

## Research Article

# Relationship between caloric intake and pregnancy outcome in diet-treated gestational diabetes mellitus

Lai-Fong Ho, BSc(N),<sup>1</sup> Iris F. F. Benzie, PhD<sup>2</sup> and Terence T. Lao, MD<sup>1</sup>

<sup>1</sup>Department of Obstetrics and Gynecology, Queen Mary Hospital and <sup>2</sup>School of Nursing, The Hong Kong Polytechnic University, Hong Kong, China

### Abstract

A prospective observational study was performed to examine the relationship between estimated caloric intake with maternal blood glucose profile and infant outcome in a group of Chinese women with gestational diabetes mellitus (GDM) treated by diet alone. Following the diagnosis of GDM according to the World Health Organization (WHO) criteria, and using a 75 g oral glucose tolerance test (OGTT), 62 non-obese Chinese women who had completed a 5-day dietary survey were recruited into the study. They were categorized by their mean caloric intake over 5 days into tertiles for comparison of gestational weight gain, glycemic control and pregnancy outcome. Subjects in the highest tertile had a mean intake close to the prescribed intake. No differences were seen in maternal characteristics, gestational weight gain, OGTT, other postprandial glucose concentrations, or infant outcome among the tertiles. Our findings indicated that as long as the caloric intake did not exceed that prescribed, the infant outcome remained satisfactory.

### Key words

caloric intake, diet therapy, gestational diabetes mellitus, glycemic control, pregnancy outcome.

## INTRODUCTION

Gestational diabetes mellitus (GDM) refers to glucose intolerance of varying degrees that is first diagnosed during pregnancy (Alberti & Zimmet, 1998). GDM impacts on pregnancy outcome across different ethnic groups (Green *et al.*, 1990; Berkowitz *et al.*, 1992; Dornhorst *et al.*, 1992; Yue *et al.*, 1996; Coustan & Carpenter, 1998). Achieving normoglycemia is the targeted standard of care in the management of GDM pregnancies (Langer *et al.*, 1989) as this is the major factor in normalizing pregnancy outcome (Hod *et al.*, 1998).

In achieving normoglycemia, dietary management forms the basis of therapy in GDM. Caloric restriction improves insulin action and fuel disposition in obese non-gravid subjects with type II diabetes mellitus (Metzger & Freinkel, 1987), and in GDM, 80% of women can be managed effectively with diet alone (Inzucchi, 1999). Following the diagnosis of GDM, the

general approach is to commence nutritional counseling and diet therapy together with blood glucose monitoring, and insulin therapy is prescribed in addition when necessary (Fagen *et al.*, 1995; Metzger & Coustan, 1998; Hollingsworth & Moore, 1999; Inzucchi, 1999).

Diet therapy is generally prescribed by a dietitian, following an assessment of intake using some form of questionnaire. However, there are few local data on the relationship between actual caloric intake and maternal glycemic control, and overall the evidence base in this area is of poor quality (Dornhorst & Frost, 2002). It is generally assumed that unsatisfactory glycemic control is due to poor dietary compliance and over-intake of calories. The usual approach is to restrict or adjust further the caloric intake before insulin therapy is considered or prescribed. However, dietary restraint could also have undesirable influences on eating patterns and gestational weight gain (Conway *et al.*, 1999), and the effects of diet therapy have not been evaluated against the potential risk in pregnancies complicated by GDM.

To explore the relationship between caloric intake and glycemic control following the commencement of diet treatment in patients with GDM, we performed a

Correspondence address: Ms Lai-Fong Ho, Department of Obstetrics and Gynecology, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong, China. Email: ho156@i-cable.com

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prospective observational study using a 5-day dietary survey (Livingstone, 1995) on subjects who had commenced diet therapy in consultation with a dietitian. The objectives of the study were to examine the relationships between: (i) estimated and prescribed caloric intake; (ii) estimated caloric intake and short-term glycemic control assessed using the postprandial blood glucose profile; and (iii) estimated caloric intake and long-term control using gestational weight gain as the surrogate. We also examined the relationship between caloric intake and infant birth weight, placental weight, and the incidence of large- and small-for-gestational age infants.

