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Repurposing Albendazole and Ivermectin in Cancer Patient Therapy

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ABSTRACT
Cancer remains a leading cause of mortality worldwide, and conventional treatments continue to face challenges related to drug resistance, adverse effects, and limited therapeutic efficacy. Drug repurposing has emerged as a promising strategy for identifying alternative anticancer agents using existing medications. This review aimed to evaluate the potential anticancer effects and mechanisms of action of albendazole and ivermectin, two anthelmintic drugs traditionally used to treat parasitic infections. A narrative literature review was conducted using PubMed, Google Scholar, and Elsevier databases to identify relevant studies published between 2020 and 2025. Studies investigating the anticancer effects of anthelmintic agents in clinical or preclinical settings were considered, and 15 studies were included in the synthesis. The extracted data covered cancer types, experimental models, treatment concentrations, exposure durations, half-maximal inhibitory concentration (IC_{50}), anticancer outcomes, and proposed mechanisms. The findings indicated that albendazole demonstrated antiproliferative activity against colorectal, gastric, breast, prostate, melanoma, and cervical cancer models. Its proposed mechanisms included G2/M cell cycle arrest, apoptosis induction, increased reactive oxygen species, and inhibition of angiogenesis and metastasis. Ivermectin exhibited anticancer effects across several cancer models, including colorectal, osteosarcoma, urothelial, breast, esophageal, and prostate cancers. Its mechanisms involved oxidative stress, mitochondrial dysfunction, DNA damage, apoptosis, and modulation of the tumor microenvironment through increased T-cell infiltration and reduced immunosuppressive activity. Both agents also demonstrated potential synergistic effects when combined with selected chemotherapeutic drugs or immune checkpoint inhibitors. However, IC_{50} values varied according to cancer cell type, drug concentration, and treatment duration. In conclusion, albendazole and ivermectin represent promising candidates for further investigation through drug repurposing. Nevertheless, the available evidence remains predominantly preclinical, and well-designed clinical trials are necessary to establish their safety, optimal dosage, pharmacokinetics, drug interactions, and therapeutic efficacy in cancer patients.
Keyword: *Albendazole, Ivermectin, Anthelmintic, Cancer therapy.*

Introduction

Cancer remains one of the leading causes of mortality worldwide, with conventional treatments such as chemotherapy, radiotherapy, and targeted therapy forming the backbone of clinical management but despite significant advancements, treatment resistance, adverse effects, and limited efficacy in certain cancer types continue to challenge therapeutic outcomes (Alavi & Ebrahimi Shahmabadi, 2021; Jay Feliciano et al., 2023)

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Before 1970, cancer treatment primarily relied on surgery, early forms of radiotherapy, and the initial chemotherapeutic agents, many of which were developed from chemical warfare compounds (Sonkin et al., 2024). Since the 1970s, significant advancements in pharmaceutical science have expanded the range of therapeutic options available to clinicians (Kaur et al., 2023). Cancer treatment comes not without any side effects and patients spent an average of 4.5 hours per visit managing these effects (Henry et al., 2008).

Off-label use is usually intentional and done to meet medical needs when other treatments are not suitable or available (Rusz et al., 2021). Anthelmintic agents such as albendazole and ivermectin, traditionally used to treat parasitic worm infections, have shown promising anti-cancer properties in preclinical and early-phase clinical studies (Huang et al., 2021). More recently, emerging research has highlighted the potential role of anthelmintic drugs as adjunctive therapies in cancer treatment (Stolfi et al., 2023).

Overcome multidrug resistance, anthelmintic drugs have emerged as promising candidates for cancer drug repurposing. However, their comparative anticancer efficacy and clinical potential remain unclear due to fragmented evidence and heterogeneous study designs. This review synthesizes current evidence on the anticancer effects of anthelmintic drugs and compares their therapeutic potential in cancer treatment.

