Is improvement in the Healthy Food Intake Index (HFII) related to lower risk of gestational diabetes?

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Running head: Dietary change and gestational diabetes
Abstract

The aim was to analyze whether changes in the Healthy Food Intake Index (HFII) during pregnancy are related to gestational diabetes (GDM) risk. The participating 251 pregnant women had a pre-pregnancy body mass index (BMI) ≥30 kg/m² and/or a history of GDM. A 75g oral glucose tolerance test was performed during the 1st and 2nd trimesters of pregnancy for assessment of GDM. A normal OGTT result at 1st trimester was an inclusion criteria for the study. Food frequency questionnaires collected at 1st and 2nd trimesters served for calculating the HFII. A higher HFII score reflects higher adherence to the Nordic Nutrition Recommendations (NNR) (score range 0-17). Statistical methods included Student’s t-test, Mann Whitney U-test, Fishers’ exact test, and logistic regression analysis. The mean HFII at first trimester was 10·1 (95% CI 9·7, 10·4) points, and the mean change from 1st to 2nd trimester 0·35 (95% CI: 0·09, 0·62) points. The odds for GDM decreased with higher HFII change (adjusted OR 0·83 per one unit increase in HFII, [95% CI 0·69, 0·99], p=0·043). In analysis of the association between HFII-subindices and GDM, odds for GDM decreased with higher HFII-Fat change (fat-% of milk and cheese, type of spread and cooking fats) but it was not significant in a fully adjusted model (p=0·058). Dietary changes towards the NNR during pregnancy seem to be related to lower risk of GDM.

Introduction

The prevalence of gestational diabetes (GDM) increases globally and is currently affecting 4-15% of pregnant women in Western countries (O’Sullivan et al. 2011, Lamberg et al. 2012). Modifiable risk factors may include diet and physical activity, which have also been the primary targets of most intervention studies (Bain et al. 2015). Even though most lifestyle intervention studies have failed in preventing GDM, we recently showed that a lifestyle intervention with diet and physical activity counselling can prevent GDM in high risk women (Koivusalo et al. 2016). At about the same time Jing et al (Jing X et al. 2010) also succeeded in preventing GDM with diet and physical activity counselling. According to epidemiological cohort studies dietary factors seem to be strongly associated with GDM risk (Tobias et al. 2012, Schoenaker et al. 2015). Observational studies have reported that a Western dietary pattern associates with an elevated risk for GDM, and dietary patterns characterized by high consumption of vegetables, fruits, dietary fiber and low consumption of high fat/high sugar foods and red and processed meat associate with lower risk of GDM (Zhang et al. 2006, Tobias et al. 2012, Karamanos et al. 2014, He et al. 2015). However, a prospective study focusing upon the relationship between an actual dietary change during pregnancy and GDM risk is still lacking.
Measuring changes in overall diet instead of traditional individual-nutrient/food analysis, is justified because firstly, several dietary factors seem to be associated with GDM risk (Schoenaker et al. 2016). Secondly, change in one dietary factor is usually followed by a compensatory change in another factor, and these changes cannot and should not be separated (Sacks et al. 1995). Thirdly, the subtle changes in intake of individual nutrients or foods may become detectable only when several changes are combined (Hu 2002). Diet quality indices can be suitable for measuring changes in overall diet (Clutter Snyder et al. 2007). Several dietary patterns measured by indices, such as Mediterranean dietary pattern (Med), alternate Mediterranean dietary pattern (aMed), Diet Approach to Stop Hypertension (DASH), and alternate Healthy eating index (aHEI), associate with GDM (Tobias et al. 2012, Karamanos et al. 2014). However, studying the association between a change in diet quality index score and GDM risk is a novel approach.

Culturally and locally acceptable dietary patterns for GDM prevention are essential. Specific Nordic cultural characteristics of diets are taken into account in food-based guidelines of the Nordic Nutrition Recommendations (NNR) (Nordic Council of Ministers 2014). A diet adherent to the NNR has similar characteristics with the aforementioned healthy diets associated with GDM. The similarities with the other dietary indices are high intake of fruits, berries, and vegetables, whole grain foods, nuts and legumes, and low intake of sodium, red and processed meats and sugar-sweetened beverages. NNR also recommends vegetable oils, such as rapeseed oil and vegetable margarines instead of animal fats, as well as low-fat instead of high-fat dairy products (Nordic Council of Ministers 2014). A recently developed and validated diet quality index, the Healthy Food Intake Index (HFII), measures adherence of a diet to the NNR (Meinila et al. 2016). The aim of the current study was to analyze whether changes in the HFII, and concurrently changes in the adherence to the NNR, from 1st to 2nd trimester of pregnancy is related to GDM among women at high risk of GDM. A further aim was to study whether any sub-index within the HFII drives/influences the possible relationship between the HFII and GDM.

