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Phytomedicine Used in Cancer

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Abstract

Phytomedicine, which involves using medicinal plants and substances made from plants, has changed the way we treat cancer. It works in many ways, helps the body's natural defenses, and is safer than some other treatments. Recent studies show that these plant-based medicines can cause cancer cells to die, stop them from growing, prevent the formation of new blood vessels that feed tumors, and change the body's environment around the tumor. These treatments not only work well with standard cancer treatments like chemotherapy and radiation but also help deal with the side effects of those treatments and make them more effective. Many studies, including large population research and clinical trials, back up the use of phytomedicine as a helpful addition to cancer care. When combined with modern technology and tailored treatment plans, phytomedicine plays a key role in improving cancer treatment today.

Keywords: Phytomedicine, Phytochemicals, Cancer, Tumour Microenvironment, Oxidative Stress, Cancer Stem Cells, Adjuvant Therapy, Nanodelivery.

1) Introduction

Cancer is one of the main reasons people die worldwide. While traditional treatments have helped improve survival rates, they still have problems like side effects, drug resistance, cancer coming back, spreading to other parts of the body, and high costs. At the same time, medicinal plants and their active parts—often called phytochemicals—are used for many diseases, including cancer. In recent years, these plant-based treatments have become more popular in cancer research. Phytomedicine is the use of plant extracts or isolated compounds to treat diseases.

But there are still many challenges. It's hard to standardize these plant extracts, clinical trials can be inconsistent, they don't always work well in the body, there aren't enough large, controlled studies, and there are legal and regulatory issues. For example, a study on prostate cancer found that even though some plant compounds, like genistein, lycopene, and curcumin, show promise in lab and animal tests, there's not enough proof they work well in humans.

Phytomedicine comes from the Greek word "phyton," meaning plant. It refers to using medicinal plants and their active compounds to prevent and treat diseases. Plants have been a big part of medicine for a long time. For example, the Pacific yew tree gives paclitaxel, which is used in the drug Taxol®. The rosy periwinkle plant gives vincristine and vinblastine, which are important in chemotherapy. Recent studies, both in the lab, in animals, and in people, show that many phytochemicals could help Tableht cancer. These compounds often work by changing how cells signal, reducing harmful substances in the body, and helping the immune system.

Phytochemicals are natural substances made by plants, like alkaloids, flavonoids, terpenoids, polyphenols, and saponins.

They can affect molecules involved in cancer development. These compounds often work on multiple targets, which makes it harder for cancer to become resistant and can make them work better with standard treatments.

2) Phytomedicine in Oncology

Phytomedicine is significant in the field of complementary and integrative oncology. This interest is driven by its diverse bioactive properties, minimal toxicity, and capacity to influence several key characteristics of cancer. Research indicates that dietary phytochemicals can function at different phases of cancer development — including initiation, promotion, and progression — making them effective as both preventive and treatment options.

Several phytochemicals exert anticancer activity through mechanisms such as:

- Induction of apoptosis (programmed cell death)
- Cell cycle arrest
- Inhibition of angiogenesis and metastasi

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- Suppression of inflammation
- Modulation of oxidative stress
- Epigenetic regulation (DNA methylation, histone acetylation)
- Sensitization to chemotherapy or radiotherapy.

3) Mechanisms of Action of Anticancer Phytochemicals

TUGAS TUTOR focuses on targeting various signaling pathways that are involved in the survival and growth of cancer cells:

1. Induction of Apoptosis

One essential method for getting rid of aberrant cells is apoptosis. Numerous phytochemicals cause apoptosis by extrinsic (death receptor) or intrinsic (mitochondrial) mechanisms. For example:

- **Curcumin** upregulates *Bax* and *p53* while downregulating *Bcl-2*, promoting cytochrome c release and caspase activation.
- **Resveratrol** activates *caspase-3* and *PARP* cleavage in breast and colon cancer cells.
- **Berberine** enhances *Fas/FasL*-mediated apoptosis in lung cancer cells.

