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Association between inflammatory potential of diet and odds of gestational diabetes mellitus among Iranian women

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Abstract

Background: The possible relationship between diet-related inflammation and the risk of gestational diabetes mellitus (GDM) requires further investigation, especially in non-Western populations. We examined the association between dietary inflammatory index (DII) scores and GDM in a case-control study conducted in Iran.

Methods: This study included 122 GDM cases and 266 controls hospitalized for acute non-neoplastic diseases. Cases were pregnant women aged 18–40 years, who visited major general hospitals in different regions of Tehran. Pregnant women were screened for gestational diabetes between the 24th and 28th week of gestation with a 50-g who 1-hr glucose challenge test (GCT). Cases were diagnosed positive for GDM. Controls were pregnant women who had normal GCT test. DII scores were computed from dietary intake assessed by a previously validated 147-item food frequency questionnaire. Logistic regression models adjusted age, gestational age, energy, exercise, BMI, smoking exposure, family history of diabetes, and history of multivitamin intake were used to estimate odds ratios (ORs) and 95% confidence intervals (CI).

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Disclosure: Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI. The subject matter of this paper will have no direct bearing on the work of CHI, nor has any CHI-related activity exerted any influence on this project.

Results: Subjects with higher DII scores (i.e., indicating a more pro-inflammatory diet) had a higher odd of GDM with the DII being used as both a continuous (OR=1.20; 95% CI=0.94–1.54) and as categorical (OR $_{\text{tertile 3vs1}}$=2.10; 95% CI=1.02–4.34, P-trend=0.03)

Conclusion: These results indicate that a pro-inflammatory diet, as evidenced by higher DII scores, is associated with increased odds of GDM among Iranian women.

Introduction

Chronic inflammation is characterized by the continuous presence of inflammatory cytokines in circulation and systemically; and this might lead to chronic diseases such as obesity, diabetes and cancer (1, 2). One of the most common complications of pregnancy is gestational diabetes mellitus (GDM) (3). The prevalence of GDM in pregnancy varies widely according to the study population and the diagnostic test used, and ranges from 2.4% to 21% (4). GDM is defined as any degree of glucose intolerance recognized first during pregnancy (4). The prevalence of GDM is about 14% in the United States (5) and in Iran it is 4.7% (95% CI, 3.91 – 5.64%) using Carpenter and Coustan criteria, and 3.97% using National Diabetes Data Group (NDDG) criteria (6).

Increasing incidence of GDM causes serious concerns for health systems worldwide (7). GDM is associated with maternal (premature birth, infectious complications, hydramnios, hypertensive complications) and fetal (still birth, altered fetus growth, metabolic disturbances, respiratory distress syndrome, obesity in childhood and diabetes) complications (8). Infants are more likely to become obese, have impaired glucose tolerance or develop diabetes in adolescence or early adulthood, compared to offspring of normoglycaemic females (9). As a result, this population needs special attention, particularly in the developing countries. There is strong evidence that specific dietary components influence both inflammation (10–13) and GDM (14, 15). Some food items such as fish and fruits exert an anti-inflammatory effect (16, 17), whereas dietary pattern rich in red meat increase inflammation (18). There is evidence showing inflammatory cytokines which regulate inflammation in GDM (19).

The literature-derived, population-based dietary inflammatory index (DII) was developed to assess the inflammatory potential of individuals' diets (20), and has been validated with various inflammatory markers, including C-reactive protein (21, 22), interleukin-6 (23–25), and tumor necrosis factor-alpha (24). In this study, we explore the association between the DII and GDM in a case-control study in Iran (26). The DII has been used in Iranian populations in the past; with higher DII scores shown to be associated with lower bone mineral density (BMD) in lumbar spine among postmenopausal women (27), and increased risk of esophageal squamous cell cancer (ESCC) (28), multiple sclerosis (29), ulcerative colitis (30), prediabetes (31) and recurrent abortion (32).

Our hypothesis is that higher DII scores (indicating a more pro-inflammatory diet) are associated with increased odds of incident GDM.

Material and methods

Study population

This hospital-based case-control study was conducted in Tehran, a province of Iran at high-risk of diabetes. Cases were pregnant women aged 18–40 years, who visited major general hospitals in different regions of Tehran (11 million inhabitants). Pregnant women were screened for gestational diabetes between the 24th and 28th week of gestation with a 50-g, 1-hr glucose challenge test (GCT). If the screening test was positive (blood glucose greater than 130 mg per ml), diagnostic testing was performed using a 100 g, 3-hour oral glucose tolerance test (OGTT). Women meeting Carpenter and Coustan criteria: fasting, 5.3 mmol/l; 1 h, 10.0 mmol/l; 2 h, 8.6 mmol/l; 3h, 7.8 mmol/l (33), were diagnosed with GDM (any two values at or above established thresholds).

