Published in final edited form as:

Ann Epidemiol. 2017 June ; 27(6): 384–390.e1. doi:10.1016/j.annepidem.2017.05.006.

Maternal Intake of Fried Foods and Risk of Gestational Diabetes Mellitus

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Abstract

Purpose—We examined the relationship of maternal periconceptional (i.e., before conception and early pregnancy) intake of fried foods with GDM risk.

Methods—In a prospective birth cohort in Seattle and Tacoma, Washington State, USA, we assessed maternal periconceptional fried food intake using a food frequency questionnaire among 3,414 participants. We used multivariable generalized linear regression models to derive estimates of relative risks (RRs) (and 95% confidence intervals, 95% CI) of GDM in relation to the intake of different types of fried foods (i.e., fried fish, fried chicken, fried potatoes, chips and doughnuts).

Results—A total of 169 GDM incident cases were identified in this cohort (4.96%). Compared with no fried fish intake, fried fish intake >1 servings/month was associated with 68% higher GDM risk [adjusted RR and 95% CI; 1.68 (1.16, 2.45); $P_{\text{trend}}=0.019$]. After adjusting for confounders, the RRs (95% CI) of GDM relative to fried chicken intake were 1.0, 1.44 (0.98, 2.09) and 1.81 (1.22, 2.70) for none, 1 and >1 servings/month intake of fried chicken, respectively ($P_{\text{trend}}=0.002$). Dietary intake of fried potatoes, snack chips or doughnuts was not significantly associated with higher GDM risk. Limitations of our study include the lack of information about frying methods and the intake of fried foods at home and away from home.

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Author Contributions

MAW designed the study and obtained funding for the study. COY, QC and MAW analyzed data and drafted the manuscript. COY, MAW, BG, QC, AC, DE and WB reviewed and edited the manuscript. COY, QC, and MAW had full access to all the data in the study and take responsibility for the integrity of the data, the accuracy of data analysis, and the decision to submit for publication.

Conclusions—Regular intake of fried fish and fried chicken are associated with elevated GDM risk.

Keywords

gestational diabetes; fried fish; fried chicken; fried potatoes; pregnant women

Introduction

Fast food is a major part of the US diet [1,2]. Approximately, one in four Americans visit a fast-food restaurant daily [1,3]. Fast food restaurants commonly include fried foods in their menus such as french fries, fried chicken, fried fish, snack chips, and doughnuts, among others [3,4]. Frying is a complex cooking process that modifies the fatty acid composition of foods; and the oils used for frying increase the energy density and reduce foods' water content [5]. Frying may also improve food palatability by making the food crunchy and aromatic, which may in turn lead to excess consumption [6]. Apart from the high-energy content, frequent consumption of fried food is considered an indicator of unhealthy lifestyle and dietary habit [7,8]. Fried food consumption has been shown to be associated with increased risks of type 2 diabetes [9], high blood pressure [10], and metabolic syndrome [11], as well as increased general and central obesity [6,10].

Gestational diabetes mellitus (GDM) is a common complication of pregnancy characterized by glucose intolerance with onset or first recognition during pregnancy [12]. Women with a history of GDM have more than a 7-fold increased risk of developing type 2 diabetes within 5 to 10 years after delivery [13]. Additionally, children born to mothers with GDM are more likely to develop type 2 diabetes later in life [14]. Risk factors for GDM include higher parity, advanced maternal age, family history of diabetes mellitus, nonwhite race/ethnicity, sleep disordered breathing, as well as pre-pregnancy overweight and obesity [15,16,17,18]. Modifiable risk factors such as diet before or during early pregnancy may serve as a target for preventing GDM. Specifically, a recent study reported that frequent fried food consumption before pregnancy was significantly associated with a greater risk of incident GDM [7]. However, few epidemiological studies have been focused on maternal outcomes associated with fried food consumption during pregnancy. Importantly, available literature, though sparse, do suggest associations of maternal habitual consumption of fried foods with adverse perinatal outcomes including infant low birth weight and preterm birth [19,20]. For example, in the European Prospective Mother-Child Study (NewGeneris), Pedersen and colleagues noted that maternal consumption of fried foods, such as fried potatoes, which are rich in acrylamide, was associated with cord blood acrylamide levels and reduced infant birth weight. Notably, a 1-unit increase in the acrylamide food score was associated with a 16 g decrease in birth weight (95% CI: -33, 1; $p=0.066$) [19]. Additionally, Martin and colleagues recently reported that maternal early pregnancy dietary pattern that includes fried fish and fried chicken consumption was associated with higher risk of preterm birth (OR=1.53; 95% CI: 1.02, 2.30) [20]. Given mounting epidemiologic evidence from studies of men and non-pregnant women supporting associations of fried food intake with higher risk of type 2 diabetes [8,9], and given these two recent studies conducted among pregnant women, we sought to study the extent to which, if at all, higher maternal fried food intake in

the periconceptional period is associated with increased GDM risk later in the pregnancy. We hypothesized that periconceptional fried food intake is associated with an increased risk of developing GDM during the pregnancy.

