The Recurrence of Gestational Diabetes: Could Dietary Differences in Fat Intake Be an Explanation?

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OBJECTIVE — To present the results of a comprehensive dietary review of a group of women with a recurrence of gestational diabetes mellitus (GDM), compared with a group of women with no recurrence of GDM during a subsequent pregnancy.

RESEARCH DESIGN AND METHODS — The dietary intake of 14 women with a recurrence of GDM was compared with 21 women with no recurrence of GDM. Women with GDM in one pregnancy have a recurrence rate of only 30–50%. While the reasons for this have not been determined, dietary factors have been considered probable.

RESULTS — The women with a recurrence of GDM consumed 38.4 (by diet history) and 41.4% (by food record) of their total energy intake as fats, compared with 34.1 ($P < 0.01$) and 33.1% ($P < 0.001$), respectively, for women with no recurrence. The percentage intake of polyunsaturated, monounsaturated, and saturated fatty acids was similar in both groups. There was a proportionate reduction in carbohydrate intake as a percentage of total energy and in fiber intake in grams for the women with a recurrence of GDM.

CONCLUSIONS — When the relationship between saturated fat intake and insulin resistance is considered, the possibility exists that dietary modification of fat intake before and during pregnancy may reduce the recurrence rate of GDM.

Gestational diabetes mellitus (GDM) is carbohydrate intolerance of variable severity with onset or first recognition during the current pregnancy (1). The recurrence rate of GDM in a subsequent pregnancy has ranged from 30 to 50% (2–6). Factors which in some studies have been associated with a recurrence of GDM include obesity (2,3), weight gain between pregnancies (6), and insulin use in the index pregnancy (2,3).

Dietary modification during pregnancy is essential for the medical management of women with GDM and can lead to outcomes equivalent to a glucose-tolerant pregnancy (7,8). While weight gain between pregnancies appears as the most consistent risk associate with a recurrence of GDM, there are no reports examining whether any dietary differences exist between women who develop a GDM recurrence and women with no recurrence. If dietary differences could be demonstrated to exist, then the potential to alter dietary intake before or early in the pregnancy to prevent a recurrence of GDM may be possible. If a subsequent pregnancy with GDM can hasten the conversion to NIDDM (5,9), then proactive dietary interventions may assume even greater importance.

This study presents the results of a comprehensive dietary review of a group of women with a recurrence of GDM, compared with a group with no recurrence of GDM during a subsequent pregnancy.

RESEARCH DESIGN AND METHODS

— This study used a cohort of women previously studied with respect to the recurrence rate of GDM (6). A total of 480 women seen by one endocrinologist (R.M.) for the medical management of their GDM over a 5-year period, January 1990 to December 1994, were the subjects for the index pregnancy. Of these 480 women, 101 had completed a second pregnancy by the end of 1995, and 100 women had been tested again for GDM.

The definition of GDM was based on the recommendations of the Australasian Diabetes in Pregnancy Society (ADIPS) (10). No preliminary glucose challenge test was used. Unless diagnosed at an earlier stage of pregnancy, all subjects at the beginning of the third trimester had a standard 75-g oral glucose tolerance test (OGTT) in the morning after a 12-h fast. (Three women were diagnosed in the second pregnancy at 12, 14, and 18 weeks of gestation.) GDM was diagnosed if the fasting plasma glucose was ≥5.5 mmol/l and/or the 2-h glucose level was ≥8.0 mmol/l. In some instances, a modification of the above-mentioned test was performed with only the 2-h sample being taken after the OGTT (11). All women received standard treatment as previously outlined (6,8). After delivery and before discharge from the hospital, the majority of women were reviewed by a dietitian and offered general advice. No post-discharge dietary follow-up was possible.

A recurrence of GDM was found in 35 (35%) of the 100 patients who had had a second pregnancy and a test for GDM. Over a 3-month period, June–August 1996, an attempt was made to contact by telephone, letter, and personal visit all of these 100 women. Those women who could be contacted and who agreed to participate in the study were visited at their homes.

