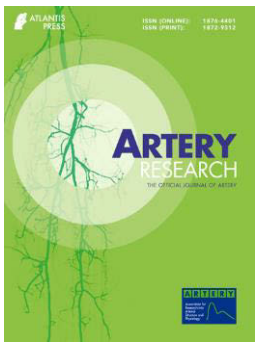

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# 13.04: LACK OF ENDOTHELIAL DYSFUNCTION IN BUERGER'S DISEASE

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**13.01**
**C-REACTIVE PROTEIN LEVELS ARE GRADUALLY ASSOCIATED WITH ADIPONECTIN AND ARTERIAL STIFFNESS IN NEWLY DIAGNOSED UNTREATED ESSENTIAL HYPERTENSIVE SUBJECTS: A UNIFYING APPROACH TO ATHEROSCLEROSIS**

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**Purpose:** To examined the plausible correlations between hs-CRP levels, adiponectin and arterial stiffness in essential hypertensive patients.
**Methods:** In 148 newly diagnosed untreated non-diabetic essential hypertensive patients [98 men, mean age = 49 years, office BP = 150/97 mmHg], aortic stiffness was evaluated on the basis of carotid to femoral pulse wave velocity (PWV), by means of a computerized method (Complior SP). Venous blood samples were drawn for estimation of lipid profile and hs-CRP and adiponectin levels. All subjects according to hs-CRP values were divided into group A (hs-CRP $\leqslant$ 1.29 mg/l), group B (hs-CRP = 1.3–2.39 mg/l) and group C (hs-CRP > 2.39 mg/l).
**Results:** Patients in group A (n = 51) compared to subjects in group B (n = 45) and C (n = 52) had lower office systolic BP and left ventricular mass index ($p < 0.005$ for all cases), while groups did not differ regarding lipid levels (p = NS). In the entire population, hs-CRP was positively associated with body mass index ($r = 0.32$, $p < 0.001$) and c-f PWV (0.412, $p < 0.0001$), while it was negatively correlated with adiponectin ($r = -0.231$, $p = 0.005$). Furthermore, patients in group C exhibited lower levels of adiponectin compared to group B and A ($7.0\pm4.0$ vs $8.9\pm5.1$ vs $9.4\pm4.9$ μg/ml, respectively; $p < 0.05$ for all cases) and more augmented PWV values ($8.6\pm1.6$ vs $8.2\pm0.9$ vs $7.8\pm1.2$ m/s, $p < 0.05$, for all cases). Analysis of covariance revealed that adiponectin and PWV values remained different between groups after adjustment for confounding factors ($p < 0.05$).
**Conclusions:** Low-grade inflammation is associated in a graded fashion with proatherogenic processes linked with hypoadiponectinemia and arterial stiffening, even in the early stages of essential hypertension.

**13.02**
**EXPOSURE TO URBAN AIR POLLUTANTS ALTERS ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS**

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Exposure to urban air pollution, ultrafine particles or gas, is associated with acute cardiovascular mortality and morbidity. We investigated the effects of ambient air pollution on endothelial function in 40 healthy Caucasian men, previously described in the KLK study (*JCI 2005*), who spontaneously breathed ambient air pollution in Paris.
Endothelial function was measured by the % of brachial artery dilatation (dDr) and the absolute variation of shear stress (dSS) after hyperhemia following 5 min hand ischemia and after 150 μg of TNT sublingual, using RF-bases echotracking device, at two visits 2 weeks apart. Air pollution level, (CO, NO, $NO_2$, $SO_2$, PM 2.5) were extracted from "Airparif" database, the day of vascular measurement (J0"Pollutant") and 5 days before (mean "pollutant"). The ranks of pollutants were added to form SPOL score.
Baseline dDr was significantly and negatively correlated with mean and J0 NO ($P < 0.001$ for both), mean and J0 SO2 ($P < 0.0001$ for both), mean and J0 CO ($P < 0.0001$ for both), and SPOL($P < 0.001$). SPOL explained 19% of the variance of baseline dDr. Baseline dSS was significantly correlated with mean NO2 ($P = 0.003$), mean SO2 ($P = 0.004$), J0 SO2 ($P = 0.05$), and J0 PM 2.5 ($P = 0.02$). Changes in dDr between the two visits was significantly correlated with delta NO ($P = 0.001$) and delta SPOL ($P = 0.006$). Delta SPOL explained 8% of the variance of Delta DdLir. Changes in diameter or shear stress after TNT were not correlated with changes in air pollutants levels.
Endothelial function is significantly impaired by ordinary levels of pollutant in healthy young males, in urban area.

