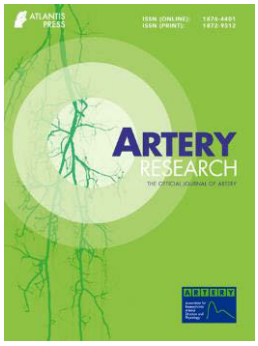

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4.2: SEX-DEPENDENT EFFECTS OF PERIVASCULAR ADIPOSE TISSUE ON VASCULAR FUNCTION

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validation prior to their implementation in clinics. For this purpose, silicone replicas of anatomically realistic geometries of AAAs are fabricated and the flow field in the aneurysmal region is experimentally measured *in vitro*, using time-resolved volumetric Particle Image Velocimetry (PIV) [3–4]. Furthermore, the experimental setup allows for strain measurements of the aneurysmal wall to be taken simultaneously using Digital Image Correlation (DIC). These data are used to validate concurrent computational simulation results and FSI analyses. The results demonstrate that the FSI computational approach can predict the patterns of flow from the PIV measurements, which arise from the geometry of the AAA. This work highlights that empirical and computational modelling can complement each other to investigate AAA development towards our goal of producing validated computational simulations that can be used for diagnostic purposes.

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Oral session IV — Clinical Aspects

4.1
PILOT STUDY ON THE PRECLINICAL VASCULAR DAMAGE IN BOLIVIAN PATIENTS WITH CHAGAS INDETERMINATE CHRONIC PHASE

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Background: In Italy, the prevalence of seropositivity for *Trypanosoma cruzi* in immigrants from endemic countries is about 11.3% (30.7% for Bolivian immigrants).
The disease acute phase is usually asymptomatic, often leading to chronic infection that may remain silent for life (chronic indeterminate phase). Chagas heart disease is the most severe and frequent (20–30%) form of chronic phase; its pathophysiology shares similar mechanisms with the arterial impairment associated to other diseases (diabetes, hypertension, aging), leading to chronic inflammation and stiffening. In literature there are no data about the possible elastic arteries deterioration in Chagas disease. Our hypothesis was that early arterial compliance modifications might be found in the chronic indeterminate phase of Chagas disease.
Methods: 35 consecutive Bolivian subjects (21 with indeterminate Chagas disease, mean age [SD] 44.2 [8.2], 5 women, and 14 controls, mean age 40.2 [8.2], 5 women) accessing the service of Tropical Medicine were enrolled. Staging of the disease, laboratory assay, and hemodynamics (central and peripheral blood pressure [BP], aortic pulse wave velocity [PWV], carotid intima media thickness, cardiac ultrasound) were assessed.
Results: No clinical nor laboratory differences were found between the cases and controls. Peripheral and central BPs components were similar. Chagas patients presented higher PWV than controls (7.87 ± 1.29 vs 6.43 ± 1.12 m/s, $p = 0.002$), even when adjusting for age, mean BP, heart rate, body mass index, smoking status ($p = 0.001$).
Conclusion: Patients with Chagas indeterminate chronic phase presented higher arterial stiffness than controls, pointing out an early arterial involvement as the possible etiological mechanism underlying the increased cardiovascular risk in these patients.

4.2
SEX-DEPENDENT EFFECTS OF PERIVASCULAR ADIPOSE TISSUE ON VASCULAR FUNCTION

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Background: Premenopausal women are relatively protected against hypertension compared to males. Estrogen levels have been identified as a potential underlying cause, but the pathophysiological mechanisms remain incompletely understood. We hypothesised that sex-dependent effects of perivascular adipose tissue PVAT mediate altered vascular function in hypertension.
Methods: The effect of PVAT was investigated on resistance vessels of 16 week old male and female stroke-prone spontaneously hypertensive rats (SHRSP).
Results: Wire-myography was used on 3rd-order mesenteric vessels (maximum contraction: male +PVAT 113.3 ± 1.1 vs. female +PVAT 91.4 ± 11.36 %). Noradrenaline mediated vasoconstriction was increased in SHRSP males compared to females. K_{ATP} channel-mediated vasorelaxation by cromakalim was impaired in males compared to females (maximum relaxation: male +PVAT 46.9 ± 3.9 % vs. female +PVAT 97.3 ± 2.7 %) A cross-over study assessing function of male PVAT on female vessels and vice versa confirmed the reduced K_{ATP} mediated vasorelaxation induced by male PVAT (maximum relaxation: female $+PVAT_{female}$ 90.6 ± 1.4 % vs. female $+PVAT_{male}$ 65.8 ± 3.5 %). An adipokine array with subsequent western blot validation identified resistin as a potential modifier of vascular reactivity. Resistin was increased by approximately 2-fold in SHRSP male PVAT. Male and female vessels pretreated with resistin (40 ng/ml) showed no difference in response to noradrenaline. However, vasorelaxation in response to cromakalim was significantly impaired in resistin treated female vessels, similar to levels observed in male vessels (maximum relaxation: female +PVAT 97.3 ± 0.9 % vs. female +PVAT +resistin 36.8 ± 2.3 %).
Conclusion: We identified a novel role for resistin in sex-dependent PVAT mediated vascular function in hypertension through a K_{ATP} channel mediated mechanism.

4.3
ABNORMAL PRESSURE WAVE REFLECTION ACCELERATES THE DEVELOPMENT OF HYPERTENSION VIA THE INCREASE OF ARTERIAL STIFFNESS

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Objectives: It is noted that not only arterial stiffness but also abnormal pressure wave reflection are risks for the development of hypertension. However, the association between arterial stiffness and pressure wave reflection in the development of hypertension has not been fully clarified. The present study was conducted to examine whether the abnormal pressure wave reflection accelerates the development of hypertension via the increase of arterial stiffness.
Methods: In 3102 middle-aged healthy Japanese men without hypertension at baseline, systolic/diastolic blood pressures, brachial-ankle pulse wave velocity (baPWV), and radial augmentation index (rAI) were annually measured during a 9-year study period.
Results: In multivariate linear regression analysis and in mixed model linear regression analysis, baPWV was not longitudinally associated with rAI. Linear regression analysis demonstrated that the higher rAI at the baseline was associated with the larger longitudinal increase of baPWV ($beta = 0.17$, $p < 0.01$). At the end of study period, 404 subjects were developed to hypertension. The prevalence rate of the development of hypertension during the study period was higher in subjects with higher baPWV and higher rAI at the baseline (220 in 939 subjects: 23%) than that in other 3 groups classified by the status of baPWV and rAI at the baseline (e.g. 52 in 942 subjects with low baPWV combined with low rAI: 6%, $p < 0.01$).
Conclusion: The abnormal pressure wave reflection, which may be derived from both arterial stiffness and peripheral vascular damages, may be an accelerator for the development of hypertension via the increase of arterial stiffness.