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Supplementary material

Drug Repurposing in Cancer Therapy: A Systematic Review

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Supplementary material

Table S1. Repurposed drugs along with their mechanism in prostate cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoroquinolones	Antibiotic	inhibits key enzymes involved 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.	[70]
Propranolol	Beta blockers	Blocks beta-adrenergic receptors and inhibits PI3K/AKT/mTOR and MAPK pathways	[71]
Digoxin	Antiarrhythmic drug	Inhibits Na^+/K^+ ATPase & modulates PI3K/AKT/mTOR signaling.	[71]
Ouabain	Antiarrhythmic drug	Inhibition of Na^+/K^+ ATPase.	[71]
Aspirin	NSAIDs	Modulation of COX signaling pathways.	[71]
Celecoxib	NSAIDs	Modulation of COX signaling pathways.	[71]
Dexamethasone	Corticosteroids	Modulation of signaling pathways, e.g., PI3K/AKT/mTOR.	[71]
Statins	Antihyperlipidemic drugs	Inhibition of HMG-CoA reductase and reduction of cholesterol synthesis.	[71]
Metformin	Antidiabetic drug	Activation of AMPK pathway and inhibition of cancer cell proliferation; Inhibiting Complex I decreases respiration and ATP production, and lowers insulin/IGF-1 levels.	[71], [49]
Itraconazole	Antifungal drugs	Hedgehog pathway inhibition & angiogenesis suppression.	[71]
Zoledronic acid	Bisphosphonates	Bisphosphonate mediated osteoclast inhibition & modification of tumor microenvironment.	[71]
Statins	Antihyperlipidemic drugs	Inhibition of HMG-CoA reductase and suppression of androgen synthesis and angiogenesis.	[72]
Diclofenac	Anticholinergics	Inhibition of COX and prostaglandin.	[72]
Chloroquine	Antibiotic	Inhibiting 20s/26s proteasome and inactivating NF-κB.	[72]
Nelfinavir	Protease inhibitors	Proteasome inhibition (20s/26s) and NF-κB inactivation & PI3K-Akt pathway blockade.	[72]
Ivermectin	Anthelmintic Agents	Ivermectin enhances anti-androgen drug Enzalutamide activity in LNCaP cells and reverses Docetaxel resistance in PC3 cells. Inhibits proliferation in DU145 cells. Restores sensitivity to endocrine therapy, suggesting a role in combination treatments.	[59]

Table S2. Repurposed drugs along with their mechanism in ovarian cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Statins	Antihyperlipidemic drugs	It inhibits HMG-CoA reductase, reducing mevalonate, downstream cholesterol and isoprenoid biosynthesis, which are essential for various cellular processes.	[73], [74], [75] [76]
Metformin	Antidiabetic drug	Reactivates the AMPK pathway, which in turn inhibits mTOR signaling leading to several anti-cancer effects, including reduced protein and lipid synthesis, slower proliferation rates, activation of autophagy, and reduced inflammatory responses.	[73], [74], [75], [49]
Bisphosphonates	Antiresorptive drugs	Inhibits the proliferation of ovarian cancer cell lines in a concentration-dependent manner. Metformin can induce apoptosis in both primary ovarian cancer cells and SKOV-3 cells by downregulating Bcl-2 and Bcl-xL expression and upregulating Bax and Cytochrome c expression	[74]
Ivermectin	Anthelmintic Agents	Ivermectin interferes with several cellular mechanisms, including multidrug resistance proteins (MDR) inhibition, Akt/mTOR, and Wnt signaling pathways modulation, p21–activated kinase (PAK-1) and yes-associated protein 1 (YAP1).	[74]
Ritonavir	Antiviral drugs	Induces cell cycle arrest and apoptosis, primarily through inhibiting the AKT pathway and potentially by modulating other signaling pathways like surviving	[75]
Aspirin	NSAIDs	inhibition of cyclooxygenase enzymes, particularly COX-2, which is often overexpressed in tumors. By blocking COX-2, aspirin reduces the production of pro-inflammatory prostaglandins that promote tumor growth, angiogenesis, and metastasis. Additionally, aspirin can disrupt tumor-promoting signaling pathways and influence cellular processes like apoptosis and angiogenesis	[76]
Artemisinin	Antimalarial drugs	Induces cell cycle arrest and autophagy via NF-κB suppression	[56]
Griseofulvin	Antifungal Drugs	Induces apoptosis and G2/M cell cycle arrest by disrupting microtubule polymerization, preventing mitotic spindle formation; suppresses spindle microtubule dynamics, leading to mitotic catastrophe and cancer cell death, particularly effective in rapidly dividing cells.	[69]

