Lipid Levels in Former Gestational Diabetic Mothers

CYNTHIA H. MEYERS-SEIFER, MD
BETTY R. VOHR, MD

OBJECTIVE — To investigate lipid levels in former gestational diabetic mothers, 5–6 years postpartum, and to evaluate the relationship of these values to glucose, insulin, BMI, and blood pressure.

RESEARCH DESIGN AND METHODS — The subjects studied were 56 former gestational diabetic mothers and 48 control mothers 5–6 years postpartum. Two hours after a 50-g carbohydrate meal, total cholesterol (TC), triglycerides (TG), HDL and LDL cholesterol, glucose, and insulin were measured and compared between the two groups (analysis of variance). BMI and blood pressure were also evaluated. The risk of finding an abnormal metabolic, anthropometric, or hemodynamic parameter in either group was assessed (χ^2 analysis and Fisher's exact test). Correlation coefficients were assessed between the lipids versus insulin, glucose, BMI, and blood pressure.

RESULTS — Mean TC, TG, LDL cholesterol, glucose, and systolic blood pressure were significantly higher in the gestational diabetic mothers than in the control mothers. In addition, there was greater likelihood of finding an abnormal TC ≥5.17 mmol/l, LDL cholesterol ≥4.14 mmol/l, and systolic blood pressure >140 mmHg in gestational diabetic mothers. Triglycerides correlated with BMI, insulin, systolic and diastolic blood pressure, and HDL cholesterol correlated inversely with insulin in gestational diabetic mothers

CONCLUSIONS — We conclude that at 5–6 years postpartum, former gestational diabetic mothers demonstrate changes in lipid levels that differ from control mothers and that specific lipids correlate with cardiovascular risk factors. Further study is needed to evaluate gestational diabetic mothers for the development of cardiovascular risk factors including insulin resistance.

Gestational diabetes identifies women who are at risk for NIDDM months to years postpartum (1). There is a well-known association between NIDDM and other cardiovascular risk factors including obesity, hypertension, dyslipidemia, hyperinsulinemia, and atherosclerotic vascular disease (2). One hypothesis suggests that the underlying cause of each of the other factors is insulin resistance. Metabolic studies have documented insulin resistance in association with obesity, hypertension, dyslipidemia, and NIDDM (3–7). In addition, studies have demonstrated insulin resistance in former gestational diabetic mothers without glucose intolerance several months to 4 years postdelivery (8–10). Former gestational diabetic mothers may therefore be at increased risk for development of other cardiovascular risk factors, such as lipid abnormalities, in addition to NIDDM.

Individuals with lipid and lipoprotein abnormalities have been shown to be at increased risk for cardiovascular disease (11). Lipids have been studied during pregnancy in diabetic and nondiabetic women, but few studies have addressed lipids in the postpartum gestational diabetic mother. While some studies have reported that NIDDM in pregnancy and gestational diabetes are associated with maternal hypertriglyceridemia and low HDL cholesterol (12–14), others have found no differences in lipoprotein parameters between nondiabetic women, women with well-controlled pregestational diabetes, and women with gestational diabetes during pregnancy (15). Mildly elevated triglycerides (TG) and reduced HDL cholesterol have been found in a group of overweight Hispanic women with a history of gestational diabetes within the first 3 years postpartum, compared with control mothers. These lipid changes were found to be associated with the development of diabetes in the former gestational diabetic mothers during the study period (16).

The primary purpose of this study was to evaluate the lipid levels in former gestational diabetic mothers and control mothers 5–6 years postdelivery; the secondary purpose was to evaluate the relationship of the lipid values with glucose, insulin, blood pressure, and obesity. We hypothesized that total cholesterol (TC), TG, and LDL cholesterol would be significantly higher, and HDL cholesterol significantly lower in former gestational diabetic mothers than in control mothers. In addition, we hypothesized that increased TC, TG, and LDL cholesterol, and decreased HDL cholesterol would correlate with increased obesity, glucose, insulin, and blood pressure in the gestational diabetic mothers.