Controls were pregnant women whose GCT tests at 24–28 weeks of pregnancy were in normal range. The exclusion criteria were multiple pregnancies, history of gestational diabetes or diabetes (pre-pregnancy) and undergoing a weight-reduction diet one year before pregnancy. Furthermore, controls were followed up until the end of pregnancy. If they developed GDM, they were excluded from the study. In this study, two controls were recruited within the same medical center for each case. Controls were matched to cases on age (within 5 years). During analysis, three patients (1 cases and 2 controls) with extreme energy intakes that probably reflected careless completion of the dietary questionnaire (below or above the mean ± 3 SD for loge-transformed calories; cutpoints: 244 kcal and 5284 kcal) were excluded. The final sample for statistical analysis was 122 cases and 266 controls.

Dietary assessment

Dietary habits of participants (one year before the interview) were obtained by trained dietitians using a valid and reproducible 147-item semi-quantitative food frequency questionnaire (FFQ) (34). The consumption frequency of each food items was gathered on a daily, weekly, or monthly basis; the portion sizes were assessed using household measures and then converted to grams (35). A validated food album [2] and a set of household measurements (e.g., cup, tablespoon, teaspoon plate, glass, small bowl, and spatula) were used to assist respondents to estimate the portion size and type of food items. Respondents were asked to report the frequency of consumption of a given serving of each food item on a daily, weekly, monthly and yearly basis and data were then converted to daily intake frequency. Portion size of each food item consumed was converted to grams. Intake of each food item in grams was then determined by multiplying the portion size by daily intake frequency. The edible fraction of foods also was considered using household measurement guidelines (35). Total energy intake was estimated by adding the energy value of each food in the FFQ. Nutrients value was based on the Nutrient Composition of Iranian Foods (36). The USDA Food Composition Data (37) was used for foods or food ingredients that were not available in Nutrients Composition of Iranian Foods.

Other variables

Information on age, pre-pregnancy weight, education level, socioeconomic status, smoking status, family history of diabetes and taking supplements were obtained from all cases and controls through a general questionnaire. Height was measured without shoes to the nearest 0.5 cm using meter installed on the wall. BMI was calculated by dividing weight in kilograms by the square of height (m). Physical activity was assessed through valid and reliable questionnaire (38). Physical activity was calculated considering Metabolic Equivalent (MET) and reported as MET-h/wk (39).

FFQ-derived dietary data were used to calculate DII scores for all participants. The DII is based on literature published through 2010 linking diet to inflammation. Individuals' intakes of food parameters on which the DII is based are then compared to a world intake standard database. A complete description of the DII is available elsewhere (20). A description of validation work, including derivation of the DII from both dietary recalls and a structured questionnaire similar to an FFQ and related to interval values of hs-CRP, also is available (21). Briefly, the literature relating to the association between dietary components and six different inflammatory markers (IL-1β, IL-4, IL-6, IL-10, TNF-α and C-reactive protein) published from 1950 to 2010 was reviewed. Each of the 45 different food parameters identified as being related to the six inflammatory biomarkers in this massive search was assigned a "food parameter-specific inflammatory effect score" through a process of counting the number of studies reporting pro-inflammatory, anti-inflammatory, and no effects on the six inflammatory markers, and weighting the scores by study design and size of the literature pool. A total of 32 food parameters were available from the FFQ and therefore could be used to calculate DII scores (energy, carbohydrate, protein, total fat, fiber, cholesterol, saturated fat, mono-unsaturated fat, poly unsaturated fat, omega-3, omega-6, trans fat, niacin, thiamin, riboflavin, vitamin B12, vitamin B6, iron, magnesium, selenium, zinc, vitamin A, vitamin C, vitamin D, vitamin E, folic acid, beta carotene, garlic, turmeric, onion, caffeine).

To calculate DII for the participants of this study, the dietary data were first linked to the regionally representative world database that provided an estimate of a mean intake and standard deviation for each food parameter (20). These then become the multipliers to express an individual's exposure relative to the "standard global mean" as a z-score. This is achieved by subtracting the "standard global mean" from the amount reported and dividing this value by the standard deviation. To minimize the effect of "right skewing" (a common occurrence with dietary data), this value was then converted to a centered percentile score. The centered percentile score for each food parameter for each individual was then multiplied by the respective food parameter-specific inflammatory effect score, which is derived from the literature review, as described above, in order to obtain a food parameter-specific DII score for an individual. All of the food parameter-specific DII scores are then summed to create the overall DII score for every participant in the study (20).

