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Cytotoxic effects of tea (*Camellia sinensis*), stevia (*Stevia rebaudiana*), moringa (*Moringa oleifera*) leaves, and their potential as functional beverages for cancer patients

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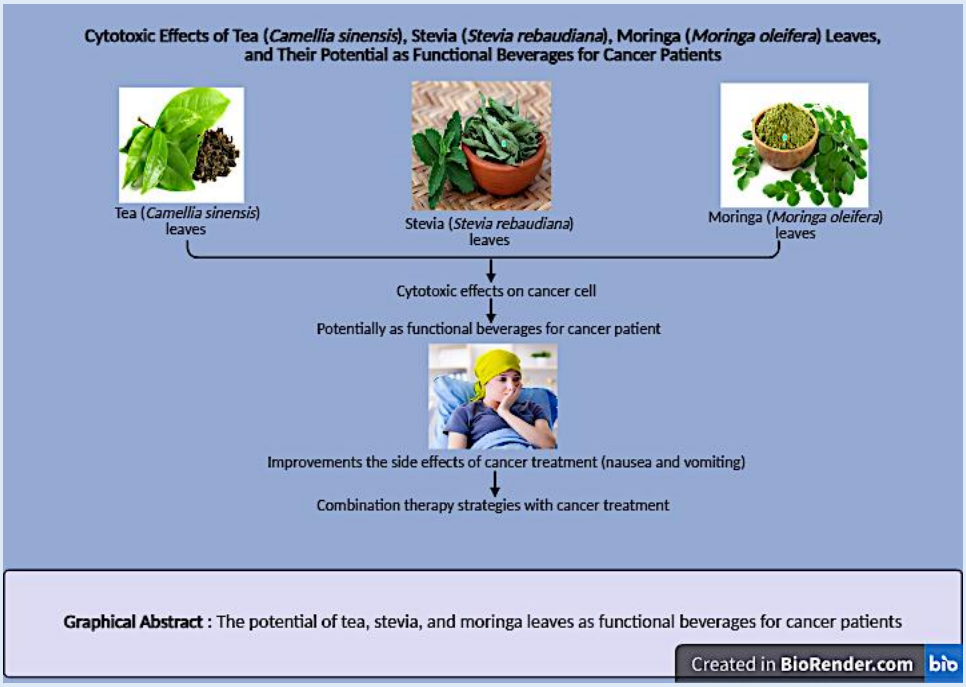
ABSTRACT

The global incidence of cancer has been rising annually, affecting various types and increasing the number of patients. Research on cancer, including its pathology, treatment, and management of side effects, continues to evolve, yet no truly satisfactory outcomes have been achieved. Pharmacological cancer therapy should be complemented with non-pharmacological approaches to improve the quality of life of cancer patients. Chemotherapy-induced nausea and vomiting (CINV) still troubles patients after all these years, making functional beverages a potential target to overcome this problem. Several natural ingredients, including tea, moringa, and stevia leaves, have been empirically used as functional beverages due to their antioxidant level. Therefore, it's necessary to determine whether these plants exhibit cytotoxic effects on cancer cells and their interaction with anticancer drugs. This review aimed to compile previous research on the potential of tea, moringa, and stevia leaves as functional beverages for cancer patients. Their cytotoxic effect has been demonstrated in several *in vitro* studies, with no reported metabolic herbal-drug interactions.

Novelty: This review highlights the underexplored potential of tea, stevia, and moringa as functional beverage components with cytotoxic properties relevant to cancer patient care.

Keywords: cancer, functional food, tea, stevia, moringa

Graphical abstract: Cytotoxic Effects of Tea (Camellia sinensis), Stevia (Stevia rebaudiana), Moringa (Moringa oleifera) Leaves, and Their Potential as Functional Beverages for Cancer Patients



INTRODUCTION

As human civilization evolves, disease and its treatments remain an inseparable aspect of life. One of the most significant "bio burdens" in the world today is cancer, with its prevalence increasing annually in both types and number of cases. According to GLOBOCAN 2022, there were 19,964,811 new cases and 9,736,779 deaths reported [1]. The cancer mortality rate is about 50% in the world, necessitating further development of effective cancer treatment strategies to reduce these mortality rates.

The National Cancer Institute defines cancer as a disease characterized by uncontrolled cell growth that can spread to other parts of the body [2]. Therefore, the primary strategy in cancer treatment is to halt this uncontrolled growth and prevent metastasis. Cancer pharmacotherapy consists of pharmacological and non-pharmacological therapies. Pharmacological therapies aim to treat the disease through chemotherapy, radiation, or targeted therapy. Non-pharmacological therapies focus on maintaining the patient's quality of life by managing the adverse effects of pharmacological treatments. Chemotherapy-induced nausea and vomiting (CINV) is one of the significant side effects associated with some treatments of cancer that still troubles patients after all these years [3]. Cancer patients are at high risk of developing malnutrition, especially in advanced stages of the disease [4]. Cancer-related malnutrition is commonly associated with clinical worsening, including reduced quality of life, poor physical function, and higher mortality [5]. Hence, functional foods and beverages could be a potential target for overcoming this problem.

The critical role and beneficial effect of functional foods and beverages on health beyond their fundamental nutritional value. They enhance wellness and lower the risk of disease [6]. The Functional Food Center (FFC) defines functional foods (FFs) as natural or processed

foods that contain biologically active compounds, which, in specified, adequate, non-toxic amounts, provide a clinically proven and documented health benefit utilizing specific biomarkers, to promote optimal health and reduce the risk of chronic/viral disease and manage their symptoms [7]. Epidemiological studies have data that many food constituents or natural products can prevent cancer. In 2020, the American Cancer Society (ACS) published guidelines on diet and physical activity for cancer prevention. Overall recommendations are: 1) achieve and maintain a healthy body weight, 2) be physically active, 3) healthy eating pattern (vegetables, fruits, whole grains, legumes), 4) limit or avoid intake of red and processed meats, sugar-sweetened beverages, highly processed foods, and refined grain products [8].

