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Maternal BMI, Parity, and Pregnancy Weight Gain: Influences on Offspring Adiposity in Young Adulthood

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Context: The prevalence of obesity among women of childbearing age is increasing. Emerging evidence suggests that this has long-term adverse influences on offspring health.

Objective: The aim was to examine whether maternal body composition and gestational weight gain have persisting effects on offspring adiposity in early adulthood.

Design and Setting: The Motherwell birth cohort study was conducted in a general community in Scotland, United Kingdom.

Participants: We studied 276 men and women whose mothers' nutritional status had been characterized in pregnancy. Four-site skinfold thicknesses, waist circumference, and body mass index (BMI), were measured at age 30 yr; sex-adjusted percentage body fat and fat mass index were calculated.

Main Outcome Measure: Indices of offspring adiposity at age 30 yr were measured.

Results: Percentage body fat was greater in offspring of mothers with a higher BMI at the first antenatal visit (rising by 0.35%/kg/m²; $P < 0.001$) and in offspring whose mothers were primiparous (difference, 1.5% in primiparous vs. multiparous; $P = 0.03$). Higher offspring percentage body fat was also independently associated with higher pregnancy weight gain (7.4%/kg/wk; $P = 0.002$). There were similar significant associations of increased maternal BMI, greater pregnancy weight gain, and parity with greater offspring waist circumference, BMI, and fat mass index.

Conclusions: Adiposity in early adulthood is influenced by prenatal influences independently of current lifestyle factors. Maternal adiposity, greater gestational weight, and parity all impact on offspring adiposity. Strategies to reduce the impact of maternal obesity and greater pregnancy weight gain on offspring future health are required. (*J Clin Endocrinol Metab* 95: 5365–5369, 2010)

It is increasingly recognized that events in early life can increase the risk of greater adiposity and its metabolic sequelae in later life (1, 2). Thus, defining the role of developmental influences could lead to effective new interventions. Factors that could cause an intergenerational cycle of obesity

include the mother's body composition and pregnancy weight gain. These are of particular importance given the increasing prevalence of obesity in women of child-bearing age (2).

Emerging evidence suggests that maternal adiposity has long-term detrimental effects on offspring obesity risk.

Several studies have demonstrated a relationship between increased maternal body mass index (BMI) during pregnancy and increased BMI in the offspring (1). Maternal adiposity has also been associated with increased fat mass in neonates, children, and at age 24 yr (1). Excessive maternal pregnancy weight gain has been linked with higher BMI in the offspring (3, 4) and with greater childhood fat mass (5).

Few studies have examined whether maternal diet impacts on adult adiposity, although men whose mothers were exposed to the Dutch famine in midpregnancy had greater BMI in early adulthood (6). Trophoblast invasion of the maternal uterine spiral arteries results in greater uteroplacental blood flow and increased fetal nutrient supply in second and subsequent pregnancies than in first pregnancies (7); the Dutch famine study (6) and experimental work linking prenatal undernutrition to offspring outcomes led us to speculate that in primiparous pregnancies reduced fetal nutrient supply could increase offspring adiposity. We have previously reported findings from follow-up studies of men and women in Motherwell, Scotland, whose mothers' weight, height, and diet in pregnancy were recorded. Mothers who ate the most unbalanced diets had offspring with higher blood pressure (8) and heightened hypothalamic-pituitary-adrenal responses to a stress challenge (9). In the current study, we examined the relationship of the mother's parity, BMI, and pregnancy weight gain with adiposity in the offspring in early adulthood and performed exploratory analyses of the role of maternal diet during pregnancy.

Subjects and Methods

We have previously described a sample of 626 Caucasian men and women born in Motherwell Maternity Hospital during 1967–68, whose blood pressures were measured in early adulthood (8). Maternal height and parity were abstracted from the antenatal records, together with weight measurements from booking (median, 111.5 d) and the end of pregnancy (median, 269 d), allowing derivation of pregnancy weight gain (kilograms per week). Summaries of the mothers' dietary intake in "early" (≤ 20 wk) and "late" (> 20 wk) pregnancy were recorded by staff at the antenatal clinic (8). The baby's birth weight and gestation at birth were abstracted from the original obstetric records.

When the offspring were aged 27–30 yr, a total of 276 attended a local clinic for measurements of body composition. Height was measured with a portable stadiometer, and weight with calibrated digital scales. Four skinfold thicknesses (biceps, triceps, subscapular, and suprailiac regions) were measured in triplicate on the nondominant side using Harpenden calipers (between-observer variation at all four sites $< 10\%$). Fat mass was estimated from skinfold thickness measurements (10). Fat mass index (FMI) was calculated as fat mass (kilograms)/height (meters) (2). Subjects completed a questionnaire assessing their smoking habits, alcohol consumption, and frequency and level of physical activity, using questions adapted from the 1991 Health

Survey of England. The study was approved by the Research Ethics Committee of the Lanarkshire Health Board, and subjects gave informed written consent.

