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Associations between maternal anthropometric characteristics and infant birth weight in Iranian population

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Abstract

Objective: To examine the (1) normal ranges of anthropometric and insulin resistance/sensitivity indices (homeostatic model assessment for insulin resistance, homeostatic model assessment for insulin sensitivity, and quantitative insulin sensitivity check index) for Iranian pregnant women and their newborns and (2) associations between maternal anthropometric and metabolic values and infants' birth weights among Iranian women.

Methods: Anthropometric and metabolic values of 163 singleton non-diabetic pregnant women in Tehran, Iran (2014) were collected before and during pregnancy and at delivery. Linear regression, multivariable regression, and Student *t* tests were used to evaluate correlations between birth weight and maternal variables.

Results: Linear regression modeling suggested that maternal serum glucose ($p = 0.2777$) and age ($p = 0.6752$) were not associated with birth weight. Meanwhile, maternal weight and body mass index before pregnancy ($p = 0.0006$ and 0.0204, respectively), weight at delivery ($p = 0.0036$), maternal height ($p = 0.0118$), and gestational age ($p = 0.0016$) were positively associated with birth weight, while serum insulin ($p = 0.0300$) and homeostatic model assessment for insulin resistance ($p = 0.0334$) were negatively associated with infant's birth weight. Using multivariate modeling, we identified severalconfounders: parity (multipara mothers delivered heavier babies compared to first-time mothers) explained as much as 24% of variation in birth weight ($p = 0.005$), maternal height explained 20.7% ($p = 0.014$), gestational age accounted for 19.7% ($p = 0.027$), and maternal body mass index explained 19.1% ($p = 0.023$) of the variation in the infant's birth weight. Maternal serum insulin and infant's sex were not observed to be associated with birth weight ($p = 0.342$ and 0.669, respectively) in the overall model.

Conclusion: Overweight/obese women may experience higher incidence of delivering larger babies. Multivariable regression analyses showed that maternal body mass index and height, parity, and gestational age are associated with newborn's birth weight.

Keywords

Neonates, birth weight, body mass index, pregnancy, low/high birth weight, homeostatic model assessment

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Introduction

Birth weight (BW) is one of the main metrics used by health care providers to measure fetal well-being and to predict future adult health. An infant's BW is strongly associated with the infant's risk of mortality, developmental and growth problems during childhood, and various diseases in adulthood.[1] For instance, infants born under 2500 g (termed low birth weight (LBW)) have an increased risk of low oxygen levels at birth, inability to maintain body temperature, difficulty feeding and gaining weight, and infection.[2] In adulthood, LBW infants have increased risk of developing

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hypertension and diabetes, and of having deficits in academic and professional achievement compared to adults with normal BWs.[3] Similarly, full term infants born at or above 4000 g (termed high birth weight (HBW)) have higher risks of complications, including higher risks of childhood and adulthood obesity, insulin resistance, heart disease, and cancer.[4]

Maternal characteristics, such as a woman's genetic makeup, socioeconomic status, demographic information, and behavioral habits, are often cited as predictors of BW.[5] For example, studies have shown that mothers who are older, who work, and/or who have lower educational levels tend to deliver LBW babies.[6] In addition, mothers who are obese or have gestational diabetes are at an increased risk of having HBW infants because the excess glucose in their serum travels across the placenta to the growing fetus.[7,8] Aside from infant complications and risks due to HBW, mothers with HBW infants also suffer harms such as having higher risk of cesarean delivery (cesarean delivery rates are twice as high for fetal weights greater than 4500 g) and of shoulder dystocia, the event where the maternal pelvis obstructs the delivery of the baby's shoulder, possibly resulting in the tearing of vagina or uterine rupture, heavy bleeding after birth, and in rare cases, an infant's paralysis.[1,9]

