

Targeting Tumor Growth and Progression in Carcinoma Prevention through Natural Catechol as Chemopreventive Agents

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ABSTRACT

Green tea is rich in catechins, which are polyphenolic chemicals that have strong anticancer effects through pro-apoptotic, anti-inflammatory, and antioxidant processes. They alter a number of molecular pathways, including as those that control the course of the cell cycle, limit angiogenesis, and reduce metastasis. With traditional Chemotherapeutics, Epigallocatechin Gallate (EGCG), the most researched catechin, exhibits synergistic actions that increase efficacy and decrease toxicity. Catechins may serve as both preventive and therapeutic agents in the treatment of carcinoma, according to data from *in vitro*, *in vivo*, and a small number of clinical investigations. However, issues like poor absorption and varying patient reactions call for sophisticated administration methods and extensive clinical testing.

Keywords: Angiogenesis, Apoptosis, Bioavailability, Cancer prevention, Cancer therapy, Carcinoma, Catechins, Epigallocatechin gallate, Green tea, Metastasis.

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INTRODUCTION

Plants naturally produce catechin, a secondary metabolite that belongs to the flavonol family and is well-known for its health-promoting qualities. Catechu, a chemical that is often produced by boiling the heartwood of the *Mimosa catechu* tree, is where the term "catechin" originates. Many different plant-based foods and drinks contain catechins. Among the most abundant sources are black tea, matcha, Korean tea, and green tea. Along with adding flavor and possible health benefits, they can also be found in grape seeds, onions, coconuts, and a variety of other fruits and vegetables.^[1] Catechin consists of three rings: a dihydropyran ring (the C ring) and two benzene rings (the A and B rings). Attached to the third carbon of this C ring is a hydroxyl group (-OH), which gives the molecule its distinctive chemical properties. The presence of two chiral centers at carbon positions 2 and 3 provides structural interest in catechin because it allows the atoms to be placed in many spatial configurations. As a consequence, four isomers are created. Compounds with the

groups on carbons 2 and 3 in a trans configuration are known as catechins, whereas epicatechins are produced when the groups are in a cis configuration.^[2] Green tea high in catechins has long been used as a natural treatment for a number of illnesses, and it is particularly valued in traditional Chinese medicine. Approximately 20% of all tea produced worldwide now is green tea. Its processing method is special because the leaves aren't fermented or severely oxidized, preserving their natural polyphenols, like as catechins, which are known to have health advantages. The high concentration of polyphenol flavonoids, particularly a class known as catechins, in tea is thought to be the primary source of the health advantages that are frequently associated with it. The distinctive chemical activity of these naturally occurring compounds is attributed to their benzopyran structure, which includes at least one aromatic ring. Due to their well-established anti-inflammatory and potent antioxidant qualities, catechins play a significant part in tea's ability to support general health.^[3] A number of studies have suggested that green tea catechins may be beneficial to combat depression,^[4-6] influenza,^[7] HIV,^[8,9] obesity,^[10] and Parkinson's and Alzheimer's diseases, e.g. by inhibiting amyloid- β fibril formation,^[11-13] several forms of cancers,^[14-17] and cardiovascular diseases.^[18-20]

The natural substances known as flavonoids are abundant in a wide variety of fruits, vegetables, roots, and flowers. The flavan core is a fundamental structure that all of them have in common. Based on the various chemical groups that are affixed to the core



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pyran ring (also known as ring C) and the degree of saturation of this ring, scientists categorize flavonoids. These minute structural variations aid in identifying the precise kind and purpose of every flavonoid.^[21] The fundamental structure of flavonoids consists of three rings: a core ring (C) with six members connected to a benzene ring (A).

This center ring is a γ -pyran ring, which is found in types such as flavonols and flavones. It has a carbonyl group at position C-4 and a phenyl group (B ring) attached at carbon position 2 (C-2). The phenyl group is bonded at C-3 rather than C-2 in isoflavonoids, which is a small difference.^[22] However, catechins differ from other flavonoids in that they are structurally simpler; they do not have a double bond between carbons 2 and 3 and do not contain the carbonyl group at C-4 (Table 1).

