from antibiotic to anti-cancer agents: An underestimated truth. Biomedicine & Pharmacotherapy, 111, 934-946.
63. Zappavigna, S., et al. (2020). Anti-Inflammatory Drugs as Anticancer Agents. International journal of molecular sciences, 21(7), 2605.
64. Nowak-Sliwinska, P., Scapozza, L., & i Altaba, A. R. (2019). Drug repurposing in oncology: Compounds, pathways, phenotypes and computational approaches for colorectal cancer. Biochimica et Biophysica Acta (BBA)-Reviews on Cancer, 1871(2), 434-454.
65. Fong, W., & To, K. K. (2019). Drug repurposing to overcome resistance to various therapies for colorectal cancer. Cellular and molecular life sciences, 76, 3383-3406.
66. Fu, Z., Zhang, X., Gao, Y., Fan, J., & Gao, Q. (2023). Enhancing the anticancer immune response with the assistance

of drug repurposing and delivery systems. Clinical and Translational Medicine, 13(7), e1320.
67. Yang, Y., et al. (2024). Small-molecule drugs of colorectal cancer: Current status and future directions. Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease, 1870(1), 166880.
68. Ali Ibrahim Mze, A., & Abdul Rahman, A. (2024). Repurposing the antipsychotic drug penfluridol for cancer treatment. Oncology Reports, 52(6), 174.
69. Weng, N., Zhang, Z., Tan, Y., Zhang, X., Wei, X., & Zhu, Q. (2023). Repurposing antifungal drugs for cancer therapy. Journal of Advanced Research, 48, 259-273.
70. Yadav, V., & Talwar, P. (2019). Repositioning of fluoroquinolones from antibiotic to anti-cancer agents: An underestimated truth. Biomedicine & Pharmacotherapy, 111, 934-946.
71. Bahmad, et al. (2022). Overcoming Drug Resistance in Advanced Prostate Cancer by Drug Repurposing. Medical sciences (Basel, Switzerland), 10(1), 15. \
72. Lourenço, T., & Vale, N. (2023). Pharmacological Efficacy of Repurposing Drugs in the Treatment of Prostate Cancer. International journal of molecular sciences, 24(4), 4154.
73. Almeida-Nunes, D. L., Silvestre, R., Dinis-Oliveira, R. J., & Ricardo, S. (2023). Enhancing immunotherapy in ovarian cancer: The emerging role of metformin and statins. International Journal of Molecular Sciences, 25(1), 323.
74. Kobayashi, Y., Banno, K., Kunitomi, H., Tominaga, E., & Aoki, D. (2019). Current state and outlook for drug repositioning anticipated in the field of ovarian cancer. Journal of gynecologic oncology, 30(1).
75. Nunes, M., Henriques Abreu, M., Bartosch, C., & Ricardo, S. (2020). Recycling the purpose of old drugs to treat ovarian cancer. International Journal of Molecular Sciences, 21(20), 7768.
76. Joharatnam-Hogan, N., et al. (2020). The role of aspirin in the prevention of ovarian, endometrial and cervical cancers. Women's Health, 16, 1745506520961710.
77. Varisli, L., Cen, O., & Vlahopoulos, S. (2020). Dissecting pharmacological effects of chloroquine in cancer treatment: interference with inflammatory signaling pathways. Immunology, 159(3), 257–278.
78. Gonzales, C. N., et al. (2024). Malignant glioma remodeling of neuronal circuits: therapeutic opportunities and repurposing of antiepileptic drugs. Trends in cancer, 10(12), 1106–1115.
79. Xia, Y., et al. (2024). Drug repurposing for cancer therapy. Signal transduction and targeted therapy, 9(1), 92.
80. Lu, C., Li, X., Ren, Y., & Zhang, X. (2021). Disulfiram: a novel repurposed drug for cancer therapy. Cancer chemotherapy and pharmacology, 87, 159-172.
81. You, F., et al. (2022). Drug repositioning: Using psychotropic drugs for the treatment of glioma. Cancer Letters, 527, 140-149.
82. Camps, I. (2025). Editorial: Opportunities and challenges in drug repurposing. Frontiers in Pharmacology, 16.
83. Ivanov, A. (2026). From laboratory to patient access: A scoping review of the multi-dimensional challenges in drug repurposing. MDPI, 14(3), 85.
84. Zafar, R., et al. (2025). Advantages, challenges, and impact of drug repurposing for cancer treatment. In Repurposed Drugs: Current State and Future Perspectives.
85. Garcia-Diaz, M. (2025). Overcoming barriers to off-patent drug repurposing: A lifecycle-based policy solution. Frontiers in Pharmacology, 16.
86. Sakis, N. (2026). Advancing the human right to health in cancer care through drug repurposing strategies. PubMed Central (PMC).
87. Van der Pol, K. H., et al. (2023). Drug repurposing of generic drugs: Challenges and the potential role for government. Applied Health Economics and Health Policy, 21(6), 831–840.

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