The Association Between Maternal Glucose Concentration and Child BMI at Age 3 Years

ANDREA L. DEIERLEIN, PHD, MPH[1]
ANNA MARIA SIEGA-RIZ, PHD, RD[2,3,4]

KIM CHANTALA, MS[5]
AMY H. HERRING, SCD[4,6]

OBJECTIVE—The objective of the study was to determine the association between child BMI at age 3 years and maternal glucose concentration among women without pre-existing diabetes or a gestational diabetes mellitus (GDM) diagnosis.

RESEARCH DESIGN AND METHODS—Data are from the Pregnancy Infection and Nutrition and Postpartum studies and include 263 mother-child pairs. Measured weights and heights at 3 years were used to calculate age- and sex-specific BMI z scores and percentiles. Multivariable linear regression models were used to examine associations of continuous BMI z scores with maternal glucose concentration. Modified Poisson regression estimated risk ratios of child overweight/obesity (BMI ≥85th percentile).

RESULTS—The mean (SD) maternal glucose concentration and prepregnancy BMI were 103.8 (23.7) mg/dL and 24.3 (5.9) kg/m^2, respectively. At 3 years, the mean (SD) child BMI z score was 0.29 (0.99), 20.9% were overweight/obese and 5.3% were obese. In the adjusted model, when compared with glucose concentration <100 mg/dL, a concentration ≥130 mg/dL was associated with significantly higher child BMI z score at 3 years (estimated z score difference of 0.39 [95% CI: 0.03–0.75]). With the use of the same reference category, a concentration ≥130 mg/dL was associated with an approximate twofold greater risk of child overweight/obesity (adjusted risk ratio 2.34 [95% CI: 1.25–4.38]).

CONCLUSIONS—Fetal exposure to high maternal glucose concentration in the absence of pre-existing diabetes or GDM may contribute to the development of overweight/obesity in the offspring, independent of maternal prepregnancy BMI.

Diabetes Care 34:480–484, 2011

P regnancies complicated by gestational diabetes mellitus (GDM) are associated with several adverse outcomes in the offspring including macrosomia and the development of obesity and type 2 diabetes later in life (1). Similar observations for early infant outcomes have been made for fetal exposure to maternal hyperglycemia without a GDM diagnosis. In the multinational, multicenter Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study, positive associations were found for high birth weight, fetal hyperinsulinemia (measured by cord serum C-peptide), and neonatal adiposity with increasing levels of maternal glucose tolerance (2,3). Fewer studies are available for associations of offspring outcomes later in life. Higher relative weights in Pima Indian offspring at ages 5–24 years (4) and increased risk of child obesity at 5–7 years in a multiethnic U.S. population (5) were reported across increasing levels of maternal glucose concentration; however, a recent analysis of data from the Belfast, U.K. center of the HAPO study found no association between maternal glucose and child obesity at 2 years (6). Discrepancies in these findings may be due to differences in the study populations, methodology, and/or ages of the offspring at the time of outcome assessment.

High maternal glucose concentration during pregnancy is a modifiable and preventable behavioral factor that may represent an early life determinant of pediatric obesity and its related comorbidities. The purpose of the current analysis was to determine the association between child BMI at age 3 years and maternal glucose concentration among women without pre-existing diabetes or a GDM diagnosis. Our results are intended to contribute to the limited amount of literature examining this association, especially for offspring anthropometric outcomes beyond the neonatal and infancy periods. This research is useful to inform prenatal intervention strategies, since there is some evidence that treatment of hyperglycemia during pregnancy attenuates adverse offspring outcomes such as macrosomia (7) and possibly later childhood obesity (5).

RESEARCH DESIGN AND METHODS

—Subjects were recruited from the third cohort of the Pregnancy Infection and Nutrition study (PIN), January 1, 2001 through June 30, 2005, and followed through 3 years postpartum by the PIN Postpartum (3 and 12 months postpartum) and PIN Kids (3 years postpartum) studies. The recruitment protocols for the studies were documented previously (8). Briefly, pregnant women who were at least 16 years of age at conception, English speaking, before 20 weeks' gestation at recruitment, and receiving prenatal care from public and private clinics at the University of North Carolina (UNC) Hospitals were eligible for participation. The PIN study protocols were approved by the Institutional

Review Boards of the School of Medicine at UNC Chapel Hill.