Table S3. Repurposed drugs along with their mechanism in glioma cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Itraconazole	Antifungal Drugs	Binding with VDAC1 reduces mitochondrial ATP.	[49]
Hydroxychloroquine	Antibiotic	Alters mitochondrial function and bioenergetics & affects proteolytic systems	[77]
Levetiracetam	Antiepileptic drugs	Inhibiting neuronal hyperexcitability; disrupting neuron-glioma synaptic communication	[78]
Valproic acid	Antiepileptic drugs	Functions as a voltage-gated sodium channel blocker and t-type calcium channel blocker	[78]
Statins	Antihyperlipidemic drugs	Inhibits angiogenesis in a dose-dependent manner through downregulation of vascular endothelial growth factor (VEGF), CD31, and Bcl-2 in glioblastoma model	[79]
Penfluridol	Antipsychotic drugs	Inhibits Akt phosphorylation and GLI1, reducing proliferation; suppresses EMT by downregulating vimentin, N-cadherin, Snail, Slug; reduces stemness markers (SOX2, OCT4, Nanog); enhances immune response by reducing Tregs and increasing M1 macrophages.	[68]
Disulfiram	Disulfide	Inhibits temozolomide-resistant GBM cells (IC鈪呪個 =100 nM), suppressing self-renewal at 500 nM; trials show DSF (500 mg/day) with temozolomide yields 5.4 months median progression-free survival	[80]
Chlorpromazine	Antipsychotic drugs	Promotes endoplasmic reticulum (ER) stress and reactive oxygen species (ROS) accumulation, triggering the unfolded protein response (UPR), leading to apoptotic cell death. Inhibits AKT/mTOR signaling pathway, reducing cell survival and proliferation. Causes cell cycle arrest by inhibiting AKT/mTOR signaling or promoting EGR-1-mediated P21 expression, halting glioma cell proliferation.	[81]
Trifluoperazine	Antipsychotic drugs	Disrupts calcium homeostasis via IP3R, inhibits IDH1-calmodulin interaction, targets mitochondrial function to reduce glioma stem cell stemness, enhances chemosensitivity and improves radiotherapy efficacy.	[81]
Pimozide	Antipsychotic drugs	Inhibits STAT5 activity, reducing glioma cell growth. Blocks EGF transcription regulated by ID1 or promotes USP1-mediated ID1 degradation, suppressing proliferation.	[81]
Olanzapine	Antipsychotic drugs	Inhibits proliferation, migration, and anchorage-independent growth. Induces autophagic cell death by suppressing NF-kB activation. Elevates ROS production to increase cell death induced by ionizing radiation, while scavenging free radicals to protect normal cells. Enhances temozolomide (TMZ) antitumor activity, though specific mechanisms are unclear.	[81]
Ivermectin	Anthelmintic Agents	Limited efficacy due to poor blood-brain barrier penetration; no specific mechanisms provided as IVM's prospects for glioma treatment are deemed unoptimistic.	[59]
5-Fluorocytosine	Antifungal Drugs	Used in gene-directed enzyme prodrug therapy (GDEPT), where it is converted by cytosine deaminase into 5-fluorouracil, a toxic metabolite that inhibits thymidylate synthase, disrupts DNA synthesis, and induces apoptosis in cancer cells.	[69]