The previously obtained demographic details were checked and current BMI calculated as weight in kilograms divided by the square of the height in meters. A positive family history was recorded if a grandparent, parent, or sibling had diabetes. Two meth-

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Abbreviations: ADIPS, Australasian Diabetes in Pregnancy Society; DCCT, Diabetes Control and Complications Trial; EEI, estimated energy intake; GDM, gestational diabetes mellitus; NRG, no recurrence of GDM; OGTT, oral glucose tolerance test; PBMR, predicted basal metabolic rate; RG, recurrence of GDM.

ods, a diet history and a food record, were applied for the assessment of dietary intake.

Diet history

Diet history used a locally adapted version of the the Diabetes Control and Complications Trial (DCCT) methodology (12). This version included a diet history interview where subjects were asked to describe their usual eating pattern for one day, beginning with the first meal or the equivalent. This description was followed by probing questions on amounts and types of foods listed, food preparation techniques, snacking and dining out patterns, variations in the dietary patterns, and the frequency of consumption of common food groups.

With respect to diet history, the two DCCT interview forms (food preparation questionnaire, food pattern questionnaire) were adapted to Australian conditions (13), and these local versions were completed at the time the subjects attended the diet history interview. The new questionnaires were developed via direct communication with the dietitian responsible for the DCCT instruments and with the reference to information on food available in large local supermarkets and via Australian food frequency questionnaires provided by the Commonwealth Scientific and Industrial Research Organization (CSIRO). Pilot studies were conducted to test for validity and utility with student dietitians at the University of Wollongong and with patients attending the Diabetes Centre.

The DCCT Food Pattern Questionnaire is semiquantified, requiring information on frequency of consumption (daily, 4–6 times per week, 1–3 times per week, 1–3 times per month, or 1–3 times a year, or never) and comments on seasonal variation, but not stipulating the serving sizes. In this study, both questionnaires were used in a qualitative sense to check the accuracy of the diet history data. For example, any food not listed in the diet history but listed as consumed more than once a week in the food pattern questionnaire was included in the diet history analysis using a single serving size from the database reference amount.

The diet history interviews were conducted by three student dietitians completing the final stage of a master's degree course. The students were specifically trained for the interview technique based on the DCCT methodology and recent research on the communication process in the dietary interview (14). In this process, each student interviewed the same person and the results were compared. The amounts of foods were recorded in standard metric household measures. All data were recorded on standardized forms.

Food record

A second measure of dietary intake was obtained using a 3-day food record. Each woman was individually instructed on how to record on standardized forms all food consumed on two typical weekdays and for one weekend day. The amounts of food eaten were recorded using standard household measures.

Data for nutrient analyses were collated to give group mean values from both measures of dietary intake (diet history and food record) for total energy, total fiber, percentage of energy from protein, fat, carbohydrate, and alcohol and the proportion of fat consumed as saturated, polyunsaturated, and monounsaturated fatty acids.

Data from diet history and 3-day food record were entered into the DIET1 nutrient analysis software program, version 4 (Xyris Software, Highgate Hill, Queensland, Australia). This program used the NUTTAB 95 Australian food composition database (15). Standardized substitutions for similar food were made when individual food items were not on the database, and recipes were entered as required. Any food not included in diet history but listed as consumed more than once a week in the food frequency checklist was included in the diet history analysis using a single serving from the database reference amount. Vitamin or mineral supplements taken by subjects were not included in the analysis.

The plausibility of reported energy intakes from the food record was assessed by calculating the ratio of estimated energy intake (EEI) to predicted basal metabolic rate (PBMR), where BMR was calculated using the Schofield equation (16). A figure of <0.88, representing the 97.5% CI, was taken as evidence of underreporting at the individual level (17).