Participants and methods

The study participants were a part of the Finnish Gestational Diabetes Prevention Study (RADIEL), a multicenter lifestyle intervention study conducted in two Southern Finnish districts, namely the Helsinki Metropolitan area and Lappeenranta, between 2008 and 2014. A total of 496 (68% all the women assessed for eligibility) Finnish women with an elevated risk for GDM due to obesity (BMI ≥30 kg/m²), a history of GDM, or both participated. They were either less than 20 weeks pregnant (n=293) or were planning pregnancy (n=203). These women were allocated to a control (n=236) or an intervention (n=260) group. Exclusion criteria were age ≤ 18 years, diabetes diagnosed before pregnancy, use of medication influencing glucose metabolism, multiple pregnancy, physical
disability, current substance abuse, severe psychiatric disorder and substantial communication
difficulties. The participants in the control group received standard antenatal care while those in the
intervention group participated in diet and physical activity counselling. Depending on the
recruitment timing (pre- or during pregnancy) the counseling started before pregnancy or at early
pregnancy. The diet intervention was given by a dietitian and it emphasized high intake on fruits,
vegetables, high fiber grains, and fish, and low intake on high fat dairy and meat, and high energy/low
nutrient foods, as well as choosing vegetable fat over animal fat (Korvusalo et al. 2015). Physical activity
counseling encouraged to moderate-intensity physical activities a minimum of 150 min per week and
to an overall active lifestyle. In the present study 45 (18%) participants were recruited before
pregnancy and 206 (82%) participants in early pregnancy. The control (n=119) and the intervention
(n=132) groups were combined to study whether an actual change in dietary pattern would be related
to the incidence of GDM. The participants had a 75g oral glucose tolerance test (OGTT) at 1st and 2nd
trimesters of pregnancy. Only participants with normal OGTT at 1st trimester were included. The final
number of participants of the current study was 251 (50% from all participants). This study was
carried out according to the guidelines laid down in the Declaration of Helsinki and all procedures
involving human subjects were approved by The Ethics Committee of the Department of Obstetrics
and Gynecology of Helsinki and Uusimaa Hospital District. Written informed consent was obtained
from all participants.

One or more pathological glucose value in a 75 g two-hour OGTT (run by a central laboratory) led
to GDM diagnosis. The diagnostic thresholds were: fasting plasma glucose concentration (PG) ≥ 5.3
mmol/l, one hour concentration (1hG) ≥ 10.0 mmol/l and two hour concentration (2hG) ≥ 8.6 mmol/l
(American Diabetes Association 2008). The OGTT was conducted at 6-18 weeks and if normal, the woman was
included in the current study and the OGTT was repeated at 24-28 weeks of pregnancy.

The Healthy Food Intake Index (HFII) was used to measure the adherence of a diet to the NNR. The
HFII has been thoroughly described previously (Meinila et al. 2016). We have shown that the HFII is valid
for ranking participants according to their adherence to the NNR. The results of the validation study
apply among pregnant obese women and women with previous GDM. The components of the HFII
reflect the content of food-based guidelines of the Nordic Nutrition Recommendations (NNR) (Nordic
Council of Ministers 2014). The HFII comprised 11 components covering the following food groups;
vegetables, fruits and berries, high-fiber grains, fish, low-fat milk, low-fat cheese, cooking fat, fat
spread, snacks, sugar-sweetened beverages, and fast food (Table 1). The highest score was set to
reflect the food-based guideline of the NNR, while the lowest was indicative of a poor quality diet.
While higher consumption of recommended foods resulted in higher scores, higher consumption of
foods recommended in moderation (fast food, sugar-sweetened beverages, and snacks) resulted in lower scores. Setting the maximum score was based on a priori assumption of the relative importance of the category for the overall diet quality. The HFII is a sum of the 11 components, and the scores range between 0 and 17. Based on the validation study (Meinila et al. 2016), the higher the HFII scores the lower the intakes of fatty acids and saturated fatty acids, and the higher the intakes of carbohydrate, dietary fiber, vitamin D, folate, and intake of MUFA and PUFA as percentages from total fat. In the validation study the HFII was found to measure primarily three dimensions of the diet: Fats (ratio of low- to high fat cheese, ratio of low- to high fat milk, fat spread quality, cooking fat quality), Healthy foods (fish, vegetables, fruit and berries, high-fiber grains), and Unhealthy foods (snacks, fast food, sugar-sweetened beverages). In the current study HFII-sub-indices for each of these dimensions were created. A sum of the component scores of each dimension resulted in sub-indices Fats, Healthy foods, and Unhealthy foods (Table 1). The data for the HFII were derived from food frequency questionnaires (FFQ). The participants filled in the FFQs at the first visit to the study nurse at 6 to 18 weeks of pregnancy (mean gestational age 12·5 (SD 1·9) and at the second visit at 22 to 30 weeks of pregnancy (mean gestational age 26 (SD 1·9).

