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23 Running title: Diet of pregnant women with gestational diabetes

Abstract

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- 2 Objectives: To determine if women with gestational diabetes (GD) modify their diet and
- 3 nutrient intake in late pregnancy and gain more weight during pregnancy compared to women
- 4 without GD.
- 5 **Subjects and methods:** Food and nutrient intake of 3 613 pregnant women was studied using
- 6 food frequency questionnaires from the Type 1 Diabetes Prediction and Prevention Nutrition
- 7 Study.
- 8 **Results:** GD was reported in 4.8% of the participating women (n=174). Women with GD
- 9 gained less weight during pregnancy than those unaffected by GD (mean 9.4 kg vs. 12.6 kg,
- p<0.001). Women with GD consumed more milk products (84 vs. 76 g/MJ, p=0.002), cereal
- 11 products (21 vs. 18 g/MJ, p<0.001), vegetables (32 vs. 22 g/MJ, p<0.001) and meat (16 vs. 14
- 12 g/MJ, p<0.001) than unaffected women. The intake of protein (18 vs. 16 E%, p<0.001) and
- dietary fibre (3.1 vs. 2.4 g/MJ, p<0.001) was higher, while the intake of sugars (13.3 g/MJ vs.
- 14 15.0 g/MJ, p<0.001) and saturated fatty acids (3.49 g/MJ vas. 3.98 g/MJ, p<0.001) was lower,
- among women with GD. The nutrient density of the diet was higher in women with GD with
- higher intakes of vitamin A and D, folate and iron.
- 17 **Conclusions:** The late pregnancy diet of women with GD differed considerably from that of
- unaffected women. Women with GD had a higher body weight at the beginning of the
- 19 pregnancy, but they gained less weight during pregnancy. These findings indicate that
- 20 abnormal glucose tolerance during pregnancy encourages women to modify their dietary
- 21 habits towards healthier food choices.

23 Keywords

Keywords: gestational diabetes, diet, weight gain

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Introduction

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2 Gestational diabetes (GD) represents the onset or first recognition of glucose intolerance 3 during pregnancy (Metzger and Coustan, 1998). It has been associated with adverse foetal outcomes such as macrosomia and birth trauma (Blank et al., 1995; Crowther et al., 2005). 4 5 Women with GD may face other pregnancy complications such as pre-eclampsia and have 6 also increased risk of developing glucose intolerance in subsequent pregnancies (Dornhorst 7 and Rossi, 1998; Metzger and Coustan, 1998). In addition, those with a history of GD are also 8 at increased risk of developing type 2 diabetes. Although glucose tolerance is impaired in all 9 women during pregnancy, only a small percentage go on to develop GD. In 2006, 8.4% of 10 pregnant Finnish women were found to have an abnormal result in an oral glucose tolerance test (National Research and Development Centre for Welfare and Health, 2007). Worldwide, 12 the estimates of GD prevalence vary widely, depending on the diagnostic criteria used.

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Age, parity, smoking, increased body weight and body mass index (BMI), along with previous GD are factors known to increase the risk of GD in pregnant women (Langer et al., 2005; Rudra et al., 2007; Solomon et al., 1997; Wang et al., 2000). Generally GD is more common among overweight and obese women than among lean women (American College of Obstetricians and Gynecologists, 2005; Chu et al., 2007; Solomon et al., 1997). It has been indicated that women with GD gain less weight during pregnancy than those without GD (Catalano et al., 1993; Couch et al., 1998; Teramo et al., 2007). This is a likely reflection of the effectiveness of lifestyle interventions available for women with GD at maternity welfare clinics when the condition is first detected.

The cornerstone of the management of GD is nutrition therapy (Gunderson, 2004; Metzger and Coustan 1998). The goal of dietary intervention is to attain normoglycemia and to maintain a moderate weight gain (around 6-8 kg) through pregnancy (Gunderson, 2004; Hasunen et al., 2004). However, those who fail to respond to the glycemic targets through diet receive insulin in conjunction with nutrition therapy. At Finnish maternity welfare clinics, women with GD are advised to follow a diet that favours vegetables, fruits, berries and whole grain cereal over high energy content food (Hasunen et al., 2004). They are also recommended to increase their intake of fibre, and to moderate their consumption of fats, while ensuring an adequate intake of unsaturated fats. The intake of protein is recommended to not exceed 20-25%, total fat 30-40% and carbohydrates 40-50% of the total daily energy intake (Virtanen et al., 2008). Furthermore, women with GD are advised to increase their level of physical activity (American Diabetes Association, 2004; Hasunen et al., 2004).

