2016

Relationship between fasting plasma glucose levels and maternal food group and macronutrient intakes in pregnancy

Laura Mullaney

Aisling Brennan

Shona Cawley

See next page for additional authors

Funder: UCD Centre for Human Reproduction; Danone Nutricia Early Life Nutrition

Authors

Laura Mullaney, Aisling Brennan, Shona Cawley, Amy C. O'Higgins, Daniel McCartney, and Michael J. Turner

Nutrition & Dietetics 2016; **73**: 441–447 DOI: 10.1111/1747-0080.12278

ORIGINAL RESEARCH

Relationship between fasting plasma glucose levels and maternal food group and macronutrient intakes in pregnancy

Laura MULLANEY,[1] Aisling BRENNAN,[1] Shona CAWLEY,[1] Amy C. O'HIGGINS,[2] Daniel MCCARTNEY[1] and Michael J. TURNER[2]

[1]School of Biological Sciences, Dublin Institute of Technology, Dublin 8, Republic of Ireland, and [2]UCD Centre for Human Reproduction, Coombe Women and Infants University Hospital, Dublin 8, Republic of Ireland

Abstract

Aim: Increased maternal body mass index (BMI) has been consistently associated with elevated blood glucose levels during pregnancy. Studies to date investigating the relationship between maternal blood glucose levels and dietary intake have shown mixed results. We investigated the association between maternal fasting plasma glucose (FPG) levels and food group and macronutrient intakes in the first trimester of pregnancy, after adjustment for maternal bodyweight.

Methods: Women were recruited after sonographic confirmation of an ongoing singleton pregnancy in the first trimester. Dietary information was collected using the validated Willett Food Frequency Questionnaire. Maternal height and weight were measured and BMI calculated. Body composition was measured using advanced bioelectrical impedance analysis. FPG levels were obtained for women who were selectively screened with a 75 g oral glucose tolerance test.

Results: No associations were observed between maternal FPG levels and food group or macronutrient intakes but higher energy and starch intakes were found in obese subjects ($P = 0.009$ and $P = 0.03$ respectively). On univariate analysis, higher FPG levels were associated positively with higher maternal bodyweight, BMI, body fat, fat free mass and visceral fat (all $P < 0.001$). However, on multivariate regression analysis, higher FPG levels remained associated only with maternal BMI > 29.9 kg/m^2 (OR 7.4, $P = 0.01$).

Conclusions: Our findings indicate that maternal BMI is the key determinant of maternal glycaemia. Interventions which focus on overall energy restriction and especially the limitation of dietary starch to optimise prepregnancy maternal bodyweight are likely to be useful in improving glycaemic control in higher risk pregnancies.

Key words: fasting plasma glucose, food group, gestational diabetes, obesity, pregnancy.

Introduction

Gestational diabetes mellitus (GDM) is associated with adverse outcomes not only for the woman, but also for her offspring.[1–3] GDM has been associated with increased caesarean section rates and pre-eclampsia, while women who develop GDM are also at increased risk of developing type 2 diabetes mellitus (T2DM) later in life.[4,5] Offspring of mothers with GDM are at risk of macrosomia, as well as obesity and T2DM later in life.[4,5] In women with GDM, higher levels of blood glucose pass through the placenta. This results in foetal hyperinsulinaemia and hyperglycaemia leading to an increase in foetal fat and protein stores, and subsequently macrosomia.[6]

While the definition of GDM as glucose intolerance with onset or first recognition during pregnancy is largely accepted, the exact level of glucose intolerance which defines GDM remains contentious.[1–4,7,8] The Hyperglycaemia and Adverse Pregnancy Outcome study found a linear association between maternal plasma glucose (PG) levels and adverse perinatal outcomes across the whole distribution of PG levels in pregnancy.[9] Thus, there is no clear PG threshold above which women and their offspring are at high clinical risk and below which they are at low risk. Criteria for the diagnosis of GDM have been developed, however, in an attempt to identify thresholds which best predict adverse maternal and neonatal outcomes. Unfortunately,

L. Mullaney, BSc, Research Dietitian
A. Brennan, BSc, Dietitian
S. Cawley, BSc, Research Dietitian
A.C.O'Higgins, MD, Obstetric Specialist Registrar
D. McCartney, PhD, Lecturer in Human Nutrition and Dietetics
M.J. Turner, FRCOG, Professor of Obstetrics and Gynaecology
Correspondence: L. Mullaney, School of Biological Sciences, Dublin Institute of Technology, Dublin 8, Republic of Ireland. Tel: +353-1-4085786; Fax: +353-1-4085760. Email: lauraemullaney@gmail.com