Tea leaves (*Camellia sinensis*) are natural ingredients with high antioxidant content, which are known for their polyphenol compounds. EGCG plays regulatory roles in the tumor microenvironment (TME) and metabolic reprogramming, which provides combined therapeutic strategies for anticancer treatment [9]. Besides tea, moringa leaves (*Moringa oleifera*) are believed to possess potent antioxidant activity, making them a viable option for a functional beverage. However, it must be both palatable and enjoyable. Formulation with specific combinations of additional ingredients is needed. The stevia leaf (*Stevia rebaudiana*) is a natural sweetener that serves as a sugar substitute, making it a safe and daily consumable alternative [10].

Given the potential of tea, moringa, and stevia leaves, further literature review is necessary to determine whether these plants exhibit cytotoxic effects and if there is any interaction with chemotherapy agents. This review aimed to compile prior research on the potential of tea, moringa, and stevia leaves as complementary beverages for cancer patients.

METHODS

This literature review was conducted using articles published in the PubMed database and in the Functional Foods in Health and Disease Journal, with the keywords “functional foods”, "stevia and cancer", "tea and cancer", and "moringa and cancer". The literature type was clinical trials, cohort studies, *in vivo* studies, and *in vitro* studies published within the last 10 years (2014-2024). Thirty-five articles met the research objective (Figure 1).

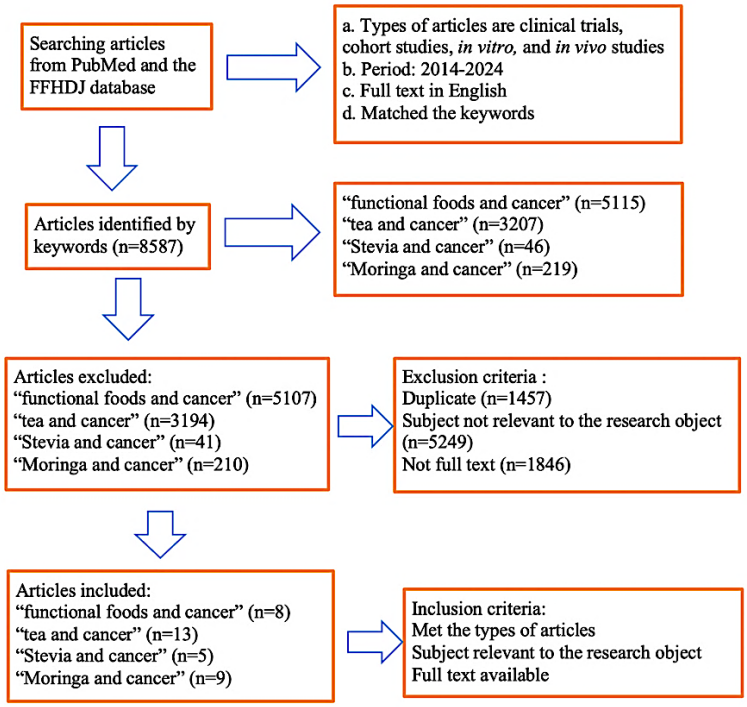


Figure 1. Flow chart literature review

Functional Foods Classification: Functional foods are classified into three categories: 1) conventional foods (whole foods without modification, naturally rich in bioactive compounds beneficial for health, e.g., vegetables, fruits, dairy, grains), 2) modified foods (foods enriched or fortified with specific nutrients, e.g., calcium-fortified drinks, antioxidant- and vitamin-enriched bread, fiber-enriched products, sterols, and omega-3 fortified foods), 3) synthetic food ingredients (functional food components synthesized in laboratories, e.g., inulin, oligofructose). Based on their sources, functional foods can be categorized into plant-based and animal-based sources (Figure 2).

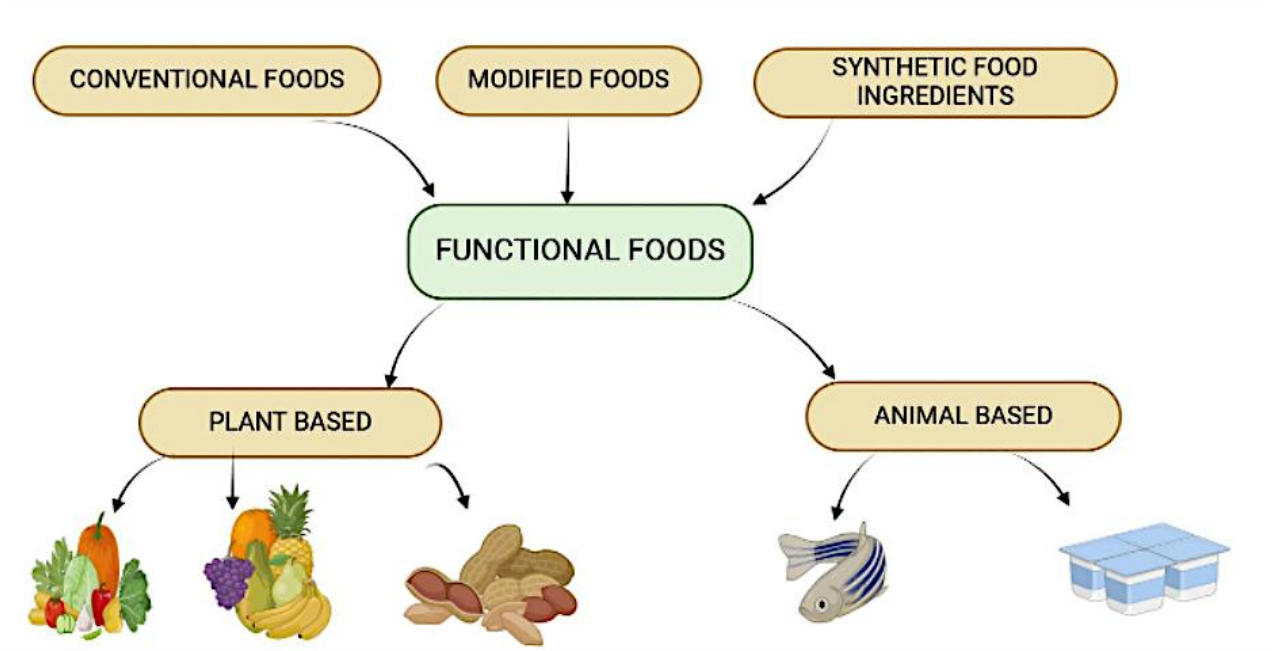


Figure 2. Classification of functional foods

The Functional Foods Center has developed a 17-step process for defining functional foods. The proposed classification system (A, B, or C) integrates post-market research, epidemiological studies, and is informed by clinical trials. The 17 individual steps are as follows [7]:

a) Establish a goal of the functional food product, b) determines relevant bioactive compound(s), c) establishes the appropriate dosage of bioactive compound(s), d) establishes the appropriate time of consumption of bioactive compound(s), e) determines the specific pathway and mechanism of action, f) establishes relevant biomarker(s), g) Chooses an appropriate food vehicle for bioactive compound(s), h) provides preclinical studies on efficacy and safety, i) provides clinical trials for dosage, efficacy, and safety, j) creates a special label that informs the consumers of the most effective way to consume product, k) publications are necessary in open-access, peer-reviewed journals, l) educates the market, m) sends information to credible third parties and/or governmental agencies for approval, n) official establishment of the accredited functional food product, o) release the functional food product to the market (receive the basic category level C), p) provides epidemiological studies (reapply for the approval for a new category level B), q) provides post-market research (reapply for the approval for a new category level A).

Established in 1998, the FFC could serve as a certification agency for FFs. Once applications for FFs certification are submitted, the FFC would be responsible for evaluating and ensuring these products meet the standards and requirements related to health benefits and safety.

Functional foods have a fascinating history that traces back to the 1950s. The concept emerged when

researchers began investigating connections between diet and degenerative diseases. One prominent figure was Ancel Keys, who published groundbreaking studies about heart disease associated with cholesterol and fatty foods. His work was a catalyst in understanding how diet influences health. The term "functional foods" gained popularity in the 1980s, initially in Japan. The Japanese government introduced a concept called "FOSHU" (Foods for Specified Health Use), labelling certain foods with scientifically proven health benefits. This paved the way for a global interest in functional foods, from probiotics to omega-3-enriched products. Since then, the field has expanded exponentially with advancements in nutrition science (see Figure 3), shifting from functional agriculture to functional plants, and finally to functional food [11].

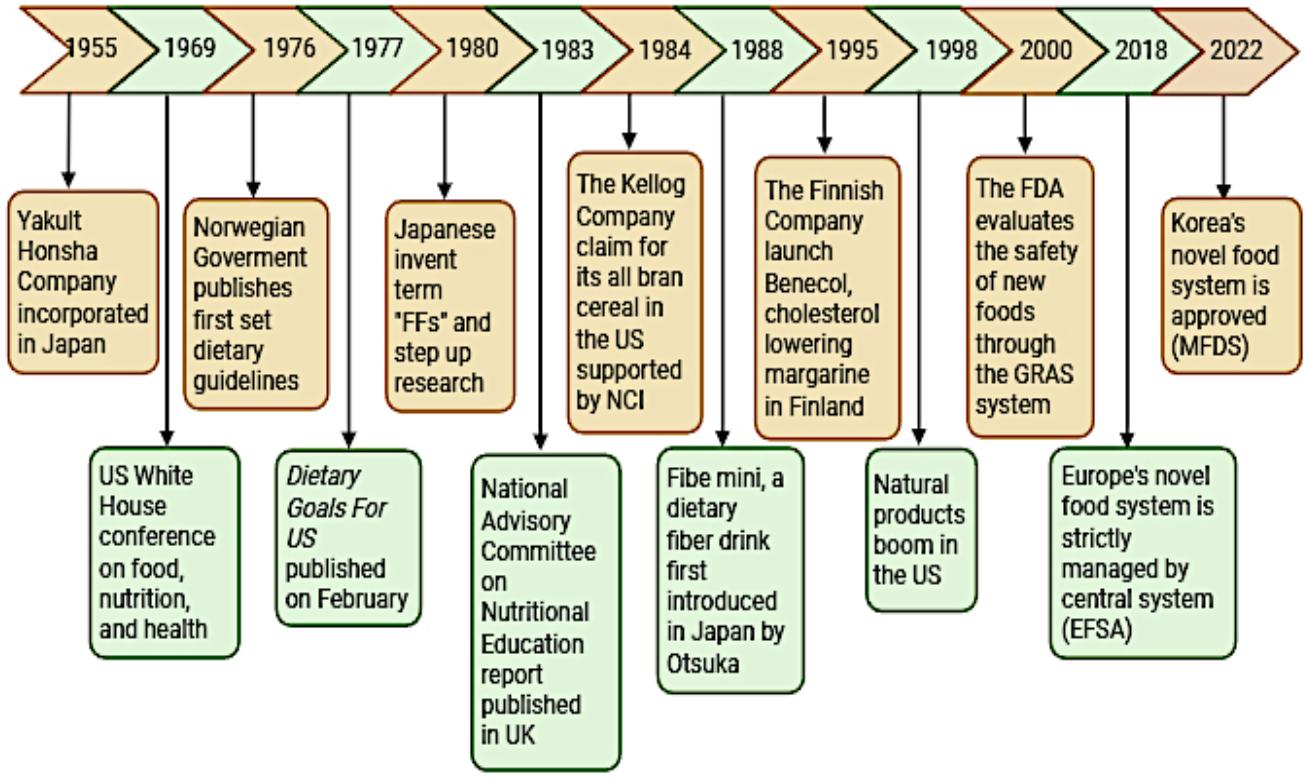


Figure 3. Key milestone of functional foods

Tea and Cancer: Tea has been widely used for centuries not just as a beverage, but also as a remedy for various ailments. Tea leaves come from the *Camellia sinensis* plant. The processing methods affect the flavor, color, and polyphenol content, contributing to their health benefits [12]. Polyphenols in green tea are monomers, epigallocatechin-3-gallate (EGCG), epigallocatechin (EGC), epicatechin (EC), epicatechin-3-gallate (ECG) [13]. EGCGs are known for their strong antioxidant properties and act as free radical scavengers [14]. It simultaneously reduces oxidative stress biomarkers by enhancing OGG1-mediated DNA repair and inducing Nrf2-dependent antioxidant genes [15]. It also interacts with endogenous antioxidants, such as glutathione, and generates reactive oxygen species (ROS) to activate the pro-oxidative pathway, leading to the selective induction of cancer cell death. This dual antioxidant and pro-oxidant functionality highlighted the potential therapeutic value of EGCG in cancer prevention and treatment [16]. Along with the oxidation process in black tea, these monomers undergo polymerization [17] into tannin, theaflavin, and thearubigins, offering benefits such as supporting heart

health and improving gut microbiota [18]. Oolong tea, in the middle of the oxidation spectrum, has a unique balance of green and black tea properties. Some studies on the effect of tea consumption are described in Table 1.

