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FILE: • Green Tea (*Camellia sinensis*) • Low-Density-Lipoprotein Concentrations • Atherosclerotic Disease

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RE: Effects of Green Tea on Low-Density-Lipoprotein Concentrations in Healthy Subjects

Hirano-Ohmori R, Takahashi R, Momiyama Y, et al. Green tea consumption and serum malondialdehyde-modified LDL concentrations in healthy subjects. *J Am Coll Nutr*. 2005;24(5):342–346.

The results of prospective studies have shown that flavonoid consumption is associated with a reduction in mortality from coronary artery disease and a reduced risk of myocardial infarction. Flavonoids are found in beverages such as tea (*Camellia sinensis*) and wine (*Vitis vinifera*) and in many fruits and vegetables. In vitro studies have shown that green tea inhibits low-density-lipoprotein (LDL) oxidation, platelet aggregation, and matrix metalloproteinase (MMP) activities, all of which are associated with a reduced risk of atherosclerotic disease (an arteriosclerosis characterized by atheromatous deposits in and fibrosis of the inner layer of the arteries). Despite these positive findings, these specific effects of green tea have not been studied in vivo. Thus, the objective of this study was to investigate the effects of green tea consumption on LDL oxidation, platelet aggregation, platelet thromboxane production, and MMP activities in healthy adults.

In this trial, 22 healthy male nonsmokers aged 32 years (\pm 5 years) were enrolled. The study consisted of a 1-week run-in period, followed by a 2-week period in which the subjects drank 7 cups of water daily, followed by a 2-week period during which the subjects drank 7 cups of green tea daily. A commercially available green tea (sarasara ryokucha; Itoen Co., Tokyo, Japan) was used; 7 cups of this tea provided 542.5 mg of catechins, 108.5 mg of caffeine, and 56.7 mg of vitamin C. The location of the study was not disclosed. Serum malondialdehyde-modified LDL (a marker of LDL oxidation), plasma LDL, platelet thromboxane, plasma total catechin, and MMP concentrations were measured. Platelet aggregation was also evaluated.

The total concentration of plasma catechins decreased significantly (P < 0.05) at the end of the water period and then increased at the end of the green tea period. Plasma LDL

concentrations did not change significantly during the study; however, serum malondialdehyde-modified LDL concentrations decreased significantly (P < 0.05) from 84 \pm 45 IU/L at the end of the water period to 76 \pm 40 IU/L at the end of the green tea period. Furthermore, the ratio of malondialdehyde LDL to LDL cholesterol decreased significantly (P < 0.02) from 0.74 \pm 0.21 at the end of the water period to 0.65 \pm 0.20 at the end of the green tea period. No significant changes in platelet aggregation, platelet thromboxane, or MMP activities were observed after green tea consumption.

The daily consumption of 7 cups of green tea daily for 2 weeks significantly decreased serum malondialdehyde LDL concentrations but had no significant effect on platelet aggregation, platelet thromboxane production, or plasma MMP activities. The lack of a significant effect on the 3 variables in question does not support previous in vitro results that suggest a protective effect of green tea consumption against cardiovascular disease. These results "suggest that the ordinary consumption of green tea in Japanese may inhibit LDL oxidation in vivo." The authors state that one limitation of the study was that it was sequential in design, which made it impossible to separate period effects from treatment effects, but do not provide details regarding this limitation. Another limitation was that healthy subjects were used. Thus, the authors recommend that a future study be conducted in subjects at high-risk of heart disease and with the use of a crossover design to clarify whether green tea has an inhibitory effect on LDL oxidation in vivo.

—Brenda Milot, ELS

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