Accepted February 2016

Nutrition & Dietetics

clear evidence demonstrating improved clinical outcomes through the use of one criterion over another has remained elusive. This has led to the use of several different criteria for the diagnosis of GDM which are arbitrary and often based on expert opinion.[7] Diagnosis of GDM can be further complicated by poorly controlled pre-analytical handling of the fasting glucose sample.[8]

Diet and physical activity level (PAL) have been proposed as modifiable risk factors for the development of GDM.[10–16] However, diet and lifestyle interventions to enhance blood glucose control in pregnancy have yielded inconsistent results.[17] Conversely, it is established that the risk of developing GDM is increased in women with higher prepregnancy body mass index (BMI), and that this risk increases progressively across the BMI categories of overweight and obesity.[18–20] Total body fat mass has also been linked to insulin resistance.[21–23] However, there is a lack of studies examining the association between maternal fat mass and glycaemic control during pregnancy.

Effective interventions to prevent and treat GDM are important to reduce the short- and long-term adverse health consequences for women and their offspring. The aim of this study was to investigate the association between maternal FPG levels and energy intake (EI), PAL, food group intake and macronutrient intake in the first trimester of pregnancy after adjustment for bodyweight and other potential confounders.

Methods

The Coombe Women and Infants University Hospital is one of the largest maternity hospitals in the European Union (EU) and cares for women from all socioeconomic groups and from across the urban–rural divide. Women were recruited at their convenience between February and August 2013 as part of a longitudinal study investigating maternal weight trajectories.[24,25] The women's clinical and socio-demographic details were computerised routinely at the first antenatal visit. The main inclusion criteria were women booking for antenatal care after an ultrasound confirmation of a singleton ongoing pregnancy in the first trimester. Exclusion criteria included multiple pregnancies, women with pre-existing diabetes or women who subsequently delivered in another hospital.

To collect habitual food and nutrient intakes, women were asked to complete the previously validated semi-quantitative Willett Food Frequency Questionnaire (WFFQ).[26–28] Socioeconomic, health behavioural and physical activity data were also collected using an online questionnaire. Height was measured to the nearest centimetre using a Seca wall-mounted digital metre stick with the woman standing in her bare feet. Weight was measured digitally to the nearest 0.1 kg and BMI calculated. Body composition was measured using an eight-lead multi-frequency bioelectrical impedance analyser (BIA) (Tanita MC 180, Tokyo, Japan).[29,30]

Of a total study population of 524 women, oral glucose tolerance tests (OGTT) were performed between weeks 24 and 28 of gestation on a cohort 180 women identified to have risk factors for GDM according to national screening guidelines.[24,31] Written informed consent was obtained from all study participants. The study was approved by the Coombe Women and Infants University Hospital Research Ethics Committee.

The FFQ used was a self-administered WFFQ adapted from the European Prospective Investigation into Cancer and Nutrition study and validated for use in Irish adults.[26,27,32] This WFFQ has also been recently validated in an Irish obstetric population.[28] Frequency of consumption of a 'standard portion' of each food or beverage item consumed was divided into nine categories, ranging from 'never or less than once per month' to 'six or more times per day'. A 'standard portion' was quantified using the Food Standards Agency's Average Portion Sizes reference text.[33] This dietary assessment protocol captured food and nutrient data reflective of the periconceptual period, as the WFFQ focuses on consumption patterns over the previous year. The WFFQ food intake data were entered into WISP version 4.0 (Tinuviel Software, Llanfechell, Anglesey, UK) to convert these reported food intakes into nutrient intakes. The food composition tables used in WISP are derived from McCance and Widdowson's *Food Composition Tables* 5th and 6th editions, and all supplemental volumes.[34]

The clinical and health behavioural data collected included any applicable medical conditions and medications, as well as the woman's smoking status. Questions collecting socioeconomic data were derived from the EU Survey on Income and Living Conditions 2012.[35,36] Material indices of disadvantage included relative income poverty, as well as relative deprivation, while consistent poverty status was also calculated using these two parameters. Relative income poverty status was calculated by comparing equivalised household income against the 60% median income threshold. Relative deprivation was assessed by determining whether women had experienced the enforced absence (due to financial constraint) of two or more basic necessities from a list of 11 over the previous year. Consistent poverty was identified if a woman's equivalised household income fell below the relative income poverty threshold, in addition to experiencing the enforced absence of two or more of the 11 basic markers of deprivation over the preceding 12 months.

