



## Original Contribution

# Epidemiologic Evidence for the Fetal Overnutrition Hypothesis: Findings from the Mater-University Study of Pregnancy and Its Outcomes

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The fetal overnutrition hypothesis proposes that greater maternal adiposity results in increased obesity throughout life in the offspring. The authors examined the associations between parental prepregnancy body mass index (BMI; weight (kg)/height (m)<sup>2</sup>), based on height and weight reported by the mother at her first antenatal clinic visit, and offspring BMI (height and weight measured at age 14 years) in 3,340 parent-offspring trios from a birth cohort based in Brisbane, Australia (mothers were recruited in 1981–1984). The maternal-offspring BMI association was stronger than the paternal-offspring BMI association. In the fully adjusted model, the increase in standardized offspring BMI at age 14 for a one-standard-deviation (SD) increase in maternal BMI was 0.362 SD (95% confidence interval: 0.323, 0.402), and the corresponding result for a one-SD increase in paternal BMI was 0.239 SD (95% confidence interval: 0.197, 0.282). There was statistical support for a difference in the magnitude of the association between maternal-offspring BMI and paternal-offspring BMI in all confounder-adjusted models tested (all *p*'s < 0.0001). In sensitivity analyses taking account of different plausible levels of nonpaternity (up to 15%), the greater maternal effect remained. These findings provide some support for the fetal overnutrition hypothesis.

body mass index; fetal development; fetal nutrition disorders

Abbreviations: BMI, body mass index; CI, confidence interval.

In addition to the influence of dietary factors and physical activity on overweight and obesity, it is increasingly being recognized that prenatal factors influence childhood and adulthood body mass index (BMI; weight (kg)/height (m)<sup>2</sup>) and obesity levels (1–6). It has been suggested that intra-uterine overnutrition affects lifelong risk of obesity (4–8). According to this hypothesis, high maternal plasma concentrations of glucose, free fatty acids, and amino acids result in permanent changes in appetite control, neuroendocrine functioning, or energy metabolism in the developing fetus and thus lead to obesity in later life. Since maternal BMI is positively associated with insulin resis-

tance and glucose intolerance, and therefore higher plasma concentrations of glucose and free fatty acids, fetal overnutrition is more likely among mothers with greater BMI during pregnancy (4–8). If this hypothesis is true, the consequences are important: “The obesity epidemic could accelerate through successive generations independent of further genetic or environmental factors” (1, p. 475).

Support for the hypothesis that greater maternal BMI during pregnancy can result in greater obesity in the offspring later in life comes from a number of lines of evidence. In vitro animal and human studies have demonstrated that fetal pancreas development and fat stores are influenced by the

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availability of fetal fuels—particularly glucose, lipids, and amino acids—which are in turn determined by maternal obesity and plasma levels of glucose and free fatty acids (7). The offspring of female rats with diet-induced obesity during pregnancy have been found to be heavier than the offspring of rats with the same genotype but without the diet-induced maternal obesity (9). However, whether these findings are relevant to human populations is unclear. In humans, a number of epidemiologic studies have found positive associations between maternal BMI in pregnancy and offspring obesity or BMI in later life (10–14), and some (15–17), though not all (13, 18), studies have found that greater weight gain in pregnancy is related to greater offspring obesity or BMI in later life. The relation between maternal BMI and offspring BMI is not necessarily due to an intrauterine effect; it may reflect transmission of genetic factors between mother and offspring or shared socioeconomic and lifestyle factors between a mother and her offspring.

One way to further explore whether the association between maternal BMI during pregnancy and offspring BMI is specifically due to an intrauterine effect is to compare the magnitude of the maternal-offspring association with that of the paternal-offspring association. Similar magnitudes of effect of maternal and paternal BMI on offspring BMI would suggest that the associations were driven by factors (genetics, family socioeconomic position, shared lifestyle factors) that are just as likely to be passed from father to offspring as they are from mother to offspring. By contrast, a stronger maternal-offspring BMI association compared with the paternal-offspring BMI association would support a specific maternal effect; intrauterine effects would be a likely explanation.

