Determinants of Body Fat in Infants of Women With Gestational Diabetes Mellitus Differ With Fetal Sex

Barbara E. Lingwood, phd[1]
Alexandra M. Henry, bsc[1]
Michael C. d'Emden, phd[2]
Amanda-Mei Fullerton, bsc[1]
Robin H. Mortimer, mbbs[2,3]
Paul B. Colditz, dphil[1]
Kim-Anh Lê Cao, phd[4]
Leonie K. Callaway, phd[3]

OBJECTIVE—Neonatal adiposity is a well-recognized complication of gestational diabetes mellitus (GDM). This study aimed to identify factors influencing adiposity in male and female infants of women treated for GDM.

RESEARCH DESIGN AND METHODS—This was a prospective study of 84 women with GDM. Daily blood glucose levels (BGLs) were retrieved from glucose meters, and overall mean fasting and mean 2-h postprandial BGLs were calculated for each woman. Infant body composition was measured at birth, and regression analysis was used to identify significant predictors of infant body fat separately in male and female infants.

RESULTS—Maternal fasting BGL was the major predictor of adiposity in male infants but had little relationship to adiposity in female infants. In male infants, percent fat was increased by 0.44% for each 0.1 mmol/L increase in mean maternal fasting BGL. Maternal BMI was the primary predictor in female infants but had little effect in males. In female infants, percent fat was increased by 0.11% for each 1 kg/m^2 increase in maternal prepregnancy BMI.

CONCLUSIONS—Fetal sex may influence the impact that treatment strategies for GDM have on infant adiposity.

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The maternal metabolic disturbance of gestational diabetes mellitus (GDM) affects fetal development and alters birth weight, BMI, and percent body fat at birth (1,2). Current treatment of GDM achieves normalization of birth weight and reduces neonatal complications (3). However, the effects of GDM on the offspring extend well beyond the fetal period and, thus, offspring of women with GDM also have an increased risk of unfavorable long-term outcomes such as obesity and diabetes, well above that explained by genetics alone (4), even after treatment.

To date, studies designed to inform optimal treatment of GDM have focused on normalization of birth weight, but neonatal adiposity may be a more sensitive marker of disturbed in utero metabolism, risk of obesity, and poor long-term health than birth weight alone (1). Body fat at birth is elevated in infants born to women with GDM even when birth weight is normal (1). In a group of 6- to 12-year-old children born to women with GDM, percent body fat in childhood was significantly correlated to body fat at birth, but there was no relationship between birth weight and weight at the time of study (5). Even though treatment of mild GDM does reduce the incidence of macrosomia, it does not reduce the incidence of obesity in the offspring at 4–5 years (6).

To interrupt the obesity cycle and reduce the risk of future poor adult health, it may be necessary to normalize neonatal adiposity as well as birth weight. To do this, it is essential to understand the factors that determine adiposity in infants of women with GDM.

While genetic factors may be the primary determinant of lean body mass, fetal fat mass may be more strongly influenced by the in utero environment (7). A range of maternal factors have been identified as determinants of neonatal size and body fat, including maternal BMI, parity, maternal glucose concentration, and insulin sensitivity (8–10). Higher gestational weight gain is associated with increased infant birth weight in lean and moderately overweight women (11) and in women with normal glucose tolerance (9) but not in obese women (11) or women with GDM (9). However, the factors influencing fetal fat accretion remain poorly understood.

Both body weight and body composition at birth are different in male and female infants (12), and sex of the infant has been reported as a significant determinant of each (9). We hypothesized that the determinants of fetal body composition may also differ with fetal sex. The aim of this study was to identify factors that influence adiposity in male and female infants born to women treated for GDM.

RESEARCH DESIGN AND METHODS

—The study was approved by the human research ethics committees of the Royal Brisbane and Women's Hospital (RBWH) and the University of Queensland. Informed parental written consent was obtained and participation was voluntary.