Flavonoids are able to carry out a variety of biological tasks due to their diverse spectrum of structures. Among them are lowering inflammation, battling bacterial and viral infections, easing allergies, avoiding blood clots, guarding against cancer and DNA damage, and even assisting in blood vessel relaxation. They are also useful in promoting general health because of their potent antioxidant qualities.^[23-25]

Molecular Mechanisms of Catechin Action in Cancer Prevention

Catechins, a subgroup of polyphenolic flavonoids abundantly present in green tea (*Camellia sinensis*), have gained significant attention for their chemopreventive and therapeutic potential against a variety of cancers. Among these, Epigallocatechin-3-Gallate (EGCG) is the most abundant and bioactive catechin, constituting approximately 50-80% of the total catechin content in green tea.^[42] EGCG exhibits a broad spectrum of anticancer effects by targeting multiple cellular and molecular pathways that are critical in cancer development, progression, and metastasis. One of the foremost mechanisms through which EGCG exerts its anticancer action is by reducing oxidative stress. It functions as a strong free radical scavenger, neutralizing Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS), which are known to cause DNA damage, genomic instability, and malignant transformation.^[43] EGCG stimulates the expression and activity of endogenous antioxidant enzymes such as Superoxide Dismutase (SOD), catalase, and Glutathione Peroxidase (GPx), thereby strengthening the cell's intrinsic antioxidant defense system and preventing oxidative DNA lesions.^[44]

In addition to its antioxidant function, EGCG modulates several intracellular signaling pathways involved in tumor cell survival and proliferation. Specifically, it inhibits the Phosphoinositide 3-Kinase (PI3K)/Akt pathway, which plays a pivotal role in promoting cancer cell growth and resistance to apoptosis.^[45] Similarly, EGCG suppresses the Mitogen-Activated Protein Kinase (MAPK)/extracellular Signal-Regulated Kinase (ERK) pathway, thereby inducing cell cycle arrest at the G1 or G2/M

phase and promoting programmed cell death in various cancer cell lines, including those of the breast, colon, prostate, and lung.^[46]

EGCG also has profound effects on apoptotic regulation. It has been shown to upregulate tumor suppressor genes such as p53 and pro-apoptotic proteins like Bax, while simultaneously downregulating anti-apoptotic proteins including Bcl-2 and Bcl-xL, tipping the balance in favor of apoptosis.^[47] This regulation of apoptotic machinery is crucial for the elimination of transformed or mutated cells before they progress to invasive cancer. EGCG exhibits anti-angiogenic and anti-metastatic properties, which are essential for inhibiting tumor growth and dissemination. It suppresses the expression of Vascular Endothelial Growth Factor (VEGF), a key factor that promotes the formation of new blood vessels in tumors (angiogenesis).^[48] Additionally, EGCG downregulates the activity of Matrix Metalloproteinases (MMP-2 and MMP-9), enzymes that degrade the extracellular matrix and facilitate cancer cell invasion and metastasis.^[49] EGCG also influences cancer-related inflammatory pathways. It inhibits the activation of Nuclear Factor-Kappa B (NF- κ B), a transcription factor that controls the expression of numerous genes involved in inflammation, proliferation, angiogenesis, and immune evasion.^[50] Inhibition of NF- κ B by EGCG leads to a decrease in the production of pro-inflammatory cytokines such as Interleukin-6 (IL-6), Tumor Necrosis Factor-alpha (TNF- α), and Cyclooxygenase-2 (COX-2), creating an unfavorable microenvironment for tumor progression. EGCG exerts epigenetic effects by modulating the activity of DNA Methyltransferases (DNMTs) and Histone Deacetylases (HDACs) (Table 2).

. These enzymes are often overexpressed in cancer cells, leading to the silencing of tumor suppressor genes via promoter hypermethylation and chromatin condensation. EGCG inhibits these epigenetic enzymes, resulting in reactivation of silenced genes and restoration of normal cell regulatory functions (Figure 1).^[51]

Antioxidant and Anti-inflammatory Properties of Catechin in Carcinogenesis

Catechins, particularly Epigallocatechin-3-Gallate (EGCG) from green tea, have shown strong protective effects against cancer, largely due to their antioxidant and anti-inflammatory properties. These two functions are closely connected and play a key role in preventing the initiation, promotion, and progression of cancer.