## METHODS

Our hospital is a referral center serving a population that is predominantly (95%) ethnic Chinese. An in-house program to screen for GDM has been implemented for more than a decade. The screening, diagnosis and management of GDM in our hospital have been described in previous reports (Lao & Lee, 1998; Lao & Tam, 2001). At the time of the first antenatal visit, women identified to have risk factors for the development of GDM, such as advanced age (> 34 years), relevant past obstetric and family history, obesity, and recurrent or significant glycosuria, undergo the 75 g oral glucose tolerance test (OGTT). In addition, all women have random plasma glucose testing at the beginning of the third trimester (28–30 weeks) to screen for glucose intolerance. Those with abnormal spot plasma glucose ( $> 5.8 \text{ mmol L}^{-1}$  if  $< 2 \text{ h}$  postprandial and  $> 5.0 \text{ mmol L}^{-1}$  if  $> 2 \text{ h}$  postprandial) also undergo OGTT. The result of the OGTT is interpreted using the World Health Organization (WHO) criteria (Alberti & Zimmet, 1998), and the categories of impaired glucose tolerance (IGT, OGTT 2-h value  $\geq 7.8 < 11.1 \text{ mmol L}^{-1}$ ) and diabetes mellitus (DM, OGTT 2-h value  $\geq 11.1 \text{ mmol L}^{-1}$ ) are both managed as GDM as recommended. Women diagnosed with GDM are referred to a dietitian for dietary advice, and a blood glucose profile (fasting and 2 h after breakfast, lunch and dinner glucose levels) is performed around 1 week after their initial dietary consultation. There are approximately 4000 deliveries in our hospital each year and the prevalence of GDM is 10–12%.

For this study, Chinese women with GDM were recruited at the time of their first blood glucose profile assessment in the day ward following the commencement of diet therapy. These subjects had blood sampling at specified times with respect to their meals, but they were free to come and go from the day ward and to pursue any activities that they would prefer. The subjects approached for recruitment for this study

were all within 90–120% of ideal body weight and in whom the prescribed caloric intake was 30 kcal/kg. The other inclusion criteria were: ethnic Chinese who could speak and understand Cantonese, singleton pregnancy, and the only complication being GDM. Women with pre-existing DM, underlying medical complications requiring dietary restriction or control, or multiple pregnancies, were excluded. A sample size of 50 was estimated to be large enough to generate meaningful data as preliminary power calculations indicated that 50 subjects are adequate to detect a 6 kcal/kg difference in dietary intake at the 5% level with 80% certainty.

Following explanation and written consent, a dietary survey form in Chinese, modified from the diet assessment form used by our dietitians to cover a 5-day period from the day prior, to the third day following the blood glucose profile was used. This 5-day period would cover one weekend. There is no gold standard for measuring food intake. Weighed intakes are the preferred method, but a 5–7 day survey is a validated method for providing an estimation of the actual caloric intake (Livingstone, 1995) and is regarded as more reliable than the 24-h recall or 2–4 days dietary diary methods used in many previous studies. The average daily intake can be used to categorize the subjects for comparison of the variable of interest. In this study, subjects were instructed to record the food and drink components in their breakfast, lunch, dinner and snacks. Portions were not weighed, but a ‘handy’ way to estimate serving and portion size by visualizing the portions and relating them to the size of the hand or fist (Faculty of Applied Health Sciences, University of Waterloo, Canada) was used in this study to obtain a more accurate estimation of the amount of intake.

Both the diet assessment form, as well as the methods of estimation of the amount of intake, have been validated and adopted for use in our hospital by the Dietetics Department. The survey forms were distributed and the food diary was started 1 week after seeing the dietitian. The total caloric intake for each day was estimated using the Nutritionists IV Diet Analysis Database (1995) together with a locally generated database for Chinese food, and the mean daily intake in the 5-day survey was calculated. Subjects were then categorized by their average daily intake into tertiles for analysis. Maternal demographic data, weight at each visit as recorded by an electronic scale, height taken during the first visit, results of blood glucose profile and the pregnancy outcome were retrieved from the hospital records after delivery. Our department uses only the glucose profile and not HbA1c to assess glycemic control in GDM as HbA1c is not sensitive enough for glucose intolerance of such a short duration.

For statistical analysis, continuous variables were analyzed with the *t*-test and one-way ANOVA method, with post-hoc analysis by Duncan's multiple range test set at the 5% level. Categorical variables were analyzed by the chi-squared test and Spearman's correlation coefficient (*rho*). Statistical calculation was performed using a commercially available statistical package (SPSS version 10.0).

