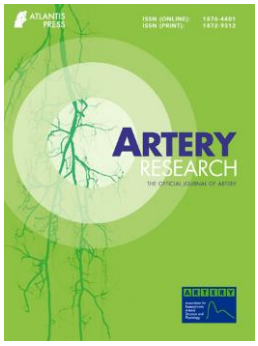

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P3.09: STIFFER ARTERIES IN "HEALTHY" SUBJECTS WITH COMPONENTS OF THE METABOLIC SYNDROME

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indices, smoking and MetS components ($p<0.01$). In contrast, AGEs measured by skin autofluorescence were not associated neither with indices of insulin resistance ($r_{HOMA-IR}$ =-0.04, r_{QUICKI} =0.03) nor PWV (r=0.02). Subjects with impaired vs. normal glucose tolerance had higher PWV (9.33±1.54 vs. 8.67±1.54 m/s) and hsCRP (3.54±3.3 vs. 2.53±2.55 mg/L), but not AGEs (2.11±0.41 vs. 2.17±0.44).
Conclusions: In the middle-aged MetS subjects without diabetes hsCRP but not AGEs measured by skin autofluorescence are related to both altered glucose metabolism and arterial stiffening. Our finding suggests that in early stages of the cardiometabolic disorder prevailing determinant of arterial damage is inflammation, but not tissue glycation.

P3.09
STIFFER ARTERIES IN "HEALTHY" SUBJECTS WITH COMPONENTS OF THE METABOLIC SYNDROME

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Objectives: To compare indices of vascular stiffness and markers of adiposity and inflammation in "healthy" subjects with/without components of the metabolic syndrome (met-x).
Methods: Subjects satisfying ≥1 (≥1 Met-X;) and no criteria (0 Met-X) were recruited (IDF 2006). All were lifelong non-smokers, normotensive, normolipidaemic and normoglycaemic. Augmentation index (AIx) and pulse wave velocity (PWV) were measured using applanation tonometry (Sphygmacor & Vicorder). Fasting leptin, adiponectin, IL-6, TNFα & MCP-1 were measured. Unpaired students t-test and Fischer's exact test was used to detect differences.
Results: Anthropometric, metabolic and haemodynamic indices of the met-x syndrome were significantly higher in the ≥1 Met-X group ($p<0.0001$). AIx and PWV were higher in the ≥1 Met-X group. The adipose related hormones, leptin and adiponectin were higher and lower, respectively, in the ≥1 Met-X group but the pro-inflammatory markers, IL-6, TNFα & MCP-1 were not different (Table 1).

Table 1 Body fat composition, arterial stiffness and humoral adipose/pro-inflammatory markers in subjects with/without early signs of met-x. Results are mean±SD.

	0 Met-X criteria	≥1 Met-X criteria	*P*
n	91	106	
Age (years)	37±10	40±8 years	0.06
Body fat (%)	22.64±6.74	30.44±7.71	<0.0001
AIx (%)	12.83±13.60	19.28±13.34	<0.001
PWV ($m.s^{-1}$)	6.82±0.85	7.14±1.20	<0.05
Leptin ($pg.mL^{-1}.10^{-2}$)	100.39±73.44	168.93±123.94	<0.001
Adiponectin ($pg.mL^{-1}.10^{-2}$)	77.54±41.10	53.91±31.98	<0.001
IL-6 ($pg.ml^{-1}$)	1.59±0.96	2.08±3.01	0.27
TNFα ($pg.ml^{-1}$)	2.98±1.10	3.29±1.50	0.21
MCP-1 ($pg.ml^{-1}$)	214.26±96.85	204.51±80.15	0.55

Conclusion: Subjects with early met-x have stiffer arteries than those with normal metabolic function. These results suggest that premature arterial stiffening may be mediated via hormonal rather than inflammatory mechanisms.

P3.10
GENETIC FACTORS VS CARDIOVASCULAR RISK FACTORS. WHAT IS MORE SIGNIFICANT IN VASCULAR AGING?

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Objective: Telomere length (TL) has been identified as a marker for biological, cardiovascular aging and cardiovascular events risk. Increased arterial stiffness and intima-media thickness (IMT) are the main signs of arterial aging and strong predictors for the development of cardiovascular disease. But the origin of the association between cardiovascular events risk and telomere biology is still unknown. The aim of this study was to determine the role of telomere lenght (a largely inherited component) and conventional cardiovascular risk factors (CVRF) (a largely acquired component) in vascular aging process.
Design: The study group included 146 patients free from established cardiovascular diseases, mean age 51,34 ± 19,02 years. Smoking, arterial hypertension, obesity, dyslipidemia, high fasting glucose level were considered as CVRF.
Methods: TL was assessed by quantitative polymerase chain reaction. IMT was determined by ultrasonography in both left and right carotid arteries. Arterial stiffness was appreciated by aortic pulse wave velocity (PWV) measuring with the help of SphygmoCor (AtCor Medical).
Results are summarized in the table.

	PWV	IMT
TL	r = -0,2657	r = -0,1861
	p = 0,0096	p = 0,0618
CVFR	r = -0,0983	r = 0,2997
	p = 0,264	p = 0,0005

Conclusions: TL has strong correlation with PWV, but not IMT. Opposite, the presence of conventional CVRF are contribute to subclinical atherosclerosis, not arterial stiffness. Thus, age-related changes in the vascular wall has different causes and requires an individual approach to the prevention and treatment.

P3.11
ETHNIC DIFFERENCES IN ARTERIAL WAVEFORM MEASURES IN A LARGE SAMPLE OF ADULTS ENROLLED IN THE VITAMIN D ASSESSMENT (VIDA) STUDY

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Few studies have examined whether central arterial waveform measures vary with ethnicity. We aimed to provide a more comprehensive examination of ethnic differences in several cardiovascular risk factors, which may reveal new ethnic variations. A cross-sectional (baseline) analysis of 5110 adults (2971 M, 2139 F; age range, 50-84 years) from multiple ethnic groups (European/Other, Maori, Pacific, South Asian) participating in a New Zealand clinical trial of the effect of vitamin D supplementation (the ViDA study) on cardiovascular disease events was carried out. Peripheral blood pressure was measured with an Omron T9P oscillometric device. Arterial pressure waveforms were derived from suprasystolic brachial measurement using a Pulsecor R6.5 device, which previously has been shown to yield central pressure measurements highly correlated with those from aortic catheterisation. These were decomposed into forward- and backward-travelling waves and reservoir wave analysis was applied to derive reservoir and excess pressures. Compared to European/Other participants, those in the other three ethnic groups had significantly higher peripheral augmentation index

Table: Preliminary data from the ViDA study.

Measure	Mean (SE)* European/Other (n=2959)	Mean difference (SE)* from European/Other			P-value#
		Maori (n=194)	Pacific (n=254)	South Asian (n=139)	
Brachial BP (mmHg)					
Systolic	139.5 (0.4)	2.9 (1.4)	2.4 (1.2)	-2.1 (1.6)	0.019
Diastolic	76.9 (0.2)	2.0 (0.7)	0.7 (0.7)	-1.5 (0.9)	0.009
Peripheral augmentation Index %	100.6 (0.9)	8.8 (2.9)	3.2 (2.7)	5.8 (3.4)	0.009

*Adjusted for age, sex and BMI; # P-value for variation across all 4 ethnic groups.