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Green Tea Catechins in Cancer Prevention and Therapy: Molecular Mechanisms, Clinical Evidence, Safety, and Future Perspectives

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Abstract

Green tea, derived from *Camellia sinensis*, is a widely consumed dietary source of bioactive polyphenols. Among its major catechins, epigallocatechin-3-gallate (EGCG) has been studied most extensively in cancer research. This review critically synthesizes phytochemical, molecular, preclinical, epidemiological, clinical, pharmacokinetic, and safety evidence to assess the potential role of green tea catechins in cancer prevention and treatment. Preclinical studies of breast, prostate, colorectal, lung, liver, oral, and hematological cancers indicate that EGCG modulates oxidative stress, inflammatory signaling, proliferation, apoptosis, cell-cycle progression, angiogenesis, epithelial–mesenchymal transition, invasion, metastasis, and epigenetic regulation. Reported molecular targets include the PI3K–AKT–mTOR, MAPK, JAK–STAT, and NF-κB signaling pathways, as well as EGFR and VEGF signaling, BCL-2 family proteins, caspases, matrix metalloproteinases, and enzymes involved in DNA methylation. A systematic review and meta-analysis reported an association between green tea or EGCG exposure and lower overall cancer risk, with variation by cancer type and study design. Clinical evidence for efficacy in established malignancies remains limited, and conventional oncological treatment remains the standard of care. By integrating mechanistic findings with human data, this review identifies gaps in clinical efficacy, pharmacokinetics, and safety that must be addressed before green tea catechins can be incorporated into evidence-based oncology.

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Keywords: Green Tea Catechins; Epigallocatechin-3-Gallate; Cancer Chemoprevention; Cancer Therapy; Clinical Translation

Introduction

Cancer is a heterogeneous group of diseases associated with the development of progressive molecular and cellular alterations that allow cancer cells to proliferate, evade their growth, survive death, become genomically unstable, adapt to metabolic changes, initiate angiogenesis, escape the immune system, invade tissues and spread to other parts of the body. Despite significant improvements in surgery, radiotherapy, cytotoxic chemotherapy, endocrine therapy, targeted agents and immunotherapy, resistance to treatment, recurrence, toxicity, and metastatic progression still remain as a driver of research for new strategies to prevent and treat many malignancies. Historically, natural products have yielded a large number of pharmacologically active molecules and dietary phytochemicals are also currently being studied as regulators of intracellular signaling, transcription, metabolism, inflammation and epigenetic regulation.

Green tea is made from Camellia sinensis leaves using relative minimal enzymatic oxidation during the tea-making process which leaves a significant amount of catechins as polyphenols. The main catechins are EGCG, epigallocatechin, epicatechin gallate and epicatechin. In the experimental oncology literature, EGCG stands as the most prominent compound due to its chemical structure, which allows it to act as a redox agent, a hydrogen bond donor and acceptor, bind to proteins and membranes and regulate a large number of enzymes and signaling pathways [1, 2]. This biochemical promiscuity accounts for some of EGCG's reported effects that are not the typical single-target effect of a drug.

Evidence refer to the scientific interest of green tea and malignancy at several levels. Antiproliferative and pro-apoptotic effects have been observed in cell-culture studies, tumour inhibition in tumor initiation or tumour growth in animal studies in selected settings, associations of tea consumption and cancer incidence in observational studies, and standardized preparations of catechins have been tested in selected populations in clinical trials. Levels of evidence do not mean that one should interchange them. Inhibitory in vitro concentrations may not be obtained in vivo in human tumors after oral administration, observations may be confounded by other variables and changes in the biomarker may not

lead to reduced recurrence or mortality. Therefore, a serious evaluation procedure should differentiate a biological plausibility from proven clinical effectiveness [3, 4, 5].

The present review summarizes the green tea as a source of bioactive compounds with anticancer activity and focuses on EGCG. It examines the chemical composition, mechanisms that are relevant to malignant transformation, evidence from major tumor types, evidence from human prevention and treatment, pharmacokinetic limitations, safety and drug interactions, and priorities toward future translational research.

