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### **6.2: IMPACT OF CHANGES IN SMOKING BEHAVIOUR BETWEEN ADOLESCENCE AND YOUNG ADULTHOOD ON ARTERIAL STIFFNESS IN YOUNG ADULTS. THE NORTHERN IRELAND YOUNG HEARTS PROJECT**

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## Oral Presentation Abstracts

### 6.1 LOW-GRADE INFLAMMATION AND ENDOTHELIAL DYSFUNCTION PRECEDE THE INCREASE IN PULSE PRESSURE IN TYPE 1 DIABETES: A 20-YEAR LONGITUDINAL STUDY

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**Purpose:** To investigate, longitudinally, whether increases in markers of inflammation (CRP, sICAM-1) and endothelial dysfunction (sICAM-1, sVCAM-1) are associated with and precede the increase in pulse pressure (PP) in individuals with type-1 diabetes (DM1).

**Methods:** Inception cohort of 277 DM1 patients (114 women; mean age at diagnosis: 27.5 ± 13.8 yrs) who were consecutively admitted, upon diagnosis, between Sep'79-Aug'84 to an outpatient diabetic clinic (Gentofte, Denmark). Throughout a follow-up period of >20 yrs, patients' PP, other risk factors, and CRP, sICAM-1 and sVCAM-1 were measured repeatedly (at 3-4 months intervals). Associations were analysed with generalized estimating equations (GEEs) and adjusted for sex, age at diagnosis, smoking, anti-hypertensive treatment and MAP.

**Results:** PP increased by 0.53 mmHg/yr, CRP by 0.033 mg/L/yr, sICAM-1 by 1.8 ng/ml/yr and sVCAM-1 by 1.8 ng/ml/yr over the 20-yr longitudinal period (all  $p < 0.001$ ). Higher levels of ln-CRP, sICAM-1 and sVCAM-1 were all significantly associated with higher levels of PP: 0.53 mmHg (95%CI: 0.05-1.00), 1.09 mmHg (0.53-1.66) and 0.94 mmHg (0.40-1.48) per SD increase in marker, respectively. In addition, levels of sICAM-1 and sVCAM-1, but not ln-CRP, at any time point, were also associated with increases in PP occurring in the 2 yrs thereafter: 0.56 mmHg (0.18-0.94), 0.53 mmHg (0.17-0.90) and -0.01 mmHg (-0.38-0.35) per SD increase in marker, respectively. Adjustments for other risk factors did not change these associations.

**Conclusion:** Life-course increases in low-grade inflammation/endothelial dysfunction are associated with and precede increases in PP, supporting the view of their involvement in the development of premature arterial stiffening in diabetes.

### 6.2 IMPACT OF CHANGES IN SMOKING BEHAVIOUR BETWEEN ADOLESCENCE AND YOUNG ADULTHOOD ON ARTERIAL STIFFNESS IN YOUNG ADULTS. THE NORTHERN IRELAND YOUNG HEARTS PROJECT

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**Purpose:** to investigate 1) the extent to which young adults who started smoking during adolescence and persisted doing so during young adulthood ('persistent smokers'—PS), and those who started smoking in young adulthood only ('starters'—SS) had greater aorto-iliac PWV than never smokers (NS); and 2) whether any such differences could be explained by greater

levels of inflammation and/or endothelial dysfunction (ED) associated with smoking.

**Methods:** We studied 408 young adults (age 22.6 ± 1.6 yr; 209 women) from the NIYHP in whom PWV and plasma levels of inflammation (i.e. CRP and fibrinogen) and ED (i.e. vWF and tPA-antigen) were measured during young adulthood, and smoking status was assessed during adolescence (age 15) and young adulthood. Data were analysed using linear regression with adjustments for potential confounders and/or mediators. **Results:** After adjustment for age, sex, height and MAP, and other lifestyle risk factors, PS, but not SS, had higher levels of PWV compared with NS [+0.20 m/s (95%CI: 0.07; 0.33),  $p = 0.003$ , and -0.02 m/s (-0.13; 0.08),  $p = 0.667$ , respectively]. Further adjustments for inflammation and ED did not materially attenuate the differences between PS and NS [to +0.19 m/s (0.06; 0.33),  $p = 0.006$ ], despite the fact that PS had higher levels of inflammation [+0.29 SD (0.05; 0.52),  $p = 0.017$ ] and ED [+0.20 SD (0.01; 0.39),  $p = 0.037$ ].

**Conclusion:** Persistent smoking between adolescence and young adulthood adversely impacts on arterial stiffness, as well as on levels of inflammation and endothelial-dysfunction. However, these pathophysiological mechanisms did not explain the increased arterial stiffness related to persistent smoking.

### 6.3 DETERMINING EARLY CARDIOVASCULAR RISK PROFILES IN PAEDIATRIC RHEUMATIC DISEASE

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**Objectives:** Increased cardiovascular risk in adults with systemic lupus erythematosus (SLE) and rheumatoid arthritis, not explained by exposure to traditional factors alone, have prompted investigation into the role of chronic inflammation. This study aimed to determine early cardiovascular risk profiles and their correlates in children with SLE, systemic juvenile inflammatory arthritis (SJIA) and juvenile dermatomyositis (JDM).

**Methods:** Disease activity and drug therapy were recorded, fasting lipid, glycemic and inflammatory profiles performed, and vascular testing including carotid intima-media thickness (CIMT), flow-mediated dilatation (FMD) and pulse wave velocity (PWV). Data within groups were compared to normal controls and between groups using parametric methods.

**Results:** Of 137 subjects, SLE ( $n = 88$ ) were older and more predominantly female than SJIA ( $n = 21$ ) and JDM ( $n = 28$ ) age 15.4 ± 2.5 vs. 13.9 ± 2.4 and 13.9 ± 2.3 years, and female 83% vs. 57% and 50%, respectively. At testing, most had relatively healthy BMI, normal lipid and glycemic profiles and over mean follow-up 3.1 ± 3.0 years, 91% received corticosteroids (mean cumulative dose/kg 0.24 ± 0.30 g). Higher ESR, but lower complement C3 and C4 and albumin were found in SLE vs. SJIA and JDM, while CRP was lower in SLE vs. SJIA. Lower CIMT in SJIA ( $p < 0.05$ ) and higher PWV in SLE and JDM (both  $p < 0.001$ ) were found vs. controls. No between group differences for CIMT, FMD or PWV were found, even when adjusting for sex, age, BMI, disease duration or cumulative corticosteroid dose.