Self-assessed habitual PALs were also collected using a self-administered, unsupervised questionnaire. Individual PAL was estimated for each woman from 1.45 metabolic equivalents (MET) (seated work with no option of moving around and no strenuous leisure time activity), up to 2.20 MET (strenuous work or highly active leisure time (e.g. competitive athletes in daily training).[37]

Women who under- and over-reported EI were excluded from the final food and nutrient intake datasets as previously described[24] so as to enhance the integrity of our analyses.[38] Data analyses were carried out using SPSS version 20.0 (IBM Corporation, Armonk, NY, USA). Descriptive analyses were initially carried out to characterise the cohort with respect to their age, parity, ethnicity, stage of gestation,

socioeconomic status, smoking status and PAL. One-way ANOVA tests were used to compare mean values for continuous variables (age, gestational age, PAL) between the FPG tertiles. Cross-tabulation with chi-square analyses were used to test differences in categorical socioeconomic and health behavioural variables across the FPG tertiles. Data for weight, BMI, body fat mass, percentage body fat and fat free mass were non-normally distributed. Kruskal–Wallis tests were used to assess differences in these parameters between the FPG tertiles. Kruskal–Wallis tests were also used to test differences in median energy-adjusted food group and macronutrient intakes among women in each FPG tertile. Binary logistic regression was used to assess factors associated with FPG levels >4.5 mmol/L. This regression model incorporated variables such as antenatal obesity, family history of diabetes, early pregnancy weight, body fat %, fat free mass, visceral fat level, age, parity, smoking status, Irish nativity, glycaemic index of the diet and EI, sugar, carbohydrate, protein, fat and dietary fibre intake. Mann–Whitney *U*-test was used to assess differences in energy-adjusted macronutrient intakes between obese and non-obese women.

Results

OGTTs were undertaken by 180 women. GDM was diagnosed in 16 women (8.9%) according to the International Association of Diabetes and Pregnancy Study Group recommendations.[39] Mean FPG levels were 4.5 mmol/L (range 3.6–8.9 mmol/L). The social and demographic characteristics of this study population both overall, and according to FPG level, are shown in Table 1. Women completed the WFFQ at 12.6 ± 2.8 weeks gestation. FPG levels increased with increasing weight, BMI, body fat mass, percentage body fat, fat free mass, and visceral fat (all $P < 0.001$) (Table 2).

EI under-reporting was observed in 57 women (31.7%). There were no EI over-reporters in the sample. EI under-reporters in this sample had a higher weight (87.1 ± 19.3 *vs* 73.9 ± 15.2 kg ($P = 0.001$)), BMI (32.0 ± 7.1 *vs* 26.9 ± 5.5 kg/m^2 ($P = 0.001$)), body fat % (37.1 ± 7.4 *vs* 32.4 ± 7.4% ($P = 0.001$)), and fat free mass (53.6 ± 7.4 *vs* 49.0 ± 5.9 kg ($P = 0.001$)) compared with plausible reporters of EI. No differences were seen in energy-adjusted food group and macronutrient intakes across FPG tertiles (Table 3).

On logistic regression only antenatal obesity (BMI > 29.9 kg/m^2; OR 7.4, $P = 0.01$) was associated with a FPG level >4.5 mmol/L. Obese plausible reporters (n = 35) had a higher EI (3254.9 *vs* 2281.5 kcal/day ($P = 0.009$)), higher starch intake (28.2 *vs* 24.2% total energy (TE) ($P = 0.03$)), higher maltose intake (0.65 *vs* 0.45% TE ($P = 0.04$)) and lower fructose intake (3.37 *vs* 3.88% TE ($P = 0.03$)) compared with non-obese women. There was no difference in self-reported PAL between obese and non-obese women (1.76 ± 0.2 *vs* 1.75 ± 0.2 ($P = 0.598$)).

One- and two-hour post glucose load PG levels also showed no association with maternal food and macronutrient intakes. The one-hour PG levels also increased as maternal weight, BMI and body composition increased. Interestingly the two-hour PG levels were not as significant as the FPG or one-hour PG levels. Only BMI increased as the two-hour PG levels increased ($P = 0.03$).