Pregnancy is an ideal time for behavioral and lifestyle modifications pertaining to diet and physical exercise (Artal et al., 2007). Although pregnant women with GD are generally advised to improve the quality of their diet, it is not well known whether women adhere to the given instructions. In the present study, we firstly wanted to determine if women with GD modify their diet and nutrient intake in late pregnancy compared to women without GD. Secondly, we wanted to determine if women with GD gain more weight during pregnancy compared to women without GD.

Materials and methods

Subjects of the present study are participating in the ongoing Type 1 Diabetes Prediction and Prevention (DIPP) study, a large population-based birth cohort study in Finland which started in 1994 (Kupila et al., 2001). The study was established to predict the development of type 1 diabetes and to search for means to prevent or delay progression of increased genetic susceptibility to clinical type 1 diabetes. Infants who carry increased genetic risk represent about 15% of the total population. Infants for the DIPP study were screened for HLA-DQB1-conferred susceptibility to type 1 diabetes using samples of cord blood, and those with a high risk were invited to participate in the DIPP study in the area of their local university hospital (Oulu, Tampere and Turku University Hospitals). The study is approved by the local Ethical Committees. All participating families have given their informed, written consent.

The DIPP Nutrition Study falls within the framework of the larger DIPP Study. The DIPP Nutrition Study aims at investigating maternal nutrition during pregnancy and lactation, and child's nutrition, in relation to the natural progression from increased genetic susceptibility to the development of β -cell autoimmunity and clinical manifestation of type 1 diabetes, allergic diseases and asthma. The subjects of the present study are women whose child was born between October 20, 1997 and December 31, 2002 in Oulu and Tampere University Hospitals (n=5 339) (Figure 1). Baseline dietary data was available from 3 783 women, of which 51 were rejected because of incomplete dietary data (for example more than 10 missing food items in the food frequency questionnaire, FFQ). A total of 3 732 women with dietary data were available for this study (70% of the women eligible to the DIPP Study). The research questions were studied between women with and without GD. We excluded those with type 1 or type 2 diabetes from the comparison group. The total study population was thereafter 3 613. For our analyses on maternal weight and weight gain, we needed to exclude further subjects based on the following criteria: women with twin and triple pregnancies, those with missing information on the number of foetuses and women with incomplete weight gain information. After this exclusion, data on 3 260 were available for weight gain analysis.

1 The food frequency questionnaire

2 Maternal diet during pregnancy was assessed retrospectively by a self-administered, semi-3 quantitative FFQ, which has been validated specifically for the present study population 4 (Erkkola et al., 2001). This 181-item questionnaire assesses the mother's usual dietary intake over the last month before maternity leave, usually the 8th month of the pregnancy in Finland. 5 6 The mothers filled in the questionnaire 1-3 months after the delivery and returned it at their 7 first visit to the study center where a trained study nurse checked the FFQ and if required, 8 completed it in consultation with the mother. All returned FFOs were also double-checked by 9 a trained nutritionist. The FFQ contained questions about the frequency (number of times per 10 day, week or month) and the amount of foods consumed, in units of common serving sizes. 11 The food consumption data was entered into a dietary database and the Finnish Food Composition Database Fineli® (National Institute for Health and Welfare Nutrition Unit, 12 13 2009) was used to calculate daily food and nutrient intakes. Besides food consumption data, 14 the FFQ also enquired about the use of dietary supplements (i.e. brand name and 15 manufacturer, dosage and gestational weeks when used). Supplement intake was divided by 16 the length of the pregnancy (days) and represents the whole time period of the pregnancy.

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18 Information on GD, pregnancy and background characteristics

In Finland, free-of-charge health care is offered to all pregnant women in maternity welfare clinics and in practice all pregnant women attend the service. During the present study an oral glucose tolerance test was performed usually at 26-28 week of gestation for those women who fulfilled one or more of the following criteria: glucosuria after fasting, overweight (BMI>25 kg/m²), age >40 years, earlier delivery of a baby with birth weight >4 500 g or macrosomic foetus (Berger et al 2002). GD is diagnosed when at least one of the values is pathological in a 75 g (2 hrs) oral glucose tolerance test (American Diabetes Association, 2004). The FFQ

1 required the women to indicate if they followed any special diets during pregnancy by

selecting one of the following alternatives: no special diets, food allergy or hypersensitivity,

3 lactose intolerance, type 1 diabetes, type 2 diabetes, gestational diabetes, coeliac disease,

vegetarian diet or other special diets. This reported information of the GD is the main

5 outcome of the study.