RESEARCH DESIGN AND METHODS

— The gestational diabetic mothers and control mothers were recruited from a cohort followed longitudinally in the Diabetes in Pregnancy Screening Program at Women and Infants' Hospital, Providence, RI. All mothers were screened between 24 and 28 weeks gestation. A diagnosis of gestational diabetes was made with a plasma glucose screen >7.2 mmol/l 1 h after 50 g of oral glucose, followed by two abnormal glucose values on a 100-g oral glucose tolerance test. The criteria of O'Sullivan and Mahan (17) modified by Carpenter and Coustan (18) were used: fasting plasma glucose >5.3 mmol/l, 1 h >10 mmol/l, 2 h >8.6 mmol/l, and 3 h >7.8 mmol/l. The gestational diabetic mothers received nutrition

From the Department of Pediatrics (C.H.M.-S.), Division of Pediatric Endocrinology and Metabolism, Rhode Island Hospital; and the Department of Pediatrics (C.H.M.-S., B.R.V.), Women and Infants Hospital, Brown University School of Medicine, Providence, Rhode Island.

Address correspondence and reprint requests to Cynthia H. Meyers-Seifer, MD, Endocrinology and Metabolism, Children's Hospital, 700 Children's Drive, Columbus, OH 43205.

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ANOVA, analysis of variance; TC, total cholesterol; TG, triglycerides.

counseling and were given a standard dietary regimen including 45–55% of calories from carbohydrates, 25% from protein, and 25% from fat. Mothers with gestational diabetes were monitored with weekly hospital-based or daily home blood glucose monitoring to keep the fasting glucose below 5.6 mmol/l and the 2-h postprandial glucose below 6.7 mmol/l. Insulin therapy was recommended on the basis of a fasting blood glucose >5.6 mmol/l or a 2-h postprandial glucose >6.7 mmol/l. Control mothers were screened between 24 and 28 weeks and had a plasma glucose <7.2 mmol/l 1 h after 50 g of oral glucose.

Mothers were recruited for longitudinal assessment and evaluated 5 or 6 years postdelivery between April 1994 and June 1995. The mothers had all delivered full-term healthy neonates. Informed consent was obtained. The mother was fasted overnight and given a mixed carbohydrate breakfast containing 50–56.5 g carbohydrate, 4.4–9.9 g protein, and 8.1–13.2 g fat (kcal range: 326–346), between 7:30 and 8:00 A.M. Two hours postprandially, 2.5 ml of blood was obtained from an anticubital vein, clotted, and centrifuged to obtain serum. Serum TC, TG, HDL cholesterol, LDL cholesterol, glucose, and insulin were assayed in the Women and Infants' Hospital research laboratory. Blood glucose was determined by the glucose oxidase method using a YSI glucose analyzer (YSI, Yellow Springs, OH). Serum insulin concentration was measured in a double-antibody radioimmunoassay, which used a recombinant human insulin standard and ^{125}I monoiodinated insulin (Diagnostic Products, Los Angeles, CA). Coefficient of variation was 8.1%. Triglycerides were determined using enzymatic hydrolysis to free fatty acids and glycerol. Glycerol was measured spectrophotometrically at 540 nm after coupled enzyme reactions catalyzed by glycerol kinase, glycerol phosphate oxidase, and peroxidase (Sigma Diagnostics, St. Louis, MO). Coefficient of variation was 2.1%. Total cholesterol was determined by hydrolyzing all cholesterol esters to cholesterol, which was then measured spectrophotometrically at 500 nm after coupled enzyme reactions catalyzed by cholesterol oxidase and peroxidase (Sigma). Coefficient of variation was 1.3%. To measure HDL cholesterol, VLDL and LDL were separated from HDL by selective precipitation using phosphotungstic acid and magnesium chloride. The cholesterol concentration in the HDL fraction was then assayed by the enzymatic method described above for total cholesterol. Coefficient of variation was 2.8%. To measure LDL cholesterol, VLDL and HDL were separated from LDL using latex beads coated with affinity-purified goat polyclonal antisera to specific human apolipoproteins. After a short incubation and centrifugation, the LDL remained in the filtrate solution. The cholesterol concentration in the LDL fraction was assayed by the enzymatic method described above for total cholesterol. Coefficient of variation was 3.2%.

Weight was obtained using a standard upright scale (Seca, model 707, Columbia, MD). Height was obtained using a permanently affixed stadiometer, and the average of two measures was reported. Right arm blood pressure was obtained with the arm fully exposed, resting on a supportive surface. The DINAMAPP vital signs monitor was used with a cuff covering 75% of the upper arm between the top of the shoulder and the olecranon. The mean of two measurements was reported for systolic blood pressure and diastolic blood pressure.