A total of 1,169 women in PIN delivered a live singleton infant and were eligible for recruitment into the postpartum study. There were 689 and 550 mother-child pairs who participated at 3 and 12 months postpartum, respectively. The most common reasons for nonparticipation included refusal/request to leave study ($n = 202$), unreachable/moved out of study catchment area ($n = 226$), and ineligibility ($n = 338$) because of medical constraints, timing issues, or pregnancy. Recruitment for PIN Kids began in 2004 and added an assessment of the index infant at 3 years. There were 409 mother-child pairs who completed the 3-year visit. Three children were not eligible for inclusion because a physician diagnosed growth-related illness. Anthropometric measurements were missing for 81 children, mostly because other data were collected by phone interview rather than home visit ($n = 58$) or the child was unavailable, such as napping, during the home interview. We further excluded mothers with pre-existing diabetes ($n = 20$), GDM ($n = 9$), or missing glucose concentration information ($n = 3$). Children born before 37 weeks' gestation ($n = 30$) were also excluded. The remaining 263 mother-child pairs were included in the current analyses.

Distributions of selected baseline characteristics between eligible mother-child pairs who participated in PIN Kids ($n = 406$), PIN Postpartum (at 3 months postpartum) pairs not included in PIN Kids ($n = 283$), and eligible PIN pairs not participating in PIN Postpartum ($n = 480$) were examined. In comparison with mothers in PIN Kids, those not participating in both PIN and PIN Postpartum were significantly younger and had infants with lower mean birth weight and gestational age. They were more likely to be obese, black, unmarried, less educated, and from low income households. Additionally, a higher percentage of PIN Postpartum mothers not participating in PIN Kids smoked during pregnancy. All other comparisons of characteristics were not significant. There were no significant differences in the baseline characteristics of the PIN Kids samples included ($n = 263$) in this analysis and those excluded ($n = 143$) with the exception that excluded women had a slightly higher prepregnancy BMI (mean SD). BMI was 26.3 (7.8) kg/m^2 and 24.3 (5.9) kg/m^2 for

excluded and included women, respectively ($P = 0.004$).

At the 3-year home visit, children's heights and weights were measured by trained PIN staff using stadiometers and scales, respectively, according to National Health and Nutrition Examination Surveys protocols (9). The mean (SD) age of the children at the home visit was 3.04 (0.15) years. Child BMI at 3 years was calculated from these measurements (in kg/m^2) and converted to age- and sex-specific BMI z scores (continuous) and percentiles (categories) using the 2000 Centers for Disease Control and Prevention/National Center for Health Statistics growth charts (10). Overweight/obesity was defined as BMI-for-age and sex ≥85th percentile (reference <85th percentile).

Maternal plasma glucose concentrations were derived from universal screens administered during the second trimester (mean gestational age ~27 weeks) on all women without pre-existing diabetes. The universal screen involved administration of a random 1-h 50-g glucose challenge test (GCT) to the women. Women with abnormal values on the GCT were administered a 3-h oral glucose tolerance test to confirm GDM using Carpenter and Coustan criteria (11). For this analysis, maternal plasma glucose concentrations from the GCT were categorized using the following cut points: <100, 100–<130, and ≥130 mg/dL. These cut points are comparable with those used in a previous study (12), and glucose concentrations ≥130 mg/dL are suggestive of hyperglycemia (11).

Maternal prepregnancy BMI (in kg/m^2) was calculated using self-reported prepregnancy weight and measured height. For quality assurance, weight measurements taken at the first prenatal clinic visit (within 15 weeks' gestation) were compared with the self-reported prepregnancy weights to identify biologically implausible weight gains. Women with implausible values ($n = 7$) had their prepregnancy weights imputed following previously published methods (8). Maternal prenatal smoking (months 1–6 of pregnancy), household income, education, marital status, age at conception, race/ethnicity, pre-existing diabetes, and parity were collected from prenatal interviews and categorized as shown in Table 1. Household income was expressed as percentage of the poverty line and calculated using the 2001 U.S. Department of Health and Human Services Federal Poverty Guidelines; a percentage ≤185% is the

cutoff for the Special Supplemental Nutrition Program for Women, Infants, and Children. Household income at the 3-month postpartum interview was used for missing information from the prenatal period ($n = 7$). Infant sex and birth weight were recorded at delivery and abstracted from the medical records. Gestational age was determined from ultrasound measurements conducted before 22 weeks gestation (up to 21 weeks, 6 days). If no ultrasound was performed or if it was not performed before the start of the 22nd week then the date of the last menstrual period was used ($n = 5$). Birth weight z scores specific for infant sex and gestational age were calculated using U.S. national reference data (13).