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The maternal weight and height information at the beginning of the pregnancy, as well as the weight gain during pregnancy, was obtained from the follow-up card kept at the maternity

welfare clinic. In Finnish healthcare system, the first antenatal visit is usually between $\mathbf{8}^{th}$ and

12th gestational week. The rate of weight gain (kg/week) was calculated as the difference in

body weight between the last and the first antenatal visit divided by the number of gestational

weeks at the time of the weight measurement. BMI was calculated based on the weight and

height information. The women's age and education details were registered by a structured

questionnaire completed after the delivery. Professional education was merged into three

groups from the seven education groups available in the questionnaire. Information about

parity, place of residence and smoking habits during pregnancy was derived from the Medical

Birth Registries of the Oulu and Tampere University Hospitals. The same registries contained

information on the municipality code, from which the degree of urbanization (urban,

population centre, rural) was derived.

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Statistical analysis

22 Stepwise logistic regression using forward elimination was used to analyse the association

between background factors and GD. The energy densities (g/MJ) of foods and nutrients were

calculated to eliminate the effects of differences in total energy intake. Foods and nutrients

that were not normally distributed were transformed into logarithmic scale for statistical

analyses. The associations between GD and food and nutrient intake were studied using linear regression analyses and were adjusted for maternal age, place of residence, degree of urbanization, vocational education, BMI and parity. T-test was used to investigate the difference in weight gain through pregnancy and the child's birth weight between women with and without GD. The proportions of vitamin and mineral supplement users between the two groups were tested using χ^2 -test. In addition, the differences of the mean supplement intakes between women with and without GD were tested using non-parametric Mann-Whitney test. A p-value of <0.05 was considered statistically significant. SPSS 15.0 for Windows (SPSS Inc., Chicago, IL, USA) was used to analyse the data.

Results

In the present study GD was reported in 174 (4.8%) of the participating women (total n=3 613). GD was more common among women in the two oldest age categories (30-34 years and ≥35 years). Furthermore, women with GD had higher BMI at the first visit (mean 28.1 kg/m²) than women without GD (mean 24.1 kg/m²). The risk of GD was higher among women living in northern Finland (Oulu University Hospital district) and those who had an upper secondary vocational education. (Table 1)

Women affected by GD gained less weight (p<0.001) (mean 9.4 kg, SD 5.1) than the unaffected women (mean 12.6 kg, SD 4.5). The mean rate of weight gain was 0.34 kg/wk in women with GD and 0.43 kg/wk in women without GD (p<0.001) (Figure 2). Infant birth weights differed between the two groups of women (mean 3 650 g in women with GD vs. 3 560 g in unaffected women; p=0.036).

1 The diet of the women with GD differed from the diet of the women without GD. The former

consumed more milk and cereal products than the latter. Those with GD consumed less fruits

and berries but more vegetables. Meat was consumed more frequently by the women with GD

than by the unaffected women. Women with GD consumed less chocolate and sweets than

5 those unaffected. (Table 2)

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7 The dietary intake of most nutrients differed statistically between the women with GD and

those without. However, for many nutrients the difference was too small to be of practical

importance. If the difference was <10%, we concluded that the difference is not of clinical

importance and did not analyse the results further. The intake of sugars was lower while the

intake of starch and dietary fibre was higher among women with GD. Their intake of protein

was higher than that of the unaffected women. Intake of saturated fatty acids was lower in

women with GD. Their intake of vitamins A and D, folate and iron was higher than that of the

unaffected women. The intake of iron was also higher in subjects with GD. (Table 3)

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Most of the women (77.2%) complemented their diet with dietary supplements during

pregnancy. No difference was found in nutrition intake from supplements between the two

groups of women. Subjects with GD took iron supplements less frequently than other

19 subjects. (Table 4)

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Discussion

22 The present study was undertaken to explore differences in food and nutrient intakes between

pregnant women with GD and those unaffected. Our study revealed that the diet in late

pregnancy among women with GD differed considerably from the diet of those without GD.