Table S4. Repurposed drugs along with their mechanism in pancreatic cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoxetine	Selective serotonin reuptake inhibitors (SSRIs)	Induces apoptosis and inhibiting proliferation, survival, and invasion pathways, including AKT/mTOR andNF-κB. It also modulates immune responses and cellular stress pathways, enhancing the effectiveness of other cancer treatments.	[60]
Canagliflozin	SGLT2 inhibitors	Reducing glucose uptake; inhibiting PI3K-Akt/β-catenin pathways	[53]
Glyburide	Antidiabetic drug	Modulating insulin/IGF pathways; ROS-dependent apoptosis	[53]
Pyrvinium pamoate (PP)	Anthelmintic Agents	PP inhibits mitocnondrial function and disrupts cancer cell energy metabolism in the hypoxic, nutrient-poor tumor microenvironment of PDAC, reducing proliferation and enhancing sensitivity to chemotherapeutic agents.	[82]
Aspirin	NSAIDs	Inhibit COX-1/COX-2; Modulate NFkB or STAT3 pathway	[83]
Metformin	Antidiabetic drug	Lower insulin/IGF-1 levels Activate AMPK which inactivates mTOR pathway	[83]
Statins	Antihyperlipidemic drugs	Prevent synthesis of mevalonic acid, which then activates small G proteins–Ras, Rho, and Rac;	[83]
Propranolol	Beta blockers	Block cAMP-dependent intracellular signaling and release of EGF Block PKA-dependent release of VEGF	[83]
Bisphosphonates	Bisphosphonates	Interfere with RAS and Rho pathways Inhibit tumor educated macrophages;	[83]
DPP-4 inhibitors	Antidiabetic drug	Stimulate pancreatic β-cells to release insulin, resulting in increased α- and β-cell mass	[83]
Penfluridol	Antipsychotic drugs	Triggers ER stress markers (BIP, CHOP, IRE1伪); activates PP2A phosphatase, leading to apoptosis; suppresses PRLR signaling, disrupting cell survival pathways; promotes autophagy-mediated apoptosis by impairing lysosomal formation.	[68]
Disulfiram	Disulfide	DSF with arsenic trioxide/ascorbic acid induces alternative cell death in Ras-mutated cell lines	[80]

Table S5. Repurposed drugs along with their mechanism in leukemia cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Thalidomide	Immunomodulatory agents	thalidomide inhibits angiogenesis by interrupting processes mediated by bFGF and/or vascular endothelial growth factor (VEGF).	[84]
Imatinib	Kinase inhibitors	Imatinib's anticancer activity stems from its ability to inhibit the BCR-ABL tyrosine kinase. This inhibition blocks the cell's ability to grow and divide, ultimately leading to cell death (apoptosis) in cancer cells	[84]
Dasatinib	Kinase inhibitors	Dasatinib disrupts cellular processes like growth, adhesion, and migration that are often dysregulated in cancer. This disruption leads to cell death (apoptosis) and inhibits tumor growth, making dasatinib effective in treating certain leukemias and some solid tumors	[84]
Metformin	Antidiabetic drug	Inhibits Complex I, which reduces ATP production, and reduces insulin/IGF-1 levels; activates AMPK pathway, and inhibits cell proliferation.	[49]
Clarithromycin	Antibiotic	Inhibition of autophagy by targeting hERG1	[67]
Artemisinin	Antimalarial drugs	Promotes ferroptosis via AMPK/mTOR/p70S6k-mediated autophagy	[56]
Penfluridol	Antipsychotic drugs	Activates PP2A, suppressing pro-survival kinases like Akt and MAPK; elevates intracellular ROS, inducing both autophagy and apoptosis.	[57]
Ivermectin	Anthelmintic Agents	Preferentially kills leukemia cells at low concentrations without affecting normal hematopoietic cells by increasing chloride ion influx, leading to plasma membrane hyperpolarization and reactive oxygen species (ROS) production. Synergizes with cytarabine and daunorubicin to enhance treatment efficacy.	[59]

Table S6. Repurposed drugs along with their mechanism in liver cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoroquinolones	Antibiotic	They target DNA gyrase and topoisomerase-II, leading to DNA damage, cell cycle arrest, and ultimately apoptosis in cancer cells	[48]
Valproic acid	Antiepileptic	Decreases Respiration & ATP production	[49]
Aspirin	NSAIDs	Reduce stress signaling pathways & COX inhibition	[50]
Statins	Antihyperlipidemic drugs	Inhibiting HMG-CoA reductase - Decrease in mevalonate and downstream cholesterol biosynthesis. Inhibition of isoprenoid metabolites required for cellular functions	[51]
Metformin	Antidiabetic drug	Inhibits Complex I, which reduces ATP production, and reduces insulin/IGF-1 levels; activates AMPK pathway, and inhibits cell proliferation.	[85]
Statins	Antihyperlipidemic drugs	Inhibits HMG-CoA reductase, affects mevalonate pathway; prolongs survival in advanced HCC with 5-FU; shows weaker pro-apoptotic activity compared to lipophilic statins.	[55]
Disulfiram	Disulfide	DSF/Cu with anti-PD-1 antibody inhibits PARP1, reducing GSK3β activity, upregulating PD-L1, and increasing T-cell infiltration, enhancing tumor suppression	[58].
Ivermectin	Anthelmintic Agents	Inhibits tumor development by blocking YAP1 activity in spontaneous liver cancer models (Mob1b−/− mice)	[59]
Terbinafine	Antifungal Drugs	Inhibits squalene epoxidase (SQLE), disrupting sterol biosynthesis and leading to accumulation of toxic intermediates; suppresses Raf-MEK-ERK signaling pathway, which regulates cell proliferation and survival, thereby inducing apoptosis in cancer cells.	[69]

