

Relationship between maternal body composition changes and heavy for date infants in pregnant women with diabetes

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ABSTRACT

Aims/Introduction: Maternal hyperglycemia is associated with heavy for date (HFD) infants. Considering the association between body composition and hyperglycemia, we investigated the changes in maternal body composition and their relationship with HFD infants in pregnant women with diabetes.

Materials and Methods: Body composition was measured during pregnancy using a bioelectrical impedance analysis system. This retrospective study included 151 pregnant women; 27 women had type 1 diabetes mellitus (DM), 21 had type 2 DM, 101 were diagnosed with gestational DM, and 2 had overt DM. The number of HFD infants was 40.

Results: In the non-type 1 DM group, change in fat mass (ΔFM) ($P < 0.01$) and pre-pregnancy BMI ($P < 0.05$) were risk factors for HFD. In the insulin group, ΔFM , pre-pregnancy BMI, and age (all $P < 0.05$) were risk factors for HFD. The area under the curve was 0.813 for the predictive model combined with ΔFM and pre-pregnancy BMI in the non-type 1 DM group and 0.818 for the model combined with ΔFM , pre-pregnancy BMI, and age in the insulin group.

Conclusions: The combination of body composition parameters and clinical data may predict HFD in pregnant women with diabetes.

INTRODUCTION

Human body composition reflects nutritional status. Assessing maternal nutritional status and providing nutritional guidance during pregnancy is essential to ensure appropriate newborn birthweight and the health of the next generation¹. Body mass index (BMI) is commonly used as an indicator of obesity. However, it does not measure fat percentage. Fat mass (FM), fat-free mass (FFM), and total body water (TBW) measured using bioelectrical impedance analysis (BIA) can accurately reflect body composition and are considered better predictors of maternal nutritional status than BMI^{2–4}.

Several studies have reported that inappropriate gestational weight gain (GWG) is associated with adverse pregnancy outcomes in women without diabetes⁵. A meta-analysis reported that maternal pre-pregnancy BMI and GWG were associated with the risk of pregnancy complications⁶. Obese mothers with

high GWG had the highest risk of pregnancy complications. Promoting appropriate GWG may thus reduce pregnancy complications, and ultimately, the risk of maternal and neonatal morbidities. In contrast, inappropriate GWG appears to be associated with an increased risk of adverse maternal and fetal outcomes in women with diabetes, regardless of pre-pregnancy BMI⁷. To our knowledge, few studies have longitudinally evaluated body composition, including GWG as well as FM, FFM, and TBW, in pregnant women with hyperglycemic disorders.

Maternal hyperglycemia is associated with several risks of adverse perinatal outcomes, and heavy for date (HFD) infant is one of these. HFD is a serious complication that can cause shoulder dystocia, labor arrest, emergency cesarean section, and so on. Measurements of body composition-related indicators, such as FFM, in the second trimester of non-diabetic pregnant women can predict the risk of macrosomia, enabling obstetricians to implement interventions earlier to reduce adverse perinatal outcomes⁸. Although the relationship between maternal body composition and newborn birth weight has been

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investigated, the results remain inconsistent. Some studies have shown a correlation with FFM and newborn birth weight^{9–11}; however, body composition is known to vary with race^{1, 12}. Few studies have reported on the maternal body composition and newborn birth weight in pregnant Asian women with diabetes. Therefore, in this retrospective study, we aimed to assess the changes in maternal body composition and investigate their relationship with HFD in Japanese pregnant women with diabetes.

MATERIALS AND METHODS

Study design

This retrospective cohort study of patients who gave birth at Okayama University Hospital was conducted according to the guidelines of the Declaration of Helsinki. All procedures involving human patients were approved by the Ethics Committee of Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences and Okayama University Hospital (approval no. 1806-009). Written informed consent was obtained from all the patients.

Participants and procedures

Maternal body composition was measured at each prenatal checkup in pregnant women with type 1 DM, type 2 DM, gestational DM (GDM), and overt DM. “non-type 1 DM group” contained type 2 DM, GDM, and overt DM. This study was limited to pregnant Japanese women. Women with preterm delivery at <37 weeks, overdue delivery at >42 weeks and multiple pregnancies were excluded from the study.

Diagnosis of GDM and overt DM

Participants except for those with type 1 and type 2 DM underwent a 2 h, 75 g oral glucose tolerance test (OGTT) when their random plasma glucose level was ≥ 100 mg/dL in the first and second trimesters. The OGTT results were used to identify women with GDM or overt DM according to the recommendation of the Japanese Society of Diabetes and Pregnancy based on the International Association of Diabetes and Pregnancy Study Groups guideline (GDM was identified by at least one OGTT value: fasting glucose level ≥ 92 mg/dL, 1 h ≥ 180 mg/dL or 2 h ≥ 153 mg/dL; overt DM was identified by fasting glucose level ≥ 126 mg/dL or hemoglobin A1c $\geq 6.5\%$)¹³.

