Effect of Black Tea on Aortic Stiffness

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Background: We have shown that caffeine increases acutely arterial stiffness. Tea is a widely consumed beverage, however, its effect on arterial stiffness has not been defined. Aortic stiffness is an important prognosticator of cardiovascular risk.

Methods: We studied 12 healthy volunteers (33±5 years) in a randomized, single-blind, crossover fashion. The subjects were studied on 3 separate occasions: a) 6 g of black tea, b) 176 mg of caffeine alone (the amount contained in 6 gr of tea) and c) placebo. Carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness using a validated automated, non-invasive device (Complior®).

Results: Tea had a biphasic effect on aortic stiffness. Initially it led to a marginal (P=0.07) increase in PWV, which, however, rapidly disappeared. In contrast, caffeine alone led to a sustained increase in PWV (P<0.05, figure). The reversal of aortic stiffness increase with tea after the initial 30 minutes can be attributed to a beneficial effect of tea flavonoids that peak later than caffeine. Pressures were increased in a similar manner both with tea and caffeine (systolic: by 8.8 and 7.8 mmHg respectively, P<0.001 for both).

Conclusions: Tea shows a tendency to increase initial aortic stiffness due to the caffeine that it contains. However, this effect is promptly counterbalanced by a beneficial effect of tea flavonoids. This finding provides valuable insights for the effects of tea consumption on the cardiovascular system.

Influence of Coenzyme Q10 and Cerivastatin on the Pharmacokinetics of Clopidogrel and Its Active Metabolite in Human Plasma

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Background: Thromboxane synthesis inhibitors reduce platelet aggregation by increasing the level of the prostacyclin/prostanoid metabolite 6-ketoPGF1α and by decreasing thromboxane A2 levels. The effects of cerivastatin on these indexes of platelet inhibition were studied.

Methods: Ten healthy volunteers (9 men, 1 women, age 25 – 48) received a single oral dose of 1 mg of cerivastatin and 300 mg of clopidogrel. Cmax, tmax and t1/2 (mean ± SEM) plasma concentrations of clopidogrel were 7.0 ± 0.8% in OSA vs 6.5 ± 1.3 in controls (p=0.73). Percent changes of brachial artery diameter to nitroglycerin were similar in both groups (15.0 ± 6.0% in OSA vs 15.3 ± 6.9% in controls; p=0.12), thus confirming the absence of structural abnormalities of conduit-vessels in OSA patients.

Conclusions: We conclude that in otherwise healthy patients with OSA, in the absence of potential confounding factors that would affect measurements of brachial artery endothelial function, there is no evidence of impaired endothelial function in conduit-vessels.

Presentation of Brachial Artery Endothelial Function in Otherwise Healthy Patients With Obstructive Sleep Apnea

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Background: Obstructive sleep apnea (OSA) has been implicated in several cardiovascular disease conditions including hypertension, heart failure and cerebrovascular disease. Impaired endothelial function is thought to be involved in risk for future cardiovascular disease in OSA. We tested the hypothesis that otherwise healthy OSA patients, in the absence of any confounding factors, have impaired brachial artery endothelial function.

Methods: We studied 16 newly diagnosed, never treated OSA patients (age: 44.7±3.7 years; apnea-hypopnea index: 45±10 events/hour) and 16 matched normal controls (age: 43.4±2.7 years; apnea-hypopnea index: 2±4 events/hour). All subjects were on no medications and free of any other diseases. None of the subjects smoked and both patients and controls had similar body mass index, blood pressure, fasting glucose and plasma lipid levels. Controls underwent complete polysomnography to exclude occult OSA. Conduit-vessel endothelial function was evaluated by high-resolution ultrasound of the brachial artery. Brachial artery diameter was measured at baseline and after five minutes of ischemia induced by cuff inflation as a measure of endothelium-dependent vasodilation, and after sublingual administration of nitroglycerin, endothelin-independently vasodilated.

Results: Data are presented as mean ± standard error. Baseline brachial artery diameters were similar in OSA and control subjects (4.34 ± 0.1 mm in OSA and 4.04 ± 0.1 mm in controls; p=0.12). Percent changes of brachial artery diameter in response to ischemia were similar in both groups (7.9 ± 0.8% in OSA vs 6.5 ± 1.3 in controls; p=0.73). Percent changes of brachial artery diameter to nitroglycerin were also similar in both groups (15.0 ± 1.8% in OSA vs 15.3 ± 1.3% in controls; p=0.91), thus confirming the absence of structural abnormalities of conduit-vessels in OSA patients.

Conclusions: We conclude that in otherwise healthy patients with OSA, in the absence of potential confounding factors that would affect measurements of brachial artery endothelial function, there is no evidence of impaired endothelial function in conduit-vessels.

Preservation of Brachial Artery Endothelial Function in Otherwise Healthy Patients With Obstructive Sleep Apnea

Maria E. Otto, Rodrigo Bello Barretto, Anna Svatkova, Simone Santos, Kevin A. Bybee, Bipy Khandheria, Viren K. Somers, Mayo Clinic, Rochester, MN

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Conclusions: We conclude that in otherwise healthy patients with OSA, in the absence of potential confounding factors that would affect measurements of brachial artery endothelial function, there is no evidence of impaired endothelial function in conduit-vessels.