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REVIEW ON "GREEN TEA IN CANCER CHEMOTHERAPY"

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ABSTRACT
The idea that cancer may be prevented by consuming naturally occurring chemicals that people could eat is becoming more and more popular. Tea is cultivated in around thirty countries and is the most widely consumed beverage in the world, behind water. Over the past 10 years, a wealth of data from many laboratories worldwide has provided compelling evidence that tea's polyphenolic antioxidants reduce the risk of cancer in a variety of animal-tumor bioassay systems. Even though the epidemiological research is inconclusive, it has also shown that drinking tea may reduce the chance of developing cancer. Black tea has received less research attention than green tea. There are other polyphenolic antioxidants found in green tea, but the main one that's thought to be responsible for the majority of the tea's cancer-preventive qualities is (–)-epigallocatechin-3-gal-late (EGCG). These effects and the chemical processes behind the biological reaction to green tea polyphenols will be covered in this review.

KEYWORDS: Epigallocatechin-3-gallate (EGCG), green tea polyphenols, cancer, and chemoprevention etc.

1. INTRODUCTION

Chemoprevention has become a viable strategy for lowering the occurrence of cancer and, consequently, its related mortality and morbidity. Based on laboratory investigations, at least thirty distinct categories of drugs are currently recognized to possess chemopreventive activities against cancer. According to epidemiological research, a few of these agents are also showing potential (Challa et al., 1997; Chung et al., 1998; Dragsted et al., 1993; Kelloff et al., 1996; Pezzuto, 1997). "Polyphenols" are one such class of agents. The most popular beverage drank by humans, after water, is tea. According to Ahmad et al. (1998) and Katiyar and Mukhtar (1996), tea includes catechins, which are polyphenolic constituents. It was about ten years ago that polyphenolic agents found in green tea were first shown to have anti-mutagenic and anti-carcinogenic properties (Khan et al., 1988, Wang et al., 1989a,b). The evergreen plant Camellia sinensis, which belongs to the Theaceae family, is the source of green tea. In certain regions of the world, tea has long been thought to have health-promoting properties (Weisburger et al., 1997). Although they are not definitive, epidemiological studies indicate that drinking green tea may reduce one's chance of developing cancer. The bulk of research on the benefits of tea. However, black tea's chemopreventive potential has also been evaluated in a few studies (Katiyar and Mukhtar, 1996; Ahmad et al., 1998). Clinical research on the relationship between green tea drinking and cancer risk have been started, drawing on data from cell culture, laboratory animal, and epidemiological findings. Green tea's cancer-chemopreventive properties are attributed to its main polyphenolic antioxidants, which include (–)-epicatechin (EC), (–)-epigallocatechin (EGC), (–)-epicatechin-3-gallate (ECG), and (–)-epigallocatechin-3-gallate (EGCG). The most protective of them is thought to be EGCG (Ahmad et al., 1998; Katiyar and Mukhtar 1996).

Tea Consumption History

According to Chinese folklore, tea was initially discovered by the emperor Shen Nung in 2737 BC. The plant Camellia sinensis was first found and farmed in Southeast Asia thousands of years ago (Harbowy & Balentine, 1997). Since then, its renown has grown to the point that it is currently the most consumed beverage globally, second only to water. Around 120 milliliters of

brewed tea are used daily per person globally (Ahmad et al., 1998; Katiyar and Mukhtar 1996). At least thirty nations now grow and cultivate tea (Ahmad et al., 1998; Katiyar and Mukhtar 1996). Today, people drink a wide variety of tea preparations that all come from the same plant (Camellia sinensis) but are made using different techniques. According to Ahmad et al. (1998) and Katiyar and Mukhtar (1996), the three most popular major tea types are oolong tea (2%, consumed in some parts of China and Taiwan) and black tea (78%, consumed primarily in Western and some Asian countries), green tea (20%, consumed primarily in Asia and a few countries in North Africa and the Middle East).