Statistical Analyses:

Study characteristics were examined by case-control status. The DII was analyzed both as a continuous variable and as tertiles with cutpoints derived from controls. The DII, as tertiles,

was examined across the following characteristics: age, gestational age, BMI, exercise, smoking exposure, family history of diabetes and history of multivitamin intake in the year before pregnancy. Student *t*-tests and ANOVA were used for continuous and χ^2 tests was used for categorical variables. Odds ratios and 95% confidence intervals (OR; 95% CI) were estimated using logistic regression models, adjusting only for age and energy, and then fitting a model with additional adjustment for gestational age, BMI, exercise, smoking, family history of diabetes and history of multivitamin intake in the year before pregnancy. P-value for trend was determined with value of the median DII in each tertile. Statistical tests were performed using SAS® 9.3 (SAS Institute Inc., Cary, NC); all p values were based on two-sided tests.

Results

DII scores in this study ranged from −4.03 (most anti-inflammatory score) to +4.72 (most pro-inflammatory score). Table 1 shows the socio-demographic and lifestyle characteristics of the 122 cases and 266 controls. The mean age of women at diagnosis of GDM was 29.76±4.26 years and 29.64±4.52 years for controls. Women with GDM had significantly higher BMI and family history of diabetes and significantly lower gestational age, physical activity and. Cases had significantly higher mean DII scores compared to controls (0.15±0.89 vs −0.07±1.05, p-value=0.04). Control characteristics across categories of DII are provided in Table 2. There were some differences in sociodemographic factors, and lifestyle habits across DII categories. In particular, participants in the third tertile of DII were less likely to have history of supplement use and had lower gestational age, but none of these results were statistically significant.

ORs and 95% CIs for the risk of GDM are shown in Table 3. Results obtained from modeling DII as a continuous variable in relation to risk of GDM showed a positive association after adjustment for age and energy intake (OR=1.25; 95% CI=1.01–1.56); and were nearly identical in the multivariable analyses (OR=1.20; 95% CI=0.94–1.54); however, the result was not longer significant. When analyses were carried out with DII expressed as tertiles, and adjusting for age and energy, subjects in tertile 3 had an OR of 2.65 (95% CI= 1.39–5.07, p_{trend}=0.007) in comparison to subjects in tertile 1. Again, after multivariable adjustment, results were essentially identical as in the model adjusting only for age and energy ($OR_{\text{tertile 3vs1}}$=2.10; 95% CI=1.02–4.34, p_{trend}=0.03).

Discussion

In this Iranian case-control study, we found that women with a higher DII score (i.e., those who had more pro-inflammatory diets) were at increased odds of GDM compared to subjects with more anti-inflammatory diets. This is the first study to explore this association. These results support the hypothesis that consuming a more pro-inflammatory diet is associated with increased GDM risk.

In a prospective study conducted in China, a vegetarian dietary pattern was associated with a decreased risk of GDM, while the sweets and seafood pattern was associated with an increased risk of GDM (40). Results from a study conducted in the US, showed high

consumption of refined grains, fat, added sugars and low intake of fruits and vegetables during pregnancy to be associated with higher odds for GDM (41). Reports from systematic review from 34 articles comprising 21 individual studies (10 prospective cohorts, 6 cross-sectional, and 5 case-control) showed dietary pattern rich in fruit, vegetables, whole grains, and fish and low in red and processed meat, refined grains and high-fat dairy to be beneficial against GDM (42). Consumption of food items such as fiber, vegetables and fruits has been shown to reduce inflammation (43, 44), while other foods, such as red and processed meat, increase inflammation (45). In a case-control study conducted in Iran, adherence to either the Dietary Approaches to Stop Hypertension (DASH) or Mediterranean diet is associated with decreased risk for GDM (46).

This is the first study in a Middle-Eastern population to examine the DII in relation to GDM. In a recently published study, after multivariate adjustment, subjects in the most pro-inflammatory DII group had 19 times higher odds of developing pre-diabetes compared to subjects in tertile 1 (DII_{T3VS1}: OR=18.88; 95%CI 7.02, 50.82) (31), in the same study subjects in tertile 3 of DII had significantly higher fasting plasma glucose (FPG) (DII_{T3VS1}: b=4.49; 95%CI 1.89, 7.09), OGT (DII_{T3VS1}: b=8.76; 95%CI 1.78, 15.73), HbA1c (DII_{T3VS1}: b=0.30; 95%CI 0.17, 0.42) (47). During pregnancy, some degree of immune system modulation occurs wherein there is a reduction of pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-α), IL-1ß, and IL-6 and an increase of counter regulatory cytokines, such as IL-10 (48). This process is disturbed during GDM wherein pancreatic ß-cell destruction occurs resulting in pro-inflammatory imbalance created by a sustained elevation of cytokines like IL-1ß and TNF-α and reduction of IL-10 (49).

