reactive protein [CRP]), physician-diagnosed chronic conditions (diabetes, hypertension, stroke and Coronary heart disease) and depressive symptoms ascertained with the Center for Epidemiologic Studies Depression Scale range 0 to 8. We controlled for age, sex and Apolipoprotein E (APOE).

Results Our findings suggest that experiencing financial hardship in childhood (20% of the sample) was associated with a higher risk of cognitive impairment (OR=1.55, 95% CI 1.15 – 2.09) in later life. A similar pattern was observed for having parents unemployed (7% of the sample) (OR=1.63, 95% CI 1.09 – 2.46) or physically abusive parents (3%) (OR=3.21, 95% CI 1.82–5.66). We also found that increased depressive symptoms were interlinked with higher cognitive impairment, while APOEe4 and inflammatory markers were not directly associated. However, inflammation was indirectly associated with cognitive impairment, via depressive symptoms (β=0.08, SE=0.03, p=0.020) and chronic conditions (β=0.39, SE=0.19, p=0.042).

Conclusion These findings support the psychosocial paradigm. They suggest that those from disadvantaged family backgrounds are more likely to have lower levels of education and be less wealthy, which in turn lead to poorer health, such as higher overall inflammation and increased depressive symptoms. These findings provide a plausible explanation for inequalities in late-life cognitive health.

OP25 THE SEX-SPECIFIC ASSOCIATION BETWEEN MITOCHONDRIAL DNA HAPLOGROUPS AND TRAJECTORIES OF CARDIOMETABOLIC RISK FACTORS DURING CHILDHOOD AND ADOLESCENCE: A PROSPECTIVE COHORT STUDY

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Background The sex-specific aetiology of cardiometabolic risk across the life course is well established but remains poorly understood. Mitochondria are essential for energy conversion in all cells. Mitochondrial DNA haplogroups have been implicated in the aetiology of cardiometabolic risk, though previous studies have not examined whether mitochondrial DNA haplogroups contribute to the sex-specific aetiology of cardiometabolic risk. We examined sex-specific associations between eight common European haplogroups and ten cardiometabolic risk factors in childhood and adolescence.

Methods Longitudinal data from the Avon Longitudinal Parents and Child Study, a prospective birth cohort study, was analysed. Cardiometabolic risk factors included systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse rate, triglycerides, high-density lipoprotein cholesterol (HDL-c), non-HDL-c, insulin, glucose, height-adjusted fat mass and height-adjusted lean mass, measured at research clinics when participants were approximately 7, 9, 10, 11, 13, 15 and 18 years old. Participants were genotyped using the SNP genotyping platform by Sample Logistics and Genotyping Facilities at the Wellcome Trust Sanger Institute and LabCorp (Laboratory Corporation of America). We used linear spline multilevel models to examine the association between mitochondrial DNA haplogroups and cardiometabolic risk factor trajectories across childhood and into adolescence. Analysis was stratified by sex to allow for differential effects between sexes. All trajectories were modelled in MLwiN version 2.36 called from Stata version 15.

Results Sample sizes for different outcomes range from 2,023 females and 1,962 males (6,518 total measures) for insulin up to 3,570 females and 3,689 males (17,039 total measures) for triglycerides. There was no strong evidence that haplogroups H,J,K,U,T and I are associated with cardiometabolic risk factors. In females, haplogroup X was associated with 3.22 mmHg (95% confidence interval (CI):0.4–6.0) lower SBP at age 7 and 1.29 kg (95%CI:0.4–2.2) lower lean mass at age 9 compared with haplogroup H. Similar associations were observed at age 18, albeit with CIs spanning the null. Haplogroup X was also associated with 15.5% (95%CI:2.5–28.5) lower fat mass at age 9 in females, although this association did not persist at age 18. Males with haplogroup W had higher HDL-c and lower non-HDL-c at birth, with decreasing and increasing rates of change, respectively, during the first 9 years of life. The associations did not persist at age 18.

Conclusion Mitochondrial DNA haplogroups X and W may play a role in the sex-specific aetiology of cardiometabolic risk during childhood and adolescence.

OP26 ADVERSE CHILDHOOD EXPERIENCES, POPULATION ATTRIBUTABLE RISK AND INCREMENTAL RISK OF CORONARY HEART DISEASE: A 13 YEARS FOLLOW-UP OF THE WHITEHALL II COHORT STUDY

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Background Adverse childhood experiences (ACEs), such as parental divorce, parental mental illness, parental separation, have been increasingly recognised as an upstream potential causal factor of development and premature mortality of coronary heart disease (CHD). No studies, however, have investigated attributable risk for incident CHD by each and all types of ACEs, and associations between combinations of ACEs and incident CHD.

Methods Among 5149 participants aged 35 to 55 at entry (1985–1988) to the Whitehall II cohort study with follow-up of 12.9 years, we examined; (i) associations between ACEs and incident CHD; (ii) what extent removal of each and all ACEs could eliminate incident CHD; and (iii) incremental risk of CHD in the association with ACEs combinations. Cox proportional hazard regression was applied to estimate hazard ratios and 95% confidence intervals. After identifying a model, we computed the average marginal effects, and hazard ratios and 95% confidence intervals for each ACEs combination. Incident CHD was identified through the Hospital Episode Statistics (HES) from Phase 5 (1997–1999) when ACEs were measured, to Phase 11 (2012–2013).

Results In the study sample, 65.6% had at least one ACE. After adjusting for sex, age, ethnicity, and childhood socioeconomic status, none of ACEs ('parental attachment', 'financial hardship', 'parental punishment', 'parental dysfunction', 'early-life parental separation', 'orphanage', 'hospitalisation') had statistically significant associations with incident CHD. All types of ACEs were attributed to approximately 13% of