Some investigators have examined associations between both maternal and offspring BMI and paternal and offspring BMI, but such studies have found inconsistent results and have had important limitations (19–31). In some studies, there does appear to be a stronger maternal effect. For example, in a study of nearly 9,000 Japanese children, the odds ratio for obesity at age 3 years in relation to maternal obesity was 2.56 (95 percent confidence interval (CI): 2.07, 3.17) and the odds ratio related to paternal obesity was 1.70 (95 percent CI: 1.43, 2.02) (29). Similarly, in a study of children aged 6–7 years, the odds ratio for the effect of maternal obesity was 5.07 (95 percent CI: 2.62, 9.79) and that for the effect of paternal obesity was 2.66 (95 percent CI: 1.51, 4.70) (30). Other studies have found stronger effects for fathers (23) or no difference in parental effects (19, 20). However, rarely have researchers presented statistical evidence for a difference in parental effects, and many previous studies have been small and unlikely to have sufficient power to detect important differences in effect between mothers and fathers. To our knowledge, no previous study comparing parental-offspring BMI associations has undertaken sensitivity analyses to account for the potential effect of nonpaternity in explaining any stronger maternal effect.

Finally, if the fetal overnutrition hypothesis is concerned with a specific effect of maternal BMI during pregnancy, the correct test of that hypothesis is a comparison of the effect of maternal BMI immediately pre-pregnancy on later offspring BMI with the effect of paternal BMI, obtained at

the same time as maternal BMI, on later offspring BMI. Most studies carried out to date have, in fact, examined parental-offspring associations with all three measurements taken at the same time (when the offspring was in childhood, adolescence, or early adulthood) (19–24, 28–31). The environment shared by all three family members from birth to this time of assessment may well mask any specific intrauterine effect.

Our aim in this study was to determine whether there is a difference in the magnitudes of the associations between maternal BMI (reported in early pregnancy) and offspring BMI and paternal BMI (also reported in early pregnancy) and offspring BMI.

## MATERIALS AND METHODS

### Participants

The Mater-University Study of Pregnancy and Its Outcomes is a prospective study of women (and their offspring) who received antenatal care at a major public hospital (Mater Misericordiae Hospital) in South Brisbane, Australia, between 1981 and 1984 (32). The cohort consists of 7,223 women (and their offspring) who delivered a live singleton baby that neither died nor was adopted prior to leaving the hospital and who completed both initial phases of data collection. These mothers and children have been followed prospectively, with mothers completing questionnaires at their first antenatal clinic visit, 3–5 days after birth, 6 months after birth, 5 years after birth, and 14 years after birth.

### Measurements

In this study, the main outcome in all analyses was the child's BMI, derived from measurements of weight and height taken at the 14-year follow-up. We also examined associations with offspring BMI derived from measurements of weight and height taken at the 5-year follow-up. For both ages, the average of two measurements of the child's weight, taken with a scale accurate to 0.2 kg while the child was lightly clothed, was used in all analyses. Height was measured using a portable stadiometer that was accurate to 1 mm.

Maternal height and weight at the first antenatal visit and the mother's estimate of her own prepregnancy weight and paternal height and weight were obtained at study initiation from obstetric records or maternal questionnaires. There was a high level of correlation between maternal estimates of prepregnancy weight and measured weight at the first antenatal visit (Pearson's correlation coefficient = 0.95). In this study, maternal BMI was calculated from the mother's self-report of prepregnancy weight and her height at the first clinic visit. Paternal BMI was calculated from the mother's report of the child's father's height and weight as reported at the first clinic visit.

Birth weight and length were measured at the time of birth, and gestational age (completed weeks) was obtained from the obstetric records. Information on maternal age at birth, parity (categorized as 0, 1, 2, 3, 4, and  $\geq 5$ ), smoking around the time of pregnancy (pregnancy and/or in early pregnancy only, throughout pregnancy, never), and maternal

and paternal educational attainment (did not complete secondary school, completed secondary school, completed further/higher education) was obtained from obstetric records and questionnaires administered at study initiation. At the start of the study, the mothers were asked about gross family income during the year of their pregnancy (Aus\$: 0–2,599; 2,600–5,199; 5,200–10,399; 10,400–15,599; 15,600–20,799; 20,800–25,999; and  $\geq 26,000$ ). This information was collapsed into three categories: low income ( $< \$10,400$ ), middle income ( $\$10,400$ – $\$15,599$ ), and high income ( $\geq \$15,600$ ). At the 14-year follow-up, the children were asked how often they ate fast food (three response options: daily, 2–3 times per week, and once per week or less) and how often they undertook exercise that made them sweat (four response options: never, once per week, twice per week, and three or more times per week).