2. Cell Cycle Arrest

By modifying cyclins and cyclin-dependent kinases (CDKs), phytochemicals stop unchecked growth. For instance:

- **Epigallocatechin gallate (EGCG)** induces G1 phase arrest in prostate and breast cancer cells.
- **Genistein** inhibits CDK1 and cyclin B, blocking G2/M transition.

3. Inhibition of Angiogenesis

Tumor angiogenesis is necessary for metastasis. Curcumin, quercetin, and resveratrol inhibit VEGF, MMP-9, and HIF-1α, reducing new blood vessel creation.

4. Anti-Inflammatory Effects

Chronic inflammation plays a role in the advancement of tumors. Phytochemicals help reduce the activity of inflammatory molecules such as COX-2, iNOS, NF-κB, and TNF-α. Curcumin is particularly effective in inhibiting NF-κB, which in turn helps lower the production of inflammatory cytokines..

5. Epigenetic Modulation

Some substances found in plants can change how DNA is modified and affect histone proteins, which helps reverse incorrect gene silencing. For example, sulforaphane, which comes from broccoli, stops histone deacetylases (HDACs) from working, which helps turn on genes that normally prevent cancer.

4) Major Phytochemicals and Their Anticancer Effects

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Curcumin (from Curcuma longa)

Chemical class: polyphenol (diferuloylmethane)

The following cancer types were studied: Breast, colon, prostate, pancreas, liver, and lung

Mechanisms of action:

- Activates caspases, modulates Bax/Bcl-2, and depolarizes mitochondria, leading to cell death.λ
- Stops cell cycle at G1/S or G2/M by inhibiting cyclins and CDKs.λ
- Inhibits angiogenesis by targeting VEGF, MMPs, and HIF-1α.
- Reduces inflammation by suppressing NF-κB, COX-2, and pro-inflammatory cytokines.
- Epigenetic regulation: inhibits histone acetyltransferase (HAT) and modifies microRNAs.λ.

Clinical status: Multiple Phase I/II trials demonstrate safety and tolerability; bioavailability remains a difficulty, which is addressed via nanoformulations..

Resveratrol (from grapes, peanuts, berries)

Chemical class: Stilbene polyphenol

Cancer types studied: Colon, breast, prostate, lung, leukemia

Mechanisms of action:

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- Activates p53, mitochondrial pathways, and caspase cascades, resulting in cell death.λ
- Cell cycle arrest in S phase due to cyclin/CDK regulation.λ
- Promotes anti-inflammatory and antioxidant action via inhibiting NF-κB and regulating ROS levels.λ
- Suppresses MMPs and EMT-related genes to prevent metastasis.
- Modifies histone acetylation and regulates miRNAs that contribute to cancer growth.

Clinical status: Oral supplementation shows low toxicity; combination with chemotherapy enhances efficacy.

Epigallocatechin Gallate (EGCG, from green tea)
Chemical class: Catechin polyphenol
Cancer types studied: Breast, prostate, lung, colorectal
Mechanisms of action:

- Stops the growth of cells by stopping them from moving past the G1 phase of their cell cycle.
- Causes cell death by activating caspase-3 and caspase-8.
- Prevents the formation of new blood vessels by reducing VEGF and MMPs.
- Acts as an antioxidant by removing harmful free radicals and controlling signals related to reactive oxygen species.
- Changes how genes are expressed by blocking DNMTs and HDACs, which helps bring back the activity of tumor suppressor genes..

Clinical status: Oral consumption has been linked to a reduced risk of cancer in epidemiological studies, and the use of nanoformulations can improve the bioavailability of these treatments.

Quercetin (found in onions, apples, berries)
Chemical class: Flavonoid
Cancer types studied: Colon, breast, lung, liver
Mechanisms of action:

- Causes cells to die by turning on caspases and turning off Bcl-2.
- Stops the cell cycle at the G2/M stage.
- Prevents cancer spread by blocking MMP-2, MMP-9, and EMT markers.
- Reduces inflammation by lowering NF-κB activity.
- Tablehts oxidative stress by removing ROS and boosting antioxidant enzymes..