The participants filled in a questionnaire on their health, lifestyle, and history of pregnancies and pregnancy-related issues. Information on age, educational attainment, smoking (yes/no), and leisure time physical activity (LTPA) was also collected. LTPA was assessed by a self-report of weekly time spent on LTPA that makes a participant at least slightly out of breath and sweaty. Body mass index (BMI) was calculated from the weight and height measured at the woman’s first visit to the study nurse during pregnancy. Weight gain from 1st to 2nd trimester was calculated from weights measured at first and second trimester visits.

Statistical methods

The descriptive analysis were performed in three groups according to the participants’ baseline HFII. The cut-off points were set to ± one standard deviation from the mean HFII; category I (score range 0-7), II (score range 8-12), and III (score range 13-17), respectively. Normality of the variables was tested by Shapiro-Wilk W test. Difference between women who did and did not develop GDM were analyzed by Student’s t-test, Mann-Whitney U-test, and Fisher’s test. Difference in HFII at 2nd trimester between women who developed GDM and women who did not develop GDM was tested by linear regression analysis. Association of change in the HFII and HFII-sub-indices with GDM were analyzed by logistic regression analysis. The logistic regression analysis were adjusted for baseline HFII (or HFII-Fat / HFII-Healthy / HFII-Unhealthy), age, BMI, previous GDM (no/yes), parity (nulliparous/parous), weight change from 1st to 2nd trimester, LTPA change, and group
assignment (control/intervention). Sensitivity analysis in groups of treatment (intervention/control), parity, previous GDM, and low and high HFII were also performed by logistic regression analysis without adjustments. For the sensitivity analysis HFII scores were divided into two groups by median as a cut-off value. Stata 13.1, StataCorp LP (College Station, TX, USA) statistical package was used for the analyses.

Results

At baseline (1st trimester of pregnancy) the proportions of the participants in the HFII categories (I, II, and III) were 18% (n=46), 60% (n=151), 22% (n=54), respectively. Eighteen percent (n=46) of the participants developed GDM. From the lowest to the highest HFII category (I, II, and III), the proportions of women who developed GDM were 15% (n=7), 17% (n=27), and 22% (n=12), respectively. BMI was higher in women who did not develop GDM compared to women who developed GDM (p=0.006) (Table 2). Most women who developed GDM had a history of previous GDM (67.4%) and this was different compared to women who did not develop GDM (34.1%). No other statistically significant differences in baseline characteristics were observed between the women who developed GDM and women who did not.

The mean HFII score at baseline was 10.1 (95% CI 9.7, 10.4), and the mean HFII change from 1st to 2nd trimester of pregnancy improved by 0.35 (95% CI: 0.09, 0.62) points. The range of the HFII changes varied from 7 to 7. The HFII score decreased, i.e. the adherence to the NNR decreased, from 1st to 2nd trimester of pregnancy in those who developed GDM (Figure 1). On the contrary, the HFII improved in those who did not develop GDM. The odds for GDM was significantly lower with higher change in HFII score from 1st to 2nd trimester of pregnancy (OR 0.83; 95% CI 0.69, 0.99; p=0.043) in the adjusted model (Figure 2). From the other variables in the model, only previous GDM (OR 4.7; 95% CI 1.28, 17.35; p=0.02) was significantly associated with GDM in the index pregnancy. When analyzed in groups according to treatment (intervention/control), parity (nulliparous/parous), previous GDM (no/yes), or HFII at baseline (low/high), the association between HFII change and GDM were parallel between the groups (Figure 3).