There are several limitations worth noting in the present study. First, the possibility of selection bias is difficult to avoid in hospital-based case–control studies. The present study minimized this problem because of the high (approximately 80%) participation rate of eligible subjects who were approached to participate. Errors in self-reported intake estimates, through which cases may recall their diets differently after a disease diagnosis, may be common in case-control studies (50, 51). The probability of recall bias may have been reduced by using hospital controls and administering a validated FFQ by trained interviewers in a hospital setting. We also know that such dietary self-reports may be plagued by disease-independent response sets, such as social approval and social desirability (47, 52, 53). These were not measured in the study, nor do we know how they would function in Iranian culture. Also, our participants were generally of low literacy and socioeconomic status, and likely to have little knowledge about the role of diet and nutrients in the abortion. This should have reduced the possibility of recall bias.

Having information on measurement errors may help to control for inaccuracies in dietary recall (53–56). Another limitation is that only 32 of possible 45 food parameters were available for DII calculation. The food parameters that are missing are flavonoids, turmeric, thyme and others that are usually consumed in relatively small amounts or not consumed at all; hence, they may not have had a major impact on the scoring.

One common practice is to use patients with another disease as a control group, with the assumption that the exposure under study (diet or DII) is unrelated to the condition of this

control group (for example orthopedics conditions). Because diet may well affect many diseases, it is often difficult to identify disease groups that are definitely unrelated to the aspects of diet under investigation. Another limitation is that pregnant women with chronic inflammation related diseases like rheumatoid arthritis and preeclampsia were not excluded from the study. However, the overall age-adjusted prevalence of rheumatoid arthritis in Iran is low (0.33% (95% CI: 0.22–0.46)) (57), similarly, reports from a recent study conducted in Iran, showed the prevalence of preeclampsia to be very low (0.05 (95% CI: 0.05, 0.06)) (58). Hence, the low prevalence of these diseases would not have affected our results.

One important strength of the study is that this is one of the first in a Middle Eastern population to explore the association between the inflammatory property of diet and GDM. As diets vary considerably across the globe, it is important to examine the association in a variety of populations with different ways of eating. Of note, the range in DII scores in this Iranian population was narrower than in other populations that we have studied in North America. This may be a reflection of the reduced number of DII food parameters available from the dietary data in this study, or a true difference in the heterogeneity of inflammatory potential of diets across populations. Future work with larger study populations and more detailed dietary assessment is warranted to confirm these findings. Other strengths include ability to control for total energy intake and major potential confounding factors.

In conclusion, subjects who consumed a more pro-inflammatory diet were at increased odds of GDM compared to those who consumed a more anti-inflammatory diet. Thus, encouraging intake of more anti-inflammatory dietary factors, such as plant-based foods rich in fiber and phytochemicals, and reducing intake of pro-inflammatory factors, such as fried or processed foods rich in saturated fat or trans-fatty acids, may be a strategy for reducing risk of GDM.

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References

1. Coussens LM, Werb Z. Inflammation and cancer. Nature. 2002 12 19–26;420(6917):860–7. PubMed PMID: . [PubMed: 12490959]
2. Philip M, Rowley DA, Schreiber H. Inflammation as a tumor promoter in cancer induction. Semin Cancer Biol. 2004 12;14(6):433–9. PubMed PMID: . [PubMed: 15489136]
3. Russo LM, Nobles C, Ertel KA, Chasan-Taber L, Whitcomb BW. Physical activity interventions in pregnancy and risk of gestational diabetes mellitus: a systematic review and meta-analysis. Obstetrics and gynecology. 2015 3;125(3):576–82. PubMed PMID: . [PubMed: 25730218]
4. Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL. Diabetes melitus williams obstetrics. New York: Mc Graw Hill 2014.
5. Metzger BE, Buchanan TA, Coustan DR, de Leiva A, Dunger DB, Hadden DR, et al. Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus. Diabetes Care. 2007 7;30 Suppl 2:S251–60. PubMed PMID: . [PubMed: 17596481]