**13.03**
**SYNERGISTIC EFFECT OF ANGIOTENSIN II TYPE 1 RECEPTOR AND ENDOTHELIAL NITRIC OXIDE SYNTHASE GENE POLYMORPHISMS ON ARTERIAL STIFFNESS**

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Angiotensin II and nitric oxide play an important role in the function of arterial system. We wondered whether the mutations of angiotensin II type 1 receptor (AGTR1) and endothelial nitric oxide synthase (eNOS) genes are associated with increased stiffness of large arteries. Two frequent polymorphisms, $A^{1166}C$ of AGTR1 and $T^{786}C$ of eNOS, were assessed in a random, population-based sample of 250 subjects aged 25 to 64 years. Pulse wave velocity was measured in the aorta (APWV, between carotid and femoral arteries) and on the lower extremity (peripheral pulse wave velocity, PPWV, between femoral and tibialis posterior/dorsalis pedis arteries). Both polymorphisms were significantly associated with PPWV: $12.4\pm0.7$, $13.8\pm0.2$, $15.2\pm2.7$ m/s for AA, AC and CC genotypes of AGTR1, respectively, $p < 0.02$ for trend; $12.3\pm0.8$, $13.4\pm1.0$, $15.1\ \pm1.6$ m/s for TT, TC and CC genotypes of eNOS, respectively, $p < 0.05$). The combined effect of the polymorphisms was further studied. Subjects with 3–4 mutant alleles (heterozygous + homozygous or homozygous + homozygous, n = 35) had signigicantly increased PPWV ($17.9\pm2.4$ m/s) than those with no mutant allele ($12.4\pm1.2$ m/s) or 1–2 alleles ($12.3\pm0.5$ m/s, $p < 0.007$ for difference). These associations remained highly significant in multiple regression models with adjustment on potential confounders. The polymorphisms did not influence APWV or blood pressure. In conclusion, both AGTR1 and eNOS gene polymorphisms are associated with increased stiffness of peripheral muscular-type large arteries and their effect is synergistic. This finding reflects an interaction between the renin-angiotensin and nitric oxide systems in their effect on arterial properties.

**13.04**
**LACK OF ENDOTHELIAL DYSFUNCTION IN BUERGER'S DISEASE**

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**Objective:** To compare the acute flow-dependent vasodilatation (FDV) to a hand warming test (from 28ºC to 44ºC) and endothelium-independent vasodilatation (EIV) to sublingual glyceryl trinitrate (GTN 150 μg) of the brachial artery (BA) in 10 patients with an acute-phase Buerger disease, defined by an ADAR score $\geqslant$4 and a recent ulcer of the lower limbs (7 current), and 10 age- and sex-matched non-smokers healthy subjects.
**Methods:** BA diameter and shear stress were recorded by high definition echotracking. FDV was estimated by the slope of diameter-shear stress relationship.
**Results:** See the table.

| | median [IQR] | | P value |
|---|---|---|---|
| | Buerger | Control | |
| FDV: Δ BA diameter (%) | 6.8 (0.1;8.1) | 3.7 (1.1;6.5) | ns |
| FDV: Δ shear stress (%) | 70 (32;100) | 93 (57;125) | ns |
| EIV: Δ BA diameter (%) | 30 (28;33) | 17 (13;22) | ns |
| EIV: Δ shear stress (%) | 98 (40;137) | 117 (76;251) | ns |
| Slope diameter-stress | 0.89 (0.55;1.24) | 0.28 (–0.01;0.58) | 0.025 |

Buerger's patients had an enhanced flow-dependent response to the increase in shear stress due to hand warming by comparison with controls as shown by the higher slope of diameter-shear stress relationship.
**Conclusions:** Acute flow-mediated changes in brachial artery diameter during hand hyperhemia and EIV to GTN were not impaired in patients compared to control, by contrast to what has been repeatedly suggested in the literature.

**14.01**
**MOLECULAR DETERMINANTS OF ARTERIAL STIFFNESS**

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Arterial stiffness has an independent predictive value for cardiovascular events. This review proposes an integrated view of the molecular determinants of arterial stiffness, based on a candidate gene approach, an analysis of the structure-function relationship in hypertension, and studies on gene expression profile in humans. In monogenic diseases of connective tissue (Marfan, Williams, and Ehlers-Danlos syndromes) and corresponding animal models, the precise characterization of arterial phenotype allows understanding the influence of abnormal, genetically-determined, wall components on arterial stiffness. These studies underline the role of extracellular matrix signaling in the vascular wall and the fact that elastin