SUPPLEMENTARY MATERIAL

Genetic evidence for causal relationships between maternal obesity-related traits and birth weight

Overview of Supplementary Material

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Funding/support of individual studies

Individual study acknowledgements
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Maximising specificity of genetic variants: excluding single nucleotide polymorphisms (SNPs) with effects on multiple traits ("pleiotropic" SNPs)

It was important to ensure that each genetic score would enable us, as far as possible, to capture specifically the respective maternal trait. To identify SNPs with pleiotropic effects, we queried each of our initial 193 selected SNPs and all SNPs in linkage disequilibrium with these ($r^2>0.2$) using the National Human Genome Research Institute (NHGRI) catalog of published genome-wide association studies (GWAS)\(^1\) and listed SNPs associated with other traits at $P<5\times10^{-8}$. Starting with this list, we excluded SNPs whose location near a candidate gene and/or the strength of association with another trait suggested that the association with the maternal exposure of interest is almost certainly secondary to the other trait (e.g. exclusion of index SNPs at FTO and MC4R from the type 2 diabetes genetic score, since these are primarily associated with BMI and secondarily with type 2 diabetes via their effect on BMI). We additionally excluded SNPs from the list with strong evidence of effects on two or more traits that are potentially relevant to the maternal environment and birth weight. Details of SNPs in the final selected list are shown in \textit{eTable 3}.

We performed an updated search of the NHGRI catalog, while writing the research paper, to check for further pleiotropic associations identified for SNPs used in our analyses, which were published after our initial search. We performed sensitivity analyses excluding these additional SNPs to check that they did not alter our findings (results available from the authors on request).

Maximising specificity of genetic variants: separating genetic scores for closely-related maternal traits

Fasting glucose and type 2 diabetes share several genetic susceptibility variants, reflecting the overlap between these two phenotypes (\textit{eTable 3}). We excluded from the type 2 diabetes genetic score the index SNPs at the two fasting glucose loci that explain the most variance in fasting glucose, but have relatively moderate effects on type 2 diabetes risk (MTNR1B and GCK). Likewise, we excluded the index SNP at the TCF7L2 locus from the fasting glucose genetic score as it has a proportionately much larger effect on type 2 diabetes risk. In this way, our fasting glucose genetic score would predominantly capture variation in maternal fasting glucose in the normal physiological range, while our type 2 diabetes genetic score would be more likely to capture pathologically-raised fasting and non-fasting maternal glucose levels.

Maternal triglycerides and HDL-cholesterol also share associations with several genetic variants. We therefore attempted to make our genetic scores for these exposures as specific as possible. For HDL-cholesterol, we included only SNPs near genes associated with known Mendelian lipid disorders (see \textit{eTable 3})\(^2\). For triglyceride levels, SNPs were included in the genetic score if they were solely associated with triglyceride levels, or if their effect on triglyceride levels was at least three times greater than that of HDL-, LDL- or total cholesterol, based on effect sizes reported in \(^2\). To facilitate these comparisons, the raw effect sizes in mg/dL were first converted to percentages of the mean of the corresponding lipid concentration.

SNPs missing from studies

When index SNPs were missing from individual studies, we used the SNP Annotation and Proxy Search tool, SNAP\(^3\) to identify suitable proxy SNPs ($r^2>0.8$). If a study had fewer than 80% of the index or proxy SNPs required to generate a specific genetic score, it was excluded from the analysis. The one exception to this was the HAPO (non-GWAS) Study, for which only 6 of 17 triglyceride SNPs had been genotyped. We included this study, despite the missing SNPs, because the 6 genotyped SNPs included those with the largest effects on triglyceride levels, covering the majority of variation captured by the 17 SNP score.

Imputation quality

For each study with GWAS data, we examined the imputation quality ($r^2$ or proper\_info\(^4\)) of SNPs selected for each score. We excluded four studies (BSUC-WTCCC, NFBC1966, QIMR and TwinsUK) from analyses of the adiponectin genetic score due to imputation quality scores $<0.8$ for either 1 or 2 of the 3 SNPs in that score. In each of the remaining 7 genetic scores, a small number of included SNPs had imputation quality scores $<0.8$, but this only affected a median of 0 to 1 SNP per study, equivalent to a maximum of 6% of the SNPs comprising the score, so we did not exclude them. Finally, we identified that 3 individual SNPs were poorly imputed ($r^2<0.8$) in multiple studies: rs10830963 (fasting glucose genetic score, 4 of 15 studies), rs1063069 (type 2 diabetes genetic score; 11 of 13 studies) and rs13238203 (triglycerides genetic score; 11 of 16 studies). To verify that these individual SNPs did not materially alter our results, we performed sensitivity analyses: (i) we repeated the meta-analyses of the fasting glucose genetic score excluding the 4 studies in which SNP rs10830963 was poorly imputed; (ii) we performed weighted meta-analyses of existing summary GWAS data\(^5\),

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\(^1\) NHGRI catalog, \(^2\) See \textit{eTable 3}, \(^3\) SNAP tool, \(^4\) SNP Annotation and Proxy Search tool, \(^5\) Existing summary GWAS data.
as described previously both including and excluding the rs11063069 and rs13238203 SNPs. Results of these analyses are available from the authors on request.

**Calculation of maternal genetic scores**
We calculated a weighted genetic score for each maternal exposure to account for the fact that some SNPs have relatively larger effects than others. Formula 1 below describes the calculation, where w is the weight and SNP is the number of trait-raising or lowering alleles at that locus. The decision to model according to the trait-raising or lowering allele was informed by the known association between each maternal trait and BMI (Box 1). The weights used for each SNP were obtained from published GWAS of non-pregnant individuals, which either did not include any of the studies used in this paper or had at most 17% of participants overlapping. These weights and their sources, are summarised in eTable 3.

\[
\text{Weighted score} = w_1 \times \text{SNP}_1 + w_2 \times \text{SNP}_2 + \ldots + w_n \times \text{SNP}_n \quad (1)
\]

We rescaled each weighted genetic score (GS) to reflect the number of available SNPs using formula 2 as described in Lin et al.²

\[
\text{GS} = \frac{\text{Weighted Score} \times \text{Number of SNPs available}}{\text{Sum of weights of available SNPs}} \quad (2)
\]

**Meta-analyses**
We meta-analysed data from all available studies to give an overall result from each side of the triangle (Figure 1): the genetic score-maternal exposure association; the genetic score-birth weight association; and the observational maternal exposure-birth weight association. We combined the regression coefficients and standard errors from individual study analyses by performing inverse variance meta-analyses with fixed effects as there was little evidence of between-study heterogeneity of effect size. All meta-analyses were performed using the user-written Stata command, metan.³ We estimated the percentages of total variation among study estimates due to between-study heterogeneity using Cochran’s Q test and the I² statistic.¹⁰ To convert the overall results from birth weight and ponderal index Z-scores into grams and kg m² respectively, we multiplied the effect size and their upper and lower 95% confidence limits by a representative value of the standard deviation of birth weight (484 g) or ponderal index (2.78 kg m²; ALSPAC study).

**Mendelian randomization analysis**
We performed instrumental variable (IV) estimation using the ratio estimator.¹² We estimated the effect of each maternal exposure on either birth weight or ponderal index by dividing the overall genetic score-birth weight or genetic score-ponderal index association by the overall genetic score-maternal exposure association. The standard error of these estimates was calculated using a Taylor series approximation; we used a 2nd order Taylor series expansion to obtain the variance of the IV estimate. We then made a normal distribution assumption by calculating the 95% confidence interval as follows: IV estimate ± 1.96*sqrt(variance of IV estimate from Taylor series expansion).

We used a Z-test to test for a difference between the instrumental variable (genetic) and observational associations. The Z-score was calculated by estimating the covariance between the observational and instrumental variable (genetic) estimates using a bootstrapping procedure. We used the following formula for our Z-test:

\[
Z = \frac{\text{(difference between IV and observational estimate)} / \text{sqrt(variance of difference between the estimates)}}{\text{sqrt(variance of difference between the estimates)}}
\]

where the variance of the difference between the estimates is given by:

\[
\text{var(IV estimate)} + \text{var(observational estimate)} - 2 \times \text{cov(IV estimate, obs estimate)}
\]

The covariance between the IV and observational estimates was estimated by nonparametric bootstrapping the IV and observational estimates using 20 replications (we chose a relatively small number of replications because we included meta-analyses with up to 18 studies). We then compared the Z-statistic with a standard normal distribution.

**Guarding against weak instrument bias**
Mendelian randomization studies may be susceptible to weak instrument bias. Bias is the difference between the estimated value of a parameter and its true value. Weak instrument bias occurs in the direction of the
phenotype (i.e. the maternal trait). The strength of each instrument used in our study is a function of (i) the proportion of variance in the maternal trait explained by the genetic score and (ii) the sample size. Since the variance in each maternal trait explained by the genetic score was modest, we maximized the sample size (Table 2). The possible causal associations identified in our study are therefore unlikely to be due to weak instrument bias.

Control for population stratification
The presence of subpopulations, which differ in mean birth weight and have genetic variants present at different frequencies, can cause artificial associations between genotypes and birth weight. To ensure that the genetic associations we tested were not confounded in this way, we took the following steps: (i) we included only women of European ancestry; (ii) where necessary, analyses in the individual studies were adjusted for ancestry principal components; (iii) in those studies that had performed a genome-wide association study of birth weight, we checked the genomic control lambda values (ratio of median of the empirically observed distribution of the test statistic to the expected median), which suggested only minimal inflation: median lambda ~ 1.006 [interquartile range: 1.004-1.012]; (iv) we combined summary statistics from individual studies by inverse variance meta-analysis, thereby controlling for any population stratification between studies in the overall sample.

Sensitivity analyses
The ascertainment of offspring birth weight or gestational age data varied among the individual studies, from measurement by trained study personnel, to ascertainment from medical records or birth registries, to self-report. To verify that our results were unaffected by the varying quality or availability of phenotypic data, we performed sensitivity meta-analyses of the associations between the 8 genetic scores and birth weight in up to 12 studies with best quality data (i.e. measured or medical record birth weight and gestational age available). Results of these analyses are available from the authors on request.

To verify that the SBP genetic score-birth weight associations were unaffected by using weights from the offspring (i.e. the maternal trait), we performed a blood pressure GWAS in 127,698 individuals of British descent using the UK Biobank data. The UK Biobank recruited over 500,000 individuals aged 37-73 years (99.5% were between 40 and 69 years) in 2006-2010 from across the country16. Two blood pressure readings were taken approximately 5 minutes apart using an automated Omron blood pressure monitor. Two valid measurements were available for most participants, and the average was taken. Individuals were excluded if the two readings differed by more than 4.56SD, and blood pressure measurements more than 4.56SD away from the mean were excluded. We accounted for blood pressure medication use by adding 15 to the systolic blood pressure measure. Valid blood pressure measurements were available for 120,008 individuals. Blood pressure was adjusted for age, sex and centre location and then inverse normalized. The weights from the blood pressure GWAS in the UK Biobank were utilised to create a genetic risk score in the ALSPAC study (n=7,304). We investigated the correlation of the two blood pressure risk scores (r2=0.77) and performed Mendelian randomization. The results are available from the authors on request.

Estimating how much of the possible causal effect of BMI on birth weight is mediated by fasting glucose
To begin to understand what proportion of the estimated causal effect of BMI on birth weight might be mediated by fasting glucose, we first estimated the causal effect of BMI on maternal fasting glucose. Using available studies (see eTable 6a), each additional allele of the BMI genetic score was associated with a 0.145 kg/m2 (95%CI: 0.126, 0.164) higher BMI and a 0.005 mmol/L (95%CI: 0.001, 0.009) higher fasting glucose. This is equivalent to 0.34 SD higher fasting glucose level per 1 SD higher genetically instrumented BMI. (To convert to SD units, we used BMI SD ~ 4 kg/m2 and fasting glucose SD ~ 0.4 mmol/L.) We then multiplied the genetic estimate and 95%CI for the effect of fasting glucose on birth weight (114g [95%CI: 80, 147g]) by 0.34 to represent the possible causal effect of fasting glucose on birth weight for every 1 SD higher maternal BMI.

Since we found genetic evidence that systolic blood pressure (SBP) was causally associated with birth weight in the opposite direction and positively associated with BMI, we additionally estimated the causal effect of BMI on SBP. Each additional allele of the BMI genetic score was associated with a 0.07 mmHg (95%CI: 0.02, 0.11) higher SBP. This is equivalent to 0.19 SD higher SBP per 1 SD higher genetically instrumented BMI. (To convert to SD units, we used SBP SD ~ 10 mmHg.) We then multiplied the IV estimate and 95%CI for the effect of SBP on birth weight (-208g [95% CI: -394, -21]) by 0.19 to represent the causal effect of SBP on birth weight for every 1 SD higher maternal BMI.