Given that some maternal characteristics are modifiable or treatable, identifying key maternal risk markers before and during a pregnancy is critical to ensure both maternal and infant's health. Some women who are considered at risk of having complicated pregnancies, such as obese women or those with gestational diabetes, are often monitored carefully and provided with resources and treatments early on in a pregnancy. However, this leads to a gap where women who may also be at risk of having complicated pregnancies are not monitored because their metabolic measurements do not reach the threshold of disease diagnosis. Most commonly, mother's serum glucose levels are measured during pregnancy because untreated diabetes diagnosis has been shown to lead to higher rates of infant morbidity and mortality. In contrast, related metabolic measures such as serum insulin, insulin sensitivity, and insulin resistance are not commonly measured, largely due to the unknown effects of these variables on the newborn's well-being and lack of established reference ranges for the latter two variables.[10,11]

Consequently, this study investigated these associations and contributed to the current body of literature by (1) providing values for maternal and neonatal anthropometric and metabolic characteristics for healthy Iranian women and their full term newborns and (2) assessing different maternal characteristics that may be used to predict full term baby's BW. In particular, since few studies report normal ranges for the insulin sensitivity and resistance markers like homeostatic model assessment for insulin resistance (HOMA2-IR), homeostatic model assessment for insulin sensitivity (HOMA2-S), and quantitative insulin sensitivity check index (QUICKI), we report the normal ranges of these markers for future clinical reference.

Methods

This longitudinal, observational study was carried out in Tehran, Iran from April to November 2014. A cohort of 178 pregnant women was followed from 24 to 28 weeks' pregnancy to the time of delivery. Only single birth pregnancies were considered to control for newborn weight differences arising from multiple births. The study sampling technique was recruiting from expecting mothers who were clients to Taleghani Hospital. Mothers with history of diabetes, gestational diabetes, hypertension, and other diseases that can affect serum glucose levels were excluded from the study. Participants were informed of the study's objectives and procedures, and subsequently completed consent forms acknowledging that their participation was voluntary. The sample size for the study was determined by the formula $n=(Z\alpha/2)^2\times\sigma^2/E^2$, where the confidence level was set at 95% (so Zα equaled 1.96), the margin of error (E) was 8% (a larger E was chosen due to limited financial resources), and standard deviation (σ) was 0.5. Based on this formula, at least 150 participant mothers were needed. Medical Research Ethics Committee of the Research Institute for Endocrine Sciences of the Shahid Beheshti University reviewed and approved this study's ethical standards and protocol (36EC-RIES-92/07/23).

Data collection

A trained midwife recorded maternal characteristics throughout each participant's pregnancy, including the type of delivery (cesarean or vaginal), parity, mother's age, height, and weight before pregnancy (i.e. the most recent documented weight in the participant's medical record prior to pregnancy), weight at delivery time (right before delivering the baby), and weight gain during pregnancy. Data on the anthropometric measurements of newborns (i.e. serum glucose level, serum insulin level, gestational age, sex, and BW) were recorded within the first 24 h after birth. Between 24 and 28 weeks of gestation, the O'Sullivan glucose tolerance test was performed on all pregnant women to screen for gestational diabetes. Women with positive glucose tests ($n=2$, 1.1%) or missing BWs for their babies ($n=13$, 7.3%) were excluded from the study sample. Consequently, this study was performed on 163 non-diabetic, healthy mothers and their full term newborns.

Assays

Serum samples for glucose and insulin measurements were collected from fasting mothers and their newborns at the time of delivery. Newborn's serum was obtained through puncture of the umbilical cord artery. Samples were placed in serum separator anticoagulant-free tubes (SST) II with separation gels and immediately sent to the laboratory, where they were centrifuged at 3500 r/min for 5 min to obtain

serum. Serum aliquots were then made and frozen at −70°C until they were ready for testing. The glucose hexokinase method was used to measure serum glucose concentrations and the electro-chemi-luminescence immunoassay (ELICIA) was used to determine insulin concentration.

Although measuring of c-peptide level is a good proxy to determine insulin levels, we did not measure c-peptide levels due to the test not being readily available at the time in our facility.

