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REVIEW ARTICLE

A Review of Green Tea– Health benefits and Effects

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ABSTRACT

Tea is one of the most widely consumed beverages in the world, second only to water, and its medicinal properties have been widely explored. Green tea is capable of improving hyperlipidaemia and the impaired kidney functions.

Green Tea Benefits: Consumption of green tea provides a protection against stroke, liver disease, bacterial infection, cancer, viral infection and lowers the risk of osteoporosis. Green tea catechin was reported to have a protective effect on mammalian hepatic cells, leading to its therapeutic use for hepatitis. World wide studies conducted thus far present mixed evidences with regard to the effects of green tea on oxidation. Some studies conclude that green tea catechins have both in vitro and in vivo antioxidant activities while others show no antioxidant benefit in vivo. In particular, EC and EGC are the isomers contributing to the regulation of lipoprotein oxidation. Much has been written elsewhere about the value of green tea (Camellia sinensis) from a health perspective. To begin with, the active principles in green tea are its catechins and caffeine. Research has demonstrated that green tea is capable of stimulating thermogenesis and promoting fat oxidation; in other words, it helps burn body fat. Research and a history of traditional use lend support to green tea's potential as weight loss botanicals.

KEY WORDS: Green Tea, Cancer, Viral infection, Atherosclerosis, Antioxidant, Thyroid gland, Obesity.

Introduction

Aside from water, tea is the most consumed beverage in the world [1,2]. In particular, green tea is consumed primarily in Japan, China, and some parts of the Middle East, North Africa, and North America. The benefits arising from drinking green tea have therefore, been the focus of some studies during recent years. The term green tea refers to the product manufactured from fresh leaf of the tea plant, Camelliasinensis[3]. Green tea is an excellent source of water-soluble polyphenol antioxidants. In particular, green tea leaves are very rich in flavonoids, which are a group of polyphenols present in vegetables, fruits and beverages such as tea and wine [4]. During manufacturing of green tea, the oxidation of polyphenolic components is precluded [1].

Green tea (Camellia sinensis) has a long history as a folk remedy, and it is the most widely consumed beverage in the world but the beneficial medicinal properties have only been elucidated in the past 20 years. Recently green tea is being widely studied

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for its beneficial effects in the treatment and prevention of human diseases. Green tea extracts contain a number of components including catechins, saponins and flavonoids and it has been demonstrated that catechins can reduce cellular oxidation, blood glucose level and cholesterol absorption. Therefore, it has been expected that an intake of green tea extract will prevent or delay the onset of diseases such as cardiovascular diseases, diabetes and cancer. Flavonoids are known to be effective in removing individual oxygen and free radicals from the lipid peroxidation step. Polysaccharides of green tea have also been reported to have hypoglycemic activities. Great advanced studies have been made to reveal the hypoglycemic effect of tea polysaccharides.

Tea is one of the most widely consumed beverages in the world, second only to water, and its medicinal properties have been widely explored.The tea plant, Camellia sinensis, is a member of the Theaceae family, and black, oolong, and green tea are produced from its leaves.Tea, particularlygreen tea, is an important source of flavonoids, namely catechins, which are strong antioxidants. Among cathechins, epigallocatechingallate is the more powerful antioxidant invitro and it is the most abundant polyphenol in green tea[5]. It has been reported that green tea polyphenols increase insulin activity in diabetic rats. Intestinal glucose uptake is mainly accomplished by the sodium-dependent glucose transporter, which was markedly inhibited by green tea polyphenols, thus decreasing the intestinal absorption of glucose which leads to hyperglycemia. In the present study, diabetic rats showed constant fasting hyperglycemia throughout the study and the administration of green tea extract to diabetic rats reduced the elevation of blood glucose, some studies found that the green tea reduced blood glucose level in both type 1 and type 2 of diabetic rat models. The antihyperglycemic effect of green tea constituents was ascribed to the activities of basal insulin, inhibition of intestinal glucose transporter and decrease the expression of genes that control gluconeogenesis. Some studies have shown that catechins can inhibit digestive enzymes such as salivary amylase, intestinal sucrase and α-glucosidase, suggesting that the reduced digestibility action of catechins may be responsible for lowering blood glucose levels. In diabetic rats, these mechanisms may be responsible for the anti-hyperglycemic effect of green tea. It has been reported that the insulin-like action of polyphenols is involved in the mechanism by which green tea decreases the blood glucose level. Alloxan produces oxygen radicals in the body, which cause pancreatic injury which is responsible for increased blood glucose seen in the diabetic rats indicated that diabetes induced by alloxan was reversed by simultaneous administration of green tea polyphenol. The consumption of green tea produced a significant reduction in glucose level of diabetic rats. Also green tea is capable of improving hyperlipidaemia and the impaired kidney functions. In addition, the pretreatment of rats with green tea for 6 weeks before induced diabetes is effective in controlling the hyperglycemia and hyperlipidaemia.