Chemoprevention of Cancer with Tea

The polyphenolic antioxidants found in tea are capable of offering protection against the initiation and subsequent development of cancer, according to a number of studies conducted in laboratories worldwide using a variety of target-organ bioassay protocols on laboratory animals (Ahmad et al., 1998; Katiyar and Mukhtar 1996). Even though the data from several epidemiological studies carried out in various populations were not conclusive, they were deemed valuable enough to initiate clinical trials assessing the relationship between drinking green tea and the risk of cancer (Ahmad et al., 1998; Katiyar and Mukhtar 1996; Kohlmeier et al., 1997). In the mouse model, it has been demonstrated that oral ingestion or topical applications of green tea and/or its polyphenolic contents provide protection against UV radiation- and chemical carcinogen-induced skin carcinogenesis (Mukhtar et al., 1994). Green tea extracts containing the polyphenolic fraction, water extract, or individual polyphenolic antioxidants have also been demonstrated in numerous animal studies to provide protection against chemically induced carcinogenesis in the lung, liver, esophagus, forestomach, duodenum, pancreas, colon, and breast (Ahmad et al., 1998; Katiyar and Mukhtar 1996). According to current research, (–)-epigallo-catechin-3-gallate (EGCG) is thought to mediate a large portion of green tea's cancer-chemopreventive effects (Ahmad et al., 1998; Katiyar and Mukhtar 1996). It is acknowledged that green tea's additional polyphenolic compounds enhance its ability to prevent cancer. It is unclear, nonetheless, if the various polyphenolic chemicals found in green tea operate via distinct processes or via a comparable biochemical route. An optimal chemopreventive agent for human use need to possess little or negligible toxicity, excellent efficiency across many locations, oral bioavailability, a well-established mechanism of action, affordability, and human acceptability. Up to 400 mg of polyphenolic antioxidants, 200 mg of which are EGCG, may be found in only one cup of brewed green tea. It's also intriguing to note that green tea extracts are currently added to a wide range of consumer goods, including beverages, ice creams, cosmetics, and health care items.

The Bioavailability of Polyphenols in Tea

It is unclear how readily the active polyphenolic elements in tea are absorbed by humans and experimental animals after ingestion. In a research by Yang et al. (1998), plasma concentrations and urine excretion of tea catechins were evaluated as a function of time in 18 participants who were given varying doses of green tea. At 1.4–2.4 hours after ingesting the tea preparation, the maximum plasma concentrations (Cmax) of EGCG, EGC, and EC were determined to be 326 ng/ml, 550 ng/ml, and 190 ng/ml, respectively, after swallowing 1.5, 3.0, or 4.5 g of decaffeinated green tea (in 500 ml of water). The Cmax values increased by 2.7–3.4 times when the dosage was raised from 1.5 to 3.0 g. However, the Cmax values did not significantly rise at 4.5 g, indicating a saturation phenomena. The half-life of EGCG was found to be longer than that of EGC or EC at all concentrations tested. Urine contained EGC and EC but not EGCG, and 90% of the total urinary EGC and EC was eliminated in less than eight hours. Although there was no discernible dose-response relationship, the amount of EGC and EC excretion rose when the tea dosage was raised. Basic pharmacokinetic characteristics of green tea polyphenols in humans were supplied by this study, and these parameters may be used to determine the amounts of these chemicals in the body after consuming green tea. The location of radiolabeled [3H]EGCG in mice organs after oral administration was recently investigated by Saganuma et al. (1998). Numerous organs were discovered to be radioactive, including those where it has previously been demonstrated that EGCG or green tea extract inhibits carcinogenesis. These findings showed that the body may sustain a high concentration of tea polyphenols by regularly consuming green tea. These research might be helpful in formulating future plans to develop green tea as a viable chemopreventive agent.