Phytochemical Composition and Biologically Active Constituents

The green tea is composed of a mixture of polyphenols, methylxanthines, amino acids, vitamins, minerals and volatile compounds. The polyphenolic fraction is the one that has received the most attention with catechins being the most studied. The most famous of the EGGs is EGCG, followed by epigallocatechin, epicatechin gallate and epicatechin. Gallate group and several hydroxyl groups of EGCG allow to interact with the reactive species, transition metals, proteins, phospholipid membranes, and signaling molecules. Green tea is also high in caffeine and theanine, and the quantities of each can differ according to cultivar, geographic location, leaf maturity, processing method, tea-brewing temperature, and brewing time as well as commercial preparation [1].

Clinically relevant compositional variability. The biological effects and systemic exposure of brewed green tea, decaffeinated tea, purified EGCG and concentrated green tea extracts cannot be assumed to be the same. Doses of catechins that can be present in standardised preparations for clinical trials can be significantly higher than those that are consumed in the diet. Therefore, the actual catechin composition and dose should be indicated instead of using a general term for 'green tea'. A summary of the main properties of the principal constituents of green tea, their importance in cancer studies, and the major drawbacks are all summarized in Table 1.

Table 1. Major bioactive constituents of green tea and their relevance to cancer research.

Constituent	Chemical group	Relevance to cancer research	Major limitation
Epigallocatechin-3-gallate (EGCG)	Catechin	Most extensively studied; affects multiple signaling, apoptotic, angiogenic and epigenetic pathways	Limited and variable oral bioavailability; high-dose extract safety concerns

Constituent	Chemical group	Relevance to cancer research	Major limitation
Epigallocatechin (EGC)	Catechin	Redox and metabolic activity; contributes to total catechin exposure	Less extensively characterized in oncology
Epicatechin gallate (ECG)	Catechin	Antioxidant and signaling effects	Lower clinical evidence than EGCG
Epicatechin (EC)	Catechin	Biological effects on vascular and cellular physiology	Not a major focus of cancer trials
Caffeine	Methylxanthine	May modify physiological responses and tolerability	Content differs markedly among products

Molecular Mechanisms Relevant to Malignant Disease

Redox Regulation and Oxidative Stress

Oxidative stress has a role in carcinogenesis through its effects on DNA damage, genomic instability, lipid and protein oxidation, inflammatory signaling and altered cell survival. EGCG has been called an antioxidant due to its ability to donate electrons and interact with reactive species. But, its behavior is more complex. In certain experimental conditions, EGCG can be auto-oxidized and lead to the formation of ROS that create a pro-oxidant environment that can help in cancer-cell damage. Lambert and Elias [6] pointed out that the biological effects of tea polyphenols could be attributed to the antioxidant and pro-oxidant effects. Final response will be dependent on the concentration, oxygen tension, metal ions, cell type, the extracellular environment and the antioxidant status.

The dual redox is significant for interpretation of anticancer experiments. Oxidative stress to normal cells may be important in prevention, while controlled increases in oxidative stress in malignant cells may be important in mitochondrial dysfunction and apoptosis. Thus, the anticancer hypothesis is not limited to an antioxidant action.

Induction of Apoptosis and Mitochondrial Dysfunction

When apoptosis fails, the genetically damaged cells survive and is a hallmark of most malignancies. EGCG has been found to affect both the intrinsic and extrinsic apoptotic pathways. Experimental studies include modulation of BCL-2 family proteins, loss of mitochondrial membrane potential, release of cytochrome-c, and activation of caspases. EGCG may decrease the balance of antiapoptotic signals and increase proapoptotic signals, leading to programmed cell death [7, 8].

There are significant variations between cell lines in the amount of apoptosis induced by exposure to concentrations or length of duration. This is a significant restriction because the concentrations used in cell culture (micromolar) can be higher than the concentrations that can be reached in vivo via normal oral intake. However, the frequent presence of apoptotic targets in various experimental models of cancer suggests that apoptosis is a general mechanism that plays a key role in cancer.

Cell-Cycle Arrest and Suppression of Proliferation

In uncontrolled proliferation, the dysregulation of cyclins, cyclin dependent kinases, checkpoint proteins, and growth factor signaling are involved. Increased expression/activity of cell-cycle inhibitory proteins like p21 and p27, and decreased expression/activity of selected cyclins and cyclin-dependent kinases, are associated with EGCG. These changes may result in arrest at the G1/S and/or G2/M transition, depending on which experimental model is used. This leads to decreased DNA replication and progression through the cell cycle and is responsible for the antiproliferative phenotype observed in breast, prostate, colorectal, lung, and hematological cancer models [7, 9].