This was a prospective study of 84 women diagnosed with GDM and treated at the RBWH. All infants were delivered at or near term (37–42 weeks' gestation). Subjects were excluded if there was a multiple pregnancy or a history of maternal illness other than GDM or if infants had congenital anomalies.

Diagnosis and treatment of GDM

The diagnosis of GDM was based on current Australian Diabetes in Pregnancy

From the [1]University of Queensland Centre for Clinical Research, The University of Queensland, Brisbane, Australia; the [2]Department of Endocrinology and Diabetes, Royal Brisbane and Women's Hospital, Brisbane, Australia; the [3]School of Medicine, The University of Queensland, Brisbane, Australia; and the [4]Queensland Facility for Advanced Bioinformatics, The University of Queensland, Brisbane, Australia.
Corresponding author: Barbara E. Lingwood, b.lingwood@uq.edu.au.
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Society (ADIPS) criteria—a 75-g oral glucose tolerance test resulting in a venous plasma glucose ≥5.5 mmol/L fasting and/or ≥8.0 mmol/L after 2 h (13). After diagnosis, women were treated according to ADIPS guidelines (13), with an initial dietitian review and advice about physical activity. Women were requested to monitor their blood glucose levels (BGLs) (Accu-Chek; Roche Diagnostics, Mannheim, Germany) four times daily—fasting and 2 h after commencing each meal. Target BGLs were set according to current ADIPS guidelines: 5.5 mmol/L or lower fasting, and 7.0 mmol/L or lower 2-h postprandial (13). Insulin treatment was begun if more than two glucose measurements exceeded the target range in 1 week. Daily BGLs were stored in an established database at RBWH. The overall mean fasting and mean 2-h postprandial during the third trimester was calculated for each participant.

Measurement of infant body composition

Infant body composition was measured within 6 days of birth, using the PEA POD body composition system (Life Measurement Inc., Concord, CA) (14). Body composition assessment methodology has been described previously (12). In brief, the infant's mass was measured (to 0.1 g) using the integrated scale, and body volume was assessed using air displacement plethysmography. Infant percent body fat was computed from body density by software integral to the PEA POD system (version 3.0.1) based on a two-compartment model—fat and fat free compartments. The density of fat is assumed to be 0.9007 kg/L. Age- and sex-specific densities of fat free mass are computed based on the data of Fomon et al. (15), taking into account reported fluctuations in hydration level occurring in the first 6 days after birth (16).

Data analysis

Birth weight *z* scores were calculated using the data of Beeby et al. (17), which are based on an Australian population and take into account infant sex and gestation. Parity was converted to a dichotomous variable as follows: nulliparous at commencement of data collection versus other. The association between infant percent body fat and potential predictor variables in male and female infants was initially investigated using univariate analysis. To ensure the assumptions of linearity were not violated, graphical checks using scatter plots and Lowess curves were used. In addition, studentized residuals were examined. In male infants, the relationship between prepregnancy BMI and neonatal fat mass was strongly influenced by one observation (the largest mother in the series had an infant with very low fat mass). The data for male infants was reexamined, removing this observation. When this sensitivity analysis was performed, the data did not vary in any material way from that presented here.

Multiple linear regression was conducted using both forward and backward stepwise selection procedures. Variables were entered into the forward analysis if the *P* value in the multivariate regression was <0.1 and removed from the backward analysis if the *P* value was >0.2. All models presented here underwent residual analysis to ensure error assumptions were not violated. Delta beta influence statistics were also examined to ensure that no single observation was substantially altering the correlation coefficient. There was no instability in the models that would indicate collinearity. It was not possible to test for interactions because of the sample size. However, results of the multivariate regression were checked using two additional methods, partial least squares regression (18) and Random Forests (19), which are less affected by highly correlated variables. Statistical analysis was conducted using STATA (version 10.0; StataCorp, College Station, TX) and R (version 2.10; R Foundation for Statistical Computing, Vienna, Austria). Significance was set at $P < 0.05$. Results are presented as mean ± SD unless otherwise indicated.