General Information

The Green tea is obtained from the tea plant Camellia sinensis belongs to the family Theaceae. Tea is the most consumed drink in the world after water. Green tea is a 'non-fermented' tea and contains more catechins than black tea or oolong tea. Catechins are invitro and invivo strong antioxidants. In addition, its content of certain minerals and vitamins increases the antioxidant potential of this type of tea. Presently, it is cultivated in atleast 30

countries around the world. Tea beverage is an infusion of the dried leaves of Camellia sinesis. It is a widely used medicinal plant by the trials throughout India, China and popular in various indigenous system of medicine like Ayurveda, Unani and Homoeopathy. Green tea has been consumed throughout the ages in India, China, Japan and Thailand.

Scientific Classification

Kingdom: Plantae

Order : Ericales

Family : Theaceae

Genus : Camellia

Species : C. sinensis

Binomial name :Camellia sinensis(L.) Kuntze.

Common Names

India : Chha contributed by polyphenols. Other compounds are

China : Cha alkaloids (caffeine, theophylline and theobromine), amino

Russia : Chai acids, carbohydrates, proteins, chlorophyll, volatile

Africa : Itye organic compounds (chemicals that readily produce

Italy : Te vapors and contribute to the odor of tea), fluoride

England : Tea plant aluminum, minerals and trace elements. Polyphenols United State

There is good evidence that green tea catechins have a role in the protection against degenerative diseases.[4] Tea drinking, by providing antioxidants, may become valuable in several chronic diseases known as oxidative stress conditions[6], such as the cardiovascular diseases (CVD)[7-8]. These pathologies are one of the major causes of mortality and morbidity in the western world[9]. The search for new factors leading to a decrease in the prevalenceof CVD and other oxidative stress diseases becomes,therefore, valuable.

Mechanisms of Action

The anticarcinogenic properties of green tea polyphenols, mainly EGCG, are likely a result of inhibition of tumor initiation and promotion, induction of apoptosis, and inhibition of cell eplication rates, thus retarding the growth and development of neoplasms[10-11]. Green tea polyphenols antioxidant potential is directly related to the combination of aromatic rings and hydroxyl groups that make up their structure, and is a result of binding and neutralization of free radicals by the hydroxyl groups. In addition, green tea polyphenols stimulate the activity of hepatic detoxification enzymes, thereby promoting detoxification of xenobiotic compounds and are also capable of chelating metal ions, such as iron, that can generate radical oxygen species[12-13].

Green tea polyphenols inhibit the production of arachidonic acid metabolites such as pro-inflammatory prostaglandins and leukotrienes,resulting in a decreased inflammatory response. Human and animal studies have demonstrated EGCG's ability to block inflammatory responses to ultraviolet A and B radiation, as well as significantly inhibiting neutrophil migration that occurs during the inflammatory process[14-16]. Research on green tea's thermogenic properties indicates a synergistic interaction between its caffeine content and catechin polyphenols that can result in prolonged stimulation

of thermogenesis. Studies have also shown green tea extracts are capable of reducing fat digestion by inhibiting the activity of certain digestive enzymes[17-18]. Although the exact mechanism is unknown, green tea catechins have been shown significantly to raise the levels of Lactobacilli and Bifidobacteria while decreasing levels of numerous potential pathogens[19]. Studies have also demonstrated green tea's antibacterial properties against a variety of gram-positive and gram-negative species[20].

Discussion

Regarding the effect of green tea on lipid profile are controversial because of the difference in the dose and duration of these studies. The lipid profiles in normal groups also did not alter following the intervention. The possible mechanisms by which green tea extract can exert cholesterol lowering effect are reducing the absorption of dietary and biliary cholesterol and promoting its fecal excretion.