Statistical analysis

Skewed variables were transformed using logarithms to satisfy statistical assumptions of normality, and Pearson correlation was used to examine associations between offspring adiposity variables (percentage body fat, waist circumference, FMI, and BMI) and maternal and offspring characteristics. Multiple linear regression analyses were performed, adjusting for potential confounding variables. Data were analyzed using SPSS version 15 (SPSS Inc., Chicago, IL).

Results

Characteristics of the 276 subjects and their mothers are available as Supplemental Table 1, published on The Endocrine Society's Journals Online web site at <http://jcem.endojournals.org>. Women had greater percentage body fat and FMI than men (both $P < 0.001$), but men had higher BMI and waist circumference than women ($P = 0.01$ and $P < 0.001$, respectively).

Current lifestyle and adiposity

Taking account of gender, percentage body fat was greater in nonsmokers ($P < 0.001$) and increased with increasing age ($P = 0.004$). Likewise, waist circumference, FMI, and BMI were greater in nonsmokers ($P = 0.02$, $P = 0.002$, and $P = 0.14$, respectively) and tended to increase with increasing age ($P = 0.13$, $P = 0.052$, and $P = 0.27$, respectively). Waist circumference was higher in those who reported not undertaking vigorous activity ($P = 0.05$) and in those of lower social class ($P = 0.023$). All subsequent analyses took account of the subject's age, gender, social class, current smoking, and activity levels.

Maternal influences on offspring adiposity

Percentage body fat was greater in offspring from mothers with a higher first antenatal visit BMI (rising by 0.35% per kg/m^2 ; $P < 0.001$) and in offspring whose mothers were primiparous (difference, 1.51% in primiparous *vs.* multiparous mothers; $P = 0.034$). Table 1 and Fig. 1, A and C, show that greater offspring percentage body fat was independently associated with higher mothers' BMI, higher pregnancy weight gain, and parity. Waist circumference was also greater in offspring of mothers with higher antenatal booking BMI and in offspring whose mothers were primiparous or had greater pregnancy weight gain. Table 1 and Fig. 1, B and D, show that greater offspring waist circumference had strong independent associations with higher mothers' BMI, higher pregnancy weight gain, and parity. Men and women whose

TABLE 1. Association of mother’s parity, early pregnancy BMI, and pregnancy weight gain with offspring percentage fat, waist circumference, BMI, and FMI

	Univariate	Multivariate
Percentage body fat		
Whether mother primiparous (0 = no, 1 = yes)	0.09 (<i>P</i> = 0.1)	0.15 (<i>P</i> = 0.01)
Mother’s early pregnancy BMI	0.22 (<i>P</i> < 0.001)	0.34 (<i>P</i> < 0.001)
Mother’s pregnancy weight gain	0.14 (<i>P</i> = 0.03)	0.26 (<i>P</i> < 0.001)
Waist circumference		
Whether mother primiparous (0 = no, 1 = yes)	0.11 (<i>P</i> = 0.07)	0.16 (<i>P</i> = 0.008)
Mother’s early pregnancy BMI	0.20 (<i>P</i> < 0.001)	0.31 (<i>P</i> < 0.001)
Mother’s pregnancy weight gain	0.13 (<i>P</i> = 0.05)	0.24 (<i>P</i> < 0.001)
BMI		
Whether mother primiparous (0 = no, 1 = yes)	0.04 (<i>P</i> = 0.6)	0.09 (<i>P</i> = 0.1)
Mother’s early pregnancy BMI	0.24 (<i>P</i> < 0.001)	0.35 (<i>P</i> < 0.001)
Mother’s pregnancy weight gain	0.12 (<i>P</i> = 0.06)	0.26 (<i>P</i> < 0.001)
FMI		
Whether mother primiparous (0 = no, 1 = yes)	0.08 (<i>P</i> = 0.2)	0.13 (<i>P</i> = 0.03)
Mother’s early pregnancy BMI	0.23 (<i>P</i> < 0.001)	0.35 (<i>P</i> < 0.001)
Mother’s pregnancy weight gain	0.14 (<i>P</i> = 0.03)	0.27 (<i>P</i> < 0.001)

The results shown are correlation coefficient and associated *P* values from multiple linear regression analyses that include the subject’s age, sex, smoking status, social class, and activity level. The predictors and outcomes are expressed in SD units, so that the regression coefficients are effectively correlation coefficients. For each measure of offspring body composition, the maternal predictors are included first in univariate and then in multivariate analyses.

mothers were primiparous had on average a 3.5-cm greater waist circumference.