Mechanism(s) of Biological Effects of Green Tea

First Mechanistic Research

It is crucial to comprehend the biological mechanisms behind the benefits of green tea, since doing so might aid in the development and enhancement of cancer chemotherapy preventive measures. The first mechanistic attempts in this direction were mainly concerned with determining how green tea polyphenols affected the following: ● reduction in biochemical markers of tumor initiation and promotion ● prevention against mutagenicity and genotoxicity of chemicals ● regulation of detoxification enzymes ● trapping of activated metabolites of carcinogens ● regulation of antioxidant and free-radical scavenging activity (Katiyar and Mukhtar, 1996).

Molecular Mechanisms

Below is a synopsis of the study done to understand the molecular processes underlying the biological reactions of polyphenols found in green tea.

Green tea polyphenols trigger the pathway known as mitogen-activated protein kinase (MAPK). The suppression of enzymes such cytochromes P450, which are involved in the bio-activation of carcinogens, has been linked to the protective benefits of green tea polyphenols (Yu et al., 1997). Phase II detoxifying enzymes have also been shown to be involved in the biological response to green tea in a few additional in vivo experiments. The MAPK pathway was implicated in the stimulation of Phase II enzymes by numerous medications because the 5' flanking regions of Phase II genes include an antioxidant-responsive element (ARE). According to Yu et al. (1997), this work showed that a possible signaling route involved in the control of ARE-mediated phase II enzyme gene expression may be the cause of the MAPK pathway's activation. Additionally, it was shown that the induction of CAT activity occurs when green tea polyphenol is applied to human hepatoma (HepG2) cells that have been transfected with a plasmid construct that contains ARE and a minimal glutathione S-transferase Ya promoter connected to the chloramphenicol acetyltransferase (CAT) reporter gene. The findings imply that green tea polyphenols use ARE to promote the transcription of Phase II detoxifying enzymes. The administration of green tea polyphenols also caused an increase in mRNA levels and a notable activation of c-Jun N-terminal kinase 1 (JNK1), extracellular signal-regulated kinase 2 (ERK2), and MAPK.

EGCG inhibits the action of urokinase

It has been shown recently that urokinase, one of the enzymes most commonly expressed in human malignancies, is inhibited by EGCG, which has anti-cancer properties (Jankun et al., 1997). It was shown by computer-based molecular modeling that EGCG binds to urokinase and extends from a positively charged loop of urokinase towards arginine 35, therefore preventing the catalytic triad of urokinase, which consists of histidine 57 and serine 195. By using the spectrophotometric amidolytic test to measure the inhibition of urokinase activity, these calculations were confirmed. However, Yang (1997) later questioned the study's viability at feasible dosage levels.

Green tea induces apoptotic cell death and arrest of the cell cycle

Chemopreventive drugs that control apoptosis may have an impact on the steady-state cell population since the rate of apoptosis greatly influences the life duration of both normal and cancer cells inside a biological system (Fesus et al., 1995). Apoptosis is induced by a number of cancer chemopreventive drugs, while tumor-promoting drugs are known to block apoptosis (Boolbol et al., 1996; Mills et al., 1995; Wright et al., 1994). As a result, it is reasonable to anticipate that chemopreventive drugs having shown effects in human epidemiology and/or animal tumor bioassay systems, as well as the capacity to cause cancer cells to undergo apoptosis, may have broader effects on the treatment of cancer. Currently, there are just a few recognized chemopreventive drugs that can induce apoptosis (Jee et al., 1998; Jiang et al., 1996). We demonstrated in our lab that EGCG causes human epidermoid carcinoma (A431) cells to undergo apoptosis and cell cycle arrest (Ahmad et al., 1997). Significantly, this apoptotic response was unique to cancer cells since normal human epidermal keratinocytes were not affected by EGCG administration, which instead caused the induction of apoptosis in human carcinoma keratinocytes HaCaT, human prostate carcinoma cells DU145, and mouse lymphoma cells LY-R.A different research (Chen et al., 1998) examined the impact of EGCG on the proliferation of human fibroblasts (WI38VA) transformed by the SV40 virus in comparison to normal WI38 cells. It was discovered in this investigation that EGCG inhibited the development of transformed WI38VA cells but not that of normal WI38VA cells. This investigation furthermore revealed a comparable differential increase.

