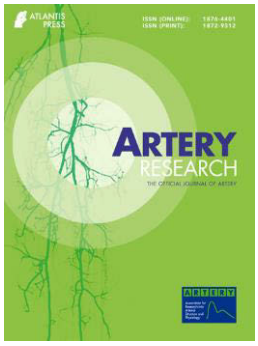

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P90: POSITIVE EFFECTS OF ANTIHYPERTENSIVE TREATMENT ON AORTIC STIFFNESS IN THE GENERAL POPULATION

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work, and increased subendocardial viability index in supine and upright positions ($p < 0.01$ for all). Stroke volume was increased in the supine ($\sim$11 ml, $p < 0.01$) but not in the upright position, while upright ($\sim$1 l/min, $p < 0.01$) but not supine cardiac output was significantly reduced. Upright increase in systemic vascular resistance was amplified after bisoprolol ($p < 0.05$). Pulse pressure amplification was reduced especially in the upright position (supine reduction 10%, upright reduction 20%). Aortic augmentation index, augmentation pressure and pulse pressure were not changed in the supine position, but were increased in the upright position (from 7 to 20%, 3 to 7 mmHg, 28 to 35 mmHg, respectively, $p < 0.01$ for all).

Conclusions: Bisoprolol decreased central and peripheral blood pressure in male subjects with grade I to grade II hypertension, but central blood pressure was reduced less efficiently than peripheral blood pressure. Importantly, the harmful influences of bisoprolol on central pulse pressure and pressure wave reflection were especially observed in the upright position.

P90
POSITIVE EFFECTS OF ANTIHYPERTENSIVE TREATMENT ON AORTIC STIFFNESS IN THE GENERAL POPULATION

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Aortic stiffness is strongly related to age and mean arterial pressure (MAP). We investigated whether antihypertensive treatment modulates the association of the aortic pulse wave velocity (PWV) with age and with MAP in the general population. In the Czech post-MONICA, we measured the PWV in 735 subjects (mean age 61.2 ± 7.8 years, 54.1% women, 44.3% on antihypertensive medication). We used a linear regression model to assess the effect of treatment on the PWV.

The independent covariates in our analysis included sex, age, MAP, body mass index, plasma glucose, low-density lipoprotein cholesterol, smoking and observer. The patients receiving treatment were older (64.1 ± 6.7 vs. 58.9 ± 7.8 years), had higher systolic blood pressure (135.9 ± 16.2 vs. 130.1 ± 16.5 mm Hg) and had higher pulse wave velocity (9.1 ± 2.2 vs. 8.2 ± 2.1 m s 1; P for all o0.0001) than untreated subjects.

After adjustment for MAP, the use of treatment modified the association between age and the PWV (regression equations, treated patients 9.68—0.009 age vs. untreated subjects 6.98 þ 0.020 age, difference of regression slopes, F $^{1}/_{4}$ 11.2; P $^{1}/_{4}$ 0.0009). In analyses adjusted for age, treatment was associated with a smaller increase of the PWV with MAP (treated patients 9.63—0.006 MAP vs. untreated subjects 7.18 þ 0.010 MAP, F $^{1}/_{4}$ 10.70; P $^{1}/_{4}$ 0.0001). These results were driven primarily by subjects whose blood pressure was below 140/90 mm Hg.

In the cross-sectional analysis from a random sample of the general population, antihypertensive treatment was associated with a less steep increase in the PWV with age and the mean arterial pressure.

P91
GREEN TEA EXTRACT REDUCES LIPID PROFILE, PERCENTAGE OF AORTIC AUGMENTATION INDEX AND INCREASES SOLUBLE RAGE CONCENTRATIONS IN NORMOTENSIVE PATIENTS WITH TYPE 2 DIABETES MELLITUS: A RANDOMIZED, DOUBLE-BLINDED, AND PLACEBO-CONTROLLED TRIAL

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Background: Type 2 diabetes mellitus is associated with premature atherosclerosis and arterial stiffening by an accumulation of advanced glycation end-products in vessel wall (1). Green tea polyphenols are considered a cardioprotective substance and may be used as an adjuvant for diabetes treatment, because its ability to stimulates the soluble RAGE secretion (2). There is no clinical evidence of the effect of green tea extract administration on metabolic parameters, arterial stiffness and the soluble RAGE expression.

Material and Methods: A double-blind, placebo-controlled, randomized clinical trial in normotensive patients with type 2 diabetes mellitus was conducted to identify the effect of green tea extract on arterial stiffness, metabolic and anthropometric parameters and on soluble RAGE (sRAGE) with the S100A1 ligand.

Results: We included 20 subjects, there was no difference between groups at baseline. There was a decrease in the green tea extract group on aortic augmentation index (21.12 ± 8.9 to 18.07 ± 9.7, $p = 0.045$), total cholesterol (203.9 ± 37.6 to 176.9 ± 25.9 mg/dl, $p = 0.019$) triglycerides (202.6 ± 146.9 to 123.2 ± 64.8 mg/dl, $p = 0.023$) and an increase in sRAGE (1358.5 ± 390.0 to 1281.1 ± 369.7 $p = 0.052$).

Table 1. Effect of 12 weeks of Green tea extract intervention or placebo on circulating parameters.

	GTE		PLACEBO		P
	Basal n = 10	Final n = 10	Basal n = 10	Final n = 10	
Fasting Glucose, mg/dl	169.9 ± 92.3	123.9 ± 69.8	168.1 ± 49.7	171.3 ± 39.8	0.089
Creatinin mg/dl	0.75 ± 0.2	0.81 ± 0.14	0.7 ± 0.2	0.78 ± 0.14	0.853
Total Cholesterol mg/dl	203.9 ± 37.6	176.9 ± 25.9	187.9 ± 44.6	216.1 ± 48.2	0.019*
Triglycerids, mg/dl	202.6 ± 146.3	123.9 ± 64.8	159.9 ± 57.0	184.3 ± 93.9	0.023*
HDLc, mg/dl	47.9 ± 7.8	44.9 ± 5.2	48 ± 8.9	46.9 ± 10.2	0.529
LDLC. mg/dl	123 ± 32.8	109.4 ± 25.1	92.3 ± 30.2	111.2 ± 53.3	0.436
TGO, U/ml	25.6 ± 10.1	25.3 ± 7.08	40.7 ± 13.8	44.4 ± 26.8	0.971
TGP, U/ml	23.8 ± 13.6	28.9 ± 11.9	35.4 ± 14.5	44.7 ± 25.4	0.912
TFG, naL/min	119.9 ± 56.3	101.8 ± 23.9	120.6 ± 50.2	102.3 ± 22.7	0.739

Values are arithmetic means ± SE except for mean differences between groups, which have been adjusted for baseline values. Between-group P values reflect the between-group comparison change-scores from Man Whitney U statistic methodology. *Significant ($p < 0.05$) within-group change.

Conclusions: Green tea extract reduces lipid levels, percentage of aortic augmentation index and increases soluble RAGE concentrations in normotensive patients with Type 2 Diabetes.

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