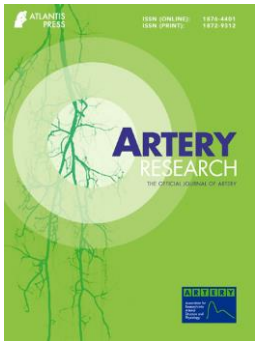

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PO-12: RACIAL DIFFERENCES OF ENOS EXPRESSION RESPOND TO C-REACTIVE PROTEIN

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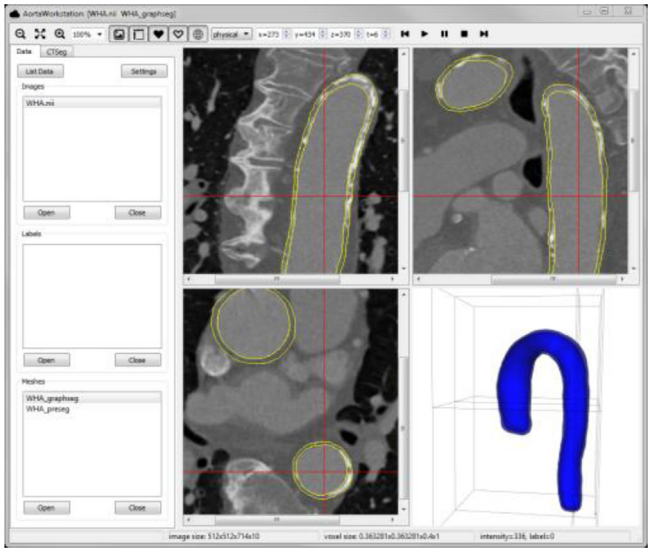

Figure 1 Example of data visualization: image data is shown on three orthogonal slices, segmented surface is shown by 3D rendering and its intersections with image slices (yellow contours).

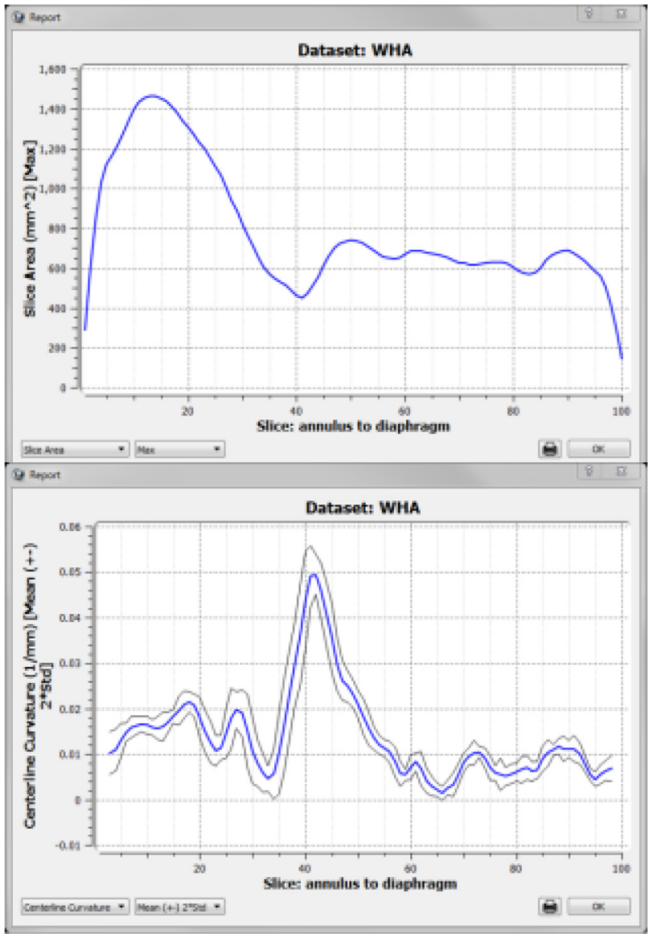

Figure 2 Examples of quantitative indices derived from a cardiac cycle. Top: maximal cross-sectional areas, bottom: range (mean and 2*standard deviation) of centerline curvature.

PO-11

SEX DIFFERENCES IN THE DEVELOPMENT OF ABNORMAL ENDOTHELIUM-DEPENDENT VASODILATION IN AORTA FROM TYPE 2 DIABETIC RATS: POSSIBLE ROLE OF NITRIC OXIDE

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Little is known about the interaction between diabetes and sex in vasculature. This study was designed to investigate whether there were sex differences in rat aortic endothelium-dependent vasodilation (EDV) in Zucker diabetic fatty (ZDF) rats, and the potential role of nitric oxide (NO). EDV to acetylcholine (ACh) was measured in aortic rings pre-contracted with phenylephrine (PE). Contractile responses to PE were generated before and after treatment with L-NAME (200 μM), a NO synthase (NOS) inhibitor. In addition, the levels of endothelial NOS (eNOS) and NADPH oxidase (NOS, a potent source of superoxide) mRNA expression were determined using real-time RT-PCR. Type 2 diabetes significantly impaired EDV in aortic rings from female ZDF rats, however, potentiated the relaxation in males. Diabetes decreased the contractile responses to PE in aortic rings from rats, regardless of sex. Moreover, diabetes enhanced the extent of PE potentiation after blocking eNOS with L-NAME in females. Accordingly, the levels of eNOS mRNA expression were substantially enhanced in aorta of female ZDF rats compared to those in lean animals. In addition, Nox1 and Nox4 mRNA expression were substantially enhanced in aorta of female ZDF rats. These data suggest that the predisposition of the female aorta to injury in type 2 diabetes may be, in part, due to the alteration of NO production (supported by NIH/NIDCR).