Statistical analysis

Descriptive statistics (i.e. mean, standard deviation (SD), and range) were calculated and recorded for all quantitative maternal and neonatal variables, and percentages with counts were calculated for all categorical variables (i.e. gender and parity). The frequency distribution of BWs was graphed on a histogram to verify that data were approximately normally distributed. Indices of insulin resistance and insulin sensitivity, measured by HOMA2-IR and HOMA2-S, respectively, were calculated by the program HOMA2 calculator v2.2.3 using the values obtained from the fasting blood samples.[12] HOMA2 is an updated computer model of HOMA that was created to account for variations between hepatic and peripheral insulin sensitivity, increases in secretion of insulin, or decreases in production of hepatic glucose when plasma glucose concentrations are above 180 mg/dL, renal losses of glucose, or the effects of circulating proinsulin. Insulin sensitivity is the reciprocal of insulin resistance.[12]

QUICKI value, another index for insulin sensitivity, was calculated by the formula 1/[(log insulin)(mIU/L)+(log glucose)(mg/dL)].[11,13] Due to some unreported laboratory values or clinical measurements, the counts for each variable were recorded separately.

Associations between each of the following maternal variables and newborn BW were explored using both linear and polynomial regression analyses: age, parity, body mass index (BMI) (before pregnancy), weight before pregnancy, weight at delivery, weight gain during pregnancy, height, serum glucose level, maternal serum insulin level, HOMA2-S value, HOMA2-IR value, and QUICKI value, as well as infant's sex and gestational age at delivery time. Linear regression equations and their respective coefficients were recorded in the form $E(\text{BW}) = \hat{b}_0 + \hat{b}_1$ [variable], where $E(\text{BW})$ is the estimated BW, $\hat{b}_0$ is a constant, and $\hat{b}_1$ is the slope. $\hat{\beta}_1$, the standardized regression coefficient, was also calculated to allow inter-variable comparisons of coefficients. The magnitude and signs of regression models' correlation coefficients were also computed to understand the strength and direction of each predictor variable in estimating BW. Since polynomial regression analyses yielded similar correlation coefficients, only the linear regression equations are reported in this article. The F test was used to test for the statistical significance of the slope coefficients under the null hypothesis $\hat{b}_1 = 0$.

To further explore possible relationships between maternal anthropometric and metabolic characteristics and newborn's BW, several maternal characteristics belonging to the lowest (Q1) and highest quartiles (Q4) of infant's BW were compared using a two-sided, two-sample Student's t test (Table 3). Also, infants' BWs related to Q1 and Q4 of maternal characteristics were compared (Table 4).

To adjust for several confounders and to identify which independent variable explains the greatest variance in the infant BW, a multivariable linear regression analysis was performed including six variables ($n = 125$). These variables had shown correlations with BW in the simple linear regression analyses. The multicollinearities of the independent variables were checked to prevent redundancy. Since maternal serum insulin levels and HOMA2-IR values showed a strong correlation (Pearson correlation of 0.995, $p < 0.001$), the HOMA2-IR variable was removed from the model. The final model included maternal serum insulin, BMI before pregnancy, parity (coded categorically), maternal height, infant's sex, and gestational age (Table 5).

All analyses were computed using STATA version 13.1. Statistical significance was set at $p < 0.05$.

Results

The final study sample size was 163 women and their infants. Their anthropometric and metabolic values are recorded in Table 1. The average age of women was 28.0 years and the average pre-pregnancy BMI was 23.7 kg/m^2, which falls under the normal BMI weight range. The majority of the newborns were male ($n = 89$, 54.6%) and slightly more than half of the births were for women's first children ($n = 89$, 54.6%). While the mean glucose values of mothers and newborns were similar, infants had lower mean insulin levels than mothers. The mean BW was 3227.6 g (standard deviation: 397.1 g). Five newborns (3.1%) were HBW (i.e. $\geq$4000 g) and three newborns (1.8%) were LBW (i.e. <2500 g).

