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Review Article

Anticancer Potential of Plant-Derived Compounds: A Comprehensive Review

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ABSTRACT

Cancer continues to pose a major global health challenge, contributing significantly to illness and death across populations. While conventional therapies have led to important advancements, limited efficacy, adverse side effects, and high treatment costs remain substantial barriers. In recent years, natural compounds—particularly those derived from plants—have attracted growing interest for their potential therapeutic value. Known for their chemical diversity and wide-ranging biological activities, these compounds are being increasingly explored for their role in cancer prevention and treatment. This review broadly examines the anticancer potential of phytochemicals, outlines their underlying biological mechanisms, and discusses recent developments in their application within modern medicine.

INTRODUCTION

Cancer is a complex, multifactorial disease marked by uncontrolled cell proliferation, resistance to apoptosis, sustained angiogenesis, and the ability to metastasize. It remains a major global health concern, affecting millions of individuals worldwide. Although conventional treatment modalities such as surgery, chemotherapy, and radiation therapy—have improved patient outcomes, they are often associated with significant side effects and the emergence of resistance. Consequently, there is an urgent need for novel therapeutic agents that are both effective and less toxic. Historically, plants have served as a rich reservoir of bioactive compounds, and more than 60% of currently approved anticancer drugs are derived from natural sources. Notable examples include paclitaxel, vincristine, and camptothecin, which have become integral components of cancer therapy. These phytochemicals exert their anticancer effects by modulating key cellular pathways suppressing proliferation, inducing programmed cell death, and inhibiting angiogenesis and metastasis.

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2. Classification of Plant-Derived Anticancer Compounds

Plant-derived anticancer compounds can be broadly categorized based on their chemical structures and mechanisms of action:

1. Alkaloids

Structure: Contain nitrogen atoms, typically in a heterocyclic ring.

Mechanism of Action: Disrupt microtubule dynamics, inhibit DNA topoisomerases, and interfere with DNA/RNA synthesis**.**

Key Compounds:

Vincristine & Vinblastine (*Catharanthus roseus*)

- **Mechanism:** Bind to tubulin, inhibit microtubule polymerization → arrest in metaphase.
- **Use:** Leukemias, lymphomas, breast cancer.

Camptothecin (*Camptotheca acuminata*)

- **Mechanism:** Inhibits topoisomerase I, causing DNA strand breaks during replication.
- **Derivatives:** Irinotecan, topotecan (clinically approved).

Berberine (*Berberis* species)

- **Mechanism:** Induces apoptosis via mitochondrial pathways; modulates p53 and caspases.
- **Use:** Preclinical studies in colon, lung, and breast cancer.

2. Terpenoids (Isoprenoids)

Structure: Derived from five-carbon isoprene units; classified into mono-, sesqui, di-, tri, and tetraterpenoids.

Mechanism of Action: Modulates signal transduction, inhibits angiogenesis, and induces apoptosis.

Key Compounds:

- **Paclitaxel (Taxol) (*Taxus brevifolia*)**
  - **Mechanism:** Stabilizes microtubules, preventing depolymerization → mitotic arrest.
  - **Use:** Ovarian, breast, lung, and prostate cancers.
- **Artemisinin (*Artemisia annua*)**
  - **Mechanism:** Generates reactive oxygen species (ROS); affects cell cycle progression.
  - **Research:** Experimental use in breast, leukemia, and colon cancers.
- **Thymoquinone (*Nigella sativa*)**
  - **Mechanism:** Suppresses NF-κB, STAT3, and angiogenesis; induces apoptosis.
  - **Use:** In vitro efficacy across multiple cancers.

3. Phenolics and Polyphenols

Structure: Aromatic rings with hydroxyl groups; includes flavonoids, stilbenes, tannins.

Mechanism of Action: Antioxidant activity, inhibition of cell proliferation, promotion of apoptosis, and modulation of key signaling pathways (PI3K/Akt, MAPK, NF-κB).