Power calculations
Using data available from the ALSPAC study, we estimated the variance explained in birth weight (BW) by each maternal genetic score as the difference in adjusted-$R^2$ values between linear regression models (i) and (ii) as follows:

(i) $BW = sex + gestational\_age$

(ii) $BW = sex + gest\_age + genetic\_score$

We then used these values to estimate (a) the power available in our included sample to detect evidence of association between maternal genetic score and birth weight at $P<0.05$, and (b) the minimum sample size needed to detect association between maternal genetic score and birth weight at $P<0.05$ with 80% power. Power calculations were performed using Quanto v.1.2 (http://biostats.usc.edu/software).
**eFigure 1.** Comparison of the observational with the genetic change in ponderal index (in kg/m\(^3\)) for a 1 standard deviation (SD) change in each maternal trait. For 25(OH)D and adiponectin, we present the change in ponderal index for a 10% change in maternal trait level because these variables were logged for analysis. The genetic change was estimated from Mendelian randomization analysis, in which a genetic score was used to estimate the possible causal effect of the maternal trait on ponderal index. The genetic estimate is presented twice: in the second case it was adjusted for fetal genotype using a subset of the available studies. The error bars represent the 95% confidence intervals around the effect size estimates.

<table>
<thead>
<tr>
<th>Trait</th>
<th>Change in Trait</th>
<th>Standard deviation</th>
<th>N studies</th>
<th>N women</th>
<th>N offspring genotypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-pregnancy BMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observational</td>
<td></td>
<td></td>
<td>4 kg/m^2 higher</td>
<td>3</td>
<td>9690 0</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>4 kg/m^2 higher</td>
<td>7</td>
<td>9628 9628</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Fasting glucose</td>
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<td>8</td>
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<td>9335 9335</td>
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<td></td>
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<td>1</td>
<td>854 0</td>
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<td>Genetic</td>
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<td></td>
<td>0.5 mmol/L lower</td>
<td>9</td>
<td>15573 0</td>
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<tr>
<td>Genetic (adjusted for fetal genotype in subset)</td>
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<td></td>
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<tr>
<td>HDL cholesterol</td>
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<td>8207 8207</td>
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<td></td>
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<td>10 mmHg higher</td>
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<td>9691 0</td>
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<tr>
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<td></td>
<td></td>
<td>10 mmHg higher</td>
<td>7</td>
<td>13527 0</td>
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<tr>
<td>Genetic (adjusted for fetal genotype in subset)</td>
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<td></td>
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<td>Systolic blood pressure</td>
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<td></td>
<td></td>
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<td>1</td>
<td>3718 0</td>
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<tr>
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<td></td>
<td></td>
<td>10% lower</td>
<td>7</td>
<td>14004 0</td>
</tr>
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<td>25(OH)D</td>
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<td>1373 0</td>
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<tr>
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<td></td>
<td>10% lower</td>
<td>6</td>
<td>11501 0</td>
</tr>
<tr>
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<td></td>
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<tr>
<td>Adiponectin</td>
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<td>5</td>
<td>6851 6851</td>
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<tr>
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<td></td>
<td>10% lower</td>
<td>6</td>
<td>11501 0</td>
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<tr>
<td>Genetic</td>
<td></td>
<td></td>
<td>10% lower</td>
<td>5</td>
<td>6851 6851</td>
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<tr>
<td>Genetic (adjusted for fetal genotype in subset)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The change in ponderal index (kg/m\(^3\)) for a standard deviation (SD) change in each maternal trait is shown in the diagram. The error bars represent the 95% confidence intervals around the effect size estimates. The table shows the number of studies (N studies) and the number of women (N women) for each trait.
eFigure 2. Estimating how much of the estimated possible causal effect of maternal BMI on birth weight is mediated by maternal fasting glucose. The solid, horizontal arrow indicates our genetic estimate [95%CI] of the causal effect of BMI on birth weight. The dashed arrows on the left side show genetic causal estimates of a 1 SD (≈ 4kg/m²) higher maternal pre-pregnancy BMI on maternal fasting glucose (≈0.34SD = 0.14 mmol/L) and maternal systolic blood pressure in pregnancy (≈0.19 SD = 2 mmHg). The dashed arrows on the right show the scaled genetic causal estimates of these changes in fasting glucose and systolic blood pressure on birth weight. The effect of maternal BMI on birth weight via fasting glucose (≈39g) is broadly similar to the total effect of maternal BMI on birth weight (≈55g), but that effect is opposed by the birth weight lowering effect of SBP (≈40g). Overall, this suggests that while maternal fasting glucose mediates part of the positive association between maternal BMI and birth weight, other BMI-related factors are likely to be involved. Abbreviations: BMI, body mass index; BW, birth weight; FPG, fasting plasma glucose; SBP, systolic blood pressure.
<table>
<thead>
<tr>
<th>STUDY INFORMATION</th>
<th>STUDY</th>
<th>ALSPAC Mothers</th>
<th>Berlin Birth Cohort (BBC) Mothers</th>
<th>1958 British Birth Cohort or NCDS (B58C-WTCCC)</th>
<th>1958 British Birth Cohort or NCDS (B58C-T1DGC)</th>
<th>CHOP Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
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<td>British/European descent</td>
<td>European descent</td>
<td>British/European descent</td>
<td>British/European descent</td>
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</tr>
<tr>
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<tr>
<td>Collection type (e.g. population-based)</td>
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<td>Population-Based</td>
<td>Community-based</td>
<td>Population-based</td>
<td>Population-based</td>
<td>Population-based</td>
</tr>
<tr>
<td>N women with birth weight of 1 child and genotypes for at least one genetic score</td>
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<td>7,304</td>
<td>1,357</td>
<td>855</td>
<td>836</td>
<td>312</td>
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<tr>
<td>Fetal genotype data available? (Y/N)</td>
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<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>BIRTH WEIGHT</td>
<td>Method by which offspring birth weight (and length, if available) were collected</td>
<td>Obstetric records / measured by trained study personnel</td>
<td>Measured by trained personnel immediately after birth</td>
<td>Maternal self-report (information from questionnaires at age 33 and 42 years)</td>
<td>Maternal self-report (information from questionnaires at age 33 and 42 years)</td>
<td>Questionnaire and EPIC medical records (9.5% of questionnaire values were checked against medical records: r=0.83)</td>
</tr>
<tr>
<td>GESTATIONAL AGE</td>
<td>Method by which gestational age was collected</td>
<td>By date of last menstrual period (LMP), paediatric assessment, obstetric assessment, ultrasound assessment.</td>
<td>Calculated from LMP and corrected by ultrasound, if the difference was &gt; 2 weeks</td>
<td>From maternal self-report at ages 33 and 42 years: a question inquiring if the child was born at term, or alternatively how many weeks in advance or late.</td>
<td>From maternal self-report at ages 33 and 42 years: a question inquiring if the child was born at term, or alternatively how many weeks in advance or late.</td>
<td>NA</td>
</tr>
<tr>
<td>Summary Maternal Characteristics, where available in the Included sample, During Pregnancy (median (IQR) given where the trait distribution deviates strongly from the normal distribution</td>
<td>ALSFAC Mothers</td>
<td>Berlin Birth Cohort (BBC) Mothers</td>
<td>1958 British Birth Cohort or NCDS (BS8C-WTCCC)</td>
<td>1958 British Birth Cohort or NCDS (BS8C-TIDGC)</td>
<td>CHOP Mothers</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Maternal age at delivery, unless otherwise stated [Mean (sd)], years</td>
<td>28.5 (4.8)</td>
<td>30.1 (5.4)</td>
<td>26.2 (5.2)</td>
<td>26.1 (5.4)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Maternal pre-pregnancy BMI [Mean (sd)], kg/m2</td>
<td>22.93 (3.73)</td>
<td>22.78 (3.93)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Maternal pregnancy BMI [Mean (sd)], kg/m2</td>
<td>26.63 (4.03)</td>
<td>28.39 (4.25)</td>
<td>NA</td>
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<tr>
<td>Fasting glucose [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>Triglycerides [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>HDL-cholesterol [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Blood pressure [Mean (sd)], mmHg</td>
<td>112.9 (7.5)/65.5 (4.8)</td>
<td>117.28 (10.85)/70.73 (7.56)</td>
<td>This was measured at the 3rd trimester.</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>25-hydroxyvitamin D [Median (IQR)], nmol/L</td>
<td>62.1 (43.6, 85.4)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
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<tr>
<td>Adiponectin [Mean (sd)], ug/mL</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>% of mothers who smoked in pregnancy</td>
<td>17.5%</td>
<td>15.6%</td>
<td>38.0%</td>
<td>34.1%</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Mean gestational week of collection of maternal characteristics</td>
<td>28</td>
<td>28</td>
<td>Retrospective</td>
<td>Retrospective</td>
<td>NA</td>
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<tr>
<td>Parity (% primiparous births)</td>
<td>34%</td>
<td>53.6%</td>
<td>100</td>
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### Summary Offspring Characteristics

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<tr>
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<th>ALSPAC Mothers</th>
<th>Berlin Birth Cohort (BBC) Mothers</th>
<th>1958 British Birth Cohort or NCDS (B58C-WTCCC)</th>
<th>1958 British Birth Cohort or NCDS (B58C-TIDGC)</th>
<th>CHOP Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Birth weight [Mean (sd)], grams</strong></td>
<td>3481 (475)</td>
<td>3472 (511)</td>
<td>3325 (483)</td>
<td>3379 (469)</td>
<td>3440 (562)</td>
</tr>
<tr>
<td><strong>Gestational age at delivery [Median (IQR)], weeks</strong></td>
<td>40 (39, 41)</td>
<td>40 (38, 40)</td>
<td>40 (40, 41)</td>
<td>40 (40, 41)</td>
<td>NA</td>
</tr>
<tr>
<td><strong>Birth length [Mean (sd)], cm</strong></td>
<td>51 (2)</td>
<td>51.31 (2.50)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td><strong>Ponderal index [Mean (sd)], kg/m3</strong></td>
<td>26 (3)</td>
<td>25.68 (3.40)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

### References

**Reference - Cohort**


**Study URL**

- http://www.bristol.ac.uk/alspac/
- NA
- www.cls.ioe.ac.uk and www.wtccc.org.uk
- www.cls.ioe.ac.uk

*Please note that the study website contains searchable details of all data, available through: http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/

NA, not available

Informed consent was obtained from all participants, and study protocols were approved by the local regional or institutional ethics committees (ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees).
<table>
<thead>
<tr>
<th>STUDY INFORMATION</th>
<th>STUDY</th>
<th>COPSAC-2000 Mothers</th>
<th>DNBC-GOYA Random Set</th>
<th>DNBC-PTB-CONTROL Mothers</th>
<th>EFSOCH Mothers</th>
<th>GEN-3G Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
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<td>Danish/European descent</td>
<td>Danish/European descent</td>
<td>British/European descent</td>
<td>Canadian/European descent</td>
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<td>Country (Sample source)</td>
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<td>Denmark</td>
<td>UK</td>
<td>Canada</td>
</tr>
<tr>
<td>Collection type (e.g. population-based)</td>
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<td>High-risk asthma birth cohort</td>
<td>Population based¹</td>
<td>Population-based</td>
<td>Community-based</td>
<td>Population-based</td>
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<tr>
<td>N women with birth weight of 1 child and genotypes for at least one genetic score</td>
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<td>282</td>
<td>1,805</td>
<td>1,649</td>
<td>746</td>
<td>676</td>
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<tr>
<td>Fetal genotype data available? (Y/N)</td>
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<td>Y</td>
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<td>Y</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>BIRTH WEIGHT</td>
<td>Method by which offspring birth weight (and length, if available) were collected</td>
<td>Medical Records</td>
<td>Obstetric data from medical birth register</td>
<td>Obstetric data from medical birth register</td>
<td>At birth using standard neonatal anthropometry measures</td>
<td>Hospital electronic medical records</td>
</tr>
<tr>
<td>GESTATIONAL AGE</td>
<td>Method by which gestational age was collected</td>
<td>Medical Records</td>
<td>The National Birth Register, where gestational age is reported by doctors and midwives at birth</td>
<td>Consensus algorithm for gestational age was developed based on information from medical birth register, hospital discharge register, LMP, LMP corrected for menstrual cycle length, Expected date of delivery (often based on ultrasound), and mother's selfreport of gestational age.</td>
<td>Hospital records and study midwives</td>
<td>Hospital electronic medical records</td>
</tr>
</tbody>
</table>
## SUMMARY MATERNAL CHARACTERISTICS, where available in the INCLUDED sample, DURING PREGNANCY (median (IQR) given where the trait distribution deviates strongly from the normal distribution.

