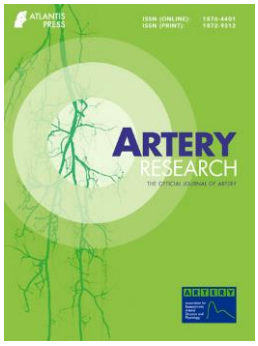

**Artery Research**

**ISSN (Online): 1876-4401** **ISSN (Print): 1872-9312**
**Journal Home Page: https://www.atlantis-press.com/journals/artres**

# P.056: HERITABILITY OF ARTERIAL WALL INTIMA MEDIA THICKNESS IN DIFFERENT VASCULAR BEDS

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**To cite this article**: V. Dzenkeviciute, L. Ryliskyte, A. Laucevicius, Z. Petrulioniene (2007) P.056: HERITABILITY OF ARTERIAL WALL INTIMA MEDIA THICKNESS IN DIFFERENT VASCULAR BEDS, Artery Research 1:2, 65–66, DOI: https://doi.org/10.1016/j.artres.2007.07.113

**To link to this article**: https://doi.org/10.1016/j.artres.2007.07.113

Published online: 21 December 2019

**Conclusions:** Patients with HCV have impaired aortic elastic properties, whereas HBV does not influence aortic stiffness. These findings are important to further characterize the increase of cardiovascular risk in patients with hepatitis C virus seropositivity and to specify the linking role of the adipose tissue-related hormones.

**P.052**
**AN INTERLEUKIN-6 POLYMORPHISM DETERMINES CHANGES IN ARTERIAL STIFFNESS CAUSED BY ACUTE INFLAMMATION**

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**Purpose:** A promoter polymorphism (-174G>C) of interleukin-6 has been linked with increased cardiovascular risk Arterial stiffness is an important predictor of cardiovascular risk. Recent data suggest that acute inflammation leads to an increase of aortic stiffness. The effect of this polymorphism on arterial stiffness has not been defined yet.
**Methods:** Nineteen healthy adults (mean age $34.7 \pm 2.2$ years old, 11 men) participated in the study (randomised, double-blind design). Salmonella Typhi vaccine was used as an inflammatory stimulus. RLFPs were performed by standard methods for IL-6 and three genotypes were determined, GG, GC and CC. Pulse wave velocity (CF-PWV) was measured as an index of aortic stiffness using a non-invasive device (Complior®). Arterial stiffness expressed by wave reflection was studied using a validated system (SphygmoCor©). Augmentation index (AIx) was measured as an index of wave reflection. Arterial stiffness was assessed before and 8 hours after vaccination as well as in 11 non-vaccinated matched volunteers.
**Results:** Eight hours after vaccination, the G allele was associated with a more prominent change of pulse wave velocity (for GG and GC, 5.70 to 5.92m/sec, $p<0.05$, for CC group, p=NS) and a significant decrease in AIx (GG and GC, 18.31% to 12.22%, $p<0.05$ and CC 23% to 14.7%, p=NS), indicating increased aortic stiffness and decreased wave reflection. There were no changes in the control group.
**Conclusions:** Acute inflammation results in changes of arterial stiffness to a different degree, depending on interleukin-6 genotype. These findings underscore the genetic significance of IL-6 gene on the pathophysiology of cardiovascular system.