PI3K/AKT/mTOR, MAPK and JAK/STAT Signaling

The phosphoinositide 3-kinase/protein kinase B/mammalian target of rapamycin pathway is a common avenue of activation in cancer and a pathway that regulates survival, protein synthesis, glucose metabolism, proliferation, and resistance to therapy. In a variety of experimental systems it has been reported that EGCG can inhibit components of the pathway. Likewise, mitogen-activated protein kinase signaling and Janus kinase/signal transducer and activator of transcription signaling have been found to be targets for modulation by catechin. These pathways are very tightly coupled and changes in a specific pathway could affect multiple phenotypes in the downstream pathways such as apoptosis, proliferation, inflammation and angiogenesis [9].

It is proposed that EGCG may interfere with a number of oncogenic pathways at once, which could be considered as an advantage in complex diseases like cancer. Conversely, the multi-target activity increases the difficulties of the pharmacological interpretation as the dominant target can change between tumor types and concentrations. For this reason,

studying the molecular effects at concentrations that are attainable in the clinic is necessary for translational studies.

Inflammatory Signaling and NF-kappa B

Chronic inflammation may contribute to the initiation and promotion of tumors through the production of reactive species, facilitation of proliferation, inhibition of anti-tumoral immune response, induction of angiogenesis and remodeling of tissues. Nuclear factor-kappa B is a key inflammatory and life-prolonging transcription factor. Experimental investigations have shown that EGCG could reduce the activation of NF-kappa B and modulate inflammatory mediators such as the cyclooxygenase-2, tumour necrosis factor alpha, interleukin 6 and associated cytokine systems [9, 10].

The anti-inflammatory properties could be especially important in cancer prevention in tissues that are chronically stimulated by inflammatory signals. Nevertheless, alterations in circulating inflammatory biomarkers should not be considered as a tumor control marker without establishing correlation with relevant oncological outcomes in clinical trials.

Angiogenesis

Solid tumors need a growing network of blood vessels to acquire oxygen and nutrients to spread metastases. Angiogenesis is a process regulated by many factors, including vascular endothelial growth factor. In experimental models, EGCG has been reported to decrease VEGF expression or signaling, and to inhibit endothelial-cell proliferation, migration and tube formation. Other effects on hypoxia-responsive signals and matrix remodeling may also contribute to the reduction of the angiogenic phenotype [3].

Antiangiogenic activity is a possible mechanism to decrease tumor growth in animals, but there is not yet enough clinical proof that green tea catechins can be considered antiangiogenic cancer drugs. The separation is relevant because in clinical oncology, one has to get the exposure right, have validated targets and have shown therapeutic benefit when trying to inhibit angiogenesis.

Invasion, Epithelial-Mesenchymal Transition and Metastasis

For metastasis to occur, cancer cells must detach, modify the extracellular matrix, move, penetrate, survive in circulation and stop at and invade other tissues. This process involves participation of matrix metalloproteinases, cell-adhesion molecules, cytoskeletal

regulators and epithelial-mesenchymal transition programs. It has been reported that EGCG can regulate MMPs, inhibit the migration and invasion of various cell models. Signaling related to the epithelial-mesenchymal transition has also been shown to be affected [9, 11].Recurrence, metastatic progression or endpoints of disease-free survival would be necessary in future trials to demonstrate clinically relevant antimetastatic activity.

Epigenetic Regulation

Abnormal epigenetic regulation contributes to cancer development, alongside DNA sequence changes. Tumor suppressor genes can be turned off and/or oncogenic programs can be activated by DNA methylation, histone modifications, chromatin remodeling, and non-coding RNAs. EGCG has been studied as a DNA methyltransferase (DNMT) inhibitor and other epigenetic enzymes. Experimental evidence indicates it can affect methylation and re-express certain genes, although concentrations and cell types will be important factors [9, 12].

Epigenetics is a promising field of research for chemoprevention because epigenetic changes are reversible. But epigenetic activity needs to be shown in realistic exposure levels in human target tissues before it can be extrapolated to clinical recommendations. The interrelated anticancer mechanisms are conceptually integrated as shown in Figure 1.