Human colorectal cancer (Caco-2) cells, breast cancer (Hs578T) cells, and their respective normal counterparts are all inhibited by EGCG. Additionally, EGCG administration increased serum-induced expression of the c-fos and c-myc genes in transformed WI38VA cells, but not in normal WI38 cells. It also caused apo-ptosis. According to this study, these varying reactions may be caused by the differential activation of specific genes, such as c-fos and c-myc. It was shown in a different research (Fujiki et al., 1998) that EGCG and other tea polyphenols suppress the proliferation of human lung cancer (PC-9) cells that had reached the G2/M phase arrest in the cell cycle. As EGCG and green tea extract have been found to have anticarcinogenic properties, this work showed that [3H]EGCG, supplied via oral intubation into the mouse stomach, resulted in tiny quantities of 3H-activity in several organs including as skin, stomach, duodenum, colon, liver, lung, and pancreas consequences. The present study proposed the participation of the tumor necrosis factor (TNF)-α pathway as a mechanism of biological reactions mediated by EGCG.

In another have a look at by using Yang et al (1998), the boom inhibitory effects of green tea polyphenols were investigated the use of four human most cancers cellular strains. boom inhibition become measured by way of 3H-thymidine incorporation after 48 h of treatment. EGCG and EGC displayed strong growth inhibitory outcomes towards lung- tumor mobile strains H661 and H1299, with estimated IC50 values of 22 μM, but were less powerful towards lung-most cancers cell line H441 and colon-cancer cellular line HT-29 with IC50 values 2- to three-fold better. ECG turned into discovered to have lower sports while EC became even much less powerful. on this look at, exposure of H661 cells to a dose of 30 μM EGCG, EGC, or theaflavins for twenty-four h resulted in a dose-structured apoptosis. The incubation of H661 cells with EGCG also resulted in a dose-structured formation of H2O2. Addition of H2O2 to H661 cells led

to an apoptotic response similar to EGCG. EGCG-induced apo- ptosis in H661 cells became found to be absolutely inhibited by way of exogenously brought catalase (50 units/ml). This inhibition sug- gests that tea polyphenol-mediated H2O2 production consequences in apoptosis of the cells, contributing to the growth inhibitory potential of tea polyphenols in vitro. in this observe, the involve- ment of H2O2, in addition to the effect of catalase, become an intrigu- ing observation due to the fact the tea polyphenols are generally regarded as antioxidants. the rationale provided through the authors is that tea polyphenols additionally possess seasoned-oxidative ac- tivities (Yang et al., 1998).

EGCG inhibits cellular proliferation and tumor progression via epidermal growth factor receptor (EGFR) pathway

The activation of the epidermal growth element receptor (EGFR) tyrosine kinase through its ligand is thought to provoke a couple of mobile reponses related to cellular proliferation on one hand. The over-expression of EGFR is proven to supply neoplastic phenotype on the other hand. based totally on those records, a current take a look at by way of Liang et al. (1997) proven that EGCG drastically inhibits each DNA synthesis and the protein tyrosine kinase sports of EGFR, platelet-derived growth-fac- tor receptor (PDGFR), and fibroblast growth-factor receptor (FGFR), but not of pp60v–src, protein kinase C (p.c), or protein kinase A (PKA), in A431 cells. EGCG was also determined to inhibit the car-phosphorylation of EGFR via EGF and to dam the binding of EGF to its receptor. This take a look at counseled that EGCG may inhibit tumor development by means of blockading the EGFR-pathway.