Key Compounds:

- **Curcumin (*Curcuma longa*)**

- **Mechanism:** Downregulates NF-κB, COX-2, and Bcl-2; induces caspase-3 activation.
- **Use:** Promising in colorectal, pancreatic, and breast cancers.

- **Resveratrol (*Vitis vinifera*)**
- **Mechanism:** Inhibits angiogenesis, induces cell cycle arrest (G1/S phase), suppresses metastasis.
- **Use:** Colon, prostate, breast, and liver cancers.

4. Flavonoids (Subclass of polyphenols)

Structure: C6-C3-C6 backbone with hydroxylation and methoxylation patterns.

Mechanism of Action: ROS modulation, inhibition of tyrosine kinases, anti-estrogenic effects.

Key Compounds:

- **Quercetin (Onions, apples, tea)**
- **Mechanism:** Induces apoptosis via p53 activation and mitochondrial pathways.
- **Use:** Effective in breast, prostate, and colon cancers.

- **Genistein (Soy products)**
- **Mechanism:** Inhibits tyrosine kinases, blocks angiogenesis, and modulates estrogen receptors.
- **Use:** Hormone-sensitive cancers like breast and prostate.

5. Lignans

Structure: Phenylpropanoid dimers with a dibenzylbutane skeleton.

Mechanism of Action: Topoisomerase II inhibition, induction of apoptosis.

Key Compounds:

- **Podophyllotoxin (*Podophyllum peltatum*)**
- **Mechanism:** Precursor of etoposide and teniposide, which inhibit topoisomerase II → DNA damage.
- **Use:** Testicular cancer, small-cell lung cancer.

6. Saponins

Structure: Glycosides with steroid or triterpenoid aglycones.

Mechanism of Action: Disrupt cell membranes, enhance immune response, induce apoptosis.

Key Compounds:

- **Diosgenin (*Dioscorea* species)**
- **Mechanism:** Inhibits proliferation, modulates NF-κB and Akt pathways.
- **Use:** Experimental; shows potential in leukemia and colon cancer.

7. Quinones

Structure: Aromatic ketones; redox-active molecules that generate ROS.

Mechanism of Action: Induces oxidative stress, damages mitochondrial membrane potential.

Key Compounds:

- **Lapachol (*Tabebuia avellanedae*)**
- **Mechanism:** ROS generation → mitochondrial dysfunction and apoptosis.

- **Emodin (*Rheum palmatum, Polygonum cuspidatum*)**

  - **Mechanism:** Targets PI3K/Akt and ERK pathways; enhances chemosensitivity.

8. Coumarins

Structure: Benzopyrone derivatives with aromatic lactone structure.

Mechanism of Action: Antioxidant effects, cell cycle arrest, anti-angiogenic activity.

Key Compounds:

- **Esculetin**

  - **Mechanism:** Induces apoptosis, inhibits STAT3 and β-catenin pathways.

- **Psoralen (*Psoralea corylifolia*)**

  - **Mechanism:** Used in PUVA therapy (photochemotherapy); forms DNA adducts under UV exposure.

9. Other Notable Classes

- **Sulfur-containing compounds:** e.g., Allicin from garlic – induces apoptosis, inhibits angiogenesis.

- **Tannins:** e.g., Ellagic acid from pomegranate – antioxidant, anti-proliferative.

3. Mechanisms of Anticancer Activity

3.1 Induction of Apoptosis

Many phytochemicals activate intrinsic (mitochondrial) or extrinsic (death receptor) apoptotic pathways, leading to cancer cell death.

3.2 Inhibition of Cell Proliferation

Compounds like paclitaxel disrupt the cell cycle, especially at the G2/M phase, halting mitosis.

3.3 Anti-Angiogenesis

Inhibition of vascular endothelial growth factor (VEGF) expression by compounds like curcumin prevents the formation of new blood vessels essential for tumor growth.

3.4 Suppression of Metastasis

Plant compounds inhibit enzymes like matrix metalloproteinases (MMPs), involved in the degradation of extracellular matrix, thereby reducing invasion and metastasis.