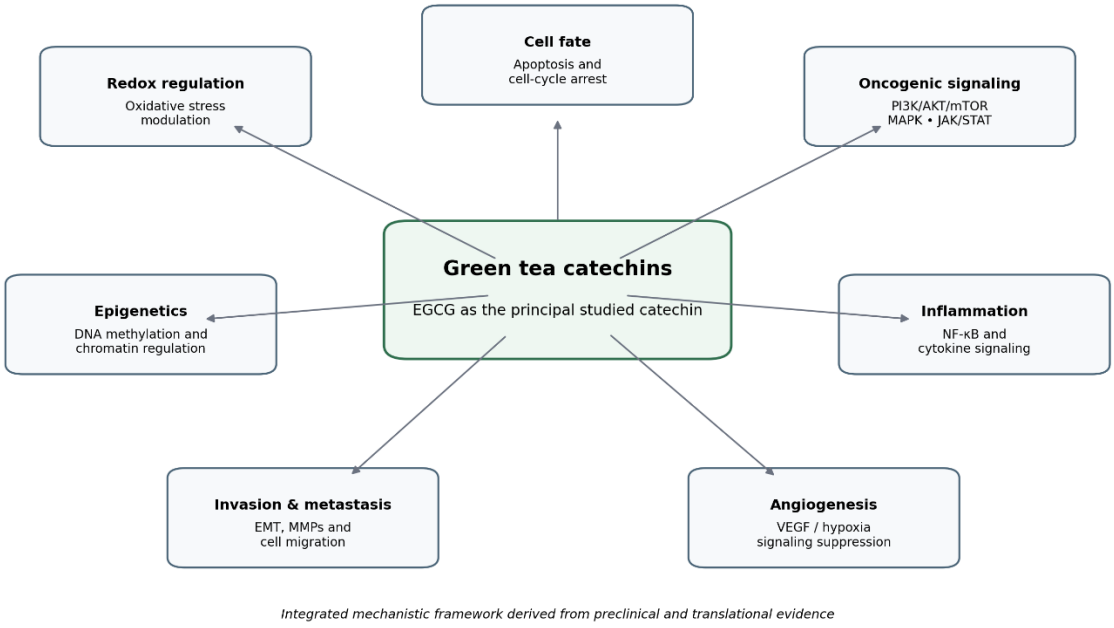


Figure 1. Integrated molecular mechanisms proposed for green tea catechins, particularly EGCG, in malignant disease. The schematic synthesizes redox, apoptotic, proliferative, inflammatory, angiogenic, metastatic, and epigenetic pathways discussed in refs. [6].

Green Tea and Breast Cancer

Breast cancer is a biologically heterogeneous disease type comprising hormone receptor-positive, HER2-positive and the triple-negative disease type. Pilot studies show that EGCG can inhibit breast cancer cell proliferation, induce cell death, disrupt receptor-mediated signaling, inhibit blood vessel growth, and inhibit migration. A recent systematic review and meta-analysis of preclinical female hormone-dependent cancer models showed that tumors were reduced in size in both breast and ovarian cancer models, but that there was little clinical evidence to support this.

Biomarker studies have long been the focus of human intervention studies, but not outcome studies. For instance, in the Minnesota Green Tea Trial, standardized green tea extract was studied in postmenopausal women who were at higher risk for breast cancer. These studies are useful for pharmacology and prevention studies, but they don't prove that green tea is an effective treatment for invasive breast cancer. Concentrated extracts should be used in patients under endocrine, chemotherapy, targeted and immunotherapy treatment, so it is better to consult a doctor about these patients.

Green Tea and Prostate Cancer

Prostate cancer is included in the most well studied cancers regarding green tea. The proposed mechanisms are indirect activation of the androgen receptor, inhibition of proliferative pathways, induction of apoptosis, reduction in inflammatory pathways and suppression of angiogenesis. A randomized placebo-controlled trial of standardized green tea catechins in men at increased risk employed a product that provided ~400mg EGCG per day for one year. The study did not show a statistically significant decrease in its primary endpoint of the incidence of prostate cancer, but selected secondary endpoints appeared to raise additional research questions [13].