EGCG inhibits the induction of nitric oxide (NO)-synthase via a down-regulation in the transcription factor nuclear fac- tor-nB (NFnB)

Nitric oxide (NO) is a bioactive molecule that performs an essential function in infection and carcinogene- sis, and in a latest take a look at, Lin and Lin (1997) assessed the outcomes of green tea polyphenols at the modulation of NO- synthase in thioglycollate-elicited and lipopolysaccharide (LPS)-activated peritoneal macrophages. Gallic acid (GA), EGC, and EGCG have been determined to inhibit the protein expression of inducible NO-synthase as well as the generation of NO. This examine further established that EGCG inhibits the activation of the transcription issue NFnB, an event this is believed to be associated with the induction of inducible NO-synthase (iNOS). Taken together, these information counseled that EGCG might also block the early occasion of NO-synthase induction via inhibiting the binding of transcription NFnB to the iNOS promoter, thereby, inhibiting the induction of iNOS transcription. The concept of involvement of NO in the organic reaction of EGCG was strengthened by way of every other look at by Chan et al. (1997), who demonstrated that EGCG causes an inhibition of lipopolysaccharide (LPS)- and interferon (IFN)-γ-activated iNOS mRNA expression in a cellular-way of life system. EGCG turned into additionally discovered to inhibit the enzyme activities of iNOS and neu- ronal NO-synthase (nNOS). Peroxynitrite (OONO) is a distinctly poisonous oxidizing and nitrat- ing species this is produced in vivo through a reaction between superoxide radical (O2-) and NO. In another examine, Pannala et al (1997) validated the potential of inexperienced tea polyphenols, viz., catechin, epicatechin, ECG, EGC, and EGCG to (i) inhibit OONO-mediated tyrosine-nitration, and (ii) limit floor price alteration of low density lipoprotein (LDL). in this take a look at, all the compounds examined had been located to be effective OONO scavengers, as they were effective in stopping the nitration of tyrosine. those polyphenols were also discovered to shield towards OONO-mediated LDL modification.

EGCG inhibits tumor promoter-mediated activator pro- tein-1 (AP-1) activation, and cell transformation

Because many studies have suggested recently that the activation of AP-1 performs an crucial function in tumor promoting, the down- law of this transcription component is now notion to be a popular therapeutic method against most cancers (McCarty, 1998). In a current observe (Dong et al., 1997) employing the JB6 mouse epidermal cellular line, an considerably utilized in vitro model machine for tumor advertising research, Dong et al investigated anti- tumor selling results of EGCG and theaflavins. each of these had been located to inhibit EGF- or TPA- prompted mobile trans- formation, in addition to AP-1-established transcriptional hobby and DNA binding activity. This observe similarly showed that the inhibition of AP-1 activation takes place via the inhibition of a c-Jun NH2-terminal kinase (JNK)-established pathway.

EGC inhibits the activity of the protein tyrosine kinase, c-jun mRNA expression, and JNK1 activation

In a latest study, Lu et al (1998) investigated a few possible mechanisms concerned with the antiproliferative capability of EGC. using rat aortic easy-muscle (A7r5) cells, it become confirmed that the ac- tivity of the serum-inspired membranous protein, tyrosine kinase (PTK), is inhibited by means of EGC. EGC was additionally determined to reduce the phosphorylation of many proteins with distinctive molecular weights, on the tyrosine web site, indicating that EGC might also inhibit activity of tyrosine kinase, or stimulate the activity of phosphatase. It become in addition demonstrated that EGC reduces the ranges of c-jun mRNA, phosphorylated JNK1, and JNK1- kinase activity. those records advise that the antiproliferative impact of EGC, as a minimum in part, is mediated via the inhibi- tion of tyrosine kinase interest, reducing c-jun mRNA expres- sion and inhibiting JNK1 activation. The involvement of PTK activity and protein phosphoryla- tion turned into in addition explained by some other examine, wherein Kennedy et al (1998) evaluated the mechanism of the antiproliferative capacity of inexperienced tea polyphenols in Ehrlich ascite tumor cells. on this

examine, EGC and EGCG treatments were located to result in a huge decrease in cellular viability. EGC, but now not EGCG, caused a stimulation of PTK pastime. EGC treatment was also determined to bring about tyrosine phosphorylations of forty two and 45 kDa proteins, and inside the pastime of ornithine decarboxylase (ODC), an essential cell enzyme in polyamine biosynthesis.