<table>
<thead>
<tr>
<th></th>
<th>COPSAC-2000 Mothers</th>
<th>DNBC-GOYA Random Set</th>
<th>DNBC-PTB-CONTROL Mothers</th>
<th>EFSOCH Mothers</th>
<th>GEN-3G Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age at delivery, unless otherwise stated [Mean (sd), years]</td>
<td>30.4 (4.3)</td>
<td>29.2 (4.2)</td>
<td>29.9 (4.2)</td>
<td>30.5 (5.3)</td>
<td>28.4 (4.4)</td>
</tr>
<tr>
<td>Maternal pre-pregnancy BMI [Mean (sd), kg/m²]</td>
<td>NA</td>
<td>23.57 (4.27)</td>
<td>23.57 (4.27)</td>
<td>24.07 (4.42)</td>
<td>24.83 (5.63)</td>
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<tr>
<td>Maternal pregnancy BMI [Mean (sd), kg/m²]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>28.01 (4.55)</td>
<td>27.98 (5.38)</td>
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<tr>
<td>Fasting glucose [Mean (sd), mmol/L]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>4.35 (0.38)</td>
<td>4.20 (0.41)</td>
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<tr>
<td>Triglycerides [Mean (sd), mmol/L]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>2.13 (0.73)</td>
<td>1.93 (0.64)</td>
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<tr>
<td>HDL-cholesterol [Mean (sd), mmol/L]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>2.08 (0.46)</td>
<td>1.91 (0.43)</td>
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<tr>
<td>Blood pressure [Mean (sd), mmHg]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>107.5 (9.2) / 67.6 (6.8)</td>
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<tr>
<td>25-hydroxyvitamin D [Median (IQR), nmol/L]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>61.7 [50.1 ; 75.5] (at ~9 weeks)</td>
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<tr>
<td>Adiponectin [Mean (sd), ug/mL]</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>12.57 (4.72)</td>
</tr>
<tr>
<td>% of mothers who smoked in pregnancy</td>
<td>12.8%</td>
<td>25.8%</td>
<td>17.8%</td>
<td>13.0%</td>
<td>8.88%</td>
</tr>
<tr>
<td>Mean gestational week of collection of maternal characteristics</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>28</td>
<td>~26</td>
</tr>
<tr>
<td>Parity (% primiparous births)</td>
<td>63.2%</td>
<td>50.0%</td>
<td>30.9%</td>
<td>49.8%</td>
<td>47.5%</td>
</tr>
<tr>
<td></td>
<td>COPSAC-2000 Mothers</td>
<td>DNBC-GOYA Random Set</td>
<td>DNBC-PTB-CONTROL Mothers</td>
<td>EFSOCH Mothers</td>
<td>GEN-3G Mothers</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------------------</td>
<td>-----------------------</td>
<td>---------------------------</td>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td><strong>Birth weight</strong> [Mean (sd), grams]</td>
<td>3560 (505)</td>
<td>3643 (495)</td>
<td>3595 (497)</td>
<td>3512 (480)</td>
<td>3448 (433)</td>
</tr>
<tr>
<td><strong>Gestational age at delivery</strong> [Median (IQR), weeks]</td>
<td>40 (39, 41)</td>
<td>40.3 (39.4, 41.1)</td>
<td>40 (39, 40)</td>
<td>40 (37, 43)</td>
<td>39.7 [38.9, 40.4]</td>
</tr>
<tr>
<td><strong>Birth length</strong> [Mean (sd), cm]</td>
<td>52.5 (2.2)</td>
<td>52.5 (2.2)</td>
<td>52 (2)</td>
<td>50 (2)</td>
<td>51.1 (2.1)</td>
</tr>
<tr>
<td><strong>Ponderal index</strong> [Mean (sd), kg/m³]</td>
<td>24.6 (2.4)</td>
<td>25.0 (2.3)</td>
<td>25 (2)</td>
<td>28 (3)</td>
<td>25.9 (2.5)</td>
</tr>
</tbody>
</table>

**REFERENCES**


**Study URL**


*Pregnant women recruited 1996-2002 as part of the Danish National Birth Cohort. A random (according to BMI) selection with genotype data are included in the current study.

NA, not available

Informed consent was obtained from all participants, and study protocols were approved by the local regional or institutional ethics committees.
### Table 1(c) Basic characteristics of study participants and their offspring (studies 11-14)

<table>
<thead>
<tr>
<th>STUDY INFORMATION</th>
<th>STUDY</th>
<th>Generation R Mothers</th>
<th>HAPO Mothers (GWAS)</th>
<th>HAPO Mothers (nonGWAS)</th>
<th>MaBa (Mothers)</th>
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<tbody>
<tr>
<td>Ethnicity</td>
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<td>Dutch/European descent</td>
<td>Northern European</td>
<td>European descent</td>
<td>Norwegian/European descent</td>
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<td>Country (Sample source)</td>
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<td>UK, Canada, Australia</td>
<td>USA, UK, Canada, Australia</td>
<td>Norway</td>
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<td>Population-based</td>
<td>Population-based</td>
<td>Population-based</td>
<td>Population based</td>
</tr>
<tr>
<td>N women with birth weight of 1 child and genotypes for at least one genetic score</td>
<td></td>
<td>3,810</td>
<td>1,380</td>
<td>3,590</td>
<td>650</td>
</tr>
<tr>
<td>Fetal genotype data available? (Y/N)</td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>BIRTH WEIGHT</td>
<td>Method by which offspring birth weight (and length, if available) were collected</td>
<td>Hospital records and community midwives</td>
<td>Medical record abstraction</td>
<td>Medical record abstraction</td>
<td>From The Medical Birth Registry of Norway</td>
</tr>
<tr>
<td>GESTATIONAL AGE</td>
<td>Method by which gestational age was collected</td>
<td>Hospital records and community midwives</td>
<td>Estimated according to last menstrual period or ultrasound gestational age and estimated date of delivery or confinement</td>
<td>Estimated according to last menstrual period or ultrasound gestational age and estimated date of delivery or confinement</td>
<td>Gestational age was obtained from ultrasound at gestational week 17-19 of pregnancy.</td>
</tr>
<tr>
<td></td>
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<td>HAPO Mothers (GWAS)</td>
<td>HAPO Mothers (nonGWAS)</td>
<td>Mollä (Mothers)</td>
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</tr>
<tr>
<td>--------------------------------</td>
<td>----------------------</td>
<td>---------------------</td>
<td>------------------------</td>
<td>-----------------</td>
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</tr>
<tr>
<td><strong>SUMMARY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MATERNAL CHARACTERISTICS</strong>, where available in the INCLUDED sample, DURING PREGNANCY (median (IQR) given where the trait distribution deviates strongly from the normal distribution</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal age at delivery, unless otherwise stated [Mean (sd)], years</td>
<td>31.2 (4.5) [=Maternal age at intake, when average gestational age = 14.4 weeks]</td>
<td>31.5 (5.3) [=Maternal age at OGTT, when average gestational age = 28 weeks]</td>
<td>30.4 (5.4) [=Maternal age at OGTT, when average gestational age = 28 weeks]</td>
<td>28.5 (3.3)</td>
<td></td>
</tr>
<tr>
<td>Maternal pre-pregnancy BMI [Mean (sd)], kg/m²</td>
<td>23.12 (3.92)</td>
<td>24.5 (5.0)</td>
<td>24.63 (5.33)</td>
<td>23.93 (3.94)</td>
<td></td>
</tr>
<tr>
<td>Maternal pregnancy BMI [Mean (sd)], kg/m²</td>
<td>26.98 (4.04)</td>
<td>28.46 (4.82)</td>
<td>28.58 (5.25)</td>
<td>24.78 (3.80)</td>
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</tr>
<tr>
<td>Fasting glucose [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>4.56 (0.37)</td>
<td>4.54 (0.37)</td>
<td>NA</td>
<td></td>
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<tr>
<td>Triglycerides [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>NA</td>
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</tr>
<tr>
<td>HDL-cholesterol [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Blood pressure [Mean (sd)], mmHg</td>
<td>120.0 (11.4) / 69.3 (9.2)</td>
<td>108.6 (9.9) / 71.4 (8.0)</td>
<td>108.3 (9.6) / 70.7 (8.1)</td>
<td>113.5 (11.8) / 68.5 (8.4)</td>
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</tr>
<tr>
<td>25-hydroxyvitamin D [Median (IQR)], nmol/L</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Adiponectin [Mean (sd)], ug/mL</td>
<td>NA</td>
<td>20.37 (12.83)</td>
<td>-</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>% of mothers who smoked in pregnancy</td>
<td>27.1%</td>
<td>13.5%</td>
<td>15.0%</td>
<td>8.1%</td>
<td></td>
</tr>
<tr>
<td>Mean gestational week of collection of maternal characteristics</td>
<td>30</td>
<td>28.5</td>
<td>28.3</td>
<td>18</td>
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<tr>
<td>Parity (% primiparous births)</td>
<td>58.7%</td>
<td>56.9%</td>
<td>50.0%</td>
<td>45.8%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Generation R Mothers</td>
<td>HAPO Mothers (GWAS)</td>
<td>HAPO Mothers (nonGWAS)</td>
<td>MoBa (Mothers)</td>
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<tr>
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<td>----------------------</td>
<td>---------------------</td>
<td>------------------------</td>
<td>-----------------</td>
<td></td>
</tr>
<tr>
<td><strong>Birth weight [Mean (sd), grams]</strong></td>
<td>3528 (494)</td>
<td>3557 (517)</td>
<td>3526 (463)</td>
<td>3679 (430)</td>
<td></td>
</tr>
<tr>
<td><strong>Gestational age at delivery [Median (IQR), weeks]</strong></td>
<td>40 (39, 41)</td>
<td>40 (39, 41)</td>
<td>40 (39, 41)</td>
<td>40.1 (39.3, 41.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Birth length [Mean (sd), cm]</strong></td>
<td>51 (2)</td>
<td>50.6 (2.3)</td>
<td>50.8 (2.3)</td>
<td>50.6 (1.8)</td>
<td></td>
</tr>
<tr>
<td><strong>Ponderal index [Mean (sd), kg/m3]</strong></td>
<td>27 (3)</td>
<td>27.48 (3.19)</td>
<td>26.87 (2.86)</td>
<td>28.31 (2.53)</td>
<td></td>
</tr>
</tbody>
</table>

**REFERENCES**

- Nutritional epidemiology: Birth weight and gestational age at delivery. Birth weight [Mean (sd), grams] | 3528 (494) | 3557 (517) | 3526 (463) | 3679 (430) |

**Study URL**

- www.generationr.nl
- http://www.hapo.northwestern.edu/index.html
- http://www.hapo.northwestern.edu/index.html
- http://www.fhi.no/morogbarn

NA, not available

Informed consent was obtained from all participants, and study protocols were approved by the local regional or institutional ethics committees.
<table>
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<th>QIMR</th>
<th>TWINSUK</th>
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<td>European descent</td>
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<td>Country (Sample source)</td>
<td>Northern Finland, Provinces of Oulu and Lapland</td>
<td>The Netherlands</td>
<td>Australia</td>
<td>UK</td>
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<td>Prospective general population-based</td>
<td>Population-based controls</td>
<td>Population-based recruitment of adult twins</td>
<td>population based</td>
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<tr>
<td>N women with birth weight of 1 child and genotypes for at least one genetic score</td>
<td>2,035</td>
<td>706</td>
<td>892</td>
<td>1,602</td>
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<td>Year(s) of birth of offspring</td>
<td>1987-2001</td>
<td>1946 - 2003</td>
<td>1929-1990</td>
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<tr>
<td>Fetal genotype data available? (Y/N)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>BIRTH WEIGHT</td>
<td>Method by which offspring birth weight (and length, if available) were collected</td>
<td>Birth Register Data</td>
<td>From longitudinal surveys by self-report/parental report. Birth weight determined as average of all valid data points.</td>
<td>Self-report through questionnaire</td>
</tr>
<tr>
<td>GESTATIONAL AGE</td>
<td>Method by which gestational age was collected</td>
<td>Last menstrual period and Scans. Based on hospital records</td>
<td>From longitudinal surveys by self-report/parental report. Gestational age determined as average of all valid data points.</td>
<td>NA</td>
</tr>
</tbody>
</table>
**SUMMARY MATERNAL CHARACTERISTICS, where available in the INCLUDED sample,**
**DURING PREGNANCY (median (IQR) given where the trait distribution deviates strongly from the normal distribution**

<table>
<thead>
<tr>
<th></th>
<th>NFBC1966</th>
<th>NTR</th>
<th>QIMR</th>
<th>TWINSUK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age at delivery, unless otherwise stated [Mean (sd)], years</td>
<td>26.5 (3.7) available for 2010 participants</td>
<td>27.1 (3.7)</td>
<td>24.5 (4.0)</td>
<td>NA</td>
</tr>
<tr>
<td>Maternal pre-pregnancy BMI [Mean (sd)], kg/m²</td>
<td>NA</td>
<td>NA</td>
<td>22.79 (5.13)</td>
<td>NA</td>
</tr>
<tr>
<td>Maternal pregnancy BMI [Mean (sd)], kg/m²</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Fasting glucose [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Triglycerides [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>HDL-cholesterol [Mean (sd)], mmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Blood pressure [Mean (sd)], mmHg</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>25-hydroxyvitamin D [Median (IQR)], nmol/L</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Adiponectin [Mean (sd)], μg/mL</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>% of mothers who smoked in pregnancy</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Mean gestational week of collection of maternal characteristics</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Parity (% primiparous births)</td>
<td>NA</td>
<td>84.0%</td>
<td>NA</td>
<td>NA</td>
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</tbody>
</table>
## SUMMARY OFFSPRING CHARACTERISTICS