**P.053**
**AN INTERLEUKIN-6 POLYMORPHISM DETERMINES CHANGES IN ARTERIAL STIFFNESS CAUSED BY ACUTE INFLAMMATION**

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**Purpose:** Arterial stiffness is an important predictor of cardiovascular events. A polymorphism in the promoter region of il-6 (-174G>C) has been associated with cardiovascular risk. However, the relationship between this polymorphism and arterial stiffness has not been investigated yet.
**Methods:** Two hundred and forty-five individuals participated in the study (mean age $40.8 \pm 0.5$ years old, 164 males). RFLP was performed and three genotypes were determined, GG, GC and CC. Arterial stiffness as expressed by wave reflection was studied using a validated system (SphygmoCor©) that employs high-fidelity arterial tonometry and appropriate computer software for pulse wave analysis. Augmentation index (AIx) was measured as an index of wave reflection. Higher values of augmentation index indicate increased wave reflection and arterial stiffness.
**Results:** The distribution of genotype was GG/GC/CC: 125/107/13, respectively. After adjustment for age and sex, multinomial logistic regression analysis revealed that GC genotype is associated with higher values of AIx compared to GG homozygocity (22.56% versus19.6%, $p<0.1$). Moreover, further analysis showed that the presence of C allele (GC or CC genotype) was linked to increased AIx compared to GG genotype (22.37% versus 19.6%, $p<0.1$), which indicates impaired elastic properties. The values of aortic and peripheral blood pressures did not differ among three groups (p=NS).
**Conclusions:** In healthy individuals, a polymorphism of the promoter region of interleukin-6 gene is associated with wave reflection and impaired arterial elastic properties. This finding provides evidence of a possible genetic link between the inflammatory cascade, arterial stiffness and the cardiovascular system.

**P.054**
**RELATIONSHIP BETWEEN, BLOOD VISCOSITY, SHEAR STRESS AND ARTERIAL STIFFNESS IN PATIENTS WITH ARTERIAL HYPERTENSION**

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**The aim** of the study was to investigate relationships between whole blood viscosity (WBW), ascending aorta shear stress (AASS) and carotid- femoral pulse wave velocity (PWV) in patients with arterial hypertension (HT).
**Material and methods:** Study group (G1): 43 pts. with primary HT - (age $53 \pm 6.4$ yrs.) was compared with control group (G2): 15 normotensives (age - $55 \pm 5.9$ yrs.) Blood pressure using ''Omron M5 I'', PWV using Complior® device, WBV using ''Brookfield DV III+pro'', aortic diameter and flow velocity (required for AASS calculation) using VIVID 7 GE ultrasonograph were measured.
**Results:** SBP ($152 \pm 11.3$ vs $124 \pm 9.7$ mmHg, $p< 0.001$), DBP ($92 \pm 6.1$ vs $83 \pm 5.2$ mmHg, $p< 0.001$), WBV at shear rate 100-400/ $s^{-1}$($5.1 \pm 1.2$ vs $4.3 \pm 0.9$ cP, $p< 0.05$) and, PWV ($11.8 \pm 1.7$ vs $8.8 \pm 1.6$ m/s, $p< 0.05$) were higher in G1 than in G2 group. Mean AASS was higher ($27.7 \pm 5.3$ vs $21 \pm 4.3$ dyne/cm$^2$, $p< 0.05$) in G1 than in G2 group. In G1 group PWV correlated positively with age: ($r = 0.37$, $p< 0.39$), SBP: ($r = 0.45$, $p <0.05$) and WBV: ($r = 0.41$, $p<0.05$), and negatively with AASS: ($r = - 0.29$, $p< 0.05$).
**Conclusions:** Hypertensive patients are characterized by stiffer aorta and higher WBW, as well as lower AASS. Low shear stress seems to be one of the factors responsible for aortic stiffness in hypertensive patients.

**P.055**
**COMMON CAROTID ARTERY STIFFNESS: MORE SENSITIVE TO AGE AND GENDER RELATED LARGE ARTERY STIFFENING THAN AORTIC PULSE WAVE VELOCITY?**

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**Background:** The relationship between global arterial stiffness measures and measures based on local diameter and pressure readings is not yet fully understood.
**Methods:** We compared the changes with age and gender of aortic stiffness parameters — pulse wave velocity (PWV) and total arterial compliance (TAC) — to stiffness indices at the common carotid and femoral arteries — compliance (CC) and distensibility coefficient (DC); β-stiffness index — in a subset of 1026 women and 938 men, all apparently healthy subjects aged 35-55 participating in the Asklepios study.
**Results:** At the carotid artery, DC and β gradually increased with age with more pronounced stiffening in women, yielding a significant age-gender interaction. A similar trend was observed for CC. Femoral arterial stiffness did not change with age and no age-gender interaction was found. PWV indicated gradual stiffening with age occurring at an equal pace in men and women with no age-gender effect. TAC on the other hand did show a significant age-gender effect next to a change with age: it decreased in women, while remaining constant in men.
**Discussion:** In healthy middle-aged subjects, the age-related evolution of carotid stiffness and TAC indicates a more rapid increase in large artery stiffness in women than in men. This evolution, however, is not reflected in PWV. We speculate that PWV, integrating the properties of a large arterial segment that gradually varies from a large elastic to a more muscular vessel, might lack the sensitivity to pick up subtle age and gender effects primarily affecting the large, elastic arties.