RESULTS—The demographic and metabolic characteristics of the cohort are shown in Table 1. There was no difference between women carrying a male fetus and those carrying a female fetus in any of the characteristics recorded. Mean maternal BMI was 28.2 ± 7.7 kg/m^2, and almost 60% of women were overweight or obese (Table 1). This was a relatively well-controlled group of women with GDM; 80% met both current fasting and postprandial ADIPS targets (5.5 and 7.0 mmol/L) (13) on average, and 75% met the lower targets of the American Diabetes Association (5.3 and 6.7 mmol/L) (20). There was no difference in the percentage of women meeting either target according to whether they had a male or female offspring.

Infant characteristics are shown in Table 2. Mean birth weight and birth

Table 1—*Maternal characteristics of the study cohort of 84 women with GDM*

Maternal characteristic	Whole group (*N* = 84)	Male infant (*n* = 42)	Female infant (*n* = 42)
Gestation at delivery (weeks)	39.3 ± 1.1	39.3 ± 1.0	39.4 ± 1.2
Maternal age (years)	32.5 ± 4.8	32.8 ± 5.0	32.3 ± 4.6
Maternal BMI (kg/m^2)	28.2 ± 7.7	28.1 ± 6.4	28.3 ± 8.8
Number of women overweight (BMI 25–30)	22 (26.2)	13 (31.0)	9 (21.4)
Number of women obese (BMI >30)	28 (33.3)	15 (35.7)	13 (31.0)
Nulliparous	42 (50)	23 (55)	19 (45)
Gestational weight gain (kg)	12.0 ± 6.1	11.8 ± 5.7	12.3 ± 6.8
Ethnicity			
White	57 (67.9)	30 (71.4)	27 (64.3)
Indian	8 (9.5)	4 (9.5)	4 (9.5)
Other*	12 (14.3)	5 (11.9)	7 (16.7)
Unrecorded	7 (8.3)	3 (7.1)	4 (9.5)
Smokers	5 (5.9)	3 (7.1)	2 (4.8)
Insulin therapy	20 (24)	11 (26)	9 (21)
Metformin therapy	6 (7.1)	5 (11.9)	1 (2.4)
GTT fasting (mmol/L)	4.88 ± 0.65	4.95 ± 0.72	4.80 ± 0.55
GTT 2 h (mmol/L)	9.01 ± 1.08	9.06 ± 0.85	8.95 ± 1.28
Gestation at diagnosis (weeks)	27.8 ± 4.9	27.7 ± 5.4	27.9 ± 4.3
Treatment commenced (weeks)	29.2 ± 4.8	29.2 ± 5.3	29.2 ± 4.4
Mean fasting BGL (mmol/L)	4.96 ± 0.47	5.01 ± 0.46	4.90 ± 0.49
Mean postprandial BGL (mmol/L)	6.06 ± 0.52	6.06 ± 0.49	6.06 ± 0.55

Data are mean ± SD or *n* (%). GTT, glucose tolerance test. *Other ethnicities (*n*) include Aboriginal (3), Filipino (2), Maori (2), Malaysian (1), Solomon Islander (1), Vietnamese (1), Chinese (1), and Afghani (1).

Table 2—*Infant characteristics of the whole study cohort and according to sex of the infant*

Infant characteristic	Whole group (N = 84)	Male infant (n = 42)	Female infant (n = 42)
Birth weight (g)	3,536 ± 513	3,592 ± 505	3,481 ± 521
Mean birth weight z score	0.436 ± 1.084	0.420 ± 1.054	0.451 ± 1.126
Birth weight <10th percentile	3 (3.6)	1 (2.4)	2 (4.8)
Birth weight >90th percentile	16 (19.0)	9 (21.4)	7 (16.7)
Birth weight >4,000 g	14 (16.7)	9 (21.4)	5 (11.9)
Fat free mass (g)	2,889 ± 329	2,943 ± 314	2,835 ± 340
Infant percent body fat	12.1 ± 4.3*	11.6 ± 4.4*	12.7 ± 4.1*
Infant percent body fat >90th percentile	21 (25.0)*	12 (28.6)*	9 (21.4)*