The fully adjusted odds for GDM were 0.72 (95% CI: 0.52, 1.01) per one unit (point) increase in HFII-Fat from 1st to 2nd trimester (p=0.058) (Figure 2). Changes in the HFII-Healthy foods (p=0.40) and in the HFII-Unhealthy foods (p=0.33) were not significantly associated with risk of GDM.
Discussion

Our findings suggest that dietary changes towards the food guidelines of the Nordic Nutrition Recommendations (NNR) made during pregnancy may be related to lower risk of GDM and that the HFII can be a practical tool for measuring the changes. The relationship between GDM and change in the HFII seem to attribute most to a change in the HFII-fat score.

The intervention applied probably affected dietary intake of the participants. However, the reasons behind the dietary changes were not our study aims but the association between the changes and later occurrence of GDM. Possible confounding of the intervention could be changes in physical activity. We have addressed this by adjusting the analysis for change in leisure time physical activity (LTPA).

We additionally adjusted the analysis for intervention assignment (intervention/control group) thereby addressing a potential unknown effect of the intervention. A source of bias could be the fact that those with a history of GDM have markedly increased risk for GDM compared to women without a history of GDM. Thus, the effect of dietary changes could be different in women with and women without a history of GDM. However, in a separate analysis according to GDM history the results were similar in women with and without a history of GDM. Association between higher HFII change and lower risk of GDM seemed to concern only parous and not multiparous women. This probably attributes, at least partly, to smaller number of nulliparous compared to multiparous participants in the present study, and higher prevalence of GDM among multiparous compared to nulliparous women. Thus, the data in multiparous women was probably more statistically powered compared to the data of nulliparous women.

Our approach provided novel information on the association between GDM and dietary changes made during pregnancy. According to observational studies GDM is associated with several acknowledged healthy dietary patterns with similar characteristics to a diet adherent to the NNR (Schoenaker et al. 2015, He et al. 2015, Tryggvadottir et al. 2015, Shin et al. 2015). These studies have, however, either prospectively studied the association of GDM to a diet measured at one time point before or during pregnancy, or to long-term dietary intake preceding pregnancy. Thus, a rationale for a comparison with the existing observational studies is limited.

Short-term effects of dietary change in preventing GDM have been studied mainly in diet or combined diet and exercise interventions (Tieu et al. 2008, Han et al. 2012, Bain et al. 2015). Individual dietary intervention studies have not succeeded in preventing GDM, possibly because of the small sample sizes. A meta-analysis found that dietary interventions lowered GDM incidence (Oostdam et al. 2011), whereas another found no effect (Rogozinska E et al. 2015). Although Rogozinska et al. (Rogozinska E et al. 2015) did not find any
Effect of dietary counselling on GDM incidence, in a sub-analysis they found that overweight and obese women benefitted from the intervention whereas in studies including women of any weight the intervention was ineffective. The result supports our findings, because the majority of the participants in our study were obese. Combined diet and physical activity intervention studies are more abundant than interventions concentrating exclusively on dietary counselling (Tieu et al. 2008, Bain et al. 2015). The majority of the several lifestyle intervention studies performed aiming at prevention of GDM, however, have failed. Unfortunately, reports of these lifestyle interventions usually lack results of effect of the actual lifestyle changes on GDM risk, as was evident in a recent Cochrane systematic review (Bain et al. 2015). Although seven out of the twelve included studies reported on dietary adherence to the intervention, none reported the level of adherence in relation to the intervention targets. Thus, whether a certain magnitude of lifestyle change would have been sufficient remained unclear. Two recent lifestyle interventions with diet and physical activity counselling have succeeded in preventing GDM (Jing et al. 2015, Koivusalo et al. 2016), which is in accordance with the current results. We recently reported that the RADIEL study was successful in preventing GDM (Koivusalo et al. 2016) but we did not yet report on the effects of the actual lifestyle changes on GDM. Changes in food intakes were seen in low-fat cheese and fish (Valkama et al. 2015). Not detectable subtle changes in other individual foods were now taken into account in the HFII change of the current study. These results suggest that compliance with dietary counselling based on the NNR could result in lower risk of GDM. The causes of the contradictory results of lifestyle intervention studies need further investigation. A somewhat surprising finding was the observation that women in the highest HFII category (who had the highest adherence to the NNR) had the highest GDM incidence. This probably reflects the inclusion criteria that allowed participants who were non-obese but had a history of GDM. These women appeared to have the highest risk for GDM (Koivusalo et al. 2016, Huvinen et al. 2016). Because of the heterogeneity of the underlying mechanisms of GDM (Huvinen et al. 2016) the same dietary strategy may not be equally effective among all high risk women (Grotenfelt et al. 2016). This area needs further studying.