Medical nutritional therapy and insulin treatment

All pregnant women with diabetes were advised of the daily energy intake (pre-pregnancy BMI <25 kg/m²: ideal bodyweight $\times 30 + 200$ kcal/day, pre-pregnancy BMI ≥ 25 kg/m²: ideal bodyweight $\times 30$ kcal/day). Participants were instructed to self-monitor their blood glucose levels using Glutest NEO[®] (PHC Corporation, Tokyo, Japan). Patients with pre-breakfast fasting blood glucose levels >95 mg/dL, pre-prandial glucose levels >100 mg/dL, and/or postprandial glucose levels >120 mg/dL received insulin treatment¹⁴. “Insulin group” contained the participants who had received insulin treatment.

Measurements

Body composition was measured in G1 (up to 15 gestational weeks), G2 (16–27 weeks), G3 (28 weeks to delivery) using a foot-to-foot BIA system (TANITA MC-180[®]; TANITA, Tokyo, Japan). The participants stood erect on the footpads of the analyzer with bare feet and measurements were obtained when the hand grips were held. Electric current was supplied from electrodes placed on the tips of the toes and fingers, and the voltage on the heels of both feet and near the sides of both hands was measured. Body weight, FM, FFM, and TBW were analyzed with the BIA. The BIA device (TANITA MC-180[®]) used a built-in “maternity mode,” which adjusts body composition estimates for gestational age by accounting for the average weights of the fetus, placenta, and amniotic fluid. This adjustment is based on proprietary algorithms provided by the manufacturer, intended to improve the accuracy of maternal fat mass and water content estimation during pregnancy. Several studies have shown the BIA device is accurate and reliable in predicting maternal body composition excluded the weight of the fetus, placenta and amniotic fluid^{3, 15, 16}. Data on maternal age, pre-pregnancy BMI, DM category, weeks and mode of delivery, newborn birth weight, and perinatal prognosis were collected from medical records. HFD was defined as newborn birth weight ≥ 90 th percentile. GWG (GWG from G1 to G3), Δ FM (FM gain from G1 to G3), Δ FFM (FFM gain from G1 to G3), and Δ TBW (TBW gain from G1 to G3) were calculated with the measurement data. Maternal height was measured at the first visit. Pre-pregnancy BMI was calculated by the data of height and the pre-pregnancy maternal weight obtained from self-reports. HFD was defined as birth weight ≥ 90 th percentile based on the “New Japanese neonatal anthropometric charts for gestational age at birth” by Itabashi *et al.*¹⁷, published by the Japan Pediatric Society.

Statistical analysis

We performed two subgroup analyses: DM type and insulin use. Differences among the BIA measures of GWG, Δ FM, Δ FFM, and Δ TBW between obese and non-obese subgroups were evaluated using the two-independent samples *t*-test. The relationship of maternal body composition and clinical data with the risk of HFD was investigated using logistic regression analysis. To evaluate the predictive performance of the risk factors, receiver operating characteristics (ROC) curve analysis was performed, and the area under the curve (AUC) was calculated. To assess the internal validity of the predictive models and mitigate overfitting, we conducted five-fold cross-validation using logistic regression. The average AUC and its 95% confidence interval (95% CI) were calculated for each model across folds. Additionally, in response to reviewer feedback, a subgroup analysis was performed among women with GDM to examine the association between Δ FM and the risk of having a HFD neonate. Logistic regression analysis was conducted within the GDM subgroup, adjusting for maternal age and pre-pregnancy BMI.

Statistical significance was set at $P < 0.05$. Analyses were performed using STATA ver. 18.0.

RESULTS

In total, 180 women participated in this study. Twenty patients with preterm delivery at <37 weeks, one patient with overdue delivery at >42 weeks, and 8 patients with multiple pregnancies were excluded. Therefore, 151 women met the inclusion criteria. The characteristics of the study population in each subgroup of DM are summarized in Table 1. Non-type 1 DM group contained type 2 DM, GDM, and overt DM. The mean ages for the type 1 DM group and non-type 1 DM group were 29.8 and 35.4 years, respectively, and the mean pre-pregnancy BMI was 23.3 and 25.2 kg/m², respectively (type 1 DM group; $n = 27$, non-type 1 DM group; $n = 124$). Among the participants, 66.7% and 50.8% were non-obese (BMI <25 kg/m²) and 33.3% and 39.2% were obese (BMI ≥ 25 kg/m²) in the type 1 DM group and non-type 1 DM group, respectively. The number of HFD infants was 17 (89.5%) and 23 (18.5%).

