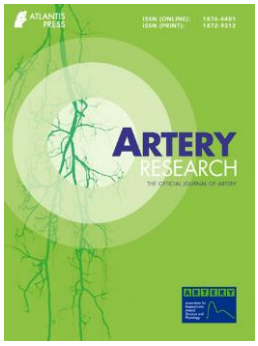

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05.03: OSTEOPROTEGERIN IS RELATED TO CAROTID-FEMORAL PULSE WAVE VELOCITY AND SURVIVAL IN HEMODIALYSIS PATIENTS

J. Nemcsik, B.C.S. Fekete, G. Speer, G. Bakonyi, J. Egresits, E. Fodor, T.E.H. Othmane, A. Szabo, I. Kiss, A. Tisler

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Background: We investigated in the same subjects, heritability and familial aggregation of various indexes of arterial stiffness and we partitioned the phenotypic correlation between these traits into shared genetic and environmental components.
Methods: Using a family-based random sampling frame, we recruited 204 parents (mean age, 51.7 years) and 290 offspring (29.4 years) from the population in Cracow, Poland (62 families), Hechtel-Eksel, Belgium (36), and Pilsen, the Czech Republic (50). We measured peripheral pulse pressure (PPp) sphygmomanometrically at the brachial artery; central pulse pressure (PPc), the peripheral (PAIx) and central (CAIx) augmentation indexes by tonometry at the radial artery; and aortic pulse wave velocity (PWV) by tonometry or ultrasound. In multivariate-adjusted analyses, we used the ASSOC and PROC GENMOD procedures as implemented in S.A.G.E. and SAS, respectively.
Results: All traits, with the exception of PPc ($P=0.79$) and PWV ($P=0.08$), showed significant heritability ($P\leq0.0001$), ranging from 0.37 for PPp to 0.41 for CAIx. The genetic correlation between PWV and the other arterial indexes were significant ($\rho_G\geq0.29$; $P<0.0001$). The corresponding environmental correlations were only significantly positive for PPp ($\rho_E=0.10$, $P=0.03$). Intrafamilial concordance was significant for all arterial indexes ($r\geq0.12$; $P\leq0.02$), with the exception of PPc ($r=-0.007$; $P=0.90$) in parent-offspring pairs. The sib-sib correlations were also significant for CAIx ($r=0.22$; $P=0.001$).
Conclusion: The observation in the same group of subjects of significant intrafamilial concordance and heritability of various indexes of arterial stiffness as well as the genetic correlations among arterial phenotypes strongly support the search for shared genetic determinants underlying these traits.

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05.03
OSTEOPROTEGERIN IS RELATED TO CAROTID-FEMORAL PULSE WAVE VELOCITY AND SURVIVAL IN HEMODIALYSIS PATIENTS

J. Nemcsik [1], B.C.S. Fekete [2], G. Speer [2], G. Bakonyi [3], J. Egresits [1], E. Fodor [3], T.E.H. Othmane [2], A. Szabo [3], I. Kiss [1], A. Tisler [2]. *[1]Division of Angiology and Nephrology, Department of Medicine, St. Imre Teaching Hospital, Budapest, Hungary, [2]1st Department of Medicine, Semmelweis University, Budapest, Hungary, [3]B Braun Avitum Nephrological Network, Budapest, Hungary*

Osteoprotegerin (OPG) is a marker and regulator of arterial calcification, and it is related to survival in hemodialysis patients. The link between OPG and aortic stiffening - a consequence of arterial calcification - has not previously been evaluated in this population, and it is not known whether OPG related mortality risk is mediated by arterial stiffening.

At baseline OPG and aortic pulse wave velocity (PWV) was measured in 98 hemodialysis patients who were then followed for a median of 18 months. The relationship between OPG and PWV was assessed by multivariate linear regression. The role of PWV in mediating OPG related mortality risk was evaluated by including both OPG and PWV in the same survival model.

At baseline mean (SD) PWV was 11.2 (3,3) m/s and median OPG (interquartile range) was 11.1 (7.5-15.9) nmol/L. There was a strong positive linear relationship between PWV and lnOPG (beta=1.48, p=0.009), independent of other covariates. During follow-up 28 patients died (mortality rate 18.4/100 patient years). In separate survival models both PWV and lnOPG were related to all cause mortality (hazard ratios 1.21[1.07-1.38] and 5.39 [2.16-13.43], respectively). When both PWV and lnOPG were entered into the same model, only OPG remained significantly associated with mortality (hazard ratios 1.12 [0.97-1.28] and 4.37 [1.62-11.80], respectively).

