Methods We did a causal mediation analysis using data from the UK CF registry, which captures 99% of all people with CF in the UK and records clinical information including weight and infection status at annual review visits. The exposure of interest was SEC in the first year of life measured by the index of multiple deprivation; the outcome was first lung function measure between ages 6 and 9. We were interested in mediation by weight trajectory during the first six years of life. Potential confounders were sex, year of birth, genotype and infection.

All children born between 2000 and 2010 and diagnosed by newborn screening were included in the analysis if they had at least one lung function measure between ages 6 and 9, at least one weight and infection measure between birth and age 6, and complete data on SECs and baseline confounders. We imputed missing data using multiple imputation by chained equations.

We used the parametric mediational g-formula to estimate the total effect of SECs on lung function, and the indirect effect mediated by weight trajectories in the first six years of life, accounting for potential time-varying confounding by infection. Confidence intervals were estimated using non-parametric bootstrap.

Results Using data from 853 children, we found a total effect of deprivation on lung function, measured by percent of predicted FEV1, of 4.53 percentage points (95% CI 3.44 to 5.77). Our results showed that if we could improve the weight of the most disadvantaged children to have the same distribution as that of the least disadvantaged children, their lung function would improve on average by 0.74 percentage points (95% CI 0.36 – 1.1).

Conclusion Only 16% (95% CI 8%-25%) of the inequalities in early lung function for people with CF were explained by weight trajectories in the first 6 years of life, suggesting that other important pathways to inequalities need exploration.

Wednesday 9 September

Life Course: Childhood

OP23 THE LESSER-KNOWN BREASTFEEDING PROBLEM: PREVALENCE AND DETERMINANTS OF PRELACTEAL FEEDING PRACTICE IN INDONESIA

LD Rahmartani*, C Carson, MA Quigley. *Nuffield Department of Population Health, University of Oxford, Oxford, UK*

10.1136/jech-2020-SSMabstracts.23

Background Prelacteal feeding (PLF) is anything other than breastmilk given to neonates before breastfeeding is established. There are numerous types of PLF and they are given for various reasons. Except when medically indicated, PLF is considered as one of the many potential barriers to optimal breastfeeding. However, PLF is practiced widely across the world. Meanwhile, it is still understudied and epidemiological research on the different types of PLF is limited in many settings, including in Indonesia. This study looks at the prevalence and determinants PLF in Indonesia, focusing on overall PLF and three common types (formula, other milk, and honey).

Methods A cross-sectional, secondary data analysis of the 2017 Indonesia Demographic and Health Survey was undertaken. The study population was 6168 ever-breastfeeding mothers whose last child was ≤23 month-old. Because PLF was a common outcome, modified Poisson regression was used to estimate the adjusted prevalence ratio (PR) for potential determinants and PLF.

Results By 2017, nearly half (45%) of mothers in Indonesia gave PLF. The most common feeds were infant formula (25%), any other milk (14%), plain water (5%), and honey (3%). Factors associated with higher prevalence of overall PLF were upper-middle (Q3-Q4) wealth quintiles (PR 1.17, 95% Confidence Interval (CI) 1.03–1.32 for Q3 and PR 1.18, 95% CI 1.04–1.33 for Q4), rural residence (PR 1.17, 95%CI 1.07–1.27), baby perceived to be small at birth (PR 1.26, 95%CI 1.14–1.38), and caesarean deliveries at either public (PR 1.34, 95%CI 1.19–1.51) or private facilities (PR 1.17, 95%CI 1.03–1.33). Conversely, mothers who gave birth to the second/subsequent child (PR 0.81, 95%CI 0.75–0.87) and mothers who possessed an antenatal card (PR 0.86, 95%CI 0.77–0.96) were less likely to give PLF. When analysed separately, formula displayed relatively similar risk factors to those of overall PLF, yet several associations varied among these three types of PLF. For instance, higher wealth quintiles and rural residence were risk factors for formula but not for other milk and honey. Furthermore, honey was more prevalent in home births than in deliveries at health facilities (PR 6.05, 95% CI 4.02–9.10), but formula and other milk were more frequent among caesarean deliveries. First-time birth was the only factor that showed a consistent association with overall PLF, formula, other milk, and honey.

Conclusion PLF is common in Indonesia but the prevalence and determinants vary by PLF type. Identifying high-risk groups, particularly by PLF type, is useful to plan more targeted interventions to improve breastfeeding practices.

OP24 THE MEDIATING PATHWAYS OF THE ASSOCIATION BETWEEN ADVERSE CHILDHOOD EXPERIENCES AND COGNITIVE HEALTH IN LATER LIFE

[1]D Cadar, [2]LK Kobayashi, [1]AP Steptoe, [3]P Demakakos*. *[1]Department of Behavioural Science and Health, University College London, London, UK; [2]Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, Michigan, USA; [3]Department of Epidemiology and Public Health, University College London, London, UK*

10.1136/jech-2020-SSMabstracts.24

Background Previous research has often shown that morbidity and disability are hinged to negative events and exposures that can accumulate over the life course, but less clear is their impact on late-life cognitive health. We assessed the biopsychosocial mechanisms influencing the associations between adverse childhood experiences and cognitive impairment at advanced ages.

Methods Data were from 3,130 dementia-free adults aged 50 + from the English Longitudinal Study of Ageing (ELSA) with data available from wave 3 (2006/07) to wave 8 (2016/17). ELSA provides a wide variety of psychosocial data collected via face-to-face computer-assisted personal interviews (CAPI) and self-completion questionnaires. All participants provided informed consent prior to their participation in the study. We used structural equation modelling to estimate direct and indirect associations between adverse childhood experiences (ACEs) and cognitive impairment (measured with 1.5 SD below the mean on the modified Telephone Cognitive Screening Interview scale range 0 to 35) via markers of SES (education and wealth), inflammation (serum fibrinogen and C-

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

[1]KN O'Neill*, [2]E Aubrey, [1]LM O'Keeffe. [1]*School of Public Health, University College Cork, Ireland*; [2]*MRC Integrative Epidemiology Unit, University of Bristol, UK*

10.1136/jech-2020-SSMabstracts.25

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

[1]M Akasaki*, [2]D Batty, [3]A Steptoe, [4]O Nicholas, [2]C Valencia-Hernández, [1]R Hardy, [3]J Abell. [1]*Department of Social Science, University College London, London, UK*; [2]*Department of Epidemiology and Public Health, University College London, London, UK*; [3]*Department of Behavioural Science and Health, University College London, London, UK*; [4]*Department of Statistical Science, University College London, London, UK*

10.1136/jech-2020-SSMabstracts.26

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