Statistical analyses were performed using STATA 11 (College Station, TX). Potential effect measure modifiers, confounders, and mediators of interest were identified a priori from a literature review and causal diagrams (14). The interaction of maternal glucose concentration and prepregnancy BMI was tested in the full models using interaction terms and Wald tests with an a priori significance P value of <0.15. Student t tests, Fisher exact tests, and analyses of variance were used to analyze distributions of baseline characteristics. Multivariable linear regression models were used to examine associations of continuous BMI z scores with maternal glucose concentration. Modified Poisson regression (Poisson regression with a robust error variance) estimated risk ratios of child overweight/obesity (BMI ≥85th percentile). This method has been validated for directly estimating relative risks for dichotomous, common outcomes in prospective studies (15). All regression analyses were adjusted for clustering at the individual level (16) since there were five women with more than one child included in the analyses. The analyses were repeated excluding the second child of these women, and there were no appreciable differences in the results.

RESULTS—The mean (SD) maternal glucose concentration was 103.8 (23.7) mg/dL. The mean (SD) prepregnancy BMI was 24.3 (5.9) kg/m^2. Approximately 30% of the women were overweight (17.6%) or obese (12.2%). The majority of women were 25–34 years at conception, nonblack, married, achieved a high school degree or higher, upper income (>350% of the 2001 Poverty Guidelines), and nonsmokers during pregnancy. Approximately half of them were nulliparous.

Table 1—Baseline distributions of population characteristics according to glucose tolerance and child BMI at 3 years in the PIN Kids Study (n = 263)

Variable	N	Mean BMI z score (SD)	P*
Age at conception (years)			
16–24	47	0.38 (0.93)	
25–29	68	0.37 (1.03)	0.51
30–34	96	0.17 (0.95)	
35–47	52	0.32 (1.07)	
Prepregnancy BMI (kg/m^2)			
Underweight (<18.5)	16	−0.25 (0.65)	
Normal weight (18.5–24.9)	168	0.19 (0.93)	<0.01
Overweight (25.0–29.9)	47	0.56 (0.93)	
Obese (≥30.0)	32	0.73 (1.28)	
Maternal height (inches)			
≤64	107	0.23 (1.06)	0.40
>64	156	0.33 (0.95)	
Race			
Non-Black	238	0.27 (1.00)	0.27
Black	25	0.50 (0.96)	
Marital status			
Married	227	0.23 (0.99)	0.02
Other	36	0.65 (0.94)	
Education			
≤Grade 12	32	0.49 (1.25)	
Grades 13–16	126	0.40 (0.93)	0.04
≥Grade 17	105	0.10 (0.95)	
Family income (% 2001 poverty guidelines)			
<185%	36	0.45 (0.87)	
185–350%	46	0.14 (1.14)	0.37
>350%	181	0.30 (0.97)	
Parity			
Nulliparous	127	0.29 (0.93)	0.95
1 or more births	136	0.29 (1.05)	
Smoking in months 1–6 of pregnancy			
No	240	0.26 (0.97)	0.01
Yes	17	0.89 (1.17)	
Infant sex			
Male	142	0.37 (1.01)	0.17
Female	121	0.20 (0.97)	
Glucose tolerance (mg/dL)			
<100 (range 44–99)	112	0.20 (1.03)	
100–<130 (range 100–129)	112	0.26 (0.94)	0.05
≥130 (range 130–188)	39	0.62 (0.97)	

*P values from ANOVA or Fisher exact test, testing for differences across strata.

The mean (SD) birth weight and gestational age of the children were 3415.8 (413.4) g and 39.2 (1.2) weeks, respectively. At 3 years, the mean (SD) child BMI z score was 0.29 (0.99), 20.9% were overweight/obese (BMI ≥85th percentile), and 5.3% were obese (BMI ≥95th percentile). Table 1 shows the distributions of selected characteristics for the sample (n = 263) according to mean child BMI z score. Mean child BMI z score significantly differed across categories of maternal glucose concentration and prepregnancy BMI, as well as maternal prenatal marital status, education, and smoking.

