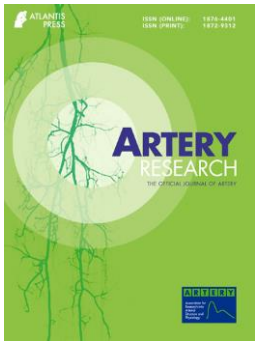

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

P4.57: STIFFNESS OF THE LARGE ARTERIES IN INDIVIDUALS WITH AND WITHOUT DOWN SYNDROME

A. Rodrigues, L. Coelho, W. Gonçalves, M. Vasconcellos, R. Cunha, S. Gouvea, G. Abreu

To cite this article: A. Rodrigues, L. Coelho, W. Gonçalves, M. Vasconcellos, R. Cunha, S. Gouvea, G. Abreu (2012) P4.57: STIFFNESS OF THE LARGE ARTERIES IN INDIVIDUALS WITH AND WITHOUT DOWN SYNDROME, Artery Research6:4, 199–199, DOI: https://doi.org/10.1016/j.artres.2012.09.204

To link to this article: https://doi.org/10.1016/j.artres.2012.09.204

Published online: 21 December 2019

subjects aged 71±6 yrs (mean±SD) from an existing community study. Incremental LV elastances at the start (E_{se}) and end of ejection (E_{ee}) were calculated as the ratio of dp/dt and dV/dt at corresponding positions in time. In our analysis we considered the inter-relationships of E_{ee} with heart rate (HR), systolic blood pressure (SBP), pressure relaxation rate (dp/dt_{ee}), arterial compliance (SV/PP), mitral annulus velocity e', E/e', ejection fraction (EF) and the classic non-invasive ventricular-vascular coupling index (Ees/Ea). Univariate correlations (Table) as well as stratification according to lower and higher E_{ee} groups showed that a higher E_{ee} was associated with a higher E_{se} and with lower arterial compliance and reduced ventricular relaxation rate (e'), despite increased dp/dt_{ee}. EF and Ees/Ea were not associated with any of these measures.
Conclusions: An increased E_{ee} reflects slowed ventricular relaxation, which may be due to the impact of reduced arterial compliance on LV diastolic performance. The classic non-invasive ventricular-vascular coupling index Ees/Ea did not reveal such a relationship.

P4.57
STIFFNESS OF THE LARGE ARTERIES IN INDIVIDUALS WITH AND WITHOUT DOWN SYNDROME

A. Rodrigues [1], L. Coelho [1], W. Gonçalves [1], M. Vasconcellos [1], R. Cunha [2], S. Gouvea [2], G. Abreu [2]
[1]*School of Medicine, University Center of Espírito Santo, Colatina, Brazil*
[2]*Postgraduate Program in Physiological Sciences, Federal University of Espírito Santo, Vitória, Brazil*

Background: Down syndrome (DS) is known to cause premature aging in several organ systems[1]. In this controlled study, the possibility of changes in the large arteries due to aging was evaluated in patients with DS[1].
Methods: Eighty-two subjects of both genders were selected. The DS group had 41 active subjects. The control group was consisted of 41 healthy matched for age and gender. Carotid–femoral pulse wave velocity was obtained as an index of aortic stiffness using an automatic noninvasive method[2].
Results: The general characteristics of the groups and the main results are shown in Table 1 and Figure.
Conclusion: Despite evidence in the literature that patients with DS undergo early aging[1], this process does not seem to affect the large arterial trunks[3]. Considering that DS presents with chronic hypotension, it is reasonable to propose that the prolonged reduction of arterial distending pressure may contribute to functional preservation of the arteries in patients with Down syndrome
Keywords: aging, Down syndrome, pulse wave velocity, arterial stiffness

References

1 Nakamura E, Tanaka S. Biological ages of adult men and women with Down's syndrome and its changes with aging. *Mech Ageing Dev.* 1998; 105:89–103.
2 Asmar R, Benetos A, Topouchian J, et al. Assessment of arterial distensibility by automatic pulse wave velocity measurement. *Hypertension.* 1995; 26:485–490.

Table 1 Anthropometric and hemodynamic characteristics in the experimental groups.

