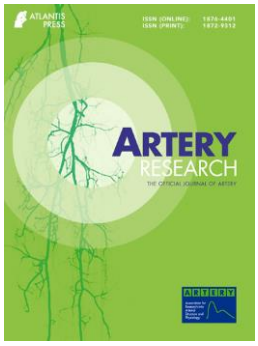

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2.5: IN SINGLETONS BORN AT TERM, LOWER GESTATIONAL AGE IS ASSOCIATED WITH INCREASED AORTIC PULSE WAVE VELOCITY IN YOUNG ADULTHOOD: THE NORTHERN IRELAND YOUNG HEARTS PROJECT (NIYHP)

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Conclusion: Ambulatory aPWV, estimated by an operator-independent method, provides additional information to cfPWV regarding the associations of arterial stiffness with the retinal microcirculation.

2.5
IN SINGLETONS BORN AT TERM, LOWER GESTATIONAL AGE IS ASSOCIATED WITH INCREASED AORTIC PULSE WAVE VELOCITY IN YOUNG ADULTHOOD: THE NORTHERN IRELAND YOUNG HEARTS PROJECT (NIYHP)

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Decreases in the mean gestational age of babies born at term have been reported over the past decade in several developed countries, linked to increases in the rates of planned births by labour induction and/or pre-labour caesarean sections. In contrast to the effects of pre-term birth, the extent to which lower gestation age within the 'at-term' range (i.e. $\geq$37−$\leq$42 weeks) affects individuals' cardiovascular heath is largely unknown, however. We have therefore examined the association between gestational age (obtained from the Northern Ireland Child Health Services' records) and aortic pulse wave velocity (aPWV) in 351 young adults from the NIYHP (50.4% women, mean age of 22.4 $\pm$ 1.6 years, all singletons and born at term, 98% with birth weight>2.5 kg). In analyses adjusted for age, sex, birth weight (in SDs relative to UK's 1990 reference), birth order, breast-feeding, maternal and paternal age at child's birth, and social economic status, we found that each week increase in gestational age was significantly associated with lower levels of aPWV [standardized ß = −0.11 (95% CI:−0.21;−0.01, p = 0.039)]. Additional adjustments for individuals' adult BMI and mean arterial pressure did not appreciably affect this association. None of the other birth covariates were independently associated with aPWV. These findings suggest that lower gestational age, even within the at-term range, may be a key determinant of early vascular ageing as each additional week conferred benefits. This aspect may have been neglected by the over-simplistic characterization of individuals as 'born at-term' and may have clinical implications for policies around planned deliveries, given the current trends.

2.6
PULSE WAVE VELOCITY AND GAIT PERFORMANCE IN OLDER SUBJECTS

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Background: Arterial stiffening is an age-related change and is a well-known cardiovascular risk factor but its association with physical decline is rarely evaluated.
The aim of this analysis was to assess the association of arterial stiffness as carotid-femoral pulse wave velocity (PWV) with gait performance in older subjects.
Methods: PWV was measured with Complior device. In all subjects was assessed: gait speed (V), Timed Up&Go test (TUG), handgrip strength, personal (ADL) and Instrumental Activities of Daily Living (IADL). Body composition was assessed with DXA, nutritional status with Mini Nutritional Assessment. Standard blood laboratory tests and markers of inflammation (hsCRP, IL-6, pentraxin3-PTX3, osteoprotegerin-OPG, TNFα soluble receptor2-sTNFR2) were determined.
Results: Mean age of 69 subjects (53—96 yrs) was 72.5 $\pm$ 9.8 yrs. Mean number of diseases was 5.3 $\pm$ 2.2, and of used medications was 7.0 $\pm$ 2.5. Subjects with PWV < 10 m/s and $\geq$10 m/s did not differ in systolic (SBP) and diastolic blood pressure, heart rate, number of diseases and medications, IADL, ADL, handgrip strength. Patients with PWV < 10 m/s were younger (67.8 $\pm$ 6.4vs74.8 $\pm$ 10.4 yrs; p = 0.004), had higher V (1.02 $\pm$ 0.31vs0.798 $\pm$ 0.23 m/s; p = 0.006), lower TUG (9.69 $\pm$ 2.6vs11.81 $\pm$ 4.56; p = 0.02), higher mdrd (76.3 $\pm$ 21.4 vs 62.87 $\pm$ 20.3 ml/min/m^2) and lower legs' fat content (LEfat) (6433.1 $\pm$ 1934.2vs 8046.4 $\pm$ 3187.5g; p = 0.047). PWV correlated positively with age (r = .47, p < 0.0001), TUG (r = 0.26, p = 0.037), negatively with V (r = −0.37, p = 0.003), handgrip strength (r = −0.30, p = 0.015), ADL (r = −0.28, p = 0.02).
In multiple regression analysis gait speed was negatively associated with PWV (β = −0.37; p = 0.0075), female gender (β = −0.36; p = 0.045) and TUG (β = −0.443; p = 0.0038), and positively with Hb (β = 0.30; p = 0.045), PTX3 (β = 0.608; p = 0.001), sTNFR2 (β = 0.374; p = 0.035).
Conclusions: Artery stiffness, apart from female gender and inflammation, may be associated with poorer gait performance in older subjects.