Table 2 displays the results from linear regression analyses for the association of maternal glucose concentration categories and child BMI z score at 3 years. Each 1 mg/dL increase in maternal glucose concentration was associated with a slight increase in BMI z score at 3 years (estimated difference in z score of 0.005, 95% CI: −0.001, 0.010, P = 0.08 in the adjusted model, data not shown in table). When compared with glucose concentration <100 mg/dL, a concentration ≥130 mg/dL was associated with significantly higher child BMI z score at 3 years in the adjusted model (estimated difference in z score of 0.39, 95% CI: 0.03–0.75). We also examined the association of maternal glucose concentration categories and risk of child overweight/obesity at 3 years (Table 3). In comparison with glucose concentration <100 mg/dL, concentration ≥130 mg/dL was associated with an approximate twofold greater risk of child overweight/obesity (adjusted risk ratio 2.34, 95% CI: 1.25–4.38). Additional adjustment of models for infant birth weight z score did not substantially alter any of the effect estimates. We also did not find an interaction between maternal glucose concentration and prepregnancy BMI; however, because of the small sample size we may have lacked adequate power to detect one if it existed.

CONCLUSIONS—With the use of data from mother-child pairs participating in a recent, prospective, longitudinal cohort study, we found a significant increased risk of child overweight/obesity at 3 years associated with maternal glucose concentration ≥130 mg/dL. These results suggest that fetal exposure to high maternal glucose concentration in the absence of pre-existing diabetes or GDM may contribute to the development of overweight/obesity in the offspring, independent of maternal prepregnancy BMI.

Nearly 60 years ago, Pedersen (17) hypothesized that fetal exposure to maternal hyperglycemia via diabetes resulted in fetal hyperinsulinemia and greater adiposity at birth. Since then both animal and human studies provided evidence to support Pedersen's observations and increased our understanding of the pathophysiology of GDM (18). GDM is associated with several adverse offspring outcomes including macrosomia; large-for-gestational age (LGA); respiratory distress (1); insulin resistance; and greater risk of obesity, type 2 diabetes, and metabolic syndrome later in life (1). There is adequate literature to suggest that the associations for early infant outcomes (macrosomia [19], LGA [12], and adiposity [3]) extend to hyperglycemia during pregnancy in the absence of pre-existing diabetes or GDM. However, there are fewer studies examining associations with later childhood outcomes, specifically obesity.

Consistent with the current study, two studies found positive associations for maternal glucose concentration and

Table 2—Estimated differences in child BMI z score at 3 years associated with categories of maternal glucose concentration in the PIN Kids Study

Glucose concentration	Model 1† (n = 263)		Model 2‡ (n = 256)		Model 3§ (n = 254)	
	β* (95% CI)	P	β (95% CI)	P	β (95% CI)	P
<100 mg/dL	Reference		Reference		Reference	
100–<130 mg/dL	0.05 (−0.21–0.31)	0.70	0.07 (−0.18–0.32)	0.59	0.01 (−0.24–0.26)	0.93
≥130 mg/dL	0.44 (0.08–0.80)	0.02	0.39 (0.03–0.75)	0.03	0.39 (0.02–0.75)	0.04

*Coefficient from linear regression analysis. †Model 1: unadjusted. ‡Model 2: adjusted for maternal education, race, prenatal smoking, prepregnancy BMI, and maternal height. §Model 3: adjusted for Model 2 and birth weight z score.

offspring anthropometric outcomes. Among nondiabetic Pima Indians, Pettitt et al. (4) showed a linear association between maternal glucose concentration (2-h, 75-g oral glucose tolerance test [OGTT]) and offspring relative weights at ages 5–24 years. There was also a higher prevalence of obesity and higher rates of abnormal glucose tolerance in offspring of mothers who had abnormal glucose tolerance during pregnancy. In a multiethnic U.S. population, Hillier et al. (5) reported significant increased odds of child BMI >85th percentile (adjusted odds ratio [aOR]: 1.22, 95% CI: 1.03–1.45) and BMI >95th percentile (aOR: 1.28, 95% CI: 1.02–1.60) at 5–7 years associated with maternal glucose concentration (1-h, 50-g GCT) of 122–140 mg/dL compared with 43–94 mg/dL.