In the sample, 5% ($n = 8$) of mothers were underweight (defined as BMI < 18.5), 61% ($n = 95$) were normal weight ($18.5 \leq \text{BMI} \leq 24.9$), 25% ($n = 40$) were overweight ($25 \leq \text{BMI} \leq 29.9$), and 8% ($n = 13$) were obese (BMI $\geq$ 30). All underweight mothers, 96% ($n = 91$) of normal-weight mothers, 95% ($n = 38$) of overweight mothers, and 85% of obese mothers gave birth to normal BW newborns. Meanwhile, 2% ($n = 2$) of normal-weight mothers, 3% ($n = 1$) of overweight mothers, and 15% ($n = 2$) of obese mothers gave birth to HBW newborns, and 2% ($n = 2$) and 8% ($n = 1$) of obese mothers gave birth to LBW newborns.

Student's t test showed the average BW for girls (3158.0 g) was lower when compared to boys (3285.5 g) ($p = 0.0408$). However, infant's sex showed no significant differences in determining BW in the multivariable model.

Table 2 shows the values of the coefficients of linear regression lines that estimate BW based on maternal variables. Some maternal variables—BMI before pregnancy,

Table 1. Metabolic and anthropometric values of healthy mothers and full term newborns.

Variable	*n*	Mean ± SD	Percentile				
			5th	25th	50th	75th	95th
Mothers							
Age (years)	162	28.0 ± 4.3	23.0	25.0	28.0	31.0	35.0
BMI before pregnancy (kg/m^2)	156	23.7 ± 3.7	18.0	21.2	23.5	25.9	31.0
Weight before pregnancy (kg)	155	62.1 ± 10.0	46.0	55.0	62.0	68.0	80.0
Weight at delivery (kg)	162	75.0 ± 10.8	57.0	68.0	74.0	81.0	96.0
Weight gain during pregnancy (kg)	155	13.1 ± 5.2	6.5	10.0	13.0	15.0	22.0
Height (cm)	158	162.3 ± 6.0	152.0	160.0	162.0	165.0	172.0
Glucose (mg/dL)	126	84.9 ± 24.7	47.0	68.0	82.5	96.0	134.0
Insulin (mIU/L)	135	16.6 ± 14.4	2.0	7.6	12.0	20.9	47.1
HOMA2-IR	125	0.64 ± 0.52	0.13	0.29	0.46	0.81	1.78
HOMA2-S (%)	125	309.6 ± 445.8	56.3	124.1	219.7	347.2	792.1
QUICKI	125	0.34 ± 0.05	0.27	0.30	0.33	0.36	0.41
Newborns							
BW (g)	163	3227.6 ± 397.1	2600	2950	3230	3500	3900
Glucose (mg/dL)	133	85.9 ± 38.6	40.0	64.0	78.0	96.0	167.0
Insulin (mIU/L)	133	8.7 ± 8.4	1.7	3.7	5.6	10.0	27.9
HOMA2-IR	125	0.64 ± 0.52	0.13	0.29	0.46	0.81	1.78
HOMA2-S (%)	129	691.4 ± 1159.1	83.9	265.8	479.0	750.6	1556
QUICKI	129	0.38 ± 0.07	0.29	0.34	0.38	0.42	0.49

HOMA2-S: homeostatic model assessment for insulin sensitivity; HOMA2-IR: homeostatic model assessment for insulin resistance; QUICKI: quantitative insulin sensitivity check index; SD: standard deviation; BMI: body mass index; BW: birth weight.

Table 2. Linear regression analyses showing the effects of maternal metabolic and anthropometric characteristics on newborn's BW.