Women with GD consumed more milk and cereal products, vegetables and meat than those

unaffected, and consequently their intake of protein, starch and dietary fibre, and that of vitamin A and D, folate and iron, was higher. In addition, women with GD had a lower intake of sugars and saturated fatty acids. Women affected by GD also gained less weight than the unaffected women. Our findings indicate that when abnormal glucose tolerance is first detected, women tend to modify their dietary habits towards healthier food choices. Since the first line of treatment of GD is through nutrition therapy in maternity welfare clinics, our findings indicate that the dietary advice women receive is in fact effective.

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The main strength of the current study was the large sample size of a population-based cohort. In addition, since virtually all deliveries in Finland take place in public hospitals, our study population is likely to be a close representation of the overall population of pregnant women in the areas of the university hospitals of the study. A further strength is our use of an FFQ which was specifically validated for the purpose of this study population (Erkkola et al., 2001). However, the study involves some limitations, of which most are related to the retrospective study design and its potential for recall bias. Firstly, since the participants needed to recall their food use retrospectively, the influence of their current diet is a possible source of error (Bunin et al., 2001). On the other hand, it could also be argued that women may recall their dietary habits during pregnancy particularly well because of this unique period in their lives. Secondly, as the women without GD mostly had a relatively uncomplicated pregnancy, while those with GD received dietary guidance, it is possible that the latter may have over-reported their consumption of healthy foods. Thirdly, since we used an FFQ to study the maternal intake during pregnancy, there is a possibility that the food consumption and nutrient intake data are overestimates, especially that of vegetables, and energy and energy-yielding nutrients (Erkkola et al., 2001; Nelson and Bingham, 1997). Then again, some studies have reported that the energy intake among certain groups of women,

such as those who are overweight, weight-conscious or pregnant, is often too low to meet the physiological energy requirements (Hill and Davies, 2001; Trabulsi and Schoeller, 2001). Hence, the reduced energy intake among the women with GD in our study may in fact to certain extent be down to the tendency of under-reporting by obese women (Hill and Davies, 2001). We therefore took steps to minimize the effects of over and underreporting in the present study, and thus estimated the energy densities of food and nutrient intakes in the analyses. Finally, as the GD status in our study was reported by the responding mothers, we

could not confirm the status independently.

Our data showed that women with GD had a lower intake of sugars and saturated fatty acids whilst having a higher intake of protein, starch and dietary fibre, as compared with the unaffected women. A recent study among British women with GD, who also received dietary advice, observed similar differences in the intakes of saturated fats, added sugar, complex carbohydrates and dietary fibre (Thomas et al., 2006). In our study, the women with GD had a total fat intake of 32% of the total energy intake, while the unaffected women had a slightly higher intake of fat at 34%. The mean intake of protein among women with GD was 18% of the total daily energy intake and hence fell below the recommended level of 20-25% (Virtanen et al., 2008). In women without GD the intake of protein was even lower at 16% of the total energy intake. However, the proportion of carbohydrates of the total energy intake was similar in both groups at 49% of the total daily energy intake.

In line with many previous studies, we observed that the women with GD were generally older and had a higher BMI at the beginning of the pregnancy than women without GD (Hackmon et al., 2007; Radesky et al., 2008; Rudra et al., 2007; Solomon et al., 1997; Wang et al., 2000). In our study, those with GD also had a higher body weight at the beginning of

the pregnancy than those not affected, but they gained less weight during the course of the pregnancy. Similar observations have been made previously in a case-control study, which evaluated weight gain during pregnancy in women with GD and those without (Catalano et al., 1993). However, although the study indicated that the weight gain in women with GD was less than in controls, the effect was considered to be attributed to their greater pregravid weight. In contrast to previous studies, parity and cigarette smoking were not associated with GD in the present study (England et al., 2004; Solomon et al., 1997).

Overall the use of dietary supplements was quite common in our study population; 77% of all women took at least one dietary supplement. Our results are similar to an earlier study among pregnant Finnish women in which 70% of women were reported to take dietary supplements (Erkkola et al., 1998). However, in the present study population women with GD took iron supplementation less frequently than the unaffected women. The high consumption of meat and cereal products, both good sources of iron, among women with GD is a likely explanation to this finding.

