Tea and Tea Polyphenols in Cancer Prevention$^{1,2}$

Chung S. Yang,$^3$ Jee Y. Chung, Guang-yu Yang, Saranjit K. Chhabra and Mao-Jung Lee

Laboratory for Cancer Research, College of Pharmacy, Rutgers, The State University of New Jersey, Piscataway, NJ 08854

ABSTRACT The inhibitory action of tea (Camellia sinensis) and tea components against cancer formation has been demonstrated in different animal models involving different organ sites in many laboratories. The possible preventive activity of tea against cancer in humans, however, is not clear. A critical question is whether the information obtained from animal studies is applicable to humans because of possible species differences or the difference in the quantity of tea used in animal studies and that consumed by humans. This article will discuss the results from animal studies and possible cancer inhibitory mechanisms that might be applicable to human cancer prevention. To provide a basis for more quantitative analyses of the effect of tea on carcinogenesis, the levels of tea polyphenols in blood, urine and tissue samples have been analyzed, and the pharmacokinetic properties of tea polyphenols studied. Studies with cell lines have demonstrated that tea polyphenols affect signal transduction pathways, inhibit cell proliferation and induce apoptosis, but the effective concentrations are usually much higher than those observed in blood and tissues. More mechanistic studies in these areas will help us to understand the inhibitory action of tea against carcinogenesis and provide background for evaluating the effects of tea consumption on human carcinogenesis. J. Nutr. 130: 472S–478S, 2000.

KEY WORDS: • tea • polyphenols • cancer chemoprevention • animal models • cancer cell lines

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3 To whom correspondence and reprint requests should be addressed.

4 Abbreviations: AP-1, activator protein 1; DGT, decaffeinated green tea; EC, (-)-epicatechin; ECG, (-)-epicatechin-3-gallate; EC, (-)-epigallocatechin; EGCG, (-)-epigallocatechin-3-gallate; IC$_{50}$, concentration at which 50% inhibition occurs; NF-kB, nuclear factor xB; NNK, 4-(methylationosamine)-1-(3-pyridyl)-1-butane; TPA, 12-O-tetradecanoylphorbol-13-acetate.

The cancer preventive activity of tea and tea constituents has been demonstrated in different animal models by different laboratories. These results have generated a great deal of enthusiasm for the use of tea as a cancer preventive agent. The scientific questions that remain to be answered are the following: 1) What are the active components for the cancer prevention activity? 2) What are the mechanisms involved? 3) Can the cancer preventive activity be demonstrated in humans? This paper will also discuss these issues.

Tea constituents and their biochemical properties

Tea is usually prepared by infusing green or black tea leaves in hot water. A typical tea beverage, prepared in a proportion of 1 g leaf to 100 mL water in a 3-min brew, usually contains 250–350 mg tea solids. Green tea is manufactured by drying fresh tea leaves; therefore, its composition resembles that of fresh tea leaves in that it contains characteristic polyphenolic compounds, (-)-epigallocatechin-3-gallate (EGCG), (-)-epigallocatechin (EGC), (-)-epicatechin-3-gallate (EGC) and (-)-epicatechin (EC). These compounds, commonly known as catechins, usually account for 30–42% of the dry weight of the solids in brewed tea (Balentine et al. 1997). The structures of the major catechins and black tea polyphenols are shown in Figure 1. EGCG is the most abundant catechin and has received by far the most attention. Flavonoids such as quercetin and their glycosides exist at lower levels. Caffeine usually accounts for 3–6% of the dry weight of brewed tea. In the manufacture of black tea, the tea leaves are crushed to allow the polyphenol oxidase to catalyze the oxidation and polymerization of catechins in a process known as “fermentation.” Some of the catechins remain, accounting for 3–10% of the dry weight in brewed black tea. Theaflavins, which includes theaflavin, theaflavin-3-gallate, theaflavin-3' -gallate and theaflavin-3,3' -digallate, are key to the characteristic color and taste of black tea and account for 2–6% of the dry weight in brewed black tea. The major fractions of black tea polyphenols, generally known as thearubigens, have higher molecular weights and are poorly characterized chemically; they account for >20% of the solid weight of brewed black tea (Balentine...
The inhibition of carcinogenesis by tea in animal models