PO-12

RACIAL DIFFERENCES OF eNOS EXPRESSION RESPOND TO C-REACTIVE PROTEIN

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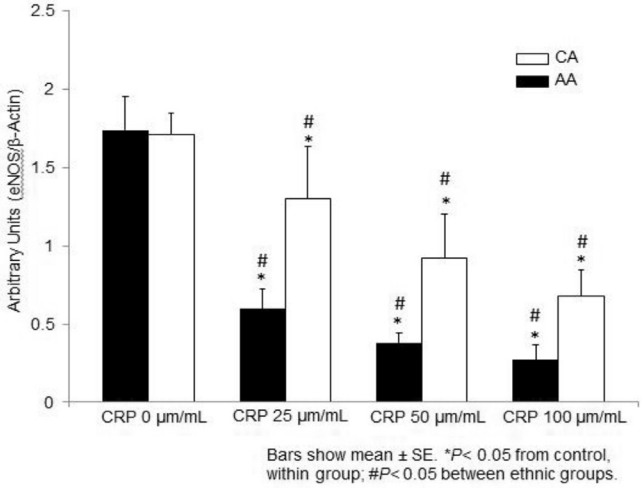
Background: African Americans (AA) have higher rates of cardiovascular diseases (CVD) compared to Caucasians (CA). Endothelial dysfunction is a common feature of CVD risk factors. Previous studies suggest racial differences in endothelial function exist at the physiological level. C-reactive protein (CRP), a risk marker for CVD, causes a reduction in eNOS expression and bioactivity in endothelial cells (ECs). AA individuals have significant higher concentrations of CRP than Caucasian (CA) individuals. The aim of this study was to investigate the racial differences of endothelial function under CRP stimulation at the cellular level.
Methods: Eight human umbilical vein endothelial cells (HUVECs) lines from African American (AA) and Caucasian (CA) donors with gender split evenly were cultured and incubated with CRP for 24-hrs. Doses of CRP were 0, 25, 50 and 100 μg/mL. Western blot was conducted to measure the expression of eNOS after CRP incubation. ImageJ densitometric analysis of eNOS bands expressed in relation to β-actin.



Results: As Figure 1 shows, at control condition, there was no difference in the eNOS protein expression between AA and CA HUVECs. The incubation of CRP significantly reduced the expression levels of eNOS on both AA and CA HUVECs in a dose-dependent manner. The reductions of eNOS protein expression in AA HUVECs at all three different concentrations were significantly greater than those in CA HUVECs.
Conclusion: AA HUVECs respond differently to CRP compared to CA HUVECs. CRP incubation causes greater reduction of eNOS expression on AA than CA HUVECs. The results suggest a possible mechanism for the racial differences in endothelial dysfunction.