Albendazole and ivermectin have attracted attention in cancer research because they may affect biological processes essential for tumor development and progression. Albendazole, a benzimidazole anthelmintic, primarily acts by disrupting microtubule formation in susceptible parasites. In cancer models, this mechanism has been associated with impaired cell division, cell cycle arrest, and the induction of apoptosis. In addition, studies have suggested that albendazole may influence oxidative stress, angiogenesis, and molecular pathways involved in tumor growth. These findings provide a scientific basis for investigating its potential application beyond its conventional antiparasitic indications.

Ivermectin, another widely recognized antiparasitic agent, has also demonstrated potential anticancer activity in experimental studies. Its reported effects include the inhibition of cancer cell proliferation, induction of apoptosis, mitochondrial dysfunction, and increased oxidative stress. Furthermore, ivermectin may influence tumor–immune interactions by modifying the tumor microenvironment and enhancing antitumor immune responses in certain experimental models. Research has also explored its use in combination with chemotherapy and immune checkpoint inhibitors, suggesting that it may enhance treatment responses under specific experimental conditions. However, the magnitude and mechanisms of these effects vary across cancer types, cell lines, drug concentrations, and treatment durations.

Despite these promising findings, important uncertainties remain regarding the translation of experimental evidence into clinical practice. Differences in study designs, experimental models, treatment protocols, and outcome measurements make it difficult to compare the anticancer potential of albendazole and ivermectin directly. Moreover, their effective concentrations in laboratory studies may not correspond to safely achievable exposures in humans. Therefore, evidence of anticancer activity in preclinical models should not be interpreted as proof of clinical effectiveness, and these drugs should not replace established cancer treatments without adequate clinical evidence. A systematic synthesis of the available literature is needed to identify consistent findings, evaluate the proposed mechanisms of action, and clarify existing research gaps. This review consequently compares the reported anticancer effects of albendazole and ivermectin, examines their potential as repurposed therapeutic agents, and identifies priorities for future research, particularly regarding safety, pharmacokinetics, optimal dosing, drug interactions, and clinical efficacy.

Method

In this review, studies were included if they evaluated the use or potential anticancer effects of anthelmintic agents, including albendazole and ivermectin, in cancer models. Eligible study designs comprised randomized and non-randomized clinical trials, cohort studies, case-control studies, and preclinical studies (in vitro or in vivo) published between 2020 and 2025. Studies were excluded if they were reviews, editorials, conference abstracts without complete data, unrelated to cancer or anthelmintic drugs, duplicates, non-peer-reviewed publications, or lacked essential information.

A comprehensive literature search was conducted in PubMed, Google Scholar, and Elsevier using combinations of the keywords “anthelmintic,” “albendazole,” “ivermectin,” “cancer,” and “tumor.” The search was limited to research articles published from 2020 to 2025. All identified records were collected and screened by title and abstract, and duplicate records were removed. Potentially relevant studies were then assessed through full-text screening according to the predefined inclusion and exclusion criteria.

For data extraction, information from the eligible studies was systematically collected, including author and publication year, anthelmintic agent, cancer type or cell line, study design, treatment dose or concentration, treatment duration, IC50, reported anticancer effects, and proposed mechanisms of action. The included studies were qualitatively synthesized by grouping findings according to the anthelmintic agent, cancer model, treatment characteristics, and reported anticancer outcomes with findings were summarized descriptively and quantitatively.

Result and discussion

This study used a narrative literature review design to synthesize evidence on the anticancer effects of anthelmintic drugs. Literature searches were conducted in PubMed, Google Scholar, and Elsevier for publications from 2020 to 2025 using the keywords "anthelmintic," "albendazole," "ivermectin," "cancer," and "tumor." Studies were selected based on predefined inclusion and exclusion criteria. Data were extracted from the 15 included studies, including publication year, anthelmintic drug, cancer type or cell line, study design, treatment concentration or dose, treatment duration, IC50, anticancer outcomes, and proposed mechanisms of action. The extracted findings were qualitatively synthesized by grouping studies according to the anthelmintic agent, cancer model, treatment characteristics, and reported anticancer effects. Due to differences in study designs, cancer models, treatment conditions, and outcome measures, the findings were compared descriptively rather than through quantitative meta-analysis.