There were significant changes in maternal body composition from G1 to G3 when patients were classified by the DM status. In the type 1 DM group, the Δ FM ($P = 0.04$) of non-obese

women was significantly greater than that of obese women (Figure 1a). In the non-type 1 DM group, GWG ($P = 0.03$), Δ FM ($P < 0.001$), and Δ FFM ($P = 0.04$) of non-obese women were significantly greater than that of obese women (Figure 1b).

To investigate the associations between HFD and body composition measurement and clinical data, a logistic regression analysis was conducted (Table 2). There were no statistically significant factors in the type 1 DM group. However, in the non-type 1 DM group, Δ FM (odds ratio [OR] 1.549, 95% CI 1.123–2.135, $P < 0.01$) and pre-pregnancy BMI (OR 1.184, 95% CI 1.013–1.385, $P < 0.05$) increased the risk of HFD. The accuracy of significant variables for predicting HFD is shown in Table 3. The AUC for Δ FM was 0.715 (95% CI 0.516–0.914), which was greater than that of pre-pregnancy BMI (AUC 0.633, 95% CI 0.415–0.850). In addition, a predictive model for HFD based on Δ FM and pre-pregnancy BMI was established (Combined Model 1). The AUC in Model 1 was 0.813 and the 95% CI was 0.631–0.995. The ROC curve for HFD prediction in the non-type 1 DM group is displayed in Figure 2. To address the potential heterogeneity among diabetes subtypes, we additionally performed a subgroup analysis limited to women with GDM, which comprised the largest group with a sufficient sample size ($n = 101$). In this analysis, Δ FM was significantly associated with the risk of HFD (OR 1.66, 95% CI 1.08–2.53, $P = 0.020$), independent of maternal age and pre-pregnancy BMI (Table S1).

Subsequently, the study population was grouped according to insulin use (Table 4). The mean age was 34.5 years in the insulin group and 34.2 years in the no insulin group, while the mean pre-pregnancy BMI was 25.5 and 23.9 kg/m² for the insulin and no insulin group, respectively (insulin group; $n = 93$, no insulin group; $n = 58$). Among the participants, 45.2% and 67.2% were non-obese and 54.8% and 32.8% were obese for the insulin and no insulin groups, respectively. The number of HFD infants born to women with insulin treatment was 31 (33.3%), while the no insulin group gave birth to 9 (15.5%) HFD infants.

Changes of maternal body composition from G1 to G3 classified by insulin use is shown in Figure 3. In both the insulin group and no insulin group, GWG ($P = 0.0016$ and $P = 0.03$) and Δ FM ($P < 0.001$ and $P < 0.001$) of non-obese women were significantly greater than that of obese women.

A logistic regression analysis was performed based on grouping of participants use of insulin. There were no statistically significant factors in the no insulin group (Table 5). In the insulin group, Δ FM (OR 1.429, 95% CI 1.080–1.892), pre-pregnancy BMI (OR 1.224, 95% CI 1.016–1.476), and age (OR 0.867, 95% CI 0.772–0.973) increased the risk of HFD. The accuracy of significant variables for predicting HFD is shown in Table 6. The AUC for age (AUC 0.662, 95% CI 0.622–0.892) was larger than the AUC for Δ FM (AUC 0.518, 95% CI 0.513–0.812). The AUC for pre-pregnancy BMI was 0.757 (95% CI 0.351–0.686), which was greater than other factors, but not significantly

Table 1 | Characteristics of the study population in each subgroup of diabetes mellitus

	Type 1 DM group ($n = 27$)	Non-type 1 DM group ($n = 124$)
Age (year)	29.8 (21–39)	35.4 (21–45)
Pre-pregnancy BMI (kg/m ²)	23.3 (17.9–33.7)	25.2 (16.9–45)
BMI category (kg/m ²)		
Non-obese (less than 25)	18 (66.7)	63 (50.8)
Obese (25 or higher)	9 (33.3)	61 (39.2)
DM category		
Type 1 DM	27 (100.0)	–
Type 2 DM	–	21 (16.9)
GDM	–	101 (81.5)
Overt DM	–	2 (1.6)
Gestation age at delivery (week)	38.3 (37–41)	38.9 (37–41)
GWG (kg)	10.5 (3.1–15.4)	5.7 (–3.3 to 15.1)
Δ FM (kg)	2.0 (–1.5 to 6.1)	–0.9 (–11.0 to –9.2)
Δ FFM (kg)	3.5 (–0.7 to 8.4)	2.9 (–3.0 to 11.9)
Δ TBW (kg)	3.3 (0.1–7.2)	2.4 (–2.9 to 8.6)
Mode of delivery (%)		
Spontaneous vaginal delivery	18 (66.7)	74 (59.7)
Instrumental	3 (11.1)	15 (12.1)
Cesarean delivery	6 (22.2)	35 (28.2)
Newborn birth weight (kg)	3.5 (2.4–5.2)	3.1 (2.0–4.4)
HFD	17 (89.5)	23 (18.5)

Data are mean (range) or number (%) unless otherwise specified. DM, diabetes mellitus; GDM, gestational diabetes mellitus; GWG, gestational weight gain from G1 to G3; HFD, heavy-for-dates; Δ FFM, fat-free mass gain from G1 to G3; Δ FM, fat mass gain from G1 to G3; Δ TBW, total body water gain from G1 to G3.