(Offspring of the INCLUDED sample of mothers)

<table>
<thead>
<tr>
<th></th>
<th>NFBC1966</th>
<th>NTR</th>
<th>QIMR</th>
<th>TWINSUK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight [Mean (sd), grams]</td>
<td>3525 (461)</td>
<td>3469 (529)</td>
<td>3344 (532)</td>
<td>3365 (581)</td>
</tr>
<tr>
<td>Gestational age at delivery [Median (IQR), weeks]</td>
<td>40 (39, 41)</td>
<td>40 (38, 42)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Birth length [Mean (sd), cm]</td>
<td>50.3 (2.0)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Ponderal index [Mean (sd), kg/m³]</td>
<td>27.61 (2.44)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

## REFERENCES


**Study URL**

- [NFBC website](http://www.oulu.fi/nfbc/)
- [Twin Register website](http://www.tweelingenregister.org)
- [QIMR website](http://www.genepl.qimr.edu.au/general/researchtopics.cgi)
- [TwinsUK website](http://www.twinsuk.co.uk)

NA, not available

Informed consent was obtained from all participants, and study protocols were approved by the local regional or institutional ethics committees
### Table 2(a) Genotyping information (studies 1-5)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>ALSPAC Mothers</th>
<th>Berlin Birth Cohort (BBC) Mothers</th>
<th>1958 British Birth Cohort or NCDS (B58C-WTCCC)</th>
<th>1958 British Birth Cohort or NCDS (B58C-T1DGC)</th>
<th>CHOP Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MATERNAL GENOME- OR EXOME-WIDE GENOTYPING</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina Human660W-Quad BeadChip</td>
<td>Human Exomechip ver1.1</td>
<td>Affymetrix Genome-wide Human SNP Array 6.0</td>
<td>Illumina 550K Infinium</td>
<td>Illumina550, Illumina610 Infinium</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Centre National de Génomotype (CNG), Evry, France</td>
<td>Oxfor Centre for Diabete, Endocrinology and Metabolism, University of Oxford, UK</td>
<td>Wellcome Trust Sanger Institute, Cambridge, UK</td>
<td>JDRF/WT DIL Lab in Cambridge, UK</td>
<td>The Center for Applied Genomics, Children's Hospital of Philadelphia, USA</td>
</tr>
<tr>
<td>N SNPs in QC’d dataset</td>
<td>526,688</td>
<td>NA</td>
<td>721,428</td>
<td>520,413</td>
<td>513,518</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>MACH v.1.0.16 / HapMap Phase II</td>
<td>NA</td>
<td>Impute /HapMap Phase II</td>
<td>Impute /HapMap Phase II</td>
<td>Impute /HapMap Phase II</td>
</tr>
<tr>
<td>N QC’d SNPs available for GWAS analysis</td>
<td>2,450,866</td>
<td>NA</td>
<td>2,543,926</td>
<td>2,451,644</td>
<td>2,546,219</td>
</tr>
<tr>
<td>Genomic control lambda from GWAS analysis of offspring birth weight</td>
<td>1.039</td>
<td>NA</td>
<td>0.984</td>
<td>1.007</td>
<td>NA</td>
</tr>
<tr>
<td><strong>FETAL GENOME- OR EXOME-WIDE GENOTYPING</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina HumanHap550 quad array</td>
<td>Human Exomechip ver1.1</td>
<td>NA</td>
<td>NA</td>
<td>Illumina550, Illumina610 Infinium</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Sample Logistics and Genotyping Facilities at the Wellcome Trust Sanger Institute and LabCorp (Laboratory Corporation of America) using support from 23andMe</td>
<td>Oxford Centre for Diabete, Endocrinology and Metabolism, University of Oxford, UK</td>
<td>NA</td>
<td>NA</td>
<td>The Center for Applied Genomics</td>
</tr>
<tr>
<td>N SNPs in QC’d dataset</td>
<td>500,541</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>513,518</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>MACH v.1.0.16 / HapMap Phase II</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Impute /HapMap Phase II</td>
</tr>
</tbody>
</table>

#### DATA ANALYSIS

<p>| Analysis software | Stata v.13 | PLINK and R | Stata, version 12 | Stata, version 12 | SNPtest and R |</p>
<table>
<thead>
<tr>
<th>REFERENCES</th>
<th>ALSPAC Mothers</th>
<th>Berlin Birth Cohort (BBC) Mothers</th>
<th>1958 British Birth Cohort or NCDS (B58C-WTCCC)</th>
<th>1958 British Birth Cohort or NCDS (B58C-T1DGC)</th>
<th>CHOP Mothers</th>
</tr>
</thead>
</table>
### Table 2(b) Genotyping information (studies 6-10)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>COPASAC-2000 Mothers</th>
<th>DNBC-GOYA Random Set</th>
<th>DNBC-PTB-CONTROL Mothers</th>
<th>EFSOCH Mothers</th>
<th>GEN-3G Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina 550K</td>
<td>Illumina Human610-Quad v1.0</td>
<td>Illumina Human 660W-quad Bead Array</td>
<td>Illumina Human Exome Beadchip v1</td>
<td>NA</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Children's Hospital of Philadelphia, Center for Applied Genomics</td>
<td>Centre National de Génomypage (CNG), Evry, France</td>
<td>Center for Inherited Disease Research, Johns Hopkins University, Baltimore, Maryland, USA</td>
<td>Centre National de Génomypage, France</td>
<td>NA</td>
</tr>
<tr>
<td>N SNPs in QC’d dataset</td>
<td>486,373</td>
<td>545,349</td>
<td>518,097</td>
<td>234,763</td>
<td>NA</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>MacH-minimac/Hapmap Phase II</td>
<td>Mach 1.0</td>
<td>MaCH/HapMap Phase II</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>N QC’d SNPs available for GWAS analysis</td>
<td>NA</td>
<td>2,449,993</td>
<td>2,543,887</td>
<td>57 QC’d SNPs available for analysis of genetic scores selected for the current project</td>
<td>NA</td>
</tr>
<tr>
<td>Genomic control lambda from GWAS analysis of offspring birth weight</td>
<td>NA</td>
<td>1.006</td>
<td>1.006</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

### MATERNAL GENOME- OR EXOME-WIDE GENOTYPING

<p>| Genotyping centre and method | NA | NA | NA | LGC Genomics (formerly Kbiosciences); KASPar | Genome Quebec Innovation Centre |
| Call rate [Median (range)]; N SNPs genotyped | NA | NA | NA | 0.953 [0.932, 0.999] (N=16 SNPs) | 0.99 [0.98 ; 1] (N=4 snps) |
| Did any SNPs deviate from HWE (Bonferroni corrected P&lt;0.05)? Y/N | NA | NA | NA | N | N |
| Duplicate concordance (%) | NA | NA | NA | &gt;99% (approx. 10%) | 100% |</p>
<table>
<thead>
<tr>
<th></th>
<th>COPSAC-2000 Mothers</th>
<th>DNBC-GOYA Random Set</th>
<th>DNBC-PTB-CONTROL Mothers</th>
<th>EFSOCH Mothers</th>
<th>GEN-3G Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina 550K</td>
<td>NA</td>
<td>Illumina Human 660W-quad Bead Array</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Children's Hospital of Philadelphia, Center for Applied Genomics</td>
<td>NA</td>
<td>Center for Inherited Disease Research, Johns Hopkins University, Baltimore, Maryland, USA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>N SNPs in QC'd dataset</td>
<td>486,373</td>
<td>NA</td>
<td>514,382</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>MacH-minimac/Hapmap Phase II</td>
<td>NA</td>
<td>MaCH/HapMap Phase II</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Genotyping centre and method</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>LGC Genomics (formerly Kbiosciences); KASP</td>
<td>NA</td>
</tr>
<tr>
<td>Call rate [Median (range)]; N SNPs genotyped</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>0.926 [0.907, 0.934] (N=13 SNPs)</td>
<td>NA</td>
</tr>
<tr>
<td>Did any SNPs deviate from HWE (Bonferroni corrected P&lt;0.05)? Y/N</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>N</td>
<td>NA</td>
</tr>
<tr>
<td>Duplicate concordance (% duplicated genotypes)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>&gt;99% (approx. 10%)</td>
<td>NA</td>
</tr>
<tr>
<td>Analysis software</td>
<td>R-project</td>
<td>Stata</td>
<td>R</td>
<td>Stata v.13</td>
<td>R</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>Reference - MATERNAL genotyping</td>
<td>REFERENCES</td>
<td>Reference - FETAL genotyping</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>---------------------------------</td>
<td>------------</td>
<td>-----------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GEN-3G Mothers</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GEN-3G Mothers</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STUDY</td>
<td>Generation R Mothers</td>
<td>HAPO Mothers (GWAS)</td>
<td>HAPO Mothers (nonGWAS)</td>
<td>MoBa (Mothers)</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>----------------------</td>
<td>---------------------</td>
<td>------------------------</td>
<td>---------------</td>
<td></td>
</tr>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>NA</td>
<td>Illumina Human 610 Quad v1 B SNP array</td>
<td>NA</td>
<td>Illumina 660Wquad</td>
<td></td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>NA</td>
<td>Broad Institute Center for Genotyping and Analysis (CGA), USA</td>
<td>NA</td>
<td>The Norwegian Cancer Hospital, Oslo</td>
<td></td>
</tr>
<tr>
<td>N SNPs in QC’d dataset</td>
<td>NA</td>
<td>559,739</td>
<td>NA</td>
<td>432,270</td>
<td></td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>NA</td>
<td>Beagle / HapMap3 CEU &amp; TSI</td>
<td>NA</td>
<td>PLINK /HapMap Phase II</td>
<td></td>
</tr>
<tr>
<td>N QC’d SNPs available for GWAS analysis</td>
<td>NA</td>
<td>1,968,447</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Genomic control lambda from GWAS analysis of offspring birth weight</td>
<td>NA</td>
<td>1.016</td>
<td>NA</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

**MATERNAL GENOME- OR EXOME-WIDE GENOTYPING**

<p>| Genotyping centre and method | LGC Genomics (formerly Kbiosciences); KASPar | NA | LGC Genomics (formerly Kbiosciences); KASPar | NA |
| Call rate [Median (range)]; N SNPs genotyped | 99.3% (N=34 snps) | NA | 0.983 [0.977, 0.988] (N=23 SNPs) | NA |
| Did any SNPs deviate from HWE (Bonferroni corrected P&lt;0.05)? Y/N | Y: rs4836133 (P = 3x10-15; excluded) | NA | N | NA |
| Duplicate concordance (% duplicated genotypes) | 99.80% | NA | &gt;99% (min 4%) | NA |</p>
<table>
<thead>
<tr>
<th>FETAL GENOME- OR EXOME-WIDE GENOTYPING</th>
<th>Generation R Mothers</th>
<th>HAPO Mothers (GWAS)</th>
<th>HAPO Mothers (non-GWAS)</th>
<th>MoBa (Mothers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina 610 Quad and 660W</td>
<td>Illumina Human 610 Quad v1 B SNP array</td>
<td>NA</td>
<td>Illumina 660W quad</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Human Genotyping Facility (HuGeF), Dept Internal Medicine, Erasmus MC, The Netherlands</td>
<td>Broad Institute Center for Genotyping and Analysis (CGA)</td>
<td>NA</td>
<td>The Norwegian Cancer Hospital, Oslo</td>
</tr>
<tr>
<td>N SNPs in QC’d dataset</td>
<td>489,879</td>
<td>559,739</td>
<td>NA</td>
<td>432270</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>Minimac and MACH</td>
<td>Beagle / HapMap3 CEU &amp; TSI</td>
<td>NA</td>
<td>PLINK / HapMap Phase II</td>
</tr>
</tbody>
</table>

| FETAL CUSTOM GENOTYPING                | Genotyping centre and method | NA | NA | LGC Genomics (formerly Kbiosciences); KASPar | NA |
| Call rate [Median (range)]; N SNPs genotyped | NA | NA | 0.983 [0.977, 0.988] (N=23 SNPs) | NA |
| Did any SNPs deviate from HWE (Bonferroni corrected P<0.05)? Y/N | NA | NA | N | NA |
| Duplicate concordance (%)             | NA | NA | >=99% (min 4%) | NA |

