International Journal of Medical Science and Pharmaceutical Research

ISSN(e): 3050-5720
ISSN(p): 3050-5712

Vol. 3 (4): 124-131, April 2026

Home Page: https://ijmspr.org/

A Review on Recent Advances of Anticancer Potentials of Some Medicinal Plants

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KEYWORDS:

Anticancer, Medicinal plants, Phytochemicals, Apoptosis, Molecular mechanisms, Treatment modalities, and Cancer types.

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DOI: 10.55677/IJMSPR/2026-3050-I404

Published: April 13, 2026

ABSTRACT

Cancer continues to represent a major global health challenge, with conventional treatment modalities often constrained by systemic toxicity, drug resistance, and high economic burden. In recent years, medicinal plants and their bioactive phytochemicals have gained considerable attention as promising anticancer agents due to their multitargeted mechanisms of action and comparatively favorable safety profiles. This review summarizes recent advances in the anticancer potential of some medicinal plants against specific cancer types, compositions of different plants' bioactive compounds as well as molecular mechanisms of the plants. Different mechanisms including induction of apoptosis, cell cycle arrest, inhibition of angiogenesis and metastasis, modulation of immune responses, and regulation of critical oncogenic signaling pathways such as PI3K/Akt, NF-κB, STAT3, and Wnt/β-catenin have been discussed. Despite promising preclinical and early clinical findings, challenges related to poor bioavailability, lack of standardization and limited large-scale clinical validation remain significant barriers to clinical translation. Advances in drug delivery systems, particularly nanotechnology-based approaches, along with standardized formulations and rational combination strategies, offer promising solutions. This review highlights the therapeutic potential of medicinal plant-derived compounds as complementary or adjunctive agents in cancer management and underscores the need for rigorous clinical studies to facilitate their integration into evidence-based oncology.

Cite the Article: *Usman, K., Bello, M., Usman, M., Hamza, A.B., Naibi, AA., Danmaliki, N.I., Abdullahi, M., Adamu, U., Bello, N., Yusuf, A., Garba, A. (2026). A Review on Recent Advances of Anticancer Potentials of Some Medicinal Plants. International Journal of Medical Science and Pharmaceutical Research, 3(4), 124-131. https://doi.org/10.55677/IJMSPR/2026-3050-I404*

1.0 INTRODUCTION

Cancer is characterized by uncontrolled cell proliferation, resistance to cell death, and metastatic spread, and remains a leading cause of global morbidity and mortality despite advances in diagnostics and therapeutics (Sung *et al.,* 2021). Although chemotherapy and radiotherapy remain central to cancer management, their clinical utility is often compromised by systemic toxicity and the emergence of resistance. Consequently, increasing attention has been directed toward plant-derived bioactive compounds as complementary or alternative anticancer agents due to their pleiotropic pharmacological effects (Newman & Cragg, 2023). Cancer encompasses a heterogeneous group of diseases that can arise in virtually any tissue or organ. Broadly, cancers are classified according to their tissue of origin. Carcinomas, which originate from epithelial cells, represent the most common category and include malignancies such as breast, lung, colorectal, and prostate cancers. Sarcomas arise from mesenchymal tissues including bone, muscle, and connective tissue. Leukemias are hematological malignancies characterized by abnormal

proliferation of white blood cells in the bone marrow and peripheral blood, whereas lymphomas and multiple myeloma originate from lymphoid tissues and plasma cells, respectively (Kumar *et al.,* 2024).

Additionally, melanomas develop from melanocytes and are notable for their aggressive metastatic potential, while central nervous system tumors, such as gliomas, arise from neural or glial cells. Each cancer type exhibits distinct molecular signatures, clinical behaviors, and therapeutic responses, underscoring the need for diverse and targeted treatment strategies. Medicinal plants have historically contributed to modern oncology, most notably through the development of chemotherapeutic agents such as vincristine, vinblastine, and paclitaxel, which were originally isolated from plant sources and remain integral components of contemporary cancer treatment regimens (Newman and Cragg, 2020). Beyond serving as sources of cytotoxic drugs, plants provide a vast reservoir of secondary metabolites including alkaloids, flavonoids, terpenoids, and phenolic compounds that can modulate multiple signaling pathways involved in carcinogenesis. Plant-derived compounds may contribute to cancer management through several mechanisms. They can inhibit tumor cell proliferation by inducing cell cycle arrest at specific checkpoints, promote apoptosis via intrinsic and extrinsic pathways, and suppress angiogenesis by downregulating pro-angiogenic factors such as vascular endothelial growth factor (VEGF) (Ahmed *et al.,* 2022).