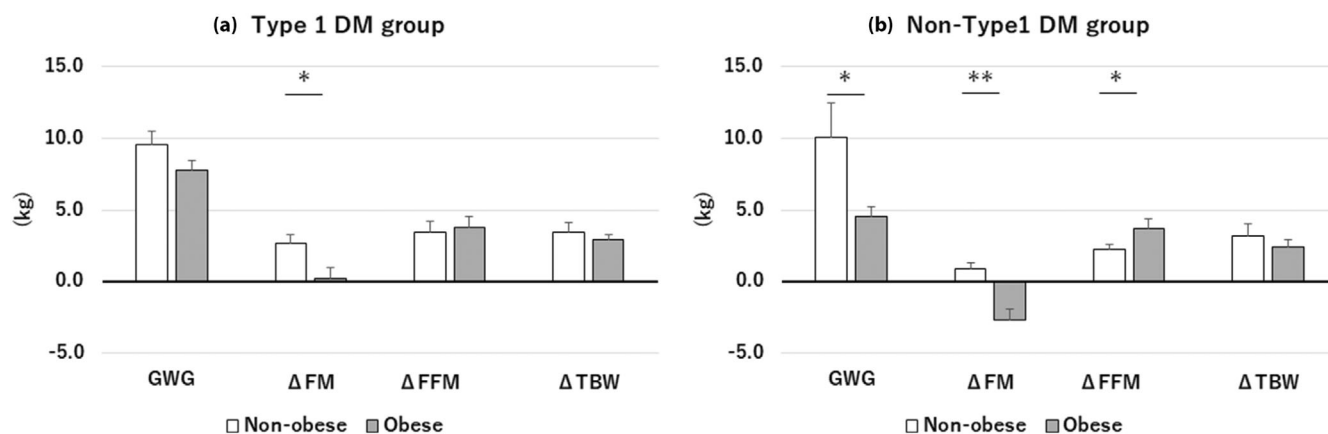


Figure 1 | Changes of maternal body composition from G1 to G3 classified by DM group. Bars represent mean \pm standard error (SE). Units are in kilograms (kg). Measurements for (a) type 1 DM group ($n = 27$) and (b) non-type 1 DM group ($n = 124$). * $P < 0.05$, ** $P < 0.01$. DM, diabetes mellitus; GWG, gestational weight gain from G1 to G3; ΔFFM, fat-free mass gain from G1 to G3; ΔFM, fat mass gain from G1 to G3; ΔTBW, total body water gain from G1 to G3.

Table 2 | Risk factors associated with HFD classified by DM type

Variables	β	SE	Wald	OR (95% CI)	P
Type 1 DM group					
ΔFM (kg)	-0.114	0.305	0.027	0.952 (0.524,1.728)	NS
ΔFFM (kg)	0.593	0.352	0.483	1.278 (0.640,2.550)	NS
ΔTBW (kg)	0.439	0.428	0.252	1.239 (0.536,2.866)	NS
Pre-pregnancy BMI (kg/m ²)	0.278	0.234	0.114	1.082 (0.684,1.713)	NS
Age (year)	0.227	0.132	0.134	1.050 (0.810,1.361)	NS
Non-type 1 DM group					
ΔFM (kg)	1.713	0.164	7.128	1.549 (1.123,2.135)	<0.01
ΔFFM (kg)	0.383	0.280	0.220	1.140 (0.659,1.974)	NS
ΔTBW (kg)	-0.443	0.366	0.312	0.815 (0.398,1.669)	NS
Pre-pregnancy BMI (kg/m ²)	0.953	0.080	4.498	1.184 (1.013,1.385)	<0.05
Age (year)	0.044	0.085	0.010	0.992 (0.840,1.171)	NS

CI, confidence interval; OR, odds ratio; SE, standard error; β , standardized partial regression coefficient.

greater. A predictive model for HFD based on ΔFM, pre-pregnancy BMI, and age was established (Combined Model 2). The AUC for Model 2 was 0.818 and the 95% CI was 0.704–0.932. The ROC curve for HFD prediction in the insulin

group is shown in Figure 4. The cross-validated AUC for Combined Model 1 (non-type 1 DM group) was 0.738 (95% CI 0.551–0.982), and for Combined Model 2 (insulin group) was 0.712 (95% CI 0.611–0.807). These results demonstrate consistent predictive performance and are summarized in Table S2.