Table 1. Clinical and Cohort studies of cancer patients associated with tea consumption

Type of Cancer	Parameter	Methods	Results	Mechanism	Citation
Pancreatic Cancer	CA 19-9	Two hundred seventy healthy individuals (220 men, 50 women) aged 25-65 years consumed 375 ml/day of black tea (equivalent to 6 cups of medium size) and then had their blood serum taken and measured for the concentration of CA 19-9 using ELISA at a wavelength of 450 nm. The normal value of CA 19-9 is ≤ 40 U/ml.	The CA 19-9 value increased in 43.3% of participants with a value range of 69-105 U/ml.	Black tea stimulates the production of CA 19-9 from the epithelial tissue. Black tea at low concentrations protects the wipes. GI epithelium, while at high concentrations induces apoptosis and cytolysis in the small intestine epithelium related to the activation of caspase 8 through the Fatty acid synthase-associated protein with death domain (FADD) pathway.	[19]
Prostate Cancer	PSA (Prostate Serum Antigen)	A phase II clinical trial RCT was carried out on 113 men who were diagnosed with prostate cancer and had undergone RP (Radical Prostatectomy), some drank green tea, black tea, and water (control), and then PSA was measured by HPLC and ELISA. The parameters tested are proliferation, apoptosis, inflammation, and oxidation.	There is no significant difference in the proliferation, apoptosis, and oxidation of the RP tissue in 93% of patients. However, there was a significant decrease in inflammatory parameters (NF κβ) and PSA in men who consumed green tea.	It is assumed that the polyphenols in tea inhibit cell proliferation and induce apoptosis through various mechanisms, including antioxidant and anti-inflammatory activities.	[18]
Breast Cancer	Estrogen Receptor (ER) Value	Conducted interviews with women in Hong Kong aged 20-84 years old (756 people with a BC diagnosis and 789 people who are not yet and have gone through menopause and regularly drink tea every day). The data were then statistically analyzed using logistic regression. The effect of tea consumption on the ER value and menopausal status of patients was also observed.	58.1% of new patients and 55% of outpatients who regularly drink tea showed OR values of 1.01 (95% CI: 0.78-1.31) and 1.2 (95% CI: 0.8-1.78), respectively. Regular tea drinking significantly reduced the risk of breast cancer in pre-menopausal women (OR 0.62, 95%CI 0.4-0.97) but increased the risk in menopausal women (OR 1.4, 95%CI 1.0-1.96) and ER-negative (OR 2.99, 95%CI 1.26-7.11)	Green tea inhibits the proliferation of cancer cells by inhibiting Vascular Endothelial Growth Factor (VEGF)	[20]
Melanocarcinoma	BCC (*Basal Cell Carcinoma*) and SCC	A population study was conducted on residents of Nambour, Australia, who were categorized into four	Tea drinkers who consumed≥ 4 cups compared to those who never drank tea for BCC obtained an RR (Relative Risk) value of 1.03, 95% CI	The polyphenols in black tea, Theaflavin and Thearubigin, can reduce skin inflammation due to	[21]

Type of Cancer	Parameter	Methods	Results	Mechanism	Citation
	(*Squamous Cell Carcinoma*)	types of black tea drinking habits (never, ever 1 cup/day, 1-3 cups/day, ≥4 cups/day), then data on residents affected by BCC and SCC were collected from 1997 to 2007. There were 323 participants out of 740 people affected by BCC and SCC.	0.7-1.53; p-trend 0.74, and for SCC, a RR value of 1.25, 95% CI 0.71-2.19; p-trend 0.29. Shows no significant difference between tea-drinking habits and BCC or SCC	UV rays, prevent DNA damage and immune suppression, and induce apoptosis and DNA repair.	
Myelodysplastic Syndrome (MDS)	Odds Ratio (OR) Logistic Regression statistical analysis	The study was conducted in China from 2012 to 2013. Patients aged 18-85 years with 208 MDS cases and 208 non-MDS patients were associated with tea drinking habits, which were divided into three categories (tea drinkers ≥20 years, ≥2 cups/day, tea drinkers ≥750g/year)	The OR and 95% CI values in these three categories are: 0,39 (0,2-0,74); 0,45 (0,25-0,79); 0,40 (0,21-0,77). Studies suggest that regular tea consumption may reduce the risk of MDS in the population of Zhejiang, China.	NA	[22]
Liver Cancer	Hazard ratios (HR) and 95% CI from Cox regression models.	Prospective cohort study in Chinese women aged 40-70 years old from an urban district in Shanghai between 1996 and 2000. 71.841 participants were included in the analysis, and 253 incident liver cancer cases were identified.	Compared to never tea drinkers, the risk of liver cancer for participants who have consumed over 30 kg of dried tea leaves cumulatively was 0,56 (95% CI: 0,32-0,97). For those who drank green tea only, HR was 0,54 (95% CI: 0,3-0,98)	NA	[23]
Ovarian Cancer	Odds Ratio (OR): Logistic regression statistical analysis	The study was conducted in Canada from 2001 to 2012. Patients aged 40-79 years with 524 ovarian cancer cases and 1587 non-ovarian cancer patients were associated with the habit of drinking tea, coffee, and caffeinated beverages.	The OR and 95% CI values in black tea drinkers are 1.56 (1.07-2.28) and the association of drinking black tea with endometrioid histotype OR value 3.19 (1.32-7.69); Drinking black tea regularly shows an increased risk of ovarian cancer in the endometriosis histotype, but not in serous and clear cells.	NA	[24]

