Effects of Tea Consumption on Nutrition and Health[1]

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ABSTRACT Beneficial health effects of tea have been demonstrated in animal experiments and some human studies. The two most extensively investigated diseases are cancer and heart disease. Although mechanisms of protective activity of tea against these diseases have been proposed, there are inconsistencies in the relationship between tea consumption and the risk of these diseases in humans. The bioavailability of active components is beginning to be understood, but further research is required to determine whether the results from animal studies are applicable to humans. Also discussed are the possible effects of tea in increasing thermogenesis and bone density as well as decreasing risk of cataracts and arthritis. The potential health benefits of tea consumption warrant further investigation. J. Nutr. 130: 2409–2412, 2000.

KEY WORDS: • tea • cancer • heart disease • health benefits

Tea, the dried leaves of the plant *Camellia sinensis*, is a popular beverage consumed worldwide. About three billion kilograms of tea are produced and consumed yearly. The possible beneficial health effects of tea are being investigated and have received a great deal of attention. This review examines the available scientific information concerning tea and health.

Chemistry of Tea Constituents. Green tea is manufactured by drying fresh tea leaves. It contains characteristic polyphenolic compounds, (–)-epigallocatechin-3-gallate (EGCG),[3] (–)-epigallocatechin (EGC), (–)-epicatechin-3-gallate (ECG) and (–)-epicatechin (EC) (**Fig. 1**). These compounds are commonly known as catechins. 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, comprised of 30–42% catechins and 3–6% caffeine (1). EGCG is the most abundant catechin and has received by far the most attention. In manufacturing black tea, the tea leaves are crushed to allow the polyphenol oxidase to catalyze the oxidation, leading to polymerization of catechins. The remaining catechins account for 3–10% of the solids in brewed black tea. Theaflavins, which include 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 solids in brewed black tea. The major fractions of black tea polyphenols, accounting for >20% of the solids in brewed black tea, are known as thearubigens. They have larger molecular weights and are poorly characterized chemically. More detailed information on the composition of green and black tea can be found in Balentine et al. (1). Of the tea produced worldwide, 78% is black tea, which is usually consumed in the Western countries, 20% is green tea, which is commonly consumed in Asian countries, and 2% is oolong tea which is produced (by partial fermentation) mainly in southern China.

The most widely recognized properties of tea polyphenols are their antioxidant activities, arising from their ability to scavenge reactive oxygen species (2). Tea polyphenols also bind to metal ions, preventing them from participating in peroxidative reactions. Green and black tea and isolated tea polyphenols have been shown to scavenge reactive oxygen and nitrogen species, reducing their damage to lipid membranes, proteins and nucleic acids in cell-free systems. The manifestation of these activities in biological systems is discussed in subsequent sections.

Absorption, Distribution, Metabolism and Elimination of Tea Polyphenols. Recent advances made in the analysis of tea polyphenols have improved our understanding of the pharmacokinetics of these compounds. In our studies, the total amount (free plus conjugated forms) of each catechin was used for pharmacokinetic analysis. Administration of 1.5, 3.0 and 4.5 g of decaffeinated green tea solids (in 500 mL of water) to human volunteers resulted in maximal plasma concentrations (C_{max}) of 326 ng, 550 ng and 190 ng/L for EGCG, EGC and EC, respectively (3). These C_{max} values were observed at 1.4–2.4 h after the ingestion of the tea preparation. The elimination half-life ($t_{1/2}$) of EGCG (5.0–5.5 h) appeared to be higher than those of EGC and EC (2.5–3.4 h). EGC and EC, but not EGCG, were excreted in the urine. Over 90% of the total urinary EGC and EC (mostly in the conjugated forms) was excreted within 8 h. Substantial amounts of the catechins were detected in colon mucosa in surgical samples from patients who consumed tea 12 h before surgery (4). After drinking green tea preparations, human volunteers had peak saliva levels of EGC, EGCG and EC two orders of magnitude higher than those in the plasma (5). The $t_{1/2}$ of the salivary catechins was 10–20 min, much shorter than that of the plasma. EGCG was converted to EGC in the oral cavity, and a salivary catechin esterase activity was characterized (5). There are indications that both catechins were absorbed through the oral mucosa.