## RESULTS

Of the 88 women initially approached, three were excluded because one was illiterate and two carried a twin pregnancy. Twelve survey forms were not returned and 11 had incomplete data. Therefore, the final number of subjects completing the survey and included in the analysis was 62. None of these subjects required insulin therapy, nor were they obese. Subjects were categorized into three groups according to the tertile ranking of their average estimated total daily caloric intake (Table 1). Weight gain for the whole pregnancy was defined as the difference between the immediate pre-delivery and the recalled pre-pregnancy weight. Weight gain following diagnosis was

defined as the difference between the immediate pre-delivery weight and the weight at the time of the OGTT. There were no significant differences in age, weight, height, body mass index (BMI) or weight gain amongst the tertiles.

The mean caloric intake on days 1–5 in the three groups is shown in Table 2. Only the highest tertile had a mean caloric intake that was close to that prescribed. The degree of glucose intolerance and glycemic control is shown in Table 3. There was no significant difference in the fasting or 2-h glucose values of, or the gestational age at, OGTT. For glucose profile, no difference was found in the post-breakfast or post-lunch glucose concentrations, but the post-dinner ( $P < 0.02$ ) and mean postprandial glucose concentrations ( $P < 0.05$ ) were significantly higher in the highest tertile group. Although no correlation between mean caloric intake with any of the postprandial glucose concentrations was found, repeat analysis using partial correlation and controlling for pre-pregnancy weight and height demonstrated a significant correlation between mean caloric intake with post-dinner glucose concentration ( $P = 0.012$ ) but not for mean postprandial glucose concentration ( $P = 0.092$ ).

**Table 1.** Maternal characteristics and gestational weight gain according to caloric intake in tertiles

	Tertiles			<i>P</i> -value
	Highest ( <i>n</i> = 21)	Middle ( <i>n</i> = 20)	Lowest ( <i>n</i> = 21)	
Age (years)	34.6 ± 4.9	34.8 ± 4.4	35.1 ± 2.7	NS
Weight (kg)	56.0 ± 6.8	55.7 ± 9.0	55.3 ± 9.1	NS
Height (cm)	156.5 ± 5.3	157.5 ± 7.0	155.8 ± 5.3	NS
BMI (kg/m <sup>2</sup> )	23.0 ± 3.2	22.4 ± 3.2	23.1 ± 4.2	NS
Weight gain (kg)				
Whole pregnancy	9.2 ± 3.6	10.0 ± 3.8	10.0 ± 4.3	NS
After diagnosis	6.3 ± 3.9	7.7 ± 3.4	5.8 ± 3.8	NS

Results expressed as mean ± SD. BMI, body mass index; NS, not significant.

**Table 2.** Caloric intake (kcal) according to tertiles

	Tertiles			Overall mean
	Highest ( <i>n</i> = 21)	Middle ( <i>n</i> = 20)	Lowest ( <i>n</i> = 21)	
Day 1	2189.5 ± 300.4	1638.0 ± 112.2	1214.4 ± 149.0	1681
Day 2	1733.8 ± 358.3	1586.6 ± 269.1	1476.0 ± 367.8	1599
Day 3	1992.7 ± 471.9	1789.4 ± 299.9	1398.0 ± 295.3	1733
Day 4	1746.7 ± 419.1	1722.4 ± 299.9	1319.9 ± 309.2	1598
Day 5	1863.2 ± 378.1	1692.1 ± 325.1	1384.1 ± 329.3	1647
Mean/day	1863	1692	1384	

Results expressed as mean ± SD.

**Table 3.** Relationship between glycemic control and caloric intake in tertiles

	Highest ( <i>n</i> = 21)	Middle ( <i>n</i> = 20)	Lowest ( <i>n</i> = 21)	<i>P</i> -value
OGTT (mmol/L)				
Fasting	4.7 ± 0.5	4.6 ± 0.4	4.7 ± 0.6	NS
2 h	9.1 ± 1.8	8.9 ± 0.9	9.2 ± 1.5	NS
Gestation at OGTT (week)	19.4 ± 8.5	16.5 ± 7.6	20.9 ± 7.4	NS
Glucose concentration (mmol/L)				
Post-breakfast	5.5 ± 1.0	5.2 ± 6.7	5.4 ± 1.0	NS
Post-lunch	6.4 ± 1.3	5.7 ± 0.9	6.2 ± 0.9	NS
Post-dinner	6.9 ± 1.2*	5.9 ± 0.9	6.3 ± 1.0	< 0.02
Mean	6.2 ± 0.8*	5.6 ± 0.6	5.9 ± 0.9	< 0.05

\* Duncan's test *P* < 0.05 compared with middle tertile. Results expressed as mean ± SD. NS, not significant; OGTT, oral glucose tolerance test.