DISCUSSION

This study revealed three clinical findings for pregnant women with diabetes. First, the obese group had less increase in FM and therefore less weight gain than the non-obese group. Second, weight gain during pregnancy was mainly due to the increase in FFM. Third, FM gain, maternal age, and pre-pregnancy BMI might contribute to HFD prediction, and combining these factors could potentially improve accuracy.

The obese pregnant women with diabetes had less increase in FM and therefore less weight gain than the non-obese group. Excessive maternal weight or obesity and GWG are known to be associated with perinatal complications of GDM. Obese mothers with excessive GWG may have the highest risk of adverse outcomes. Promoting a healthy pre-pregnancy BMI and GWG is effective to reduce perinatal complications and ensure the health of women with GDM^{18, 19}. Nutritional therapy and exercise interventions to control blood glucose levels are also useful for appropriate GWG and to decrease adverse

Table 3 | Accuracy of different variables and combination model for predicting HFD in non-type 1 DM group

Variables	AUC	95% CI	P	Cutoff	Sensitivity	Specificity
ΔFM (kg)	0.715	0.516–0.914	<0.01	1.25	0.667	0.741
Pre-pregnancy BMI (kg/m ²)	0.633	0.415–0.850	<0.05	27.70	0.556	0.741
Combined Model 1	0.813	0.631–0.995	<0.001	0.10	0.889	0.685

Combined Model 1, predictive model for HFD combined with ΔFM and pre-pregnancy BMI. AUC, area under curve.

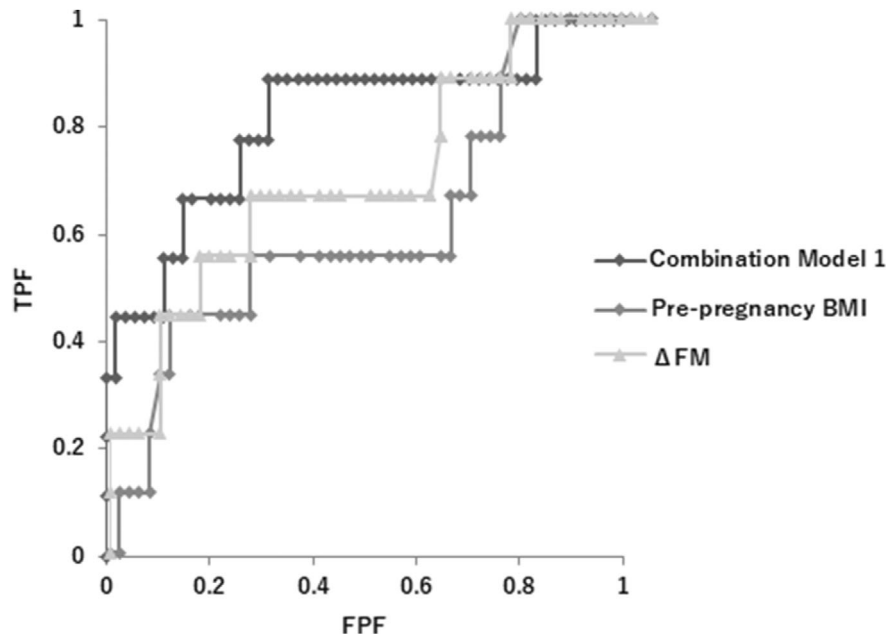


Figure 2 | ROC curve of the risk factors for HFD in the non-type 1 DM group. The pre-pregnancy BMI, Δ FM and Combined Model 1 are displayed. FPF, false positive fraction; TPF, true positive fraction; Δ FM, fat mass gain from G1 to G3.

pregnancy outcomes. In general, obese pregnant women are known to gain less weight than the non-obese pregnant women without hyperglycemic disorders.²⁰ The present study revealed a similar trend in pregnant women with diabetes. Furthermore, the FM loss was greater in the obese group than in the non-obese group. The FM loss was greater in the non-type 1 DM group than in the type 1 DM group, and it was also greater in the insulin group than in the no insulin group. Many obese pregnant women might complicate type 2 diabetes or GDM with poor glucose control. They had more strict nutritional therapy and optimal GWG guidance. Nutritional therapy might have a relationship for the FM loss.

The weight gain during pregnancy was mainly caused by the FFM gain in pregnant women with diabetes. Previous studies reported that normal healthy women gained 12.5 kg of body weight, that is a weight gain associated with the best clinical outcome, and 3–5 kg of FM was stored during pregnancy to satisfy the total requirement of 30,000 kcal²¹. However, it has been unclear whether changes in weight, FM, and FFM in pregnant women with abnormal glucose metabolism are similar to those in normal pregnant women. This study revealed the longitudinal changes in diabetic maternal body composition, specializing in Asians. Medical nutritional therapy is based on the optimal weight gain calculated based on the pre-pregnancy BMI. In the United States, weight recommendations for the gestational period are provided by the Institute of Medicine, which aims to achieve a birth weight of 3,000–4,000 g at 39–40 weeks of gestation. In Japan, the optimal GWG is recommended by the Japan Society of Obstetrics and Gynecology²².