The inhibition of carcinogenesis by tea has been demonstrated by different investigators in many animal models. These include cancers of the skin, lung, esophagus, stomach, liver, duodenum and small intestine, pancreas, colon, bladder, prostate and mammary gland (Conney et al. 1999, Dreosti et al. 1997, Katiyar and Mukhtar 1996, Yang and Wang 1993, Yang et al. 1996 and 1999). For example, inhibition of lung tumorigenesis by tea preparations has been demonstrated in A/J mice that have been treated with the tobacco carcinogen, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butaneone (NNK). Administration of decaffeinated green or black tea to mice during the NNK treatment period, or after the NNK treatment period, markedly reduced the number of tumors formed in the mice (Wang et al. 1992a). The inhibitory activity of black tea theaflavins was also demonstrated in this model (Yang et al. 1997a). When the mice were given black tea 16 wk after the NNK injection, at which time almost all of the mice had developed adenomas, the progression of these tumors to adenocarcinomas was significantly inhibited (Yang et al. 1997a). Brewed black tea and green tea infusions, when given to A/J mice as the sole source of drinking fluid, also inhibited spontaneous lung tumorigenesis (Landau et al. 1998). The inhibitory action of caffeine against lung tumorigenesis has been demonstrated previously in mice (Xu et al. 1992) and in rats (Chung et al. 1998). In the latter study, caffeine, at a concentration corresponding to that in a 2% black tea extract, displayed inhibitory activity comparable to, or stronger than that of 2% black tea. The above experiments indicate that tea has a broad inhibitory activity against both spontaneous and chemically induced lung tumorigenesis and is effective when administered during the initiation, promotion or progression stages of carcinogenesis. This conclusion may also apply to other animal models such as chemically and UV light–induced skin carcinogenesis (Huang et al. 1997, Katiyar and Mukhtar 1996, Wang et al. 1992b and 1994).

Conflict results have been reported concerning the effects of tea on colon carcinogenesis. Colon cancer formation induced by azoxymethane in rats and by 1,2-dimethylhydrazine in mice was inhibited by low concentrations of green tea polyphenols (0.01 or 0.1% solution as drinking fluid) and EGCG (reviewed in Yang et al. 1996). On the other hand, treatment of rats with black tea (0.6–2.5% solution) in drinking fluid did not inhibit azoxymethane-induced colon carcinogenesis (Weisburger et al. 1998). One possible interpretation of these two different types of results is that green tea polyphenols inhibit colon carcinogenesis, whereas some black tea constituents are much less effective. The effect of tea on mammary carcinogenesis was not demonstrated in several studies. In a recent study, black tea (1.25 or 2.5% solution) was found not to inhibit 7,12-dimethylbenz[a]anthracene-induced mammary gland tumorigenesis in rats fed an AIN-76A diet, but to reduce mammary tumorigenesis in rats fed a high fat diet (Rogers et al. 1998). On the other hand, EGCG has been shown to inhibit the growth of human breast and prostate cancer cells in athymic mice (Liao et al. 1995).

Effects of tea consumption on human cancers

Many epidemiologic studies have been conducted to investigate the effects of tea consumption on human cancer incidence, but no clear-cut conclusion could be drawn (Blot et al. 1997, Buschman 1998, Katiyar and Mukhtar 1996, Yang and Wang 1993, Yang et al. 1996). For example, studies in northern Italy have suggested a protective effect of tea against oral, pharyngeal and laryngeal cancer. In a case-control study in Shanghai, frequent consumption of green tea was shown to be associated with a lower incidence of esophageal cancer, especially among nonsmokers and nondrinkers of alcohol. Similar studies in Shanghai have suggested that green tea consumption is associated with lower risk of pancreatic and colorectal cancer. The protective effect against gastric cancer by tea has also been suggested from studies in Kyushu (Japan), northern Turkey, and central Sweden, but not from many other studies in different geographic areas (Yang et al. 1996). In studies in Saitama, Japan, women consuming >10 cups of tea daily were shown to have lower risk for cancer (all sites combined) and...
increased tea consumption was associated with lower risk for breast cancer metastasis and recurrence (Imai et al. 1997, Nakachi et al. 1998). In a prospective cohort study of post-menopausal women in Iowa, tea (mostly black tea) drinking was shown to be associated with a lower risk for digestive tract cancers and urinary tract cancers (Zheng et al. 1996). On the other hand, in the Netherlands Cohort Study on Diet and Cancer, consumption of black tea was not found to affect the risk for stomach, colorectal, lung and breast cancers (Goldbohm et al. 1996). A recent study of middle-aged Finnish men indicated a positive association between increased tea consumption and colon cancer risk (Hartman et al. 1998).