From a human biology perspective, our bodies are constantly exposed to oxidative stress—a condition caused by an imbalance between free radicals (such as reactive oxygen species or ROS) and the body's ability to neutralize them with antioxidants. These free radicals can damage DNA, proteins, and cell membranes, leading to mutations and the start of cancer development.^[52] Catechins like EGCG act as natural antioxidants, meaning they

can scavenge or neutralize these harmful molecules before they cause damage. Additionally, they boost the activity of internal antioxidant enzymes, such as Superoxide Dismutase (SOD), Glutathione Peroxidase (GPx), and catalase, which are vital for maintaining cellular health and preventing oxidative damage.^[53,54]

Alongside oxidative stress, chronic inflammation is another major driver of cancer. Inflammatory processes create an environment where damaged cells are more likely to survive, proliferate, and eventually transform into cancer cells. Persistent inflammation is often associated with increased production of cytokines (like TNF- α and IL-6), pro-inflammatory enzymes (like COX-2 and iNOS), and activation of transcription factors such as NF- κ B and STAT3, all of which are commonly elevated in cancerous tissues.^[55] Catechins help by suppressing these inflammatory pathways. For example, EGCG can block the NF- κ B signaling pathway, preventing it from turning on genes that promote inflammation, cell survival, and proliferation.^[56] This suppression leads to a decrease in pro-inflammatory mediators, creating an environment that is less favorable for cancer growth. Catechins contribute to reducing inflammation-induced DNA damage and angiogenesis (formation of new blood vessels) two processes that cancer cells rely on to grow and spread.^[57]

Catechin-Mediated Modulation of Cell Signaling Pathways in Carcinoma

Catechins, especially Epigallocatechin-3-Gallate (EGCG) found in green tea, help in preventing and slowing down cancer by modifying key cell signaling pathways that are often hijacked in cancer cells. These pathways act like “communication highways” inside the cell, sending messages that tell the cell when to grow, divide, repair damage, or die. In carcinoma (cancers that originate from epithelial tissues), these messages often become faulty, leading to uncontrolled growth and resistance to cell death. Catechins step in and disrupt these harmful signals, helping to restore balance. One of the major pathways affected by EGCG is the PI3K/Akt pathway, which normally helps cells survive and grow. In many cancers, this pathway is overactive, allowing cancer cells to keep growing unchecked and avoid apoptosis (programmed cell death). EGCG can block the activation of Akt, leading to reduced cell survival and increased cell death.^[58]

Another important pathway is the MAPK/ERK pathway, which controls how cells respond to growth signals. When this pathway is too active, it can cause rapid cell division a hallmark of cancer. Catechins help suppress ERK activation, slowing down this uncontrolled cell division.^[59] EGCG also supports cancer prevention by acting on the Wnt/ β -catenin signaling pathway, which is often involved in colon and breast cancers. By reducing β -catenin levels, EGCG prevents the activation of genes that promote cancer cell growth and spread.^[60] In addition, EGCG

inhibits NF- κ B, a protein complex that turns on genes related to inflammation and cell survival. Since inflammation is closely linked with cancer, blocking NF- κ B reduces the inflammatory environment that helps tumors grow.^[61] Similarly, EGCG can suppress the STAT3 pathway, which when overactive, supports tumor development and immune evasion.^[62]

Induction of Apoptosis and Cell Cycle Arrest by Catechins

Catechins especially Epigallocatechin-3-Gallate (EGCG) found in green tea play a powerful role in fighting cancer by triggering apoptosis (programmed cell death) and halting the cancer cell cycle. These are two of the most important ways the body controls abnormal or dangerous cells. In cancer, these systems are often broken, allowing cancer cells to multiply rapidly and avoid dying. Catechins help to restore these natural defense mechanisms.

Catechins Induce Apoptosis (Cell Death)

Apoptosis is like the cell's built-in self-destruct system. It allows the body to eliminate old, damaged, or potentially dangerous cells in a clean and controlled way. In many cancers, this system is turned off meaning cells grow unchecked.

Catechins such as EGCG can reactivate apoptosis in cancer cells through several key actions:

1. Increase in pro-apoptotic proteins like Bax and p53, which encourage cells to die when they're abnormal,^[63]
2. Decrease in anti-apoptotic proteins such as Bcl-2, which normally protect cancer cells from dying,^[64]
3. Activation of caspases, especially caspase-3 and caspase-9, which are enzymes that cut up and dismantle cancer cells during apoptosis,^[65]
4. Release of cytochrome c from the mitochondria, triggering the final steps of the apoptotic process.^[66]

Catechins Cause Cell Cycle Arrest

Catechins can pause the cell cycle, which is the process cells go through to grow and divide. In cancer, the cell cycle is often out of control. Catechins interfere with proteins that regulate the cell cycle, causing it to stall at specific checkpoints.