Clinical status: Preclinical studies show promise; clinical trials are currently taking place. Nanocarriers help improve solubility and target specific tissues better.

Genistein (from soy products)
Chemical class: Isoflavone
Cancer types studied: Prostate, breast, colon, lung

Mechanisms of action:

- Blocks tyrosine kinase signaling pathways like PI3K/Akt and MAPK.
- Causes cell death and stops new blood vessel growth by lowering VEGF levels.
- Stops the cell cycle at the G2/M stage.
- Changes how genes are expressed through epigenetic effects, affecting DNA methylation and microRNA levels..

Clinical status: Widely used in Asia; Phase II trials suggest synergistic effects with chemotherapy.

Berberine (from Berberis species)
Chemical class: Isoquinoline alkaloid
Cancer types studied: Liver, colon, breast, lung
Mechanisms of action:

- Causes cells to die by affecting the mitochondria.
- Stops the cell cycle at the G1 stage.
- Prevents cancer cells from spreading by reducing MMP-2 and MMP-9.
- Reduces inflammation by blocking NF-κB and the production of certain proteins.
- Changes how cancer cells use sugar and fat, which slows their growth..

Clinical status: Preclinical studies promising; human trials limited.

Sulforaphane (from broccoli, cruciferous vegetables)
Chemical class: Isothiocyanate
Cancer types studied: Prostate, breast, lung, colon

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Mechanisms of action:

- It causes cells to die and stops them from dividing during the G2/M phase of the cell cycle.
- It stops the formation of new blood vessels by reducing VEGF and HIF-1α.
- It changes how genes are expressed by inhibiting HDAC and altering DNA methylation.
- It also helps reduce oxidative stress and inflammation..

Clinical status: Phase I studies demonstrate tolerability and biomarker modulation.

Thymoquinone (from Nigella sativa)

Chemical class: Monoterpene

Cancer types studied: Breast, colon, liver, leukemia

Mechanisms of action:

- Triggers cell death through the mitochondrial pathway.
- Reduces inflammation by blocking the NF-κB pathway.
- Slows cell growth by stopping the cell cycle in the G1 stage.
- Prevents cancer spread by reducing EMT and MMP activity.

Clinical status: Limited human studies; preclinical results promising.

Compound/Herb	***Cancer Target***	***Clinical/Preclinical Evidence***	***Mechanism***
Curcumin (Curcuma longa)	Breast, Colon, Prostate	Combination therapy, reduced toxicity	Anti-inflammatory, apoptosis
Withaferin A (Withania somnifera)	Breast, Lung, Colon	Sensitizes to chemotherapy	Apoptosis enhancement
EGCG (Green Tea Polyphenol)	Breast, Multiple	Synergistic with curcumin, suppresses VEGF	Antioxidant, angiogenesis inhibition
Thymoquinone (Nigella sativa)	Breast, Multiple	Modulates p53, NF-κB, ERK1/2	Apoptosis, metastasis inhibition
Paclitaxel (Pacific Yew)	Ovarian, Breast, Lung	FDA-approved chemotherapeutic	Microtubule stabilization
Camptothecin (Camptotheca acuminata)	Multiple	Drug derivatives in clinical use	Topoisomerase I inhibition

Table no 1

5) Medicinal Plants with Demonstrated Anticancer Properties

Breast Cancer

Breast cancer is the most common cancer among women globally. Several phytochemicals and plant extracts have shown promising anticancer activity in vitro, in vivo, and some clinical studies.

Plant/Extract	Active Compounds	Mechanisms	Evidence
Curcuma longa (Turmeric)	Curcumin	Apoptosis induction, cell cycle arrest, NF-κB inhibition	Preclinical investigations; Phase I/II clinical trials show safety and efficacy in combination with chemotherapy.
Camellia sinensis (Green Tea)	EGCG	Anti-proliferative, anti-angiogenic, epigenetic modulation	Epidemiological and interventional studies suggest lower breast cancer risk.