The results suggest that for GDM prevention the foods included in the HFII-Fat (milk fat-%, cheese fat-%, and spread fat, cooking fat) may be the most important target of dietary change during pregnancy. This may also reflect a possible higher accuracy of the HFII-Fat components compared to other components of the HFII. In the reproducibility analysis of our recent paper (Meinila et al. 2016) the components of the HFII-fat showed slightly higher reproducibility compared to other components. The components measured choices between high- vs. low-fat and vegetable vs. animal fat products. HFII-Fat may measure a dietary aspect more constant over time than intake frequencies of foods.
which the other components measured. Thus, the sub-indices may not be equally comparable with each other.

This study has some limitations. Originally the HFII was not designed to measure dietary change, and it may be rather insensitive for measuring change. This suggests that the magnitude of the relationship between the HFII and GDM could be even stronger than what was detected in the present study. In addition, all the components of the HFII do not measure change equally. For example, decreasing the frequency of snacks (candy, pastry, chocolate, chips, and ice cream) from four times a week does not produce any change in the HFII score. Similarly, if one consumes vegetables once a day at baseline and increases it to two times, the HFII score remains unchanged. Subtle changes remaining unnoticed may result in differences in the ability of the HFII-sub-scores to detect changes. It is also important to notice that the HFII does not measure changes in all foods. Thus, some possibly relevant aspects of diets in relation to GDM may have remained unnoticed. The underlying FFQ for the HFII was not validated. However, the HFII was validated against 3-day food records using the same FFQ (Meinila et al. 2016). As this is an observational study we cannot conclude on causality because of possible residual confounding. In addition, LTPA was self-reported which may not be a reliable measure of true physical activity (Evenson et al. 2012). The study group did not represent the general population but those at high risk for GDM due to obesity and/or to a history of GDM. Nonetheless, these criteria concern a large group of the population; one third of Finnish women at reproductive age are overweight or obese (National Institute for Health and Welfare 2013). The results can, however, be generalized to only obese pregnant women and pregnant women with a history of GDM.

Strengths of the current study include a thoroughly validated HFII shown to measure adherence of diet to the NNR in a high GDM risk population. A longitudinal setting allowed us to study change within an individual instead of differences between individuals, which cannot be assumed to reflect true change. Prospective setting with dietary measures at two different time points provide stronger evidence of causality than dietary measure at one single time point only. By testing and excluding those with pathologic OGTT at baseline we excluded the possible effect of the GDM diagnosis on diet and excluded the possibility of reverse causality. The RADIEL main trial and the current study uniquely included participants who were non-obese but had a history of GDM. By including these women we took into account the heterogeneity of the pathophysiology of GDM (Huvinen et al. 2016).

**Conclusions**

Dietary change towards the Nordic Nutrition Recommendations during pregnancy may be related to lower risk of GDM. This supports the idea that GDM prevention by dietary changes started during
early pregnancy could be effective. Well-powered interventions with detailed measures of intervention adherence, as well as descriptive studies on obstacles for intervention adherence are necessary. Studies on interaction of diet and different pathophysiologic factors of GDM are also needed.

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**Conflicts of interest:** None.

**Authorship:** JM designed, conducted research, performed statistical analysis, wrote the paper, had primary responsibility for final content, AV designed and conducted research, wrote the paper, and read and approved the final manuscript, SK designed and conducted research, critically revised the manuscript with expert advice and comments, and read and approved the final manuscript, KR designed and conducted research, critically revised the manuscript with expert advice and comments, and read and approved the final manuscript, HK designed research, performed statistical analysis, critically revised the manuscript with expert advice and comments, read and approved the final manuscript, BS-L designed, conducted research, critically revised the manuscript with expert advice and comments, and read and approved the final manuscript, JE designed and conducted research, critically revised the manuscript with expert advice and comments, read and approved the final manuscript, ME designed, performed statistical analysis, wrote the paper, had primary responsibility for final content, and read and approved the final manuscript.
References:


Figure 1. Mean and 95% confidence interval of the Healthy Food Intake Index (range 0-17) at 1st and 2nd trimesters of pregnancy in non-GDM (GDM-) and GDM (GDM+) groups.