1. Catechins can block the G1/S and G2/M phases, preventing DNA replication and division of cancer cells,^[67]
2. They suppress the activity of cyclins and Cyclin-Dependent Kinases (CDKs) the green lights of the cell cycle,^[68]
3. They increase CDK inhibitors like p21 and p27, which act like "red lights", stopping the cycle when needed.^[69]

Catechin and Angiogenesis Inhibition in Tumor Progression

The process by which tumors stimulate the formation of new blood vessels. These vessels act like highways, delivering oxygen and nutrients to the tumor and allowing it to grow, survive, and eventually spread (metastasize) to other parts of the body. Without new blood vessels, tumors can't grow beyond a small size. This is where catechins, especially Epigallocatechin-3-Gallate (EGCG) from green tea, come into play. Catechins have been shown to inhibit angiogenesis, thereby cutting off the tumor's lifeline and slowing down its growth and spread. Angiogenesis is a healthy process used in wound healing and tissue growth. But in cancer, it becomes hijacked. Tumors release pro-angiogenic factors like Vascular Endothelial Growth Factor (VEGF), which signal nearby blood vessels to grow toward the tumor.^[70] This creates a tumor-friendly environment, helping cancer cells not only thrive but also travel through the bloodstream to form new tumors (Metastasis).

Disruption of Angiogenesis by Catechin

Inhibition of VEGF/EGCG blocks the expression of VEGF, one of the most important proteins responsible for triggering new blood vessel formation. Without VEGF, the tumor cannot effectively signal for more blood supply.^[71]

Blocking VEGF receptors

Even if VEGF is present, EGCG can inhibit the activity of VEGF receptors (VEGFR) on endothelial cells (the cells that line blood

vessels), preventing the message from being received and acted upon.^[72]

Inhibition of MMPs

Catechins also suppress Matrix Metalloproteinases (MMP-2 and MMP-9) enzymes that break down surrounding tissues and allow blood vessels to grow into the tumor. These enzymes also help cancer cells invade nearby tissues, so inhibiting them reduces both angiogenesis and metastasis.^[73]

Suppression of HIF-1 α

Under low Oxygen (hypoxic) conditions, tumors activate hypoxia-inducible factor 1-alpha (HIF-1 α), a transcription factor that increases VEGF production. Catechins inhibit HIF-1 α activity, further reducing VEGF levels and angiogenesis.^[74]

Experimental Evidence

Studies using various cancer models, including breast, prostate, and colon cancers, have shown that treatment with EGCG results in fewer new blood vessels around tumors, smaller tumor sizes, and reduced metastasis. In some cases, combining catechins with chemotherapy drugs improved the anti-angiogenic effects and made tumors more sensitive to treatment.^[75,76] Pharmacokinetics and Metabolic Fate of Catechins in Humans: Catechins, especially Epigallocatechin-3-Gallate (EGCG) from green tea, are widely studied for their health-promoting properties. However, their actual effectiveness in the human body depends on how they are absorbed, distributed, metabolized, and excreted a process known as pharmacokinetics.

Table 1: Biological botanical Sources and concentration of Catechin.

Common Name	Botanical Name	Plant Part Used	Approx. Content (mg/100g or mL)	Reference
Green Tea	<i>Camellia sinensis</i>	Leaves (dried)	50-100 mg/100 mL	[26]
Black Tea	<i>Camellia sinensis</i>	Leaves (fermented)	20-50 mg/100 mL	[26,27]
Cocoa	<i>Theobroma cacao</i>	Seeds (dried)	50-100 mg/100 g	[28]
Arjuna	<i>Terminalia arjuna</i>	Bark	~10-25 mg/100 g	[29]
Gooseberry (Amla)	<i>Phyllanthus emblica</i>	Fruit	~20-40 mg/100 g	[30]
Witch Hazel	<i>Hamamelis virginiana</i>	Bark, leaves	~30-50 mg/100 g	[31]
Hawthorn	<i>Crataegus monogyna</i>	Berries, leaves	~10-20 mg/100 g	[32]
Grape Seed	<i>Vitis vinifera</i>	Seeds	100-500 mg/100 g	[33]
Cinnamon	<i>Cinnamomum verum</i>	Bark	~40-100 mg/100 g	[34]
Oak Tree	<i>Quercus robur</i>	Bark	~50-100 mg/100 g	[35]
Blackberries	<i>Rubus fruticosus</i>	Fruit	5-20 mg/100 g	[36]
Raspberries	<i>Rubus idaeus</i>	Fruit	5-20 mg/100 g	[37]
Fava Beans	<i>Vicia faba</i>	Seeds	~12 mg/100 g	[38]
Cinnamon Bark	<i>Cinnamomum cassia</i>	Bark	~80-120 mg/100 g	[39]
Pomegranate	<i>Punica granatum</i>	Peel	~20-60 mg/100 g	[40]