It appears that most reports on the cancer prevention effects were from studies of Asians, who drink predominantly green tea, whereas studies of black tea-drinking Europeans observed a protective effect infrequently. The reviews on green tea and human cancer by Buchman (1998) and on black tea and cancer by Blot et al. (1997) seem to be consistent with this suggestion. One possibility is that the cancer prevention activity of green tea is stronger than that of black tea. In studies on the inhibition of cancer formation by tea in animal models, the effective components appear to be catechins, theaflavins and caffeine. Although black tea contains all three classes of compounds, its catechin content is much lower than that of green tea. The bioavailability and biological activity of the major components of black tea, theauginogen, are not known. It is also possible that the different results on tea and cancer are due to different etiological factors involved in different geographic areas (Yang and Wang 1993).

The lack of a clear-cut conclusion concerning the protective effect of tea against cancer in humans is in contrast to the conclusions obtained from animal studies. Some possible reasons for this difference are as follows: 1) tea may have a weak protective effect, but it is masked by confounding factors associated with life style in certain populations; 2) the amount of tea consumed by humans is much lower than the tea concentrations used in animal studies and therefore cannot produce a significant effect; and 3) tea inhibits carcinogenesis in animals by mechanisms that may not be applicable to the prevention of human cancer. To address these points, information on the active components, their bioavailability and tissue levels, and their mechanisms of action is of great importance.

**Absorption and tissue distribution of tea polyphenols**

**Studies in animals.** The absorption and tissue distribution of tea catechins are just beginning to be understood. After intravenous injection of decaffeinated green tea (DGT) into rats, the plasma concentration-time curves of EGCG, EGC and EC could be fit into a two-compartment model. The β elimination half-lives ($t_{1/2B}$) were 212, 45 and 41 min for EGCG, EGC and EC, respectively. When pure EGCG was given intravenously, however, a shorter $t_{1/2B}$ (135 min) for EGCG was observed, suggesting that other components in DGT could affect the plasma concentration and elimination of EGCG. After intragastric administration of DGT, ~14% of EGCG and 31% of EC appeared in the plasma, but <1% of EGC was bioavailable. Conversion of intragastrically administered EGCG to EGC was not observed. After intravenous administration of DGT, the level of EGCG was found to be the highest in the intestines, whereas the highest levels of EGC and EC were observed in the kidney.

When tea (0.6% green tea polyphenols) was administered to rats through the drinking fluid, the blood levels of EGC and EC were much higher than that of EGCG (unpublished results). A large amount of EGCG was found in the feces. The blood EGCG and EC levels increased in the first 2 wk (peaked at 800–1000 μg/L), and then the levels decreased markedly to 300–350 μg/L on d 28. A similar decrease in urinary excretion of EGC and EC was also observed. When a second cycle of tea administration was initiated after a 10-d washout period, the EGC and EC levels were much lower than the high levels found on d 8 and 14 in the first cycle. After administration of tea to the rats for 8 d, substantial amounts of EGC and EC were found in the rat esophagus (185–195 ng/g tissue) large intestine (300–930), kidney (400–500), bladder (800–810), lung (190–230), and prostate (240–250), but the levels of EGC and EC were low in the liver, spleen, heart and thyroid. The amount of EGCG was higher in the esophagus and large intestine because of direct contact, but lower in other organs because of poor systemic absorption of EGCG by rats. A similar pattern of increase and then decrease in blood catechin levels was also seen in mice, except that the decrease took place 4 d after treatment with tea. In mice, the bioavailability of EGCG was much higher than that in rats. The highest levels of EGCG and other catechins were in the low micromolar range.