3.5 Modulation of Signaling Pathways

Phytochemicals modulate multiple signaling cascades, including:

PI3K/Akt/mTOR

Wnt/β-catenin

MAPK/ERK

NF-κB

p53

4. Synergistic Effects and Combination Therapies

Combining plant-derived compounds with conventional chemotherapeutics has shown promise in enhancing efficacy and reducing toxicity. For example:

Curcumin + Doxorubicin: Enhances apoptosis and reduces cardiotoxicity.

Resveratrol + Cisplatin: Improves chemosensitivity and reduces nephrotoxicity.

5. Challenges and Future Directions

5.1 Bioavailability and Pharmacokinetics

Many plant compounds suffer from poor solubility, low absorption, and rapid metabolism. Nanotechnology-based delivery systems (e.g., liposomes, nanoparticles) are being explored to overcome these barriers.

5.2 Standardization and Quality Control

Variability in plant sources and extraction methods poses challenges in consistent efficacy and safety.

5.3 Clinical Validation

While many compounds show promise in preclinical studies, large-scale clinical trials are necessary to validate their therapeutic potential.

6. CONCLUSION

Plant-derived compounds represent a promising avenue for the development of novel anticancer agents. Their ability to target multiple pathways involved in cancer progression with minimal toxicity to normal cells makes them ideal candidates for adjunctive or alternative therapies. Future research focusing on formulation improvements, mechanistic insights, and clinical validations will be crucial in harnessing their full therapeutic potential.

REFERENCES

1. Liu RH. Health-promoting components of fruits and vegetables in the diet. Advances in nutrition. 2013 May 1;4(3):384S-92S.
2. Willett WC. Balancing lifestyle and genomics research for disease prevention. Science. 2002;296:695–8.
3. Liu RH. Potential synergy of phytochemicals in cancer prevention: mechanism of action. J Nutr. 2004; 134:3479S–85S.
4. Doll R, Peto R. Avoidable risks of cancer in the United States. J Natl Cancer Inst. 1981;66:1191–308. 4. Willett WC. Diet, nutrition, and avoidable cancer. Environ Health Perspect. 1995;103:165 70.
5. USDA. Dietary Guidelines for Americans 2010. USDA Human Nutrition Information Service, Hyattsville, MD. 2010.
6. PBH, Produce for Better Health Foundation. State of the plate: 2010 Study on America's consumption of fruits and vegetables. 2010.
7. Liu RH. Health benefits of fruits and vegetables are from additive and synergistic combination of phytochemicals. Am J Clin Nutr. 2003;78: 517S–20S.
8. Liu RH, Finley J. Potential cell culture models for antioxidant research. J Agric Food Chem. 2005;53:4311–4.
9. Wolfe KL, Kang XM, He XJ, Dong M, Zhang QY, Liu RH. Cellular antioxidant activity of common fruits. J Agric Food Chem. 2008;56:8418–26.
10. Song W, Derito CM, Liu KM, Liu H X, Dong M, Liu RH. Cellular antioxidant activity of common vegetables. J Agric Food Chem. 2010;58: 6621–9.
11. Chu Y-F, Sun J, Wu X, Liu RH. Antioxidant and antiproliferative activities of vegetables. J Agric Food Chem. 2002;50:6910–6.
12. Yang, L., et al. (2012). The anticancer potential of tea catechins. Pharmacology & Therapeutics, 133(1), 33-46.
13. Yang, C.S.; Wang, H. Mechanistic issues concerning cancer prevention by tea catechins. Mol. Nutr. Food Res. 2011, 55, 819–831.
14. Crespy, V.; Williamson, G. A review of the health effects of green tea catechins in in vivo animal models. J. Nutr. 2004, 134, 3431S–3440S.
15. Sang, S.; Lambert, J.D.; Ho, C.T.; Yang, C.S. The chemistry and biotransformation of tea constituents. Pharmacol. Res. 2011, 64, 87–99.