Figure 2. Association of Healthy Food Intake Index (HFII) change (range from -7 to 7), HFII-Fat change (range from -4 to 5), HFII-Healthy foods change (range from -5 to 4), and HFII-Unhealthy foods change (range from -3 to 3) with risk (Log(OR) with 95% confidence interval) of gestation diabetes (GDM) in obese women and women with a history of gestational diabetes. The models were adjusted for baseline HFII (or HFII-Fat/HFII-Healthy foods/HFII-Unhealthy foods), age, BMI, previous GDM (no/yes), parity (nulliparous/parous), weight change from 1st to 2nd trimester, leisure time physical activity change, and group assignment (control/intervention).

Figure 3 Association of Healthy Food Intake Index (HFII) change (range from -7 to 7) with risk (crude log (OR) with 95% confidence interval) of gestation diabetes in obese women and women with a history of gestational diabetes in subgroups of treatment group, parity, previous gestational diabetes, and baseline HFII (median cut-off).
Table 1. The HFII-sub-indices among the components of the Healthy Food Intake Index (HFII) and their scoring studied among pregnant Finnish women at high risk of gestational diabetes.

<table>
<thead>
<tr>
<th>Components</th>
<th>Scoring and cut-off limits</th>
<th>Scoring range of the sub-index</th>
</tr>
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<tbody>
<tr>
<td>HFII-Fat</td>
<td></td>
<td></td>
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<tr>
<td>Cheese fat %*</td>
<td>low-fat (&lt;17%) 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>high fat (≥17%) 0</td>
<td></td>
</tr>
<tr>
<td>Milk fat %*</td>
<td>low-fat (≤1%) 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>low- and high fat 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>high fat (&gt;1%) 0</td>
<td></td>
</tr>
<tr>
<td>Fat spread*</td>
<td>exclusively vegetable fat 2</td>
<td>sum of the component scores: 0-6</td>
</tr>
<tr>
<td></td>
<td>both vegetable and animal fat 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>exclusively animal fat / no fat 0</td>
<td></td>
</tr>
<tr>
<td>Cooking fat*</td>
<td>vegetable fat 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>animal fat 0</td>
<td></td>
</tr>
<tr>
<td>HFII-Healthy foods</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish†</td>
<td>≥1x/d 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&lt;1x/d 0</td>
<td></td>
</tr>
<tr>
<td>Vegetables†</td>
<td>&gt;2x/d 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1–2 x/d 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&lt;1x/d 0</td>
<td></td>
</tr>
<tr>
<td>Fruit and berries†</td>
<td>≥1x/d 1</td>
<td>sum of the component score: 0-7</td>
</tr>
<tr>
<td></td>
<td>&lt;1x/d 0</td>
<td></td>
</tr>
<tr>
<td>High-fiber grains†</td>
<td>≥3x/d 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1-2x/d 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&lt;1x/d 0</td>
<td></td>
</tr>
<tr>
<td>HFII-Unhealthy foods</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snacks‡</td>
<td>≤4 x / wk 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5–6 x / wk1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥1 x / d 0</td>
<td>sum of the component scores: 0-4</td>
</tr>
<tr>
<td>Fast food§</td>
<td>&lt;1 x / wk 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥1 x / wk 0</td>
<td></td>
</tr>
<tr>
<td>Sugar-sweetened beverages§</td>
<td>&lt;1 x / wk 1</td>
<td></td>
</tr>
</tbody>
</table>
≥1/wk 0

*Cutoffs are based on the NNR. †Cut-offs are based on the Finnish Nutrition Recommendations 2014 and a consensus-panel decision. ‡Cut-offs are based on tertiles. §Cut-offs based on median.
Table 2. Baseline (1st trimester of pregnancy) demographic and clinical characteristics of pregnant women at high risk of gestational diabetes (GDM) (n=251).