**Blood and saliva levels in humans**

Administration of 1.5, 3.0 and 4.5 g of DGT (in 500 mL of water) resulted in maximal plasma concentrations of 326, 550 and 190 μg/L for EGCG, EGC and EC in human volunteers, respectively (Yang et al. 1998a). These values were observed at 1.4–2.4 h after the ingestion of the tea preparation. A good dose-response relationship was not observed between the doses of 3 and 4.5 g. The half-life of EGCG (5.0–5.5 h) appeared to be higher than those of EGC and EC (2.5–3.4 h). EGCG and EC (and lower levels of EGCG in some individuals) were excreted through the urine. Over 90% of the total urinary EGCG and EC was excreted within 8 h. Substantial amounts of the catechins were detected in colon mucosa and prostate tissues in surgical samples from patients who consumed tea 12 h before surgery. The results suggest that in heavy tea drinkers (e.g., five cups green tea per day), the plasma levels of catechins are comparable to those found in mice used in cancer prevention studies with tea.

Because of the possible application of tea in the prevention of oral and esophageal cancers, the salivary levels of tea catechins were determined in six human volunteers after drinking tea (Yang et al. 1998a). Saliva samples were collected after the mouth was rinsed thoroughly with water. After drinking green tea preparations (equivalent to 2–3 cups of tea), peak saliva levels of EGCG (11.7–43.9 mg/L), EGCG (4.8–22.8 mg/L) and EC (1.8–7.5 mg/L) were observed after a few minutes. These levels were two orders of magnitude higher than those in the plasma. The $t_{1/2}$ of the salivary catechins was 10–20 min, much shorter than that of the plasma. Holding a tea solution in the mouth for a few minutes (without swallowing) produced even higher salivary catechin levels, but taking tea solids in capsules resulted in no detectable salivary catechin level. Holding EGCG solution in the mouth resulted in EGCG and EGC in the saliva and subsequently EGCG in the urine. The results suggest that EGCG was converted to EGC in the oral cavity, and both catechins were absorbed through the oral mucosa. A catechin esterase activity that converts EGCG to EGC was found in the saliva (Yang et al. 1998a). The results suggest that slowly drinking tea is a very effective
way of delivering rather high concentrations of catechins to the oral cavity and then the esophagus.

Studies with cell lines

Green and black tea polyphenols generally result in the growth inhibition of many cell lines. Some of the results are summarized in Table 1. The efficacy of inhibition varied, depending on the cell line used. EGCG was generally the best inhibitor in most of the cell lines tested, with 50% inhibition (IC50) values ranging between 22 and 130 μmol/L. EGC and GC were better inhibitors toward A427 cells with IC50 values of 34 and 38 μmol/L, respectively. Our laboratory has also shown that EGCG and EGC inhibit the growth of H661 and H1299 with IC50 values of 22 μmol/L. The growth inhibition of Ha-ras–transformed 21 BES cells by the black tea polyphenol, theaflavin-3–3′-digallate, was similar to the growth inhibition caused by green tea polyphenols, EGCG and EGC (Yang et al. 1998b). These studies demonstrate the biological activities of tea polyphenols, but the effective concentrations observed are generally 1–2 orders of magnitude higher than peak human plasma concentrations. The lowest effective concentration of EGCG (1–2 μmol/L) observed was in the inhibition of the transformation of preneoplastic human mammary epithelial cells by benzo[a]pyrene (Katdare et al. 1998).

Tea polyphenols may inhibit cell growth through a variety of mechanisms. One such mechanism is through the induction of apoptosis. Human cancer cell lines that have shown changes indicative of apoptosis after EGCG or EGC treatment include PC-9, H661, KATO III, DU 145, A431, HaCat and Molt-43 (see Table 1) (Ahmad et al. 1997, Hibasami et al. 1996 and 1998, Okabe et al. 1997). EGCG may also act as a prooxidant through H2O2 production to inhibit EGCG-induced apoptosis (Yang et al. 1998b). In combination with other chemopreventive drugs such as sulindac and tamoxifen, EGCG induced a synergistic apoptotic effect (Suganuma et al. 1999). Other mechanisms for the growth inhibition of cancer cells may be through the induction of cell cycle arrest by EGCG and the inhibition of signal transduction pathways leading to the activation of important transcription factors activator protein 1 (AP-1) and nuclear factor κB (NF-κB) (Ahmad et al. 1997, Dong et al. 1997, Lin and Lin 1997, Okabe et al. 1997).