**P.056**
**HERITABILITY OF ARTERIAL WALL INTIMA MEDIA THICKNESS IN DIFFERENT VASCULAR BEDS**

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**Background and purpose:** Genetic and environment factors have been linked to the cause of atherosclerosis. Carotid and femoral intima media

thickness (IMT) is a marker of early atherosclerosis that is correlated with traditional risk factors and is predictive of subsequent myocardial infarction and stroke. Furthermore, possible differences in the pathophysiology of common carotid artery IMT and femoral IMT might allow the exploration of differential gene regulation in specific vascular beds.
**Methods:** The current data included 63 probands (mean age 44.83 ± 7.41) and 77 relatives (mean age 45.35 ± 8.14). from 63 families. B-mode carotid and femoral ultrasonography was used to definite mean IMT of commom carotid (CCA) and common femoral artery (CFA). Variance component methods were used to estimate heritability from the normalized deviates.
**Results:** Variances explained by all final covariates (includes sex, age, blood pressure, smoking, total cholesterol, HDL cholesterol, triglicerides, diabetus status, body mass index) for mean CCA and mean CFA IMT were 0.393 and 0.394, respectively. Multivariable −adjusted heritability were 0.232 for mean CCA and 0.141 for mean CFA IMT (all P <0.005).
**Conclusion:** These data suggest that genetic factors independent of traditional cardiovascular risk factors more influence to CCA IMT than to CFA IMT. Although we found that acquired risk factors contribute progressively to IMT. Future studies of genetic linkage and gene candidate association are warranted to identify specific genetic variants predisposing early symptoms of atherosclerosis in specific vascular beds.

## P.057
## AORTIC DISTENSIBILITY BY NUCLEAR MAGNETIC RESONANCE IN ESENTIAL HYPERTENSION

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It is demonstrated that Essential Hypertension is accompanied by a reduction of large artery distensibility (Dist), which represents a marker of demonstrated prognostic significance. Dist can be assessed by echotracking derived systo-diastolic changes in aortic diameter versus blood pressure changes. However, a less operator dependent and more precise assessment might be obtained by nuclear magnetic resonance (NMR). This study was done to compare aortic (Ao) Dist obtained by echotracking and NMR in normotensive and hypertensive patients. We studied 14 treated essential hypertensives (age 36 ± 3.5 years, blood pressure 126 ± 3/78 ± 1.7 mmHg means ± SE) and 15 matched normotensives controls (blood pressure 116 ± 3.0/73 ± 2.4 mmHg). Systodiastolic changes in thoracic (T, 1 cm above the celiac tripode) and abdominal (A, 1 cm above bifurcation) Ao were obtained by either method. Dist was calculated via the Reneman formula using tonometric carotid pulse pressure. NMR Dist values were systematically greater than the echotracking one (Hypertensives T 7.2 ± 0.5 vs 3.0 ± 0.5 , A 6.0 ± 0.5 vs 2.9 ± 0.4 1/mmHg $10^{-1}$ ,Normotensives, T, 8.6 ± 0.5 vs5.2 ± 0.5, A 7.5 ± 0.5 vs3.6 ± 0.3 1/mmHg $10^{-1}$ p<0.05). The correlation between RMN and echotracking obtaind values was significant. Dist was systematically and significantly lower in H than N (p always <0.05) . No significant differences were observed between NMR T and A aAo arterial diameter. Thus, Ao Dist may be underestimated by echotracking method. Data obtained by the two approaches are similarly capable of detecting Dist reduction in hypertension. This scores in favour of continuing use of the much low expensive echotracking method.

