with healthy non-obese participants only the metabolically unhealthy obese participants had elevated odds of incident depression (OR=1.56, 95% CI, 1.09 – 2.22), but not their metabolically healthy obese counterparts (OR=1.45, 95% CI, 0.92 – 2.30) nor unhealthy non-obese participants (OR=1.38, 95% CI, 0.98 – 1.94). In further analysis we examined the associations between individual metabolic risk factors and depression. There was a dose-response association between the number of metabolic risk factors and risk of depression, although the risk only became significant in participants with more than one risk factor. Adverse triglycerides, impaired glycaemic control, and low grade inflammation were associated with depression at follow-up in models adjusted for age, sex and baseline CES-D score.

Conclusion The association between obesity and risk of depressive symptoms appears to be partly dependent on metabolic health, although further work is required to confirm these findings.

Population Based Studies: Intergenerational

OP61 IS MATERNAL IRON STATUS ASSOCIATED WITH OFFSPRING'S BLOOD PRESSURE AND ADIPOSITY? A MENDELIAN RANDOMIZATION STUDY

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Background Iron deficiency during pregnancy is a common problem. Experimental animal studies suggest that mothers deficient in iron during pregnancy are more likely to have offspring who become obese and have higher blood pressure. The use of random assortment of genes from parents to offspring can provide a method for assessing the causal impact of nutritional exposures, which is less likely to be influenced by confounding and reverse causality. The C282Y mutation in the *HFE* gene is robustly associated with iron stores, with those who carry the mutation having higher iron stores. Thus, this variant could be used as an instrumental variable to examine whether the association of maternal iron with offspring body mass index (BMI), waist circumference (WC) and blood pressure (BP) is causal.

Methods We conducted a Mendelian randomization study to examine the association between maternal iron status with offspring adiposity and BP in adulthood. Instrumental variable (IV) analysis, using maternal C282Y as a genetic instrument for mother's ferritin, was performed. IV analysis uses the proportion of the variation in maternal ferritin that is explained by C282Y to provide an unconfounded estimate of the relationship with offspring outcomes. The results were compared to the results of multivariable ordinary least squares (OLS) regression examining the same relationship. Male and female offspring of mothers from the UK Women Cohort Study (UKWCS) were approached, of whom 348 with mean age of 41 years completed the study. About half were offspring of C282Y carriers. Offspring's BP, height and weight were measured at their local medical practice. Participants were also asked to self-measure their WC at home.

Results Maternal C282Y was associated with maternal ferritin (mean difference per allele=84 g/l, 95% CI 31, 137, P=0.002). Using IV analyses, maternal ferritin was not associated with offspring's BP, BMI or WC. The first stage F statistic for the strength of the instrument was 10 (Kleibergen-Paap *rk* LM P-value=0.009). Maternal ferritin was associated with offspring diastolic BP, WC and BMI in univariable, but not in multivariable OLS analysis. There was no strong statistical evidence of a difference between the OLS and the IV models coefficients for any of the outcomes considered.

Conclusion We found no association between maternal iron status and offspring's BP and adiposity using both multivariable OLS and IV modeling with maternal C282Y mutation as the instrument. Further exploration of this relationship is needed in larger studies that have genetic variation assessed in both mother and offspring.

OP62 THE INFLUENCE OF PRENATAL MATERNAL AND PATERNAL ANXIETY AND DEPRESSION ON CARDIOVASCULAR BIOMARKERS IN THE CHILD AT AGE 10: FINDINGS FROM THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN (ALSPAC)

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Background The aim of the current study was to investigate whether exposure to prenatal maternal anxiety and depression influenced later offspring glucose, lipid and inflammatory markers via intrauterine mechanisms.

Methods Data from a prospective birth cohort based in the South West of England were used. Our analysis included 2839 mother-children duos and 2361 father-child duos for outcomes assessed at mean age 9.9 years (non-fasting cholesterol, triglycerides, low density and high density lipoprotein cholesterol (LDLc and HDLc), C-reactive protein (CRP) and interleukin 6 (IL–6) and 2011 and 1726 parent-child duos for outcomes at mean age 15.4 years (fasting glucose, insulin, lipids and CRP). We compared associations of maternal exposures with offspring outcomes to those of the same paternal exposures with offspring outcomes. The rationale for this comparison was that if maternal depression/anxiety influenced offspring outcomes via intrauterine mechanisms we would expect stronger maternal compared with paternal associations. We also examined whether any association of exposures during pregnancy reflected a postnatal effect, with persistence of depression/anxiety into the postnatal period.

Results Maternal anxiety at 18 and 32 weeks gestation, and maternal depression at 32 weeks gestation were associated with increased CRP in children at 9.9 years (mean difference (95% CI): 0.031 (0.005 to 0.057), 0.030 (0.004 to 0.056), and 0.021 (0.003 to 0.040) respectively), but not at 15.4 years. These associations remained when adjusting for potential confounders (maternal age, ethnicity, pre-pregnancy BMI, parity, social class, smoking and alcohol consumption). Paternal anxiety and depression (measured at 18 weeks gestation)were also associated with increased CRP in children at 9.9 years (mean difference (95% CI): 0.039 (0.003 to 0.076) and 0.026 (0–0.052) respectively), but not at 15.4 years. The magnitudes of the paternal associations were similar to those seen in mothers. Maternal and paternal postnatal depression/anxiety symptoms were also associated with offspring CRP at age 9.9 and appeared to explain much of the antenatal association.

There were no consistent associations between maternal or paternal anxiety or depression during the antenatal or postnatal periods and any of offspring glucose, insulin, IL-6 or lipids at either age.

Conclusion We have found evidence of a relationship between maternal and paternal anxiety and depression during pregnancy and CRP levels in childhood, which does not persist to adolescence. Our results suggest that these associations are unlikely to be explained by intrauterine mechanisms and may be explained by shared familial confounding or postnatal effects.

OP63 ASSOCIATIONS OF ALL-CAUSE AND CAUSE-SPECIFIC MORTALITY WITH BODY MASS INDEX IN A LARGE NORWEGIAN COHORT: USE OF OFFSPRING BODY MASS INDEX AS AN INSTRUMENTAL VARIABLE

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