Maternal variable	b_0	b_1	β_1	*R*	*p* value
Age (years)	3149.46	2.99	0.03	0.033	0.6752
BMI before pregnancy (kg/m^2)	2757.91	19.67	0.19	0.185	0.0204*
Weight before pregnancy (kg)	2544.41	10.91	0.27	0.273	0.0006*
Weight at delivery (kg)	2597.34	8.41	0.23	0.228	0.0036*
Weight gain during pregnancy (kg)	3257.01	−2.69	−0.03	−0.035	0.6663
Height (cm)	1080.95	13.19	0.20	0.200	0.0118*
Glucose (mg/dL)	3110.71	1.62	0.10	0.097	0.2777
Insulin (mIU/L)	3334.23	−5.34	−0.19	−0.187	0.0300*
HOMA2-IR	3339.37	−148.84	−0.19	−0.191	0.0334*
HOMA2-S (%)	3216.07	0.09	0.10	0.100	0.2679
QUICKI	2902.48	1011.47	0.12	0.124	0.1679
Parity	0.82	0.0003	0.116	0.116	0.1390
Gestational age (week)	36.58	0.0007	0.246	0.246	0.0015*

HOMA2-S: homeostatic model assessment for insulin sensitivity; HOMA2-IR: homeostatic model assessment for insulin resistance; QUICKI: quantitative insulin sensitivity check index; BMI: body mass index; BW: birth weight; β_1 : standardized regression coefficient.
Formula: $E(BW) = b_0 + b_1$ [variable].
p values were calculated using the *F* test for the null hypothesis that the slope (b_0) was 0 (i.e. H_0: $b_1 = 0$).
*Significance was set at alpha = 0.05.

weight before pregnancy, weight at delivery, height, and gestational age—as well as being male or born to multipara mothers showed positive association with BW, while maternal serum insulin levels and HOMA2-IR results were negatively associated with newborn's BW.

When maternal glucose levels, serum insulin levels, insulin sensitivity and resistance markers, BMI before pregnancy, weight before pregnancy and at delivery, age, parity, and height belonging to Q1 and Q4 of newborn BW were compared, all maternal variables except glucose, parity, height, and age showed significant differences in values between Q1 and Q4 (Table 3).

Similarly, as Table 4 shows, the BWs of newborns belonging to the Q1 and Q4 of different maternal characteristics

Table 3. Comparisons between the maternal characteristics (serum glucose and insulin levels, insulin sensitivity and resistance markers, BMI before pregnancy, and weight before/at pregnancy, age, parity, and height) belonging to Q_1 and Q_4 of infant BW.

Maternal variable for BW Q1/Q4	*n*	Mean maternal variable	*p* value
Glucose (mg/dL)			
Q1	125	83.68	0.4426
Q4		88.71	
Insulin (mIU/L)			
Q1	134	20.25	0.0230*
Q4		12.88	
Parity			
Q1	162	1.48	0.0913
Q4		1.8	
HOMA2-S (%)			
Q1	122	186.79	0.0163*
Q4		291.15	
HOMA2-IR			
Q1	122	0.85	0.0116*
Q4		0.51	
QUICKI			
Q1	122	0.32	0.0459*
Q4		0.34	
BMI before pregnancy (kg/m²)			
Q1	155	22.74	0.0349*
Q4		24.73	
Weight before pregnancy (kg)			
Q1	155	58.31	0.0039*
Q4		65.50	
Weight at delivery (kg)			
Q1	162	71.08	0.0049*
Q4		78.11	
Maternal height (cm)			
Q1	158	161.46	0.1511
Q4		163.37	
Maternal age (years)			
Q1	162	27.46	0.6949
Q4		27.8	

HOMA2-S: homeostatic model assessment for insulin sensitivity; HOMA2-IR: homeostatic model assessment for insulin resistance; QUICKI: quantitative insulin sensitivity check index; BMI: body mass index; BW: birth weight; Q1: lowest quartile; Q4: highest quartile.
p value was calculated using a two-sided Student's *t* test for the null hypothesis that the difference between the associated maternal characteristic for two groups of BW Q1 and Q4 is 0.
*Significance was set at alpha = 0.05.