Possible active components and their bioavailability

Many authors have considered EGCG as the active component of green tea because EGCG is the most abundant catechin, and cancer inhibitory activity of EGCG has been demonstrated (Chung et al. 1998, Katiyar and Mukhtar 1996, Xu et al. 1992, Yamane et al. 1995). However, other components should also be considered on the bases of their biological activities and bioavailabilities. For example, EGC, which is also abundant in green tea, is more bioavailable; it has anti-proliferative and inhibitory activities similar to those of EGCG and could also be important.

From our studies with three human lung cancer cell lines (H661, H1299 and H441) and one colon cancer cell line (HT-29), we observed that the potency of inhibiting cell growth by green tea polyphenols had the rank order of EGCG > EGC > EC (Yang et al. 1998b). The potency of theaflavin-3–3′-digallate was similar to that of EGCG and higher than that of theaflavin-3 (3′)-gallate, which was still higher than theaflavin. The growth inhibitory activity of green tea extracts appeared to be due to the summation of activities of EGCG, EGC, ECG and EC. These catechins, together with the theaflavins, may account for most of the growth inhibitory activity of black tea extracts. The growth inhibitory activity of theaflavins, the major components of black tea, is not known.

The inhibition of EGCG against skin, stomach, colon, and lung carcinogenesis (Chung et al. 1998, Xu et al. 1992, Yamane et al. 1995) as well as the growth of human prostate and breast tumors in athymic mice have been demonstrated (Liai et al. 1995). Theaflavins have been shown to inhibit lung and

### Table 1

<table>
<thead>
<tr>
<th>Human cancer</th>
<th>Cell line</th>
<th>Biological activity</th>
<th>EC50&lt;sup&gt;1&lt;/sup&gt; EGCG, μmol/L</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human oral</td>
<td>1483 HNSCC</td>
<td>Growth Inhibition</td>
<td>18</td>
<td>(Khafif, et al. 1998)</td>
</tr>
<tr>
<td>Human breast</td>
<td>MCF-7</td>
<td>Growth Inhibition</td>
<td>120</td>
<td>(Valcic, et al. 1996)</td>
</tr>
<tr>
<td>Human lung</td>
<td>PC-9</td>
<td>Growth Inhibition</td>
<td>140</td>
<td>(Suganuma, et al. 1999)</td>
</tr>
<tr>
<td></td>
<td>A-427</td>
<td>Apoptosis</td>
<td>100</td>
<td>(Okabe, et al. 1997)</td>
</tr>
<tr>
<td></td>
<td>H441</td>
<td>Growth Inhibition</td>
<td>94</td>
<td>(Valcic, et al. 1996)</td>
</tr>
<tr>
<td></td>
<td>H661</td>
<td>Growth Inhibition</td>
<td>60</td>
<td>(Yang, et al. 1998b)</td>
</tr>
<tr>
<td></td>
<td>H1299</td>
<td>Apoptosis</td>
<td>22</td>
<td>(Yang, et al. 1998b)</td>
</tr>
<tr>
<td>Human stomach</td>
<td>H1299</td>
<td>Apoptosis</td>
<td>22</td>
<td>(Yang, et al. 1998b)</td>
</tr>
<tr>
<td>Human colon</td>
<td>Caco-2</td>
<td>Apoptosis</td>
<td>2000</td>
<td>(Hibasami, et al. 1998)</td>
</tr>
<tr>
<td>Human skin</td>
<td>DU145</td>
<td>Apoptosis</td>
<td>175</td>
<td>(Ahmad, et al. 1997)</td>
</tr>
<tr>
<td></td>
<td>A431</td>
<td>Apoptosis</td>
<td>87</td>
<td>(Ahmad, et al. 1997)</td>
</tr>
<tr>
<td></td>
<td>HaCat</td>
<td>Apoptosis</td>
<td>175</td>
<td>(Ahmad, et al. 1997)</td>
</tr>
<tr>
<td></td>
<td>UACC-375</td>
<td>Growth Inhibition</td>
<td>130</td>
<td>(Valcic, et al. 1996)</td>
</tr>
<tr>
<td>Human blood</td>
<td>Molt-43</td>
<td>Growth Inhibition</td>
<td>100</td>
<td>(Hibasami, et al. 1996)</td>
</tr>
</tbody>
</table>