**Table 4.** Relationship between pregnancy outcome and caloric intake in tertiles

	Highest ( <i>n</i> = 21)	Tertiles		<i>P</i> -value
		Middle ( <i>n</i> = 20)	Lowest ( <i>n</i> = 21)	
Gestation (week)	38.7 ± 1.7	38.4 ± 1.4	38.6 ± 1.1	NS
Birthweight (kg)	2.99 ± 0.46	3.09 ± 0.57	3.17 ± 0.37	NS
Crown heel length (cm)	49.4 ± 1.7	49.4 ± 2.2	49.9 ± 1.3	NS
Apgar score				
1st minute	9.0 ± 1.2	9.1 ± 0.5	8.8 ± 0.8	NS
5th minute	9.8 ± 0.4	9.9 ± 0.5	9.8 ± 0.5	NS
LGA (%) <sup>†</sup>	2 (9.5)	3 (15.0)	4 (19.0)	NS
SGA (%) <sup>†</sup>	4 (19.0)	3 (15.0)	3 (14.3)	NS
Placental weight (g)	576 ± 157	617 ± 115	653 ± 133	NS

Results expressed as mean ± SD and in number (%) where indicated by <sup>†</sup>. LGA, large-for-gestational age (> 90th percentile); NS, not significant; SGA, small-for-gestational age (= 10th percentile).

When the pregnancy (infant) outcome was analyzed, there was no significant difference between caloric intake tertiles in gestational age, birthweight, crown heel length, Apgar scores at the first and fifth minute, incidence of large-for-gestational age (LGA, birthweight > 90th percentile) or small-for-gestational age (SGA, birthweight < 10th percentile) infants, or the placental weight (Table 4). Neither the incidence of LGA nor SGA infants correlated significantly with caloric intake. When birthweight and placental weight were correlated with mean caloric intake, a significant inverse correlation was found only for placental weight ( $\rho = -0.331$ ,  $P = 0.016$ ). However, the correlation between mean caloric intake with placental weight failed to maintain statistical significance ( $P = 0.078$ ) when repeat analysis was performed with partial correlation and controlling for pre-pregnancy weight and height.

## DISCUSSION

Low energy dietary therapy has been used successfully in the management of GDM, and infants of diabetic mothers treated with diet alone have birthweights similar to those in the control group (Dornhorst *et al.*, 1991). However, dietary restriction cannot be taken for granted as the optimal treatment for GDM as tight dietary control may result in acute and chronic fetal nutritional deprivation which is linked to retarded growth and substrate limitations (Langer *et al.*, 1989). Hence there is a safety concern for the developing infant (Jovanovic-Peterson & Peterson, 1996; Langer & Hod, 1996). Furthermore, in women with GDM managed with diet alone, carbohydrate restriction may potentiate the accelerated ketosis of pregnancy and increase ketone body concentrations above those of normal control subjects (Jovanovic, 1999).

As there is paucity of randomized clinical trials on diet therapy in GDM, it is only sensible that the optimal dietary prescription should be a diet that provides adequate nourishment for the mother and fetus while limiting weight gain and avoiding postprandial hyperglycaemia (Dornhorst & Frost, 2002). It has been shown that with a prescribed diet between 1200 kcal and 1800 kcal, gestational weight gain was significantly less for the gestational diabetic mothers (mean  $\pm$  SD  $8.6 \pm 4.9$  kg), when compared with the general prenatal population ( $9.3 \pm 5.3$  kg) and control (non-GDM) subjects ( $9.7 \pm 5.3$  kg) (Dornhorst *et al.*, 1991). This suggests that gestational weight gain might be a surrogate for the degree of dietary compliance and diabetic control. However, it remains unclear whether maternal glucose profile or gestational weight gain are correlated with total caloric intake in actual practice, and whether one is superior to the other in the monitoring of the maternal response to diet therapy.