Discussion

This study found that maternal FPG levels at 24–28 weeks gestation were not associated with food group and macronutrient intakes in the periconceptional period. Obesity in early pregnancy was associated with higher FPG levels after adjusting for important confounding variables. This suggests that weight management interventions should be targeted at women of child-bearing age in the prepregnancy period, especially those who are obese. These weight management programmes should incorporate limitations on overall dietary EI particularly that derived from starchy foods, as high intakes of both were associated with maternal obesity.

Our study has a number of strengths. Maternal weight was measured, not self-reported. While the accurate assessment of bodyweight is critical, women, particularly those who are obese, have been shown to commonly underestimate their weight when self-reporting, which may lead to BMI mis-categorisation.[40,41] BIA was used to measure maternal weight and body composition.[29,30] The maternal weight was taken in the first trimester, which has been shown to be the optimal time for weight measurement in pregnancy, as maternal weight and body composition only begin to change after 18 weeks of gestation.[30] The availability and use of the women's body composition data is another strength of this study. Given the lack of clear consensus around the exact level of glucose intolerance which defines GDM, FPG levels were investigated in this study.[1–4,7,8]

A possible limitation of this study is the difficulty associated with accurate assessment of dietary intake. The WFFQ is a semi-quantitative FFQ and, therefore, does not facilitate portion size estimation for individuals. Nonetheless, the WFFQ has been validated as a dietary data collection instrument in several Irish population studies, including a recent study on pregnant women in Dublin.[26–28,32] Another potential weakness is that convenience recruitment may introduce an unforeseen self-selection bias that was not addressed in the multivariate analysis.

Women who under-reported their EI were excluded from the final food and nutrient intake datasets to enhance the integrity of the population's nutrient intake data.[38] Under-reporting of EI is a phenomenon associated with dietary surveys and must be taken into account when interpreting the results of such surveys.[24] Specifically, under-reporting of EI is increased amongst women in higher BMI categories and, therefore, needs to be considered when conducting research into GDM as increased BMI is associated with the development of GDM. Disproportionate exclusion

Table 1 Social and demographic characteristics of the study population analysed by fasting plasma glucose (FPG) levels in early pregnancy (n = 180)

	Total (n = 180)	*Lower FPG (<4.3 mmol/L) (n = 63)*	*Moderate FPG (4.3–4.59 mmol/L) (n = 63)*	*Higher FPG (≥4.6 mmol/L) (n = 54)*	P
Age[(a)] (years)	30.6 (5.5)	30.4 (5.4)	30.2 (5.8)	31.2 (5.1)	0.58
Nulliparous % (n)	41.1 (74)	38.1 (24)	39.7 (25)	46.3 (25)	0.64
Relative income poverty[(b)] % (n)	22.6 (39)	19.1 (12)	20.6 (13)	25.9 (14)	0.55
Relative deprivation % (n)	32.2 (58)	33.3 (21)	31.8 (20)	31.5 (17)	0.97
Consistent poverty[(b)] % (n)	11.1 (19)	11.5 (7)	8.2 (5)	14.0 (7)	0.62
Under-reporters % (n)	31.7 (57)	25.4 (16)	34.9 (22)	35.2 (19)	0.41
Gestational age[(a)] (weeks)	12.6 (2.8)	12.5 (2.6)	12.6 (3.3)	12.6 (2.5)	0.96
Irish-born % (n)	74.4 (134)	69.8 (44)	74.6 (47)	79.6 (43)	0.48
Current smoker % (n)	11.1 (20)	11.1 (7)	11.1 (7)	11.1 (6)	1.00
Physical activity level[(a)] (MET)	1.75 (0.3)	1.70 (0.2)	1.70 (0.2)	1.80 (0.2)	0.06

(a) Mean (SD).
(b) Data available on n = 172.
FPG, fasting plasma glucose; MET, metabolic equivalents.