Bioactive compounds in tea prevent carcinogenesis through anti-pathogen, anti-inflammatory, and cell survival pathway mechanisms [25]. Bacteria and viruses are included in the pathogenic substances that cause carcinogenesis. For example, *Escherichia coli* (E. coli) encoding colibactin (a genotoxin polyketide peptide) can damage DNA directly and induce carcinogenesis. *Helicobacter pylori* (*H.pylori*) and Enterotoxigenic *Bacteroides fragilis* (ETBF) are associated with gastric and colon cancer because the bacteria produce spermine oxidase, which causes DNA damage. Pathogenic bacteria that cause inflammation can initiate the incidence of

cancer. EGCGs have demonstrated cytoprotective effects against *H. pylori* by interfering with Toll-like receptor 4 (TLR-4) signaling [26]. It also inhibits the growth of the hepatitis B virus (HBV) by interfering with HBV DNA replication. EGCGs reduce the virus's ability to multiply within liver cells. Additionally, EGCGs prevent HBV entry into hepatocytes by modulating the endocytosis and degradation receptor of HBV infection, sodium taurocholate co-transporter polypeptide (NTCP) [27]. Its action on autophagy mediates lysosome acidification in hepatocytes, disrupting the formation of autophagosomes required for HBV replication [28]. Green tea has therapeutic potential in the prevention and management of HBV viral infection.

The anti-inflammatory mechanism of EGCG is a key regulation in its chemopreventive properties [29]. Inflammation is often closely linked with cancer progression, as inflammatory cells can create an environment that supports tumor growth, invasion, and metastasis. EGCGs work by suppressing the expression of pro-inflammatory cytokines, such as Interleukin (IL)-6, IL-8, and IL-12, which are signaling molecules involved in inflammation [30]. It reduces chemokines, CXC motif chemokine ligand 1 (CXCL1), and the enzyme cyclooxygenase-2 (COX-2), which play essential roles in promoting inflammation. EGCGs inhibit COX-2 without affecting COX-1 expression in human prostate cancer cells. COX-1 is a housekeeping enzyme that is expressed and controls homeostasis in many tissues. COX-2 is an inducible isoform readily upregulated by inflammatory stimuli [31].

The anticancer activity of EGCG involves the regulation of several key cell survival pathways, such as antiproliferative, pro-apoptotic, anti-angiogenic, and anti-invasive functions [32]. EGCGs induce apoptosis in cancer cells through 3 mechanisms: 1) general apoptosis pathway, 2) decreased telomerase activity, and 3) harmful modulation of mitochondrial function. EGCGs influence pathways such as Bax/Bcl-2 (pro-apoptotic vs anti-apoptotic protein) and activate caspase enzymes (caspase-3, -8, and 9), key players in apoptosis. In prostate cancer cells, LNCaP, it increases the Bax/Bcl-2 ratio and caspase activity. In breast (MDA-MB-231), lung (H1299), and head/neck (A549) cancer cells, EGCGs decrease Wnt/β-catenin and Akt activity, enhancing apoptosis [33]. Signal transducer and activators of transcription (STAT) signaling, which often supports cancer survival, is also disrupted, promoting cancer cell death in prostate and breast cancers.

Telomerase elongates telomeres, extending cell life and promoting cell division. EGCGs inhibit this enzyme, shortening the telomeres and limiting the number of divisions of tumor cells [34]. EGCGs disrupt mitochondrial respiratory chains (complexes I, II, and ATP synthase) to reduce cells' energy production. It also induces cell death through the autophagy process and endoplasmic reticulum stress. Theaflavin and thearubigins, polyphenols found in black tea, also demonstrate anticancer activity by halting the cell cycle at critical phases G0/G1 or G1/S, effectively preventing cancer cells, such as leukemia cells (U937 and K562). These compounds regulate key signaling pathways (PI3K/Akt and Wnt/β-catenin) and interact with related molecules (PTEN, p53, p21, CDK-Cyclin D1) to control the cell cycle. Additionally, tea compounds inhibit the p38/JNK/STAT pathway, which in turn reduces AP-1 activity —a factor associated with cancer cell proliferation and survival [29].

Angiogenesis, the formation of new blood vessels, is a critical process in tumor growth and metastasis. Tumors rely on angiogenesis to obtain nutrients and oxygen for rapid growth. The release of angiogenic factors such as epidermal growth factor (EGF), vascular endothelial growth factor (VEGF), matrix metalloproteinase (MMP), intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1) promotes this process, leading to

uncontrolled vascular growth. EGCGs and theaflavins play a significant role in inhibiting tumor angiogenesis [35]. EGCG in cervical and liver cancer can inhibit hypoxia-inducible factor-1α (HIF-1α), a key player in responding to low oxygen levels in tumors. This results in decreased expression of VEGF and MMP, suppressing tumor angiogenesis [36].

Although chemotherapy agents are effective in treating cancer, they often have unfavorable side effects. It has been demonstrated that natural products can contribute to the prevention and suppression of cancer by modulating various biological processes, which may help mitigate the side effects of treatment [37]. In addition, a meta-analysis involving 386,610 participants showed that moderate tea consumption (1.5-2.0 cups/day) was associated with lower all-cause, cardiovascular disease (CVD), and cancer mortality compared to no tea consumption [38]. Since EGCG has shown substantial advantages in cancer prevention and treatment, its efficacy still has a particular gap compared with first-line chemotherapy drugs, and the possibility of developing it as a separate anticancer drug is limited. However, when EGCG is used as an adjuvant chemotherapeutic agent, it exhibits strong chemosensitizing effects and mitigates the side effects of chemotherapeutic agents [39]. For instance, Flutamide-EGCG can be used for the immunotherapy and Androgen Deprivation Therapy (ADT) of prostate cancer. EGCG-modified cancer cells and probiotics-derived nanoparticles are used as vehicles for targeted delivery of Flutamide to the tumor site [40]. Furthermore, doxorubicin, a chemotherapeutic on cervical cancer, has been used with EGCG in studies as an autophagy regulator that boosted photothermal therapy's therapeutic effectiveness [41]. Anticancer mechanisms of EGCGs are described in Figure 4.

