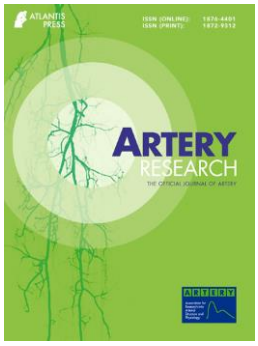

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P2.13: ALTERED MICROVASCULAR RESPONSES TO ANGIOTENSIN II INFUSION INDICATING ENDOTHELIAL DYSFUNCTION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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Purpose: HIV infection is linked to heightened cardiovascular risk; this is partly mediated through endothelial dysfunction. We investigated the interplay of endothelial function with markers of inflammatory and oxidation in two groups of HIV infected patients: on ART and naïve to ART.
Methods: We recruited 47 HIV infected patients (46 male, mean age 35±10 years, mean CD4 count: 579±271). All were free from overt cardiovascular disease. 31 patients were naïve to ART; 17 were on ART. FMD of the brachial artery was used as an index of endothelial function. Markers of inflammatory (CRP, IL6), oxidative (ADMA) and functional (CD4, viral load) status were measured. Between groups comparisons (ART vs naïve patients) were performed using the Mann-Whitney U test.
Results: Endothelial function and inflammatory markers did not differ across groups. ART group had higher ADMA levels, lower viral load and higher CD4 count, thereby a favorable oxidative/functional status compared to naïves.

	Naïve (n=31)	ART (n=17)	P value
FMD (%)	4.2 (2.3, 7.5)	5.2 (3.1, 8.2)	0.740
CRP (mg/dL)	1.37 (0.98, 2.79)	1.05 (0.28, 3.32)	0.249
IL6 (pg/mL)	1.65 (0.83, 3.39)	1.00 (0.77, 1.65)	0.258
ADMA (μmol/L)	0.63 (0.25, 0.79)	0.84 (0.78, 1.06)	0.006
Viral load	19419 (8144, 54025)	50 (50, 50)	0.001
CD4 count	450 (378, 627)	635 (510, 779)	0.011

Conclusion: In HIV infection, ART does not change endothelial function; nevertheless it is linked to a favorable oxidative and functional status.

P2.13
ALTERED MICROVASCULAR RESPONSES TO ANGIOTENSIN II INFUSION INDICATING ENDOTHELIAL DYSFUNCTION IN SUBJECTS WITH FAMILIAL HYPERCHOLESTEROLEMIA

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Purpose: Angiotensin II (ANG) is implicated in the development of cardiovascular disease. We examined vascular responses to ANG in subjects with familial hypercholesterolemia (FH), a group at high cardiovascular risk.
Methods: The effects of ANG (3h iv infusion, 10 g/kg/min) on brachial blood pressure and forearm skin microvascular function were studied in 8 female and 8 male FH (mean age 43±8 ys) and in 16 matched healthy controls. Skin microcirculation was studied by laser Doppler fluxmetry during rest and local heating of the skin to 44°C (microvascular hyperaemia). Microvascular resistance was assessed by mean arterial pressure/microvascular hyperaemia, and macrovascular reactivity by blood pressure changes. Measurements were performed before, at 1 and 3h of ANG, and 1h after stopping infusion. Mean values ±SD.
Results: Baseline systolic blood pressure was higher in FH (127±14 vs 115±12 mm Hg; p=0.02), while pressure responses to ANG were similar in both groups (eg +24±10 vs +21±7 mm Hg, ANG 3 h). There were no baseline differences in microvascular hyperaemia or resistance between the groups. However, during and after ANG microvascular hyperaemia was impaired (p=0.01; eg 126±95 vs 184±102 units, ANG 3 h), and microvascular resistance higher (p=0.01; eg 1.9±0.9 vs 0.9±0.8 mm Hg/units; ANG, 3 h) in FH. Saline infusion verified stability of the experimental design (n=8).
Conclusions: Despite similar blood pressure responses to ANG in FH and controls, microvascular dilatation capacity was impaired in FH, indicating endothelial dysfunction. These findings and increased microvascular resistance may lead to hypertension and cardiovascular complications in FH.

P2.14
RELATION OF HEART RATE VARIABILITY TO ENDOTHELIAL FUNCTION IN HEALTHY SUBJECTS – DEPENDENCE UPON CARDIAC CYCLE LENGTH

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In various diseased states reduced cardiac vagal modulation (CVM) is accompanied by impaired endothelial function. It is unclear whether CVM and endothelial function undergo dysregulation independently, or these systems affect each other negatively as a consequence of the disease process. Since the respective physiology is unclear, we aimed to investigate if such relationship between CVM and endothelial function exists in healthy subjects.
46 young males were studied. From 10 minute long ECG recordings mean RR interval (RRI) and time and frequency domain vagal heart rate variability (HRV) indices (SDNN; RMSSD; pNN50 and HF, respectively) were determined. HRV indices were used to define CVM. Endothelial function was assessed by measuring brachial artery flow mediated dilation (FMD). Hyperemic, diastolic shear rate was used to normalize FMD (nFMD).
RRI was related to most HRV indices, but not to FMD. On the other hand, all HRV indices correlated significantly and positively with FMD across subjects ($r = 0.49$, $p<0.01$ for HF–nFMD). After adjusting for potential confounders, RMSSD and HF remained significantly associated with nFMD. When subjects were dichotomized according to median RRI, the HRV–nFMD relations lost significance at higher (RRI<910 ms), and gained further significance at lower (RRI>910 ms) heart rates ($r = 0.57$, $p<0.01$ for HF–nFMD).
Our data demonstrate that vagal HRV indices are related to FMD across healthy male subjects. Although RRI is not related to FMD, the HRV-FMD relation is dependent upon RRI. The underlying mechanism may involve centrally released endothelial mediators, which enhance CVM through vasculo-neural communication.

P3 – Interventional studies 1

P3.01
A COMPARISON OF ARTERIAL FUNCTION OF HIV INFECTED (TREATED AND NEVER-TREATED) AND UNINFECTED BLACK SOUTH AFRICANS AFTER FIVE YEARS: PURE STUDY

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The prevention and treatment of hypertension is marginalized in South Africa by the overwhelming prevalence of HIV. HIV-1 infection and the treatment thereof paradoxically affect cardiovascular risk factors and may add to the cardiovascular risk of these individuals.
We aimed to compare the 5 year cardiovascular changes of black South Africans who were (a) HIV-1 infected without treatment, (b) HIV-1 infected with antiretroviral therapy and (c), uninfected. In this study 164 uninfected and 145 HIV-1 infected (77 never-treated and 68 treated) participants were followed-up after a 5 year period. Two hundred and ninety one participants were lost to follow-up, 11 were newly HIV infected, 39 died and 241 did not partake. The cardiovascular and anthropometric variables were assessed and the percentage change determined. Follow-up analysis (cross-sectional) showed a lower IMT ($p<0.01$), central systolic blood pressure ($p<0.01$) and augmentation index ($p=0.03$) in the HIV infected compared to the HIV uninfected participants. After 5 years (2005-2010) the treated HIV-1 infected participants showed an increase in pulse pressure ($p=0.03$) and no change in pulse wave velocity, whilst a decrease ($p=0.02$) was encountered in the never-treated HIV infected participants. No difference in the % change was seen between the treated and uninfected participants.
In conclusion, the cardiovascular profile of the treated HIV-1 infected Africans show signs of an early development of arterial dysfunction over 5 years which is not seen in the never-treated participants. How the