More detailed pharmacokinetic studies have been conducted in rats (6). After intravenous injection of decaffeinated green tea, the $t_{1/2}$ was 212, 45 and 41 min for EGCG, EGC, and EC, respectively. The highest level of EGCG was found in the intestines and the highest levels of EGC and EC were observed in the kidney. After intragastric administration of

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[3] Abbreviations used: C_{max}, maximal plasma concentrations; EC, (–)-epicatechin; ECG, (–)-epicatechin-3-gallate; EGC, (–)-epigallocatechin; EGCG, (–)-epigallocatechin-3-gallate; M4, [5-(3′,4′-dihydroxyphenyl)-γ-valerolactone]; M6, [5-(3′,4,′,5′-trihydroxyphenyl)-γ-valerolactone]; $T_{1/2}$, elimination half-life; TNF, tumor necrosis factor.

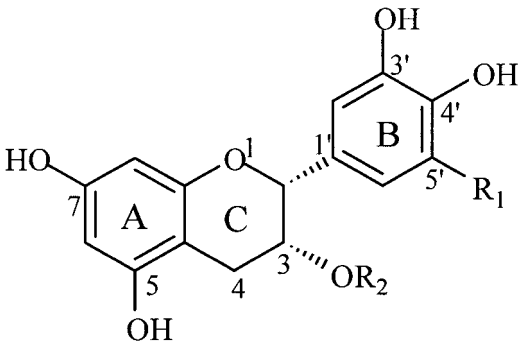

FIGURE 1 Structures of catechins. For EGCG, R_1 = OH and R_2 = galloyl; for ECG, R_1 = H and R_2 = galloyl; for EGC, R_1 = OH and R_2 = H; for EC, R_1 = H and R_2 = H.

decaffeinated green tea, ~14% of EGC and 31% of EC appeared in the plasma, but <1% of EGCG was bioavailable in rats. When green tea solutions were given to rats in the drinking fluid, the blood levels of EGC and EC were much higher than that of EGCG, and the level of EGC and EC declined after prolonged feeding (7). A similar pattern of decrease in blood catechin levels was also seen in mice. In mice, the plasma level of EGCG was much higher than that in rats. This species difference is probably due to the poor absorption of EGCG by rats. The highest levels of these catechins were in the low micromolar range (7).

Catechins, especially those without the gallate moiety, are readily conjugated to glucuronide and sulfate; the conjugated forms may account for two thirds of the catechins found in the plasma and urine. *O*-Methyl EGC (mainly in the glucuronide or sulfated forms) has recently been found in our laboratory to be a major metabolite, present at levels 4–5 times higher than EGC in human plasma and urine. *O*-Methylated EGCG derivatives, with methylation occurring at the one or two of the 3′, 4′, 3″ and 4″ positions, have been found in the bile of rats (8). The conversion of EGCG to EGC (and presumably ECG to EC) takes place in the intestine. Substantial amounts of catechins are degraded by microorganisms in the intestine of humans and animals, leading to the formation of [5-(3′,4′-dihydroxyphenyl)-γ-valerolactone] (M4) and [5-(3′,4,′,5′-trihydroxyphenyl)-γ-valerolactone] (M6) (9). These metabolites are the ring fusion products of EGC and EC, respectively. Both M4 and M6 (mainly in the glucuronide and sulfate form) have been detected in human urine and plasma; in some individuals, the amounts of urinary M4 and M6 were several fold higher than their respective precursors (9). These metabolites were also found in various rodent tissues. The biological activities of these catechin metabolites requires investigation.

Tea and Cardiovascular Diseases. Many epidemiologic studies have investigated the effects of tea consumption on cardiovascular disease (reviewed in 10,11). Earlier cohort studies in California and Norway yielded inconsistent results. In a long-term study of a Dutch cohort, the highest tertile of tea consumption was associated with a lower risk of death from coronary heart disease and lower incidence of stroke. In a follow-up study in Rotterdam, an inverse association of tea intake with severity of aortic atherosclerosis was observed (12). The Boston Area Health Study found that subjects who drank one (200–250 mL) or more cups of black tea per day had approximately half the risk of a heart attack compared with those who did not drink tea at all (13). Welsh men, however, had a positive association between black tea consumption and ischemic heart disease. It was thought that the addition of milk to tea, common among the Welsh, might have abolished the antioxidant potential of the tea. In two subsequent studies on this topic, however, the presence of milk did not affect the plasma level or urinary excretion of catechins (14).

One of the proposed mechanisms for the possible protective effect of tea against cardiovascular diseases is that tea polyphenols inhibit the oxidation of LDL, which is known to be involved in the development of atherosclerosis (2); however, such an antioxidative effect was not demonstrated in three recent human studies (reviewed in 11,14). A fourth study indicated that consumption of black tea slightly protected LDL against oxidation ex vivo. Tea polyphenols accumulated in LDL particles after 3 d of green or black tea consumption, but their levels were not sufficient to enhance resistance to LDL oxidation (14).