A systematic review and meta-analysis reported that diet plus physical activity intervention was the most effective therapy for the prevention of both GDM and excessive GWG²³. Further studies are needed to investigate whether nutritional therapy and glycemic control decrease FM gain in pregnant women with diabetes.

FM gain, maternal age, and pre-pregnancy BMI might contribute to HFD prediction, and the combination of these factors could potentially improve accuracy. Although the cross-validated AUCs were slightly lower than the original model AUCs, this is expected due to the conservative nature of cross-validation. The results support the internal stability of both predictive models. One of the most serious complications caused in pregnancy with diabetes is HFD, which may lead to stressful situations for obstetricians, including shoulder dystocia or emergency cesarean section. Although HFD is generally predicted by ultrasound examination, measurement errors may occur. The ability to predict the possibility of HFD using maternal body composition could be clinically useful. Previous studies have reported that newborn birth weight is associated with maternal FFM during the first trimester of pregnancy²⁴. However, it is unclear why the FFM correlates with newborn birth weight. The FFM includes TBW, bones, and proteins. An increase in FFM is primarily because of an increase in TBW. Several studies have shown that increased TBW is associated with birth weight in pregnant women without diabetes^{25–28}. Furthermore, decreased TBW may lead to increased blood viscosity and inadequate oxygen supply to the tissues²⁹. In our study, weight gain during pregnancy was mainly caused by an

Table 4 | Characteristics of the study population in each subgroup of insulin use

	Insulin group (n = 93)	No insulin group (n = 58)
Age (year)	34.5 (21–45)	34.2 (21 to 43)
Pre-pregnancy BMI (kg/m ²)	25.5 (16.9–42.2)	23.9 (16.9 to 42.3)
BMI category (kg/m ²)		
Non-obese (less than 25)	42 (45.2)	39 (67.2)
Obese (25 or higher)	51 (54.8)	19 (32.8)
DM category		
Type 1 DM	26 (28.0)	–
Type 2 DM	22 (23.7)	–
GDM	43 (46.2)	58 (100.0)
Overt DM	2 (2.1)	–
Gestation age at delivery (week)	38.6 (37–41)	39.0 (37 to 41)
GWG (kg)	7.1 (0.0–15.4)	5.4 (–3.3 to 14.5)
ΔFM (kg)	0.0 (–11.0 to 7.1)	–0.7 (–7.5 to 9.2)
ΔFFM (kg)	3.4 (–3.0 to 11.9)	2.5 (–1.6 to 11.5)
ΔTBW (kg)	3.1 (–2.9 to 8.6)	1.9 (–19.5 to 7.4)
Mode of delivery (%)		
Spontaneous vaginal delivery	57 (61.3)	35 (60.3)
Instrumental	11 (11.8)	7 (12.1)
Cesarean delivery	25 (26.9)	16 (27.6)
Newborn birth weight (kg)	3.3 (2.0–5.2)	3.1 (2.0 to 4.4)
HFD	31 (33.3)	9 (15.5)

Data are mean (range) or number (%) unless otherwise specified. BMI, body mass index; DM, diabetes mellitus; GDM, gestational diabetes mellitus; GWG, gestational weight gain from G1 to G3; HFD, heavy-for-dates; ΔFFM, fat-free mass gain from G1 to G3; ΔFM, fat mass gain from G1 to G3; ΔTBW, total body water gain from G1 to G3.

increase in FFM, and FM gain was less than FFM gain. In addition, it was shown that FM decreased during pregnancy in the obese group. Therefore, the risk of HFD would increase if

Table 5 | Risk factors associated with HFD classified by insulin use

Variables	β	SE	Wald	OR (95% CI)	P
Insulin group					
ΔFM (kg)	1.396	0.143	6.223	1.429 (1.080, 1.892)	<0.05
ΔFFM (kg)	0.486	0.231	0.548	1.186 (0.755, 1.864)	NS
ΔTBW (kg)	0.168	0.263	0.090	1.082 (0.646, 1.812)	NS
Pre-pregnancy BMI (kg/m ²)	1.018	0.095	4.523	1.224 (1.016, 1.476)	<0.05
Age (year)	–0.897	0.059	5.893	0.867 (0.772, 0.973)	<0.05
No insulin group					
ΔFM (kg)	3.692	0.969	1.150	2.825 (0.423, 2.060)	NS
ΔFFM (kg)	8.072	2.534	1.445	1.582 (0.327, 7.663)	NS
ΔTBW (kg)	–9.432	3.830	1.344	0.018 (0.00, 21.474)	NS
Pre-pregnancy BMI (kg/m ²)	–4.314	0.733	1.066	0.469 (0.112, 1.974)	NS
Age (year)	1.237	0.520	0.220	1.276 (0.460, 3.537)	NS

CI, confidence interval; OR, odds ratio; SE, standard error; β, standardized partial regression coefficient.