16. Hong, J.; Lu, H.; Meng, X.; Ryu, J.H.; Hara, Y.; Yang, C.S. Stability, cellular uptake, biotransformation, and efflux of tea polyphenol (−)-epigallocatechin-3-gallate in HT-29 human colon adenocarcinoma cells. Cancer Res. 2002, 62, 7241–7246.
17. Hou, Z.; Sang, S.; You, H.; Lee, M.J.; Hong, J.; Chin, K.V.; Yang, C.S. Mechanism of action of (−)-epigallocatechin-3-gallate: Auto-oxidation-dependent inactivation of epidermal growth factor receptor and direct effects on growth inhibition in human esophageal cancer KYSE 150 cells. Cancer Res. 2005, 65, 8049–8056.
18. Yang, C.S.; Sang, S.; Lambert, J.D.; Lee, M.J. Bioavailability issues in studying the health effects of plant polyphenolic compounds. Mol. Nutr. Food Res. 2008, 52, S139–S151.
19. Li, C.; Lee, M.J.; Sheng, S.; Meng, X.; Prabhu, S.; Winnik, B.; Huang, B.; Chung, J.Y.; Yan, S.; Ho, C.T.; et al. Structural identification of two metabolites of catechins and their kinetics in human urine and blood after tea ingestion. Chem. Res. Toxicol. 2000, 13, 177–184.
20. Clifford, M.N.; van der Hooft, J.J.; Crozier, A. Human studies on the absorption, distribution, metabolism, and excretion of tea polyphenols. Am. J. Clin. Nutr. 2013, 98, 1619S–1630S.
21. Roth, M.; Timmermann, B.N.; Hagenbuch, B. Interactions of green tea catechins with organic anion-transporting polypeptides. Drug Metab. Dispos. 2011, 39, 920–926.
22. Zhang, Y.; Hays, A.; Noblett, A.; Thapa, M.; Hua, D.H.; Hagenbuch, B. Transport by OATP1B1 and OATP1B3 enhances the cytotoxicity of epigallocatechin 3-O-gallate and several quercetin derivatives. J. Nat. Prod. 2013, 76, 368–373.
23. Jemnitz, K.; Heredi-Szabo, K.; Janossy, J.; Ioja, E.; Vereczkey, L.; Krajcsi, P. ABCC2/Abcc2: A multispecific transporter with dominant excretory functions. Drug Metab. Rev. 2010, 42, 402–436.
24. Hong, J.; Lambert, J.D.; Lee, S.H.; Sinko, P.J.; Yang, C.S. Involvement of multidrug resistance-associated proteins in regulating cellular levels of (−)-epigallocatechin-3-gallate and its methyl metabolites. Biochem. Biophys. Res. Commun. 2003, 310, 222–227.
25. Lipinski, C.A.; Lombardo, F.; Dominy, B.W.; Feeney, P.J. Experimental and computational approaches to estimate solubility and permeability in drug discovery and development settings. Adv. Drug Deliv. Rev. 2001, 46, 3–26.
26. Lambert, J.D.; Lee, M.J.; Lu, H.; Meng, X.; Hong, J.J.; Seril, D.N.; Sturgill, M.G.; Yang, C.S. Epigallocatechin-3-gallate is absorbed but extensively glucuronidated following oral administration to mice. J. Nutr. 2003, 133, 4172–4177.
27. Chow, H.H.; Hakim, I.A.; Vining, D.R.; Crowell, J.A.; Ranger-Moore, J.; Chew, W.M.; Celaya, C.A.; Rodney, S.R.; Hara, Y.; Alberts, D.S. Effects of dosing condition on the oral bioavailability of green tea catechins after single-dose administration of Polyphenon E in healthy individuals. Clin. Cancer Res. 2005, 11, 4627–4633.
28. Ju, J.; Hong, J.; Zhou, J.N.; Pan, Z.; Bose, M.; Liao, J.; Yang, G.Y.; Liu, Y.Y.; Hou, Z.; Lin, Y.; et al. Inhibition of intestinal tumorigenesis in Apcmin/+ mice by (−)-epigallocatechin-3-gallate, the major catechin in green tea. Cancer Res. 2005, 65, 10623–10631.
29. Ju, J.; Liu, Y.; Hong, J.; Huang, M.T.; Conney, A.H.; Yang, C.S. Effects of green tea and high-fat diet on arachidonic acid metabolism and aberrant crypt foci formation in an azoxymethane-induced colon carcinogenesis mouse model. Nutr. Cancer 2003, 46, 172–178.