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Glycyrrhiza glabra (Licorice)	Glabridin, Isoliquiritigenin	Anti-estrogenic, apoptosis induction	Preclinical studies suggest inhibition of ER-positive breast cancer cell proliferation.
Nigella sativa	Thymoquinone	Apoptosis, EMT suppression, anti-metastatic	Preclinical mouse models showed tumor volume decrease.
Vitis vinifera (Grape Seed Extract)	Resveratrol, Procyanidins	Anti-inflammatory, apoptosis induction, angiogenesis inhibition	Preclinical studies suggest the usage as adjuvant therapy.
Camellia japonica	Quercetin	ROS modulation, apoptosis	In vitro research, possibility for adjuvant therapy.

Table no 2

Colorectal Cancer (CRC)

CRC is strongly influenced by diet and gut microbiota. Phytochemicals exert chemopreventive and therapeutic effects through modulation of oxidative stress, inflammation, and apoptosis.

Plant/Extract	Active Compounds	Mechanisms	Evidence
Broccoli (Brassica oleracea)	Sulforaphane	HDAC inhibition, apoptosis, cell cycle arrest	Preclinical and small clinical trials; chemopreventive potential
Camellia sinensis	EGCG	Anti-proliferative, apoptosis, anti-angiogenic	Epidemiological studies reveal a lower CRC risk.
Curcuma longa	Curcumin	Anti-inflammatory, apoptosis, cell cycle arrest	Phase II trials indicate potential synergy with FOLFOX chemotherapy.
Allium sativum (Garlic)	Allicin	Anti-proliferative, antioxidant	Preclinical research and epidemiological evidence for reduced CRC incidence.
Punica granatum (Pomegranate)	Ellagic acid, Polyphenols	Apoptosis, NF-κB inhibition	Preclinical CRC models are synergistic with chemotherapy.

Table no 3

Prostate Cancer

Prostate cancer is influenced by hormonal regulation and chronic inflammation. Several phytochemicals modulate androgen signaling and tumor progression.

Plant/Extract	Active Compounds	Mechanisms	Evidence
Soy (*Glycine max*)	Genistein	Tyrosine kinase inhibition, apoptosis, cell cycle arrest	Clinical studies suggest reduced PSA levels in early-stage prostate cancer

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Broccoli (*Brassica oleracea*)	Sulforaphane	HDAC inhibition, apoptosis	Phase I/II trials show epigenetic biomarker modulation.
Green Tea (*Camellia sinensis*)	EGCG	Anti-proliferative, anti-angiogenic	Epidemiological studies and small trials show PSA reduction.
Turmeric (*Curcuma longa*)	Curcumin	Apoptosis, anti-inflammatory	Preclinical studies and pilot clinical trials.

Table no 4

Ovarian Cancer

Ovarian cancer is often diagnosed late; phytochemicals may help enhance apoptosis and overcome chemoresistance.

Plant/Extract	Active Compounds	Mechanisms	Evidence
Turmeric (Curcuma longa)	Curcumin	Apoptosis induction, chemosensitization	Preclinical and pilot clinical studies
Green Tea (Camellia sinensis)	EGCG	Anti-proliferative, anti-angiogenic	In vitro studies show reduced proliferation
Nigella sativa	Thymoquinone	Anti-metastatic, apoptosis	Preclinical studies
Punica granatum	Ellagic acid	Anti-proliferative, antioxidant	Preclinical ovarian cancer models

Table no 5

Leukemia

Phytochemicals can induce apoptosis and modulate signaling in hematologic malignancies.