6. Dyck R, Klomp H, Tan LK, Turnell RW, Boctor MA. A comparison of rates, risk factors, and outcomes of gestational diabetes between aboriginal and non-aboriginal women in the Saskatoon health district. Diabetes Care. 2002 3;25(3):487–93. PubMed PMID: . [PubMed: 11874935]
7. Ferrara A Increasing prevalence of gestational diabetes mellitus: a public health perspective. Diabetes Care. 2007 7;30 Suppl 2:S141–6. PubMed PMID: . [PubMed: 17596462]
8. Dempsey JC, Sorensen TK, Williams MA, Lee IM, Miller RS, Dashow EE, et al. Prospective study of gestational diabetes mellitus risk in relation to maternal recreational physical activity before and during pregnancy. Am J Epidemiol. 2004 4 01;159(7):663–70. PubMed PMID: . [PubMed: 15033644]
9. Deierlein AL, Siega-Riz AM, Evenson KR. Physical activity during pregnancy and risk of hyperglycemia. Journal of women's health. 2012 7;21(7):769–75. PubMed PMID: Pubmed Central PMCID: 3387759. [PubMed: 22537020]
10. Santos S, Oliveira A, Lopes C. Systematic review of saturated fatty acids on inflammation and circulating levels of adipokines. Nutrition research. 2013 9;33(9):687–95. PubMed PMID: . [PubMed: 24034567]
11. Bordoni A, Danesi F, Dardevet D, Dupont D, Fernandez AS, Gille D, et al. Dairy Products and Inflammation: A Review of the Clinical Evidence. Critical reviews in food science and nutrition. 2015 8 19:0 PubMed PMID: . [PubMed: 26287637]
12. Barbaresko J, Koch M, Schulze MB, Nothlings U. Dietary pattern analysis and biomarkers of low-grade inflammation: a systematic literature review. Nutr Rev. 2013 8;71(8):511–27. PubMed PMID: . [PubMed: 23865797]
13. Simopoulos AP. The importance of the ratio of omega-6/omega-3 essential fatty acids. Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie. 2002 10;56(8):365–79. PubMed PMID: . [PubMed: 12442909]
14. Tieu J, Shepherd E, Middleton P, Crowther CA. Dietary advice interventions in pregnancy for preventing gestational diabetes mellitus. The Cochrane database of systematic reviews. 2017 1 03;1:CD006674 PubMed PMID: . [PubMed: 28046205]
15. Bain E, Crane M, Tieu J, Han S, Crowther CA, Middleton P. Diet and exercise interventions for preventing gestational diabetes mellitus. The Cochrane database of systematic reviews. 2015 4 12(4):CD010443. PubMed PMID: . [PubMed: 25864059]
16. Pot GK, Geelen A, Majsak-Newman G, Harvey LJ, Nagengast FM, Witteman BJM, et al. Increased Consumption of Fatty and Lean Fish Reduces Serum C-Reactive Protein Concentrations but Not Inflammation Markers in Feces and in Colonic Biopsies. The Journal of Nutrition. 2010 2 1, 2010;140(2):371–6. [PubMed: 20032491]
17. Root MM, McGinn MC, Nieman DC, Henson DA, Heinz SA, Shanely RA, et al. Combined fruit and vegetable intake is correlated with improved inflammatory and oxidant status from a cross-sectional study in a community setting. Nutrients. 2012 1;4(1):29–41. PubMed PMID: eng. [PubMed: 22347616]
18. Wood AD, Strachan AA, Thies F, Aucott LS, Reid DM, Hardcastle AC, et al. Patterns of dietary intake and serum carotenoid and tocopherol status are associated with biomarkers of chronic low-grade systemic inflammation and cardiovascular risk. The British journal of nutrition. 2014 10 28;112(8):1341–52. PubMed PMID: Epub 2014/10/15. eng. [PubMed: 25313576]
19. Skorzynska-Dziduszko KE, Kimber-Trojnar Z, Patro-Malysza J, Olszewska A, Zaborowski T, Malecka-Massalska T. An Interplay between Obesity and Inflammation in Gestational Diabetes Mellitus. Current pharmaceutical biotechnology. 2016;17(7):603–13. PubMed PMID: . [PubMed: 26813307]
20. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hebert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public health nutrition. 2014 8;17(8):1689–96. PubMed PMID: Pubmed Central PMCID: 3925198. [PubMed: 23941862]
21. Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, et al. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood Cholesterol Study (SEASONS). Public health nutrition. 2014 8;17(8):1825–33. PubMed PMID: Pubmed Central PMCID: 3983179. [PubMed: 24107546]