Antioxidant Applications

Many chronic disease states and inflammatory conditions are a result of oxidative stress and subsequent generation of free radicals. Some of these include heart disease (resulting from LDL oxidation), renal disease and failure, several types of cancer, skin exposure damage caused by ultraviolet (A and B) rays, as well as diseases associated with aging. Green tea polyphenols are potent free radical scavengers due to the hydroxyl groups in their chemical structure.

The hydroxyl groups form complexes with free radicals and neutralize them, preventing the progression of the disease process.

It has been attributed to green tea drinking a decrease in lipid peroxidation, in free radical generation, in LDL-oxidation and in the development of oxidative stress [21-24]. However, conflicting results have been reported about the antioxidant activity of green tea consumption, in what concerns the effect on plasma lipid profile and plasma lipid peroxidation[25-28]. The controversial reported data is probably confounded by dietary and lifestyle habits, by the way tea is consumed, in a regular way or not, and by the way tea is prepared and the concentration and volume of green tea consumption. Therefore, further studies are warranted to clarify the value of green tea drinking.Tea polyphenols may act as antioxidants by scavenging reactive oxygen and nitrogen species and by chelating redox-active transition metal ions, and may also act indirectly as antioxidants through, among other mechanisms, the inhibition of "pro-oxidant" enzymes and induction of antioxidant enzymes[29]. The antioxidant properties of flavonoids may therefore, protect tissues,cells and plasma constituents against oxidative damage[30-31].

Presenting a limited biosynthesis capacity and poor repair mechanisms, the circulating red blood cells (RBC) suffers and accumulates physical and chemical changes, which become more pronounced with cell age or with cell damage occurring whenever an unusual physical or chemical stress develops[32-33]. The removal of senescent or damaged RBCs seems to involve the development of a senescent neoantigen on the plasma membrane surface[34]. This neoantigen is immunologically related to band 3, a RBC transmembrane protein[35]. Modifications in this protein by proteolytic cleavage, clustering or exposure of unusual epitopes, triggers the binding of specific anti-band 3 autoantibodies and complement activation, marking the cell for death[36].

The degradation of the RBC metabolism and of the antioxidant defences, in senescent or damaged RBC by favouring the development of oxidative stress, allows haemoglobin oxidation and its linkage to band 3, promoting the aggregation of band 3 protein and the binding of natural anti-band 3 auto antibodies[37]. Previous studies performed in several oxidative stress conditions, namely in myocardial infarction, in ischemic stroke, in high competition physical exercise, in pregnancy, and in psoriasis, showed that the evaluation of membrane bound haemoglobin (MBH) and of the band 3 profile may provide good markers of erythrocyte ageing or damage[38-41].

Lipid peroxidation is a well-established mechanism of cellular injury and has been used as an indicator of oxidative stress. Measurement of MDA and 4-HNE, both products of lipid peroxidation,were also used in the present study as that kind of markers. The antioxidant activity and the effect of green tea drinking upon oxidative stress have been widely studied[25-28]. These studies suggest that the changes in antioxidant capacity and in oxidative stress are related to differences in the studied population, namely in dietary and lifestyle habits, and/or in the experimental protocols. The several reported ways for preparation of tea, in what concerns the temperature, time of infusion and concentration, besides the time period of green tea consumption, may explain the controversial data. Previous data suggest for green tea drinking a beneficial effect, by reducing the development or the enhancement of oxidative stress and, therefore, protecting the individual for oxidative stress diseases. The value of regular green tea consumption and the way it should be prepared to reach a healthy effect.

LDL Oxidation

In vitro the abilities of green tea extract and its three major components to inhibit lipid oxidation in low-density lipoprotein (LDL) catalyzed by copper. Experiments performed by few studies shown that green tea intake by rats in studies significantly prolongs the lag phase during the invitro oxidation of LDL. The lag phase is a very sensitive indicator of resistance LDL to invitro oxidation [42]. The longer the lag phase, the more resistance the substance is to oxidation. The observations that green tea intake significantly increases the lag time suggest that the polyphenolic compounds such as certain catechins may also be transported in lipoproteins and inhibit their oxidation. On the other hand, the rate of conjugated diene formation (another indicator of lipoprotein oxidation) is only slightly slower with ingestion of green tea compared to the control diet (vitamin E deficient). In addition, green tea diet does not significantly decrease lipid peroxide values. The formation of thiobarbituric reactive substances (TBARS) is a nonspecific measure of oxidative damage to lipoproteins. Green tea diet shows a slightly lower TBARS value than the control group. An invivo study shows that green tea is a powerful inhibitor of plasma lipid peroxides and LDL oxidizability. It is found to be an invivo antioxidant in both the normal and cholesterol-fed groups. As expected, the cholesterol feeding produced an increase in plasma lipid peroxides and LDL oxidizability; the latter as shown by the decrease in lag time and the increased maximal oxidation rate. Green tea significantly lengthened the lag phase in both the normal and cholesterol-fed groups. When compared to black tea, green tea is significantly better. Green tea increased the lag time 71% in normal animals and 63% in animals with high cholesterol. Infact,

