Effects of Black Tea on Blood Pressure: A Randomized Controlled Trial

High blood pressure (BP) is a leading risk factor contributing to the global burden of disease. Small changes in BP due to dietary modification may have a significant impact on the prevalence of hypertension and risk of cardiovascular disease.1

Tea is a popular beverage worldwide and is usually the major source of population flavonoid intake, often providing more than half of total intake.2 There is mounting evidence that tea and its flavonoids can make an important contribution to vascular health.3 However, the effects of regular consumption of black tea on BP remain unclear.

Our objective was to assess the effects of regular black tea consumption (3 cups/d) for 6 months on 24-hour ambulatory BP. We found that black tea consumption resulted in significantly lower systolic BP (SBP) and diastolic BP (DBP).

Methods. A randomized placebo-controlled double-blind 6-month parallel designed trial was performed. Men and women aged 35 to 75 years, who were regular tea drinkers, with a body mass index of 19 to 35 (calculated as weight in kilograms divided by height in meters squared) and a daytime ambulatory SBP between 115 and 150 mm Hg at screening were recruited from the general population.

Following randomization, participants followed a low-flavonoid diet during a 4-week run-in period and throughout the 6-month intervention. During run-in, participants consumed 3 cups/d of regular leaf tea. During the 6-month intervention, participants consumed 3 cups/d of either 1493-mg powdered black tea solids containing 429 mg of polyphenols and 96 mg of caffeine (tea), or a placebo matched in flavor and caffeine content, containing no tea solids. Intake of regular leaf tea ceased during this time. The primary outcome of 24-hour ambulatory BP,4 biochemical measurements, and dietary assessments were performed at baseline, 3 months, and 6 months.

Intent-to-treat analysis was performed by a biostatistician blinded to treatment allocation. A prespecified statistical analysis plan, which was finalized prior to breaking the randomization code, was followed. The effect of black tea on baseline-adjusted BP at 3 and 6 months was assessed using linear mixed models in STATA 11 (StataCorp) using “xtmixed” and “margins” commands. The trial was approved by the University of Western Australia Ethics Committee and registered at the Australian New Zealand Clinical trials Registry (see eAppendix [eMethods] for additional detail of study design and statistical analysis; http://www.archinternmed.com).

Results. The trial profile is provided in the eFigure. The intent-to-treat population included 95 participants randomized to the trial who completed baseline. There were no significant differences at baseline between the placebo and tea groups, respectively, in numbers of men and women (18 men/31 women; 15 men/31 women, respectively), mean (SD) age (56.3 [10.6] years; 56.9 [10.7] years, respectively), body mass index (25.2 [3.3]; 25.0 [3.7], respectively), and 24-hour SBP/DBP (121.4 [9.7]/72.9 [6.6] mm Hg; 121.2 [9.0]/71.5 [7.2] mm Hg, respectively). Energy and nutrient intakes, urinary sodium and potassium excretion, and body weight were not different between groups at baseline and were not significantly altered during the 6-month intervention.

The unadjusted mean hourly 24-hour ambulatory BP data are summarized in the Figure. Compared with placebo, regular ingestion of black tea over 6 months resulted in lower 24-hour SBP and DBP. Net effects (mean difference [95% CI]) for 24-hour SBP were −2.7 (−4.7 to −0.8) mm Hg (P = .006) at 3 months and −2.0 (−4.0 to −0.0) mm Hg (P = .05) at 6 months. Net effects for 24-hour DBP were −2.3 (−3.6 to −0.9) mm Hg (P < .001) at 3 months and −2.1 (−3.5 to −0.7) mm Hg (P = .003) at 6 months. Significant differences in BP were also observed for daytime and nighttime BPs separately, but effects on the overall 24-hour BP were mainly driven by daytime BP (Figure and eTable).

Comment. Regular consumption of 3 cups/d of black tea over 6 months, supplying approximately 429 mg/d of polyphenols, resulted in lower SBP and DBP of between 2 and 3 mm Hg. A large proportion of the general population have BP within the range included in this trial, making results of the trial applicable to individuals at increased risk of hypertension. Most previous short-term randomized controlled trials of regular black tea consumption have not shown significant effects on BP.3 The main limitation of these trials is that they were generally underpowered to detect small but clinically important effects on BP.3 It is also possible that longer-term regular consumption of black tea is needed for significant falls in BP to become apparent.

There are a number of potential mechanisms for BP lowering by black tea. Hypertension and endothelial dysfunction are integrally related, and endothelial dysfunction may be an early marker for BP changes. A recent meta-analysis found that tea consumption can improve endothelial function,6 and we have previously shown that tea flavonoids can augment nitric oxide status and reduce plasma concentrations of endothelin-1.7 This could contribute to reduced vascular tone and lower BP. An-
other possible mechanism involves effects of tea flavonoids to alter body weight and/or visceral fatness. A recent meta-analysis suggests that green tea and its flavonoids—many of which are structurally similar to black tea flavonoids—together with caffeine can reduce body weight and abdominal fatness. Although in the present study we found no significant effect on body weight, effects on abdominal fatness remain a possible explanation for the observed effects on BP.

In conclusion, our study has demonstrated for the first time to our knowledge that long-term regular consumption of black tea can result in significantly lower BPs in individuals with normal to high-normal range BPs. At a population level, the observed differences in BP would be associated with a 10% reduction in the prevalence of hypertension and a 7% to 10% reduction in the risk of cardiovascular disease. Therefore, given the high prevalence of hypertension worldwide and the importance of hypertension as a risk factor for cardiovascular and total mortality, these findings have important public health implications.