Table S7. Repurposed drugs along with their mechanism in lung cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoroquinolones	Antibiotic	They target DNA gyrase and topoisomerase-II, leading to DNA damage, cell cycle arrest, and ultimately apoptosis in cancer cells	[70]
Metformin	Antidiabetic drug	Inhibits Complex I results in decreased respiration & ATP production leads to Reducing insulin/IGF-1 levels	[49]
Canagliflozin	SGLT2 inhibitors	Reducing glucose uptake; inhibiting PI3K-Akt/β-catenin pathways	[53]
Dexamethasone	Corticosteroids	Suppresses inflammatory cytokines, alters chemotherapeutic pharmacokinetics	[63]
Statins	Antihyperlipidemic drugs	Blocks mevalonate pathway, impairing YAP/TAZ activity; reduces GGPP and RhoA activity, inhibiting proliferation, migration, and invasion (e.g., gastric cancer); enhances efficacy of combination therapies (e.g., FAC in breast cancer); promotes apoptosis.	[55]
Artemisinin	Antimalarial drugs	ER stress induction, STAT3 suppression, ferroptosis, AMPK activation	[56]
Penfluridol	Antipsychotic drugs	Causes mitochondrial ATP depletion and downregulates PGC-1α and SIRT1; induces autophagy through ER stress and unfolded protein response (UPR); inhibits AKT/MMP signaling and EMT, reducing metastasis; triggers intrinsic apoptosis via caspase activation.	[68]
Ivermectin	Anthelmintic Agents	Inhibits proliferation of H1299 lung cancer cells by targeting YAP1 activity. Synergizes with erlotinib by regulating EGFR activity, enhancing cell killing in HCC827 cells.	[59]
Sertaconazole	Antifungal Drugs	Induces proapoptotic autophagy by triggering endoplasmic reticulum stress and reactive oxygen species accumulation, leading to autophagic cell death; may also inhibit fungal-like pathways in cancer cells, though specific targets remain unclear.	[69]

Table S8. Repurposed drugs along with their mechanism in gastric cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Metformin	Antidiabetic drug	Inhibits Complex I results in decreased respiration & ATP production leading to reduced insulin/IGF-1 levels; Inhibits cell proliferation, migration, and stemness; induces apoptosis and G0/G1 cell cycle arrest	[49]
Fluoxetine	Antidepressant (SSRI)	Inhibits cell proliferation, induces apoptosis via caspase activation, PARP cleavage, and endoplasmic reticulum stress (CHOP expression) in AGS cells	[86]
Valproic acid	Antiepileptic	Inhibits cell proliferation and migration, induces apoptosis, autophagy, and G1 phase cell cycle arrest; in vivo, inhibits growth via autophagy and apoptosis	[86]
Statins	Antihyperlipidemic drugs	Inhibit cell proliferation, induce apoptosis; Lovastatin suppresses HDAC2 in vivo	[86]
Propranolol	Beta blockers	Inhibits cell proliferation, induces apoptosis, and G0/G1 cell cycle arrest; suppresses proliferation in vivo	[86]
Verteporfin	Photosensitizing agents	Inhibits YAP1/TAZ-TEAD transcriptional activity, suppresses tumor growth"	[86]
Ivermectin	Anthelmintic Agents	Inhibits proliferation of gastric cancer cells (e.g., MKN1, SH-10-TC) in vivo and in vitro by targeting Yes-associated protein 1 (YAP1) expression, which plays an oncogenic role in tumorigenesis (Page 3, [39]). Cells with high YAP1 expression are more sensitive to IVM.	[59]