Table 2 Univariate comparison of maternal anthropometric characteristics in early pregnancy analysed by fasting plasma glucose (FPG) levels (n = 180)

	Lower FPG (<4.3 mmol/L) (n = 63)	*Moderate FPG (4.3–4.59 mmol/L) (n = 63)*	*Higher FPG (≥4.6 mmol/L) (n = 54)*	P
Weight (kg)	68.0 (15.0)	80.0 (22.0)	82.4 (23.3)	<0.001
BMI (kg/m^2)	24.0 (5.0)	28.0 (10)	30.0 (8.3)	<0.001
% Body fat (kg)	32.0 (9.0)	36.0 (11.0)	36.6 (9.0)	<0.001
Fat mass (kg)	21.0 (9.0)	29.0 (16.0)	30.5 (16.6)	<0.001
Fat free mass (kg)	46.0 (7.0)	51.0 (7.0)	53.0 (11.3)	<0.001
Visceral fat level	4.0 (2.0)	5.8 (3.2)	6.0 (4.0)	<0.001

All values reported are median (interquartile range).

of obese women on the basis of dietary under-reporting may therefore result in bias and erroneous conclusions regarding the nutritional intakes and GDM risk profile of obese women, and this is an important limitation of the current study. However, as the inclusion of under-reported food group and nutrient intakes from these women would have significantly distorted the inferential associations between population food and nutrient intake estimates and GDM risk in the current cohort, their exclusion was necessary to preserve the veracity of findings from the remaining dataset.[24]

It is established that the risk of developing GDM is increased in women with higher prepregnancy BMI.[18–20] Visceral fat and total body fat mass have been linked to insulin resistance among general adult populations.[21–23] However, there is a lack of studies investigating body fat mass in pregnancy and how it affects the risk of developing GDM. A cross-sectional study (n = 79) found that women with GDM had higher body fat mass levels compared with women with normal blood glucose levels.[42] Univariate analysis in our study suggested that increased adiposity in early pregnancy was associated with higher blood glucose levels. However, after controlling for important confounding factors, only antenatal obesity as measured by BMI remained associated with higher blood glucose levels.

Recent meta-analysis found no difference in the likelihood of developing GDM between women receiving diet and exercise interventions, and those allocated to control

Table 3 Comparison of energy-adjusted food group macronutrient intakes in plausible reporters analysed by FPG tertiles (n = 123)

Food group (g/MJ energy)	*Low FPG (<4.3 mmol/L) (n = 47)*	*Moderate FPG (4.3–4.59 mmol/L) (n = 41)*	*High FPG (≥4.6 mmol/L) (n = 35)*	P
Breads	4.7 (7.1)	4.5 (5.2)	4.1 (7.1)	0.16
Breakfast cereals	4.1 (8.2)	4.1 (5.5)	3.9 (4.9)	0.50
Rice/pasta	9.0 (8.8)	10.2 (9.8)	11.4 (9.9)	0.32
Eggs	1.9 (1.7)	1.9 (1.5)	2.2 (1.9)	0.58
Potatoes	10.1 (7.1)	10.6 (6.4)	9.7 (7.8)	0.93
Fats/oils	0.6 (1.0)	0.6 (0.7)	0.5 (0.5)	0.32
Alcoholic drinks	1.9 (9.4)	0.8 (6.2)	1.2 (4.3)	0.74
Sugar groups	12.2 (11.0)	15.5 (13.4)	12.3 (11.5)	0.31
Fruit and vegetables	62.2 (36.2)	54.8 (46.3)	51.1 (35.9)	0.94
Milk/cream/cheese	4.0 (5.5)	3.1 (3.6)	4.4 (4.7)	0.08
Fish	2.89 (4.6)	5.01 (6.93)	2.09 (3.97)	0.21
Meat	13.3 (6.6)	13.4 (6.4)	14.6 (9.3)	0.39
Other drinks	61.3 (64.4)	60.0 (59.5)	54.2 (67.1)	0.96
Other foods	11.6 (9.9)	12.8 (12.5)	10.5 (13.7)	0.90
Energy (MJ/day)	10.0 (5.8)	9.8 (4.7)	9.5 (3.3)	0.38
Carbohydrate (% TE)	45.2 (8.3)	48.6 (8.9)	47.1 (9.4)	0.45
Sugars (% TE)	18.9 (6.2)	21.2 (7.6)	19.0 (7.1)	0.45
Starch (% TE)	25.2 (10.2)	26.9 (9.2)	27.0 (7.8)	0.67
NMES (% TE)	5.6 (2.5)	6.5 (4.9)	6.7 (4.1)	0.62
Fructose (% TE)	3.8 (2.4)	3.7 (2.9)	3.6 (2.0)	0.94
Sucrose (% TE)	5.9 (3.4)	6.5 (2.8)	6.1 (3.4)	0.76
Lactose (% TE)	0.7 (0.7)	0.5 (0.5)	0.6 (0.5)	0.93
Maltose (% TE)	0.5 (0.7)	0.5 (0.6)	0.5 (0.6)	0.95
Oligosaccharides (% TE)	0.02 (0.1)	0.06 (0.1)	0.06 (0.2)	0.29
Fat (% TE)	36.4 (7.8)	34.7 (6.2)	35.6 (10.3)	0.91
Saturated fat (% TE)	13.4 (4.2)	13.1 (2.8)	13.3 (4.2)	0.34
Monounsaturated fat (% TE)	11.3 (2.3)	10.9 (2.7)	10.8 (3.1)	0.96
Polyunsaturated fat (% TE)	6.5 (2.8)	7.2 (3.1)	6.8 (2.4)	0.25
Dietary fibre (per MJ energy)	5.0 (1.8)	4.8 (2.9)	4.6 (2.4)	0.35
Protein (% TE)	18.0 (5.8)	18.2 (4.2)	18.4 (4.7)	0.92
Alcohol (g/day) (% TE)	0.4 (2.0)	0.3 (1.2)	0.4 (1.6)	0.82