<table>
<thead>
<tr>
<th>DATA ANALYSIS</th>
<th>Analysis software</th>
<th>Stata version 12</th>
<th>R 3.0.2</th>
<th>Stata v.13</th>
<th>IBM SPSS Statistics 20</th>
</tr>
</thead>
</table>

| REFERENCES                             | Reference - MATERNAL genotyping | NA | NA | NA | NA |

**Table 2(d) Genotyping information (studies 16-18)**

<table>
<thead>
<tr>
<th>STUDY</th>
<th>NFBC1966</th>
<th>NTR</th>
<th>QIMR</th>
<th>TWINSUK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>Illumina HumanCNV-370DUO Analysis BeadChip</td>
<td>Perlegen-Affymetrix, Affymetrix 6.0, Illumina 370K, 600K, 1M Omni</td>
<td>HumanCNV370-Quadv3</td>
<td>HumanHap3001,2, HumanHap610Q, 1M-Duo and 1.2MDuo 1M</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>Broad Institute</td>
<td>Perlegen Sciences Mountain View CF USA, Finnish Genome Center Helsinki Finland, SNP technology Platform Uppsala Sweden, Molecular Epidemiology Leiden The Netherlands, Translational Genomics Research Institute Phoenix AZ USA, Institute of Human Genetics LIFE &amp; BRAIN Center Bonn Germany</td>
<td>CIDR</td>
<td>Sanger</td>
</tr>
<tr>
<td>MATERNAL GENOME OR EXOME-WIDE GENOTYPING</td>
<td>N SNPs in QC’d dataset</td>
<td>324,896</td>
<td>312,214-814,708</td>
<td>323,093, reduced to a common set of 274,604 SNPs (across the QIMR sample)</td>
</tr>
<tr>
<td>Imputation software / reference panel</td>
<td>Impute version 2 / HapMap2</td>
<td>Impute 1.0 / Build 36r24 Hapmap 2</td>
<td>MACH/HapMap Phase II</td>
<td>IMPUTE software package (v2) 5 using two reference panels, P0 (HapMap2, rel 22, combined CEU+YRI+ASN panels) and P1 (610k+, including combinedHumanHap610k and 1M reduced to 610k SNP content).</td>
</tr>
<tr>
<td>N QC’d SNPs available for GWAS analysis</td>
<td>2,487,934</td>
<td>2385474</td>
<td>2,454,244</td>
<td>2,401,373</td>
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<tr>
<td>Genomic control lambda from GWAS analysis of offspring birth weight</td>
<td>1.020</td>
<td>1.002</td>
<td>1.012</td>
<td>1</td>
</tr>
<tr>
<td>GENOTYPING PLATFORM AND SNP PANEL</td>
<td>NFBC1966</td>
<td>NTR</td>
<td>QIMR</td>
<td>TWINSUK</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>----------</td>
<td>-----</td>
<td>------</td>
<td>---------</td>
</tr>
<tr>
<td>Genotyping platform and SNP panel</td>
<td>NA</td>
<td>Perlegen-Affymetrix, Affymetrix 6.0, Illumina 370K, 600K, 1M Omni</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Genotyping centre</td>
<td>NA</td>
<td>Perlegen Sciences Mountain View CF USA, Finnish Genome Center Helsinki Finland, SNP technology Platform Uppsala Sweden, Molecular Epidemiology Leiden The Netherlands, Translational Genomics Research Institute Phoenix AZ USA, Institute of Human Genetics LIFE &amp; BRAIN Center Bonn Germany.</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

| N SNPs in QC’d dataset           | NA       | 312214-814708 | NA | NA |
| Imputation software / reference panel | NA       | Impute 1.0 / Build 36r24 Hapmap 2 | NA | NA |

| DATA ANALYSIS | Analysis software | R 2.14.2 | Stata | R | SNPTEST, Stata |

<table>
<thead>
<tr>
<th>REFERENCES</th>
<th>Reference - MATERNAL genotyping</th>
</tr>
</thead>
</table>
## Table 3. Details of single nucleotide polymorphisms (SNPs) used to construct the genetic scores

<table>
<thead>
<tr>
<th>Trait</th>
<th>SNP</th>
<th>Nearest/nearby gene</th>
<th>Trait-raising allele*</th>
<th>Trait-lowering allele*</th>
<th>Beta for weighting genetic score</th>
<th>Units of beta and source (including any extra details)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adiponectin</td>
<td>rs3774261</td>
<td>ADIPOQ</td>
<td>A</td>
<td>G</td>
<td>0.354</td>
<td></td>
</tr>
<tr>
<td>Adiponectin</td>
<td>rs3821799</td>
<td>ADIPOQ</td>
<td>C</td>
<td>T</td>
<td>0.352</td>
<td></td>
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</tbody>
</table>

<table>
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<td></td>
</tr>
<tr>
<td>Triglyceride main effect</td>
<td>rs5756931</td>
<td>PLA2G5</td>
<td>T</td>
<td>C</td>
<td>1.54</td>
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</tr>
<tr>
<td>Triglyceride main effect</td>
<td>rs645040</td>
<td>MSL2L1</td>
<td>T</td>
<td>G</td>
<td>2.22</td>
<td></td>
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<tr>
<td>Triglyceride main effect</td>
<td>rs714052</td>
<td>BAZ1B</td>
<td>A</td>
<td>G</td>
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<tr>
<td>Triglyceride main effect</td>
<td>rs964184</td>
<td>APOA1</td>
<td>G</td>
<td>C</td>
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<tr>
<td>Triglyceride main effect</td>
<td>rs9686661</td>
<td>MAP3K1</td>
<td>T</td>
<td>C</td>
<td>2.57</td>
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</table>

Units: mg/dL per triglyceride raising allele
<table>
<thead>
<tr>
<th>Trait</th>
<th>SNP</th>
<th>Nearest/nearby gene</th>
<th>Trait-raising allele</th>
<th>Trait-lowering allele</th>
<th>Beta for weighting genetic score</th>
<th>Units of beta and source (including any extra details)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 2 diabetes</td>
<td>rs10203174</td>
<td>THADA</td>
<td>C</td>
<td>T</td>
<td>0.131</td>
<td>Units: Natural logarithm of odds ratio for type 2 diabetes per type 2 diabetes increasing allele. Source: Morris et al., (2012) Nature Genetics.</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>rs10758593</td>
<td>GLIS3</td>
<td>A</td>
<td>G</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs10811661</td>
<td>CDKN2A/B</td>
<td>T</td>
<td>C</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs10842994</td>
<td>KLHC5</td>
<td>C</td>
<td>T</td>
<td>0.095</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs10923931</td>
<td>NOTCH2</td>
<td>T</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs11063069</td>
<td>CCND2</td>
<td>G</td>
<td>A</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs1111875</td>
<td>HHEX/IDE</td>
<td>C</td>
<td>T</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs11257655</td>
<td>CDC123/CAMK1D</td>
<td>T</td>
<td>C</td>
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<td>Type 2 diabetes</td>
<td>rs11634397</td>
<td>ZFAND6</td>
<td>G</td>
<td>A</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs11717195</td>
<td>ADCY5</td>
<td>T</td>
<td>C</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs12242953</td>
<td>VPS26A</td>
<td>G</td>
<td>A</td>
<td>0.068</td>
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</tr>
<tr>
<td>Type 2 diabetes</td>
<td>rs12427353</td>
<td>HNF1A (TCF1)</td>
<td>G</td>
<td>C</td>
<td>0.077</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs12497268</td>
<td>PSMD6</td>
<td>G</td>
<td>C</td>
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<td>rs12571751</td>
<td>ZMIZ1</td>
<td>A</td>
<td>G</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs1289811</td>
<td>PC1</td>
<td>G</td>
<td>A</td>
<td>0.077</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs1359790</td>
<td>SPRY2</td>
<td>G</td>
<td>A</td>
<td>0.077</td>
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<td>Type 2 diabetes</td>
<td>rs1496653</td>
<td>UBE2E2</td>
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<td>G</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs152224</td>
<td>ARAPI (CENTD2)</td>
<td>A</td>
<td>C</td>
<td>0.104</td>
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<td>Type 2 diabetes</td>
<td>rs163184</td>
<td>KCNJ1</td>
<td>G</td>
<td>T</td>
<td>0.086</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs16927668</td>
<td>PTPRD</td>
<td>T</td>
<td>C</td>
<td>0.039</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs17684886</td>
<td>DGKB</td>
<td>T</td>
<td>C</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs1781515</td>
<td>ST6GaL1</td>
<td>A</td>
<td>G</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs1791515</td>
<td>TLE4</td>
<td>A</td>
<td>G</td>
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<td>Type 2 diabetes</td>
<td>rs1801282</td>
<td>GCC1</td>
<td>T</td>
<td>G</td>
<td>0.086</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs2007084</td>
<td>APIS</td>
<td>G</td>
<td>A</td>
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<tr>
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<td>PROX1</td>
<td>G</td>
<td>T</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs2261181</td>
<td>HMG42</td>
<td>T</td>
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<td>rs2334499</td>
<td>DUSP8</td>
<td>T</td>
<td>C</td>
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<tr>
<td>Type 2 diabetes</td>
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<td>BCL11A</td>
<td>T</td>
<td>A</td>
<td>0.068</td>
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<tr>
<td>Type 2 diabetes</td>
<td>rs2447090</td>
<td>SBR</td>
<td>A</td>
<td>G</td>
<td>0.039</td>
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<td>G</td>
<td>A</td>
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<td>rs3734621</td>
<td>KCNK16</td>
<td>C</td>
<td>A</td>
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<td>SLC30A8</td>
<td>G</td>
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<td>Type 2 diabetes</td>
<td>rs4299828</td>
<td>ZFAND3</td>
<td>A</td>
<td>G</td>
<td>0.039</td>
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<td>Type 2 diabetes</td>
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<td>IGF2BP2</td>
<td>T</td>
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<td>WFS1</td>
<td>G</td>
<td>T</td>
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<td>C2CD4A</td>
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<td>Type 2 diabetes</td>
<td>rs459193</td>
<td>ANKRD55</td>
<td>G</td>
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<td>Type 2 diabetes</td>
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<td>C</td>
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<td>KCNJ11</td>
<td>C</td>
<td>T</td>
<td>0.068</td>
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<tr>
<td>Trait</td>
<td>SNP</td>
<td>Nearest/nearby gene</td>
<td>Trait- raising allele</td>
<td>Trait- lowering allele</td>
<td>Beta for weighting genetic score</td>
<td>Units of beta and source (including any extra details)</td>
</tr>
<tr>
<td>-------</td>
<td>-----------</td>
<td>---------------------</td>
<td>----------------------</td>
<td>-----------------------</td>
<td>---------------------------------</td>
<td>-------------------------------------------------------</td>
</tr>
<tr>
<td>Vitamin D - synthesis</td>
<td>rs10741657</td>
<td>CYP2R1</td>
<td>A</td>
<td>G</td>
<td>0.03</td>
<td>Units: nmol/L per vitamin D raising allele Source: Wang et al., (2010) Lancet</td>
</tr>
<tr>
<td>Vitamin D - synthesis</td>
<td>rs12785878</td>
<td>DHCR7/ADSYN1</td>
<td>T</td>
<td>G</td>
<td>0.05</td>
<td>Source: Wang et al., (2010) Lancet</td>
</tr>
</tbody>
</table>