22. Wirth MD, Burch J, Shivappa N, Violanti JM, Burchfiel CM, Fekedulegn D, et al. Association of a dietary inflammatory index with inflammatory indices and metabolic syndrome among police officers. Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine. 2014 9;56(9):986–9. PubMed PMID: Pubmed Central PMCID: 4156884. [PubMed: 25046320]
23. Ruiz-Canela M, Zazpe I, Shivappa N, Hebert JR, Sanchez-Tainta A, Corella D, et al. Dietary inflammatory index and anthropometric measures of obesity in a population sample at high cardiovascular risk from the PREDIMED (PREvencion con DIeta MEDiterranea) trial. The British journal of nutrition. 2015 3 28;113(6):984–95. PubMed PMID: Pubmed Central PMCID: 4870040. [PubMed: 25720588]
24. Tabung FK, Steck SE, Zhang J, Ma Y, Liese AD, Agalliu I, et al. Construct validation of the dietary inflammatory index among postmenopausal women. Annals of Epidemiology. 2015 6//;25(6):398–405. [PubMed: 25900255]
25. Wood LG, Shivappa N, Berthon BS, Gibson PG, Hebert JR. Dietary inflammatory index is related to asthma risk, lung function and systemic inflammation in asthma. Clinical and experimental allergy : journal of the British Society for Allergy and Clinical Immunology. 2015 1;45(1):177–83. PubMed PMID: Pubmed Central PMCID: 4190104. [PubMed: 24708388]
26. Safari A, Shariff ZM, Kandiah M, Rashidkhani B, Fereidooni F. Dietary patterns and risk of colorectal cancer in Tehran Province: a case-control study. BMC public health. 2013;13:222 PubMed PMID: Pubmed Central PMCID: 3605096. [PubMed: 23497250]
27. Ramallal R, Toledo E, Martinez-Gonzalez MA, Hernandez-Hernandez A, Garcia-Arellano A, Shivappa N, et al. Dietary Inflammatory Index and Incidence of Cardiovascular Disease in the SUN Cohort. PloS one. 2015;10(9):e0135221 PubMed PMID: Pubmed Central PMCID: 4560420. [PubMed: 26340022]
28. Shivappa N, Jackson MD, Bennett F, Hebert JR. Increased Dietary Inflammatory Index (DII) Is Associated With Increased Risk of Prostate Cancer in Jamaican Men. Nutrition and cancer. 2015 Aug-Sep;67(6):941–8. PubMed PMID: Pubmed Central PMCID: 4596719. [PubMed: 26226289]
29. Shivappa N, Hebert JR, Behrooz M, Rashidkhani B. Dietary Inflammatory Index and Risk of Multiple Sclerosis in a Case-Control Study from Iran. Neuroepidemiology. 2016;47(1):26–31. PubMed PMID: . [PubMed: 27362443]
30. Shivappa N, Hebert JR, Rashvand S, Rashidkhani B, Hekmatdoost A. Inflammatory Potential of Diet and Risk of Ulcerative Colitis in a Case-Control Study from Iran. Nutr Cancer. 2016;68(3): 404–9. PubMed PMID: Pubmed Central PMCID: 4883059. [PubMed: 27030369]
31. Vahid F, Shivappa N, Karamati M, Jafari Naeini A, Hebert JR, Davoodi SH. Association between Dietary Inflammatory Index (DII) and Risk of Pre-Diabetes: A Case-Control Study. Applied Physiology, Nutrition, and Metabolism. 2016.
32. Vahid F, Shivappa N, Hekmatdoost A, Hebert JR, Davoodi SH, Sadeghi M. Association between Maternal Dietary Inflammatory Index (DII) and Abortion in Iranian Women and Validation of DII with Serum Concentration of Inflammatory Factors: Case-Control Study. Applied Physiology, Nutrition, and Metabolism. 2017.
33. Screening for Gestational Diabetes Mellitus. Journal of Obstetrics and Gynaecology Canada. 24(11):894–903.
34. Asghari G, Rezazadeh A, Hosseini-Esfahani F, Mehrabi Y, Mirmiran P, Azizi F. Reliability, comparative validity and stability of dietary patterns derived from an FFQ in the Tehran Lipid and Glucose Study. The British journal of nutrition. 2012 9 28;108(6):1109–17. PubMed PMID: . [PubMed: 22251608]
35. Ghaffarpour M, Houshiar-Rad A, Kianfar H. The manual for household measures, cooking yields factors and edible portion of food. Tehran: Nashre Olume Keshavarzy 1999.
36. Azar M, Sarkisian E. Food composition table of Iran. Tehran: National Nutrition and Food Research Institute, Shaheed Beheshti University, 1980.
37. Food and Nutrition Information Center US Department of Agriculture: Food composition table (FCT) http://www.nal.usda.gov/fnic/foodcomp. accessed on 20 September 20112009.