Table S9. Repurposed drugs along with their mechanism in bladder cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Metformin	Antidiabetic drug	Metformin can inhibit BC by inducing apoptosis or cell cycle arrest, by activating various signaling pathways, blocking BC stem cell proliferation, and enhancing the efficacy of targeted therapy and chemotherapy drugs in BC cells.	[87]
Fluoroquinolones	Antibiotic	inhibits key enzymes involved 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	[48]
Ciclopirox	Antifungal Drugs	CPX works by chelating iron, and this influences many pathways such as ribonucleotide reductase, Myc, DJ-1, Wnt/β-catenin, DOHH/eIF5A/PEAK1, VEGFR-3/ERK1/2, ATR/Chk1/Cdc25A, and AMPK/TSC/mTORC1 signaling	[56]
Artemisinin	Antimalarial drugs	Induces autophagy-dependent apoptosis via AMPK-mTOR-ULK1 pathway	[56]
Penfluridol	Antipsychotic drugs	Induces lysosomal membrane permeabilization, releasing enzymes that lead to apoptosis; causes phosphatidylserine redistribution, an early apoptosis marker.	[57]

Table S10. Repurposed drugs along with their mechanism in melanoma.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Azacitidine	Demethylating agent	Induces hypomethylation by inhibiting DNMT1, thereby reactivating silenced genes. Increased expression of antigen-presenting molecules (e.g., HLA-A) and interferon (IFN) response genes	[88]
Carboplatin	Alkylating agent	DNA-damaging agent; synergizes with azacitidine to enhance immune visibility of tumors	[88]
Artemisinin	Antimalarial drugs	Suppresses IL-6-JAK-STAT3 pathway	[56]
Penfluridol	Antipsychotic drugs	Causes accumulation of unesterified cholesterol, disrupting membrane integrity; reduces tumor volume and inhibits proliferation in vivo.	[59]

Table S11. Repurposed drugs along with their mechanism in multiple myeloma.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Chloramphenicol	Antibiotic	Inhibits mitochondrial protein synthesis & decreased ATP	[49]
Leflunomide	Antirheumatic agents	DHODH Inhibition & Tyrosine Kinase Inhibition	[89]
Disulfiram	Disulfide	Copper-dependent glycolysis inhibition, ROS induction	[79]
Thalidomide	Immunomodulatory agents	Immunomodulation; targeting inflammatory pathways	[79]

Table S12. Repurposed drugs along with their mechanism in adenocarcinoma.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Aspirin	NSAIDs	Inhibits COX-1/COX-2 enzymes, reducing prostaglandin synthesis and modulating inflammatory pathway	[90]
Statins	Antihyperlipidemic drugs	Block HMG-CoA reductase, disrupting the mevalonate pathway and protein prenylation	[90]
Metformin	Antidiabetic drug	Activates AMPK, inhibiting mammalian target of rapamycin (mTOR) pathway and cellular proliferation	[90]

Table S13. Repurposed drugs along with their mechanism in NSCLC.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Auranofin	Antirheumatic agents	disrupt the redox balance in cancer cells by inhibiting thioredoxin reductase (TrxR). This inhibition leads to increased reactive oxygen species (ROS) levels, ultimately triggering cell death through apoptosis and other mechanisms	[49]
Artemisinin	Antimalarial drugs	Induces ER stress, suppresses asparagine synthetase, triggers apoptosis	[56]
Disulfiram	Disulfide	DSF/Cu inhibits NF-kB signaling, proteasome activity, and ALDH activity, inducing apoptosis; evokes ER stress and autophagy; binds NPL4, causing immobilization and cell death; generates ROS via copper chelation.	[58]

Table S14. Repurposed drugs along with their mechanism in squamous cell carcinoma.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Atovaquone	Anthelmintic Agents	Blocking Complex III leads to less respiration, less ATP, and reduced mitochondrial mass.	[49]
Disulfiram	Disulfide	DSF/Cu (50 mg/kg) induces autophagic cell death, inhibiting tumor growth in xenografts	[58]
Ketoconazole	Antifungal Drugs	Interferes with drug metabolism by inhibiting cytochrome P450 enzymes (e.g., CYP3A4), leading to increased exposure of co-administered drugs like lapatinib and reduced clearance of docetaxel, enhancing their anticancer effects.	[69]