The hypocholesterolemic activity of tea could also contribute to the protection against heart disease. In animals fed diets high in fat and cholesterol, green tea, black tea and tea polyphenols prevented elevations in serum and liver lipids, decreased serum total cholesterol or atherogenic index, and increased fecal excretion of total lipids and cholesterol (15–17). When hamsters were fed a high fat diet, those drinking green tea or green tea polyphenols had lower serum total cholesterol and triacylglycerol levels but higher fecal fat excretions than the control group (18). Nevertheless, epidemiologic studies and human trials failed to show a serum cholesterol–lowering effect from the consumption of green or black tea (11). Of the 13 recently published epidemiologic studies on this topic, only four reported a significant inverse relationship (11,19–21). Another potential mechanism may be via the effects of tea on body weight and fat. Such effects will be described in subsequent sections.

The recent observations that intragastric administration of black tea inhibited platelet aggregation and prevented experimental coronary thrombosis in dogs and that consumption of green tea polyphenols decreased ADP-induced platelet aggregation provide another possible mechanism for preventing cardiovascular diseases (reviewed in 22). Green tea extract equivalent to 10 cups (2 L) of tea for 4 wk, however, did not have significant effects on several indicators related to cardiovascular diseases (23). Both black and green tea caused larger acute (30 min after ingestion) increases in blood pressure than caffeine alone (24). Regular tea consumption, however, did not alter blood pressure.

Tea and Cancer. The public press heralds tea as a cancer-preventive beverage because such activity has been demonstrated in many animal models. These models include cancers of the skin, lung, esophagus, stomach, liver, small intestine, pancreas, colon, bladder, prostate and mammary glands (reviewed in 25–27). Tea solutions are usually given to animals as the sole source of drinking fluid. The extensive studies on UV light–induced and chemically induced skin tumorigenesis as well as chemically induced and spontaneously generated lung tumors in mice indicate that tea has broad inhibitory activity against tumorigenesis and is effective when administered during the initiation, promotion or progression stages of carcinogenesis. This conclusion may also apply to other animal models. Conflicting results have been reported concerning the effects of tea on colon carcinogenesis; both inhibition and a lack of inhibition have been reported. Inhibition of chemically induced mammary gland tumorigenesis by black tea was not observed in rats fed an AIN-76A diet, but was observed in rats fed a high fat diet. EGCG has been shown to inhibit the growth of human breast and prostate cancer cells in athymic mice.

Many epidemiologic studies have been conducted to investigate the effects of tea consumption on human cancer incidence, yet the results have been inconclusive (25–30). 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 has been shown to be associated with a lower incidence of esophageal cancer, especially among those who neither smoke nor consume alcohol. A protective effect against gastric cancer by tea has also been suggested from studies in Japan, northern Turkey and central Sweden, but not from many other studies in different geographic areas. In Japan, women consuming >10 cups (2 L) of tea daily have been shown to have lower risk for all cancers, and increased tea consumption was associated with lower risk for breast cancer metastasis and recurrence (31). In a prospective cohort study of postmenopausal 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. On the other hand, many studies did not suggest a protective effect of tea against cancer. For example, 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 (32). It appears that most reports showing positive cancer preventive effects were from studies of Asians who drink predominantly green tea, whereas studies of black-tea drinking Europeans observed protective effects infrequently. One possibility is that the cancer preventive activity of green tea is stronger than that of black tea. The effective components in tea appear to be catechins, theaflavins and caffeine; the catechin content in black tea is much lower than that in green tea. The consumption of tea is also associated with different life styles in different regions. It is possible that the different results on tea and cancer are due to the different etiological factors present in different populations.