Table 1. Anthelmintics effects on cancer cells

Reference	Drug	Result
(Fatima et al., 2024)	Albendazole	Albendazole inhibited colorectal cancer growth and sensitized chemoresistant cells to 5-FU and oxaliplatin. It promoted apoptosis by modulating RNF20, inducing G2/M arrest, and downregulating anti-apoptotic Bcl-2 family genes.
(Yang et al., 2021)	Albendazole	Albendazole suppressed key oncogenic pathways in gastric cancer cells by inhibiting STAT3/5 activation, blocking upstream JAK1/2 and Src phosphorylation, and increasing SHP-1 expression. These effects, driven by elevated ROS levels from GSH/GSSG imbalance, led to enhanced apoptosis.
(Javdan et al., 2023)	Albendazole	Albendazole significantly reduced Tau and Stathmin gene expression and inhibited cell migration in MCF-7 and MDA-MB-231 metastatic breast cancer cell lines, indicating its potential to prevent metastasis in breast cancer.
(Gaikwad et al., 2023)	Albendazole	Albendazole can bind strongly and stably to VEGFR-2, a receptor involved in tumor angiogenesis. By interacting with key residues like Asp1046 and demonstrating stable molecular

Reference	Drug	Result
		dynamics, albendazole may inhibit VEGFR-2 activity, thereby potentially preventing tumor growth through anti-angiogenic mechanisms.
(Kim et al., 2021)	Albendazole	Albendazole inhibited prostate cancer cell growth, migration, and colony formation by increasing ROS levels and suppressing antioxidant and Wnt/β-catenin signaling genes. Its effects were ROS-dependent and selective to cancer cells, suggesting its potential as an anticancer agent for prostate cancer.
(Zhu et al., 2022)	Albendazole	Albendazole inhibited melanoma cell proliferation by inducing G2/M cell cycle arrest and apoptosis. It also showed synergistic effects with the CDK4/6 inhibitor palbociclib in vitro and in vivo, suggesting its potential use in combination melanoma therapy.
(Hernández-Castillo et al., 2021)	Albendazole	Significantly reduced proliferation (>85%) in cervical cancer cell lines after 72 hours, indicating enhanced bioavailability and potential for sustained anticancer delivery.
(Zhou et al., 2021)	Ivermectin	Ivermectin suppressed colorectal cancer cell growth by inducing ROS-dependent mitochondrial apoptosis and S phase arrest. It upregulated proapoptotic markers and ROS levels, while downregulating Bcl-2. These effects were reversed by NAC, confirming a ROS-mediated mechanism and supporting ivermectin's anticancer potential in CRC.
(Hu et al., 2022)	Ivermectin	Ivermectin inhibited osteosarcoma cell growth and migration, induced caspase-dependent apoptosis, and acted synergistically with doxorubicin. In vivo, the combination significantly enhanced tumor suppression without notable toxicity. Mechanistically, ivermectin triggered oxidative stress and mitochondrial dysfunction, supporting