All statistical analyses were conducted using JMP version 3.0.2 (1994, SAS Institute, Cary, NC). Statistical tests included the χ^2 test for discrete variables and the two-tailed t test for continuous variables. Results have been expressed as mean ± SD. Results were considered significant if $P < 0.05$.

Ethical approval for this research was provided by the Human Research and Ethics Committee of the University of Wollongong and the Illawarra Area Health Service.

RESULTS — Of the 100 eligible women, 82 were able to be contacted, and 49 of the 82 (60%) women agreed to participate in the study. Of these 49 women, 1 was excluded owing to a current pregnancy and an additional 5 were excluded because they were on weight-loss diets. Of the remaining 43 women, 3 did not complete the questionnaire, and 5 were excluded from the final data analysis because they had an EEI:PBMR ratio <0.88 and were thus considered to be underreporting their dietary intake.

The final sample contained 35 women. Of the 65 women with no recurrence of GDM (NRG), 21 (32.3%) were included in this study. Of the 35 women with a recurrence of GDM (RG), 14 (40%) were included in this study. The percentage of women included in the study from both the RG and NRG original groups were not significantly different.

The 21 women in the NRG group were age 32.7 ± 3.4 years (mean ± SD) and had a parity of 2.4 ± 0.8 and a BMI of 28.0 ± 8.0, which was not significantly different from the 14 women in the RG group with a mean age of 34.9 ± 4.2 years, a parity of 3.0 ± 1.0, and a BMI of 27.0 ± 5.3. There was also no difference between the two groups with respect to the percentage of women with a positive family history of diabetes. The 21 participating women in the NRG group were not significantly different from the 44 nonparticipating women with no recurrence of GDM, with respect to age, parity, and last recorded BMI. The 14 participating women in the RG group were not significantly different from the 21 nonparticipating women with a recurrence of GDM, with respect to age, parity, and last recorded BMI. The average interval between the two pregnancies was 2.4 years, and the average time between the last pregnancy and the interview was also 2.4 years. Nutritional data obtained by diet history and food record for women in the NRG group and the RG group are shown in Table 1.

The diet history and food record both demonstrated a significantly higher intake of fat as a percentage of energy intake in the RG group, with a lower intake of both carbohydrate (percentage) and fiber (grams). No significant differences were found in the type of fat eaten between the NRG and RG groups by either method.

In terms of food choice patterns, there were no remarkable differences in the types of food chosen by either the NRG or RG groups. The main dietary sources of fat for both groups were meat, poultry, and fish,

Table 1—*Nutritional data obtained by diet history and food record for NRG and RG women*

	NRG group (n = 21)		RG group (n = 14)	
	DH	FR	DH	FR
Total energy (cal)	2,253 ± 580	1,979 ± 406	1,903 ± 432	1,975 ± 409
Protein (% kcal)	18.5 ± 2.1	18.1 ± 2.5	19.2 ± 3.3	17.9 ± 3.2
Fat (% kcal)	34.1 ± 3.9	33.1 ± 4.7	38.4 ± 3.2†	41.4 ± 6.2‡
Carbohydrate (% kcal)	46.0 ± 2.7	47.5 ± 3.8	41.7 ± 4.1*	40.4 ± 5.3‡
Alcohol (% kcal)	1.8 ± 2.7	1.2 ± 2.7	0.7 ± 1.3	0.6 ± 1.5
Fat profile (% total fat)				
Polyunsaturated	16.2 ± 5.5	17.2 ± 6.9	13.1 ± 4.8	15.9 ± 7.4
Monounsaturated	38.4 ± 3.5	37.9 ± 4.8	38.3 ± 4.9	39.4 ± 7.6
Saturated	45.5 ± 4.8	44.8 ± 7.4	48.6 ± 6.6	44.8 ± 9.5
Other nutrients				
Fiber (g)	26.3 ± 6.8	22.2 ± 5.4	19.1 ± 4.3†	18.2 ± 4.3*