the green tea increased the lag time of the cholesterol-fed animals to the extent that it was not significantly different from the control group. In addition, the maximal oxidation rate in the high cholesterol-fed animals, but not the normal group, is decreased significantly by green tea. There is evidence that green tea polyphenols bind to LDL when spiked in plasma. This incorporation, subsequent to tea polyphenol absorption into the plasma, would explains increase in lag time after consumption. Green tea also protects human LDL from oxidative modification [43].

Conflicting evidences also exist and should be taken into consideration. One study demonstrates that green tea consumption has a potent antioxidant activity on LDL oxidation invitro, but found no effect invivo [44]. In another study performed on humans, evidence shows that consumption of 6 cups of green tea per day did not affect resistance of LDL to oxidation, or markers of oxidative damage to lipids invivo, but slightly increased total antioxidant activity of plasma[45]. The invitro experiment shows that resistance of isolated LDL to oxidation increased only after incubation of plasma with very high amounts of green tea. These amounts, when converted to tea catechin concentrations, were much higher than those expected invivo.

Atherosclerotic Plaque Formation

Green tea reduces the maximum rate of LDL oxidation,Green tea antioxidants may accumulate in the arterial wall and lower LDL oxidation in the arterial wall by increasing cellular antioxidant status or by inhibiting activities of oxidizing enzymes. Another factor mediating LDL oxidation in the arterial wall is the presence of free metal ions, and there is evidence that flavonoids are strong chelators of free metal ions [46].

Green Tea Benefits

Consumption of green tea provides a protection against stroke [47], liver disease [48], bacterial infection [49-50], cancer [51], viral infection [52-53] and lowers the risk of osteoporosis [54-55]. Green tea catechin was reported to have a protective effect on mammalian hepatic cells, leading to its therapeutic use for hepatitis [56-57]. The catechin incorporated in cell membranes was shown to prevent or reduce the morphological and biochemical alterations of hepatocytes induced by hepatotoxicity agents [58-59]. Tsuchiya [60] suggested that the reduction in membrane fluidity is responsible for the anti-plaque and hepatoprotective effects of green tea catechins.

Anticancer effects of green tea

Worldwide interest in green tea as a cancer preventive agent for humans has increased, because it is non-toxic and it is effective in wide range of organs. Consumption of green tea is a practical and effective cancer preventive both before cancer onset and after cancer treatment [61]. Several researches indicate that polyphenolic antioxidants present in green and black tea can reduce risk in a variety of animal tumor bioassay systems [61-62]. The consumption of tea and its polyphenolic constituents affords protection against chemical carcinogen or ultraviolet radiation-induced skin cancer in the mouse model. Tea consumption also affords protection against cancers induced by chemical carcinogens that involve the lung, fore stomach, esophagus, duodenum, pancreas, liver, breast, colon, and skin in mice, rats, and hamsters. Recent studies have indicated that green tea and its polyphenolic constituents impart inhibitory effects on the activities of many enzymic, metabolic, and signaling pathways that have relevance to cancer development and progression [63-67].

Effect of green tea on blood pressure

Hodgson et al. [68] concluded that tea ingestion caused larger acute increases in blood pressure than caffeine alone. However, any acute effects of tea on blood pressure did not translate into significant alterations in ambulatory blood pressure during regular tea consumption.

Effect of green tea on thyroid glands

The green tea extract catechins was confirmed to have antithyroid and aromatase inhibition effects [69], that is obvious when 5% polyphenone-60 (P-60), green tea extract catechins, was administered to male rats for 2-8 weeks induced goiters and decreased weights of the body. Endocrinologically, elevating plasma thyroid stimulating hormone (TSH), luteinizing hormone (LH) and testosterone levels and decreasing tri-iodothyronine (T3) and thyroxine (T4) levels were induced by this treatment. It was also found that P-60 as a whole and some of its constituents exhibited inhibitory effects on human placental aromatase activity [69].

