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06.01: LIFE-COURSE OF MEAN ARTERIAL PRESSURE AND ITS IMPACT ON ARTERIAL STIFFNESS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY (AGAHLS)

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differences for AIx were not observed (change -0.62% and +0.44%, $p=0.48$). As expected, a significant reduction in CRP (-9.2 mg/l, $p=0.011$) and DAS28 for the RA patients (-0.73, $p=0.002$) was observed in the treatment group, but we did not find significant correlations between change in aPWV and CRP in the entire treatment group ($r=0.055$, $p=0.785$) and between change in aPWV and DAS28 in the RA group ($r=0.091$, $p=0.737$).

Conclusion: These findings indicate that anti-TNF-alpha therapy ameliorates functional parameters of early atherosclerosis. However, changes in aPWV were not correlated to improvement in markers of inflammatory activity.

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04.03

LIFE-COURSE HABITUAL PHYSICAL ACTIVITY AND ITS IMPACT ON ARTERIAL STIFFNESS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY (AGAHLs)

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Purpose: To examine how the development over time (i.e. from adolescence to adulthood) of habitual physical activity (HPA) impacts on arterial stiffness (AS) of both the elastic carotid (CCA) and the muscular femoral (CFA) arteries in adulthood.

Methods: Longitudinal data on HPA (expressed in metabolic equivalents/week – METs/wk) were retrieved from the AGAHLs ($n=373$, 196 women, 8 follow-up measures between the ages of 13 and 36 yrs). AS (i.e. CCA and CFA distensibility and compliance coefficients and CCA's Young's elastic modulus) was assessed by non-invasive ultrasonography when subjects were 36 yrs old; a sex-specific AS score for each artery was calculated by averaging the height and MAP-adjusted z-scores of each of these estimates. Generalized estimating equations were used to compare the mean levels of HPA throughout the 24-yr follow-up period between those subjects with 'stiffer' (i.e. lowest quartile) vs. 'normal' (highest 3 quartiles of AS score) arteries at the age of 36.

Results: Compared to subjects with 'normal', those with 'stiffer' CCA and CFA arteries had spent, on average and throughout the longitudinal period, 376 and 500 less METs/week on HPA (corresponding to @ 9 to 12 min/day of light-to-moderate intensity bicycling), respectively (Table). Adjustments for other risk factors (RFs), in particular cardiopulmonary fitness, explained these differences to a large extent for the CCA, but other RFs may also explain the association between HPA and CFA stiffness.

Conclusion: Promoting increases in HPA during adolescence and throughout the course of life may prevent the development of AS, partially due to its beneficial effects on fitness and other cardiovascular RFs.

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06.01

LIFE-COURSE OF MEAN ARTERIAL PRESSURE AND ITS IMPACT ON ARTERIAL STIFFNESS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY (AGAHLs)

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Model: adjustments	Stiff vs. normal CCA		Stiff vs. normal CFA	
	β	95% CI	β	95% CI
1. time, sex, height, smoking, alcohol, energy intake	-376	-724; -27	-500	-839; -161
2. + body fatness (sum of 4 skinfolds)	-317	-668; 34	-471	-810; -132
3. + cardiopulmonary fitness (VO_2 max)	-172	-510; 166	-386	-712; -60
4. + blood lipids (total-to-HDL cholesterol ratio)	-234	-585; 117	-489	-826; -152
5. + resting heart rate	-328	-670; 15	-461	-794; -128
6. + systolic blood pressure	-350	-709; 8	-482	-828; -136
7. + all variables in models 2 to 6	-145	-490; 201	-422	-750; -93

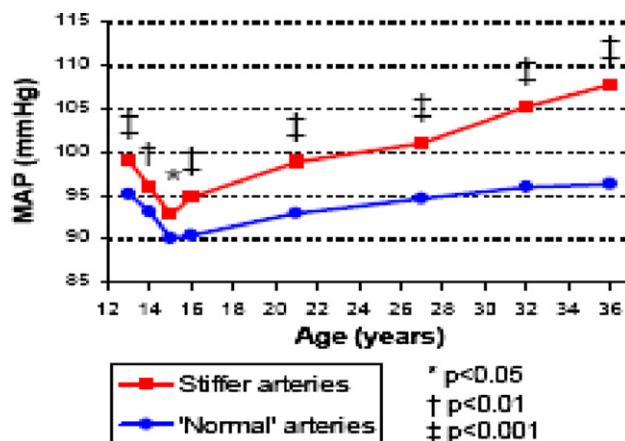
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Purpose: To investigate how the development over time (i.e. from adolescence to adulthood) of mean arterial pressure (MAP) impacts on arterial stiffness in adulthood.

Methods: Longitudinal data on systolic (SP) and diastolic (DP) blood pressure were retrieved from the AGAHLs ($n=373$, 196 women; 8 follow-up measures between the ages of 13 and 36 yrs). MAP was calculated as $[(2 \times DP) + SP]/3$. Arterial stiffness (i.e. carotid, brachial and femoral distensibility and compliance coefficients) was assessed by non-invasive ultrasonography when subjects were 36 yrs old; a sex-specific total stiffness score was calculated by averaging the height and local MAP-adjusted z-scores of each of these estimates. Generalized estimating equations were used to compare the mean levels (and the patterns of development) of MAP throughout the 24-yr follow-up period between subjects with 'stiffer' (i.e. lowest quartile) vs. 'normal' arteries (highest 3 quartiles of the total stiffness score) at the age of 36.

Results: Compared to subjects with 'normal', those with 'stiffer' arteries had, on average, 6.36 mmHg (95%CI: 5.04; 7.67) greater levels of MAP throughout the longitudinal period. These differences were already present in adolescence and were further amplified thereafter with subjects with stiffer arteries showing a steeper increase in MAP between adolescence and age 36 (Figure). Adjustments for other risk factors (i.e. smoking behaviour, energy and alcohol intake, physical activity, body fatness, blood lipids and heart rate) only slightly attenuated these differences.



Conclusion: Blood pressure monitoring should start already in early age in order to avoid/delay arterial stiffening and related cardiovascular complications.

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06.02

RELATION OF AORTIC STIFFNESS WITH ECHOCARDIOGRAPHIC INDICES OF LEFT VENTRICULAR DIASTOLIC FILLING AND LONGITUDINAL VELOCITIES IN SUBJECTS FREE OF CLINICAL CARDIOVASCULAR DISEASE

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Introduction: a relation between aortic stiffening (AS) and LV systolic dysfunction and hypertrophy is established in the elderly. A relation between AS and LV diastolic function can be hypothesized, mediated by age and increased LV mass (LVM).

Aim: to verify whether AS may affect LV diastolic function independently of LVM and age in subjects with preserved systolic function.

Methods: 144 subjects below 65 years, (59 controls: age 40 ± 12 , MBP 84 ± 7 mmHg; and 85 patients with at least one major risk factor, free of CV disease, age 42 ± 16 , mean BP 96 ± 12 mmHg). LV mass, systolic function, diastolic filling