3.1
PREDIABETES IS ASSOCIATED WITH IMPAIRED RETINAL VASODILATION: THE MAASTRICHT STUDY

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Aim: Type 2 diabetes (DM2) causes microvascular dysfunction (MVD). In addition, MVD can contribute to insulin resistance, predisposing to DM2. This hypothesis predicts that MVD should be present in impaired glucose metabolism (IGM; prediabetes). However, population-based studies of MVD and glucose metabolism are not available. We investigated this using the retinal arteriolar dilator response to flicker light.
Methods: In a population-based study (n = 2205), we determined retinal %-dilation (Dynamic Vessel Analyzer; Imedos, Germany) and glucose metabolism status (OGTT; classified as normal (NGM), IGM or DM2). Differences were compared with multivariable regression adjusted for age, sex, BMI, smoking, systolic-BP, lipid profile, retinopathy, (micro)albuminuria, the use of lipid-modifying and/or blood-pressure-lowering medication and prior cardiovascular disease.
Results: 1263 individuals had NGM (42% men, aged 58 $\pm$ 8 years (mean $\pm$ SD)), 336 IGM (61% men, aged 61 $\pm$ 7 years) and 606 (due to oversampling) DM2 (69% men, aged 63 $\pm$ 8 years). Arteriolar %-dilation was median 3.51, IQR 1.47 to 5.95, range −5.69 to +19.71. %-dilation (mean $\pm$ SD) was 4.42 $\pm$ 3.45 in NGM, 3.77 $\pm$ 3.06 in IGM, and 3.26 $\pm$ 3.27 in DM2. Adjusted analyses showed decreased %-dilation in IGM (β = −0.461, p = 0.03) and DM2 (β = 0.559, p = 0.01) vs NGM.
Conclusion: IGM and DM2 are associated with reduced flicker-light-induced retinal arteriolar dilation, independently of major cardiovascular risk factors. These findings support the concept that MVD precedes and thus may contribute to DM2.

3.2
ORIGINS OF THE BACKWARD TRAVELING WAVE IN THE ARTERIAL TREE

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Backward traveling waves, an important determinant of central haemodynamics, are usually regarded as being due to reflections from discontinuities in the arterial tree. However, consideration of a single tube model of the arterial with a single site of reflection shows that a backward pressure wave may be generated by elastic recoil of large arteries, in which case the magnitude of the backward wave is proportional to that of the forward wave. A 55-segment 1-D model of the arterial which allows reflection as a continuum along the arterial tree and, for a given prescribed aortic flow, generates physiological aortic pulse waveforms was used to examine the relation of the backward to forward pressure waves in 4107 "virtual subjects" with arterial parameters spanning the physiological range. Backward pressure wave was closely correlated with the forward wave (R = 0.931, P < 0.001). Clinical data was obtained by carotid tonometry and aortic Doppler sonography during modulation of cardiovascular function in healthy volunteers (n = 13, age 46.5 $\pm$ 10.1 years with inotropic, vasopressor and vasodilator drugs (dobutamine, norepinephrine phentolamine and nitroglycerin). The magnitude of backward pressure was highly correlated with forward pressure over a range 5—15 mmHg (R = 0.824, P < 0.001) with a constant ratio of backward to forward wave magnitude except during treatment with nitroglycerin, a vasodilator known to be highly selective for large muscular arteries. These numerical and experimental data suggest that backward pressure waves can