In conclusion, our findings indicate that women diagnosed with GD are capable of modifying their dietary habits when given nutrition guidance at the maternity welfare clinics. This observation is of great importance from the perspective of type 2 diabetes prevention. Thus by means of dietary advise, regular exercise, stabilizing weight and monitoring blood glucose levels, it is possible to delay or prevent the progression of GD to type 2 diabetes and its further complications in women with a history of GD. Since the maternal nutritional status before pregnancy, as reflected by pre-pregnancy BMI, is known to be more important than the diet during pregnancy in the development of GD, efforts to reduce rates of GD should continue to focus on reducing the prevalence of obesity among women in fertile age.

- 1 Furthermore, any efforts to promote physical activity and healthy eating habits among women
- 2 at this age should be strengthened.

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Conflict of interest

15 There are no conflicts of interest.

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Table 1 Characteristics of women with and without gestational diabetes (GD) in the DIPP nutrition study, with child born in 20.10.1997-31.12.2002 in Tampere and Oulu University Hospital districts.

	Women with GD (n=174)		Women w		
-		1 /4) %	(n=3 439) n %		OR ^a (95% CI)
Age (years) ^b	n	/0	11	/0	OK (7570 CI)
<25	18	10.3	678	19.7	1
25-29	48	27.6	1 185	34.5	1.53 (0.83-2.82)
30-34	56	32.2	1 017	29.6	2.02 (1.11-3.68)
≥35	52	29.9	559	16.3	3.20 (1.73-5.93)
Place of residence	32	27.7	337	10.5	3.20 (1.73 3.73)
District of Tampere	68	39.1	2 062	60.0	1
District of Oulu	106	60.9	1 377	40.0	2.33 (1.66-3.26)
Degree of urbanization	100	00.7	1377	10.0	2.33 (1.00 3.20)
Urban	117	67.2	2 642	76.8	NS
Population centre	20	11.5	332	9.7	110
Rural	35	20.1	438	12.7	
Missing data	2	1.1	27	0.8	
BMI at the first antenatal					
visit (kg/m ²)					
1. quarter (<21.4)	12	6.9	847	24.6	1
2. quarter (21.4-23.4)	22	12.6	839	24.4	1.60 (0.76-3.41)
3. quarter (23.4-26.1)	35	20.1	826	24.0	2.77 (1.38-5.54)
4. quarter (>26.1)	100	57.5	760	22.1	8.58 (4.51-16.31)
Missing data	5	2.9	167	4.9	,
Smoking during					
pregnancy					
No	153	87.9	2 962	86.1	NS
Yes	15	8.6	359	10.4	
Missing data	6	3.4	118	3.4	
Professional education					
Academic	28	16.1	748	21.8	1
Upper secondary					
vocational education	92	52.9	1 377	40.0	1.65 (1.03-2.65)
None or vocational					
school or course	49	28.2	1 221	35.5	1.01 (0.60-1.70)
Missing data	5	2.9	93	2.7	
Parity					
0	55	31.6	1 581	46.0	NS
1	56	32.2	1 081	31.4	
2	25	14.4	476	13.8	
≥3	36	20.7	277	8.1	
Missing data	2	1.1	24	0.7	· 1 NG 4

Abbreviations: BMI, body mass index; OR, odds ratio; CI, confidence intervals; NS, not significant

^aLogistic regression stepwise analysis for developing GD. All estimates are simultaneously adjusted for all other characteristics in the table.

^bAt the time of the delivery.

Table 2 Consumption of foods (g/MJ) in women with gestational diabetes (GD) and unaffected women.

		with GD 174)		Women without GD (n=3 439)		
_	mean	SD	mean	SD	p-value ^a	
Milk products (g/MJ)	84.3	33.5	75.5	31.6	0.002	
Cereal products (g/MJ)	20.7	5.2	17.9	4.4	< 0.001	
Fruits and berries (g/MJ)	44.3	24.5	52.8	28.3	0.004	
Vegetables (g/MJ)	32.3	18.8	21.5	11.6	< 0.001	
Meat (g/MJ)	16.3	5.1	14.2	5.0	< 0.001	
Fish (g/MJ)	2.6	2.0	2.2	1.7	0.062	
Dietary fats (g/MJ)	4.0	1.4	4.0	1.4	0.344	
Chocolate and sweets (g/MJ)	1.1	1.4	2.6	2.2	< 0.001	

Abbreviations: SD, standard deviation

^aLinear regression analyses adjusted for maternal age, place of residence, degree of urbanization, vocational education, BMI and parity.