Table S15. Repurposed drugs along with their mechanism in cervical cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Fluoroquinolones	Antibiotic	Exhibit anticancer activity by inhibiting key enzymes involved 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	[70]
Aspirin	NSAIDs	Inhibition of COX-2 reduces the production of pro-inflammatory prostaglandins that promote tumor growth, angiogenesis, and metastasis. Additionally, aspirin can disrupt tumor-promoting signaling pathways and influence cellular processes like apoptosis and angiogenesis.	[76]

Table S16. Repurposed drugs along with their mechanism in osteosarcoma.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Auranofin	Antirheumatic agents	Auranofin disrupts the redox balance in cancer cells by inhibiting thioredoxin reductase (TrxR) leading to increased reactive oxygen species (ROS) levels, ultimately triggering cell death through apoptosis and other mechanisms	[49]
Metformin	Antidiabetic drug	Blocking Complex I lowers respiration and ATP production, and also reduces insulin/IGF-1 levels.	[49]

Table S17. Repurposed drugs along with their mechanism in renal cancer.

Repurposed drug	Classification of the drug	Mechanism of action	Reference
Penfluridol	Antipsychotic drugs	Induces ER stress-mediated UPR, triggering autophagy and subsequent apoptosis; suppresses GLI1 oncogene and Ki-67 proliferation marker.	[57]
Ivermectin	Anthelmintic Agents	Ivermectin reduces mitochondrial membrane potential, inhibits mitochondrial respiration, and decreases ATP production. It also increases the expression of mitochondrial stress marker HEL in tumor tissues. Effects are reversed by mitochondrial fuel (acetyl-L-carnitine) and antioxidants (N-acetyl-L-cysteine).	[59]

Table S18. Repurposed drugs along with their mechanism in other various cancers.

Cancer type	Repurposed drug	Classification of the drug	Mechanism of action	Reference
GIST	Auranofin	Antirheumatic agents	disrupts the redox balance in cancer cells by inhibiting thioredoxin reductase (TrxR) leading to increased reactive oxygen species (ROS) levels, ultimately triggering cell death through apoptosis and other mechanisms.	[49]
Cholangiocarcinoma	Ivermectin	Anthelmintic Agents	Inhibits proliferation of KKU214 cholangiocarcinoma cells in a dose and time dependent manner. Induces cell cycle arrest in S phase and promotes apoptosis. Shows efficacy against gemcitabine-resistant KKU214 cells, suggesting utility against chemotherapy-resistant tumors.	[59]
B-cell lymphoma	Artemisinin	Antimalarial drugs	Activates CaMKK2-AMPK-ULK1 axis, triggers autophagy.	[56]
Endometrial cancer	Aspirin	NSAIDs	By inhibits COX-2, Aspirin reduces the production of pro-inflammatory prostaglandins that promote tumor growth, angiogenesis, and metastasis.	[76]
B- cell lymphoma & gallbladder cancer	Penfluridol	Antipsychotic drugs	Activates AMPK/PFKFB3 signaling, suppressing glycolysis and energy metabolism. Shows synergistic effect when combined with 2-deoxy-D-glucose (2-DG) or AMPK inhibitor Compound C.	[57]
Laryngeal carcinoma	Artemisinin	Antimalarial drugs	Suppresses STAT3, inhibits cancer stem cell-induced metastasis.	[56]
Medulloblastoma	Itraconazole	Antifungal Drugs	Itraconazole inhibits Hedgehog pathway by blocking smoothened receptor and reduces tumor growth by inhibiting AKT/mTOR signaling, which suppresses cell proliferation and survival. It also inhibits angiogenesis by reducing VEGF signaling, and by limiting tumor blood supply and blocking HER2/AKT signaling.	[69]
Nasopharyngeal carcinoma	Ivermectin	Anthelmintic Agents	Exerts cytotoxic effects in vitro by reducing PAK1 kinase activity, which inhibits the MAPK pathway.	[59]
Esophageal cancer	Penfluridol	Antipsychotic drugs	Inhibits glycolysis via activation of AMPK/FOXO3a/BIM pathway, leading to energy stress and apoptosis.	[68]

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Other References are listed in the main article.