### Statistical analyses

**Dealing with missing data.** Of the original 7,223 cohort participants, 3,795 (53 percent) attended the physical examination at age 14 years and had adequate height and weight measurements from which BMI could be calculated. Those with data on BMI at age 14 were less likely to be from low-income families, had more highly educated parents, and had lower birth weights, and the mean age of their mothers at their birth was older than that of participants without these data (all  $p$ 's  $< 0.05$ ). Maternal BMI (22.1 kg/m<sup>2</sup> vs. 22.0 kg/m<sup>2</sup>) and paternal BMI (23.7 kg/m<sup>2</sup> vs. 23.6 kg/m<sup>2</sup>) did not differ between subjects who had BMI data at age 14 years and those who did not have these data (either because they were lost to follow-up or did not complete this part of the examination adequately). Of the 3,795 offspring with BMI data, 3,748 (99 percent) had information on maternal BMI and 3,360 (89 percent) had information on paternal BMI; 3,340 (88 percent) had BMI data on both parents. The associations between maternal and offspring BMI and those between paternal and offspring BMI were identical whether maximal data sets were used or the analyses were conducted only in the 3,340 persons with data on all three family members.

For the main analyses presented here, we used only these 3,340 persons. However, we undertook sensitivity analyses using inverse probability (of having missing data) weights to determine whether our results were biased by missing data (33). The probability weights were computed from a logistic regression model with the outcome being “has complete data.” The fitted values from this logistic regression model estimate the probability of having missing data, and the weights used in the regression models of interest are the inverses of these values (33). We compared the results from these weighted analyses with those from the main (unweighted) analyses. In further sensitivity analyses, we used multivariate multiple imputation to deal with all missing data. We used switching regression in Stata, as described by Royston (34); we carried out 20 cycles of regression switching and generated 10 imputation data sets. The results from the inverse probability weighting and the multivariate multiple-imputation models did not differ from the results

presented here. The findings from the multivariate multiple-imputation models were more precisely estimated.

**Comparing maternal-offspring BMI associations with paternal-offspring BMI associations.** In order to take account of the difference in the range of BMIs between mothers and fathers, as well as differences between different genders and ages, in both the parents and the children we derived age- and sex-internally-standardized BMI  $z$  scores for the offspring and age-internally-standardized BMI  $z$  scores for the mothers and fathers. For the offspring, the age standardization was done using 6-month age intervals; for both parents, it was done using 1-year age intervals. Multiple linear regression was used to assess the associations of parental BMI with offspring birth weight and BMI at ages 5 and 14 years, with adjustment for potentially confounding factors. We computed an  $f$  statistic to formally compare the adjusted (for each other as well as other confounders) coefficients of maternal and paternal associations for each model. It is important in these models that we compared the maternal coefficient adjusted for paternal effect (and vice versa), because maternal and paternal BMI are correlated with each other (Pearson's correlation coefficient = 0.15,  $p < 0.001$ ); if we compared the unadjusted coefficients, they might appear the same simply because of this correlation.

To examine the potential role of nonpaternity in generating greater associations between maternal and offspring BMI than between paternal and offspring BMI, given the nonbiologic relationship between some fathers and their apparent offspring, we conducted a sensitivity analysis modeling the effects of nonpaternity rates of 1–15 percent, using the equation given in the Appendix (35). All analyses were conducted using Stata, version 9.0 (Stata Corporation, College Station, Texas).

### RESULTS

Mean prepregnancy maternal BMI was 22.0 kg/m<sup>2</sup> (standard deviation, 4.1); mean paternal BMI based on height and weight reported at the same time was 23.7 kg/m<sup>2</sup> (standard deviation, 4.7). Mean offspring BMI assessed at the 14-year follow-up was 20.3 kg/m<sup>2</sup> (standard deviation, 3.6) for boys and 21.0 kg/m<sup>2</sup> (standard deviation, 4.0) for girls. The pairwise Pearson correlations between family BMIs were 0.15 for the two parents, 0.33 for maternal-offspring BMI, and 0.23 for paternal-offspring BMI.