FM gain increased excessively. In the subgroup analysis among women with GDM, FM gain remained a significant risk factor for HFD, even after adjustment for age and pre-pregnancy BMI. This suggests that GDM pregnancies may be particularly sensitive to maternal fat accretion, and that FM gain could serve as a clinically relevant predictor of neonatal overgrowth. We used a foot-to-foot bioelectrical impedance analysis (BIA) system with a built-in maternity mode that adjusts for gestational age. While this algorithm improves measurement accuracy by accounting for the weights of the fetus, placenta, and amniotic fluid, trimester-specific fluid shifts and peripheral edema may still affect fat mass estimation. Future studies could enhance body composition assessment by incorporating more advanced techniques such as bioelectrical impedance spectroscopy or clinical edema scoring.

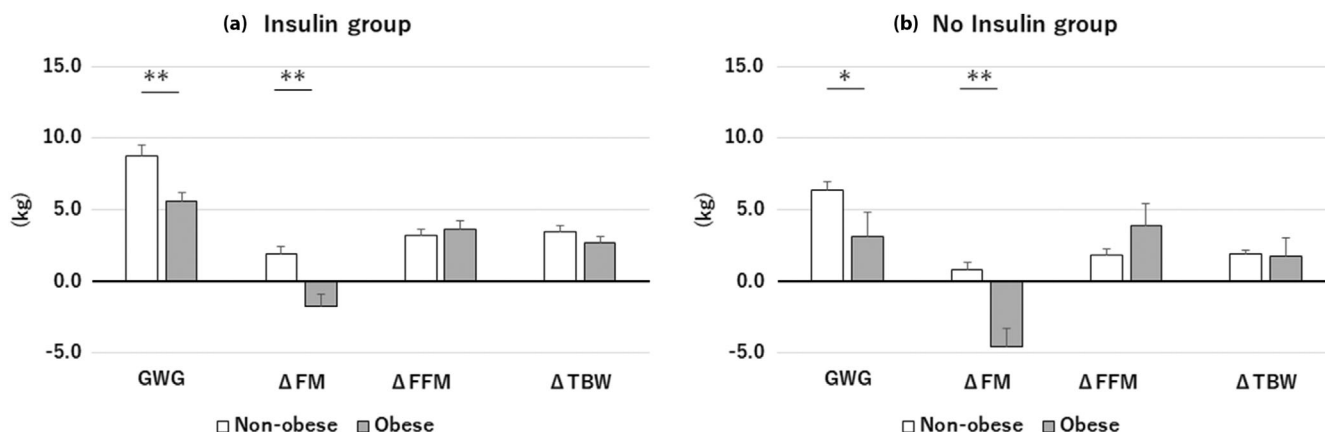
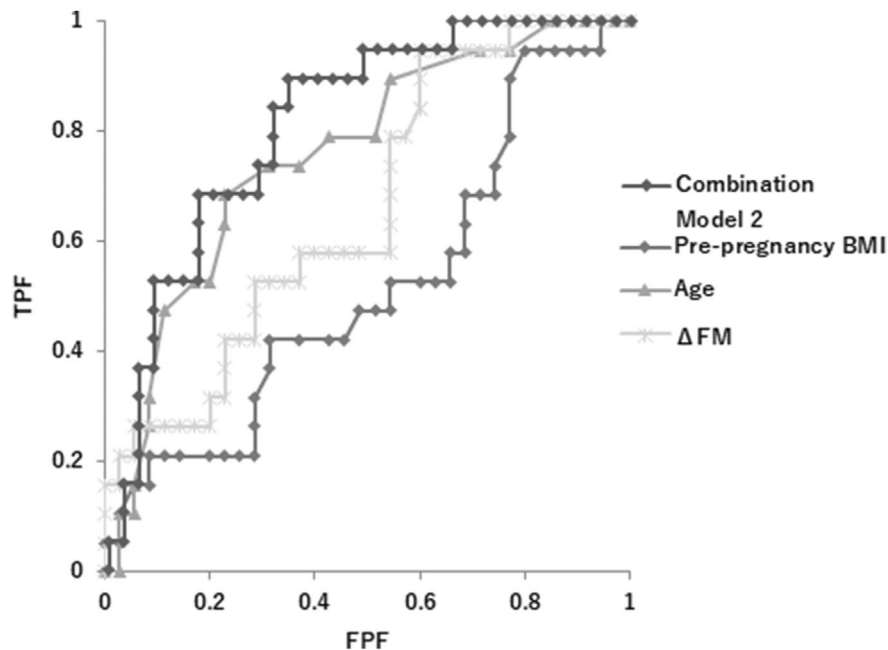


Figure 3 | Changes of maternal body composition from G1 to G3 classified by insulin use. Bars represent mean \pm standard error (SE). Units are in kilograms (kg). (a) Insulin group (n = 93) and (b) no insulin group (n = 58). **P* < 0.05, ***P* < 0.01. GWG, gestational weight gain from G1 to G3; ΔFFM, fat-free mass gain from G1 to G3; ΔFM, fat mass gain from G1 to G3; ΔTBW, total body water gain from G1 to G3.