Table 2: Physical and chemical properties.^[41]

Property	Details
Chemical Formula	C ₁₅ H ₁₄ O ₆
Molecular Weight	290.27 g/mol
Appearance	White to pale yellow crystalline powder
Melting Point	175-177°C
Solubility in Water	Slightly soluble
Solubility in Ethanol	Soluble
Boiling Point	Decomposes before boiling
Optical Rotation	[α] _D ²⁰ +15.6° (c = 1 in methanol)
Stability	Light- and air-sensitive; stable dry
Log P	0.44
UV Absorption Maxima	279 nm

Absorption: Only a Small Amount Enters the Bloodstream

When we drink green tea or take catechin supplements, catechins pass through our digestive system. But unfortunately, only a small portion is absorbed into the blood. This is because:

Catechins are sensitive to pH and enzymes in the digestive tract.

Their chemical structure makes it hard to pass through the intestinal wall. Studies show that less than 5% of the consumed EGCG actually gets into the bloodstream.^[77,78]

Distribution: Reaches Body Tissues in Tiny Amounts

Once in the blood, catechins are circulated to various organs. However, their concentration remains low, even after drinking several cups of green tea. Typically, blood levels of EGCG stay under 0.3 μM, which is much lower than the doses used in lab experiments.^[79]

Metabolism: Quickly Broken Down

After absorption, catechins are rapidly broken down (metabolized), mainly by the liver and gut bacteria:

In the liver, they undergo changes like methylation, sulfation, and glucuronidation, which help make them more water-soluble for excretion.^[80]

In the colon, gut microbes break down unabsorbed catechins into smaller compounds. Some of these byproducts may still have health benefits.^[81]

Excretion: Removed from the Body Quickly

Catechins and their breakdown products are excreted mostly through urine and feces. Their half-life (how long it takes for

the body to eliminate half the amount) is short about 1 to 4 hr, meaning they don't stay in the body for long.^[82]

Clinical Trials and Epidemiological Evidence of Catechin Efficacy

Catechins especially Epigallocatechin Gallate (EGCG) found in green tea have gained widespread attention for their potential to prevent or slow the growth of cancer. This is where clinical trials and epidemiological studies come in. These studies help scientists understand how catechins behave in the human body, and whether people who consume more catechins actually have lower cancer risks.

Clinical Trials: Testing Catechins in Real Patients

Clinical trials involve giving catechins or green tea extracts to patients under controlled conditions.

i. Prostate cancer prevention: A study on men with pre-cancerous prostate conditions (high-grade prostatic intraepithelial neoplasia) showed that those who took green tea catechins (600 mg/day) had a 90% lower chance of developing prostate cancer after one year.^[83]

ii. Oral cancer prevention: In people with oral leukoplakia (a precancerous condition), EGCG supplements helped reduce lesion size and cancer progression.^[84]

iii. Colorectal cancer recurrence: A Japanese study followed patients who had surgery for colorectal cancer. Those who consumed high amounts of green tea had a significantly lower recurrence rate and better survival outcomes.^[85]

iv. Side effects and tolerance: Catechins are well-tolerated by most people, but high doses may cause nausea or liver enzyme elevations, so dosing must be monitored.^[86]

Epidemiological Studies

These studies look at large populations to find links between green tea consumption and cancer rates.

(i) Lower cancer risk in Asia: In Japan and China, where green tea consumption is high, several studies found a reduced risk of cancers such as breast, prostate, and lung cancer.^[87,88]

(ii) Dose matters: People who drank more than 5 cups of green tea daily were less likely to develop various cancers compared to those who drank less.^[89]

(iii) Gender and lifestyle differences: Some studies suggest that catechins may be more effective in women, and their protective effect might be influenced by factors like smoking, diet, or genetics.^[90]

Safety, Toxicity, and Therapeutic Limitations of Catechins

- 1. General Safety at Dietary Levels:** Drinking 2-3 cups of green tea per day is safe and may offer health benefits without side effects. Catechins consumed through the diet are well-tolerated, and green tea has a long history of use in traditional medicine.^[91]
- 2. Toxicity at High Doses:** When catechins are consumed in high concentrations, especially as purified supplements, there is potential for toxicity.