Table 1 also shows that there were similar associations between maternal parity, BMI, and pregnancy weight gain

and offspring BMI and FMI. Offspring percentage fat mass, waist circumference, BMI, and FMI were not related to the mother’s age, height, smoking status, or social class. Smoking during pregnancy was associated with greater

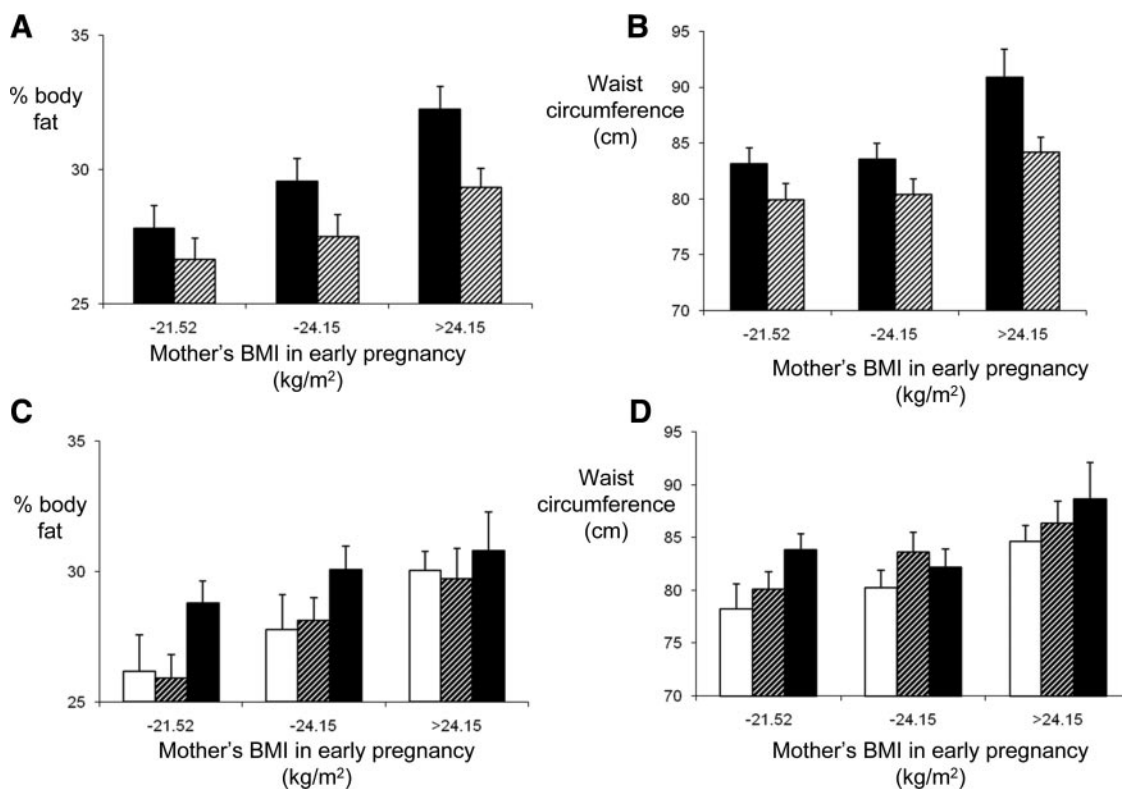


FIG. 1. Maternal BMI at time of antenatal booking, pregnancy weight gain, and parity in relation to offspring adiposity (A and C, offspring percentage body fat; B and D, waist circumference) at age 30 yr. A and B, Parity: ■, primiparous; ▨, multiparous. C and D, Pregnancy weight gain: □, no more than 0.185 kg/wk; ▨, -0.3 kg/wk; ■, more than 0.3 kg/wk. Data are expressed as mean (\pm SEM); all analyses were adjusted for age, gender, and current smoking status. A, Parity, *P* = 0.004; mother’s BMI, *P* < 0.001. B, Parity, *P* = 0.002; mother’s BMI, *P* < 0.001. C, Mother’s BMI, *P* < 0.001; pregnancy weight gain, *P* = 0.002. D, Mother’s BMI, *P* < 0.001; pregnancy weight gain, *P* < 0.001.

offspring BMI ($P = 0.038$), but not with percentage fat mass, FMI, and waist circumference. Inclusion of birth weight adjusted for gestational age in the models did not alter the associations between maternal influences and offspring adiposity (data not shown).