Table 4. Comparisons between BWs belonging to Q1 and Q4 of mother's serum glucose levels, serum insulin levels, insulin sensitivity and resistance markers, BMI before pregnancy, and weight before/at pregnancy.

Q1/Q4 maternal variable	*n*	Mean BW ± SE (g)	*p* value
Glucose (mg/dL)	126		
Q1		3210.32 ± 64.23	0.2331
Q4		3338.29 ± 82.24	
Insulin (mIU/L)	134		
Q1		3364.55 ± 79.89	0.0403*
Q4		3152.65 ± 62.74	
HOMA2-IR	123		
Q1		3352.26 ± 80.51	0.0693
Q4		3159.38 ± 66.77	
HOMA2-S (%)	123		
Q1		3156.45 ± 68.89	0.0773
Q4		3344.38 ± 78.35	
QUICKI	123		
Q1		3174.84 ± 70.11	0.1954
Q4		3311.88 ± 77.48	
BMI before pregnancy (kg/m²)	156		
Q1		3122.75 ± 263.80	0.0346*
Q4		3297.63 ± 361.56	
Weight before pregnancy (kg)	156		
Q1		3062.05 ± 251.24	0.0090*
Q4		3354.00 ± 319.30	
Weight at delivery (kg)	162		
Q1		3044.63 ± 240.29	0.0038*
Q4		3304.39 ± 336.57	

HOMA2-S: homeostatic model assessment for insulin sensitivity; HOMA2-IR: homeostatic model assessment for insulin resistance; QUICKI: quantitative insulin sensitivity check index; BMI: body mass index; BW: birth weight; Q1: lowest quartile; Q4: highest quartile; SE: standard error of mean.
p value was calculated using a two-sided Student's *t* test for the null hypothesis that the difference between the associated BWs for the maternal glucose of Q1 group and maternal glucose of Q4 group is 0.
*Significance was set at alpha = 0.05.

were compared. Both comparisons suggested that lower serum insulin levels in mothers was associated with heavier babies; the BWs of infants delivered by mothers whose serum insulin levels were in Q1 were higher than the BWs of infants delivered by mothers whose serum insulin levels were in Q4 ($p = 0.040$). This observation was consistent with the insulin model's negative coefficient in regression analyses. Also, BW of babies born to mothers from Q1of weight before pregnancy showed statistically significant lower mean values when compared to babies born to mothers from Q4 of weight before pregnancy ($p = 0.0090$). The same appeared for weight at delivery ($p = 0.0038$) and BMI before pregnancy ($p = 0.0346$). Meanwhile, no significant differences in BWs of babies delivered by women whose serum glucose levels ($p = 0.2331$), HOMA2-IR ($p = 0.0693$), HOMA2-S ($p = 0.0773$), and QUICKI ($p = 0.1954$) were in Q1and Q4were found.

Maternal serum insulin and glucose levels in our sample were positively correlated ($Y = 0.1803X + 1.6771$, $R = 0.3209$, $p = 0.0002$).

To find the average BMI (per kg/m² and its SD) which increases the chance of delivering babies with normal BW (from 2500 to less than 4000 g) the statistical difference between the average BMI for mothers with low BW (23.60 ± 2.41) or high BW (27.29 ± 2.92) babies were compared

Table 5. Multiple regression analysis of infant BW and its correlations with maternal serum insulin, BMI before pregnancy, maternal height, parity and infant's sex, and gestational age. ($n = 125$).

