GUEST EDITORIAL

Has the tea been ruined?

Medicinal effects of tea (*Camellia sinensis*) were noted by the Chinese as early as 350 AD (IARC, 1991). More recent research has suggested that regular tea consumption may influence cardiovascular disease and lower circulating lipid levels (Thelle, 1995). These findings are based on Japanese reports of a cholesterol-reducing effect of drinking green tea in cross-sectional studies of their populations (Imai & Nakachi, 1995; Kono *et al.* 1996), animal models of hypercholesterolaemia, and Dutch reports of less coronary heart disease among tea drinkers in a cohort study of adult men (den Hertog *et al.* 1993). These observational population studies are vulnerable to confounding by lifestyle. People who drink more tea may be different in other significant ways from people who drink less tea within the same population (Schwartz *et al.* 1994). Well-designed human experiments are necessary to isolate the tea effect and attribute it exclusively to this drink, the tea leaves themselves or some active component therein.

Bingham *et al.* (1997) have taken the first step towards testing the effect of tea drinking on known cardiovascular disease risk factors, including cholesterol, LDL-cholesterol and blood pressure. In this edition of the Journal Sheila Bingham and her co-investigators report on the effects on lipid and lipoprotein levels of a black tea *v.* placebo intervention with a cross-over design. In this study a heavy-tea-drinking population, with mean intakes of 1·4 litres of black tea per d, was asked to drink six or more cups of tea daily or asked to abstain for a duration of 4 weeks. The group was studied to determine changes in their HDL-, LDL- and total cholesterol and triacylglycerol levels during the intervention. The authors report no significant reduction in serum levels of cholesterol or LDL-cholesterol after the tea intervention. How is this to be understood in light of the epidemiological findings? Was it the right population, the right dose, the right substance? Did the subjects adequately adhere to the intervention?

The researchers cleverly devised a means by which they could monitor adherence to the minimal dose of tea by using *p*-aminobenzoic acid (PABA)-coated teabags and examining urinary levels from 24 h collections. This led to the concern that fifteen of sixty-five subjects were consuming much less than suggested. The low PABA collection in a subgroup might reflect one of two things: reduced consumption or incomplete collection of a 24 h urine sample. The result of the former would be an underestimation of effect due to less tea drinking than expected. Incomplete collections would probably not affect the outcome as the urine collection was also imperfect in the placebo group. Of equal interest in the interpretation of these findings is the adherence to the other arm of the intervention. Was abstinence truly achieved in this group of habitual tea drinkers? A lack of effect could equally be explained by continued tea drinking 'on the sly' during the placebo phase of the trial. Measurements of catechin levels in blood or urine would have been a valuable reassurance of adherence to the intervention. Given that adequate compliance was achieved, there are at least three other alternative explanations of the findings presented.

A close look at the data shows a mild change in lipid levels during the trial. Cholesterol levels increased 1–2% during the placebo arm of the trial. Could it be that placing tea drinkers on tea does not elicit a change in cholesterol, but removing their tea for a long enough period of time might result in an increase? Stated differently, tea drinkers might already have an induced reduction in serum cholesterol and LDL levels which only slowly

returns to 'normal'. The magnitude of change here is, however, very small and would not be of importance unless it reflects other important issues: lack of adherence, an extremely slow return to greater effect levels, or a genetic difference in susceptibility to the influence of tea which may be present in one subset and absent in another. A longer intervention would be needed to test this. Also, a definitive trial of black tea might be better conducted by slipping tea into the diets of non-drinkers and monitoring their lipid levels rather than by trying to wean tea drinkers away from their habit. The susceptibility of the population, the sufficiency of the dose and the appropriateness of the substance should be considered.

Perhaps it would be helpful to remember the wisdom of George Mikes (Mikes, 1964) who described some differences in the drink between Japan, where effects are reported, and the UK. He wrote 'The trouble with tea is that originally it was quite a good drink. So a group of the most eminent British scientists put their heads together and made complicated biological experiments to find a way of spoiling it. To the eternal glory of British science, their labor bore fruit. They suggested that if you do not drink it clear or with lemon, but pour a few drops of cold milk into it and no sugar at all, the desired object is achieved. Once this refreshing, aromatic, Oriental beverage was successfully transformed into colorless and tasteless gargling-water, it suddenly became the national drink . . . '.

Japanese differ from the British in a number of ways. They do, for one, prefer green tea, which is three times as rich in catechins (75% of dry weight compared with 25% in black tea). Genetic differences governing lipoprotein responsiveness to the active ingredients in tea may explain some of the difference in response. And, as noted by Mikes (1964), other cultures do not add milk to the drink. The dose however can not be questioned. The British have been drinking tea since 1657, and rank third worldwide in their average *per capita* consumption (2·8 kg/person per year; IARC, 1991). If the existing dose in the UK, which was the same as used in the trial, is not adequate to detect an effect, there is little reason to believe that a black tea effect is detectable elsewhere. *Per capita* consumption of tea in the Netherlands, for example, is only a quarter as high. If there is a linear black tea effect it should be detectable at these consumption levels provided in this trial.

What we have learned from this study is that black tea may serve as a mild laxative, softening stool consistency and perhaps reducing somewhat the duration of bowel movement. Whether there is an effect of black tea on cardiovascular disease development in human populations remains to be confirmed. With respect to lipid-lowering effects, experiments on non-drinkers are needed, biomarkers of adherence should be used, and the effect of both green and black tea drinking tested. In the meanwhile it appears prudent to keep up this 340-year-old habit or switch to green tea.

LENORE KOHLMEIER
Departments of Nutrition and Epidemiology
Schools of Public Health and Medicine
Office 2105e, McGavran-Greenberg
The University of North Carolina at Chapel
Hill, Chapel Hill, NC 27599-7400, USA

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