Effect of green tea on brain

Hong et al. [70] suggested that the minimizing effect of green tea extract on the eicosanoid accumulation and oxidative damage in addition to the reduction of neuronal cell death could eventually result in protective effect on ischemia/reperfusion-induced brain injury and behaviour deficit [71-72].

Obesity and Weight Loss

Green tea extract standardized to 8.35% caffeine and 24.7% catechins has been shown to stimulate brown adipose tissue invivo, with thermogenesis greater than the effect the caffeine content accounts for long term ingestion of tea catechins stopped the accumulation of body fat in mice with high fat diet induced obesity, possibly due to the activation of hepatic lipid metabolism. Human studies suggest that green tea may contribute to a reduction in the risk of cardiovascular disease and some forms of cancer, as well as to the promotion of oral health and other physiological functions such as antihypertensive effect, body weight control, antibacterial and antivirasic activity, bone mineral density increase, antifibrotic properties and europrotective power. Increasing interest in its health benefits has led to the inclusion of green tea in the group of beverages with functional properties. Other traditional uses of green tea include treating flatulence (gas), regulating body temperature and blood sugar, promoting digestion and improving mental processes. As an herbal remedy, green tea is often recommended to ease stomach discomfort,vomiting and to stop diarrhea. The antibacterial action of tea is useful in treating infections and wounds. The research interest based on tea components may provide an approach to decrease the incidence of and mortality from various diseases. Overall tea is an affordable beverage of natural origin compared to modern beverages such as soft drinks.

Recent studies on green tea's thermogenic properties have demonstrated a synergistic interaction between caffeine and catechin polyphenols that appears to prolong sympathetic stimulation of thermogenesis. A human study of green tea extract containing 90 mg EGCG taken three times daily concluded that men taking the extract burned 266 more calories per day than did those in the placebo group and that green tea extract's thermogenic effects may play a role in controlling obesity.

Green tea polyphenols have also been shown to markedly inhibit digestive lipases invitro, resulting in decreased lipolysis of triglycerides, which may translate to reduced fat digestion in humans.

Other Applications

Sickle cell anemia is characterized by a population of "dense cells" that may trigger vasoocclusion and the painful sickle cell "crisis." Ultraviolet A radiation is highly effective shown to substantially increase the risk for developing squamous cell carcinoma and melanoma.

Side Effects

Green tea is generally considered a safe, non-toxic beverage and consumption is usually without side effects. The average cup of green tea contains from 10-50 mg of caffeine, and over-consumption may cause irritability, insomnia, nervousness, and tachycardia.

Conclusion

Tea is the most popular drink after water. Green tea has been consumed everyday by millions of people around the world since ancient times in order to maintain and improve health. Most modern medicines used to treat cancer have serious side effects, high costs, and other associated risks. Green tea, on the other hand, is safe and widely available as a beverage and a nutritional supplement. While no single food item can be expected to provide a significant effect on public health, it is important to note that a modest effect between a dietary component and a disease having a major impact on the most prevalent causes of morbidity and mortality, i.e., cancer and heart disease, should merit substantial attention. This being said, it should also be stated that long-term successful weight loss has only been proven to occur with an ongoing program of dietary modification and exercise. Worldwide studies conducted thus far present mixed evidences with regard to the effects of green tea on oxidation. Some studies conclude that green tea catechins have both invitro and invivo antioxidant activities while others show no antioxidant benefit invivo. In particular, EC and EGC are the isomers contributing to the regulation of lipoprotein oxidation. Much has been written elsewhere about the value of green tea (Camellia sinensis) from a health perspective. To begin with, the active principles in green tea are its catechins and caffeine. Research has demonstrated that green tea is capable of stimulating thermogenesis and promoting fat oxidation; in other words, it helps burn body fat. Research and/or a history of traditional use lend support to green tea's potential as weight loss botanicals.