Covariate	Unstandardized coefficients (B)	Standardized coefficients (β)	*p* value	95% CI[a] for *B*	
				Lower bound	Upper bound
Maternal serum insulin (mIU/L)	−2.531	−0.084	0.342	−7.781	2.720
Maternal BMI before pregnancy (kg/m^2)	21.203	0.191	0.023*	2.973	39.432
Parity (primipara/multipara)	197.023	0.240	0.005*	60.93	333.11
Sex	−30.030	−0.037	0.669	−168.76	108.70
Maternal height (cm)	14.188	0.207	0.014*	2.928	25.45
Gestational age (weeks)	66.808	0.197	0.027*	7.810	125.806

CI: confidence interval; BW: birth weight; BMI: body mass index.
*Significance was set at alpha = 0.05.

to the average BMI for mothers of average BW (23.61 ± 2.91) babies using Student's *t* test. There was a statistically significant difference between the average BMI of mothers with high BW babies compared to mothers with average BW babies ($p = 0.0362$). The statistical difference between BMI of mothers with low BW compared to average BW babies did not reach to significance ($p = 0.8070$) maybe due to very small number of low BW babies in the sample population.

Babies born to primipara (3170.1 g) mothers had significantly lower average BW compared to those born to multipara mothers (3296.8 g, $p = 0.0423$). Although number of parity did not show an association with infant BW in our simple linear regression model ($Y = 0.0003X + 0.819$, $R = 0.116$, $p = 0.139$), in the multiple regression model (where parity was coded into two groups: primipara and multipara), parity counts for 24% (standardized coefficient beta) of variation in the BW ($p = 0.005$).

Maternal HOMA2-IR value ($p = 0.0334$) and infant's gestational age ($p = 0.0016$) were associated with infant BW. Although the linear regression models did not show HOMA2-S ($p = 0.2679$) and QUICKI ($p = 0.1679$) to be associated with infant BW, these variables were significantly associated when BW was divided into Q1 and Q4 (Table 3).

The unstandardized coefficient (*B*) predicts how much BW will change for every unit change in the corresponding independent variable. For instance, for the maternal BMI with $B = 21.203$, each unit increase in the mother's BMI, will result in 21.203 g increase in the infant's BW (Table 5).

In the multivariable model, parity (multipara mothers delivered heavier babies compared to primipara) explains as much as 24% of variation in BW ($p = 0.005$), maternal height explains 20.7% ($p = 0.014$), gestational age accounts for 19.7% ($p = 0.027$), and maternal BMI explains 19.1% ($p = 0.023$) of the variation in infant BW. Maternal serum insulin and infant's sex did not show significance in predicting BW in the model ($p = 0.342$ and 0.669, respectively).

In summary, the multivariable model had R-square value of 0.213 and adjusted R^2 value of 0.173, meaning 17.3% of BW variability could be explained by this model. The *p* value for the *F* test for the total model was <0.001, indicating that the model had a strong prediction power.

Discussion

Previous studies cite that several factors influence a newborn's BW: an infant's gestational age, gender, and his or her intrauterine growth rate. LBW is caused by premature birth and/or restricted intrauterine growth while HBW is caused by excess fuel delivery from the mother to the fetus.[2,14] To investigate associations between maternal anthropometric characteristics and newborn's BW, we only looked at full term (>37 weeks), single birth infants to eliminate the confounding effects of short gestational period and multiple birth pregnancies on BWs. In this study, we report the mean values of anthropometric characteristics of non-diabetic Iranian women and their full term infants, as well as their respective HOMA2-IR, HOMA2-S, and QUICKI ranges, which very few neonatal studies have reported to date. Additionally, we provide evidence to support that higher maternal BMI before pregnancy, weight before pregnancy, weight at delivery, height, gestational age, being male, and being born to a multipara mother are associated with higher newborn BW while higher maternal serum insulin levels and HOMA2-IR values are associated with lower newborn BW.

Our findings are consistent with other studies. Our average BW of 3228 g is comparable to the values reported in southern Iran (3060 g), Spain (3304 g) and the United States (3389 g), although ethnic and genetic differences may explain why the Iranian newborns have slightly lower BWs.[11,15,16] Furthermore, multiple studies have found positive associations between infant BW and BMI before pregnancy, maternal weight before pregnancy and at delivery, and maternal height.[5,17,18] A study by Breschi et al.[17] also found an inverse relationship between BW and maternal insulin level and a 2008 study of over 3000 women found that lower maternal insulin secretion was related to greater adiposity at birth, which gets translated to higher BW.[19] However, some studies have found no significant association between the two variables.[20] These discrepant findings