Analyzing the offspring's BMI as a dichotomous outcome, Supplemental Table 2 demonstrates the combined effect size of factors predicting offspring overweight (BMI > 25 kg/m²). The mother's first antenatal BMI, antenatal weight gain, and parity were all independent predictors of overweight.

In exploratory analyses, accounting for maternal BMI, parity, and pregnancy weight gain, the offspring's percentage body fat and FMI were higher in those whose mothers had reported lower fish intake in early pregnancy (percentage body fat, $P = 0.032$; FMI, $P = 0.037$), but were not related to fish intake in late pregnancy or to intakes of the other foods assessed.

Discussion

This study adds to the increasing evidence that maternal factors during pregnancy program lifelong effects on offspring health (1, 2). We show associations between prenatal influence and adiposity in early adulthood that are independent of current lifestyle factors. Higher maternal BMI in pregnancy, greater gestational weight gain, and being first born were all independent predictors of offspring estimated fat mass, waist circumference, BMI, and FMI. Our findings suggest that early life interventions could contribute to reducing the ongoing epidemic of obesity.

Our findings add to recently published observations that the impact of maternal adiposity on offspring obesity risk persists into adulthood (1). Although the mechanisms are unknown, the “developmental overnutrition hypothesis” states that high maternal glucose, free fatty acid, and amino acid concentrations result in permanent changes in appetite control, neuroendocrine functioning, and/or energy metabolism in the developing fetus, thus increasing the risk of obesity in later life. Animal studies support this hypothesis (1). The majority of mothers in this study were within the normal BMI range at antenatal booking (mean BMI, 23.3 kg/m²), and mean gestational weight gain was within current Institute of Medicine recommendations (11). Nonetheless, higher gestational weight gain independently predicted greater adiposity. Although there are studies demonstrating a link between maternal gestational weight gain and later adiposity in childhood (12), adolescence (3), and early adulthood (13), there are also studies showing no effect (14) or that the extremes of gestational weight gain are associated with offspring adiposity (4),

highlighting the importance of good maternal nutrition during pregnancy.

Our findings suggest an influence of maternal parity on offspring adiposity in adulthood. Although firstborn offspring are lighter at birth, being firstborn is associated with increased fat mass in both childhood (15) and adolescence (16). The mechanisms are unknown, but recent experimental data report resetting of the leptin and glucocorticoid axis within the adipocyte, contributing to increased adipogenesis during late gestation and continuing after birth (17). This suggests that some mechanisms underlying obesity are established before, or soon after, birth. This may contribute to the obesity epidemic in communities where there are restrictions on family size and a generation with a greater proportion of first-born children.

Our exploratory analyses of maternal diet and offspring adiposity showed weak associations between lower maternal fish intake in early pregnancy and greater offspring percentage body fat and FMI. This finding needs replication in other studies because it was not an *a priori* hypothesis of our study. Nonetheless, low maternal fish intake in pregnancy is associated with altered fatty acid status (18), and there is some evidence in rodents that maternal fatty acid status influences offspring adiposity (19).

Strengths of our study are the accurate measurements of maternal weight and weight gain during pregnancy, rather than self-recalled prepregnancy weights, and the measurements of offspring skinfold thicknesses, allowing estimation of fat mass. We do not think our findings simply reflect tracking of body size throughout life because, although birth weight was associated with adult waist and BMI after adjustment for adult lifestyle factors, the influence of birth weight was not significant when the maternal influences on obesity were included in the model. However, in an observational study, the direct effect of maternal adiposity on offspring adiposity compared with postnatal lifestyle and shared genetic influences cannot be determined. In addition, we do not think that offspring obesity arises because of lifestyle factors within the family that are maintained in later life because the effects were independent of social class and activity levels. Although we have not measured potential genetic influences on adiposity risk, a recent study found that later life, as well as early life, parental weight gain was associated with offspring adiposity, a finding consistent with an *in utero*, rather than genetic, influence on offspring adiposity (20). Finally, although we were unable to assess the potential impact of paternal adiposity, studies have generally shown a greater effect of maternal than paternal BMI on offspring BMI (1) and fat mass (5).

In conclusion, we have shown that increased maternal BMI in pregnancy, increased gestational weight gain, and primiparity were independent predictors of greater adiposity in adulthood. This study highlights the importance of maternal influences during pregnancy to prevent the intergenerational cycle of obesity. Strategies to raise public awareness of the risks of maternal adiposity and pregnancy weight gain on offspring future health are required.

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