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References

1. Graham HN. 1992. Green tea consumption, composition, and polyphenol chemistry. Preventive Medicine.21:334-50.
2. Vinson JA, Dabbagh YA. 1998. Effect of green and black tea supplementation on lipids, lipid oxidation and fibrinogen in the hamster: mechanisms for the epidemiological benefits of tea drinking. Federation of European Biochemical Societies, Elsevier.433(1-2):446.
3. Tijburg LB, Wiseman SA, Meijer GW, Weststrate JA. 1997. Effects of green tea, black tea and dietary lipophilic antioxidants on LDL oxidizability and atherosclerosis in hypercholesterolaemic rabbits. Atherosclerosis.135(1):37-47. Nutrition Noteworthy, 2(1), Article 7 (1999).
4. Pearson DA, Frankel EN, Aeshbach R, German JB. 1996. Inhibition of endothelial cell mediated oxidation of low-density lipoproteins by green tea antioxidants.Federation of American Societies for Experimental Biology Journal. 10:A476.

5. Feng Q, Torii Y, Uchida K, Nakamura Y, Hara Y, Osawa T.2002. Blacktea polyphenols, theaflavins, prevent cellular DNA damage by inhibiting oxidative stress and suppressing cytochromeP450 1A1 in cell cultures. J Agric Food Chem. 50(1):213–20.
6. Crespy V, Williamson G. 2004. A review of the health effects of green tea catechins in invivo animal models. J Nutr.134(12Suppl.):3431S–40S.
7. Itoh Y, Yasui T, Okada A, Tozawa K, Hayashi Y, Kohri K. 2005. Preventive effects of green tea on renal stone formation and the role of oxidative stress in nephrolithiasis. J Urol;173(1):271–5.
8. Lee W, Min WK, Chun S.2005. Long-term effects of green tea ingestion on atherosclerotic biological markers in smokers.Clin Biochem;38(1):84–7.
9. Ferrie`res J. E´pidemiologieetathe´roscle´rose. 1998. Sang ThromboseVaisseaux.10:339–42.
10. Nihal A, Hasan M. 1999. Green tea polyphenols and cancer: biological mechanisms and practical implications.Nutr Rev. 57:78-83.
11. Ahmad N, Feyes DK, Nieminen AL. 1997. Green tea constituent epigallacatechin-3-gallate and induction of apoptosis and cell cycle arrest in human carcinoma cells. J Natl Cancer Inst. 89:1881-1886.
12. Serafini M, Ghiselli A, Ferro-Luzzi A. 1996. Invivo antioxidant effect of green and black tea in man. Eur J ClinNutr. 50:28-32.
13. Erba D, Riso P, Colombo A, Testolin G.1999. Supplementation of Jurkat T cells with green tea extract decreases oxidative damage due to iron treatment. J Nutr.129:2130-2134.
14. Katiyar SK, Matsui MS, Elmets CA Mukhtar H.1999.Polyphenolic antioxidant (-)-epigallocatechin -3-gallate from green tea reduces UVB-induced inflammatory responses and infiltration of leukocytes in human skin.Photochem Photobiol.69:148-153.
15. Zhao JF, Zhang YJ, Jin XH.1999. Green tea protects against psoralen plus ultraviolet A-induced photochemical damage to skin. J Invest Dermatol;113:1070-1075.
16. Hofbauer R, Frass M, Gmeiner B. 1999. The green tea extract epigallocatechingallate is able to reduce neutrophil transmigration through monolayers of endothelial cells. Wien KlinWochenschr.111:276-282.
17. Dulloo AG, Seydoux J, Girardier L. 2000. Green tea and thermogenesis: interactions between catechin polyphenols,caffeine, and sympathetic activity. Int J ObesRelatMetabDisord. 24:252-258.
18. Juhel C, Armand M, Pafumi Y. 2000. Green tea extract (AR25) inhibits lipolysis of triglycerides in gastric and duodenal medium in vitro. J NutrBiochem.11:45-51.
19. Goto K, Kanaya S, Nishikawa T.1998. Green tea catechins improve gut flora. Ann Long-Term Care.6:1-7.
20. Chou CC, Lin LL, Chung KT.1999. Antimicrobial activity of tea as affected by the degree of fermentation and manufacturing season. Int J Food Microbiol.48:125-130.
21. Ishikawa T, Suzukawa M, Toshimitsu I. 1997. Effect of tea flavonoid supplementation on the susceptibility of low density lipoprotein to oxidative modification.Am J ClinNutr.66(2):261–6.
22. Klauning JE, Xu Y, Han C. 1999.The effect of tea consumption on oxidative stress in smokers and nonsmokers.ProcSocExpBiol Med. 220(4):249–54.
23. Hashimoto R, Yaita M, Tanaka K, Hara Y, Kojo S. 2000. Inhibition of radical reaction of apolipoprotein B-100 and a-tocopherol in human plasma by green tea catechins. J Agric Food Chem. 48(12):6380–3.
24. Pearson DA, Frankel EN, Aeschbach R, German JB. 1998. Inhibition of endothelial cell mediated low-density lipoprotein oxidation by green tea extracts. J Agric. Food Chem. 46:1445–9.
25. Erba D, Riso P, Bordoni A, Foti P, Biagi PL, Testolin G. 2005. Effectiveness of moderate green tea consumption on antioxidative status and plasma lipid profile in humans.J NutrBiochem. 16(3):144–9.
26. Kimura M, Umegaki K, Kasuya Y, Sugisawa A, Higuchi M. 2002. The relation between single/ double or repeated tea catechin ingestions and plasma antioxidant activity in humans. Eur J ClinNutr. 56(12):1186–93.
27. Sung H, Nah J, Chun S, Park H, Yang SE, Min WK. 2000. Invivo antioxidant effect of green tea. Eur J ClinNutr. 54(7):527–9.
28. Freese R, Basu S, Hietanen E, Jagadeesan N, NakachiK,Bartsch H, Mutanen M.1999. Green tea extract decreases plasma malondialdehyde concentration but does not affect other indicators of oxidative stress, nitric oxide production, or hemostatic factors during a high-linoleic acid diet in healthy females. Eur J Nutr. 38(3):149–57.
29. Frei B, Higdon JV. 2003. Antioxidant activity of tea polyphenols invivo: evidence from animal studies. J Nutr.133(10):3275S–84S.