between maternal insulin levels and BW may be a consequence of different sample characteristics since these studies only looked at women with particular conditions such as gestational diabetes, hypertension, or BMI >25 kg/m^2. To our best knowledge, no studies have looked at associations between BW and maternal HOMA2-IR, HOMA2-S, and QUICKI values thus far. However, most studies have used HOMA-IR instead to find correlations with BW and have reported conflicting findings, with some reporting that higher HOMA-IR is associated with higher incidence of HBW newborns while others found no relation between the two variables.[10,21] Again, these different findings may be due to different samplings among studies since some include non-full term infants and diabetic mothers. Nevertheless, our study contributes to the existing literature by further supporting the hypotheses that several maternal variables are associated with BW.

If a woman who is planning to become pregnant is overweight or obese, she is more likely to have a HBW newborn.[22] Given the complications and risks of having LBW or HBW newborns, we recommend future research to examine whether weight loss before pregnancy reduces the incidence of HBW babies. This is especially important for smaller-framed women whose babies may suffer shoulder dystocia alongside their maternal chorioamnionitis, postpartum hemorrhage, and longer hospital stays when delivering HBW infants.[23] Furthermore, on the basis of this study's linear regression model, we hypothesize that non-diabetic women who have high insulin levels at the time of delivery may have a higher risk of giving birth to smaller size newborns.

Although our multivariable model did not show an association between the maternal serum insulin and BW, we found a negative association between the two variables both in linear regression modeling and *t* tests comparing BW Q1and Q4. A possible explanation for our observation that higher maternal insulin levels and HOMA2-IR values at the time of delivery are associated with lower BW may be that higher insulin levels in non-diabetic mothers may be considered below a threshold of insulin resistance, consequently, lead to lower serum glucose levels, leading to less glucose delivery to the fetus and hence lower BW. Possibly, above this threshold, cells may resist taking up glucose. This will lead to an increase in the maternal plasma glucose levels, hence providing the fetus with extra glucose to grow.

This study had several limitations. First, we did not collect data on maternal socioeconomic factors such as income and education, which studies have shown to affect BW.[24] Second, we did not collect information on mothers' dietary and behavioral habits, which also influence BW; studies have shown that mothers who smoke are nearly twice as likely to give birth to LBW infants compared to non-smoking mothers.[25] Since multiple regression analyses demand complete data files for all variables simultaneously, the sample size was reduced to 125 for that method. Finally, since we had much fewer HBW infants in the sample when compared to normal weight infants, our finding of higher maternal BMI among HBW infants may change if more women with HBW infants were included in the sample.

Finally, because of the observational nature of our study, we cannot conclude causal relationships although such relationships may be plausible for some of our findings. Similarly, we cannot extrapolate our regression equations outside the range of maternal variables used to build the model since values outside of this range may follow different patterns for estimating BW.

We recommend that future studies measure socioeconomic factors, mothers' diets, and mothers' smoking habits, data that we were unable to collect due to pragmatic and financial reasons, so that these variables are incorporated into regression models. Using a larger sample size will also increase our confidence in the associations found in our study: larger sample size may allow us to detect statistically significant predictors of BW that have a small influence in changing BW or, alternately, distinguished variables that may be predictors of BW but did not reach our alpha threshold for significance.

In conclusion, overweight/obese women may experience a higher incidence of delivering larger babies. The multivariable linear regression analysis model showed that maternal BMI and height, parity, and gestational age are positively associated with newborn's BW.

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Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics approval

The Medical Research Ethics Committee of Research Institute for Endocrine Sciences (RIES) Shahid Beheshti University reviewed and approved this study's ethical standards and protocol. The reference approval letter number is 36EC-RIES-92/07/23.

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Informed consent

All participants were informed of the study's objectives and procedures and subsequently completed and signed consent forms acknowledging that their participation was voluntary.

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