Data are means ± SD. DH, diet history; FR, food record. *$P < 0.05$, †$P < 0.01$, ‡$P < 0.001$. The fat intake, as a percentage of total energy, was higher in the RG group compared with the NRG group by both DH ($P < 0.01$) and FR ($P < 0.001$). The carbohydrate intake, as a percentage of total energy, was lower in the RG group compared with the NRG group by both DH ($P < 0.05$) and FR ($P < 0.001$). The total fiber intake in grams was lower in the RG group compared with the NRG group by both DH ($P < 0.01$) and FR ($P < 0.05$).

followed by "takeaway" meals with overall no significant differences in the percentage of fat derived from different food groups. There was no significant difference between the two groups with respect to the frequency of consumption of "takeaway" foods, which averaged 0.65 and 0.62 meals each week for the NRG and RG groups, respectively. However, "takeaway" meals were still responsible for 13.8% of the total fat intake for the NRG group and 13.5% for the RG group.

CONCLUSIONS — The five substantive reports about the recurrence of GDM (2–6) have all postulated a potential effect of diet as being one of the responsible factors. However, no systematic examination of this aspect has been carried out. In an attempt to address this issue, all of the women who were part of the most recent report on the recurrence rate of GDM were considered for inclusion in a study involving a comprehensive dietary review. When considered that the interviews took place for most women more than 2 years after the second pregnancy, it was encouraging that contact could be made with 82% of subjects. The number of women who eventually agreed to participate was nearly half of the original group. After exclusion of subjects who could have compromised the data, 35 women were used for the final analyses. These women appeared to be representative of the original group. While the final number of women may appear small, this has to be seen in perspective. They were distilled from the second largest reported series representing 480 index pregnancies seen over a 5-year period.

In this study, we have used two different methods to estimate dietary intake. While no unequivocal "gold standard" exists (18), diet history is considered a reliable reference method and was the method of choice for the DCCT (12). In our hands, the results obtained by the food record method had a high correlation with the results of the diet history method and could be considered a viable alternative for prospective epidemiological studies of the diet of women with GDM.

Surprisingly, for a disorder where diet is the key to treatment, there is no information available about the usual diet of women who develop GDM, compared with women who do not. There is also no information available about whether there are any differences in dietary intake between women who develop a recurrence of GDM and women who do not. Without this type of information, it is not possible to judge whether dietary advice offered in pregnancy is having any long-term impact, and whether prospective dietary advice may affect the incidence rate of GDM.

All of the women considered for inclusion in this study had an index pregnancy with GDM. They received dietary advice during and immediately after the pregnancy. While this advice has been shown to have little influence on subsequent diet (19), it is possible that some of the women did make changes to their diet after the first pregnancy. However, current dietary intake is considered a better predictor of past intake than recollection (20), and the ability to assess diet successfully from the past has been demonstrated over a period of 4 years (21). This study, for very apparent reasons, was conducted some years after the completion of the second pregnancy. The dietary information obtained therefore is probably representative of, or at least a reasonable reflection of, the dietary intake before the subsequent pregnancy.

In the series herein reported, we have found that women who develop a recurrence of GDM have a significantly higher fat intake as a percentage of total energy than women who did not have a recurrence. The proportions of polyunsaturated, monounsaturated, and saturated fats were the same in both groups, and there were no remarkable differences in the dietary sources of fat. The women in the RG group ate more fat of all types as a percentage of energy intake than women in the NRG group. In association with this higher percentage of fat intake, there was also a proportionate reduction in the amount of carbohydrate and fiber eaten.

The women in this study had GDM diagnosed with the ADIPS criteria using a 75-g glucose load and a 2-h glucose level of ≥8.0 mmol/l. The ADIPS criteria are similar to the World Health Organization (WHO) criteria, except that the 2-h glucose level has been rounded up from 7.8 to 8.0 mmol/l. However, criteria using a 100-g glucose load and higher glucose levels for diagnosis have a lower incidence rate of GDM and therefore are likely to include patients with a more severe degree of glucose intolerance. Women diagnosed with a 100-g glucose load also have a slightly higher recurrence rate of GDM (2,3), compared with women diagnosed using lesser glucose loads (4–6). Whether women with more severe degrees of glucose intolerance will also have a higher fat intake, or whether our findings will be applicable to different populations, remains to be determined.