Table 3 Intake of energy and nutrients from foods adjusted for energy (per MJ) in women with gestational diabetes (GD) and unaffected women.

	Women	with GD	Women without		
	(n=174)		GD (n=3 439)		_
	mean	SD	mean	SD	p-value ^a
Energy (MJ)	10.8	2.9	11.5	3.4	
(kcal)	2579	699	2759	814	0.009
Protein (E%)	18		16		< 0.001
Carbohydrates (E%)	49		49		0.712
Starch (g/MJ)	15.0	3.0	13.2	2.5	< 0.001
Sugars (g/MJ)	13.3	3.3	15.0	3.4	< 0.001
Dietary fibre (g/MJ)	3.08	0.91	2.40	0.66	< 0.001
Total fat (E%)	32		34		< 0.001
Saturated fatty acids (g/MJ)	3.49	0.77	3.98	0.76	< 0.001
Trans fatty acids (g/MJ)	0.13	0.04	0.14	0.04	0.003
Monounsaturated fatty acids (g/MJ)	3.06	0.53	3.23	0.54	0.001
Polyunsaturated fatty acids (g/MJ)	1.31	0.32	1.26	0.29	0.016
N-3 fatty acids (g/MJ)	0.29	0.08	0.28	0.08	0.408
N-6 fatty acids (g/MJ)	1.00	0.27	0.95	0.23	0.007
Vitamin A (μg/MJ)	189	110	141	72	< 0.001
Vitamin D (μg/MJ)	0.50	0.21	0.43	0.19	< 0.001
Vitamin E (mg/MJ)	1.38	0.31	1.25	0.25	< 0.001
Folate (µg/MJ)	37.4	8.37	32.1	6.40	< 0.001
Vitamin C (mg/MJ)	17.5	8.3	17.3	8.7	0.134
Calcium (mg/MJ)	174	41.7	161	41.9	< 0.001
Iron (mg/MJ)	1.67	0.32	1.44	0.27	< 0.001

Abbreviations: SD, standard deviation

^aLinear regression analyses adjusted for maternal age, place of residence, degree of urbanization, vocational education, BMI and parity.

Table 4 Proportion of vitamin and mineral supplement users and nutrient intake from supplements in women with gestational diabetes (GD) and unaffected women.

	Proportion of users				_	Intake from supplements				
	Women		_			Women		-		
	Women with without GD			Women with		without GD				
	GD (n=174)		(n=3 439)		_	GD (n=174)		(n=3439)		_
	n	%	n	%	p value ^a	mean	SD	mean	SD	p-value ^b
Vitamin A (μg)	12	6.9	210	6.1	0.673	39.6	204.3	16.9	103.0	0.605
Vitamin D (μg)	46	26.4	1 043	30.3	0.273	1.12	2.57	1.30	2.59	0.248
Vitamin E (mg)	49	28.2	1 199	34.9	0.069	0.84	1.97	1.13	5.04	0.093
Folic acid (µg)	51	29.3	1 254	36.5	0.119	53.0	95.3	57.1	98.4	0.223
Vitamin C (mg)	45	25.9	1 075	31.3	0.132	20.6	66.4	23.2	83.3	0.172
Calcium (mg)	33	19.0	594	17.3	0.343	81.0	178.5	68.2	177.2	0.311
Iron (mg)	105	60.3	2 388	69.4	0.011	25.2	35.1	27.0	33.3	0.067
Any supplement	128	73.6	2 663	77.4	0.235					

Abbreviations: SD, standard deviation

 $^{^{}a}\chi^{2}$ -test

^bMann-Whitney Test

Figure legends

Figure 1 Study population of pregnant women in the DIPP Nutrition Study.

Figure 2 Rate of weight gain during pregnancy (kg/week) in women with gestational diabetes (GD) (n=160) and without gestational diabetes (n=3100).

Women invited to the DIPP Nutrition Study after giving birth to a child at increased genetic risk for type 1 diabetes at the University Hospital of Oulu or Tampere (Oct 20, 1997- Dec 31 2002)





