greater influence of multinational corporations and neoliberal trade organisations.

Conclusion Following a realist re-synthesis of a large systematically collected dataset, three final programme theories were supported that offer an account of how the welfare state, political tradition and globalisation may exert an important effect on child and maternal health. Limitations include lack of stratification by level of development, contextual effects in interpreting fertility rates, and complexity in mapping political exposures to political parties internationally. Future realist analyses could consider other political exposures, such as governance and political capacity.

P11 HOW DO PERINATAL RISK FACTORS MEDIATE THE RELATIONSHIP BETWEEN MATERNAL SOCIOECONOMIC STATUS AND PRETERM BIRTH? ANALYSIS OF ROUTINE HEALTHCARE DATA FROM THE NORTH WEST REGION OF ENGLAND

[1]Philip McHale*, [1]Daniela Schlüter, [1]Ben Barr, [2]Shantini Paranjothy, [3]Angharad Care, [1]David Taylor-Robinson. *[1]Department of Public Health, Policy and Systems, University of Liverpool, Liverpool, UK; [2]Aberdeen Health Data Science Research Centre, University of Aberdeen, Aberdeen, UK; [3]Centre for Women's Health Research, University of Liverpool, Liverpool, UK*

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Background Prevalence of preterm birth (PTB) is increasing in England. Given significant inequalities in prevalence by maternal socioeconomic status (SES) and subsequent negative consequences on health and educational attainment for children, this represents a significant risk of widening population health inequalities. The aim is to understand how different exposures to risks mediate the relationship between SES and PTB.

Methods Analysis of routinely collected data for all births at Liverpool Women's Hospital between April 2009 and March 2020, covering an area of North West England with high levels of deprivation. The exposure was a dichotomised measure of maternal SES at birth (two most deprived Indices of Multiple Deprivation deciles compared to other eight deciles), and the outcome was PTB (gestational age <37 weeks) compared with term. Mediators were maternal smoking status, medical conditions, obstetric conditions, and BMI at booking. Covariates included maternal age at birth, method of labour onset, parity, baby sex, and previous PTB. Mediation was assessed using multivariable logistic regression for modelling the effect of SES on PTB after adjusting for covariates (minimally adjusted) with and without adjustment for the potential mediators and applying difference-of-coefficients method; the difference in odds ratio (OR) for SES between these models. Proportion mediated is ($OR_{mediator\text{-}adjusted}$ - $OR_{minimally\text{-}adjusted}$)/($OR_{minimally\text{-}adjusted}$-1). All analysis was done in R.

Results Analysis sample consisted of 81,680 births; 7.4% were preterm, while 64.1% were to low SES mothers. Of mothers, 19.1% were current smokers, 4.3% had medical conditions, 10.1% had obstetric conditions and 48.6% had healthy weight BMI. The OR for the effect of low SES on PTB in the minimally adjusted model was 1.26 (95% CI 1.19–1.34). When mediators were included, OR reduced by 42% for smoking. OR for low SES did not change when maternal medical conditions or obstetric conditions were included, while OR increased when BMI was included (1.32, 95% CI 1.22–1.44).

Discussion Our findings demonstrate that significant inequalities exist in the prevalence of PTB. The initial results from the mediation analysis suggest that smoking is partly explains these inequalities. Over half of this inequality is not explained by mediators included here. Limitations include unavailable data on other mediators (e.g. maternal mental health) and in the analysis approach. The next step is to analyse the data using the counterfactual approach to mediation. Reducing PTB is a priority for Maternity and Children's services, and the NHS' Long Term Plan. These results suggest that action on smoking during pregnancy will reduce inequalities.

P12 INVESTIGATING CAUSALITY BETWEEN ADIPOSITY AND WOMEN'S REPRODUCTIVE FACTORS: A MENDELIAN RANDOMIZATION ANALYSIS

Claire Prince*, Gemma C Sharp, Laura D Howe, Abigail Fraser, Rebecca C Richmond. *MRC Integrative Epidemiology Unit, Bristol Medical School, University of Bristol, Bristol, UK*

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Background Reproductive factors (RFs) in women, such as menarche and menopause, the age at which women start and stop having children, and the number of children they have, have been implicating in disease risk including, breast cancer, cardiovascular, respiratory diseases. While body mass index (BMI) has similarly been identified as a risk factor in such diseases. Studies have identified strong bidirectional associations between adulthood BMI and RFs in women, and higher body size in girls was reported to be associated with earlier age at menarche and menopause. Considering the evidence of interplay between BMI and RFs, it is important to consider causal relationships which may underlie these associations. This study aims to appraise causality between both childhood and adulthood adiposity and the RFs, as well as establishing the magnitude of any identified relationships and direction of causality.

Methods We used data from the UK Biobank and genetic consortia with data available for RFs and BMI. We then applied Mendelian randomization (MR) methods to estimate the causal relationships between RFs and BMI both in childhood and adulthood. Sensitivity analyses were used to investigate directionality of the effect observed, to test for evidence of pleiotropy and to account for any sample overlap.

Results MR revealed body size and BMI in childhood and adulthood respectively have potentially causal relationships with a number of RFs, as well as a number of reproductive having a potentially effect on BMI in adulthood. Notably higher body size during childhood appears to lead to an earlier age at menarche (Beta (B)=-0.65, 95% confidence intervals (CI)=-0.74, -0.56), age at first, (B=-0.07, CI=-0.12, -0.01) and last birth (B=-0.09, CI=-0.13, -0.04). While higher BMI during adulthood seems to lead to an earlier age at menopause (B=-0.53, CI=-0.82, -0.23), age at first live birth (B=-1.72, CI=-2.06, -1.39), age at last live birth (B=-1.40, CI=-1.68, -1.13), earlier age at first sexual intercourse (B=-1.09, CI=-1.39, -0.78) and increased likelihood of having children (OR=1.11, CI=1.01, 1.21). Finally, findings suggests that earlier menarche (B=-0.03, CI= -0.04, -0.03) and age at first birth (B=-0.05, CI=-0.06, -0.03) and age first had sexual