Reference	Drug	Result
		its potential as a repurposed therapy for osteosarcoma, including in chemoresistant cases.
(Tung et al., 2022)	Ivermectin	Ivermectin inhibited cell growth, induced cell cycle arrest, and triggered apoptosis in urothelial carcinoma (UC) cells through both intrinsic and extrinsic caspase pathways. Apoptosis was mediated via activation of JNK signaling, highlighting ivermectin's potential as a novel therapeutic agent for UC treatment.
(GÜLER & GÜNAYDIN AKYILDIZ, 2023)	Ivermectin	Ivermectin induced apoptosis in breast cancer cells by increasing oxidative stress and causing DNA damage. These findings support its potential as a repurposed anticancer agent for breast cancer therapy.
(Jiménez-Gaona et al., 2023)	Ivermectin	19% of cancer patients reported using ivermectin alongside standard cancer therapies as an alternative treatment. While some perceived health improvements, medical professionals emphasized the lack of scientific validation and regulatory approval for its use in cancer, highlighting the need for further pharmacological research through in vitro studies.
(Xu et al., 2021)	Ivermectin	Ivermectin significantly inhibited ESCC cell proliferation in vitro and in vivo by inducing apoptosis through mitochondrial dysfunction. This effect was associated with ROS accumulation, suppression of the NF-κB pathway, and an increased Bax/Bcl-2 ratio, suggesting ivermectin's potential as a therapeutic agent for ESCC.
(Lv et al., 2022)	Ivermectin	Ivermectin inhibited prostate cancer growth by inducing G0/G1 arrest, apoptosis, and DNA damage, while suppressing AR signaling. It directly targeted FOXA1 and Ku70/Ku80, reducing chromatin accessibility and impairing DNA repair. These combined effects led to increased DNA damage and cell death, supporting

Reference	Drug	Result
		ivermectin's potential as a targeted therapy for prostate cancer.
(Draganov et al., 2021)	Ivermectin	In combination with anti-PD1 therapy, it synergistically suppressed tumor growth, reduced relapse, and achieved cures in metastatic models, suggesting ivermectin's strong potential as an immunomodulatory partner in breast cancer immunotherapy.

Demonstrated in table 1, Albendazole, reported in 7 studies, inhibited tumor growth through mechanisms such as G2/M cell cycle arrest, apoptosis induction, ROS generation, and inhibition of angiogenesis and metastasis. It showed potential in colorectal, gastric, breast, prostate, melanoma, and cervical cancers, with enhanced effects when combined with other therapies or delivered via nanoparticles. Ivermectin, in 8 studies, suppressed cancer cell proliferation by inducing oxidative stress, mitochondrial dysfunction, DNA damage, and apoptosis. It acted through pathways like JNK, NF-κB, and AR signaling, and demonstrated synergy with chemotherapies and immune checkpoint inhibitors.

Table 2. Anthelmintics drugs and Inhibition concentration range

Reference	Drug	Cells used	Treatment duration	IC50
(Fatima et al., 2024)	Albendazole	SW620	48 hours	3.8 µM
		HCT116	48 hours	2.7 µM
		SW48	48 hours	3.7 µM
		RKO	48 hours	3.5 µM
		MC38	48 hours	3.3 µM
(Zhu et al., 2022)	Albendazole & palbociclib	A375 (Human melanoma cells)	48 hours	1.588 µM
		SK-MEL-28 (Human melanoma cells)	48 hours	3.183 µM
	Ivermectin	SW480	12 hours	16.17 µM

Reference	Drug	Cells used	Treatment duration	IC50
(Zhou et al., 2021)			24 hours	15.34 µM
			36 hours	12.11 µM,
		SW1116	12 hours	7.60 µM
			24 hours	6.27 µM
			36 hours	5.76 µM
(Tung et al., 2022)		T24	24 hours	20.5 µM
			48 hours	17.4 µM
			72 hours	16.6 µM
		RT4	24 hours	26.7 µM
			48 hours	14.9 µM
			72 hours	10.0 µM
(GÜLER & GÜNAYDIN AKYILDIZ, 2023)		MCF	24 hours	24.04 µM
		MDA MB231	24 hours	34.12 µM
		184A1	24 hours	68.51 µM
(Lv et al., 2022)		LNCaP	24 hours	14.13 µM
			48 hours	8.585 µM
			72 hours	3.074 µM
		C4-2	24 hours	14.06 µM
			48 hours	7.391 µM
			72 hours	3.447 µM
		22RV1	24 hours	6.845 µM
			48 hours	5.004 µM
			72 hours	1.972 µM
		DU145	72 hours	9.232 µM
		PC-3	72 hours	8.855 µM