Skin Effects of Green Tea

pores and skin is the largest body organ and serves as a protecting barrier towards the deleterious consequences of environmental insults, consisting of the ones resulting from ultraviolet (UV) radiation. an awful lot of the deleterious effect of solar UV radiation is due to UVB (290 –320 nm). even though the long-time period abnormalities of UVB usually emerge as evident inside the populace elderly 50 years and past, epidemiological studies suggest that a lot of the crucial daylight exposure chargeable for those damaging outcomes is received at a younger age. current epidemiological observa- tions suggest that people with a records of non-cancer pores and skin most cancers have increased chance of melanoma and sure non- cutaneous cancers.

UVB induces skin cells to produce reactive oxygen species, eicosanoids, proteinases, and cytokines, and inhibition of these mediators is notion to lessen skin damage. proof for this comes from the demonstration that antioxidants such as ascor- bic acid and alpha tocopherol produce photoprotective effects in some in vitro and in vivo studies (Elmets and Mukhtar, 1996; Mukhtar and Elmets, 1996).studies have recommended that green tea polyphenols may bebeneficial in affording protection in opposition to inflammatory responses and against pores and skin-cancer hazard. Topical utility of inexperienced tea polyphenols to mouse pores and skin inhibits 12– zero-tetradecanoylphor- bol-thirteen-acetate and different skin tumor-promoter-precipitated induc- tion of protein and mRNA expression of the seasoned-inflammatory cytokines interleukin (IL)-1 α and TNF-α. skin utility of green tea polyphenols inhibits UV-radiation-triggered neighborhood and systemic suppression of contact allergic reaction and edema responses in C3H/chicken mice. in many in vitro studies, inexperienced tea polyphenols or crude extracts of green tea have additionally proven preventive outcomes in systems taken into consideration important in inflam- matory and carcinogenic techniques (Ahmad et al., 1997; Kati- yar and Mukhtar, 1996).

The relevance of the vast in vitro and in vivo laboratory facts on negative consequences as a result of solar UVB in human skin isn't always clean. This facts may be derived either based totally on epidemiological studies in a excessive-risk population, or based totally on using quick-term assays with noninvasive strategies and ac- ceptable protocols in human volunteers. currently, we assessed the protecting effect on skin of the utility of inexperienced tea polyphenols in opposition to UV-prompted erythema in human volun- teers. on this observe, a polyphenolic fraction acquired from green tea turned into applied, in exceptional strengths, at the untanned backs of everyday volunteers. Thirty min later the sites had been exposed to twice the minimum erythemogenic dose (MED) of UV radiation from a solar simulator. websites pretreated with inexperienced tea poly- phenols exhibited considerably much less erythema when compared to vehicle-handled websites. The photoprotective outcomes of green tea polyphenols have been depending on the electricity of the dose implemented, with maximum protection located from two hundred µl of a five% solution. In time route research, the green tea polyphenol- mediated cutaneous photoprotective effect changed into obtrusive, even whilst UV irradiation turned into behind schedule for many h. the protective results lasted for at the least 72 h, as a result indicating a surprisingly lengthy-time period safety, especially in opposition to continual low-dose en- vironmental insult. skin software of green tea polyphenols to human volunteers also led to giant protection against 2 MED-triggered enhancement of sunburn-mobile forma- tion and depletion of CD1 a+ Langerhans mobile density (Mukhtar et al., 1996). In additional studies, we investigated (Katiyar et al., 1999) whether or not or no longer the topical utility of EGCG could guard in opposition to UVB-triggered unfavorable outcomes in human pores and skin. in this observe, we assessed the impact of EGCG remedy on inhibition of UVB-prompted infiltration of leukocytes (macrophage/neu- trophils), a potential source of era of ROS and prosta- glandin (PG) metabolites, which play a important role in pores and skin- tumor promotion in multistage pores and skin carcinogenesis. Human topics were exposed to UVB radiation (at 4 MED doses) on sun-protected skin, and pores and skin biopsies or keratomes had been ob- tained 24 h or 48 h later. We found that topical application of EGCG (3 mg/2.5 cm2) before UVB exposure to human skin drastically blocked UVB-caused infiltration of leukocytes and reduced myeloperoxidase activity. The infiltration of leu-kocytes is considered the main source of technology of ROS. within the identical set of experiments, we located that the topical software of EGCG before UVB publicity decreased UVB- precipitated erythema. In extra experiments, we determined that EGCG treatment earlier than UVB exposure produced substantially decrease PG metabolites, particularly PGE2, in the epidermal microsomal fraction of the pores and skin, compared to non-pretreated pores and skin. Histological examination of pores and skin found out that EGCG- pretreated and UVB-exposed human skin contained fewer dead cells inside the dermis when in comparison to non-pretreated, UVB- exposed skin. these facts exhibit that EGCG has the capability to dam the UVB-precipitated infiltration of leukocytes and the following technology of ROS in human skin. this could be accountable, at least in part, for the 07b031025f5f96dfa8443f843db463b6 results of green tea. based on the work described above, it is tempting to indicate that using GTP in cosmetic