<sup>1</sup> Effective concentrations of EGCG used for 50% or greater activity are provided as examples. Concentrations of other tea polyphenols can be found in some of the references.
Possible mechanisms for the inhibitory actions of tea on tumorigenesis

Many mechanisms have been proposed concerning the inhibitory action of tea against tumorigenesis. As was pointed out previously (Yang et al. 1996, Yang 1997), in the search for relevant mechanisms, it is necessary to consider the tissue concentrations of the effective components. Inhibitory activities elicited by low micromolar or lower concentrations are likely to be more relevant than activities demonstrated with higher concentrations of tea components. Many studies have demonstrated the inhibition of carcinogen activation by tea and tea polyphenols in vitro, but such activities have been observed only in some studies in vivo. Moderate enhancement of glutathione peroxidase, catalase, glutathione S-transferase, NADPH-quione oxidoreductase, UDP-glucuronosyl transferase and methoxyresorufin O-dealkylase activities by tea administration has also been reported (Bu-Abbas et al. 1994, Khan et al. 1992, Sohn et al. 1994); the effects of these enzyme inductions on carcinogenesis are not clear. The inhibitory activities of tea and tea polyphenols may be due in many cases to their ability to inhibit growth-related signal transduction pathways. For example, EGCG and theaflavins inhibit epidermal growth factor– or TPA-induced transformation of JB6 cells, and this inhibition was correlated with the inhibition of AP-1–dependent transcriptional activity (Dong et al. 1997). The inhibition of AP-1 activation occurs through the inhibition of a c-jun NH2-terminal kinase–dependent pathway. In H-ras-transformed JB6 (the 30.7b Ras 12 cells), the H-ras–activated AP-1 pathway is a major growth stimulant. In these cells, the AP-1 activation was inhibited by EGCG, EGC, theaflavin-3,3'-digallate and other polyphenols, and the phosphorylation of both c-jun and an extracellular signal-regulated protein kinase (Erk) was inhibited (unpublished results). Because the ras genes are activated in many animal carcinogenesis models and in human cancers, the inhibition of the phosphorylation of c-jun and Erk could be an important mechanism for the inhibition of cancer formation and growth. A recent study by Cao and Cao (1999) demonstrated that EGCG can inhibit angiogenesis by inhibiting the growth of endothelial cells. In addition, the administration of 1.25% green tea as the drinking fluid to mice injected with vascular endothelial growth factor reduced corneal neovascularization significantly by 35–70%. This can be a potent mechanism for the inhibition of cancer growth in vivo. Inhibition of tumor necrosis factor alpha has also been proposed as a possible mechanism for the cancer preventive activity of EGCG (Sugano et al. 1996). The inhibition of NF-kB by EGCG, which has been demonstrated recently (Lin et al. 1997), may also contribute to the anticarcinogenic effect of tea polyphenols in many situations.

SUMMARY

The effects of tea on human carcinogenesis are still not clear. Judging from the diverse inhibitory activities observed in different animal carcinogenesis systems and different cancer cell lines, it is likely that there are multiple mechanisms by which tea constituents elicit their inhibitory effects against carcinogenesis. The challenge is to determine which mechanisms are relevant to human cancer prevention. In this context, only activities displayed by tea constituents at concentrations that are achievable in human tissues are important. Therefore, information on the bioavailability and tissue levels of EGCG, EGC, theaflavins and other tea constituents should be a vital part of future mechanistic studies. The knowledge recently gained on the tissue levels and biological activities of tea polyphenols would be useful in the planning of future epidemiologic studies and human cancer prevention trials. Further studies are required to determine the best dosage and route by which to deliver particular tea constituents to an certain organ. Large doses of pure compounds in capsules may not be the best way to deliver the active components effectively to many tissues.

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LITERATURE CITED


The effects of green tea on cancer prevention have been extensively studied. Several mechanisms of action have been identified, including inhibition of carcinogenesis, induction of apoptosis, and modulation of the immune system. Inhibitory effects have been observed on various cancer types, such as breast, colon, and skin cancer. The antioxidant properties of green tea polyphenols, such as catechins, have been linked to cancer prevention. Further research is needed to fully understand the complex interactions between green tea and the biological processes that prevent cancer.