Table 6 | Accuracy of different variables and combination model for predicting HFD in insulin group

Variables	AUC	95% CI	P	Cutoff	Sensitivity	Specificity
Δ FM (kg)	0.518	0.513–0.812	<0.05	1.60	0.526	0.714
Pre-pregnancy BMI (kg/m^2)	0.757	0.351–0.686	NS	27.70	0.421	0.686
Age (year)	0.662	0.622–0.892	<0.001	33.00	0.684	0.771
Combined Model 2	0.818	0.704–0.932	<0.001	0.29	0.842	0.686

Combined Model 2, predictive model for HFD combined with Δ FM, pre-pregnancy BMI, and age. AUC, area under curve.

**Figure 4** | ROC curve of the risk factors for HFD in the insulin group. The pre-pregnancy BMI, age, Δ FM and Combined Model 2 are displayed. FPF, false positive fraction; TPF, true positive fraction; Δ FM, fat mass gain from G1 to G3.

Pregnant women with non-type 1 DM or insulin treatment had the risk of HFD because the proportion of obese pregnant women was relatively high in the non-type 1 DM and insulin groups. To the best of our knowledge, this is the first study to assess the relationship between HFD infants and maternal body composition in pregnant women with diabetes. We believe that these results add to the evidence for Asian data. Several studies have reported that dietary interventions favorably influence outcomes related to maternal glycemia and newborn birth weight³⁰. Further studies focusing on blood glucose levels are needed. In the future, it may be worthwhile to incorporate maternal age, pre-pregnancy BMI, as well as maternal body composition, especially FM, into medical nutritional therapy.

This study had several limitations. First, the pre-pregnancy BMI was self-reported in most cases; however, the values were obtained in early pregnancy when weight changes are minimal, and none of the participants experienced significant

weight loss due to hyperemesis. Second, the small number of women with type 1 DM may have limited the power to detect significant associations in this group. Third, several potentially important confounders—such as HbA1c, insulin dosing, parity, lifestyle factors, and socioeconomic status—were not available due to the retrospective design. Fourth, although HFD was defined using the Japanese national growth chart endorsed by the Japan Pediatric Society, sensitivity analyses using other international standards (e.g., WHO or INTERGROWTH-21st) could not be performed. Finally, as this was a single-center study involving a Japanese population, external validation is needed to assess generalizability. Despite these limitations, our study is one of the first to investigate longitudinal body composition changes and their association with neonatal fat deposition in Asian women with diabetes. The findings provide valuable insights that may inform future multicenter and prospective studies to optimize nutritional and glycemic management during pregnancy. We are

currently planning a prospective, multicenter validation study. The study will aim to recruit a modest number of pregnant women with diabetes across several clinical sites, enabling evaluation of model performance in more diverse settings.

In conclusion, these results suggest that a combination of body composition parameters and clinical data may have a potential association in predicting HFD in pregnant women with diabetes.

AUTHOR CONTRIBUTIONS

Eriko Eto: conceptualization; data curation, formal analysis, investigation; methodology; project administration, supervision, writing, review, and editing. Masakazu Kato, Satoe Kirino, Chiaki Kuriyama, Syujiro Sakata, Hikari Nakato, Sakurako Mishima, Akiko Ohira: conceptualization; investigation. Hisashi Masuyama: conceptualization; project administration, supervision.

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DISCLOSURE

The authors declare no conflict of interest.

Approval of the research protocol: The study design was approved by the appropriate ethics review board; the Ethics Committee of Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences and Okayama University Hospital (approval no. 1806-009 and approval date of registry, June 15, 2018).

Informed consent: All study participants provided informed consent.

Registry and the registration no. of the study/trial: N/A.

Animal studies: N/A.

ETHICS STATEMENT

This study was conducted according to the guidelines of the Declaration of Helsinki. All procedures involving human subjects/patients were approved by the Ethics Committee of Okayama University Graduate School of Medicine, Dentistry, and Pharmaceutical Sciences and Okayama University Hospital (approval no., 1806-009).

CONSENT TO PARTICIPATE

Written informed consent was obtained from all the patients.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1 | Logistic regression analysis of the association between Δ FM and HFD in women with GDM.

Table S2 | Results of fivefold cross-validation for predictive models of HFD infants.