Statistical analysis

Comparisons between the means of former gestational diabetic mothers and control mothers were performed using analysis of variance (ANOVA). Pearson–product moment correlation coefficients were computed between lipids, BMI, insulin, glucose, and blood pressure for both the gestational diabetic mothers and the control group. When calculating the Pearson–product moment correlation coefficients, we used the Bonferroni correction. χ^2 and Fisher's exact tests of significance were used to compare the incidence of abnormal lipid levels, BMI, glucose level, insulin concentration, and blood pressure between the gestational diabetic mothers and control groups. Upper limits of normal for TC and LDL cholesterol were chosen according to the National Cholesterol Education Program guidelines, which define borderline-high and high TC as ≥5.17–6.18 mmol/l and ≥6.21 mmol/l, respectively, and borderline-high and high LDL cholesterol as 3.36–4.11 mg/dl and 4.14 mmol/l, respectively (19). These values are defined for both fasting and nonfasting samples. The lower limit of normal for HDL cholesterol, ≤0.91 mmol/l, was based on an expected range provided by Sigma for the assay used. The abnormal TG level of ≥2.83 mmol/l was chosen based on the proceedings of two consensus conferences on hypertriglyceridemia (20,21). The normal HDL cholesterol and TG levels were defined for fasting samples, and may not be accurate in the postprandial state for clinical assessment of an individual's cardiovascular risk. Because there are no postprandial standards available, however, we have used the normal fasting values for the purpose of comparing gestational diabetic mothers and control groups, both of which consumed the same breakfast. The abnormal glucose level of 7.8 mmol/l was selected on the basis of the National Diabetes Data Group guidelines, which define impaired glucose tolerance in part by a glucose level >7.8 mmol/l 2 h after a 75-g oral glucose load. Our subjects consumed 50 g carbohydrate with a meal and their glucose responses may have been less than would have been anticipated after a 75-g test (22). In clinical practice, postprandial insulin concentration is not useful in diagnosing diabetes or impaired glucose tolerance, in part due to the variability among individuals with normal as well as abnormal glucose tolerance. Insulin concentration 2 h after ingesting 75 g carbohydrate was between 90 and 1,080 pmol/l in 22 normal subjects, according to data provided by Diagnostic. We chose an insulin concentration of 240 pmol/l to test for an increased incidence of hyperinsulinemia in gestational diabetic mothers. Abnormal BMI was defined as >27 kg/m^2. Abnormal systolic and diastolic blood pressure were defined as >140 and >90 mmHg, respectively.

RESULTS — Data were collected on 56 gestational diabetic mothers and 48 control mothers. The characteristics of the study subjects are shown in Table 1. There were no differences in age, race, or socioeconomic status between the gestational diabetic mothers and the control group. There were four Hispanic, one African-American, and 51 Caucasian gestational diabetic mothers; all control subjects were Caucasian. Weight, height, and BMI were similar between groups. Birth weight of the index newborn did not differ between the two groups. Gravity and parity were similar between the two groups. Seventy-seven percent of gestational diabetic mothers and 71% of control mothers had had at least one pregnancy before the index preg-

Table 1—*Characteristics of the study subjects*

	Gestational diabetic mothers	Control subjects
n	56	48
Age (years)	36.9 ± 4.2	36.1 ± 3.6
SES	47.9 ± 12.7	46.6 ± 11.2
Weight (kg)	68.5 ± 12.4	68.8 ± 17.7
Height (cm)	163.0 ± 6.8	164.9 ± 6.9
BMI (kg/m^2)	25.9 ± 4.7	25.1 ± 5.3
Birth weight (kg) (index infant)	3.7 ± 0.5	3.8 ± 0.5

Data are means ± SD. SES, Hollingshead Social and Environmental Status, Four Factor Index.

nancy. A significantly higher percentage of gestational diabetic mothers than control subjects, (40 vs. 15%), reported a family history of diabetes (χ^2 = 7.0, P = 0.008).

Metabolic parameter and blood pressure results are shown in Table 2. TC, TG, LDL cholesterol, and glucose were significantly higher in gestational diabetic mothers. HDL cholesterol and insulin did not differ significantly between groups. Systolic blood pressure was significantly higher in the gestational diabetic mothers than in the control group.