- Based on the positive strand according to HapMap Phase 2
- HDL-specific SNPs were selected due to being near genes with known Mendelian lipid disorder
- Triglyceride main effect SNPs were included in the genetic score if they were solely associated with triglyceride levels, or if their effect on triglyceride levels was at least three times greater than that of HDL-, LDL-, or total cholesterol (using Teslovich et al 2010, Nat Genet).
<table>
<thead>
<tr>
<th>Trait</th>
<th>Total N women</th>
<th>N Studies</th>
<th>Study Name</th>
<th>Time of ascertainment</th>
<th>Brief description of maternal phenotype ascertainment and reference, if available</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI</strong></td>
<td>11,822</td>
<td>5</td>
<td>ALSPAC Mothers</td>
<td>Pre-pregnancy</td>
<td>Self-reported weight and height, weight validated with clinic measure (Lawlor et al., (2010) Diabetologia 53: 89-97)²²</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>EFSCOCH Mothers</td>
<td>Pre-pregnancy</td>
<td>Measured height 3 times and averaged, pre-pregnancy weight self reported (Knight et al., (2006) Paediatric Perinatal Epidemiology 20:172-179)²⁶</td>
</tr>
<tr>
<td><strong>Fasting glucose</strong></td>
<td>5,402</td>
<td>3</td>
<td>EFSCOCH Mothers</td>
<td>Gestational week 28</td>
<td>Fasting blood samples (10 hours fasting minimum) (Knight et al., (2006) Paediatric Perinatal Epidemiology 20:172-179)²⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HAPO Mothers (non-GWAS)</td>
<td>Gestational week 28</td>
<td></td>
</tr>
<tr>
<td><strong>Gestational or existing</strong></td>
<td>6,827</td>
<td>1</td>
<td>ALSPAC Mothers</td>
<td>Gestational week 28</td>
<td>Questionnaire at recruitment about existing diabetes and history of gestational diabetes. Data abstracted on gestational diabetes and glycosuria (Lawlor et al., (2010) Diabetologia 53: 89-97)²²</td>
</tr>
<tr>
<td><strong>Triglycerides</strong></td>
<td>663</td>
<td>1</td>
<td>EFSCOCH Mothers</td>
<td>Gestational week 28</td>
<td>Fasting blood samples (10 hours fasting minimum) (Knight et al., (2006) Paediatric Perinatal Epidemiology 20:172-179)²⁶</td>
</tr>
<tr>
<td><strong>HDL</strong></td>
<td>733</td>
<td>1</td>
<td>EFSCOCH Mothers</td>
<td>Gestational week 28</td>
<td>Fasting blood samples (10 hours fasting minimum) (Knight et al., (2006) Paediatric Perinatal Epidemiology 20:172-179)²⁶</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>9,100</td>
<td>3</td>
<td>ALSPAC Mothers</td>
<td>Gestational week 28</td>
<td>Data abstracted from obstetric medical charts at various time points in pregnancy. Data for 28 weeks gestation predicted using fractional polynomials and spline multilevel models. (Macdonald-Wallis et al., (2011) Journal of Hypertension 29: 1703-1711)²⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Molilla Mothers</td>
<td>Gestational week 18</td>
<td>Self-reported blood pressure from medical card <a href="http://www.fhi.no/dokumenter/1f32a49514.pdf">http://www.fhi.no/dokumenter/1f32a49514.pdf</a></td>
</tr>
<tr>
<td><strong>Vitamin D status</strong></td>
<td>5,305</td>
<td>2</td>
<td>ALSPAC Mothers</td>
<td>Gestational week 28</td>
<td>Measured in non-fasting blood samples. Data for 28 weeks gestation predicted using fractional polynomials and spline multilevel models. (Lawlor et al., (2013) Lancet 6736: 62203)³⁷</td>
</tr>
<tr>
<td>(25(OH)D levels)</td>
<td></td>
<td></td>
<td>GEN-3G</td>
<td>Yes (9 weeks)</td>
<td>Measured in fasting serum samples (Lowe et al., (2010) Journal Clinical Endocrinology and Metabolism 95: 5427-5434)³⁷</td>
</tr>
<tr>
<td><strong>Adiponectin</strong></td>
<td>1,376</td>
<td>1</td>
<td>HAPO Mothers</td>
<td>Gestational week 28</td>
<td>Measured in fasting serum samples (Lowe et al., (2010) Journal Clinical Endocrinology and Metabolism 95: 5427-5434)³⁷</td>
</tr>
</tbody>
</table>

*Blood pressure was measured in the Molilla study and showed strong evidence of association with the genetic score (P<0.001), but since it was measured at 18 weeks of gestation, we chose not to meta-analyse with ALSPAC and HAPO data (measured at 28 weeks). Vitamin D status was measured in the GEN-3G study and showed strong evidence of association with the genetic score (P<5x10^-5), but since it was measured at 9 weeks of gestation, we chose not to meta-analyse with ALSPAC data above (measured at 28 weeks).*
Table 5. Associations between maternal genetic scores and maternal traits during and post-pregnancy in the same individuals

<table>
<thead>
<tr>
<th>Trait</th>
<th>Study</th>
<th>N women with both pregnancy and post-pregnancy data available</th>
<th>Mean (SD) of trait measured during pregnancy</th>
<th>Change in maternal trait per trait-increasing allele (95% CI) during pregnancy</th>
<th>P value (during pregnancy)</th>
<th>Mean (SD) of trait measured post-pregnancy</th>
<th>Mean (SD) age of mother when post-pregnancy measurement taken</th>
<th>Change in maternal trait per trait-increasing allele (95% CI) post-pregnancy</th>
<th>P value (post-pregnancy)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>ALSPAC</td>
<td>2,927</td>
<td>26.02 (3.55)</td>
<td>29.7 (4.4)</td>
<td>0.13 (0.09, 0.16)</td>
<td>26.54 (5.21)</td>
<td>48.0 (4.4)</td>
<td>0.14 (0.09, 0.19)</td>
<td>1x10⁻⁴</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>EFSOCH</td>
<td>456</td>
<td>27.55 (4.19)</td>
<td>31.5 (4.7)</td>
<td>0.21 (0.11, 0.32)</td>
<td>25.03 (4.60)</td>
<td>36.8 (4.9)</td>
<td>0.16 (0.05, 0.28)</td>
<td>0.006</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>EFSOCH</td>
<td>312</td>
<td>4.39 (0.38)</td>
<td>32.0 (4.4)</td>
<td>0.04 (0.02, 0.05)</td>
<td>4.60 (0.48)</td>
<td>37.1 (4.7)</td>
<td>0.04 (0.02, 0.06)</td>
<td>2x10⁻⁴</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>EFSOCH</td>
<td>360</td>
<td>2.13 (0.70)</td>
<td>31.6 (4.7)</td>
<td>0.05 (0.03, 0.08)</td>
<td>0.91 (0.40)</td>
<td>36.9 (4.9)</td>
<td>0.03 (0.01, 0.05)</td>
<td>4x10⁻⁴</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/L)</td>
<td>EFSOCH</td>
<td>408</td>
<td>2.10 (0.46)</td>
<td>31.5 (4.7)</td>
<td>0.02 (0.01, 0.03)</td>
<td>1.71 (0.42)</td>
<td>36.8 (4.9)</td>
<td>0.03 (0.02, 0.04)</td>
<td>7x10⁻⁴</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>ALSPAC</td>
<td>2,930</td>
<td>112.3 (7.2)</td>
<td>29.6 (4.5)</td>
<td>0.17 (0.10, 0.24)</td>
<td>117.9 (12.2)</td>
<td>47.9 (4.4)</td>
<td>0.43 (0.31, 0.56)</td>
<td>2x10⁻¹²</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure
Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

(a) BMI genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, EFSOCH, HAPO (GWAS), DNBC-GOYA-RANDOM, DNBC-PTB-CONTROLS [0.18]</td>
<td>11,822</td>
<td>0.145 (0.126, 0.164)</td>
<td>&lt; 2x10^{-16}</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>-</td>
<td>EFSOCH</td>
<td>438</td>
<td>0.001 (-0.001, 0.003)</td>
<td>0.18</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO, EFSOCH [0.14]</td>
<td>2,104</td>
<td>0.005 (0.001, 0.009)</td>
<td>0.026</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>1.04 (0.97, 1.12)</td>
<td>0.28</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>735</td>
<td>0.009 (-0.006, 0.023)</td>
<td>0.25</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>732</td>
<td>-0.008 (-0.017, 0.002)</td>
<td>0.11</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>727</td>
<td>-0.004 (-0.026, 0.019)</td>
<td>0.76</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO [0.08]</td>
<td>8,450</td>
<td>0.07 (0.02, 0.11)</td>
<td>0.003</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td></td>
<td>ALSPAC</td>
<td>4,767</td>
<td>0.002 (-0.001, 0.006)</td>
<td>0.25</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>-0.001 (0.000, 0.000)</td>
<td>0.08</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO, EFSOCH [0.08]</td>
<td>9,212</td>
<td>1.00 (1.00, 1.01)</td>
<td>0.19</td>
</tr>
<tr>
<td>Highest educational qualification attained*</td>
<td></td>
<td>ALSPAC</td>
<td>6,855</td>
<td>-0.00 (-0.01, 0.01)</td>
<td>0.63</td>
</tr>
<tr>
<td>Occupational position*</td>
<td></td>
<td>ALSPAC</td>
<td>5,766</td>
<td>1.00 (0.99, 1.02)</td>
<td>0.67</td>
</tr>
<tr>
<td>Townsend deprivation score*</td>
<td></td>
<td>EFSOCH</td>
<td>612</td>
<td>0.015 (-0.003, 0.033)</td>
<td>0.11</td>
</tr>
</tbody>
</table>

*Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

*Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.

*National Statistics Socio Economic Class Occupation Code (3). Subjects grouped as 1=managerial & professional; 2=intermediate; 3=routine & manual

*Townsend deprivation score, a continuous variable based on UK postal code: 0=average; >0=more deprived; <0=more affluent
Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

(b) Fasting glucose genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m$^2$</td>
<td>ALSPAC, EFSOCH, HAPO (GWAS) [0.89]</td>
<td>8,232</td>
<td>0.007 (-0.025, 0.039)</td>
<td>0.68</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>-</td>
<td>EFSOCH</td>
<td>320</td>
<td>0.000 (-0.002, 0.003)</td>
<td>0.74</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO, EFSOCH [0.70]</td>
<td>5,402</td>
<td>0.029 (0.025, 0.032)</td>
<td>&lt; 2x10^-16</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>1.06 (0.95, 1.17)</td>
<td>0.29</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>537</td>
<td>-0.007 (-0.029, 0.016)</td>
<td>0.57</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>535</td>
<td>-0.004 (-0.018, 0.010)</td>
<td>0.57</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>531</td>
<td>-0.014 (-0.049, 0.022)</td>
<td>0.45</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.69]</td>
<td>8,450</td>
<td>0.038 (-0.026, 0.102)</td>
<td>0.25</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td>-</td>
<td>ALSPAC</td>
<td>4,767</td>
<td>0.003 (-0.003, 0.008)</td>
<td>0.34</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>-0.001 (-0.006, 0.004)</td>
<td>0.72</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS), EFSOCH [0.49]</td>
<td>9,012</td>
<td>0.97 (0.99, 1.00)</td>
<td>0.32</td>
</tr>
<tr>
<td>Highest educational qualification attained$^a$</td>
<td>-</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>-0.01 (-0.02, 0.01)</td>
<td>0.24</td>
</tr>
<tr>
<td>Occupational position$^b$</td>
<td>-</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>1.01 (0.98, 1.03)</td>
<td>0.68</td>
</tr>
<tr>
<td>Occupational position$^c$</td>
<td>-</td>
<td>EFSOCH</td>
<td>447</td>
<td>-0.018 (-0.046, 0.011)</td>
<td>0.22</td>
</tr>
<tr>
<td>Townsend deprivation score$^d$</td>
<td>-</td>
<td>EFSOCH</td>
<td>542</td>
<td>-0.050 (-0.151, 0.051)</td>
<td>0.33</td>
</tr>
</tbody>
</table>

$^a$Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

$^b$Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.

$^c$National Statistics Socio Economic Class Occupation Code (3). Subjects grouped as 1=managerial & professional; 2=intermediate; 3=routine & manual

$^d$Townsend deprivation score, a continuous variable based on UK postal code: 0=average; >0=more deprived; <0=more affluent
### cTable 6. Associations between each maternal genetic score and potentially confounding or mediating variables

#### (c) Type 2 diabetes genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, HAPO (GWAS) [0.09]</td>
<td>7,901</td>
<td>0.010 (-0.008, 0.028)</td>
<td>0.28</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.002 (-0.002, 0.006)</td>
<td>0.25</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>1.08 (1.03, 1.14)</td>
<td>0.003</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.27]</td>
<td>8,450</td>
<td>0.037 (0.004, 0.071)</td>
<td>0.028</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td></td>
<td>ALSPAC</td>
<td>4,767</td>
<td>0.001 (-0.002, 0.004)</td>
<td>0.37</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.001 (-0.002, 0.003)</td>
<td>0.63</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS) [0.24]</td>
<td>8,471</td>
<td>1.00 (1.00, 1.00)</td>
<td>0.55</td>
</tr>
<tr>
<td>Highest educational qualification attaineda</td>
<td></td>
<td>ALSPAC</td>
<td>6,855</td>
<td>-0.01 (-0.01, 0.00)</td>
<td>0.10</td>
</tr>
<tr>
<td>Occupational positionb</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>1.00 (0.99, 1.01)</td>
<td>0.95</td>
</tr>
</tbody>
</table>

*aSubjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

*bDerived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.
### Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