PO-13
ARTERIAL HEMODYNAMICS IN OVERWEIGHT YOUNG ADULT MALES FOLLOWING MAXIMAL EXERCISE

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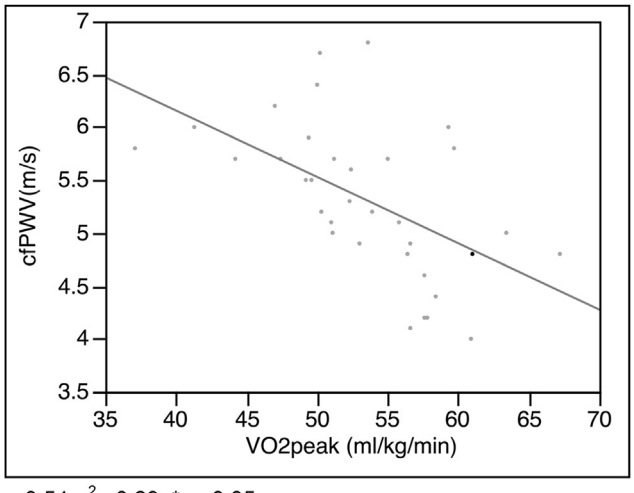
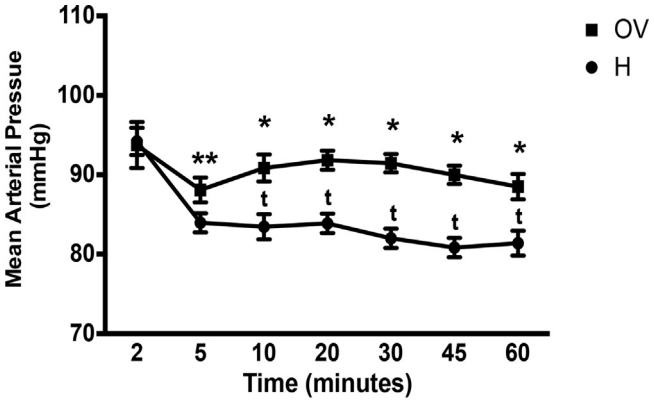
Objective: Overweight (OV), defined by body mass index (BMI), is related to increased cardiovascular risk and greater aortic stiffening. In contrast, enhanced cardiorespiratory fitness (CRF) is associated with reduced cardiovascular risk, and lower aortic stiffness. It is unknown whether CRF is related to aortic stiffness in young OV adult males. We hypothesized CRF would be inversely associated with aortic stiffness, and the post-exercise hemodynamic response would be impaired in OV males.
Methods: Thirty-four apparently healthy, young adult males (22.12 $\pm$ 0.09 years) were categorized based on BMI as healthy weight (H, $\leq$24.9 kg/m^2), or OV (24.9-29.9 kg/m^2). Resting measures of arterial stiffness (carotid-femoral pulse wave velocity, cfPWV), heart rate (HR), blood pressure (BP), pulse pressure (PP), mean arterial pressure (MAP), percent body fat (BF%), waist (WC) and hip circumference (HC), and waist-to-hip ratio (W:H) were obtained. Peak oxygen consumption (VO_{2peak}), a measure of CRF, was assessed with a maximal exercise treadmill test (EX). cfPWV and BP were obtained at 2, 5, 10, 20, 30, 45 and 60 minutes following EX.
Results: Compared with H at rest, OV had greater cfPWV, BMI, BF%, systolic BP (SBP), PP, MAP, WC, HC, and W:H ($p<0.05$, all). VO_{2peak} was greater in H compared with OV ($p<0.05$). A positive association was observed between resting cfPWV and SBP, whereas cfPWV was inversely related to VO_{2peak} ($p<0.05$, both). Compared with H, post EX MAP was increased in OV at 10, 20, 30, 45 and 60 minutes ($p<0.05$). A main effect of weight was observed for cfPWV, SBP and DBP, and a main effect of time for PP, SBP and DBP ($p<0.05$, all).
Conclusion: Increased resting aortic stiffness in young OV adult males is, in part, attributable to lower levels of CRF and increased SBP. In addition, post EX arterial hemodynamics is impaired in young adult OV males.



r=0.54, r^2= 0.29. *p <0.05.

Figure 1 Peak Volume of Oxygen Consumption (VO_2) vs. carotid-Femoral Pulse Wave Velocity (cfPWV) (n=34).



*** P< 0.05, H vs. OV at 10, 20, 30, 45, 60 minutes**
**** P<0.05, H and OV at 5 vs. 2 minutes**
t P<0.05, H at 10, 20, 30, 45, 60 vs. 2minutes

Figure 2 Post EX MAP.

PO-14
RELATIONSHIP BETWEEN CAROTID ARTERY STIFFNESS AND ALTERED CEREBROVASCULAR HEMODYNAMICS IN SOUTH ASIAN INDIAN OLDER ADULTS

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Objectives: To investigate whether differences exist in common carotid artery (CCA) stiffness between South Asian (SA) and white Caucasian (CA) older adults, and its association with cerebrovascular hemodynamic properties.
Methods: Carotid artery stiffness indicators, including pulse pressure (PP), distensibility coefficient (DC), and compliance coefficient (CC), were measured by applanation tonometry and ultrasound imaging. Continuous blood pressure (MAP), heart rate, and middle cerebral artery blood flow velocity (MFV) using non-invasive transcranial Doppler ultrasound, were monitored in 44 age- and gender-matched SA and CA community-dwelling older adults free of cardio- and cerebrovascular diseases (22 CAs/SAs: 11 M/F in each group, aged 64-82 years). Cerebrovascular resistance index (CVRi) and pulsatility index (PI) were also calculated for evaluation of cerebrovascular hemodynamics.
Results: Carotid artery stiffness was higher in SA compared to CA group, as evidenced by lower arterial compliance (CC=601$\pm$282 vs. 789$\pm$323 mm^2/MPa, respectively, p=0.048), and greater PP (59$\pm$18 vs. 46$\pm$10 mmHg, respectively, p=0.005). A significant interaction effect between ethnic group and arterial compliance on PP was observed (r^2=0.562, $p<0.001$), indicating that less compliant arteries resulted in higher PP amplitudes in SA compared to CA group. Furthermore, a moderate negative relationship between arterial compliance and CVRi was found only in the SA group (r=-0.574, p=0.025). Correspondingly, CVRi was strongly associated with lower MFV (r=-0.925, $p<0.001$).
Conclusions: SA group presented greater stiffness and less compliant arteries compared to CA group independent of age and gender. SA older adults appear to have impaired dampening capacity of central arteries to the changes in arterial pressure, thereby increasing the risk of hemodynamic pulsatility transmission into the brain. Consequently, an increase in CVRi might be a compensatory mechanism to protect the cerebral microcirculation, or reflect prior damage, resulting in lower CBF. These findings may aid in understanding the increased risk of cardio- and cerebrovascular diseases in people of SA origin.