GDM is a condition where insulin secretion and action are unable to overcome the increasing insulin resistance which normally accompanies pregnancy (22). Any factor that may also increase insulin resistance could have a potentially deleterious additive effect during pregnancy. Variations in the dietary intake of fats and carbohydrates can have a profound influence on insulin resistance and sensitivity.

In humans, short-term dietary manipulations involving different proportions of total energy derived from carbohydrates, fats, and differing compositions of fatty acids have so far yielded limited and equiv-

ocal results (23). Long-term epidemiological studies, however, have revealed some positive associations. A recent report has demonstrated in women that the relative risk of developing NIDDM was increased if the usual diet contained foods with a high glycemic index and a low fiber content (24). Other studies have shown that long-term ingestion of a high-fat diet, particularly one containing either an increased amount or a proportion of saturated fats, can be associated with insulin resistance and a progression to glucose intolerance and diabetes (25–27). This effect may be brought about by changes to the saturation of muscle membrane phospholipids related to the habitual dietary intake of fat over many months or years (28).

Women who have GDM are at very high risk of converting to impaired glucose tolerance and NIDDM in future years. This risk appears enhanced if there is a recurrence of GDM in a subsequent pregnancy (5,9). In this study, women who developed a recurrence of GDM had a significantly higher fat intake than women who did not have a recurrence. This would suggest that a high-fat diet, by its effect on insulin resistance, may be linked to the development of GDM, and a low-fat diet could be protective. The hypothesis could be advanced to state that in women at high risk of developing GDM, dietary manipulation for an as yet to be determined time before as well as during the pregnancy may reduce the incidence of GDM.

To our knowledge, this study is the first to look at the diet of women who have had a recurrence of GDM, compared with women who did not have a recurrence in a subsequent pregnancy. It is also the first to identify a specific macronutrient, which may be associated with why some women have a recurrence and some do not. The higher fat intake found for women who have a recurrence of GDM sits comfortably with all of the available information about the relationship between the dietary intake of saturated fats and insulin resistance. Prospective studies regarding the dietary intake of pregnant women and the relationship of different nutrient intakes to the development and recurrence of GDM are required.