#### (d) Triglycerides genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, EFSOCH, HAPO (GWAS) [0.06]</td>
<td>8,353</td>
<td>-0.007 (-0.041, 0.027)</td>
<td>0.70</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>-</td>
<td>EFSOCH</td>
<td>392</td>
<td>0.000 (-0.002, 0.003)</td>
<td>0.84</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO (GWAS), EFSOCH [0.07]</td>
<td>2,036</td>
<td>0.002 (-0.003, 0.008)</td>
<td>0.42</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>0.93 (0.83, 1.04)</td>
<td>0.19</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>660</td>
<td>0.003 (-0.018, 0.012)</td>
<td>0.71</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>656</td>
<td>0.003 (-0.033, 0.039)</td>
<td>0.88</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>656</td>
<td>0.003 (-0.033, 0.039)</td>
<td>0.88</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.97]</td>
<td>8,450</td>
<td>0.002 (-0.065, 0.069)</td>
<td>0.96</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td>-</td>
<td>ALSPAC</td>
<td>4,767</td>
<td>0.003 (-0.003, 0.009)</td>
<td>0.27</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>ln[log10(ug/ml)]</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.000 (-0.004, 0.004)</td>
<td>0.93</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS), EFSOCH [0.23]</td>
<td>9,142</td>
<td>1.00 (0.99, 1.01)</td>
<td>0.87</td>
</tr>
<tr>
<td>Highest educational qualification attained</td>
<td>-</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>0.00 (-0.01, 0.01)</td>
<td>0.78</td>
</tr>
<tr>
<td>Occupational position</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>1.01 (0.98, 1.04)</td>
<td>0.47</td>
</tr>
<tr>
<td>Occupational position</td>
<td>Odds ratio</td>
<td>EFSOCH</td>
<td>556</td>
<td>-0.004 (-0.034, 0.026)</td>
<td>0.81</td>
</tr>
<tr>
<td>Townsend deprivation score</td>
<td>-</td>
<td>EFSOCH</td>
<td>671</td>
<td>0.009 (-0.097, 0.115)</td>
<td>0.87</td>
</tr>
</tbody>
</table>

*Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree
*Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.
*National Statistics Socio Economic Class Occupation Code (3). Subjects grouped as 1=managerial & professional; 2=intermediate; 3=routine & manual.
*Townsend deprivation score, a continuous variable based on UK postal code: 0=average; >0=more deprived; <0=more affluent
### Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

#### (e) HDL-cholesterol genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, EFSOCH, HAPO (GWAS) [0.10]</td>
<td>8,420</td>
<td>-0.048 (-0.097, 0.002)</td>
<td>0.06</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>-</td>
<td>EFSOCH</td>
<td>438</td>
<td>0.002 (-0.002, 0.005)</td>
<td>0.40</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO (GWAS), EFSOCH [0.48]</td>
<td>2,107</td>
<td>-0.002 (-0.011, 0.007)</td>
<td>0.70</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>0.83 (0.70, 0.99)</td>
<td>0.04</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>736</td>
<td>0.001 (-0.035, 0.037)</td>
<td>0.96</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>733</td>
<td>0.050 (0.027, 0.072)</td>
<td>1x10⁻³</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>728</td>
<td>-0.032 (-0.088, 0.024)</td>
<td>0.26</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.77]</td>
<td>8,450</td>
<td>-0.013 (-0.112, 0.085)</td>
<td>0.79</td>
</tr>
<tr>
<td>Vitamin D, ln(25(OH)D)</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>-0.000 (-0.009, 0.008)</td>
<td>0.95</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>-0.001 (-0.007, 0.005)</td>
<td>0.81</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS), EFSOCH [0.38]</td>
<td>9,215</td>
<td>1.00 (0.99, 1.00)</td>
<td>0.29</td>
</tr>
<tr>
<td>Highest educational qualification attained*</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>0.01 (-0.01, 0.02)</td>
<td>0.55</td>
</tr>
<tr>
<td>Occupational position*</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>1.00 (0.96, 1.04)</td>
<td>0.93</td>
<td></td>
</tr>
<tr>
<td>Occupational position*</td>
<td>EFSOCH</td>
<td>613</td>
<td>0.004 (-0.042, 0.049)</td>
<td>0.87</td>
<td></td>
</tr>
<tr>
<td>Townsend deprivation score*</td>
<td>EFSOCH</td>
<td>744</td>
<td>0.081 (-0.081, 0.243)</td>
<td>0.33</td>
<td></td>
</tr>
</tbody>
</table>

*Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

*Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.

*National Statistics Socio Economic Class Occupation Code (3). Subjects grouped as 1=managerial & professional; 2=intermediate; 3=routine & manual

*Townsend deprivation score, a continuous variable based on UK postal code: 0=average; >0=more deprived; <0=more affluent
### Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

#### (f) Systolic blood pressure genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, HAPO (GWAS) [0.78]</td>
<td>7,741</td>
<td>-0.011 (-0.030, 0.008)</td>
<td>0.27</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.002 (-0.003, 0.007)</td>
<td>0.45</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>0.98 (0.91, 1.05)</td>
<td>0.52</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.04]</td>
<td>8,450</td>
<td>0.186 (0.140, 0.231)</td>
<td>&lt; 2x10⁻¹⁶</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td>ALSPAC</td>
<td>4,767</td>
<td>-0.001 (-0.005, 0.002)</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log10(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.001 (-0.002, 0.004)</td>
<td>0.46</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS) [0.36]</td>
<td>8,471</td>
<td>1.00 (0.99, 1.00)</td>
<td>0.17</td>
</tr>
<tr>
<td>Highest educational qualification attaineda</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>0.00 (-0.00, 0.01)</td>
<td>0.32</td>
<td></td>
</tr>
<tr>
<td>Occupational positionb</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>0.99 (0.97, 1.01)</td>
<td>0.43</td>
<td></td>
</tr>
</tbody>
</table>

aSubjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

bDerived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.
### Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

#### (g) Vitamin D genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, EFSOCH, HAPO (GWAS) [0.86]</td>
<td>8,420</td>
<td>0.073 (-0.019, 0.164)</td>
<td>0.12</td>
</tr>
<tr>
<td>Waist/Hip Ratio</td>
<td>-</td>
<td>EFSOCH</td>
<td>438</td>
<td>-0.001 (-0.008, 0.005)</td>
<td>0.68</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO (GWAS), EFSOCH [0.96]</td>
<td>2,109</td>
<td>-0.001 (-0.019, 0.016)</td>
<td>0.96</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>1.19 (0.88, 1.62)</td>
<td>0.25</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>736</td>
<td>0.001 (-0.060, 0.061)</td>
<td>0.98</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>733</td>
<td>-0.034 (-0.072, 0.005)</td>
<td>0.09</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>mmol/L</td>
<td>EFSOCH</td>
<td>728</td>
<td>0.048 (-0.046, 0.141)</td>
<td>0.32</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.98]</td>
<td>8,454</td>
<td>-0.030 (-0.211, 0.151)</td>
<td>0.98</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td>-</td>
<td>ALSPAC</td>
<td>4,767</td>
<td>0.024 (0.009, 0.039)</td>
<td>0.002</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>log₁₀(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,339</td>
<td>-0.029 (-0.772, 0.713)</td>
<td>0.94</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS), EFSOCH [0.83]</td>
<td>9,217</td>
<td>1.00 (0.95, 1.05)</td>
<td>0.98</td>
</tr>
<tr>
<td>Highest educational qualification attained&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>0.033 (0.001, 0.065)</td>
<td>0.05</td>
</tr>
<tr>
<td>Occupational position&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>0.95 (0.88, 1.02)</td>
<td>0.17</td>
</tr>
<tr>
<td>Townsend deprivation score&lt;sup&gt;d&lt;/sup&gt;</td>
<td>-</td>
<td>EFSOCH</td>
<td>613</td>
<td>0.057 (-0.017, 0.130)</td>
<td>0.13</td>
</tr>
</tbody>
</table>

### Notes

<sup>a</sup>Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

<sup>b</sup>Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.

<sup>c</sup>National Statistics Socio Economic Class Occupation Code (3). Subjects grouped as 1=managerial & professional; 2=intermediate; 3=routine & manual

<sup>d</sup>Townsend deprivation score, a continuous variable based on UK postal code: 0=average; >0=more deprived; <0=more affluent
Table 6. Associations between each maternal genetic score and potentially confounding or mediating variables

(h) Adiponectin genetic score

<table>
<thead>
<tr>
<th>Outcome variable tested for association (measured or ascertained during pregnancy, except BMI and WHR)</th>
<th>Units of outcome variable</th>
<th>Study(ies) [Phet from meta-analysis]</th>
<th>Total N women</th>
<th>Estimated change in outcome variable per trait-raising allele (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>kg/m²</td>
<td>ALSPAC, HAPO (GWAS) [0.79]</td>
<td>7,741</td>
<td>-0.04 (-0.23, 0.14)</td>
<td>0.61</td>
</tr>
<tr>
<td>Waist-Hip Ratio</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>mmol/L</td>
<td>HAPO</td>
<td>1,376</td>
<td>-0.01 (-0.06, 0.04)</td>
<td>0.69</td>
</tr>
<tr>
<td>Gestational/existing diabetes</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>6,827</td>
<td>1.57 (0.94, 2.64)</td>
<td>0.09</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>NA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>mmHg</td>
<td>ALSPAC, HAPO (GWAS) [0.66]</td>
<td>8,450</td>
<td>-0.047 (-0.389, 0.295)</td>
<td>0.79</td>
</tr>
<tr>
<td>Vitamin D, ln[25(OH)D]</td>
<td>NA</td>
<td>ALSPAC</td>
<td>4,767</td>
<td>-0.015 (-0.043, 0.012)</td>
<td>0.28</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>ln(ug/ml)</td>
<td>HAPO (GWAS)</td>
<td>1,376</td>
<td>0.17 (0.11, 0.23)</td>
<td>1x10⁻⁸</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>Odds ratio</td>
<td>ALSPAC, HAPO (GWAS) [0.91]</td>
<td>8,471</td>
<td>0.97 (0.93, 1.02)</td>
<td>0.22</td>
</tr>
<tr>
<td>Highest educational qualification attaineda</td>
<td>NA</td>
<td>ALSPAC</td>
<td>6,855</td>
<td>-0.04 (-0.10, 0.02)</td>
<td>0.16</td>
</tr>
<tr>
<td>Occupational positionb</td>
<td>Odds ratio</td>
<td>ALSPAC</td>
<td>5,766</td>
<td>0.96 (0.84, 1.10)</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

bDerived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.
Table 7. Associations between maternal genetic scores and ponderal index of offspring at birth

<table>
<thead>
<tr>
<th>Maternal exposure for which genetic score was constructed</th>
<th>N Studies</th>
<th>Total N women</th>
<th>Change in ponderal index z-score per additional maternal trait raising/lowering allele (95% CI)</th>
<th>Equivalent change in PI (kgm²) per allele (95% CI)</th>
<th>P value</th>
<th>Heterogeneity P Value (I²) from meta-analysis</th>
<th>N Studies with fetal genotype</th>
<th>Total N offspring with genotype data</th>
<th>Change in PI z-score per additional maternal trait raising/lowering allele (95% CI)</th>
<th>Equivalent change in PI (kgm²) per allele (95% CI)</th>
<th>P value</th>
<th>Heterogeneity P Value (I²) from meta-analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher pre-pregnancy BMI</td>
<td>10</td>
<td>17,743</td>
<td>0.006 (0.002, 0.010)</td>
<td>0.02 (0.01, 0.03)</td>
<td>0.003</td>
<td>0.48 (0)</td>
<td>7</td>
<td>9,628</td>
<td>0.006 (0.000, 0.012)</td>
<td>0.02 (0.00, 0.03)</td>
<td>0.05</td>
<td>0.38 (6.5)</td>
</tr>
<tr>
<td>Higher fasting glucose</td>
<td>10</td>
<td>17,818</td>
<td>0.007 (0.001, 0.012)</td>
<td>0.02 (0.00, 0.03)</td>
<td>0.02</td>
<td>0.49 (0)</td>
<td>8</td>
<td>9,492</td>
<td>0.014 (0.005, 0.022)</td>
<td>0.04 (0.01, 0.06)</td>
<td>0.001</td>
<td>0.78 (0)</td>
</tr>
<tr>
<td>Higher odds of type 2 Diabetes</td>
<td>7</td>
<td>13,518</td>
<td>0.002 (-0.002, 0.005)</td>
<td>0.01 (-0.01, 0.01)</td>
<td>0.3</td>
<td>0.20 (29.6)</td>
<td>5</td>
<td>6,800</td>
<td>0.005 (0.000, 0.011)</td>
<td>0.01 (0.00, 0.03)</td>
<td>0.05</td>
<td>0.11 (47.5)</td>
</tr>
<tr>
<td>Higher odds of type 2 Diabetes (excluding pre-existing and gestational diabetes)</td>
<td>6</td>
<td>11,653</td>
<td>0.002 (-0.001, 0.006)</td>
<td>0.01 (0.00, 0.02)</td>
<td>0.18</td>
<td>0.32 (14.9)</td>
<td>4</td>
<td>5,330</td>
<td>0.008 (0.002, 0.014)</td>
<td>0.02 (0.01, 0.04)</td>
<td>0.01</td>
<td>0.38 (2.9)</td>
</tr>
<tr>
<td>Higher triglycerides</td>
<td>9</td>
<td>17,440</td>
<td>0.000 (-0.006, 0.007)</td>
<td>0.00 (-0.02, 0.02)</td>
<td>0.89</td>
<td>0.11 (39.0)</td>
<td>6</td>
<td>9,335</td>
<td>-0.004 (-0.014, 0.005)</td>
<td>-0.01 (-0.04, 0.01)</td>
<td>0.41</td>
<td>0.07 (51.3)</td>
</tr>
<tr>
<td>Lower HDL-cholesterol</td>
<td>9</td>
<td>15,573</td>
<td>0.005 (-0.004, 0.014)</td>
<td>0.01 (-0.01, 0.04)</td>
<td>0.27</td>
<td>0.45 (0)</td>
<td>6</td>
<td>8,207</td>
<td>-0.001 (-0.013, 0.012)</td>
<td>0.00 (-0.04, 0.03)</td>
<td>0.92</td>
<td>0.13 (41.4)</td>
</tr>
<tr>
<td>Higher systolic blood pressure</td>
<td>7</td>
<td>13,527</td>
<td>-0.005 (-0.010, -0.001)</td>
<td>-0.01 (-0.03, 0.00)</td>
<td>0.03</td>
<td>0.74 (0)</td>
<td>5</td>
<td>6,821</td>
<td>-0.003 (-0.011, 0.004)</td>
<td>-0.01 (-0.03, 0.01)</td>
<td>0.43</td>
<td>0.14 (42.3)</td>
</tr>
<tr>
<td>Higher systolic blood pressure (excluding pre-eclampsia and hypertension)</td>
<td>6</td>
<td>10,770</td>
<td>-0.005 (-0.010, -0.000)</td>
<td>-0.01 (-0.03, 0.00)</td>
<td>0.07</td>
<td>0.60 (0)</td>
<td>4</td>
<td>4,735</td>
<td>-0.005 (-0.014, 0.004)</td>
<td>-0.01 (-0.04, 0.01)</td>
<td>0.25</td>
<td>0.28 (22.2)</td>
</tr>
<tr>
<td>Lower vitamin D status</td>
<td>7</td>
<td>14,004</td>
<td>-0.007 (-0.025, -0.011)</td>
<td>-0.02 (-0.07, 0.03)</td>
<td>0.44</td>
<td>0.22 (27.9)</td>
<td>3</td>
<td>7,292</td>
<td>-0.026 (-0.054, 0.002)</td>
<td>-0.07 (-0.15, 0.01)</td>
<td>0.07</td>
<td>0.04 (68.7)</td>
</tr>
<tr>
<td>Lower adiponectin</td>
<td>6</td>
<td>11,501</td>
<td>0.017 (-0.020, 0.054)</td>
<td>0.05 (-0.06, 0.15)</td>
<td>0.37</td>
<td>0.83 (0)</td>
<td>5</td>
<td>6,851</td>
<td>0.039 (-0.016, 0.094)</td>
<td>0.11 (-0.04, 0.26)</td>
<td>0.17</td>
<td>0.89 (0)</td>
</tr>
</tbody>
</table>