All values reported are median (interquartile range).
FPG, fasting plasma glucose; NMES, non-milk extrinsic sugars; TE, total energy.

groups.[17,43] There was a trend towards a beneficial effect among women receiving primarily diet-based interventions, however, with a potentially significant reduction in GDM risk observed when these interventions were limited to obese and overweight women.[43]

Our study showed no association between energy adjusted food group or macronutrient intakes and FPG levels. However, while PAL levels were similar across all BMI categories, overall dietary EI and starch consumption were both higher among obese subjects. While causation cannot be confirmed, these findings suggest that excessive dietary EI, especially that derived from starchy carbohydrate, may contribute to the development of obesity, the main driver of GDM. This suggests that both excessive EI and high starchy food intake are important targets for dietary interventions in this area.

Previous studies investigating dietary intakes in early pregnancy and the risk of developing GDM have yielded inconsistent findings. In relation to macronutrients, some studies have shown that the type and quantity of carbohydrate may influence maternal blood glucose concentrations.[13] In non-obstetric populations, high fructose intake has been linked with adverse metabolic effects.[44] However, there is a lack of studies investigating fructose consumption and the development of GDM. While glycaemic index and energy-adjusted carbohydrate or fructose intakes were not associated with blood glucose levels in this study, high starch intakes were associated with obesity, the main

predictor of elevated maternal glucose. While further studies are needed to investigate the possible detrimental effects of excessive fructose intake on maternal blood glucose levels in pregnancy, research exploring the effect of high starchy carbohydrate intake is also warranted.

Our findings indicate that weight management in the prepregnancy period may have a more beneficial effect on FPG than altering diet in early pregnancy. Obesity was the main driver of higher FPG levels. Obese women had higher energy and starch intakes than non-obese women. Weight loss prior to pregnancy in obese women, particularly through a reduction in overall energy and starch intakes, may be more effective in improving maternal glycaemic control than attempts to adjust diet in early pregnancy.

Funding source

This project was supported by the UCD Centre for Human Reproduction and was partially funded by an unlimited educational grant from Danone Nutricia Early Life Nutrition for the first author. Danone Nutricia Early Life Nutrition had no role in the study design, data analysis, data interpretation or writing of this article.

Conflict of interest

The authors have no conflict of interests to declare.

Authorship

All authors contributed to this work. LM, AB, ACOH, DMcC and MJT formulated the research question and developed the experimental design. LM, ACOH, SC collected the data. LM and AB analysed the data. All authors contributed to drafting and revisions of the manuscript and approved the final version prior to submission.