Reference	Drug	Cells used	Treatment duration	IC50
		nontumorigenic BPH-1 cells	72 hours	11.10 µM
		benign prostatic hyperplasia (BPH)	72 hours	11.85 µM

Based in Table 2, IC_{50} values varied according to cancer type, cell line, and treatment duration. For colorectal cancer, albendazole showed IC_{50} values of 2.7–3.8 µM at 48 hours across SW620, HCT116, SW48, RKO, and MC38 cells. Ivermectin showed IC_{50} values of 12.11–16.17 µM in SW480 and 5.76–7.60 µM in SW1116, with lower IC_{50} values at longer exposure times. In melanoma, albendazole combined with palbociclib showed IC_{50} values of 1.588 µM in A375 and 3.183 µM in SK-MEL-28 at 48 hours. For bladder cancer, ivermectin IC_{50} values decreased from 20.5 to 16.6 µM in T24 and from 26.7 to 10.0 µM in RT4 between 24 and 72 hours. In breast cancer, IC_{50} values at 24 hours were 24.04 µM (MCF-7), 34.12 µM (MDA-MB-231), and 68.51 µM (184A1). In prostate cancer, ivermectin IC_{50} values generally decreased with longer exposure, with the lowest value of 1.972 µM in 22Rv1 at 72 hours. Non-malignant prostate cells showed higher IC_{50} values of 11.10–11.85 µM at 72 hours.

1.1 Albendazole proliferation ability

G2/M cell cycle is one of the cycles that plays a critical role in maintaining genomic integrity by ensuring that cells do not proceed to mitosis with damaged DNA. Disruption or dysregulation of this regulatory mechanism can compromise cell cycle control and contribute to oncogenesis (Wang et al., 2024). Albendazole mechanism of action in cancer treatment works by blocking the G2/M which cause a hold in mitosis and elevation of apoptosis (Fatima et al., 2024). In RNA sequencing (RNA-seq) analysis that was conducted on ABZ-treated A375 cells, identified 1,478 significantly upregulated and 887 downregulated which revealed a marked accumulation of A375 and SK-MEL-28 cells in the G2/M and sub-G1 phases, along with a corresponding reduction in the G0/G1 population, confirming ABZ-induced G2/M phase arrest and apoptotic activity (Zhu et al., 2022). Within the In vitro study of 50 µM ABZ for 24 hours used on SNU-16 and SNU-1 cells revealed population of dead cells had appeared in treated cells and none in non-treated cells ± SD. *** $p < 0.001$ (Yang et al., 2021). In anti-proliferative effects of albendazole, Albendazole significantly reduced the colony-forming ability of PC3 and DU145 prostate cancer cells in a dose-dependent manner. In a 7-day clonogenic assay, ABZ at 0.1 µM and

0.5 μM led to marked decreases in colony area (PC3) and colony number (DU145). The surviving fraction, calculated based on plating efficiency, was significantly reduced compared to control, with *P < 0.05 and ***P < 0.001 (Kim et al., 2021). Antiproliferative effects of albendazole also tested in an albendazole-loaded mesoporous silica nanoparticles chitosan coated and non-coated shows after 72 hours treatment against four human cervical cancer cell lines CaSki cells showed a 40% reduction in proliferation at 1 μg/mL and up to 70% at 15 μg/mL nanoparticles, with chitosan-coated particles slightly more effective. HeLa, SiHa, and C33-A cells showed 20–30% inhibition below 15 μg/mL and nearly 50% inhibition in HeLa cells above 15 μg/mL. Compared to free albendazole (474.8 nmol), nanoparticles reduced viability by an additional 15–25%, likely due to improved uptake (Hernández-Castillo et al., 2021).