Many phytochemicals also exhibit antioxidant properties that reduce oxidative stress–mediated DNA damage, thereby potentially preventing tumor initiation and progression. Additionally, certain plant metabolites modulate inflammatory pathways, including NF-κB and STAT3 signaling, which are often dysregulated in cancer. Importantly, some phytochemicals have demonstrated the ability to sensitize tumor cells to conventional chemotherapy and radiotherapy, thereby overcoming drug resistance and enhancing therapeutic efficacy while potentially reducing required dosages and associated toxicity. These multifaceted mechanisms highlight the therapeutic promise of medicinal plants not only as sources of novel anticancer agents but also as adjuvants in integrative cancer treatment strategies. This review highlights recent information about selected medicinal plants, their active constituents, molecular mechanisms of action, and relevance to specific cancer types.

2.0 MEDICINAL PLANTS WITH ANTICANCER POTENTIALS

2.1 Garlic *(Allium sativum)*

Garlic (*Allium sativum*) is a perennial bulbous plant belonging to the family of Amaryllidaceae (formerly classified under Liliaceae). It is one of the oldest cultivated medicinal and culinary plants and has been used for thousands of years in traditional systems of medicine, including Ayurveda, Traditional Chinese Medicine, and Greco-Arab medicine. Garlic is believed to have originated in Central Asia, particularly regions surrounding present-day Kazakhstan, Uzbekistan, and Western China. Today, it is widely cultivated across temperate and subtropical regions worldwide, including Asia, Europe, North Africa, and the Americas, with major production in countries such as China, India, and Spain. Garlic's therapeutic properties are largely attributed to its rich content of organosulfur compounds, particularly allicin, which is formed enzymatically when garlic cloves are crushed or chopped. These bioactive constituents have demonstrated significant antiproliferative, anti-inflammatory, and antioxidant activities in various experimental models. Organosulfur compounds derived from garlic have shown antiproliferative and pro-apoptotic effects in colorectal and gastric cancer cell lines (McAlpine, 2024). Preclinical studies indicate that garlic extracts suppress tumor growth through modulation of oxidative stress, induction of apoptosis, inhibition of cell cycle progression, and enhancement of phase II detoxification enzymes (Amagase and Petesch, 2019). Additionally, these compounds may inhibit angiogenesis and reduce the activation of carcinogens in the gastrointestinal tract. Epidemiological studies suggest an inverse association between garlic consumption and the risk of gastrointestinal and prostate cancers. However, while laboratory and observational data are promising, causality and optimal therapeutic dosing remain to be established through well-designed randomized controlled clinical trials.

Bioactive constituents: Allicin, organosulfur compounds (e.g., diallyl sulfide, diallyl disulfide, diallyl trisulfide).

Cancer types: Colorectal, gastric, and prostate cancers.

2.2 Green Tea (*Camellia sinensis*)

Green tea is a beverage produced from the leaves of Camellia sinensis, a perennial evergreen shrub in the family Theaceae. Unlike black tea, green tea is unfermented and minimally oxidized during processing, which helps preserve its high content of polyphenolic compounds, especially catechins such as epigallocatechin-3-gallate (EGCG) (Radeva-Ilieva, 2025).The tea plant is believed to have originated in East Asia, particularly in China and Northeast India, and is now widely cultivated across subtropical and tropical regions of Asia, Africa, and South America, with China and Japan being major producers of green tea (Knowles *et al.,* 2025). The principal bioactive constituent of green tea, EGCG, has been extensively studied for its anticancer properties. EGCG exhibits a range of biological activities that can suppress carcinogenesis, including induction of apoptosis, cell cycle arrest, and inhibition of angiogenesis and metastasis. In vitro and animal studies show that EGCG modulates key signaling pathways implicated in cancer cell survival and proliferation, notably PI3K/Akt, MAPK, and NF-κB, which are involved in cell growth, apoptosis regulation, and inflammatory responses. EGCG has also been shown to downregulate matrix metalloproteinases and vascular endothelial growth factor (VEGF), thereby limiting tumor invasion and neovascularization (Chen *et al.,* 2021).