Plant/Extract	Active Compounds	Mechanisms	Evidence
Berberis vulgaris	Berberine	ROS-mediated apoptosis, cell cycle arrest	In vitro and in vivo leukemia models
Thymoquinone	Nigella sativa	Apoptosis, anti-proliferative	Preclinical leukemia studies
Resveratrol	Grapes	Caspase activation, apoptosis	In vitro leukemia studies
Curcumin	Turmeric	Anti-proliferative, NF-κB inhibition	Preclinical leukemia models

Table no 6

6) Key Takeaways from Plant-Based Anticancer Studies

- Multi-targeted approach: Most plant-based chemicals work on multiple cancer-related features at the same time.
- Dietary relevance: Many of these active chemicals come from common foods like turmeric, broccoli, green tea, and soy, which shows their potential for cancer prevention.
- Synergy with conventional therapy: These plant chemicals can help make chemotherapy and radiation more effective and also help reduce their side effects.
- Limitations: They are not very easily absorbed by the body, there are differences in their levels based on the plant source, and there is not enough large-scale research to support their use.

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- Future potential: Using nano-formulations, combining them with other treatments, and integrating them into standard cancer care could improve their use in real-world medical settings..

7) Nanotechnology and Delivery of Phytochemicals

One big problem with phytochemicals is that they don't dissolve well, aren't easily absorbed by the body, and get broken down quickly, which makes them less effective in real-world situations. Nanotechnology helps by allowing these chemicals to be delivered more precisely, released over time, and taken up better by cells.

6.1 Types of Nanocarriers

Liposomes:

- Spherical vesicles encapsulating hydrophilic or hydrophobic compounds.
- Example: Curcumin-loaded liposomes increase bioavailability and tumor accumulation in breast and colon cancer models.

Polymeric nanoparticles:

- Biodegradable polymers like PLGA (poly(lactic-co-glycolic acid)) allow controlled release.
- EGCG and resveratrol nanoparticles show enhanced apoptosis and reduced tumor growth.

Solid lipid nanoparticles (SLNs):

- Lipid-based carriers improving solubility of poorly water-soluble compounds.
- Curcumin SLNs show superior uptake in liver and lung cancer cells.

Nanomicelles:

- Amphiphilic molecules form micelles encapsulating hydrophobic phytochemicals.
- Example: Genistein micelles improve oral absorption and antitumor efficacy.

Gold and metal nanoparticles:

- Enable targeted photothermal therapy combined with phytochemicals.
- EGCG-coated gold nanoparticles induce selective apoptosis in melanoma cells.

8) Advantages of Nanotechnology in Phytomedicine

- Better ability to dissolve and stay stable
- Delivered directly to cancer cells, lowering harmful effects on the whole body
- Drug is released slowly and steadily over time
- Works better when used with chemotherapy and radiation treatment
- May help Tableht against resistance to multiple drugs

9) Challenges and Future Prospects

❖ Current Challenges

- Low bioavailability and solubility – makes it hard to reach effective levels in the body.
- Differences in plant sources – leads to varied amounts of active plant chemicals.
- Not enough big studies – most information comes from lab research, not real-world use.
- Possible interactions with other medicines – could affect chemotherapy or targeted treatments.
- Complex ways it works – affects multiple targets, making it hard to set standard doses and measure results..

❖ Future Directions

- Using nanotechnology and special delivery methods to make treatments work better.
- Combining plant-based medicines with traditional cancer drugs to make treatment more effective and less harmful.
- Creating customized herbal medicines based on the genetic makeup of tumors and how a person's body processes drugs.
- Studying how plant chemicals can change gene activity and help the immune system, which is a new and growing area of research.
- Working together around the world to set clear guidelines, run good quality studies, and create rules for getting these treatments approved.

10) Conclusion

Phytomedicine provides a hopeful extra or different option for preventing and treating cancer. Certain active ingredients from plants, such as curcumin, resveratrol, EGCG, quercetin, genistein, berberine, sulforaphane, and thymoquinone, work in many ways to Tableht cancer. These ways include:

1. Making cancer cells die off naturally
2. Stopping cells from dividing and growing out of control
3. Preventing the formation of new blood vessels that feed tumors

4. Blocking the spread of cancer cells to other parts of the body
5. Reducing inflammation and protecting cells from damage caused by harmful substances
6. Changing how genes are expressed, which can affect cancer growth

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