Categorical analyses using the χ^2 and Fisher's exact tests were done to identify the incidence of abnormal values in the gestational diabetic mothers compared with the control mothers. Former gestational diabetic mothers had a higher incidence of elevated TC ≥5.17 mmol/l (39 vs. 17%) and LDL cholesterol ≥4.14 mmol/l (13 vs. 2%) compared with control mothers. The incidence of abnormal values for the remaining metabolic parameters did not differ between groups. One (gestational diabetic mother) subject had a serum glucose >7.8 mmol/l (8.2 mmol/l). Gestational diabetic mothers also had a greater likelihood of having an abnormal systolic blood pressure >140 mmHg (9 vs. 0%) than the control mothers. The incidence of obesity, defined as a BMI >27 kg/m^2 was similar in the gestational diabetic mothers (34%) and control mothers (31%) (Table 3).

We compared the need for insulin during pregnancy, fasting glucose on the pregnancy glucose tolerance test, prepregnancy weight, prepregnancy BMI, weight gain during pregnancy, and weight at the end of pregnancy between the gestational diabetic mothers with abnormal lipid parameters versus the gestational diabetic mothers with normal lipid parameters as defined in Table 3. The gestational diabetic mothers with abnormal HDL cholesterol ≤0.91 mmol/l had a higher prepregnancy weight and lower pregnancy weight gain than the gestational diabetic mothers with HDL cholesterol >0.91 mmol/l, shown in Table 4. No other pregnancy characteristic differed among gestational diabetic mothers with abnormal TC, TG, and LDL cholesterol versus gestational diabetic mothers with normal TC, TG, or LDL cholesterol.

Analyzed relative to specific characteristics of the index pregnancy, lipid levels did not differ in the gestational diabetic mothers based on either a need for insulin, nor a fasting glucose >5.6 mmol/l during pregnancy.

Correlation coefficients for gestational diabetic mothers and control mothers were calculated to assess the relationships among maternal lipid levels with BMI, insulin, glucose, and blood pressure. Increased TG correlated with BMI (r = 0.38, P = 0.004), insulin (r = 0.60, P = 0.0001), systolic (r = 0.45, P = 0.0005) and diastolic blood pressure (r = 0.38, P = 0.004) in gestational diabetic mothers only (Table 5). HDL cholesterol correlated inversely with insulin (r = −0.38, P = 0.006) in gestational diabetic mothers only. Total cholesterol and LDL cholesterol did not correlate with any of these parameters in either the gestational diabetic mothers or control groups.

We then evaluated the relationship between BMI and the remaining metabolic parameters (glucose and insulin) as shown in Table 6. Within the group of gestational diabetic mothers, although there was no relationship between BMI and glucose or insulin, BMI did correlate with systolic (r = 0.32, P = 0.02) and diastolic blood pressure (r = 0.34, P = 0.02). In the control group, increased BMI correlated with insulin only (r = 0.52, P = 0.0007).

To further investigate the interactive relationships of the study parameters, we performed correlation analyses between insulin and the remaining variables. In the group of gestational diabetic mothers, insulin did not correlate with glucose, BMI, or blood pressure. In contrast, for the control group, increased insulin correlated with glucose (r = 0.34, P = 0.03) and BMI (r = 0.52, P = 0.0007).

CONCLUSIONS — The focus of the present study was to assess former gesta-

Table 2—*Metabolic parameters and blood pressure of gestational diabetic mothers and control groups*

	Gestational diabetic mothers	Control subjects	*P*
TC (mmol/l)	4.91 ± 1.24 (56) (2.93–8.29)	4.29 ± 0.93 (48) (2.48–7.46)	0.005
TG (mmol/l)	1.48 ± 1.24 (56) (0.47–7.97)	0.96 ± 0.76 (48) (0.32–5.23)	0.02
HDL cholesterol (mmol/l)	1.27 ± 0.39 (55) (0.49–2.38)	1.16 ± 0.31 (46) (0.60–2.07)	NS
LDL cholesterol (mmol/l)	3.15 ± 0.93 (55) (1.92–6.45)	2.72 ± 0.75 (47) (0.96–5.18)	0.01
Glucose (mmol/l)	4.6 ± 0.8 (53) (3.2–8.2)	4.2 ± 0.6 (47) (3.1–6.1)	0.008
Insulin (pmol/l)	102 ± 96 (51) (35–580)	96 ± 48 (40) (21–344)	NS
Systolic blood pressure (mmHg)	122 ± 12 (55) (97–154)	117 ± 7 (47) (100–138)	0.01
Diastolic blood pressure (mmHg)	72 ± 11 (55) (46–102)	69 ± 8 (47) (52–89)	NS

Data are means ± SD (*n*) (range). The subject number (*n*) was variable because of technical limitations of blood sample collection. *P* values determined by ANOVA.