Preclinical evidence supports anticancer effects of EGCG across several cancer types. For example, EGCG inhibits proliferation and induces apoptosis in lung cancer cell lines and animal models; in prostate cancer, it interferes with androgen and growth factor signaling and reduces tumor growth; in leukemia models, EGCG has been shown to provoke apoptosis and DNA damage, partly through modulation of signaling pathways and oxidative stress (Sharma *et al.,* 2024). Epidemiological and early clinical observations suggest that regular green tea consumption or green tea polyphenol supplementation may be associated with a reduced risk of certain cancers, including prostate and lung cancers. Some clinical studies involving green tea extracts in patients with chronic lymphocytic leukemia have reported decreases in leukemic cell counts and improvements in disease biomarkers, although results are variable across populations and study designs (Shanafelt *et al.,* 2013). These inconsistencies may be related to differences in tea dosage, preparation methods, genetic and lifestyle factors, and the bioavailability of catechins in humans, highlighting the need for well-controlled, large-scale trials to confirm efficacy and establish optimal therapeutic strategies (clinical evidence reviews).

Bioactive constituent: Epigallocatechin-3-gallate (EGCG)

Cancer types: Lung, prostate, and leukemia

2.3 Turmeric *(Curcuma longa)*

Turmeric (*Curcuma longa*) is a perennial rhizomatous herbaceous plant belonging to the family Zingiberaceae, which also includes ginger (Zingiber officinale) and cardamom (Elettaria cardamomum). The plant is primarily valued for its underground rhizome, which is dried and ground to produce the characteristic yellow-orange spice widely used in culinary and medicinal applications. Turmeric is believed to have originated in South Asia, particularly India, where it has been cultivated for over 4,000 years and holds an important role in Ayurvedic and traditional medicine (Mishra, 2023). Today, it is extensively cultivated in tropical and subtropical regions, especially in India (the largest global producer), as well as China, Southeast Asia, Africa, and parts of Central America. Curcumin, a polyphenolic compound responsible for turmeric's distinctive color, is the principal bioactive constituent contributing to its pharmacological properties. Extensive in vitro and in vivo studies have demonstrated that curcumin possesses broad-spectrum anticancer activity across multiple tumor types (Akter *et al.,* 2025). Its anticancer effects are attributed to its ability to suppress tumor cell proliferation, induce apoptosis, inhibit angiogenesis, and prevent invasion and metastasis. At the molecular level, curcumin acts as a multitargeted agent, modulating numerous signaling pathways involved in carcinogenesis. Notably, it inhibits activation of nuclear factor kappa B (NF-κB), a transcription factor that regulates genes associated with inflammation, cell survival, and tumor progression.

Curcumin also suppresses signal transducer and activator of transcription 3 (STAT3), thereby reducing tumor cell proliferation and resistance to apoptosis. Additionally, curcumin interferes with the Wnt/β-catenin signaling pathway, which plays a critical role in colorectal and other cancers by promoting uncontrolled cellular proliferation (Hewlings & Kalman, 2023). Beyond these pathways, curcumin modulates PI3K/Akt, MAPK, and p53 signaling, highlighting its pleiotropic mode of action. In breast cancer models, curcumin has been shown to downregulate HER2 expression, inhibit estrogen receptor signaling, and sensitize tumor cells to chemotherapeutic agents such as paclitaxel and doxorubicin (Lai *et al.,* 2024). In colorectal cancer, curcumin suppresses tumor growth by targeting inflammatory mediators and β-catenin–mediated transcription. In pancreatic cancer, one of the most aggressive malignancies, curcumin inhibits desmoplastic reactions and reduces tumor cell resistance to gemcitabine. Similarly, in bladder cancer, curcumin has demonstrated antiproliferative and pro-apoptotic effects, partly through modulation of oxidative stress and inflammatory pathways.