References

1 American Diabetes Association. Clinical practice recommendations 2001: gestational diabetes mellitus. *Diab Care* 2001; **24**: S77–9.
2 Metzger BE, Coustan DR. Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus. *Diab Care* 1998; **21**: B161–7.
3 Ashwal E, Hod M. Gestational diabetes mellitus: where are we now? *Clin Chim Acta* 2015; **451**: 14–20.
4 Wendland EM, Torloni MR, Falavigna M *et al.* Gestational diabetes and pregnancy outcomes—a systematic review of the World Health Organization (WHO) and the International Association of Diabetes in Pregnancy Study Groups (IADPSG) diagnostic criteria. *BMC Pregnancy Childbirth* 2012; **12**: 23.
5 Buchanan TA, Xiang AH, Page KA. Gestational diabetes mellitus: risks and management during and after pregnancy. *Nat Rev Endocrinol* 2012; **8**: 639–49.
6 Kc K, Shakya S, Zhang H. Gestational diabetes mellitus and macrosomia: a literature review. *Ann Nutr Metab* 2015; **66**: 14–20.
7 Farrar D, Duley L, Medley N, Lawlor DA. Different strategies for diagnosing gestational diabetes to improve maternal and infant health. *Cochrane Database Syst Rev* 2015; **1**: CD007122.
8 Daly N, Stapleton M, O'Kelly R, Kinsley B, Daly S, Turner MJ. The role of preanalytical glycolysis in the diagnosis of gestational diabetes mellitus in obese women. *Am J Obstet Gynecol* 2015; **213**: 84e1–5.
9 HAPO Study Cooperative Research Group, Metzger BE, Lowe LP *et al.* Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med* 2008; **358**: 1991–2002.
10 Zhang C, Ning Y. Effect of dietary and lifestyle factors on the risk of gestational diabetes: review of epidemiologic evidence. *Am J Clin Nutr* 2011; **94**: 1975S–9.
11 Tobias DK, Zhang C, van Dam RM, Bowers K, Hu FB. Physical activity before and during pregnancy and risk of gestational diabetes mellitus: a meta-analysis. *Diab Care* 2011; **34**: 223–9.
12 Russo L, Nobles C, Ertel KA, Chasa-Taber L, Whitcomb BW. Physical activity interventions in pregnancy and risk of gestational diabetes mellitus: a systematic review and meta-analysis. *Obstet Gynecol* 2015; **125**: 576–82.
13 Walsh JM, McGowan CA, Mahony R, Foley ME, McAuliffe FM. Low glycaemic index diet in pregnancy to prevent macrosomia (ROLO study): randomised control trial. *BMJ* 2012; **345**: e5605.
14 Zhang C, Liu S, Solomon CG, Hu FB. Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus. *Diab Care* 2006; **29**: 2223–30.
15 Bao W, Bowers K, Tobias DK, Hu FB, Zhang C. Prepregnancy dietary protein intake, major dietary protein sources, and the risk of gestational diabetes mellitus: a prospective cohort study. *Diab Care* 2013; **36**: 2001–8.
16 Bowers K, Tobias DK, Yeung E, Hu FB, Zhang C. A prospective study of prepregnancy dietary fat intake and risk of gestational diabetes. *Am J Clin Nutr* 2012; **95**: 446–53.
17 Bain E, Crane M, Tieu J, Han S, Crowther CA, Middleton P. Diet and exercise interventions for preventing gestational diabetes mellitus. *Cochrane Database Syst Rev* 2015; **4**: CD010443.
18 Torloni MR, Betrán AP, Horta BL *et al.* Prepregnancy BMI and the risk of gestational diabetes: a systematic review of the literature with meta-analysis. *Obes Rev* 2009; **10**: 194–203.
19 Heude B, Thiébaugeorges O, Goua V *et al.* Pre-pregnancy body mass index and weight gain during pregnancy: relations with gestational diabetes and hypertension, and birth outcomes. *Matern Child Health J* 2012; **16**: 355–63.
20 Morisset AS, St-Yves A, Veillette J, Weisnagel SJ, Tchernof A, Robitaille J. Prevention of gestational diabetes mellitus: a review of studies on weight management. *Diabetes Metab Res Rev* 2010; **26**: 17–25.
21 Gastaldelli A, Miyazaki Y, Pettiti M *et al.* Metabolic effects of visceral fat accumulation in type 2 diabetes. *J Clin Endocrinol Metab* 2002; **87**: 5098–103.
22 Gómez-Ambrosi J, Silva C, Galofré JC *et al.* Body adiposity and type 2 diabetes: increased risk with a high body fat percentage even having a normal BMI. *Obesity* 2011; **19**: 1439–44.
23 MacKay MF, Haffner SM, Wagenknecht LE, D'Agostino RB Jr, Hanley AJ. Prediction of type 2 diabetes using alternate anthropometric measures in a multi-ethnic cohort: the insulin resistance atherosclerosis study. *Diab Care* 2009; **32**: 956–8.
24 Mullaney L, O'Higgins AC, Cawley S, Doolan A, McCartney D, Turner MJ. An estimation of periconceptional under-reporting of dietary energy intake. *J Public Health (Oxf)* 2015; **37**: 728–36.