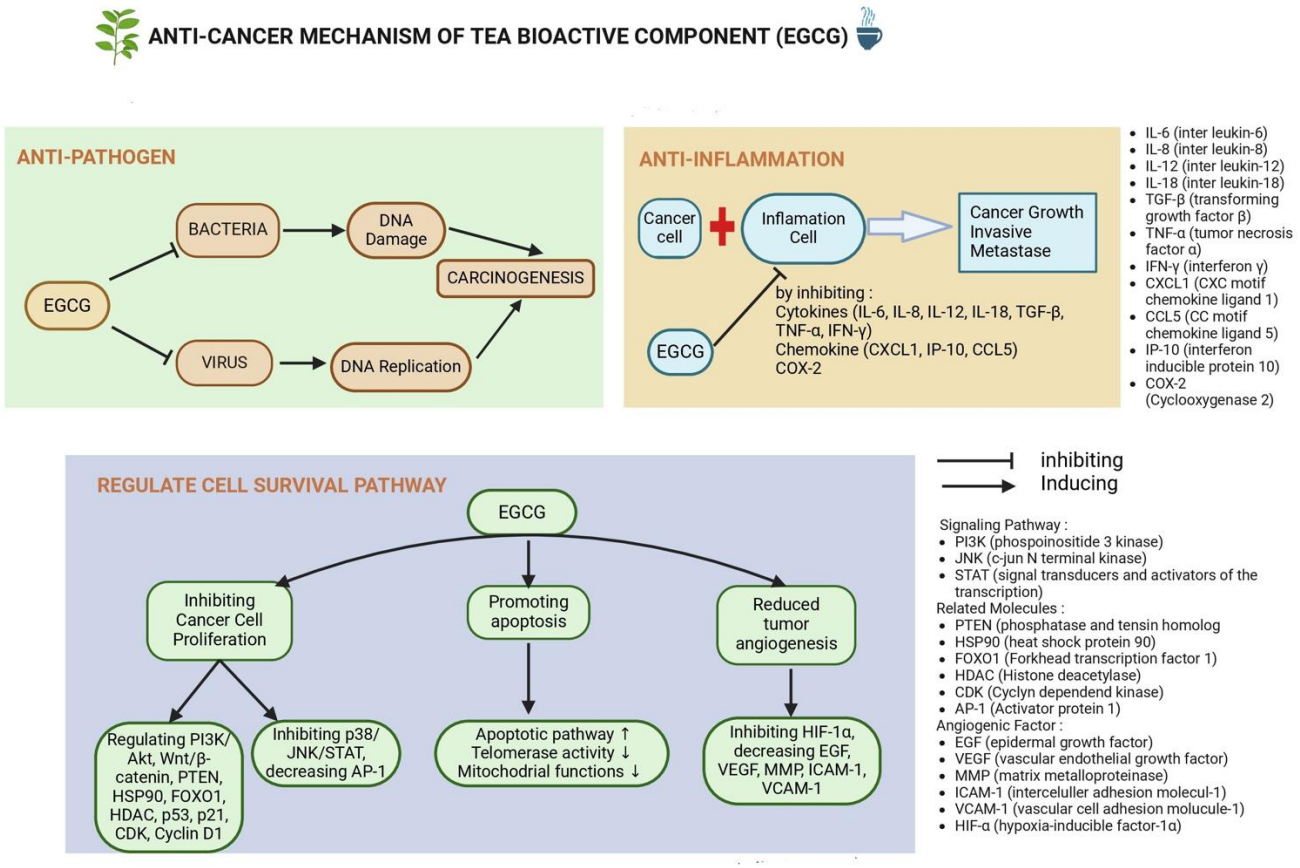


Figure 4. Anticancer mechanisms of EGCG.

Stevia and Cancer: Stevia is derived from *Stevia rebaudiana*, a perennial herb widely utilized in the food and pharmaceutical industries as a natural sweetener and potential health booster [42]. Stevia leaf extract contains numerous bioactive ingredients, including phenols, flavonoids, sterols, terpenes, tannins, vitamins, and minerals, as well as steviol glycosides, such as stevioside, rebaudioside A, and rebaudioside C, which exhibit antioxidant, anti-inflammatory, and anticancer properties. The extracts were reported to be non-cytotoxic against healthy cell lines at concentrations up to 5 µg/mL but showed cytotoxicity against HeLa cancer cells with an IC_{50} of 50 µg/mL [43]. These compounds are metabolized in the colon by gut microflora, which breaks down β-glycoside bonds to release free steviol. These are then partially absorbed, processed in the liver, and excreted via the kidneys [44]. An in vitro study noted that the IC_{50} value of steviol was higher than that of chemotherapy drugs (5-fluorouracil and doxorubicin). With further research, steviol could be developed into a chemotherapy agent in larger doses [44]. Some *in vitro* studies of stevia are described in Table 2.

Table 2. *In vitro* studies of stevia

Type of cell lines	Methods	Results	Mechanism	Citation
U2OS Osteosarcoma	Cytotoxicity Assay	The IC_{50} value of steviol 200µg/ml is almost the same as the IC_{50} value of 5-FU (positive control) 250µg/ml	Steviol inhibits U2OS cells through the G1 phase of the cell cycle, characterized by an increased Bax/Bcl-2 ratio, activation of CDKI1, p53, survivin, and Caspase-3.	[45]
MDA-MB-231 and SKBR3 Breast cancer cell lines	Cytotoxicity Assay	The IC_{50} values of stevioside against MDA-MB-231 cells were 55µg/ml and 66µg/ml for SKBR3	Stevioside inhibits the growth of MDA-MB-231 and SKBR3 cells through the mitochondrial apoptotic pathway, characterized by an increase in the Bax/Bcl-2 ratio, as well as activation of caspase-3 and caspase-9.	[46]
MCF-7 Breast cancer cell lines	Cytotoxicity Assay	The IC_{50} value of steviol was 185µg/ml	Steviol inhibits the growth of MCF-7 cells in the resting phase of G2/M and sub-G0/G1 at specific doses.	[47]
Gastrointestinal cancer cell line	Cytotoxicity Assay	The IC_{50} value of steviol 100µg/ml is almost the same as the IC_{50} value of 5-FU (positive control) 250µg/ml	Steviol inhibits cancer cells through the mitochondrial apoptotic pathway with indications of increased Bax/Bcl-2 ratio, activation of p21, p53, caspase 8, caspase 9, and caspase 3. The expression of miRNAs in the emerging steviol was miR-203a-3p and miR-6088 in HCT-116, miR-1268b and miR23c in MKN-45 cells	[48]