In this study we confined the recruitment to subjects who were prescribed a daily intake of 30 kcal/kg, and the maternal pre-pregnancy weight in the three groups indicated that subjects in the three tertile groups were essentially similar. However, despite the similarity in weight, the majority appeared to over-restrict their caloric intake to below the prescribed level, and the average intake was close to that prescribed only in the highest tertile group. This unexpected observation suggested that recruitment into this study could have been a cause for the inadvertent over-restriction, perhaps in order to ensure good results with their dietary survey and glucose profile; a phenomenon likened to the Hawthorne Effect (Wickstrom & Bendix, 2000).

Contrary to expectation, there was no significant difference in gestational weight gain between the three intake groups, whether assessed over the whole pregnancy or following the diagnosis of GDM, as has been reported previously (Dornhorst *et al.*, 1991). This finding suggests, nevertheless, that the small differences and variations in calorie intake following the commencement of diet therapy are unlikely to be reflected in changes in maternal weight in subjects with near-ideal weight, and that weight gain is of limited value as a surrogate for dietary compliance in these subjects.

The only significantly different glycemic parameter found between the three groups after controlling for maternal weight and height was the post-dinner glucose concentration, with the concentration in the highest tertile significantly higher compared with the other tertiles. It is tempting to explain this difference on the basis of their higher caloric intake, but higher caloric

intake could not have explained the absence of any difference between the other postprandial glucose concentrations, or the lack of any difference between the middle and lowest tertile groups. There is therefore potential risk for the fetus if we only monitor the glucose profile because subjects who had over-restricted their intake could not be identified in this way. Future studies on this issue are warranted.

When pregnancy outcome was analyzed, there was no difference in the mean birthweight, the mean placental weight, or prevalence of SGA or LGA infants, or Apgar scores, among the three groups. It is likely that in gestational diabetic women with near-ideal weight who are managed successfully with diet alone, the infant outcome is not significantly affected by the caloric intake as long as the glucose profile is within the therapeutic range. However, of these 62 women, 10 (16.1%) and nine (14.5%) gave birth to SGA and LGA infants, respectively. The SGA figures are high when compared with those of an earlier study of 568 diet-treated GDM women in our hospital, in which the prevalence of SGA and LGA infants was 6.9% and 19.4% (Leung & Lao, 2000). The much higher prevalence of SGA infants in the current study was unexpected and could not be explained on account of caloric intake alone. It was possible that because most of our subjects were non-obese, a calorie-restricted diet as prescribed here could have significantly reduced the calories available to the fetus. While the underlying mechanism remained to be elucidated, our results would support the concept that glycemic control by tight dietary control may result in acute and chronic fetal nutritional deprivation which is linked to retarded growth and substrate limitations (Langer *et al.*, 1989), and suggest that the efficacy of diet therapy as the sole treatment for GDM in non-obese subjects needs to be reappraised.

In conclusion, this study attempted to study the food assessment/glycemia/birthweight interface in Chinese women with GDM. The multiple problems of dietary assessment are well recognized, and our study has shown that neither the postprandial blood glucose profile nor gestational weight gain in non-obese gestational diabetic women managed with diet therapy necessarily reflect their actual caloric intake. There was a tendency for our patients to over-restrict their intake, and the prevalence of SGA infants was unexpectedly high. Nevertheless, our results also suggest that apart from the incidence of SGA infants, the perinatal outcome remained satisfactory as long as the caloric intake did not exceed that prescribed. This study may stimulate others in the field to try to further address the problems of dietary therapy in non-obese women with GDM.