The linear regression associations of parental BMI with offspring birth size or BMI at either age 5 or age 14 were the same regardless of whether offspring were females or males ( $p$  values for interactions with sex of offspring were greater than 0.7 for both parents and in all of the regression models). Therefore, all results are presented for male and female offspring combined. Table 1 shows the associations of parental BMI with offspring birth size. Maternal prepregnancy BMI was positively associated with birth weight and length irrespective of whether absolute size or sex- and gestational-age-standardized weight and length were used in these analyses. By comparison, paternal prepregnancy BMI was not associated with birth size, with the exception of a weak and borderline statistically significant positive

**TABLE 1. Associations of prepregnancy maternal and paternal body mass index\* with birth size among 3,340 parents and offspring from the Mater-University Study of Pregnancy and Its Outcomes, Brisbane, Australia, 1981–1984**

Birth size outcome	Regression of offspring birth size on maternal BMI† z score		Regression of offspring birth size on paternal BMI z score		p value for difference between effects of maternal and paternal BMI on offspring birth size
	β	95% CI†	β	95% CI	
Birth weight (g)‡	81.7	69.0, 94.4	5.9	−7.2, 19.0	<0.0001
Birth length (cm)‡	0.30	0.21, 0.39	−0.02	−0.11, 0.07	<0.0001
Birth weight for sex and gestational age (z score)	0.17	0.15, 0.20	0.03	0.00, 0.05	<0.0001
Birth length for sex and gestational age (z score)	0.10	0.07, 0.13	0.00	−0.03, 0.03	<0.0001

\* Weight (kg)/height (m)<sup>2</sup>.

† BMI, body mass index; CI, confidence interval.

‡ These β coefficients were adjusted for the sex of the child.

association with birth weight standardized for sex and gestational age. For all birth size outcomes, there was strong statistical evidence that the maternal BMI associations differed from the paternal BMI associations (all *p*'s < 0.0001).

Table 2 shows the adjusted associations of parental BMI with offspring BMI at age 14 years. The magnitude of the association was greater for maternal-offspring associations than for paternal-offspring associations in all confounder-adjusted models (all *p* values for a sex difference were less than 0.0001). When we further adjusted for birth weight and length, the maternal effect was attenuated slightly and the paternal effect became slightly stronger. However, statistical evidence of a stronger maternal effect remained. Additional adjustment of these results for the child's report of frequency of eating fast food and exercising at age 14 did not alter any of these associations (data not shown). Similarly, adjustment for maternal exercise during pregnancy and family diet at age 14 did not affect the results (data not shown).

Table 3 shows the results of a series of sensitivity analyses assuming levels of nonpaternity of 1–15 percent. There was no evidence that taking account of possible nonpaternity at

levels of up to 15 percent had any important effect on the stronger maternal-offspring association compared with the paternal-offspring association. Indeed, the paternal-offspring BMI association approached the magnitude of the maternal-offspring BMI association only at assumed nonpaternity levels of 30 percent.

Findings for BMI at age 5 years were similar to those presented for BMI at age 14 years. When we repeated our analyses using absolute BMI for both parents and their offspring, as opposed to age- and sex-standardized *z* scores, the results confirmed a stronger maternal-offspring association than paternal-offspring association in all models (all *p*'s < 0.0001). Fifty-one women had diabetes (either preexisting or gestational); when the analyses were repeated with these women removed, the results were not changed.

## DISCUSSION

The fetal overnutrition hypothesis suggests that greater maternal BMI during pregnancy will result in greater obesity

**TABLE 2. Associations of prepregnancy maternal and paternal body mass index\* with offspring body mass index among 3,340 parents and offspring from the Mater-University Study of Pregnancy and Its Outcomes, Brisbane, Australia, 1981–1984**

Adjustment factors	Regression of offspring BMI† z score on maternal BMI z score		Regression of offspring BMI z score on paternal BMI z score		p value for difference between effects of maternal and paternal BMI on offspring BMI
	β	95% CI†	β	95% CI	
Adjusted for sex and the other parent's BMI	0.363	0.324, 0.403	0.241	0.199, 0.283	<0.0001
Plus family income, parental education, and maternal age at birth	0.360	0.320, 0.401	0.243	0.200, 0.286	<0.0001
Plus parity	0.360	0.320, 0.400	0.242	0.199, 0.285	<0.0001
Plus maternal smoking around the time of pregnancy	0.362	0.323, 0.402	0.239	0.197, 0.282	<0.0001
Plus birth weight and length (sex- and gestational-age-standardized z scores)	0.353	0.304, 0.401	0.251	0.199, 0.304	0.009

\* Weight (kg)/height (m)<sup>2</sup>.