arrangements may be a singular approach for preventing the negative results related to UV radiation in people. it's far of hobby that many 56eab742a58e3778cd3080d979724cd6 cosmetics advertised with the aid of small agencies, as well as less costly lines of cosmetics marketed by using the call brand organizations, are supplementing their merchandise with inexperienced tea extracts.

Modulatory Effects of Green Tea for Cancer Chemotherapy

A current look at (Sadzuka et al., 1998) has shown that inexperienced tea also can modulate the efficacy of cancer-chemotherapeutic capsules in any such way that it increases the medicine' efficacy. in this study, the oral administration of green tea superior the tumor- inhibitory outcomes of doxorubicin on Ehrlich ascite carcinomas implanted in CDF1 and BDF1 mice. green tea treatment re- sulted in an improved availability of doxorubicin in tumor, but not in regular tissue. If verified inside the human population, these observations may also have relevance to cancer chemotherapy

Closure and Prospective Pathways

seeing that 1990, approximately ten million new cases of most cancers had been recognized and 4 million most cancers-related deaths have came about. it is believed that almost one 1/3 of the cancers are as a result of dietary conduct and the manipulation in weight-reduction plan is in- creasingly being identified as a potential method in opposition to cancer (Katiyar and Mukhtar, 1996; Weisburger, 1996). the use of tea, especially green tea, as a cancer chemopreventive agent has handiest been preferred in the last ten years. inexperienced tea is a popularly consumed beverage, is notably less expensive and non-poisonous, and has been shown to afford protection in opposition to many most cancers kinds. The epidemiological in addition to laboratory studies have proven an inverse affiliation of inexperienced tea con- sumption with the development of positive cancer kinds. Al- although compelling evidence is now available that shows the preventive capacity of green tea in opposition to most cancers, a clear beneath- status of the mechanisms associated with its movement is some distance from entire. A entire understanding of the molecular mech- anism(s) involved with the anti- carcinogenic efficacy of greentea polyphenols may be beneficial in devising higher chemopre- ventive strategies against most cancers.

In view of the to be had information from laboratory and epidemi- ological research, medical trials are now warranted to assess the usefulness of inexperienced tea and the polyphenolic antioxidants present therein. Vastag (1998) realized that a cup of tea seasoned- vides an antioxidant enhance which can guard in opposition to several most cancers sorts, and that those tea antioxidants are lots stronger than vitamins C and E in their capability to scavenge probably carcinogenic unfastened radicals. it is essential to empha- size here that section I scientific Trials to evaluate the possible efficacy of formulated inexperienced tea, in patients with superior solid tumors.

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