The mechanism of stevia's bioactive compounds in cancer has been widely publicized. Stevia is emerging as much more than a natural sweetener. Its direct effects, such as cytotoxicity, target cancer cells, impeding their growth and proliferation. Meanwhile, its indirect effects, which include protecting cells from oxidative damage through antioxidants and aiding in lipid regulation, create an internal environment less favorable for cancer progression. Polyphenols and flavonoids in stevia play a crucial role in this function [43]. These properties highlight its potential as a preventive measure and a complementary therapy alongside conventional cancer treatment.

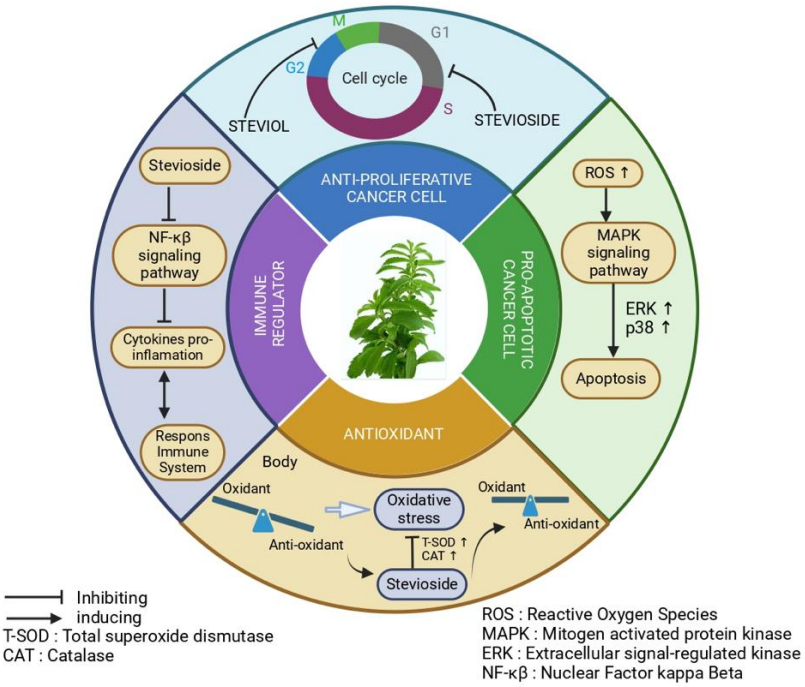


Figure 5. Biological effects of *Stevia rebaudiana*

Moringa and Cancer: *Moringa oleifera* (MO) has gained attention for its high nutritional value and medicinal properties [49]. All parts of the plant have potential bioactive compounds for cancer treatment [50]. MO contains secondary metabolites such as glucomoringin isothiocyanate (GMC-ITC), quercetin-3-glucoside, isorhamnetin-hexose, and kaempferol-3-glucoside, which have the highest anticancer activity by inducing apoptosis primarily through intrinsic apoptotic pathways [51]. It inhibits tumor cell growth and induces apoptosis in MCF-7 breast cancer cell lines and Caco-2 colorectal cancer cell lines [52]. Extracts from the leaves and bark of this plant significantly reduce cancer survival and motility. Additionally, its seed oil has been shown to exhibit antiproliferative effects on oral cancer cell lines. The other in vitro studies of moringa pod extracts showed that at concentrations of 62.5-250 µg/ml, they significantly reduced inflammation in LPS-stimulated SaOS-2 osteoblast cells, leading to decreased levels of inflammatory cytokines (IL-6 and IL-8) and ROS, enhanced cell viability, and increased calcium production. These findings suggest its potential to promote bone health [53]. Moringa's unique combination of antioxidants, vitamins, minerals, and amino acids makes it a powerhouse for overall health [54]. Some *in vitro* studies of moringa are described in Table 3.

Table 3. In vitro studies of moringa

Type of cancer cell lines	Methods	Results	Mechanism	Citation
Esophagus cancer SNO	Cytotoxicity assay	The IC_{50} value was 389,2 µg/ml	*M. oleifera* extract has antiproliferative effects on SNO cells by increasing lipid peroxidation, DNA fragmentation, and induction of apoptosis.	[55]

Type of cancer cell lines	Methods	Results	Mechanism	Citation
Hepar cancer HepG2	Chaperone-mediated Autophagy (CMA)	Elevated gene expression of p53 and Caspase 3	*M. oleifera* extract has a high antioxidant effect, which triggers oxidative stress in cells. Oxidative stress triggers various cellular responses, including the activation of mTORC2, which in turn activates Akt1. Akt1 inhibits the CMA pathway. As a result, an accumulation of toxic proteins becomes excessive, leading to apoptosis.	[56]
Lung cancer 549	Cytotoxicity assay	The IC_{50} value was 50 µg/ml	Apoptosis induction was indicated by increased ROS generation and increased activities of caspases 3 and 9.	[57]
Breast cancer MCF-7	Cytotoxicity assay	The IC_{50} value was 100 µg/ml	NA	[58]

Although MO is not yet considered a commercial chemotherapy drug, previous studies have indicated that it could become a chemotherapeutic agent [50]. Moringa has remarkable potential in cancer-related research (see Figure 6). Its bioactive compounds contribute to its ability to inhibit carcinogen activation, promote carcinogen detoxification, and reduce inflammation. Furthermore, its antiproliferative properties hinder the growth of tumor cells, while its capacity to induce apoptosis helps eliminate cancerous cells [59].