† BMI, body mass index; CI, confidence interval.

**TABLE 3. Adjusted  $\beta$  coefficients\* for the regression of offspring body mass index† z score on parental body mass index z score with mutual adjustment for the other parent's BMI, assuming various proportions of nonpaternity, among 3,340 parents and offspring from the Mater-University Study of Pregnancy and Its Outcomes, Brisbane, Australia, 1981–1984**

Assumed rate of nonpaternity (%)	Fully adjusted coefficient ( $\beta$ ) for regression of offspring BMI‡ on:		<i>p</i> for difference between parental effects
	Maternal BMI	Paternal BMI	
0	0.363	0.241	<0.0001
1	0.363	0.244	<0.0001
2	0.362	0.247	<0.0001
3	0.362	0.250	<0.0001
4	0.361	0.253	<0.0001
5	0.361	0.257	0.0001
6	0.360	0.260	0.0002
7	0.360	0.263	0.0003
8	0.359	0.267	0.0006
9	0.359	0.270	0.0008
10	0.358	0.274	0.0015
11	0.358	0.278	0.0016
12	0.357	0.284	0.0021
13	0.356	0.287	0.0049
14	0.356	0.290	0.0091
15	0.355	0.294	0.0153

\* Coefficients were calculated using the formula presented in the Appendix.

† Weight (kg)/height (m)<sup>2</sup>.

‡ BMI, body mass index.

in the offspring in later life (1, 4–8). If this is so, it has potentially important consequences, since the more obese female offspring would go on to produce more obese offspring of their own, and the obesity epidemic would be programmed throughout subsequent generations (1). The finding of a greater maternal-offspring BMI association than paternal-offspring BMI association presented here provides some support for this hypothesis.

We interpret our greater maternal effect on offspring BMI as reflecting an intrauterine effect, but our findings suggest that this does not operate through an effect on offspring birth size. It has been suggested that the greater delivery of maternal fuels to the developing fetus may result in permanent changes in appetite control, neuroendocrine functioning, or energy metabolism in the developing fetus and that such programming effects may result in a greater risk of later obesity (5). This concept of fetal overnutrition does not necessitate a mechanism that involves increased birth size. Adjustment for offspring consumption of fast food and frequency of exercise at age 14 in our study did not alter the associations, but these measures are likely to be too crude to capture neuroendocrine pathways, energy metabolism, and appetite control, and more detailed physiologic studies are required to establish the exact mechanisms underlying a greater maternal effect on offspring BMI. The greater association

with maternal BMI as compared with paternal BMI may also be explained by epigenetic effects. Again this mechanism would not require an effect on fetal size.

The greater association between mothers and their offspring could reflect the greater role of mothers in childhood nutrition and feeding habits, rather than an intrauterine effect. While this is a possibility, surprisingly few studies have examined differences in maternal-offspring and paternal-offspring behavior. In a study using data from the Norwegian National Health Survey, both maternal and paternal dietary fat intakes were strongly associated with offspring dietary fat intake, but the magnitude of association was the same for mothers and fathers (36). By contrast, mothers' level of exercise had a much weaker effect on offspring's exercise levels (whatever the sex of the offspring) than did fathers' exercise levels in that study (36). Similarly, in a recent study of Australian families, fathers' exercise levels had a stronger effect on both sons' and daughters' exercise levels and objective measures of cardiorespiratory fitness than did mothers' exercise levels (37). Thus, there does not appear to be strong evidence in the literature that mothers have a stronger effect on offspring diet and physical activity (behaviors that would affect offspring BMI) than do fathers.

### Study limitations

In this study, the participation rate at age 14 years was just over 50 percent, and children who did not attend the year 14 examination were more likely to be from poorer backgrounds and to have younger mothers and mothers who smoked throughout pregnancy. Parental BMIs did not differ between subjects with and without BMI data at age 14, and sensitivity analyses using weighted regression or multivariate imputation suggested that our findings were not biased by missing data. Maternal prepregnancy weight and paternal height and weight were all reported by the mothers and may have been inaccurate. The correlation between maternal reports of prepregnancy weight and actual weights at the first antenatal visit was very high (Pearson's correlation coefficient = 0.95). However, it is possible that greater misclassification for paternal reports could have contributed to the weaker effect in fathers as compared with mothers. The distribution of paternal BMIs in this study was similar to that reported for men of a similar age in a national Australian survey conducted in the 1980s (the time at which mothers reported heights and weights for fathers in the present study) in which heights and weights were measured (38), and the maternal-paternal correlation for BMI in this study was similar to that reported for spouses in studies with measured height and weight (39).