(i) Liver toxicity: Several case reports and clinical trials have reported hepatotoxicity (liver injury) in individuals taking green tea extract supplements, particularly on an empty stomach. The liver damage appears to be dose-dependent, and reversible when the supplement is discontinued.^[92,93]

(ii) Gastrointestinal upset: High doses of EGCG (>500 mg/day) may cause nausea, abdominal discomfort, or diarrhea in some people.^[94]

(iii) Iron absorption inhibition: Catechins can bind to dietary iron and may reduce its absorption, potentially worsening anemia in susceptible individuals.^[95]

Therapeutic Limitations

Although EGCG has shown anticancer activity *in vitro*, translating those effects to humans faces several issues.

(i) Poor bioavailability: Catechins, especially EGCG, are poorly absorbed in the intestine, rapidly metabolized in the liver, and quickly eliminated from the body. This limits their therapeutic impact *in vivo*.^[96]

(ii) Instability in digestive tract: EGCG is chemically unstable under the pH conditions found in the stomach and small intestine, which further reduces the amount available for biological action.^[97]

(iii) Inconsistent results in clinical trials: While some small studies show benefits, larger and longer-term clinical trials often produce inconclusive or modest effects, especially in cancer prevention.^[98]

Risk in Sensitive Populations

(i) Pregnancy and breastfeeding: While moderate tea consumption is safe, green tea extracts are not recommended during pregnancy due to limited safety data.

(ii) People with liver conditions: Those with pre-existing liver issues or taking other hepatotoxic drugs should avoid catechin supplements.

(iii) Drug interactions: Catechins may interact with medications, including beta-blockers, anticoagulants, and chemotherapy drugs, affecting their efficacy or metabolism.^[99]

Future Perspectives and Challenges in Catechin-Based Cancer Therapy Epigallocatechin Gallate (EGCG), in particular, is a catechin that has shown promise as a bioactive substance with potential uses in cancer treatment and prevention. Through mechanisms like antioxidant activity, angiogenesis suppression, and cell signaling pathway regulation, extensive preclinical research shows their capacity to disrupt several stages of carcinogenesis, including initiation, promotion, and progression.^[100-102]

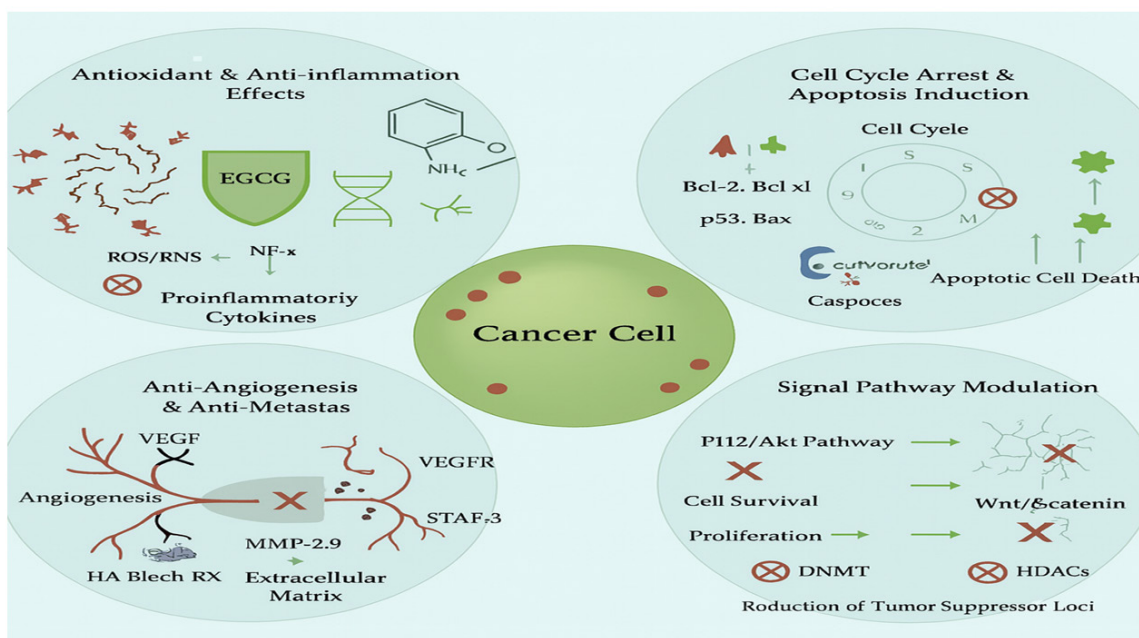


Figure 1: Flavanol-3-ol prevent the cell apoptosis in molecular pathway in cancer and anti-inflammatory regulation.^[51]