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References

1. Metzger BE (Ed.): Summary and recommendations of the Third International Workshop-Conference on Gestational Diabetes Mellitus. *Diabetes* 40 (Suppl. 2):197–201, 1991
2. Philipson EH, Super DM: Gestational diabetes mellitus: does it recur in subsequent pregnancy? *Am J Obstet Gynecol* 160:1324–1331, 1989
3. Gaudier FL, Hauth JC, Poist M, Corbett D, Cliver SP: Recurrence of gestational diabetes mellitus. *Obstet Gynecol* 80:755–758, 1992
4. Coelingh Bennink HJT: Recurrence of gestational diabetes. *Eur J Obstet Gynecol Reprod Bio* 7:359–363, 1977
5. Grant PT, Oats JN, Beischer NA: The long-term follow-up of women with gestational diabetes. *Aust N Z J Obstet Gynaecol* 26:17–22, 1986
6. Moses RG: The recurrence rate of gestational diabetes mellitus in subsequent pregnancies. *Diabetes Care* 19:1348–1350, 1996
7. Drexel H, Bichler A, Sailer S, Breier C, Lisch H, Braunsteiner H, Patsch JR: Prevention of perinatal morbidity by tight metabolic control in gestational diabetes mellitus. *Diabetes Care* 11:761–768, 1988
8. Moses RG: The medical management of gestational diabetes in Australia within a solo private practice. *Diabet Med* 11:597–600, 1994
9. Peters RK, Kjos SL, Xiang A, Buchanan TA: Long-term diabetogenic effect of single pregnancy in women with previous gestational diabetes mellitus. *Lancet* 347:227–230, 1996
10. Martin FIR: The diagnosis of gestational diabetes. *Med J Aust* 155:112, 1991
11. Moses RG: Screening for gestational diabetes mellitus. *Med J Aust* 157:500, 1992
12. Schmidt LE, Cox MS, Buzzard M, Cleary PA: Reproducibility of a comprehensive diet history in the Diabetes Control and Complications Trial. *J Am Diet Assoc* 94:1392–1397, 1994
13. Tsivis E: *The Australianisation of American Dietary Research Materials and the Development of an Extensive Food Data Base, Focussing on Fatty Acid Composition Including Omega Three and Omega Six Fatty Acids.* MSc (Nutrition and Dietetics) thesis. Wollongong, Australia, University of Wollongong, 1995
14. Tapsell L: *The Dietetic Interview: Towards a Critical Social Pedagogy.* PhD thesis. Wollongong, Australia, University of Wollongong , 1995
15. Commonwealth Department of Community Services and Health: *Nutrient Tables for Use in Australia: NUTTAB95.* Commonwealth Department of Health, Canberra, Australia, 1995
16. Schofield C, James WPT: Basal metabolic rate: review and prediction together with annotated bibliography of source material. *Human Nutrition* 39C (Suppl. 1):1–96, 1985
17. Goldberg G, Black A, Jebb S, Cole T, Murgatroyd P, Coward W, Prentice A: Critical evaluation of energy intake using fundamental principles of energy physiology. 1. Derivation of cut-off limits to identify under-recording. *Eur J Clin Nutr* 45:569–581, 1991
18. Mertz W: Food intake measurements: is there a "gold standard"? *J Am Diet Assoc* 92:1463–1465, 1992
19. Neilsen R, Tapsell L: Gestational diabetes: weight control and dietary intake after pregnancy. *Aust J Nutr Diet* 51:125–127, 1994
20. Van Staveren WA, West CE, Hoffmans MDAF, Bos P, Kardinaal AFM, van Popel GAFC, Schipper HJ, Hautvast JGAJ, Hayes RB: Comparison of contemporaneous and retrospective estimates of food consumption made by a diet history method. *Am J Epidemiol* 123:884–893, 1986
21. Willett WC, Sampson L, Brown ML, Stampfer MJ, Rosner B, Hennekens CH, Speizer FE: The use of a self administered questionnaire to assess diet four years in the past. *Am J Epidemiol* 127:188–189, 1988
22. Kühl C: Insulin secretion and insulin resistance in pregnancy and GDM: implications for diagnosis and management. *Diabetes* 40 (Suppl. 2):18–24, 1991
23. Storlien L, Baur LA, Kriketos AD, Pan DA, Cooney GJ, Jenkins AB, Calvert GD, Campbell LV: Dietary fats and insulin action. *Diabetologia* 39:621–631, 1996
24. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 277:472–477, 1997
25. Marshall JA, Hamman RF, Baxter J: High-fat, low-carbohydrate diet and the etiology of non-insulin-dependent diabetes mellitus: the San Luis Valley Diabetes Study. *Am J Epidemiol* 134:590–603, 1991
26. Feskens EJM, Kromhout D: Habitual dietary intake and glucose tolerance in euglycemic men: the Zutphen Study. *Int J Epidemiol* 19:953–959, 1990
27. Feskens EJM, Stengärd J, Virtanen SM, Pekkanen J, Räsänen L, Nissinen A, Tuomilehto J, Kromhout D: Dietary factors determining diabetes and impaired glucose tolerance. *Diabetes Care* 18:1104–1112, 1995
28. Borkman M, Storlien LH, Pan DA, Jenkins AB, Chisholm DJ, Campbell LV: The relationship between insulin sensitivity and the fatty acid composition of phospholipids of skeletal muscle. *N Engl J Med* 328:238–234, 1993