Bioactive constituent: Curcumin

Cancer types: Breast, colorectal, pancreatic, and bladder cancers

2.4 Ginger *(Zingiber officinale)*

Ginger (Zingiber officinale) is a perennial herbaceous plant belonging to the family Zingiberaceae, which also includes turmeric (Curcuma longa) and cardamom (Elettaria cardamomum). The plant is primarily cultivated for its underground rhizome, commonly referred to as ginger root, which is widely used as a culinary spice and traditional medicinal agent. Ginger is believed to have originated in Southeast Asia, particularly in regions of present-day India and China. Today, it is extensively cultivated in tropical and subtropical regions worldwide, including India (one of the largest producers), China, Nigeria, Indonesia, Thailand, and other parts of Asia and Africa (Garza-Cadena *et al.,* 2023)). The pharmacological activity of ginger is largely attributed to its bioactive phenolic compounds, particularly 6-gingerol (the major pungent compound in fresh ginger) and 6-shogaol (a dehydration product formed during drying or heating). These compounds have demonstrated significant anticancer potential in various in vitro and in vivo models. Phenolic constituents of ginger exhibit antiproliferative, pro-apoptotic, anti-inflammatory, and anti-angiogenic properties across multiple cancer types, including ovarian and gastrointestinal cancers (Plengsuriyakarn *et al.,* 2019).

Mechanistically, 6-gingerol and 6-shogaol suppress cancer cell proliferation by inducing cell cycle arrest and promoting apoptosis through both intrinsic (mitochondrial) and extrinsic apoptotic pathways. They modulate key oncogenic signaling cascades such as PI3K/Akt/mTOR and NF-κB, which are frequently dysregulated in cancer and contribute to tumor growth, survival, and resistance

to therapy (Singh & Shukla, 2025). In ovarian cancer models for example, 6-gingerol has been shown to inhibit cellular migration and invasion, partly by downregulating matrix metalloproteinases (MMPs) and inflammatory mediators. Both 6-gingerol and 6-shogaol suppress the production of angiogenic factors, including vascular endothelial growth factor (VEGF) and interleukin-8 (IL-8), thereby limiting neovascularization required for tumor progression and metastasis. Additionally, 6-shogaol has demonstrated potent anti-angiogenic activity by directly inhibiting endothelial cell proliferation, migration, and tube formation, effectively impairing the vascular support system essential for tumor expansion (Bischoff-Kont *et al.,* 2022).

Bioactive constituents: 6-Gingerol, 6-Shogaol (phenolic compounds)

Cancer types: Ovarian, gastrointestinal, and other solid tumors

2.5. Grape *(Vitis vinifera)*

Grape (*Vitis vinifera*) is a deciduous woody climbing vine belonging to the family Vitaceae. It is one of the oldest cultivated fruit crops in human history, primarily valued for fresh consumption, juice, raisins, and wine production (Springer, 2025). The plant is believed to have originated in the Mediterranean region and Western Asia, particularly around the Near East and the Caucasus. Today, Vitis vinifera is extensively cultivated in temperate regions worldwide, including Southern Europe (e.g., Italy, Spain, and France), North and South America, South Africa, Australia, and parts of Asia (Marei, 2025). Beyond its nutritional importance, grape skins, seeds, and leaves are rich sources of bioactive polyphenolic compounds with significant therapeutic potential. Resveratrol (3,5,4′-trihydroxy-trans-stilbene) is a naturally occurring polyphenolic compound predominantly found in grape skins, seeds, and red wine. It has attracted considerable scientific interest due to its antioxidant, anti-inflammatory, cardioprotective, and anticancer properties. In oncology research, resveratrol has demonstrated the ability to interfere with multiple stages of carcinogenesis, including initiation, promotion, and progression.