30. Hollman PCH, Feskens EJM, Katan MB.1999. Tea flavonols in cardiovascular disease and cancer epidemiology. ProcSocExpBiol Med. 220(4):198–202.

31. Huang Q, Wu LJ, Tashiro S, Gao HY, Onodera S, Ikejima T. 2005. (+)-catechin, an ingredient of green tea, protects murine microglia from oxidative stress-induced DNA damage and cell cycle arrest. J Pharmacol Sci. 98(1):16–24.

32. Lutz HU. 1990. Erythrocyte clearance. Harris JA, editor. Blood cell biochemistry. Erythroid cells, vol. 1. Plenum Press: New York; 81–120.

33. Kay MMB, Marchalonis JJ, Schluter SF, Bosman G. 1991. Human erythrocyte ageing: cellular and molecular biology. Trans Med Rev. 3:173–95.

34. Kay MMB. 1978. Role of physiological autoantibody in the removal of senescent human red cells. J Supramol Struct:555–67.

35. Kay MMB, Goodman SR, Sorensen K.1983. Senescent cell antigen is immunologically related to band 3. Proc Natl Acad Sci. 80(6):1631–5.

36. Lutz HU, Bussolino F, Flepp R. 1987. Naturally occurring antiband 3 antibodies and complement activation together mediate phagocytosis of oxidatively stressed human erythrocytes. Proc Natl Acad Sci. 84(21):7368–72.

37. Low PS. 1991.Role of heamoglobin denaturation and band 3 clustering in initiating red cell removal. In: Magnani M,DeFloraA, editors. Red blood cell ageing. Plenum Press:New York; 173–83.

38. Santos-Silva A, Rebelo I, Castro EMB. 2001. Leukocyte activation, erythrocyte damage, lipid profile and oxidative stress imposed by high competition physical exercise in adolescents. ClinChimActa.306(1–2):119–26.

39. Santos-Silva A, Rebelo I, Castro E. 2002. Erythrocyte damage and leukocyte activation in ischemic stroke.ClinChimActa.320(1–2):29–35.

40. Belo L, Rebelo I, Castro EMB. 2002. Band 3 as a marker of erythrocyte changes in pregnancy. Eur J Haemat. 69(3): 145–51.

41. Rocha-Pereira P, Santos-Silva A, Rebelo I, Figueiredo A,Quintanilha A, Teixeira F. 2004. Erythrocyte damage in mild and insevere psoriasis. Br J Dermatol.150(2):232–44.