Data are mean ± SD or n (%). *Indicates significantly different to that in infants born to normal-weight, nondiabetic women (12).

weight z score were not significantly different to infants born to a group of nondiabetic women of normal weight, at the same hospital over a similar time period, and assessed by the same methods (12). Mean infant percent body fat (Table 2) was higher than that of infants born to normal-weighted nondiabetic women (12) for both the whole group (P = 0.003) and for the sexes separately (11.7 ± 4.3 vs. 9.4 ± 3.4, t test, P = 0.027 for males and 12.7 ± 4.3 vs. 10.1 ± 4.4, P = 0.031 for females) despite the slightly lower gestational age. There was no significant difference in birth weight or infant percent fat between white and non-white babies, and percent body fat remained high despite normal birth weight when only white babies were considered. The number of infants with high body fat (21 of 84 or 25%) was significantly different from the expected 10% (χ^2, P = 0.02) (Table 2). Almost half (43%) of babies with high body fat had a birth weight <90th percentile, indicating that elevated body fat was not confined to the large babies. Of the 21 babies with excessive body fat, 14 (67%) of the mothers had both fasting and postprandial mean BGLs within both current ADIPS and American Diabetes Association targets.

Predictors of infant body fat

Univariate analysis of the whole group indicated that mean maternal third trimester fasting BGL (R = 0.30), mean third trimester postprandial BGL (R = 0.25), maternal prepregnancy BMI (R = 0.23), and parity (R = 0.35) were all significantly correlated with infant body fat. When multiple regression analysis was performed, only mean maternal third trimester fasting BGL and parity contributed significantly to the model, and together, these accounted for 19% of the variability in infant percent body fat.

The results of univariate analysis for male and female infants separately are shown in Table 3. The factors that were significantly associated with percent body fat in male infants were mean maternal third trimester fasting, postprandial BGL, and parity. The only factor that was significantly associated with percent body fat in female infants was maternal prepregnancy BMI.

In the male infants, forward stepwise multiple regression analysis indicated that the only significant predictor of infant percent body fat was mean maternal third trimester fasting BGL. Backward stepwise analysis added parity to fasting BGL. Infant percent fat was increased by 0.44% for each 0.1 mmol/L increase in mean maternal fasting BGL and 1.9% in women who were in their second or subsequent pregnancy. Addition of maternal prepregnancy BMI to the regression did not alter the R^2 for the model, and the standardized regression coefficient for BMI was very small and not significant (Table 4). Additional methods of multivariate analysis (partial least squares and Random Forests) also identified maternal third trimester fasting BGL and parity as the most significant predictors of male infant adiposity.

In female infants, forward stepwise analysis revealed that the only significant predictor of infant percent body fat was

Table 3—*Univariate analysis of predictors of infant percent body fat in male and female infants arranged by strength of the association*