## P.058
## ENDOTHELIAL DYSFUNCTION AS MEASURED BY FLOW MEDIATED DILATATION (FMD) IN CARDIOVASCULAR RISK ASSESSMENT

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**Introduction:** FMD has been proposed as a tool in cardiovascular (CV) risk stratification. However, this has only been studied in relatively small, high risk populations.
**Objective:** We investigated whether FMD was related to CV risk factors and intima media thickness (IMT) in a low risk population, the Nijmegen Biomedical Study (NBS).
**Method:** FMD and IMT were measured non-invasively in the brachial and the common carotid artery, respectively, using ultrasound.The NBS is a low risk population based cohort, aged 50—70 years. Interim analysis of FMD and IMT were performed in 337 subjects (mean age 60.3 year, % male 57.5). All traditional clinical and biochemical CV risk factors were determined.
**Results:** Mean IMT was 0.83 ± 0.11 mm. Mean FMD was 1.45 ± 2.45%. After correction for age and gender, all CV risk factors were significantly correlated to IMT (Pearson's R 0.11-0.33), explaing 42% of the variance in IMT. However, for FMD no significant correlations with any of the CV risk factors were found (Pearson's R 0.007 -0.13), except for TC and apoB . All CV risk factors explained only 9,5% of the variance in FMD whereas 6% of the variation in FMD was explained by IMT.
**Conclusion:** In our low risk population based cohort, aged 50-70 years old, FMD was not related to most of the CV risk factors whereas IMT contributed significantly to the variation in FMD. Our results question the additive value of FMD compared to IMT in terms of CV risk assessment, in older low risk populations.

## P.059
## IS INCREASED SYSTOLIC BLOOD PRESSURE THE MAIN REASON FOR CARDIOVASCULAR DISEASES?

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It is very well known that women in reproductive period are naturally better protected against cardiovascular diseases than men of the same age. The reasons for these are not clear.

For better determination of possible differences between gender we measured ECG, systolic (SBP), diastolic (DBP) and pulse pressure (PP), heart rate (HR), variability of HR (HRV) and pressure, cardiac output (CO), stroke volume (SV) and peripheral vascular resistance (SVR) at rest, during mental stress (3 minutes of standard arithmetic challenge) and during recovery in the group of 24 healthy males and 38 aged-matched females (19-22 years old).

HR did not differ between both groups at rest and recovery after stress, but was statistically significant higher (p<0,05) during mental stress in females. There was no difference in SBP, DBP or PP at rest, during stress and during recovery. There were no differences in HRV, variability of SBP or DBP and in SV also. But CO was greater in females, especially during mental stress (p<0.05), and SVR was smaller in females at rest, during mental stress and during recovery (p<0.05) when compared to the group of males.

There were no differences in SBP or DBP between males and females. The main differences observed were in CO and SVR. The question arises: is increased systolic or diastolic pressure the main reason for cardiovascular diseases or the reason lies somewhere in the different CO or SVR between males and females?

## P.060
## BAROREFLEX SENSITIVITY AND THE QUALITY OF BLOOD PRESSURE REGULATION

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The slope of the simultaneously changing blood pressure with heart rate (baroreflex sensitivity - BRS) is supposed to be a measure of the quality of arterial pressure regulation. In an attempt to discover early mechanisms of cardiovascular alterations we monitored different parameters in healthy, young volunteers: physically trained (N = 24) and aged-matched sedentary controls (N = 35).

We measured ECG, systolic (SBP) and diastolic blood pressure (DBP), cardiac output (CO), stroke volume (SV), peripheral vascular resistance (SVR) and BRS first at rest and then during slow breathing. Slow breathing (6 breaths/min) is one of the manoeuvres that are believed to increase vagal tone.

A spectral analysis of RR intervals was done by the autoregression method. We determined the area under the power spectrum curves over the high frequency (HF) band (0.15-0.4 Hz) and the low frequency (LF) band (0.04-0.15 Hz).

RR intervals, SBP, DBP and BRS were nearly the same in trained and untrained volunteers at rest and during slow breathing. There were also no difference in heart rate variability parameters except the significant difference (p<0,05) between groups in LF/HF during slow breathing. The