Future Perspectives

The next phase of catechin-based cancer therapy is expected to focus on improving delivery systems and bioavailability. Nanotechnology offers a significant opportunity in this regard. Encapsulating catechins in nanoparticles, liposomes, micelles, or polymeric carriers can protect them from gastrointestinal degradation, improve systemic absorption, and facilitate targeted delivery to tumor sites.^[103,104]

Catechins have been demonstrated to make cancer cells more sensitive to common chemotherapeutic medicines including 5-fluorouracil, doxorubicin, and cisplatin, which may reduce the need for higher dosages of the medication and lessen adverse effects.^[105,106] Their capacity to lessen oxidative stress, encourage apoptosis in cancerous cells, and alter drug-resistance pathways is what is responsible for this synergistic action.^[107]

Catechin treatment has potential in personalized medicine as well. Developments in metabolomics and genetics may make it possible to pinpoint patient subgroups who benefit most from catechin administration, allowing for customized treatments that minimize dangers and optimize benefits.^[108]

CONCLUSION

Catechins are a special class of natural substances that may be used in conjunction with traditional cancer treatments. Future cancer preventive and treatment efforts could benefit from their multi-targeted mode of action, dietary safety, and possible incorporation into precision oncology. They must, however, overcome obstacles like low bioavailability, inconsistent patient response, and scant clinical trial data if they are to succeed. With more multidisciplinary research and technology advancements, catechin-based cancer treatment may soon go from encouraging lab results to successful clinical treatments.

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ABBREVIATIONS

EGCG: Epigallocatechin Gallate; **-OH:** Hydroxyl Group; **PI3K:** Phosphoinositide 3-Kinase; **Akt:** Protein Kinase B; **MAPK:** Mitogen-Activated Protein Kinase; **ERK:** Extracellular Signal-Regulated Kinase; **p53:** Tumor Suppressor Protein p53; **Bax:** Bcl-2-Associated X Protein; **Bcl-2:** B-Cell Lymphoma 2; **Bcl-xL:** B-Cell Lymphoma-Extra Large; **VEGF:** Vascular Endothelial Growth Factor; **MMP:** Matrix Metalloproteinase; **NF-κB:** Nuclear Factor-Kappa B; **IL-6:** Interleukin-6; **TNF-α:** Tumor Necrosis Factor-Alpha; **COX-2:** Cyclooxygenase-2; **DNMTs:** DNA Methyltransferases; **HDACs:** Histone Deacetylases; **ROS:** Reactive Oxygen Species; **RNS:** Reactive Nitrogen Species; **SOD:** Superoxide Dismutase; **GPx:** Glutathione Peroxidase; **iNOS:**

Inducible Nitric Oxide Synthase; **STAT3:** Signal Transducers And Activators Of Transcription 3; **CDKs:** Cyclin-Dependent Kinases; **HIF-1α:** Hypoxia-Inducible Factor 1-Alpha; **μM:** Micromolar.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

SUMMARY

Catechins, notably Epigallocatechin Gallate (EGCG) sourced from green tea, utilize a multi-faceted strategy to serve as formidable chemopreventive compounds against carcinoma. They provide cellular defense through robust anti-inflammatory and antioxidant attributes (for instance, by inhibiting NF-κB). They actively combat malignant proliferation by enforcing cell cycle cessation and triggering apoptosis, or programmed cell death. Furthermore, these compounds thwart tumor progression by impeding both metastasis (cancer spread) and angiogenesis (the formation of new blood vessels, achieved by blocking VEGF). The principal impediment to their clinical utility is their limited human bioavailability. Consequently, forthcoming endeavors will focus intently on advanced delivery modalities, such as nanoparticle encapsulation, to augment systemic absorption and thus optimize their therapeutic potential.

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