42. Anderson JW, Diwadkar VA, Bridges SR. 1998. Selective effects of different antioxidants on oxidation of lipoproteins from rats. Proceedings of the Society for Experimental Biology and Medicine.218(4):376-81.

43. Zhang A, Zhu QY, Luk YS, Ho KY, Fung KP, Chen ZY. 1997. Inhibitory effects of jasmine green tea epicatechin isomers on free radical-induced lysis of red blood cells. Life Sciences.61(4):383-94.

44. Princen HM, van Duyvenvoorde W, Buytenhek R, Blonk C, Tijburg LB, LangiusJA, Meinders AE, Pijl H. 1998. No effect of consumption of green and black tea on plasma lipid and antioxidant levels and on LDL oxidation in smokers.Arteriosclerosis, Thrombosis, and Vascular Biology.18(5):833-41.

45. Van het Hof KH, de Boer HS, Wiseman SA, Lien N, Westrate JA, Tijburg LB. 1997. Consumption of green or black tea does not increase resistance of low-density lipoprotein to oxidation in humans. American Journal of Clinical Nutrition.66(5):1125-32.

46. Yokozawa T, Dong E. 1997. Influence of green tea and its three major component supon low-density lipoprotein oxidation. Experimental and Toxicologic Pathology.49(5):329-35.

47. Weisburger JH. Tea antioxidants and health. In Cadenas E, Packer L, Eds. 1996.Handbook of antioxidants. New York: Marcel Dekker. 469-86.

48. Jonathan MH.2008. Asia Pac J ClinNutr. 17(S1): 288-290.

49. Horiba N, Maekawa Y, Ito M, Matsumoto T, Nakamura H.1991. J Endo. 17: 122-125.

50. Terada A, Hara H, Nakajyo S. 1993. Microbial Ecol Health Dis. 6: 3-9.

51. Yang CS, Wang ZY. 1993. J Natl Cancer Inst. 85: 1038-1049.

52. Nakayama M, Toda M, Okubo S, Shimamura T. 1990.Letter ApplMicrobiol. 11: 38-40.

53. Tao P. Chung Kuo I Hsueh Yuan HsuehPao.1992. 14: 334-38.

54. Fujita T. 1994. AdvNutr Res. 9: 89-99.

55. Kao PC, P,eng FK. 1995. Chung Hua I HsuehTsaChih (Taipei).55: 209-213.

56. Blum AL, Berthet P, Doelle W, Goebell H, Kortum K, Pelloni S.1977.Lancet.ii: 1153-1155.

57. Kaul TN, Middleton E Jr, Ogra PL. 1985. J Med Virol. 15: 71-79.

58. Perrissoud D, Maignan MF, Auderset G. 1981.Int Cong SympSer R Soc Med.47: 21-25.

59. Varga M, Buris L.1990. ExpMolPathol.52: 249-257.

60. Tsuchiya H. 1999. Pharmacology. 59: 34-44.

61. Kohlmeier L, Weterings KGC, Steck S, Kok FJ.1997.Nutr Cancer. 27: 1-13.

62. Suganuma M, Okbe S, Sueoka N, Sueoka E, Matsuyama S, Imai K. 1999. MutatRes.v428 (1-2):339-344.
63. Liao S, Hiipakka RA. 1995. BiochemBiophys Res Comm. 214: 833-838.
64. Jankun J, Selmon H, Swiercz R, Skrzypczak-Jankun E.1997. Nature.387: 391.
65. Cao Y, Cao R. 1999. Nature.398: 381.
66. Garbisa S, Biggin S, Cavallarin N, Sartor L, Benelli R Albini A. 1999. Nat. Med. 5:1216.
67. Nam S, Smith DM, Duo OP. 2001. J Biol Chem. 276: 13322-13330.
68. Hodgson JM, Puddey IB, Burke V, Beilin LJ, Jordan N. 1999.J Hypertens.17: 457-63.
69. Satoh K, Sakamoto Y, Ogata A, Nagai F, Mikuriya H, Numazawa M.2002. Food ChemToxicol. 40: 925-933.
70. Hong JT, Ryu SR, Kim HJ, Lee JK, Lee SH, Kim DB.2000. Brain Res Bull. 53: 743-751.
71. Lee S, Suh S, Kim S. 2000. Neurosci. 287: 191-914.
72. Hong JT, Ryu SR, Kim HJ, Lee JK, Lee SH, Yun YP. 2001. Brain Res. 888: 11-18.