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1.4: CHILDHOOD DETERMINANTS OF EARLY ADULT ARTERIAL STIFFNESS IN DIFFERENT ETHNIC GROUPS

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time elapsed since smoking (<5 years, 5–15 years and > 15 years). Subjects had at the beginning and end of the study determinations of carotid-femoral pulse wave velocity (PWV) and common carotid intima-media thickness. Based on these measurements the annual absolute changes were calculated. **Results:** Smoking at baseline was not associated with statistically significant differences in PWV and CIMT. However, the annual change of PWV was statistically different between the groups of smokers, non-smokers and the 3 groups of ex-smokers ($p = 0.041$) after adjustment for relevant confounders. Specifically, smokers had 0.23 m/s/year (95% CI: 0.10 to 0.35), non-smokers 0.17 m/s/year (95% CI: 0.08 to 0.25), quitters (<5 years) had 0.28 m/s/year (95% CI: 0.07 to 0.49), quitters (5–15 years) had 0.35 m/s/year (95% CI: 0.11 to 0.59) and quitters (>15 years) -0.07 m/s/year (95% CI: -0.26 to 0.13). Similar trend for slower progression was observed for CIMT in past smokers (>5 years) but this was not statistically significant.

Conclusions: Quitting smoking slows down progression of vascular aging after many years, implying a period of adjustment for former deleterious effects of smoking.

1.4

CHILDHOOD DETERMINANTS OF EARLY ADULT ARTERIAL STIFFNESS IN DIFFERENT ETHNIC GROUPS

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Childhood determinants of aortic pulse wave velocity [PWV] are poorly understood. We tested how factors measured twice previously in childhood in the MRC 'DASH' study, particularly body mass (BMI) components and BP, affected PWV in young adults.

Methods: Of 6643 London children, aged 11–13 y, from 51 schools in samples of about 1000 in 6 ethnic groups, with markedly different adult cardiovascular risk, 4785 (72%), were seen again at 14–16 y. In 2013, 666 (97% of invited) took part in a young-adult pilot (21–23 y) follow-up. With psychosocial, anthropometric and BP measures, PWV was recorded via an upper arm cuff on the calibrated Arteriograph device. In a subsample ($n = 334$) PA was measured over 5 days via the ActivPal.

Results: Unadjusted PWVs in Black Caribbean and White UK young men were similar (mean±SD 7.9 + 0.3 vs 7.6 + 0.4 m/sec) and lower in other groups at similar systolic (s)BPs (120 mmHg) and BMIs (24.6 kg/m²). In fully adjusted regression models, independent of BP effects, while Black Caribbean (higher BMIs and waists), Black African and Indian young women had lower PWV (by 0.5–0.8, 95%CI 0.1–1.1 m/sec) than White UK women (6.9 + 0.2 m/sec), values were still increased by age, BP, powerful impacts from waist/height and time spent sedentary but a reducing impact of time walking >100steps/min (both $p < 0.01$), with a racism effect (+0.4 m/sec) in women. Childhood effects of waist/hip were also detectable.

Conclusion: Even by young adulthood, increased waist/height ratios, lower physical activity, BP and psychosocial variables (eg: perceived racism) are independent determinants of arterial stiffness, likely to increase with age.

1.5

RELATION OF ARTERIAL STIFFNESS WITH LEFT VENTRICULAR DIASTOLIC FUNCTION IN GENERAL POPULATION

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Left ventricular diastolic function declines with aging and hypertension. It is well known that elevated blood pressure results in increased arterial stiffness. The study aims to determine the relationship between arterial stiffness and left ventricular diastolic function in general population.

Methods: We recruited 303 (mean age, 46.9 years; 167 women, 155 normotensives) members of randomly recruited families. Normotension and

hypertension were diagnosed based on both office and ambulatory blood pressure measurements, or history of antihypertensive treatment. Pulse wave velocity (PWV), peripheral and central pulse pressure (pPP; cPP) were evaluated by means of pulse wave analysis. Left ventricle (LV) diastolic function was determined by measuring transmitral (early (E) and late (A) diastolic peak velocities and E/A ratio) and pulmonary (peak systolic (S) and diastolic (D) velocity and S/D ratio) flow velocities and diastolic velocities of septal and lateral mitral annulus (E' wave and E/E' ratio) obtained in tissue doppler. Additionally we measured left atrium diameter (LAD).

Results: After adjusting for relatedness, pPP, cPP and PWV were negatively associated with E/A, and positively with E/E' and S/D ($P < 0.001$). In multivariate analysis the most closely related parameters were: cPP with E/E' ($\beta = 0.04$, $P = 0.001$), cPP with S/D ($\beta = -0.004$, $P = 0.011$), and pPP with E/E' ($\beta = -0.03$, $P = 0.003$). Additionally pPP was associated with LAD ($\beta = 0.058$, $P = 0.011$). In hypertensives pPP and cPP related both to E/E' and S/D ($p < 0.01$).

Conclusions: Our study suggested that increased arterial stiffness as estimated by pulse pressure measurement might be considered as a determinant of left ventricular diastolic dysfunction.

1.6

THE BODE INDEX PROGNOSTIC SCORE IS AN INDEPENDENT DETERMINANT OF ARTERIAL STIFFNESS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Introduction: COPD is associated with increased cardiovascular risk, independent of established risk factors. Arterial stiffness is a surrogate of cardiovascular risk and we sought to determine its relationship with COPD severity and prognosis in the ERICA (Evaluation of role of inflammation in airways disease) multi-site UK study: the largest cohort study focusing on cardiovascular manifestations in COPD.

Methods: Spirometry and haemodynamic measures (aortic pulse wave velocity (aPWV), augmentation index (AIx)) were performed in 729 COPD subjects aged ≥ 40 years. COPD severity was classified by BODE Index [BMI (low BMI worse prognosis), Obstruction (FEV1), Dyspnoea (mMRC score), Exercise tolerance (6-minute walk distance) high BODE index: worse outcome], a validated score based on clinical variables and an independent predictor of mortality in COPD.

Results: Mean aPWV was 10.3 (SD 2.6) m/s, AIx 27 (10)%. BODE correlated with aPWV ($R = 0.2$, $p = 0.0001$) and this was maintained when adjusted for study site, age, supine HR and MAP, years smoked and cardiovascular comorbidities (MI, stroke, diabetes, peripheral vascular disease), ($\beta = 0.2$, $p = 0.0001$). BODE was also a determinant of AIx when adjusted for site, age, seated HR and MAP, years smoked and cardiovascular comorbidities ($\beta = 0.1$, $p = 0.02$).

Conclusions: BODE is associated with arterial stiffness in COPD, independent of traditional risk factors. Its composite variables are not on the causal pathway for vascular stiffness, so its association likely reflects patient susceptibility to smoke injury in the lungs and vasculature. BODE may also enhance cardiovascular risk stratification in COPD, since its relationship with stiffness was independent of self-reported cardiovascular comorbidities.

2.1

A METHOD FOR THE MEASUREMENT OF PRESSURE SENSITIVITY OF CAROTID-FEMORAL PULSE WAVE VELOCITY IN HUMANS

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Background: Carotid-femoral pulse wave velocity (cfPWV), a marker of cardiovascular disease, is modified by both blood pressure and changes in