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RE: Green Tea Offers Health-promoting Benefits


Scientific evidence supports the health benefits of green tea (Camellia sinensis). Green tea catechins, especially epigallocatechin-3-gallate (EGCG), have exhibited anticancer, antiobesity, antiatherosclerotic, antidiabetic, antibacterial, antiviral, and anti-dental caries properties. These authors conducted a review of the health-promoting effects of green tea, mainly focusing on published studies of the past 20 years. Included in this review are 103 cited articles.

Several animal and cell experiments have focused on the anticancer activity of green tea and its catechins. Among their studies, the authors have reported that EGCG inhibited the adhesion of cancer cells to endothelial cell layers\(^1\) and that EGCG prevented cancer cells from attaching to fibronectin\(^2\) and laminin,\(^3\) two components of the endothelial basement membrane. These findings are regarded as supporting green tea’s antimetastatic effect.

Inducing apoptosis (programmed cell death) in tumor cells is a primary mechanism of action of certain antitumor drugs. These authors observed that EGCG induced apoptosis in human lymphoma cells, as evidenced by the formation of apoptotic bodies and degradation of DNA into nucleosomal units.\(^4\)

Noting that epidemiological and intervention studies are important to reveal the anticancer effects of green tea and catechins, the authors cite several studies with conflicting results, mentioning that the discrepancy may arise from factors such as differences in the type of tea consumed, in cancer etiology, and in confounding lifestyle and genetic factors (See HC 051235-454).

In one of the cited clinical trials, the investigators reported on their study\(^5\) to assess the safety and efficacy of green tea catechins for the chemoprevention of prostate cancer in individuals with high-grade prostate intraepithelial neoplasias. After one year of daily treatment of three capsules containing 200 mg of catechins, only one tumor was
diagnosed among the 30 catechin-treated men, whereas nine cancers were found among the 30 men treated with placebo.

One study reported that green tea rich in catechins inhibited the action of galactosamine (known to induce hepatic injury) in rats.6 The authors also reported that green tea prevented liver fibrosis after hepatic injury induced by galactosamine.7

Research into the relationship between green tea and obesity-related insulin resistance syndrome has shown that green tea enhances insulin activity in vitro, enhances insulin sensitivity in human subjects and in rats, and reduces triglyceride levels in mice. A large-scale retrospective cohort study revealed that consumption of green tea and coffee, as well as total caffeine, was associated with a reduced risk for type 2 diabetes mellitus.8

Research also indicates that the ingestion of green tea and tea catechins leads to reduced body fat and offers antiatherosclerotic effects. A population-based, prospective cohort study among 40,530 Japanese adults over 11 years indicated that green tea consumption was inversely associated with mortality due to all causes and particularly to cardiovascular disease.9

Because of the evidence supporting its anti-inflammatory and neuroprotective properties, EGCG may offer help for young, disabled adults with inflammatory brain disease. Supporting the beneficial effects of green tea catechins on brain functions, an epidemiological study indicated that consumption of green tea is associated with a lower prevalence of cognitive impairment in humans.10 With evidence from other human studies and some animal studies, the authors suggest that the "effects on brain function are a very important target for future investigations of green tea."

The authors conclude that epidemiological studies indicate that the intake of green tea contributes to human health promotion and suggest that future clinical intervention studies will provide more convincing evidence for the effects of green tea.

—Shari Henson

References


Referenced article can be found at https://www.jstage.jst.go.jp/article/pjab/88/3/88_3_88/_pdf.