Mechanistically, resveratrol modulates key regulators of the cell cycle, including cyclins and cyclin-dependent kinases, leading to cell cycle arrest commonly at the G1/S or G2/M checkpoints (Najafiyan *et al.,* 2024). It promotes apoptosis by activating intrinsic mitochondrial pathways, increasing Bax/Bcl-2 ratios, triggering cytochrome c release, and activating caspases. Additionally, resveratrol inhibits angiogenesis by suppressing vascular endothelial growth factor (VEGF) expression and reducing endothelial cell proliferation (Shukla & Singh, 2018). Recent investigations have further elucidated the molecular complexity of resveratrol's anticancer actions. In breast cancer models, resveratrol has been shown to downregulate Wnt/β-catenin and Hedgehog signaling pathways, both of which are critical drivers of tumor growth, stemness, and metastasis (Fang *et al.,* 2025). It also exerts epigenetic effects by modulating DNA methylation and histone acetylation, thereby silencing oncogenes and reactivating tumor suppressor genes (Kurzava Kendall *et al.,* 2024). These epigenetic modifications contribute to long-term regulation of gene expression involved in cancer progression. In colon cancer, resveratrol suppresses tumor cell proliferation and invasion by inhibiting matrix metalloproteinases (MMPs) and pro-inflammatory mediators (Wu *et al.,* 2023). Furthermore, it interferes with survival signaling pathways such as PI3K/Akt and STAT3, which are frequently overactivated in colorectal and breast cancers. It is through the inhibition of these pathways that resveratrol enhances apoptotic signaling and reduces cellular resistance to programmed cell death. Its anti-inflammatory properties, mediated through suppression of NF-κB activation, further contribute to its chemopreventive potential.

Bioactive constituent: Resveratrol

Cancer types: Breast and colon cancers

3.0 MECHANISMS OF ANTICANCER ACTION OF PLANTS

3.1 Induction of Apoptosis

Phytochemicals such as curcumin, epigallocatechin-3-gallate (EGCG), and thymoquinone can initiate apoptosis in cancer cells through multiple, complementary pathways, making them attractive candidates for anticancer therapy. These compounds activate both the intrinsic (mitochondrial) and extrinsic (death receptor) apoptotic cascades. Mechanistically, they modulate key regulatory proteins of the apoptotic machinery by increasing the Bax/Bcl-2 ratio, promoting mitochondrial outer membrane permeabilization, and facilitating cytochrome c release into the cytosol, which subsequently activates initiator caspase-9 and the executioner caspase-3 (Singh & Kaur, 2022). In addition, curcumin and EGCG have been shown to upregulate pro-apoptotic proteins including p53 and p21 while suppressing anti-apoptotic factors such as survivin and Bcl-xL, thereby lowering the threshold for programmed cell death in transformed cells (Liu *et al.,* 2023; Wang *et al.,* 2024).

Beyond the mitochondrial pathway, several phytochemicals influence death receptor–mediated apoptosis by enhancing expression of Fas (CD95), tumor necrosis factor–related apoptosis-inducing ligand (TRAIL) receptors, and associated adaptor proteins (Anderson Luiz-Ferreira *et al.,* 2023). This sensitization increases tumor cell susceptibility to extrinsic apoptotic triggers, resulting in activation of caspase-8 and downstream effectors (Gupta *et al.,* 2024). Thymoquinone, for example, has been reported to elevate Fas ligand expression and augment TRAIL-induced apoptosis in resistant cancer models, suggesting a dual-pathway engagement (Alam *et al.,* 2023). Importantly, many of these compounds also induce selective oxidative stress in cancer cells. Through generating reactive oxygen species (ROS) beyond the adaptive capacity of malignant cells, phytochemicals amplify apoptotic signaling while normal cells, which possess more robust antioxidant defenses, are comparatively spared (Prasad *et al.,*

2018). Elevated ROS can trigger JNK and p38 MAPK pathways, further promoting apoptosis and inhibiting survival pathways such as PI3K/Akt and NF-κB, which are frequently overactive in cancer (Hassan *et al.,* 2023).