Table 3—*Incidence of abnormal metabolic, hemodynamic, and anthropometric parameters in gestational diabetic mothers and control groups*

	Gestational diabetic mothers	Control subjects	*P*
TC ≥5.17 mmol/l	22/56 (39)	8/48 (17)	0.01*
TC ≥6.21 mmol/l	8/56 (14)	2/48 (4)	NS
TG ≥2.83 mmol/l	4/56 (7)	1/48 (2)	NS
LDL cholesterol ≥3.36 mmol/l	17/55 (31)	9/47 (19)	NS
LDL cholesterol ≥4.14 mmol/l	7/55 (13)	1/47 (2)	0.05†
HDL cholesterol ≤0.91 mmol/l	8/55 (15)	9/46 (20)	NS
Glucose >7.8 mmol/l	1/53 (2)	0/47 (0)	NS
Insulin >240 pmol/l	4/51 (8)	1/40 (3)	NS
Systolic blood pressure >140 mmHg	5/55 (9)	0/47 (0)	0.04†
Diastolic blood pressure >90 mmHg	4/55 (7)	0/47 (0)	NS
BMI >27 kg/m²	19/56 (34)	15/48 (31)	NS

Data are *n* (%). *Determined by χ^2 analysis. †Determined by Fisher's exact test.

tional diabetic mothers, a population known to be at risk for NIDDM (1), for lipid abnormalities 5–6 years postdelivery and to evaluate the relationship of these findings to BMI, insulin, glucose, and blood pressure. We found that at 5–6 years postpartum, former gestational diabetic mothers had higher 2-h postprandial TC, TG, LDL cholesterol, and glucose than control mothers, confirming our first hypothesis. The gestational diabetic mothers also had higher systolic blood pressure than control mothers and were more likely to have abnormal TC ≥5.17 mmol/l, LDL cholesterol ≥4.14 mmol/l, and systolic blood pressure >140 mmHg.

The higher TC, TG, and LDL cholesterol noted in the former gestational diabetic mothers in our study may have been a consequence of metabolic changes associated with insulin resistance, inherited disorder(s) of lipid metabolism in some subjects, or a combination of these factors. In NIDDM, the typical lipid abnormalities are elevated free fatty acids and elevated TG, (mostly in the form of VLDL) and decreased HDL cholesterol (26,27). Although the mean TG was higher in the former gestational diabetic mothers in our study, we did not find an increased incidence of TG ≥2.83 mmol/l in the group of gestational diabetic mothers. In addition, we found no difference in HDL cholesterol between groups.

The difference in TG levels may in part reflect a lifestyle difference between the gestational diabetic mothers and control mothers, since factors such as prior diet and exercise status are known to alter TG level (20). A genetic predisposition to lipid abnormalities may also contribute to the difference in TG between the former gestational diabetic mothers and control mothers, as there is evidence to suggest that a genetic predisposition may contribute to dyslipidemia in NIDDM (28). For example, a heterozygous mutation in the lipoprotein lipase gene, which impairs triglyceride processing, may become clinically significant only when triglyceride is overproduced, as in NIDDM (29). Similarly, inheritance of certain apolipoprotein E alleles (apolipoprotein E mediates the binding of lipoproteins to their receptors) may promote the expression of dyslipidemia in NIDDM (30).

Although elevated TC and LDL cholesterol have not consistently been associated with NIDDM (31), our study found the TC and LDL cholesterol to be significantly higher in the former gestational diabetic mothers than in control subjects. In addition, there was an increased incidence of TC ≥5.17 mmol/l and LDL cholesterol ≥4.14 mmol/l in the group of gestational diabetic mothers. Increased LDL cholesterol is widely recognized as a risk factor for coronary artery disease (11). Our findings suggest that former gestational diabetic mothers may be more likely than control mothers to develop a lipid profile that confers increased cardiovascular risk.

The increases in TC and LDL cholesterol found in gestational diabetic mothers may or may not be related to the increased risk of NIDDM in this group. A growing body of literature addresses alterations in LDL associated with the insulin resistance syndrome and NIDDM (32–35). Peroxidation and glycation of LDL, processes that are increased in NIDDM and enhanced by hyperglycemia, alter the LDL molecule, reducing LDL receptor-mediated clearance and potentially raising circulating LDL levels. These alterations in LDL composition are also thought to increase cardiovascular risk in NIDDM by increasing the atherogenic potential of the LDL molecule (32,33).