In contrast, using data from the Belfast, U.K. center of the HAPO study, which was designed to clarify the risks of adverse outcomes associated with various degrees of maternal glucose intolerance less severe than that in overt diabetes mellitus (3), Pettitt et al. (6) found no association between maternal glucose concentration (assessed using fasting glucose and 1-h and 2-h OGTT) and child BMI ≥85th and ≥95th percentiles at 2 years. The only exception was a significant increase in prevalence of child BMI ≥85th percentile across strata of maternal 1-h glucose concentration. Similarly, in a nondiabetic homogeneous Caucasian population in Exeter, U.K. (20), despite finding significant correlations between fasting plasma glucose (measured at 28 weeks' gestation) and offspring weight, length, and BMI at birth, none of the estimates remained significant when measurements were repeated at ages 12 weeks, 1 year, and 2 years.

It has been suggested that the effects of maternal glucose concentration are long term and differ depending on the age of the offspring (6). For example, when comparing offspring of women with and without GDM, there are significant differences in weight at birth, which are not apparent at 1.5 years but then recur later in childhood at 7.7 years (21). In this study, children were ~3 years, which is younger than the ages of the populations used in studies that reported positive associations (4,5) but older than those used in studies reporting null findings (6,20). Our results add to the current evidence suggesting that the effects of fetal glucose exposure on the development of child overweight/obesity are apparent at later ages. However, there is a lack of consistency across studies in methodology concerning maternal glucose concentration measurements, study populations, as well as adjustment for potential confounding variables, which makes it difficult to compare results. More research is necessary to understand the influence of maternal glucose concentration on offspring anthropometric development among women without diabetes.

There are several limitations of the current study that must be considered when interpreting the results. A main limitation is the loss to follow-up between birth and 3 years that resulted in a disproportionate loss of women from high risk groups and likely weakened the observed associations. Additionally, the loss in sample size may have diminished the power necessary to detect some associations and interactions, if they existed. The generalizability of our sample may be limited because women were mostly white, well educated, and upper income, resulting in a lower prevalence of overweight and obesity compared with national estimates for women of reproductive ages (22).

Other limitations are due to our measurements of maternal glucose concentration and child anthropometrics. A standard cut-point of 140 mg/dL defined an abnormal value on the universal screen and resulted in further testing for GDM; however, a cut-point of 130 mg/dL is more sensitive and may be considered a better diagnostic tool for GDM (23). Therefore, some of the women with GCT values ≥130 mg/dL but <140 mg/dL (n = 20) may have been missed as GDM cases and included in our analyses. This type of misclassification would likely strengthen the observed effect estimates, especially if these women remained untreated. Child

Table 3—Risk ratios for maternal glucose concentration and child BMI ≥85th percentile at 3 years in the PIN Study

	Model 1* (n = 263)		Model 2† (n = 256)		Model 3‡ (n = 254)	
	Risk ratio (95% CI)	P	Risk ratio (95% CI)	P	Risk ratio (95% CI)	P
Glucose concentration (per mg/dL)	1.01 (1.00–1.02)	0.15	1.01 (1.00–1.02)	0.08	1.01 (1.00–1.02)	0.11
<100 mg/dL	Reference		Reference		Reference	
100–<130 mg/dL	1.00 (0.56–1.78)	1.00	1.04 (0.60–1.81)	0.88	0.98 (0.56–1.71)	0.94
≥130 mg/dL	2.15 (1.21–3.84)	0.01	2.34 (1.25–4.38)	0.01	2.48 (1.27–4.82)	0.01

Risk ratios for maternal glucose concentration are per mg/dL and categories. *Model 1: unadjusted. †Model 2: adjusted for maternal education, race, prenatal smoking, prepregnancy BMI, and maternal height. ‡Model 3: adjusted for Model 2 and birth weight z score.

BMI status is not a direct measure of adiposity but it is correlated ($r = 0.75$) with percent body fat in children aged 3–8 years (24). Finally, it is likely that the observed associations between maternal glucose concentration and child BMI partly reflect the effects of genetic or environmental factors (such as child diet and physical activity) related to a woman's glucose concentration and health behaviors in the postnatal period, which we were unable to consider in our etiologic model.

The results from this study suggest that high maternal glucose concentration (≥130 mg/dL) in the absence of a pre-existing diabetes or GDM diagnosis is a modifiable and preventable behavioral factor associated with offspring overweight/obesity at 3 years. Prenatal intervention efforts may consider monitoring women who meet such criteria as a way to decrease fetal exposure to high maternal glucose concentration and possibly prevent excess weight development. Future studies, such as the HAPO study (2), that collect longitudinal data before pregnancy through late childhood, including comprehensive measures of maternal glucose concentration, child adiposity, and potential confounders, are needed.