In hemodialysis patients OPG is strongly related to PWV and OPG related mortality risk may, in part, mediated by increased PWV.

05.04
ACUTE EFFECTS OF PASSIVE SMOKING ON PERIPHERAL VASCULAR FUNCTION

D. Adamopoulos [1], J.F. Argacha [1], M. Gujic [1], N. Amai [1], D. Fontaine [2], G. Berkenboom [1], P. van de Borne [1]. *[1]Department of Cardiology, Erasme Hospital, Brussels, Belgium, [2]Laboratory of Pharmacology and Physiology, Erasme Hospital, Brussels, Belgium*

Background: Environmental tobacco smoke (ETS) acutely affects vascular function through many pathophysiological mechanisms including nicotine sympathoexcitatory effects and oxidative stress. However, a secondary vascular reflex following smoke sensory stimulation cannot be excluded, since the vascular effects of ETS exposure have never been compared to these of a non-tobacco smoke. We therefore tested the hypothesis that acute ETS exposure, when compared to non-tobacco smoke, is responsible for a specific increase in aortic wave reflection and that this is accompanied by an alteration of endothelium dependent microvascular function.
Materials and methods: We examined the vascular effects of one hour ETS exposure, compared to a non-tobacco smoke and a normal-air exposure, in 11 healthy non-smokers men, using a randomized, single blind cross over study design. Augmentation index (AIx), and wave transit time (Tr) have been used to assess aortic wave reflection, while skin microvascular response to a local heating stimulation has been measured with a laser doppler flowmeter to assess endothelial function.
Results: Air particle densities did not differ during the ETS and non-tobacco smoke sessions. We observed no effect of ETS or non-tobacco smoke on central and peripheral blood pressures. However, AIx increased both during ($p=0.01$) and after ($p<0.01$) the ETS session, but remained unchanged in the non tobacco smoke session as compared to normal air. A strong correlation between serum nicotine levels ($n=10$) after ETS exposure and Aix change ($r=0.84$, $p<0.01$) was also noted. Tr decreased both during ($p=0.02$) and after ($p<0.01$) ETS, but remained unchanged in the non tobacco smoke session as compared to normal air. ETS exposure reduced the skin blood flow response to heating ($p=0.03$), which was not seen during the non tobacco smoke and the normal air sessions.
Conclusions: Passive exposure to tobacco smoke increases aortic wave reflection and impairs endothelium dependent microvascular function as compared to passive inhalation of non tobacco smoke. The increase in wave reflection after ETS exposure is strongly related to the rise in serum nicotine levels.

Free Communications

06.01
THE INFLUENCE OF CARDIOVASCULAR DISEASE AND RISK FACTORS ON AGE-RELATED CHANGES IN AORTIC PULSE WAVE VELOCITY

C.M. McEniery [1], Y. Yasmin [1], M. Munnery [2], S.M.L. Wallace [1], B. McDonnell [2], K.M. Maki-Petaja [1], S. Hickson [1], J.R. Cockcroft [2], I.B. Wilkinson [1]. *[1]University of Cambridge, Cambridge, United Kingdom, [2]Wales Heart Research Institute, Cardiff, United Kingdom*

We have demonstrated previously that age-related changes in aortic pulse wave velocity (PWV) are more prominent in individuals over the age of 50 years, suggesting that aortic PWV might provide a sensitive marker of risk in older subjects. Therefore, the aim of this investigation was to assess the impact of cardiovascular disease and risk factors on age-related changes in aortic PWV.

Data from 4219 participants in the ACCT Study[†] cohort were analysed (aged 18-92 years). In all subjects, seated and supine brachial BP was measured following at least 10 minutes of rest. Central (aortic) BP was derived by pulse wave analysis, and aortic (carotid-femoral) and brachial PWV were recorded (SphygmoCor). Subjects were then divided into groups based on the presence of cardiovascular disease ($n=445$) or risk factors: diabetes ($n=311$), hypertension ($n=952$), hypercholesterolaemia ($n=196$) and smoking ($n=318$), leaving 1997 control subjects, all of whom were free of cardiovascular risk factors and medication. Peripheral and central blood pressure and aortic PWV all increased significantly with age ($P<0.001$). However, compared with healthy controls, there was a steeper age-related rise in aortic PWV after the age of 50 years in subjects with cardiovascular disease or risk factors ($P<0.001$, Figure), even after adjusting for differences in mean