30. Xiao, H.; Hao, X.; Simi, B.; Ju, J.; Jiang, H.; Reddy, B.S.; Yang, C.S. Green tea polyphenols inhibit colorectal aberrant crypt foci (ACF) formation and prevent oncogenic changes in dysplastic ACF in azoxymethane-treated F344 rats. Carcinogenesis 2008, 29, 113–119.
31. Shimizu, M.; Shirakami, Y.; Sakai, H.; Adachi, S.; Hata, K.; Hirose, Y.; Tsurumi, H.; Tanaka, T.; Moriwaki, H. (−)-Epigallocatechin gallate suppresses azoxymethane-induced colonic premalignant lesions in male C57BL/KsJ-db/db mice. Cancer Prev. Res. 2008, 1, 298–304.
32. Hao, X.; Sun, Y.; Yang, C.S.; Bose, M.; Lambert, J.D.; Ju, J.; Lu, G.; Lee, M.J.; Park, S.; Husain, A.; et al. Inhibition of intestinal tumorigenesis in Apc(min/+) mice by green tea polyphenols (polyphenon E) and individual catechins. Nutr. Cancer 2007, 59, 62–69.
33. Lu, G.; Liao, J.; Yang, G.; Reuhl, K.R.; Hao, X.; Yang, C.S. Inhibition of adenoma progression to adenocarcinoma in a 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced lung tumorigenesis model in A/J mice by tea polyphenols and caffeine. Cancer Res. 2006, 66, 11494–11501.
34. Xu, Y.; Ho, C.T.; Amin, S.G.; Han, C.; Chung, F.L. Inhibition of tobacco-specific nitrosamine-induced lung tumorigenesis in A/J mice by green tea and its major polyphenol as antioxidants. Cancer Res. 1992, 52, 3875–3879.
35. Chung, F.L.; Wang, M.; Rivenson, A.; Iatropoulos, M.J.; Reinhardt, J.C.; Pittman, B.; Ho, C.T.; Amin, S.G. Inhibition of lung carcinogenesis by black tea in Fischer rats treated with a tobacco-specific carcinogen: Caffeine as an important constituent. Cancer Res. 1998, 58, 4096–4101.
36. Wang, Z.Y.; Hong, J.Y.; Huang, M.T.; Reuhl, K.R.; Conney, A.H.; Yang, C.S. Inhibition of N-nitrosodiethylamine- and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone-induced tumorigenesis in A/J mice by green tea and black tea. Cancer Res. 1992, 52, 1943–1947.
37. Gupta, S.; Hastak, K.; Ahmad, N.; Lewin, J.S.; Mukhtar, H. Inhibition of prostate carcinogenesis in TRAMP mice by oral infusion of green tea polyphenols. Proc. Natl. Acad. Sci. USA 2001, 98, 10350–10355.
38. Adhami, V.M.; Siddiqui, I.A.; Ahmad, N.; Gupta, S.; Mukhtar, H. Oral consumption of green tea polyphenols inhibits insulin-like growth factor-I-induced signaling in an autochthonous mouse model of prostate cancer. Cancer Res. 2004, 64, 8715–8722.
39. Yuan, J.M.; Sun, C.; Butler, L.M. Tea and cancer prevention: Epidemiological studies. Pharmacol. Res. 2011, 64, 123–135.
40. Gao, Y.T.; McLaughlin, J.K.; Blot, W.J.; Ji, B.T.; Dai, Q.; Fraumeni, J.F., Jr. Reduced risk of esophageal cancer associated with green tea consumption. J. Natl. Cancer Inst. 1994, 86, 855–858.

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