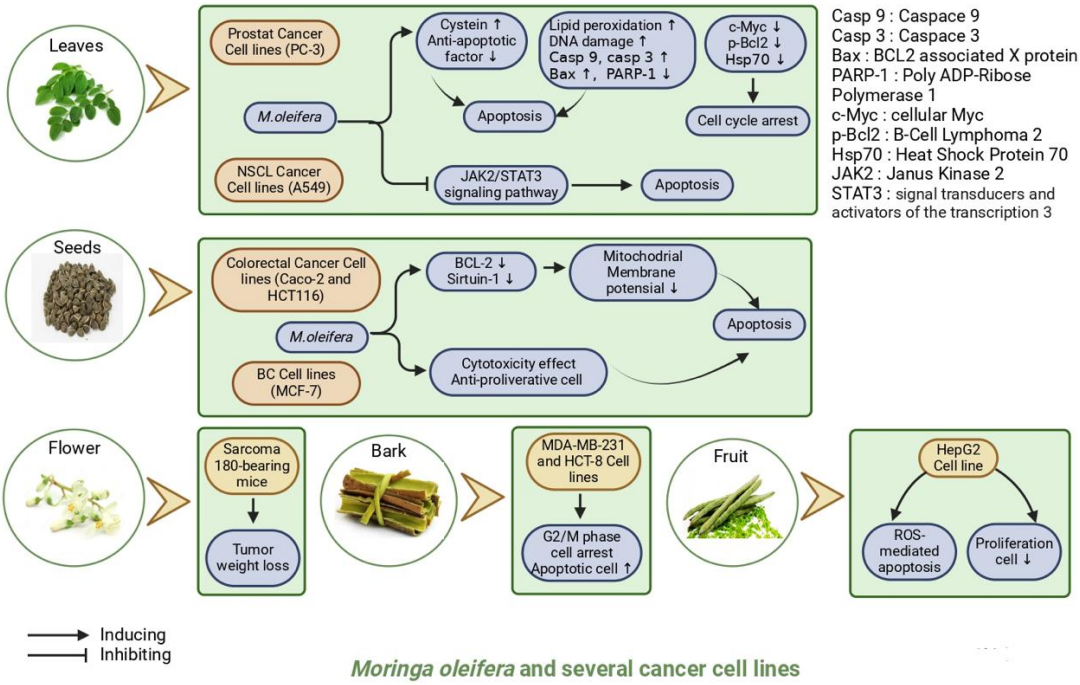


Figure 6. Biological effects of *Moringa oleifera*

CONCLUSION

Chemotherapy drugs often cause side effects such as nausea and vomiting, lowering cancer patients' quality of life. Hence, in addition to pharmacological therapies, non-pharmacological interventions such as dietary support are necessary to enhance immune function. Functional foods and beverages, especially those rich in antioxidants, play a crucial role as complementary options for cancer patients. The underexplored potential of tea, stevia, and moringa as functional beverage components with cytotoxic properties relevant to cancer patient care. Gougis et al. compiled clinical and preclinical data on pharmacokinetic interactions between herbs, food supplements, and anticancer drugs [60]. The study highlighted that stevia acts as an inhibitor and inducer of CYP3A4, with weak induction of CYP1A2. Moringa weakly inhibits CYP3A4, while green tea moderately inhibits CYP3A4. Since certain chemotherapy drugs rely on CYP450 enzymes for metabolism, understanding these interactions is crucial for their safe use in conjunction with functional beverages. It is described in Table 4.

Table 4. Herbal-drugs interaction with the enzyme CYP450

Name	CYP3A4	CYP2D6	CYP1A2	CYP2C8
Tea (*Camellia sinensis*)	Moderate inhibitor	No interaction	No interaction	Weak inhibitor
Stevia (*Stevia rebaudiana*)	Inhibitor & inducer	No interaction	Weak inducer	Not available
Moringa (*Moringa oleifera*)	Weak inhibitor	No interaction	Not available	Not available
Anastrozole	No influence	No influence	No influence	No influence
Capecitabine	No influence	No influence	No influence	No influence
Cisplatin	No influence	No influence	No influence	No influence
Epirubicin	No influence	No influence	No influence	No influence
5-Fluorouracil	No influence	No influence	No influence	No influence
Daunorubicin	No influence	No influence	No influence	No influence
Methotrexate	No influence	No influence	No influence	No influence
Melphalan	No influence	No influence	No influence	No influence

To optimize both benefits and taste, further formulation studies are needed. Since commercially available functional food products for cancer patients remain limited, additional research and development in this field are essential.

Abbreviations: Cytochrome 450 (CYP450); cytochrome 3A4 (CYP3A4); cytochrome 2D6 (CYP2A6); cytochrome 1A2 (CYP1A2); cytochrome 2C8 (CYP2C8); Inhibitory concentration 50 (IC_{50}); deoxynucleotide acid (DNA); Odds ratio (OR); confidence interval (CI); ultraviolet (UV); Cyclin-dependent kinase (CDK); Cyclin-dependent kinase inhibitor 1 (CDKI 1); epidermal growth factor (EGF); vascular endothelial growth factor (VEGF); matrix metalloproteinase (MMP); intercellular adhesion molecule-1 (ICAM-1); vascular cell adhesion molecule-1 (VCAM-1); hypoxia-inducible factor-1α (HIF-1α); epigallocatechin-3-gallate (EGCG); epigallocatechin (EGC); epicatechin (EC), epicatechin-3-gallate (ECG); reactive oxygen species (ROS); Chaperone-mediated Autophagy (CMA); CXC motif chemokine ligand 1 (CXCL1); cyclooxygenase-1 (COX-1); cyclooxygenase-2 (COX-2); interleukin (IL); activated protein 1 (AP-1); B-cell lymphoma 2 (Bcl-2); Bcl-2 associated X protein (Bax); Poly ADP-Ribose Polymerase 1 (PARP-1); Heat Shock Protein 70 (Hsp70); Janus kinase 2 (JAK2); Signal transducers and activators of the transcription 3 (STAT3); cellular Myc (c-Myc); phospoinositide 3 kinase (PI3K); c-Jun N terminal kinase (JNK); Phosphatase and tensin homolog (PTEN); forkhead transcription factor 1 (FOXO1); histon deacetylase (HDAC); transforming growth factor β (TGF-β); tumor necrosis factor α (TNF- α); interferon γ (IFN- γ); interferon inducible protein 10 (IP-10); CC motif chemokine ligand 5 (CCL5).

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