A narrative review published in 2026 suggested that although results from mechanistic and preclinical studies are promising, definitive evidence of therapeutic efficacy is lacking due to the limited number of human trials, relatively small size of human trials, and variation in formulations. A good illustration of the larger body of green tea literature: the biological plausibility is much greater than clinical evidence.

Green Tea and Colorectal Cancer

The mechanisms of colorectal carcinogenesis include genetic mutations, inflammatory processes, metabolic abnormalities, and a gradual process of transformation of the colon epithelial cells. The catechins found in green tea have been studied for their antioxidant effects, anti-inflammatory effects, pro-apoptotic effects, anti-proliferative effects and effects on intestinal microbial metabolism.

But there has not been a consistent epidemiological evidence. The conclusions of the meta-analyses and prospective cohort studies vary by gender, population, exposure and study design [14, 15]. Therefore, currently, green tea should not be recommended as a definite colorectal cancer prevention or adenoma recurrence prevention agent apart from general dietetic recommendations and research.

Green Tea and Liver Cancer

The most common background for the HCC is chronic liver damage, alcoholic liver disease and viral hepatitis or metabolic dysfunction. Since inflammation and oxidative stress are major contributors to hepatocarcinogenesis, green tea catechins have been considered as compounds of interest for prevention. Some populations have shown inverse relations between green tea consumption and liver cancer risk in the form of meta-analyses of observational studies [16, 17].

The anti-cancer associations observed in these preventive studies are difficult to interpret since no evidence has been shown to support the use of green tea for established HCC. The liver is also important to the safety of concentrated catechin supplements. High doses of green tea extracts have been linked to increases in aminotransferases and, in rare cases, serious damage to the liver. Therefore the organ that is of concern for supplement toxicity is the same organ where a preventive association was hypothesized.

Green Tea and Lung Cancer

In the models of lung cancer, EGCG has been shown to inhibit proliferation, induce apoptosis and modulate epidermal growth factor receptor-related signaling and angiogenic pathways. However, interpreting epidemiologic data is complicated because tobacco exposure is often a major risk factor for lung cancer and may also mask any diet effect. The inconsistent human findings are due to differences in smoking status, tea preparation, genetics, sex and population characteristics.

Green Tea and Oral Malignancy

Experimental studies are characterized by anti-proliferative, pro-apoptotic, anti-migratory and anti-invasive properties of EGCG. A recent systematic review of oral cancer studies found that there is an optimistic amount of molecular evidence, although it is still largely cellular and preclinical, and has only limited clinical validation. This field is a reminder that trials need to be carefully designed, with standard formulations and clinically relevant endpoints.

Green Tea and Hematological Malignancies

The green tea catechins also have been studied as treatment for leukemia, lymphoma, chronic lymphocytic leukemia and multiple myeloma. Apoptosis and growth inhibition of selected malignant hematopoietic cells have been shown in vitro. The studies of Polyphenon E in asymptomatic CLL (ACLL) showed biological activity in some patients, but were not designed to prove the efficacy of green tea as a cure [18,19].

Many lessons were learned in the study of multiple myeloma. Experimental studies have shown that EGCG itself has activity against myeloma cells, but Golden et al. [20] reported that green tea polyphenols may counteract the anticancer effects of the boronic acid proteasome inhibitor bortezomib in preclinical systems. This discovery reinforces the notion that it is important to consider using botanical supplements with care when taking anti-cancer medications, and to ensure they are reviewed by a health care professional.

Human Evidence for Cancer Prevention

Human prevention investigators are conducting prospective cohort and case-control studies as well as randomized trials. The last published broad systematic review and meta-analysis identified 43 eligible studies, comprised of randomized trials and cohort studies and showed pooled associations showing reduced overall cancer risk among the green tea/ECG group. The analysis also found more signals for some cancer sites and a dose-response relationship for some analyses [4]. The findings support the need for ongoing prevention research and don't prove a cause-and-effect relationship for all types of cancer.

The literature is still relatively varied, as it may refer to cups of green tea/day, length of time green tea has been consumed, concentration of catechins, green tea extract, or purified EGCG. Geographic variations of diet and tea preparation make it difficult to compare. Observational studies are still subject to healthy-user bias and residual confounding

due to smoking, alcohol consumption, physical activity, socioeconomic status and other dietary factors.