Elevated postpartum TG (16), indexes of maternal obesity, fasting glucose during pregnancy and insulin therapy during pregnancy (23–25), have been associated with subsequent abnormal glucose tolerance in former gestational diabetic mothers. In the present study, the gestational diabetic mothers with abnormally low HDL cholesterol had higher prepregnancy weight than the gestational diabetic mothers with normal HDL cholesterol. Maternal obesity may therefore be associated not only with future glucose intolerance but also with future lipid abnormalities in gestational diabetic mothers. Further investigation is required as abnormalities in TC, TG, and LDL cholesterol in the gestational diabetic mothers were not related to the pregnancy characteristics examined. In addition, neither fasting glucose nor insulin therapy during pregnancy predicted a difference in postpartum lipids.

Gestational diabetic mothers were found to have a higher mean serum glucose than control subjects 5–6 years post-

Table 4—*Pregnancy characteristics of gestational diabetic mothers with abnormal and normal HDL cholesterol*

	Abnormal (≤0.91 mmol/l)	Normal (>0.91 mmol/l)	*P*
Subject number	8	46	—
Prepregnancy weight (kg)	70.6 ± 15.7	61.5 ± 10.7	0.04
Pregnancy weight gain (kg)	10.0 ± 5.5	15.2 ± 6.3	0.03
Prepregnancy BMI (kg/m²)	25.7 ± 6.4	23.3 ± 3.4	NS
End of pregnancy weight (kg)	80.6 ± 12.4	76.6 ± 11.7	NS

Data are means ± SD. *P* values determined by ANOVA.

Table 5—*Correlation of triglycerides with BMI, glucose, insulin, and blood pressure in gestational diabetic mothers and control study groups*

	Gestational diabetic mothers		Control subjects	
	r	*P*	*r*	*P*
BMI	0.38	0.004	0.33	NS
Glucose	−0.14	NS	0.02	NS
Insulin	0.60	0.0001	0.11	NS
Systolic blood pressure	0.45	0.0005	0.08	NS
Diastolic blood pressure	0.38	0.004	0.21	NS

r, Pearson's product moment correlation coefficient.

partum without a significant difference noted in insulin level. One gestational diabetic mother in our study had a glucose level of >7.8 mmol/l compared with none in the control group. The incidence of insulin concentration >240 pmol/l was 8% in the group of gestational diabetic mothers and 3% in the control group. This difference was not significant. Glucose intolerance cannot be ruled out in any of our subjects, because glucose tolerance testing was not done for this study. Although the present study was not designed to determine insulin resistance, other studies have demonstrated insulin resistance in former gestational diabetic mothers without glucose intolerance several months to 4 years postdelivery (8–10).

In support of our second hypothesis, we found that in gestational diabetic mothers, TG correlated with BMI, insulin, and blood pressure, while HDL cholesterol correlated inversely with insulin. It is of interest that these associations between cardiovascular risk factors tended to cluster in the former gestational diabetic mothers, given previous data linking NIDDM and other cardiovascular risk factors (2).

Our final investigations revealed no correlation between BMI and insulin in the group of gestational diabetic mothers, while in the control group BMI correlated strongly with insulin. In addition, there was no correlation between insulin and glucose in the former gestational diabetic mothers, while this correlation was significant in the control mothers. The significance of these associations, which are present in the control mothers and lost in the gestational diabetic mothers, remains speculative.

In conclusion, our results demonstrate that 5–6 years postpartum, former gestational diabetic mothers show increased TC, TG, LDL cholesterol, and glucose compared with control subjects. In addition, in former gestational diabetic mothers, increased TG is associated with increased BMI, and increased blood pressure, while both increased TG and decreased HDL cholesterol are associated with increased insulin concentration. Former gestational diabetic mothers are also more likely than control subjects to have abnormalities in TC, LDL cholesterol, and systolic blood pressure. Further longitudinal study of metabolic, anthropometric, and hemodynamic parameters in gestational diabetic mothers postpartum will better define the evolution of lipid and other abnormalities in individuals predisposed to NIDDM.

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Table 6—*Correlation of BMI with glucose, insulin, and blood pressure in gestational diabetic mothers and control study groups*

	Gestational diabetic mothers		Control subjects	
	r	*P*	*r*	*P*
Glucose	−0.05	NS	0.29	NS
Insulin	−0.06	NS	0.52	0.0007
Systolic blood pressure	0.32	0.02	0.28	NS
Diastolic blood pressure	0.33	0.02	0.30	NS

r, Pearson's product moment correlation coefficient.

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