3.2 Cell Cycle Arrest

Many phytochemicals exert potent anticancer effects by interrupting the tightly regulated process of cell cycle progression, effectively halting proliferation at critical checkpoints (Aljabali *et al.,* 2025). These compounds modulate key regulators of the cell cycle such as cyclins, cyclin-dependent kinases (CDKs), and CDK inhibitors to enforce arrest at the G1/S or G2/M phases, thereby preventing replication of damaged or transformed DNA (Singh *et al.,* 2020). For example, resveratrol has been shown to downregulate cyclin D1 and CDK4, leading to G1 phase arrest in breast and colon cancer cells, while EGCG suppresses cyclin B1 and CDK1 expression, resulting in G2/M blockade in multiple tumor models (Alam *et al.,* 2023). In addition to modulating core cyclins and cyclin-dependent kinases (CDKs), phytochemicals often upregulate CDK inhibitors, such as p21^Cip1 and p27^Kip1, thereby enhancing checkpoint control and preventing aberrant cell cycle progression. Upregulation of p21 and p27 has been observed in response to curcumin, EGCG, and resveratrol treatment, leading to sustained cell cycle arrest that limits proliferation and facilitates repair of oncogenic lesions (Gautam *et al.,* 2019). Increased expression of these inhibitors often occurs alongside activation of tumor suppressor pathways, including p53, further amplifying cell cycle blockade and tipping the balance toward growth arrest rather than unchecked progression.

Beyond effects on regulatory kinases, several phytochemicals interfere with structural and enzymatic components of mitosis and DNA repair (Huang *et al.,* 2022). For instance, certain stilbenes and flavonoids disrupt mitotic spindle assembly by altering microtubule dynamics, creating prolonged arrest at the M phase that can trigger apoptosis or senescence if unresolved. Others have been shown to impair homologous recombination and non-homologous end-joining DNA repair pathways, exacerbating replication stress and reinforcing checkpoint activation (Tanaka *et al.,* 2024).

3.3 Anti-Angiogenesis and Anti-Metastasis

Tumor progression, invasion, and metastasis are critically dependent on the formation of new blood vessels (angiogenesis) and the remodeling of the extracellular matrix (ECM) (Winkler e*t al.,* 2020). Without adequate vascular support, rapidly growing tumors cannot obtain sufficient oxygen and nutrients, and they are less likely to disseminate to distant sites. Phytochemicals such as curcumin and α-mangostin have demonstrated significant anti-angiogenic and anti-metastatic activity across multiple experimental cancer models by targeting key drivers of these processes (Obolskiy *et al.,* 2022). One primary mechanism by which phytochemicals inhibit angiogenesis is through downregulation of vascular endothelial growth factor (VEGF) and its receptors. VEGF is a central pro-angiogenic cytokine produced by hypoxic tumor cells to stimulate endothelial cell proliferation, migration, and new vessel formation. Curcumin has been reported to reduce VEGF expression and secretion in various cancer cell lines, including colorectal and breast cancer, thereby attenuating endothelial tube formation and vascular sprouting in vitro and in vivo (Torres *et al.,* 2024). Similarly, α-mangostin, a xanthone derived from Garcinia mangostana, suppresses VEGF production and impairs endothelial cell motility, resulting in reduced microvessel density in tumor xenograft models (Hernández *et al.,* 2022). Beyond VEGF, phytochemicals also modulate matrix metalloproteinases (MMPs), a family of zinc-dependent proteases that degrade ECM components and facilitate tumor invasion and metastasis. Curcumin, resveratrol, and other plant-derived agents have been shown to decrease expression and activity of MMP-2 and MMP-9, enzymes particularly implicated in basement membrane breakdown and metastatic dissemination (Singh *et al.,* 2021). By suppressing MMP activity, these compounds preserve ECM integrity and limit cancer cell migration and invasion.

3.4 Modulation of Signaling Pathways

Plant-derived compounds exert broad spectrum anticancer activity by modulating multiple dysregulated signaling pathways that drive cancer initiation, progression, and therapy resistance. Among the most frequently targeted cascades are PI3K/Akt, NF-κB, STAT3, and Wnt/β-catenin, all of which play critical roles in regulating cell proliferation, survival, inflammation, and tumor stemness (Zhang *et al.,* 2024). The PI3K/Akt pathway, for example, promotes cell survival and growth downstream of growth factor receptors; many phytochemicals such as EGCG and curcumin inhibit Akt phosphorylation, thereby decreasing downstream mTOR signaling and sensitizing tumor cells to apoptosis (Sharma *et al.,* 2024). Similarly, curcumin and resveratrol suppress NF-κB activation, reducing the expression of pro-inflammatory cytokines, anti-apoptotic proteins (e.g., Bcl-2, survivin), and metastasis-associated genes effects that collectively dampen the malignant phenotype (Hassan *et al.,* 2023).