38. Kriska AM, Knowler WC, LaPorte RE, Drash AL, Wing RR, Blair SN, et al. Development of questionnaire to examine relationship of physical activity and diabetes in Pima Indians. Diabetes Care. 1990 4;13(4):401–11. PubMed PMID: . [PubMed: 2318100]
39. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ, et al. Compendium of physical activities: an update of activity codes and MET intensities. Medicine and science in sports and exercise. 2000 9;32(9 Suppl):S498–504. PubMed PMID: . [PubMed: 10993420]
40. He JR, Yuan MY, Chen NN, Lu JH, Hu CY, Mai WB, et al. Maternal dietary patterns and gestational diabetes mellitus: a large prospective cohort study in China. The British journal of nutrition. 2015 4 28;113(8):1292–300. PubMed PMID: . [PubMed: 25821944]
41. Shin D, Lee KW, Song WO. Dietary Patterns during Pregnancy Are Associated with Risk of Gestational Diabetes Mellitus. Nutrients. 2015 11 12;7(11):9369–82. PubMed PMID: Pubmed Central PMCID: 4663600. [PubMed: 26569302]
42. Schoenaker DA, Mishra GD, Callaway LK, Soedamah-Muthu SS. The Role of Energy, Nutrients, Foods, and Dietary Patterns in the Development of Gestational Diabetes Mellitus: A Systematic Review of Observational Studies. Diabetes Care. 2016 1;39(1):16–23. PubMed PMID: . [PubMed: 26696657]
43. Defago MD, Elorriaga N, Irazola VE, Rubinstein AL. Influence of food patterns on endothelial biomarkers: a systematic review. Journal of clinical hypertension. 2014 12;16(12):907–13. PubMed PMID: Pubmed Central PMCID: 4270900. [PubMed: 25376124]
44. Baena R, Salinas P. Diet and colorectal cancer. Maturitas. 2015 3;80(3):258–64. PubMed PMID: . [PubMed: 25619144]
45. Lopez-Garcia E, Schulze MB, Fung TT, Meigs JB, Rifai N, Manson JE, et al. Major dietary patterns are related to plasma concentrations of markers of inflammation and endothelial dysfunction. Am J Clin Nutr. 2004 10;80(4):1029–35. PubMed PMID: . [PubMed: 15447916]
46. Izadi V, Tehrani H, Haghighatdoost F, Dehghan A, Surkan PJ, Azadbakht L. Adherence to the DASH and Mediterranean diets is associated with decreased risk for gestational diabetes mellitus. Nutrition.32(10):1092–6.
47. Hebert JR, Ebbeling CB, Matthews CE, Hurley TG, Ma Y, Druker S, et al. Systematic errors in middle-aged women's estimates of energy intake: comparing three self-report measures to total energy expenditure from doubly labeled water. Ann Epidemiol. 2002 11;12(8):577–86. PubMed PMID: . [PubMed: 12495831]
48. Brogin Moreli J, Cirino Ruocco AM, Vernini JM, Rudge MV, Calderon IM. Interleukin 10 and tumor necrosis factor-alpha in pregnancy: aspects of interest in clinical obstetrics. ISRN obstetrics and gynecology. 2012;2012:230742 PubMed PMID: Pubmed Central PMCID: 3302063. [PubMed: 22462002]
49. Moller DE. Potential role of TNF-alpha in the pathogenesis of insulin resistance and type 2 diabetes. Trends in endocrinology and metabolism: TEM. 2000 8;11(6):212–7. PubMed PMID: . [PubMed: 10878750]
50. Lindsted KD, Kuzma JW. Long-term (24-year) recall reliability in cancer cases and controls using a 21-item food frequency questionnaire. Nutrition and Cancer. 1989 1989/1/01;12(2):135–49. [PubMed: 2710656]
51. Holmberg L, Ohlander EM, Byers T, Zack M, Wolk A, Bruce A, et al. A search for recall bias in a case-control study of diet and breast cancer. International journal of epidemiology. 1996 4;25(2): 235–44. PubMed PMID: . [PubMed: 9119547]
52. Hebert JR, Ma Y, Clemow L, Ockene IS, Saperia G, Stanek EJ, 3rd, et al. Gender differences in social desirability and social approval bias in dietary self-report. Am J Epidemiol. 1997 12 15;146(12):1046–55. PubMed PMID: . [PubMed: 9420529]
53. Ockene JK, Ockene IS, Quirk ME, Hebert JR, Saperia GM, Luippold RS, et al. Physician training for patient-centered nutrition counseling in a lipid intervention trial. Preventive medicine. 1995;24:563–70. [PubMed: 8610079]
54. Heitmann BL. Social desirability bias in dietary self-report may compromise the validity of dietary intake measures. Implications for diet disease relationships (letter). Int J Epidemiol. 1996;25(1): 223.