The decision to model the association in relation to the trait-raising or trait-lowering allele depended on the known direction of association of each trait with higher BMI (see Box 1). Column 1 specifies each of these directions of association. Results are per average weighted allele, adjusted for sex and gestational age. "Standard deviation of ponderal index from ALSPAC study was used for these estimates (2.78 kg/m²). "Results are per average weighted allele, adjusted for sex, gestational age and fetal genotype."
<table>
<thead>
<tr>
<th>Maternal trait (value of 1 SD with units)</th>
<th>Study/ies(^a) used for observational estimates [Total N women]</th>
<th>N women</th>
<th>Observational estimate of the change in ponderal index (kg/m(^3)) per 1 SD (or 10% (^b)) change in maternal trait, adjusted for sex and gestational age (95%CI)</th>
<th>Genetic estimate of the change in ponderal index (kg/m(^3)), adjusted for sex and gestational age, per 1 SD (or 10% (^b)) change in maternal trait, unadjusted for fetal genotype (95%CI)</th>
<th>P value (^c) comparing observational with genetic ponderal index associations (unadjusted for fetal genotype)</th>
<th>Genetic estimate of the change in ponderal index (kg/m(^3)), adjusted for sex, gestational age and fetal genotype, per 1 SD (or 10% (^b)) change in maternal trait (95%CI)</th>
<th>P value (^c) comparing observational with genetic ponderal index associations (adjusted for fetal genotype)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher prepregnancy BMI (4 kg/m(^2))</td>
<td>ALSPAC Mothers, EFSOCH Mothers, HAPO Mothers</td>
<td>9,690</td>
<td>0.24 (0.19, 0.29)</td>
<td>0.45 (0.14, 0.75)</td>
<td>0.20</td>
<td>0.47 (0.14, 0.79)</td>
<td>0.21</td>
</tr>
<tr>
<td>Higher fasting glucose (0.4 mmol/L)</td>
<td>EFSOCH Mothers, HAPO Mothers</td>
<td>4,917</td>
<td>0.31 (0.22, 0.39)</td>
<td>0.27 (0.05, 0.48)</td>
<td>0.72</td>
<td>0.53 (0.20, 0.87)</td>
<td>0.24</td>
</tr>
<tr>
<td>Higher triglycerides (0.7 mmol/L)</td>
<td>EFSOCH Mothers</td>
<td>857</td>
<td>0.15 (-0.03, 0.33)</td>
<td>0.02 (-0.21, 0.24)</td>
<td>0.35</td>
<td>-0.14 (-0.48, 0.20)</td>
<td>0.14</td>
</tr>
<tr>
<td>Lower HDL-cholesterol (0.5 mmol/L)</td>
<td>EFSOCH Mothers</td>
<td>854</td>
<td>0.12 (-0.08, 0.31)</td>
<td>0.14 (-0.11, 0.39)</td>
<td>0.91</td>
<td>-0.02 (-0.37, 0.34)</td>
<td>0.51</td>
</tr>
<tr>
<td>Higher Systolic blood pressure (10 mmHg)</td>
<td>ALSPAC Mothers, HAPO Mothers</td>
<td>9,691</td>
<td>0.00 (-0.08, 0.06)</td>
<td>-0.77 (-1.80, 0.25)</td>
<td>0.16</td>
<td>-0.46 (-1.95, 1.03)</td>
<td>0.56</td>
</tr>
<tr>
<td>Lower vitamin D status (10%)(^d)</td>
<td>ALSPAC Mothers</td>
<td>3,718</td>
<td>-0.02 (-0.03, 0.00)</td>
<td>-0.08 (-0.28, 0.13)</td>
<td>0.56</td>
<td>-0.29 (-0.65, 0.07)</td>
<td>0.14</td>
</tr>
<tr>
<td>Lower adiponectin (10%)(^d)</td>
<td>HAPO Mothers (GWAS only)</td>
<td>1,373</td>
<td>0.05 (0.02, 0.08)</td>
<td>0.03 (-0.03, 0.09)</td>
<td>0.49</td>
<td>0.06 (-0.03, 0.15)</td>
<td>0.82</td>
</tr>
</tbody>
</table>

\(^a\)Heterogeneity statistics from the meta-analyses of observational associations were: Phet = 0.35 and I\(^2\) = 9.1% for BMI; Phet = 0.23 and I\(^2\) = 32.7% for fasting glucose; Phet = 0.67 and I\(^2\) = 0% for SBP.

\(^b\)For 25\([OH]\)D and adiponectin, we present the estimated change in ponderal index per 10% reduction in maternal trait level because these variables were logged for analysis.

\(^c\)P-values <0.05 are considered to indicate evidence that the genetic effect size estimate is different from the observational estimate, suggesting that the observational estimate is subject to confounding or bias.
### (a) Observational associations between offspring birth weight and maternal socio-economic status or maternal smoking in the ALSPAC study

<table>
<thead>
<tr>
<th>Maternal trait</th>
<th>N women</th>
<th>Change in birth weight (g) per unit change in maternal trait (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest educational qualification attained(^a)</td>
<td>6,855</td>
<td>19 (10, 29)</td>
<td>0.00004</td>
</tr>
<tr>
<td>Occupational position(^b)</td>
<td>5,588</td>
<td>-34 (-68, 0)</td>
<td>0.06</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>7,021</td>
<td>-208 (-237, -179)</td>
<td>1x10^-43</td>
</tr>
</tbody>
</table>

\(^a\) Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

\(^b\) Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.

### (b) Observational associations between maternal BMI and maternal socio-economic status or maternal smoking in the ALSPAC study

<table>
<thead>
<tr>
<th>Maternal trait</th>
<th>N women</th>
<th>Change in BMI (kgm-2) per unit change in maternal trait (95%CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest educational qualification attained(^a)</td>
<td>6,115</td>
<td>-0.35 (-0.42, -0.27)</td>
<td>9x10^-20</td>
</tr>
<tr>
<td>Occupational position(^b)</td>
<td>5,128</td>
<td>0.38 (0.11, 0.65)</td>
<td>0.005</td>
</tr>
<tr>
<td>Smoking (current smoker vs non-smoker)</td>
<td>6,238</td>
<td>0.01 (-0.24, 0.26)</td>
<td>0.95</td>
</tr>
</tbody>
</table>

\(^a\) Subjects grouped as: 1=CSE; 2=Vocational; 3=Ordinary Level; 4=Advanced level; 5=Degree

\(^b\) Derived from Office of Population Censuses & Surveys Standard Occupational Classification (1). Subjects dichotomized as: 0=I, II & III (non-manual); 1=III (manual), IV & V.
**eTable 10. Power calculations**

<table>
<thead>
<tr>
<th>Maternal trait</th>
<th>Total N women</th>
<th>Adjusted-(R^2) from BW~GA sex</th>
<th>Adjusted-(R^2) from BW~GA sex GS</th>
<th>Estimated proportion of variance in birth weight explained by maternal genetic score</th>
<th>Power available in our included sample to detect evidence of association between maternal genetic score and birth weight at (P&lt;0.05)</th>
<th>Minimum sample size needed to detect association between maternal genetic score and birth weight at (P&lt;0.05) with 80% power</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>25265</td>
<td>0.1308</td>
<td>0.1312</td>
<td>0.0004</td>
<td>0.89</td>
<td>19618</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>23902</td>
<td>0.1308</td>
<td>0.1319</td>
<td>0.0011</td>
<td>1.00</td>
<td>7131</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>18670</td>
<td>0.1308</td>
<td>0.1312</td>
<td>0.0004</td>
<td>0.78</td>
<td>19618</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>24985</td>
<td>0.1308</td>
<td>0.1307</td>
<td>-0.0001*</td>
<td>0.35</td>
<td>78485</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>22167</td>
<td>0.1308</td>
<td>0.1307</td>
<td>-0.0001*</td>
<td>0.32</td>
<td>78485</td>
</tr>
<tr>
<td>Systolic Blood Pressure</td>
<td>20062</td>
<td>0.1308</td>
<td>0.1324</td>
<td>0.0016</td>
<td>1.00</td>
<td>4902</td>
</tr>
<tr>
<td>25-hydroxy vitamin D</td>
<td>30340</td>
<td>0.1308</td>
<td>0.1309</td>
<td>0.0001</td>
<td>0.41</td>
<td>78485</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>14920</td>
<td>0.1308</td>
<td>0.1307</td>
<td>-0.0001*</td>
<td>0.23</td>
<td>78485</td>
</tr>
</tbody>
</table>

*Where the estimated variance explained was negative, we assumed a value of 0.0001 for the calculations. BW, birth weight; GA, gestational age; GS, genetic score.*
**eTable 1. Association between father’s phenotypes and offspring birth weight using data from the ALSPAC study**

<table>
<thead>
<tr>
<th>Father’s phenotype</th>
<th>N men</th>
<th>Correlation coefficient (95% CI) of father’s phenotype with offspring birth weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI*</td>
<td>7491</td>
<td>0.04 (0.02, 0.06)</td>
</tr>
<tr>
<td>BMI</td>
<td>1721</td>
<td>0.03 (-0.02, 0.07)</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>1732</td>
<td>-0.03 (-0.07, 0.01)</td>
</tr>
<tr>
<td>Glucose</td>
<td>1656</td>
<td>-0.01 (-0.06, 0.03)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1656</td>
<td>-0.02 (-0.07, 0.02)</td>
</tr>
<tr>
<td>HDLc</td>
<td>1656</td>
<td>0.02 (-0.03, 0.06)</td>
</tr>
</tbody>
</table>

* Based on paternal report of weight and height at the time that their partner was in early pregnancy; all other phenotypes were assessed at a clinic visit ~18-19 years after the child’s birth. Correlation coefficients of paternal phenotypes with offspring birth weight were all weak and mostly null (Pearson correlation coefficients all ≤ 0.04). The correlation between and offspring birth weight and father’s BMI, assessed when the mothers were pregnant, was similar to that between offspring birth weight and father’s BMI 18 years later, suggesting that the postnatal measures for other phenotypes are a reasonable approximation for them before/at the time of their partner’s pregnancy.
References


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