A recent analysis of the Avon Longitudinal Study of Parents and Children that undertook an analytical approach similar to the one used here and included comparable numbers of family trios also found a greater maternal-offspring BMI association compared with the paternal-offspring BMI association (35). However, in that study, the difference between the two effects was smaller than in our study and may have been explained by levels of nonpaternity of 10 percent. When absolute BMI measures rather than standard

measures were used, there was no difference between mothers and fathers in their effects on offspring BMI. In that study, mothers reported their own heights and weights at the start of pregnancy, but unlike in our study, fathers also reported their own heights and weights, and it is possible that the somewhat greater difference in our study reflects some weakening of the paternal effect due to misclassification bias. However, correlations between maternal and paternal BMI in the two studies were the same, and correlations between paternal BMI and offspring birth weight were the same in both studies.

There are no reliable estimates of nonpaternity rates in Western populations; rates between 2 percent and 15 percent have been quoted (40). In our study, the strength of the paternal-offspring associations did not reach that of maternal-offspring associations until nonpaternity levels of 30 percent, which seem implausible, were assumed in sensitivity analyses.

While BMI is easy to assess and has been shown to be related to a range of adverse vascular and metabolic outcomes in both children and adults, it cannot differentiate between lean mass and fat mass (41). A recent review of the developmental origins of obesity called for future research to formally assess associations with fat mass and fat distribution (4). Such measures are currently not available in the Mater-University Study of Pregnancy and Its Outcomes and to our knowledge have not been used in any previous studies of the association of maternal BMI or weight change during pregnancy, or paternal BMI, with later offspring size. It will be important in future studies to determine whether there are differences between maternal and paternal size and offspring body composition. If maternal BMI is more strongly associated with offspring fat mass than paternal BMI and the absolute differences in associations with fat mass are large, this would have considerable public health importance.

### Implications

There is currently an epidemic of obesity in Western societies. The potential importance of the suggestion, from our study, that greater maternal size during pregnancy, either through programming of neuroendocrine pathways or through epigenetic or other mechanisms, results in greater offspring BMI in later life means that this issue warrants further investigation. Evidence from a randomized controlled trial would provide the strongest evidence of effects of maternal obesity and glucose and lipid levels on offspring obesity, but clearly ethical and practical considerations would make designing such a study difficult. However, the principle of Mendelian randomization could be used to design a study that would, in effect, be a natural randomized controlled trial (42–44). Studies of the association of common maternal genetic variants known to affect maternal adiposity and glucose and lipid levels (believed to be important in fetal overnutrition) with offspring adiposity, while accounting for the offspring's genotype, would provide an unbiased and unconfounded means of examining the fetal overnutrition hypothesis. Such a study, however, would require the demonstration of common genetic variants that

have replicated (in several studies) associations with the maternal intermediate phenotypes and that are known not to have pleiotropic effects (43).

### Conclusion

We have found some epidemiologic evidence in favor of the suggestion that maternal size during pregnancy has an effect on offspring BMI over and above that of shared familial or other mechanisms that would result in similar associations of paternal BMI with offspring BMI. The potential importance of this hypothesis means that it warrants further investigation.

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## APPENDIX

Formulae used for sensitivity analysis of nonpaternity were taken from the paper by Davey Smith et al. (35):

$$\begin{pmatrix} (1-p)\sigma_{ff} & (1-p)\sigma_{fm} \\ \sigma_{fm} & \sigma_{mm} \end{pmatrix}^{-1} \begin{pmatrix} \sigma_{ff} & a\sigma_{fm} \\ a\sigma_{fm} & \sigma_{mm} \end{pmatrix},$$

where  $\sigma_{ff}$  is the variance of the reported father's body mass index (BMI),  $\sigma_{mm}$  is the variance of the mother's BMI,  $\sigma_{fm}$  is the covariance of reported father's and mother's BMIs,  $p$  is the probability that the reported father is not the biologic father, and  $a$  is used to indicate the possible covariances between the mother's and biologic father's BMIs; we assumed it to be equal to the covariance between the mother's and reported father's BMIs and used  $a = 1$ .