Variable	N	R (R^2)	Slope estimate	P value
Male				
Mean third trimester fasting BGL (mmol/L)	42	0.54 (0.30)	5.27	<0.001
Mean third trimester postprandial BGL (mmol/L)	42	0.39 (0.15)	3.47	0.01
Parity (1 = nulliparous, 2 = other)	42	0.34 (0.12)		0.02
Ethnicity (white/other)	42	0.30 (0.09)		0.05
Insulin (0 = no, 1 = yes)	42	0.25 (0.06)		0.10
GTT fasting (mmol/L)	38	0.22 (0.05)	0.04	0.17
Gestation at diagnosis (weeks)	39	0.21 (0.04)	−0.18	0.19
Maternal age (years)	42	0.14 (0.02)	0.12	0.36
Maternal prepregnancy BMI (kg/m^2)	42	0.13 (0.02)	0.09	0.39
Gestational age (weeks)	42	0.10 (0.009)	0.42	0.54
Gestational weight gain (kg)	42	0.10 (0.009)	−0.07	0.53
GTT 2 h (mmol/L)	39	0.09 (0.007)	−0.44	0.61
Female				
Maternal prepregnancy BMI (kg/m^2)	42	0.32 (0.10)	0.14	0.04
GTT 2 h (mmol/L)	37	0.30 (0.08)	−0.93	0.07
Parity (1 = nulliparous, 2 = other)	42	0.24 (0.06)		0.11
Gestational age (weeks)	42	0.17 (0.03)	0.60	0.29
Gestation at diagnosis (weeks)	39	0.13 (0.02)	−0.12	0.42
Mean third trimester postprandial BGL (mmol/L)	42	0.12 (0.02)	0.96	0.42
Mean third trimester fasting BGL (mmol/L)	42	0.11 (0.01)	0.90	0.51
GTT fasting (mmol/L)	36	0.10 (0.01)	0.74	0.55
Maternal age (years)	42	0.08 (0.007)	0.007	0.59
Insulin (0 = no, 1 = yes)	42	−0.07 (0.004)		0.66
Ethnicity (white/other)	42	0.03 (0.0009)		0.82
Gestational weight gain (kg)	42	0.02 (0.0003)	0.007	0.91

GTT, glucose tolerance test.

Table 4—*Multiple regression analysis of predictors of infant percent body fat in male and female infants*

Model	Overall R (R^2)	Standardized coefficient (β)	Slope estimate
Male			
Best model: fasting BGL + parity	0.58 (0.34)		
Fasting BGL (mmol/L)		0.45	4.39
Parity (1 = nulliparous, 2 = other)		0.22	1.94
Best model + BMI	0.58 (0.34)		
Fasting BGL		0.44	
Parity		0.24	
BMI		0.04	
Best female model: BMI + GA + parity	0.47 (0.22)		
Female			
Best model: BMI + parity + GA	0.44 (0.19)		
BMI (kg/m^2)		0.24	0.11
Parity (1 = nulliparous, 2 = other)		0.25	2.07
GA (weeks)		0.20	0.71
Best model + fasting BGL	0.44 (0.19)		
BMI		0.23	
Parity		0.24	
GA		0.20	
Fasting BGL		0.04	
Best male model: fasting BGL + parity	0.33 (0.11)		

Fasting BGL, mean maternal third trimester fasting BGL; BMI, maternal prepregnancy BMI; GA, gestational age.

maternal prepregnancy BMI. Backward stepwise analysis added gestational age and parity. Infant percent fat was increased by 0.11% for each 1 kg/m^2 increase in maternal prepregnancy BMI, 2.1% in women who were in their second or subsequent pregnancy, and 0.7% for each 1-week increase in gestational age at birth. Addition of mean maternal fasting BGL to the model did not improve the model (Table 4). Additional methods of multivariate analysis also identified maternal prepregnancy BMI, parity, and gestational age as significant predictors of female infant adiposity.

When the best model derived in female infants was applied to male infants, its predictive ability was considerably less than the best model for male infants (Table 4). Likewise, when the best model derived in male infants was applied to female infants, its predictive ability was reduced compared with the best model for female infants (Table 4). Predictors of infant body were not altered in either male or female babies when only white babies were considered.

CONCLUSIONS—Increased maternal BMI and elevated maternal BGLs both have been previously reported to be associated with increased neonatal adiposity (1,5,10,21). The current study demonstrates, however, that the influence of these factors may be different in male and female infants born to women with GDM. Glycemia is the primary predictor of adiposity in male infants but has little effect on adiposity in female infants, whereas maternal BMI is the primary predictor in female infants but has little effect in males. By analyzing data for male and female infants separately, we were able to account for a greater proportion of the variability in infant adiposity in male infants than was possible in the combined group.