Clinical Evidence in Patients with Established Cancer

Only seven clinical trials with 371 patients were found in a systematic review of green tea catechins for cancer treatment. There was significant inter-study variation in doses, and in the duration of intervention ranging from days to months, and methodological diversity did not allow for quantitative meta-analysis. Several studies showed alteration in the biomarkers, or supportive-care outcomes, but the evidence was not enough for clear therapy.

A compound can have any or all of those effects, but it might also have no effect on the response to the tumor, PFS, OS, or cure rates. Currently, green tea and EGCG are regarded as investigational adjunct and chemopreventative drugs and not as proven agents for anticancer treatment. A summary of the steps required to go from evidence to application and the significant hurdles to cross the pathway is provided in Figure 2.

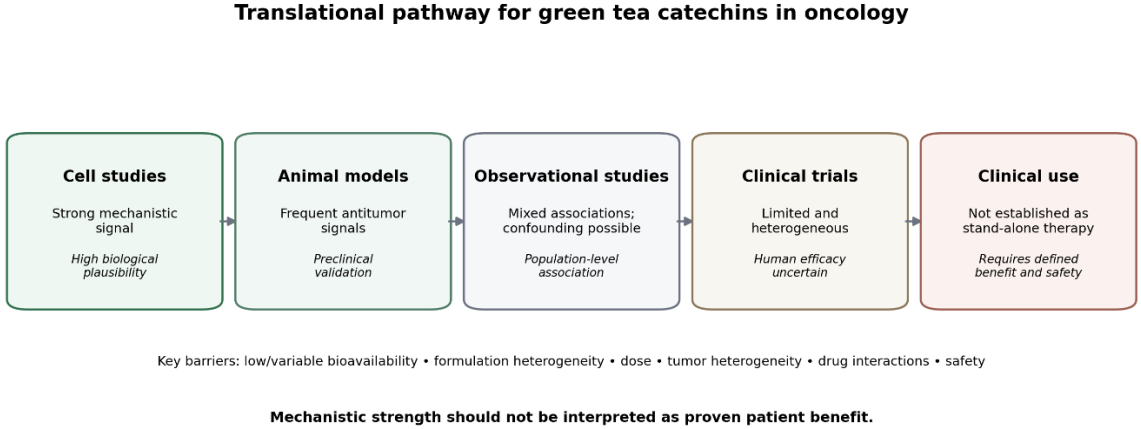


Figure 2. Translational pathway from mechanistic evidence to clinical oncology for green tea catechins. The figure emphasizes the decline in evidentiary certainty from preclinical signals to established clinical use and highlights key translational barriers [18].

Pharmacokinetics and Bioavailability

The bioavailability of EGCG in humans is one of the significant challenges in the development of EGCG as an anticancer agent. Human pharmacokinetic studies have shown that catechins are absorbed in the intestine, metabolized by microbially,-glucuronidated, sulfated, metabolized in the liver, distributed in various tissues and eliminated. Oral plasma

concentrations after oral administration are very variable between and within subjects and can be affected by dose, formulation, fasting and food effects [21, 22, 23, 24].

This pharmacokinetic limitation accounts for some of the differences between cell-culture experiments and clinical results. It is not an easy solution to simply increase the oral dose, as systemic exposure and toxicity might also increase. Thus, there is a need for formulations with enhanced delivery properties without causing unacceptable adverse effects in order to enable translational development.

Various delivery systems such as nanotechnology based delivery systems, liposomes, polymeric nanoparticles and other delivery systems have been suggested to increase the stability of EGCG, absorption, targeting of tumors and its intracellular delivery [25]. These methods are potentially effective, but are not widely used. Ultimately their value will need to be determined by pharmacokinetic, toxicological and clinical outcome studies.

Safety and Hepatotoxicity

This is because the safety profile of regular green tea differs from that of green tea concentrates. Overall, EFSA determined that catechins are generally safe for consumption as traditional green tea beverages, within regular consumption levels. However, the interventional evidence revealed that when supplemental doses of EGCG are administered at or above ~800 mg/day, there was a significant increase in serum transaminases in some individuals [26]. Uncertainty associated with universal supplemental dose definitions due to formulation, dosing conditions and individual susceptibility were also discussed.