1.2 Ivermectin

Ivermectin has the capability to be used as an additional or as monotherapy for cancer treatment. Preclinical studies show that on cellular level, ivermectin induces immunogenic cell death and enhances T cell infiltration in breast tumours. Acting through the ATP/P2X4/P2X7 axis, it reduces immunosuppressive cells and increases the Teff/Treg ratio. It is also can be found when ivermectin combined with anti-PD1 therapy, it significantly reduced tumour growth ($p < 0.03$), relapse ($p < 0.001$), and improved complete response rates ($p < 0.01$), with synergistic effects confirmed in both adjuvant ($p < 0.007$) and metastatic models ($p < 0.001$) (Draganov et al., 2021). In other metastatic model, IVM significantly reduced lung metastasis in mice, decreased serum MPO-DNA and neutrophil elastase-DNA levels, suppressed MDSCs, and increased $CD8^+$ T cell infiltration (Zhang et al., 2022). To be used as a cancer treatment, ivermectin dose should be adjusted to achieve the effects the patient need Low doses (2.5 and 5 μM) caused S phase arrest (Zhou et al., 2021). Other research also found that an increase of GSH is more significant on subject that was treated with low dose ivermectin compared to the control ($p< 0.05$) but it shows lower GSH in a higher dose of ivermectin treatment ($p< 0.01$) (Alghamdi et al., 2022). Combination of IVM with other medication were giving significant result. Research on the use of IVM with Ovarian cancer a study indicates that a combination of ivermectin and pitavastatin managed to inhibit cell growth within the ivermectin concentration of FUOV-1 (4.4, 6.2, and 8.8 μM), Ovcar-4 (4.2, 5.9, and 8.4 μM), Ovcar-8 (4.6, 6.1, and 8.3 μM), COV-318 (4.4, 6.6, and 9.0 μM), COV-362 (4.5, 6.0, and 8.0 μM), and Ovsaho (4.6, 6.1, and 8.3 μM). The combination index (CI), representing the interaction between the two drugs, showed significant deviations from 1 (indicating synergy or antagonism) at several concentrations, with statistical significance marked by *$p \leq 0.05$ and **$p < 0.01$ (paired t-test, mean ± SD, n = 3–4) (Jawad & Richardson, 2023). Other combination with chemotherapy

medications, showed that ivermectin (IVM), alone or combined with adriamycin (MCF-7, MDA-MB-231) or vincristine (HCT-8, HCT-116), significantly inhibited cancer cell migration over 24 hours. Compared to controls, IVM treatment reduced wound closure by over 50% in all cell lines (**P < 0.01) (Jiang et al., 2022). Another combination of ivermectin (3 µM) with antineoplastic paclitaxel (0.001 µM) and significantly enhanced antiproliferative effects compared to either agent alone, with statistical significance noted (*p<0.001, **p=0.008, ***p=0.006) and apoptotic cell percentages (*p=0.002, **p=0.001), indicating a synergistic effect on inhibiting viability and inducing apoptosis (Morinaga et al., 2025).

Conclusion

Both albendazole and ivermectin demonstrate significant anticancer potential through diverse mechanisms and show promise as either monotherapy or in combination with other agents. Albendazole exerts antiproliferative effects by inducing G2/M cell cycle arrest and apoptosis, as evidenced by RNA-seq analysis, clonogenic assays, and caspase activation. It also reduces viability in various cancer cell lines, including prostate, gastric, and cervical cancers, with enhanced efficacy when delivered via nanoparticles. Ivermectin, on the other hand, induces immunogenic cell death, modulates the tumor microenvironment by enhancing T cell infiltration and reducing suppressive immune cells, and shows synergistic effects with immune checkpoint inhibitors and chemotherapeutic agents. Its combination with drugs like pitavastatin, adriamycin, vincristine, and paclitaxel significantly enhances cancer cell inhibition and apoptosis. Overall, the compelling preclinical data suggest that both albendazole and ivermectin warrant further investigation as effective, low-cost repurposed agents in cancer.

Suggestion

Future studies should focus on clinical trials to evaluate the potential and safety of albendazole and ivermectin in cancer patients. Further research is also needed to determine their optimal dosage, treatment duration, pharmacokinetics, and potential drug interactions.

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