<table>
<thead>
<tr>
<th></th>
<th>GDM- (n=205)</th>
<th>GDM+ (n=46)</th>
<th>P†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>32,1 4,6</td>
<td>32,9 3,9</td>
<td>0,28</td>
</tr>
<tr>
<td>BMI at 1st trimester (kg/m²)</td>
<td>32,0 5,3</td>
<td>29,0 6,6</td>
<td>0,006</td>
</tr>
<tr>
<td>Leisure time physical activity (min/week)</td>
<td>144,1 218,1</td>
<td>105,0 141,5</td>
<td>0,46‡</td>
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<tr>
<td>Education (years)</td>
<td>14,5 2,4</td>
<td>14,3 2,5</td>
<td>0,69</td>
</tr>
<tr>
<td>HFII score (points)</td>
<td>10,0 2,8</td>
<td>10,5 2,8</td>
<td>0,27</td>
</tr>
<tr>
<td>Fats (points)</td>
<td>4,0 1,7</td>
<td>4,3 1,3</td>
<td>0,22</td>
</tr>
<tr>
<td>Healthy foods (points)</td>
<td>3,7 1,7</td>
<td>3,6 1,6</td>
<td>0,82</td>
</tr>
<tr>
<td>Unhealthy foods (points)</td>
<td>2,3 1,0</td>
<td>2,6 1,1</td>
<td>0,15</td>
</tr>
<tr>
<td>Fasting PG concentration (mmol/l)</td>
<td>4,88 0,24</td>
<td>4,88 0,25</td>
<td>0,90</td>
</tr>
<tr>
<td>PG 1 hour post load (mmol/l)</td>
<td>6,80 1,36</td>
<td>7,79 1,22</td>
<td>&lt;0,001</td>
</tr>
<tr>
<td>PG 2 hour post load (mmol/l)</td>
<td>5,75 1,07</td>
<td>6,31 1,23</td>
<td>0,002</td>
</tr>
<tr>
<td>n %</td>
<td></td>
<td>n %</td>
<td>P§</td>
</tr>
<tr>
<td>Previous GDM</td>
<td></td>
<td></td>
<td>&lt;0,001</td>
</tr>
<tr>
<td>No</td>
<td>52 25,4</td>
<td>4 8,7</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>70 34,1</td>
<td>31 67,4</td>
<td></td>
</tr>
<tr>
<td>Nullipara</td>
<td>83 40,5</td>
<td>11 23,9</td>
<td></td>
</tr>
<tr>
<td>At least one parent has a history of diabetes (type 1 or 2)</td>
<td>44 22</td>
<td>15 32,6</td>
<td>0,13</td>
</tr>
<tr>
<td>Mother has a history of GDM</td>
<td>18 8,8</td>
<td>2 4,3</td>
<td>0,55¶</td>
</tr>
</tbody>
</table>

GDM-, group of women who did not develop GDM; GDM+, group of women who developed GDM; BMI, body mass index; HFII, Healthy Food Intake Index; PG, plasma glucose. †Tested by Student’s t-test if not stated otherwise, ‡Mann-Whitney test, §Chi square test if not stated otherwise, ¶Fisher’s exact test.
Figure 1. Mean and 95% confidence interval of the Healthy Food Intake Index (range 0-17) at 1st and 2nd trimesters of pregnancy in women who did not develop gestational diabetes (GDM-) and women who developed GDM (GDM+) groups.
Figure 2. Association of Healthy Food Intake Index (HFII) change (range from -7 to 7), HFII-Fat change (range from -4 to 5), HFII-Healthy foods change (range from -5 to 4), and HFII-Unhealthy foods change (range from -3 to 3) with risk (Log(OR) with 95% confidence interval) of gestation diabetes (GDM) in obese women and women with a history of gestational diabetes. The models were adjusted for baseline HFII (or HFII-Fat/HFII-Healthy foods/HFII-Unhealthy foods), age, BMI, previous GDM (no/yes), parity (nulliparous/parous), weight change from 1st to 2nd trimester, leisure time physical activity change, and group assignment (control/intervention).
Figure 3 Association of Healthy Food Intake Index (HFII) change (range from -7 to 7) with risk (crude log (OR) with 95% confidence interval) of gestation diabetes in obese women and women with a history of gestational diabetes in subgroups of treatment group, parity, previous gestational diabetes, and baseline HFII (median cut-off).