	DOWN SYNDROME (n=41)	CONTROL (n=41)	*P* value
Age (years) Variance	21 ± 1 (13-42)	21 ± 1 (13-42)	-
Weight (kg)	55 ± 2	61 ± 2	< 0.05
Height (m)	1.47 ± 0.01	1.64 ± 0.01	< 0.001
BMI ($kg.m^{-2}$)	25 ± 1	22 ± 1	< 0.01
WHR	0.88 ± 0.01	0.80 ± 0.01	< 0.001
SBP (mmHg)	106 ± 2	117 ± 2	< 0.001
DBP (mmHg)	66 ± 2	77 ± 2	< 0.001
MAP (mmHg)	80 ± 1	90 ± 1	< 0.001
HR(bpm)	74 ± 2	76 ± 2	NS
PWV (m/s)	7.51 ± 0.14	7.84 ± 0.12	< 0.05

Data are expressed as mean ± standard error (SEM).
Abbreviations: DS = Down syndrome; BMI = body mass index; WHR = waist-hip ratio; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; PWV= pulse wave velocity; NS = not significant.

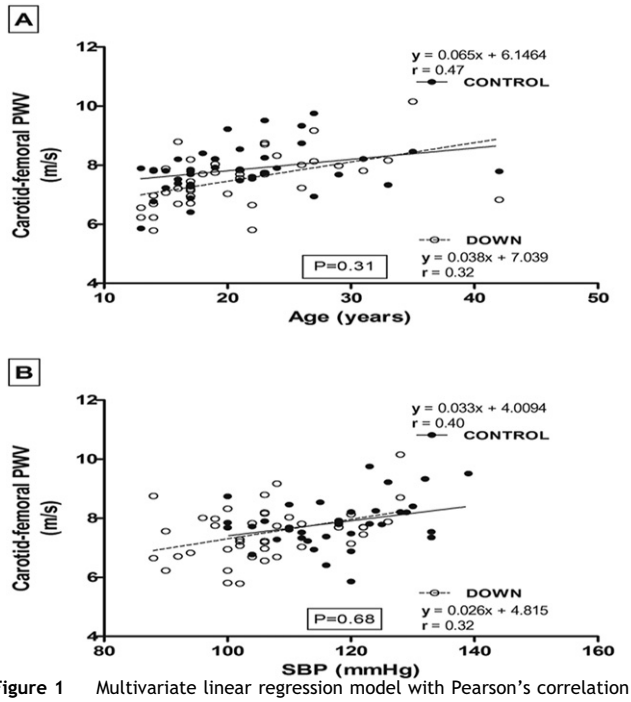

Figure 1 Multivariate linear regression model with Pearson's correlation coefficient between age, systolic blood pressure, and carotid–femoral pulse wave velocity in controls and subjects with Down syndrome.

3 Rodrigues AN, Coelho LC, Gonçalves WLS, at al. Stiffness of the large arteries in individuals with and without Down syndrome. *Vascular Health and Risk Management* 2011; 7: 375–381

P4.59
ASSOCIATION BETWEEN ENDOTHELIAL NO SYNTHASE POLYMORPHISM AND AORTIC STIFFNESS

J. Seidlerova [1], J. Filipovsky [1], O. Mayer[1], R. Cifkova [1,2], M. Pesta [3], J. Vanek [1]
[1]*Department of Internal Medicine II, Faculty of Medicine, Charles University, Pilsen, Czech Republic*
[2]*Department of Preventive Cardiology, Thomayer Faculty Hospital, Prague, Czech Republic*
[3]*Laboratory of Genetics, Faculty of Medicine, Charles University, Pilsen, Czech Republic*

Background: Recently, rs3918226 polymorphism in the promoter region of endothelial NO synthase (NOS3) was strongly associated with arterial hypertension in a large genome-wide association study*. We investigated whether this polymorphism is associated with arterial phenotypes in a Czech general population.
Methods: In a pilot study, we genotyped 101 untreated subjects (age, 54.0 years; 51.5% women, 30.7% smokers). Arterial properties were measured using SphygmoCor. In multivariate-adjusted analyses, we assessed effect of **rs3918226 on** aortic pulse wave velocity (aPWV) and augmentation index (AIx). As independent covariates we considered sex, age, MAP, heart rate and smoking.
Results: Frequency of rs3918226 genotypes were CC 85.2%, CT 14.8%, and TT 0%. Carriers of mutated T allele tended to have higher both aPWV (8.59±0.45 vs. 7.77±0.18 m/s; P=0.098) and AIx (91.77±3.56 vs. 85.89±1.45%; P=0.13) compared to CC homozygotes. These associations were modified by smoking. In smokers we observed similar trend as in the whole population ($0.067<P<0.19$), while in nonsmokers we did not find any association ($P\geq0.50$). We did not observe any association between blood pressure and the polymorphism under study ($P\geq0.67$).
Conclusion: This is first study to explore the association of rs3918226 polymorphism in NOS3 gene with arterial properties. We found marginally higher aPWV and AIx in carriers of mutated T allele in this pilot study. We hypothesize that genetic modulation of intermediate arterial phenotypes might lead to higher blood pressure. As the prevalence of