Umbilical cord blood insulin levels correlate significantly with birth weight and also with maternal BGL (22), supporting the Pedersen hypothesis that maternal BGL is a major determinant of fetal insulin and, thus, fetal growth and adiposity. This explains the macrosomia and increased adiposity that occurs in babies exposed to GDM and why, in this study, maternal BGLs are a strong predictor of adiposity in male infants. This is consistent with findings from the HAPO (Hyperglycemia and Adverse Pregnancy Outcome) study, although that study did not stratify by infant sex (10). Further reductions in adiposity in male infants may be achieved by more aggressive approaches to glucose control. The small sample size of this study does not permit further analysis of BGLs required to normalize adiposity in male infants.

The normalization of adiposity in female infants may be more complex. Higher concentrations of cord blood insulin in girls, despite similar cord BGLs to boys, and smaller size suggest they are more insulin resistant than boys at birth. They are also more insulin resistant from 5 years of age (22). This intrinsic insulin resistance in females is thought to be genetic and possibly associated with genes linked to diabetes and glucose intolerance on the X chromosome (23). Thus, it may result in hyperinsulinemia, which is partially independent of BGLs, leading to infant adiposity, which is not as strongly associated with maternal hyperglycemia as that in male infants. Maternal obesity is known to be associated with increased infant adiposity (9), but it is unclear what is responsible for this association or why it may be more apparent in female than in male infants.

The current study also confirms other findings that infant adiposity may remain elevated even when birth weight is normal (1). This underlines the importance of assessing infant body composition and aiming to normalize this rather than birth weight alone. If adiposity is not normalized, risks of poor long-term health in the child may remain. The current study suggests that different strategies may be required to normalize adiposity in male and female infants. The current focus on maternal blood glucose control may be appropriate for normalization of adiposity in male infants, but our results suggest that reduction of maternal BGLs may have little effect on adiposity in female infants. Normalization of adiposity in female infants may require different strategies, and there is a need for continuing research in this area. Normalization of the in utero metabolic environment may be particularly important in female infants because these are the infants who will grow up to become mothers themselves and influence the next generation—with the potential for an ever-enlarging intergenerational cycle of obesity.

A number of strengths add to the value of this study. We have used a robust method of assessing neonatal body composition that has been validated in a number of studies (14). Air displacement plethysmography is now considered a gold standard method for assessment of body composition in children (24,25). In addition, we have used mean maternal BGLs across the third trimester during

treatment rather than the results of a single glucose tolerance test, providing a long-term picture of maternal metabolic conditions. There are also a number of limitations. The low sample size prevents some additional interesting analysis, such as whether lower targets would prevent excess body fat in either male or female infants and what these targets should be. We also were not able to perform separate analysis of non-white ethnicities or analysis of adiposity in infants of women not meeting targets. Additional information could have been obtained if paternal height and weight had been recorded.

Different factors predict adiposity in male and female infants born to women with GDM. The observation that female infant adiposity is more highly influenced by maternal BMI may be important in understanding the potential generational cycle of obesity. Control of maternal glucose levels does not appear to be an adequate approach to reducing female infant adiposity, and future research needs to examine this issue and whether GDM treatment should be altered according to the sex of the infant to normalize infant adiposity.

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B.E.L. designed the study, supervised data collection, analyzed data, and wrote the manuscript. A.M.H. assisted with data collection and analysis. M.C.d'E. contributed to data collection and discussion and reviewed and edited the manuscript. A.-M.F. assisted with data collection and analysis. R.H.M. and P.B.C. contributed to study design and discussion and reviewed and edited the manuscript. K.-A.L.C. provided statistical advice regarding study design and assisted with data analysis. L.K.C. assisted with study design and data analysis, contributed to discussion, and reviewed and edited the manuscript.

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