Another pivotal target, STAT3, is constitutively activated in many cancers and drives transcription of genes involved in proliferation and immune evasion. Phytochemicals such as withaferin A and thymoquinone have been shown to inhibit STAT3 phosphorylation, decrease downstream target expression (e.g., cyclin D1, c-Myc), and impair tumor growth in preclinical models (Mir *et al.,* 2021). In the Wnt/β-catenin pathway central to stemness and self-renewal bioactives like resveratrol destabilize β-catenin in the nucleus, reducing expression of oncogenic transcriptional programs in colorectal and breast cancers (Kurzava Kendall *et al.,* 2024).

Importantly, many phytochemicals also activate tumor-suppressive pathways that restore cell cycle control and promote apoptosis. For instance, several polyphenols enhance p53 activity, leading to increased expression of CDK inhibitors (e.g., p21^Cip1) and

pro-apoptotic factors, thereby enforcing checkpoints and facilitating programmed cell death (Gupta *et al.,* 2024). Likewise, activation of AMP-activated protein kinase (AMPK) by compounds like resveratrol and metformin analogues reduces anabolic signaling, inhibits mTOR, and promotes autophagic cell death in stressed tumor cells.

Table 1: Summary of Some Medicinal Plants, Bioactive Compounds, Targeted Cancer Types, and Molecular Mechanisms of Anticancer Action

Plants (scientific name)	Bioactive compounds	Cancer type	Molecular Targets/Signaling pathways	Mechanism of Action
(1) *Allium sativum* (Garlic)	Allicin, Organosulfur compounds	Colorectal, Gastric, Prostate	ROS, Caspases, Bax/Bcl-2, Detoxification enzymes	Induces apoptosis, inhibits proliferation, modulates oxidative stress, enhances detoxification
(2) *Camellia sinensis* (Green Tea)	Epigallocatechin-3-gallate (EGCG)	Lung, Prostate, Leukemia	PI3K/Akt, MAPK, NF-κB, Cyclins/CDKs	Induces apoptosis, arrests cell cycle, inhibits angiogenesis, suppresses oncogenic signaling
(3) *Curcuma longa* (Turmeric)	Curcumin	Breast, Colorectal, Pancreatic, Bladder	NF-κB, STAT3, Wnt/β-catenin, VEGF, MMPs	Suppresses proliferation, induces apoptosis, inhibits invasion & angiogenesis, modulates multiple oncogenic pathways
(4) *Vitis vinifera* (Grape)	Resveratrol	Breast, Colon	PI3K/Akt, Wnt/Hedgehog, VEGF, MMPs, Epigenetic regulators	Promotes apoptosis, arrests cell cycle, inhibits angiogenesis, suppresses oncogenic signaling, epigenetic modulation
(5) Withania somnifera (Ashwagandha)	Withanolides (e.g., Withaferin A)	Prostate, Lung	NF-κB, STAT3, Akt/mTOR, Notch-1	Activates intrinsic apoptosis, suppresses proliferation, inhibits tumor growth, synergizes with chemotherapy
(6) Zingiber officinale (Ginger)	6-Gingerol, 6-Shogaol	Ovarian, Gastrointestinal	PI3K/Akt/mTOR, NF-κB, VEGF, IL-8	Induces apoptosis, inhibits proliferation & migration, anti-angiogenic, modulates oncogenic signaling
(7) Thymus vulgaris (Thyme)	Thymol, Carvacrol	Oral squamous cell carcinoma	MAPK/ERK, PI3K/Akt, Bax/Bcl-2, CDK4, MMPs	Antioxidant, induces apoptosis, inhibits proliferation & invasion, modulates cell cycle & signaling pathways

4.0 CONCLUSION

Many plants have anticancer potentials courtesy of the compounds they contain such as curcumin, epigallocatechin-3-gallate, resveratrol, thymoquinone, α-mangostin, withanolides, and taxanes. These compounds target multiple hallmarks of cancer, including uncontrolled cell proliferation, resistance to apoptosis, angiogenesis, metastasis, immune evasion, and dysregulated oncogenic signaling. Their ability to modulate several molecular pathways simultaneously allows them to overcome resistance mechanisms that often limit the effectiveness of conventional anticancer therapies.

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