Many mechanisms have been proposed concerning the inhibitory action of tea against carcinogenesis (reviewed in 25,27,33). The most commonly cited mechanism is the antioxidative activities, but many other mechanisms are also important. The antiproliferative effect of tea catechins has been demonstrated in lung and skin tumorigenesis models in mice. Inhibition of cell transformation and cell growth by purified catechins and theaflavins has also been reported. These activities have been attributed to the inhibition of activator protein 1 (AP-1) activity, possibly due to the inhibition of mitogen-activated protein kinase activities. Because of the frequent activation of AP-1 in many human cancers, this action may be applicable for human cancer prevention. Tea polyphenols have been shown to inhibit the phosphorylation of retinoblastoma protein by cyclin-dependent kinase 2/4 (Cdk 2/4), nuclear factor κB (NFκB) activity, tumor necrosis factor (TNF)-α release, and the binding of epidermal growth factor and 12-*O*-tetradecanoylphorbol-13-acetate to their respective receptors, thus inhibiting tumor promotion. Inhibition of tumor promotion–related enzymes, such as ornithine decarboxylase, protein kinase C, lipoxygenase and cyclooxygenase, by tea has been shown. An association between lowering of body fat by tea and inhibition of skin tumorigenesis has been observed (A. H. Conney, Rutgers University, personal communication). We have observed that mice drinking either black tea or green tea had fewer lung tumors and weighed significantly less than controls, although they consumed the same amount or more food (34). Retroperitoneal fat pads also weighed less in these tea-drinking mice. On the basis of the diverse inhibitory activities observed in different animal models and different cancer cell lines, it is likely that multiple tea constituents and mechanisms are involved in the inhibition of carcinogenesis.

Effects of Tea on Nutrition and Other Health Issues. In diet-induced obese mice, consumption of oolong tea for 10 wk prevented obesity and fatty liver (35). Decreased nutrient absorption and increased energy expenditure may both contribute to these effects. Green tea extracts stimulated brown adipose tissue thermogenesis in rats to a greater extent than could be attributed to caffeine alone (36). Ingestion of green tea extract by healthy young men with each meal resulted in a significant increase in 24-h energy expenditure and a significant decrease in the 24-h respiratory quotient compared with both placebo and caffeine treatments (37). These authors suggested that tea polyphenols inhibit the activity of catechol-*O*-methyltransferase and act synergistically with caffeine to prolong sympathetic stimulation of thermogenesis.

Tea polyphenols have a strong affinity for proteins and minerals, and thus may affect nutritional status (reviewed in 38). The various phenolic groups of tea can bind to more than one place on a protein via hydrophobic interactions and hydrogen bonding. Polyphenols have a strong affinity for proteins with a high proline content, such as milk caseins, gelatin and salivary proline-rich proteins. Whether tea consumption impairs protein absorption in humans remains to be investigated. Because of the strong binding affinity of tea polyphenols to metal ions, the possible effects of tea on the absorption of these nutrients is of importance. Decreased iron absorption due to drinking tea has been reported (38). Apparently, this effect is mainly on nonheme iron, especially when tea and iron are consumed simultaneously. The absorption of heme iron from cooked meats was not affected by tea consumption. Tea drinking was found to be a risk factor in infant microcyte anemia. In the National Health and Nutrition Examination Survey II study with 11,684 participants, however, anemia was not associated with consumption of tea and coffee. When methanol extract of black tea was given to rats, the apparent calcium absorption was lower than that in the control rats during d 11–18, but by wk 4, there was no difference; the treatment did not affect the apparent absorption of magnesium or protein.

Among women 65–76 y of age, tea consumption was associated with greater bone mineral density measurements (39), which is consistent with previous work reporting that tea was protective against hip fracture. These data suggested that components other than polyphenols, such as phytoestrogens or fluoride, may influence bone mineral density. Tea was found to inhibit glucosyltransferase activity of oral streptococci and the development of dental caries in rats (40). Tea contains fluoride, which may strengthen tooth enamel and improve dental health.

In a collagen-induced arthritic mouse model, green tea polyphenols significantly reduced the incidence and severity of arthritis (41). The expression of inflammatory mediators including cyclooygenase-2, interferon-γ and TNF-α was markedly lower in the arthritic joints of green tea polyphenol-fed mice. Cataract, which develops as a result of protein precipitation in the lens of the eye, may be reduced by increased tea consumption (42).

Concluding Remarks. The possible beneficial health effects of tea consumption have been suggested by some epidemiologic studies and supported by some laboratory studies. Other studies, however, are not consistent with such beneficial effects. A difficulty in human studies is the possible confounding factors related to life style, such as smoking, coffee intake and fat intake. In animal studies, the doses required for demonstrating the disease prevention effects are usually higher than the amounts consumed by humans who drink tea. Caution is

required, however, in the use of high concentrations of tea for disease prevention. Ingestion of large amounts of tea may cause nutritional and other problems because of the strong binding activities of tea polyphenols and the caffeine content, although no solid data exist concerning harmful effects of tea consumption. More research is warranted to elucidate the biological activities of green and black tea for possible health benefits in humans.

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