## REFERENCES

- Alberti KGMM, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus, Provisional Report of a WHO Consultation. *Diabet. Med.* 1998; **15**: 539–553.
- Berkowitz GS, Lapinski RH, Wein R, Lee D. Race/ethnicity and other risk factors for gestational diabetes. *Am. J. Epidemiol.* 1992; **135**: 965–973.
- Conway R, Reddy S, Davies J. Dietary restraint and weight gain during pregnancy. *Eur. J. Clin. Nutr.* 1999; **53**: 849–853.
- Coustan DR, Carpenter MW. The diagnosis of gestational diabetes. *Diabetes Care* 1998; **21**: B5–B8.
- Dornhorst A, Frost G. The principles of dietary management of gestational diabetes: Reflection on current evidence [Research Papers]. *J. Hum. Nutr. Diet.* 2002; **15**: 145–156.
- Dornhorst A, Nicholls JS, Probst F *et al.* Calorie restriction for treatment of gestational diabetes. *Diabetes* 1991; **40** (Suppl 2): 161–164.
- Dornhorst DA, Paterson CM, Nicholls JSD *et al.* High prevalence of gestational diabetes in women from ethnic minority groups. *Diabet. Med.* 1992; **9**: 820–825.
- Faculty of Applied Health Sciences, University of Waterloo, Canada. A ‘Handy’ Way to Estimate Serving and Portion Size. [Revised on 2001, cited March 2002] Available from URL: <http://www.ahs.uwaterloo.ca/~kh346/html/handy.htm>.
- Fagen C, King JD, Erick M. Nutritional management in women with gestational diabetes mellitus: A review by ADA’s Diabetes Care and Education Dietetic Practice Group. *J. Am. Diet. Assoc.* 1995; **95**: 460–467.
- Green JR, Pawson IG, Schumacher LB, Perry J, Kretchmer N. Glucose tolerance in pregnancy: Ethnic variation and influence of body habitus. *Am. J. Obstet. Gynecol.* 1990; **163**: 86–92.
- Hod M, Itzhak M, Bar J *et al.* Antepartum management protocol. Timing and mode of delivery in gestational diabetics. *Diabetes Care* 1998; **21** (Suppl 2): B113–B117.
- Hollingsworth DR, Moore TR. Diabetes in pregnancy. In: Creasy RK, Resnick R (eds). *Maternal-Fetal Medicine: Principles and Practice*, 4th edn. Philadelphia: WB Saunders, 1999; 939–954.
- Inzucchi SE. Diabetes in pregnancy. In: Burrows G, Duffy T (eds). *Medical Complications During Pregnancy*, 5th edn. Philadelphia: WB Saunders, 1999; 25–51.
- Jovanovic L. Time to reassess the optimal dietary prescription for women with gestational diabetes. *Am. J. Clin. Nutr.* 1999; **70**: 1–4.
- Jovanovic-Peterson L, Peterson CM. Review of gestational diabetes mellitus and low-calorie diet and physical exercise as therapy. *Diabetes Metab. Rev.* 1996; **12**: 287–308.
- Langer O, Hod M. Management of gestational diabetes mellitus. *Obstet. Gynecol. Clin. North Am.* 1996; **23**: 137–159.
- Langer O, Levy J, Brustman L, Anyaegbunam A, Merkatz R, Divon M. Glycemic control in gestational diabetes mellitus: How tight is tight enough: small for gestational age versus large for gestational age? *Am. J. Obstet. Gynecol.* 1989; **161**: 646–653.
- Lao TT, Lee CP. Gestational ‘impaired glucose tolerance’: Should the cut-off be raised to 9 mmol<sup>-1</sup>? *Diabet. Med.* 1998; **15**: 25–29.
- Lao TT, Tam KF. Gestational diabetes mellitus diagnosed in third trimester pregnancy and pregnancy outcome. *Acta Obstet. Gynecol. Scand.* 2001; **80**: 1003–1008.
- Leung TW, Lao TT. Placental size and large-for-gestational-age infants in women with abnormal glucose tolerance in pregnancy. *Diabet. Med.* 2000; **17**: 48–52.
- Livingstone MB. Assessment of food intakes: Are we measuring what people eat? *Br. J. Biomed. Sci.* 1995; **52**: 58–67.
- Metzger B, Coustan DR. Summary and recommendations of the Fourth International Workshop-Conference on gestational diabetes mellitus. *Diabetes Care* 1998; **21**: B161–B187.
- Metzger BE, Freinkel N. Accelerated starvation in pregnancy: Implications for dietary treatment of obesity and gestational diabetes mellitus. *Biol. Neonate* 1987; **51**: 78–85.
- First Data Bank Division, The Hearst Corporation. *Nutritionist IV Diet Analysis Database (version 4.1)*. San Bruno, CA: First Data Bank Division, The Hearst Corporation, 1995.
- Wickstrom G, Bendix T. The ‘Hawthorne effect’—what did the original Hawthorne studies actually show? *Scan. J. Work Environ. Health* 2000; **26**: 363–367.
- Yue DK, Molyneaux LM, Ross GP, Constantiano MI, Child AG, Turtle JR. Why does ethnicity affect prevalence of gestational diabetes? The underwater volcano theory. *Diabet. Med.* 1996; **13**: 748–752.