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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**Background** People with high body mass index (BMI) suffer elevated rates of mortality from a variety of causes, particularly from cardiovascular disease (CVD). Some studies have also found elevated mortality at low BMI, but it is unclear whether this represents a causal association. Potential confounding by existing ill-health is of particular concern. To avoid this problem, we used the BMI of an offspring as an instrument for the parent's BMI, as well as conducting conventional analyses of parental mortality against their own BMI.
**Methods** We extracted 33,011 mother-offspring and 28,142 father-offspring pairs from HUNT, a large prospective cohort study conducted in Nord-Trøndelag county, Norway. Participants' BMI was adjusted for age, sex, secular trends and smoking status. Associations of parental mortality rates with their own BMI were estimated as hazard ratios using Cox regression. Age was the time axis, and we adjusted for smoking, alcohol, exercise, education and employment. Additionally, adjusted causal hazard ratios between parental mortality and BMI were estimated using offspring BMI as an instrument for parental BMI and cubic splines were fitted to all associations to assess their linearity.
**Results** There were 9,271 maternal and 9,889 paternal deaths within the follow-up period of 1984–2009. The associations of all-cause, CVD and cancer mortality with parents' own BMI were substantially non-linear, with elevated mortality at both extremes and minima at 21–25 kg $m^{-2}$. Hazard ratios for all-cause mortality per standard deviation (4.1 kg $m^{-2}$) of own BMI were 1.04 (95% CI: 1.02, 1.06) and 1.05 (1.02, 1.08) in mothers and fathers, respectively. In contrast, associations of mortality with offspring BMI were approximately linear and positive. Causal hazard ratios per standard deviation of BMI estimated from the instrumental variable analyses were 1.20 (1.11, 1.30) and 1.11 (1.02, 1.22) for all-cause mortality in mothers and fathers, respectively. For CVD they were 1.26 (1.13, 1.41) and 1.15 (1.01, 1.30), and for cancer they were 1.23 (1.05, 1.45) and 1.07 (0.90, 1.27).
**Conclusion** These results confirm the elevated mortality from all causes, CVD and cancer in subjects with high or low BMI. The use of an offspring's BMI as an instrument suggests that the elevated mortality at low BMI in this and other studies may be the result of confounding by existing ill-health, and that the causal relationship between BMI and mortality is positive and approximately linear.

### OP64 PARENTAL SUICIDE ATTEMPT AND OFFSPRING SELF-HARM AND SUICIDAL THOUGHTS: RESULTS FROM THE ALSPAC BIRTH COHORT

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**Background** Exposure to parental self-harm has been linked to an increased risk of self-harm and suicidal thoughts in their offspring. Much of the available evidence is from population registers or clinical samples and relates to parental death by suicide; few studies have investigated associations of non-fatal suicide attempts in parents with community presenting non-fatal self-harm and suicidal thoughts in offspring. We studied the association of parental suicide attempt (SA) with offspring self-harm and suicidal thoughts using a large two-generational prospective cohort.
**Methods** The sample comprised 4,396 children, their mothers and 2,541 of the mothers' partners from the Avon Longitudinal Study of Parents and Children (ALSPAC). Parents were asked to report on incident episodes of SA on 10 separate occasions from pregnancy until the children were 11 years old. Information on lifetime childhood self-harm, with and without suicidal intent, and suicidal thoughts, with and without suicidal plan, was collected through child-completed questionnaires at the age of 16–17 years.
**Results** Based on preliminary results, SA was reported by 1.5% of the mothers and 0.7% of the partners. Adjusting for sociodemographic factors and parental depression, maternal SA was associated with 3-fold increased risk of self-harm with suicidal intent in their children [Adjusted odds ratio (AOR) 3.0, 95% confidence interval (CI) 1.4–6.1] but not with self-harm with no suicidal intent (AOR 0.8, 95% CI 0.3–1.9). Children exposed to maternal SA were more likely than unexposed children to report both suicidal thoughts with and without suicidal plan (AOR 5.2, 95% CI 2.3–11.6; AOR 2.2, 95% CI 1.1–4.4, respectively). Partner SA was associated with 2.3-fold increased risk of self-harm with suicidal intent (95% CI 0.5–10.6) and with an increased risk of suicidal thoughts with suicidal plan (AOR 3.5, 95% CI 0.8–16.0) but these results are consistent with chance. There was no evidence of an association between partner suicide attempt with offspring self-harm with no suicidal intent (AOR 0.4, 95% CI 0.1–3.5) or with offspring suicidal thoughts without a suicidal plan (AOR 0.6, 95% CI 0.1–4.5).
**Conclusion** Parental SA in childhood increases the risk of self-harm with suicidal intent in offspring but is unrelated to risk of self-harm without suicide intent. Parental SA is associated with larger effect size on suicidal thoughts with a suicidal plan than without a plan. Findings provide limited evidence that maternal SA is a more potent risk factor than partner SA.

## HSR: Professional Behaviour and Health Care

### OP65 PARENT UNDERSTANDING AND MANAGEMENT OF RTIs IN CHILDREN: IMPLICATIONS FOR HEALTH PRACTITIONER COMMUNICATION

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**Background** Respiratory tract infections (RTIs) are among the commonest reason parents consult their GP, representing a significant burden on primary health care services. Many of the symptoms associated with RTIs are a cause of anxiety for parents, but they are often unsure when to consult. This study investigated parents' experiences of consulting for RTI in their child as part of a wider TARGET programme to improve the care of children with RTIs.
**Methods** Parents were recruited through 6 practices in areas of high, middle and low deprivation to capture a range of patient populations. Parents with a child aged between 3 months and 12 years who had consulted for acute RTI within the previous 3 months (excluding children with serious or chronic health problems) were invited to participate. Sampling ensured that parents with younger and older children and parents who were more or less frequent consulters were sampled. Semi-structured interviews explored parent's experience of consulting for RTIs in their children, information and advice needs; and their understanding of RTIs and treatment options. Interviews were audio-recorded, transcribed and imported into NVivo8 for coding. A thematic analysis was conducted using constant comparison techniques.
**Results** Thirty parents were interviewed and the sample captured a range of socio-economic backgrounds, both single and double parent families, with between 1 and 4 children. Consultation rates ranged from 1 to 24 times per year. New parents often had little knowledge of RTIs in children; felt uncertain about which