55. Hebert JR. Social desirability bias in dietary self-report may compromise the validity of dietary intake measures [letter; author's response]. Int J Epidemiol. 1996 4;25:223–5.

56. Fewell Z, Davey Smith G, Sterne JA. The impact of residual and unmeasured confounding in epidemiologic studies: a simulation study. Am J Epidemiol. 2007 9 15;166(6):646–55. PubMed PMID: . [PubMed: 17615092]

57. Jamshidi AR, Tehrani Banihashemi A, Roknsharifi S, Akhlaghi M, Salimzadeh A, Davatchi F. Estimating the prevalence and disease characteristics of rheumatoid arthritis in Tehran: A WHO - ILAR COPCORD Study (from Iran COPCORD study, Urban Study stage 1). Medical journal of the Islamic Republic of Iran. 2014;28:93 PubMed PMID: Pubmed Central PMCID: 4301204. [PubMed: 25664294]

58. Kharaghani R, Cheraghi Z, Okhovat Esfahani B, Mohammadian Z, Nooreldinc RS. Prevalence of Preeclampsia and Eclampsia in Iran. Archives of Iranian medicine. 2016 1;19(1):64–71. PubMed PMID: . [PubMed: 26702751]

Table 1.

Characteristics of patients in an Iranian GDM Case-Control Study, Tehran, Iran (n=388).

Characteristics	**Controls**	**Cases**	**[a]P-value**
	***N* =266**	***N* =121**	
Age, (years): mean ± sd	29.76±4.26	29.64±4.52	0.81
Gestational age, (months): mean± sd	31.19±3.53	29.39±4.74	<0.0001
Body Mass Index, (kg/m^2): mean ± sd	24.64±3.32	27.25±3.82	<0.0001
Dietary Inflammatory Index (DII) Score: mean ± sd*	−0.07±1.05	0.15±0.89	0.04
Physical activity, (MET, h/d); mean ± sd*	21.75±26.37	12.92±16.43	0.001
Family history of diabetes, n (%)	89 (33.46)	66 (54.55)	<0.0001
Supplement use a year before pregnancy, n (%)	84 (31.58)	45 (37.19)	0.28
Smoking exposure n (%)	24 (9.02)	8 (6.61)	0.42

** P-values were estimated using chi-square ($\chi 2$) statistics, independent t-test for the difference between case and control groups.

Table 2.

Participant characteristics by level of dietary inflammatory index (DII) among controls, Iranian GDM Case-Control Study, Tehran, Iran (n=266).

	Tertile 1 ≤ −1.32	**Tertile 2 −1.31 to −0.38**	**Tertile 3 >−0.38**	**P-Value**[a,b]
Age, (years): mean ± sd	30.10±4.13	29.54±4.10	29.62±4.56	0.46
Gestational age, (months): mean± sd	31.39±3.46	31.65±3.80	30.52±3.25	0.10
Body Mass Index (kg/m^2): mean ± sd	24.48±2.79	24.86±3.75	24.59±3.38	0.82
Physical activity, (MET, h/d)	21.11±22.39	20.35±23.20	23.81±32.56	0.50
Family history of diabetes, n (%)	27 (30.34)	31 (34.83)	31 (35.23)	0.74
Supplement use a year before pregnancy, n (%)	35 (39.33)	29 (32.58)	20 (22.73)	0.06
Smoking exposure n (%)	8 (8.99)	8 (8.99)	8 (9.09)	0.99

[a] Student t-test was used for continuous variables;

[b] Chi-square test was used for categorical variables

Table 3.

Odds ratios and 95% confidence intervals for the association between DII and GDM in an GDM case-control study, Tehran, Iran, (n=388).

	Dietary Inflammatory Index (Tertiles) OR (95% CI)			P_{trend}-value[a]	DII (Continuous)[b] OR (95% CI)	P-Value
DII	**Tertile 1 ≤ −1.32**	**Tertile 2 −1.31 to −0.38**	**Tertile 3 >−0.38**			
Cases/controls	40/89	31/89	51/88		122/266	
Age and energy-adjusted	1 (ref.)	1.16 (0.62, 2.18)	2.65 (1.39, 5.07)	0.007	1.25 (1.01, 1.56)	0.04
Multivariable-adjusted [c]	1 (ref.)	0.82 (0.42, 1.57)	2.10 (1.02, 4.34)	0.03	1.20 (0.94, 1.54)	0.15

[a] P-value for trend derived using the median approach.

[b] One unit increase corresponding to ≈34% of its range in the current study.

[c] Adjusted for age, energy, gestational age, exercise, BMI, history of diabetes, history of exposure to smoking and history of supplemental intake.