Author Affiliations: School of Medicine and Pharmacology, University of Western Australia, and the WAIMR Centre for Food and Genomic Medicine, Perth, Western Australia, Australia (Drs Hodgson, Puddey, and Croft and Ms Scott); Discipline of General Practice, Flinders University, Adelaide, South Australia, Australia (Dr Woodman); and Unilever Research and Development, Vlaardingen, the Netherlands (Drs Mulder and Fuchs).

Correspondence: Dr Hodgson, School of Medicine and Pharmacology, Royal Perth Hospital Unit, GPO Box X2213, Perth 6847, Australia (Jonathan.Hodgson@uwa.edu.au).

Author Contributions: Dr Hodgson had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The primary data analysis was performed independent of Unilever Research and Development by Dr Woodman, employed by Flinders University, Adelaide, Australia, who followed the prespecified statistical analysis plan. He did not receive compensation of funding to perform the analysis. Study concept and design: Hodgson, Puddey, Mulder, Fuchs, Scott, and Croft. Acquisition of data: Hodgson, Scott, and Croft. Analysis and interpretation of data: Hodgson, Puddey, Woodman, Mulder, Fuchs, and Croft. Drafting of the manuscript: Hodgson and Scott. Critical revision of the manuscript for important intellectual content: Hodgson, Puddey, Woodman, Mulder, Fuchs, Scott, and Croft. Statistical analysis: Hodgson, Woodman, Mulder, and Fuchs. Obtained funding: Hodgson, Puddey, Mulder, Fuchs, and Croft. Administrative, technical, and ma-
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Online-Only Material: The eAppendix, eTable, and eFigure are available at http://www.archinternmed.com.


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Validation of 7 Type 2 Diabetes Mellitus Risk Scores in a Population-Based Cohort: CoLaus Study

One of the challenges for public health in the coming years is the expected increase of type 2 diabetes mellitus (T2DM) prevalence and its resulting health burden and costs. For the physician, although recommendations regarding who to screen for T2DM are available, the application of a validated risk score would lead to an extra cost of US$ 12.02 per screened patient relative to the FINDRISC score. The performance of the 7 T2DM risk scores is given in the Table. Most risk scores had a high AROC, specificity, and negative predictive value, while their sensitivity and positive predictive values were low.

Methods. Seven T2DM risk scores were selected in the present study. Four were based on clinical data: the 10-year risk score from Kahn et al10 (Kahn clinical); the 9-year risk score from Balkau et al11; the prevalent undiagnosed diabetes risk score from Griffin et al12; the Finnish Type 2 Diabetes Risk Score (FINDRISC), which has been developed in 2 cohorts followed for 5 and 10 years; and finally the risk score from the Swiss Diabetes Association, available online,7 which is actually adapted from FINDRISC. The 2 remaining risk scores were based on the association of clinical and biological data: the 10-year risk score from Kahn et al10 (Kahn clinical + biologic) and the 8-year risk score from Wilson et al11. We used the thresholds provided by the authors, and each score had its area under the receiver operating characteristic curve (AROC), sensitivity, specificity, and negative and positive predictive values assessed. We tested these scores in 3060 nondiabetic participants from Lausanne, Switzerland (44.6% men; mean [SD] age, 52.6 [10.6] years), followed up for 5 years (study period, 2003-2011).9 Incident diabetes was defined as fasting plasma glucose level greater than or equal to 126.13 mg/dL (to convert to millimoles per liter, multiply by 0.0555) and/or presence of oral hypoglycemic or insulin treatment.

Results. A total of 169 patients (5.5%) developed T2DM during follow-up. Compared with participants who did not develop T2DM, they were more frequently male (69.8% vs 43.1%); were older (mean [SD] age, 57.1 [9.4] vs 52.3 [10.6] years); had a higher frequency of family history of T2DM (31.4% vs 19.3%) (all P < .001); and had a higher resting heart rate (69 [10] vs 67 [9] beats/min [P < .05]). They practiced less leisure-time physical activity (45.6% vs 60.3%); had higher body mass index (29.0 [3.9] vs 23.1 [4.0] [calculated as weight in kilograms divided by height in meters squared]), waist circumference (100.3 [10.9] vs 86.8 [12.2] cm), and fasting plasma glucose (110.45 [9.37] vs 95.32 [9.37] mg/dL), triglyceride (189.38 [184.07] vs 111.50 [79.65] mg/dL [to convert to millimoles per liter, multiply by 0.0113]), and uric acid (6.03 [1.32] vs 5.11 [1.35] mg/dL [to convert to micromoles per liter, multiply by 59.485] levels; and had lower high-density lipoprotein cholesterol levels (53.28 [13.51] vs 64.09 [16.60] mg/dL [to convert to millimoles per liter, multiply by 0.0259]) (all P < .001). The performance of the 7 T2DM risk scores is given in the Table. Most risk scores had a high AROC, specificity, and negative predictive value, while their sensitivity and positive predictive values were low.

Comment. Most variables included in the risk scores were significantly different between participants who developed T2DM and those who did not, which confirms their prognostic role. The best results were obtained by the Kahn clinical + biologic risk score. However, a risk score based on simple clinical data (FINDRISC) also had a high AROC, which could be more convenient regarding health costs and acceptability by patients. Indeed, using data from our hospital, applying the Kahn clinical + biologic risk score would lead to an extra cost of US$ 12.02 per screened patient relative to the FINDRISC score.

Our study has several limitations. Follow-up time was limited to 5 years; still, our findings are in agreement with the performances reported in the original studies, suggesting that our results should also be reliable after a 10-year follow-up. Some factors such as fruit consumption and second-degree familial history could not be assessed in this study owing to lack of information; although we corrected for such missing data, it is possible that the performance of the corresponding risk scores might have been reduced. Still, one of these risk scores (FINDRISC) ranked second best in our study, suggest-