Concentrated extracts should be monitored, as evidenced by clinical trials. A group of women who took a high-EGCG dose showed liver enzyme abnormalities in the Minnesota Green Tea Trial. Generally elevations were cleared after discontinuation, supporting a causal relationship in susceptible individuals [27]. Thus, it is not possible to extrapolate the safety of a cup of tea to high dose capsules or tea extracts.

Interactions with Anticancer Therapy

Oncology, in particular, is prone to potential herb-drug interactions due to the narrow therapeutic window of many anticancer medications and reliance on specific pathways for metabolism and/or activity. Theoretically, EGCG could interact with drugs through a mechanism affecting their transport, metabolism, oxidative status, or molecular targets. The best known example is the preclinical antagonism that was reported between

EGCG and bortezomib, where an anticancer agent is seen to negatively interact with another anticancer agent even if it has an anticancer effect alone [20].

Green tea extracts and other supplements should thus be reported to the oncology team for patients receiving chemotherapy, targeted therapy, immunotherapy, endocrine therapy or radiotherapy. For concentrated products, where the exposure to catechins is far more than in a normal diet, this recommendation is particularly relevant.

Major Limitations of the Evidence

There are some common methodologic weaknesses in the green tea-cancer research literature. First, there is a lack of standardization of exposure across studies. The dose of tea is not fixed and there are variations in the amount of catechins and caffeine present in commercial extracts. Second, there are numerous lab studies that employ concentrations that may not be attainable in human plasma or tumor tissue. Third, observational studies can be confounded and can suffer from reverse causation. Fourth, Malignant diseases are biologically heterogeneous, causing differences in clinically relevant subtypes to be masked when they are pooled together. Fifth, most human intervention studies are small and short, and are designed to measure surrogate markers, not recurrence or survival. Lastly, there could be publication bias for positive experimental results.

Future Therapeutic Perspectives

The development of EGCG should take a step further, from the general antioxidant or anticancer to precision pharmacology. Different tumors may be more sensitive to modulation by catechins. Genomic, transcriptomic, proteomic, metabolomic and epigenetic profiling of these may be useful in defining responsive biological subgroups. Another advantage of biomarker-driven trials would be the ability to assess whether or not the intended molecular target is actually altered in human tumor tissue.

Another significant area is drug delivery studies. Chemical stability and protecting the EGCG product from fast metabolism may be achieved by encapsulation or targeted nanocarriers which can also facilitate tumour accumulation and reduce non-specific exposure. Combination therapy should also be explored, but with careful consideration of mechanistic compatibility and formal interaction studies and not by simply assuming combinations of two anticancer agents would be synergistic.

The same should apply to prevention research. Future trials should establish dose and composition of tea products, baseline dietary intake, smoking and alcohol status, genetic factors, microbiome factors and adherence. Long-term studies should focus on the incidence of cancer and validated precancerous endpoints and should have a systematic monitoring of hepatic and other adverse effects.

Conclusion

Green tea and EGCG are significant molecules in the line of nutrition, natural-product pharmacology, and oncology. They have the potential to modulate several molecular pathways that are associated with carcinogenesis, making them intriguing for research in prevention and therapy. Mechanistic and preclinical evidence is strongest, but there is inconsistent epidemiological evidence and limited clinical treatment evidence to support clinical use.

An interpretation of this action that is clinically responsible should separate normal use of green tea from medication and should separate prevention of cancer from treatment of cancer. Green tea should not be used as an alternative to surgery, radiotherapy, chemotherapy, endocrine therapy, targeted therapy or immunotherapy. The EGCG in concentrated preparations needs special attention due to the fact that it has to be used in a specific manner as exposure to the body depends on the formulation and high doses of EGCG have been linked to increased levels of liver enzymes. Medical supervision in cancer patients is further warranted by the possible drug interactions.

Standardized formulations, adequate pharmacokinetic studies, targeted patient selection based on biomarkers, well-designed randomized clinical trials, clinically relevant endpoints, and systematic safety assessment are key factors for future progress. Assuming they can be fulfilled, green tea catechins could then eventually be developed into a well-defined part of any cancer prevention program and/or rational add-on to selected therapeutic treatments. Until there is such evidence, their role as scientificly exciting compounds being investigated as treatments for malignant disease rather than as stand-alone treatments remains valid.

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