References
1. Reece EA. The fetal and maternal consequences of gestational diabetes mellitus. J Matern Fetal Neonatal Med 2010;23:199–203
2. Metzger BE, Lowe LP, Dyer AR, et al.; HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008;358:1991–2002
3. HAPO Study Cooperative Research Group. Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study: associations with neonatal anthropometrics. Diabetes 2009;58:453–459
4. Pettitt DJ, Bennett PH, Saad MF, Charles MA, Nelson RG, Knowler WC. Abnormal glucose tolerance during pregnancy in Pima Indian women. Long-term effects on offspring. Diabetes 1991;40(Suppl. 2):126–130
5. Hillier TA, Pedula KL, Schmidt MM, Mullen JA, Charles MA, Pettitt DJ. Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia. Diabetes Care 2007;30:2287–2292
6. Pettitt DJ, McKenna S, McLaughlin C, Patterson CC, Hadden DR, McCance DR. Maternal glucose at 28 weeks of gestation is not associated with obesity in 2-year-old offspring: the Belfast Hyperglycemia and Adverse Pregnancy Outcome (HAPO) family study. Diabetes Care 2010;33:1219–1223
7. Gillman MW, Oakey H, Baghurst PA, Volkmer RE, Robinson JS, Crowther CA. Effect of treatment of gestational diabetes mellitus on obesity in the next generation. Diabetes Care 2010;33:964–968
8. Mehta UJ, Siega-Riz AM, Herring AH. Effect of body image on pregnancy weight gain. Matern Child Health J. 5 March 2010 [Epub ahead of print]
9. Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). National Health and Nutrition Examination Protocol 1999–2000 [article online]. Available from http://www.cdc.gov/nchs/data/nhanes/bm.pdf. Accessed 19 November 2010
10. Vidmar S, Carlin J, Hesketh K, Cole T. Standardizing anthropometric measures in children and adolescents with new functions for egen. Stata J 2004;4:50–55
11. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. Am J Obstet Gynecol 1982;144:768–773
12. Scholl TO, Sowers MF, Chen X, Lenders C. Maternal glucose concentration influences fetal growth, gestation, and pregnancy complications. Am J Epidemiol 2001;154:514–520
13. Oken E, Kleinman KP, Rich-Edwards J, Gillman MW. A nearly continuous measure of birth weight for gestational age using a United States national reference. BMC Pediatr 2003;3:6
14. Greenland S, Pearl J, Robins JM. Causal diagrams for epidemiologic research. Epidemiology 1999;10:37–48
15. Zou G. A modified poisson regression approach to prospective studies with binary data. Am J Epidemiol 2004;159:702–706
16. Rogers W. Regression standard errors in clustered samples. Stata J 1993;13:19–23
17. Pedersen J. Weight and length at birth of infants of diabetic mothers. Acta Endocrinol (Copenh) 1954;16:330–342
18. Catalano PM, Kirwan JP, Haugel-de Mouzon S, King J. Gestational diabetes and insulin resistance: role in short- and long-term implications for mother and fetus. J Nutr 2003;133(Suppl. 2):1674S–1683S
19. Lapolla A, Bonomo M, Dalfrà MG, et al; GISOGD. Prepregnancy BMI influences maternal and fetal outcomes in women with isolated gestational hyperglycaemia: a multicentre study. Diabetes Metab 2010;36:265–270
20. Knight B, Shields BM, Hill A, Powell RJ, Wright D, Hattersley AT. The impact of maternal glycemia and obesity on early postnatal growth in a nondiabetic Caucasian population. Diabetes Care 2007;30:777–783
21. Touger L, Looker HC, Krakoff J, Lindsay RS, Cook V, Knowler WC. Early growth in offspring of diabetic mothers. Diabetes Care 2005;28:585–589
22. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. JAMA 2010;303:235–241
23. Cheng YW, McLaughlin GB, Esakoff TF, Block-Kurbisch I, Caughey AB. Glucose challenge test: screening threshold for gestational diabetes mellitus and associated outcomes. J Matern Fetal Neonatal Med 2007;20:903–908
24. Eisenmann JC, Heelan KA, Welk GJ. Assessing body composition among 3- to 8-year-old children: anthropometry, BIA, and DXA. Obes Res 2004;12:1633–1640