25 Mullaney L, O'Higgins AC, Cawley S, Kennedy R, McCartney D, Turner MJ. Breast-feeding and postpartum maternal weight trajectories. *Public Health Nutr* 2015; **15**: 1–8.
26 Harrington J. Validation of a food frequency questionnaire as a tool for assessing nutrient intake (MA Thesis). Galway: Department of Health Promotion, National University of Ireland, 1997.
27 Kaaks R, Slimani N, Riboli E. Pilot phase studies on the accuracy of dietary intake measurements in the EPIC project: overall evaluation of results. European Prospective Investigation into Cancer and Nutrition. *Int J Epidemiol* 1997; **26**: S26–36.
28 McGowan CA, Curran S, McAuliffe FM. Relative validity of a food frequency questionnaire to assess nutrient intake in pregnant women. *J Hum Nutr Diet* 2014; **27**: 167–74.
29 Fattah C, Farah N, O'Connor N, Stuart B, Turner MJ. The measurement of maternal adiposity. *J Obstet Gynaecol* 2009; **29**: 686–9.
30 Fattah C, Farah N, Barry S, O'Connor N, Stuart B, Turner MJ. Maternal weight and body composition in the first trimester of pregnancy. *Acta Obstet Gynecol Scand* 2010; **89**: 952–5.
31 National Institute for Clinical Health and Excellence. *Diabetes in pregnancy: management of diabetes and its complications from pre-conception to the postnatal period 2008*; 2008. (Available from: http://www.nice.org.uk/nicemedia/pdf/CG063Guidance.pdf, accessed 2 March 2015).
32 Morgan K, McGee H, Watson D *et al*. SLÁN 2007: survey of lifestyle, attitudes and nutrition in Ireland. Main report. Dublin: Department of Health and Children, The Stationery Office; 2008.
33 Food Standards Agency. *Food portion sizes*, 3rd edn. London: TSO, 2006.
34 McCance RA, Widdowson EM. *The Composition of Foods*, 6th edn. Great Britain: Food Standards Agency and Royal Society of Chemistry, 2002.
35 Central Statistics Office. *EU Survey on Income and Living Conditions (EU-SILC) 2011 and Revised 2010 Results*. Dublin: Central Statistics Office, 2013.
36 European Commission Working Group. Statistics on income poverty and social exclusion. *Laeken Indicators Detailed Calculation Methodology*; 2003. (Available from: http://www.cso.ie/en/media/csoie/eusilc/documents/Laeken,Indicators,-,calculation,algorithm.pdf, accessed 11 June 2015).
37 Food and Agricultural Organisation/World Health Organisation/United Nations University. Human energy requirements. Report of a Joint FAO/WHO/UNU Expert Consultation. Rome: Food and Agricultural Organisation; 2001
38 Livingstone MB, Black AE. Markers of the validity of reported energy intake. *J Nutr* 2003; **133**: 895S–920.
39 Metzger BE, Gabbe SG, Persson B *et al*. International association of diabetes and pregnancy study groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. *Diab Care* 2010; **33**: 676–82.
40 Fattah C, Farah N, O'Toole F, Barry S, Stuart B, Turner MJ. Body mass index (BMI) in women booking for antenatal care: comparison between selfreported and digital measurements. *Eur J Obstet Gynecol Reprod Biol* 2009; **144**: 32–4.
41 Turner MJ. The measurement of maternal obesity: can we do better? *Clin Obes* 2011; **1**: 127–9.
42 Moreno Martinez S, Tufiño Olivares E, Chávez Loya V, Rodríguez Morán M, Guerrero Romero F, Levario CM. Body composition in women with gestational diabetes mellitus. *Ginecol Obstet Mex* 2009; **77**: 270–6.
43 Rogozińska E, Chamillard M, Hitman GA, Khan KS, Thangaratinam S. Nutritional manipulation for the primary prevention of gestational diabetes mellitus: a meta-analysis of randomised studies. *PLoS One* 2015; **10**: e0115526.
44 Stanhope KL, Schwarz JM, Havel PJ. Adverse metabolic effects of dietary fructose: results from the recent epidemiological, clinical, and mechanistic studies. *Curr Opin Lipidol* 2013; **24**: 198–206.