RESEARCH DESIGN AND METHODS

The Omega study is a prospective pregnancy cohort designed to examine dietary risk factors of adverse pregnancy outcomes. Participants were women receiving prenatal care at clinics affiliated with the Swedish Medical Center and Tacoma General Hospital in Seattle and Tacoma, WA [21,22]. Eligible women were those who began prenatal care before 20 weeks of gestation, were able to speak and read English, were 18 years or older, and planned on delivering at either of the two hospitals. During early pregnancy (mean gestational age of 15 weeks), participants were asked to complete an interviewer-administered questionnaire. Participants also completed a 121-item previously validated semi-quantitative Food Frequency Questionnaire (FFQ) [23] to assess dietary habits in the past three months. Information on pregnancy outcomes was abstracted from medical records. Participants provided written informed consent and all procedures and study protocols were approved by the institutional review boards of the study hospitals.

Analytical Population

The analytical study population was derived from participants enrolled in the Omega Study between 1996 and 2008. During this period, 5,825 eligible women were approached and 4,602 (79%) agreed to participate. Women found to have physician-diagnosed pre-gestational diabetes (i.e., type 1, type 2 diabetes) and previous history of GDM (n=48), multi-fetal pregnancies (n=136), pregnancies lasting <20 weeks of gestation (n=45), and those with iron deficiency anemia (n=156) were excluded. We also excluded women who did not complete the FFQ (n=566), those who reported extreme levels of daily total energy intake [24] (<500 calories/day [n=27] or >3,500 calories/day [n=52]) and 158 women who moved out of the study area. A cohort of 3,414 women remained for analysis. The demographic and lifestyle characteristics of those who were included and excluded were similar (data not shown).

Data Collection

From structured questionnaire and medical records, we obtained covariate information including maternal age, educational attainment, height, pre-pregnancy weight, reproductive and medical histories, and medical histories of first-degree family members. We also collected information on maternal smoking during pregnancy. Self-reported pre-pregnancy weight and height were used to calculate pre-pregnancy body mass index (BMI: weight in kilograms divided by height in meters squared). Participants completed a self-administered, validated, and semi-quantitative food frequency questionnaire (FFQ) [23] at a mean gestational age of 15 weeks to assess periconceptional (i.e., three months before conception and up to three months post conception) diet. The WHI FFQ allows for assessment of intake, portion size, and food additives. Participants were provided clear instructions including photos of portion sizes for some of the items. The WHI FFQ has documented reliability of accurately recording intake over an extended period of observation [23]. Food composition

values were obtained from the University of Minnesota Nutrition Coding Center nutrient database (Nutrition Coordinating Center, Minneapolis, MN).

We selected five fried food items measured in our study: fried potatoes, fried chicken, fried fish, doughnuts and snack chips. Fried fish was described as “fried fish, fish sandwich and fried shellfish (shrimp and oysters).” Fried potatoes included “french fries and fried potatoes.” Fried chicken was listed as “fried chicken, including nuggets and tenders”. The doughnut category included “doughnuts, cakes, pies and pastries,” and snack chips “potato chips, corn chips, cheese crackers and tortilla chips.” We assumed that all these categories comprised primarily of deep-fried foods. There were some exceptions, such as listing for “cheese crackers”; however, the contribution of these single items within its general category is likely to be small [23,25]. Maternal medical records were reviewed to collect detailed clinical information. As part of the routine antenatal follow-up of all women at participating clinics, a 50-g, 1-hr oral glucose challenge test was administered between 24 and 28 weeks of gestation to screen for GDM. Women who failed the screening test [glucose 7.8 mmol/L (140 mg/dL)] completed a diagnostic 100-g, 3-hr oral glucose tolerance test within two weeks of the screening test. According to ADA 2004 guidelines, women were diagnosed with GDM if two or more 100-g, 3-hr oral glucose tolerance test levels exceeded the following criteria: fasting 5.3 mmol/L (95 mg/dL); 1-hr 10.0 mmol/L (180 mg/dL); 2-hr 8.6 mmol/L (155 mg/dL); 3-hr 7.8 mmol/L (140 mg/dL) [26].

Statistical Analysis

We examined frequency distributions of maternal socio-demographic, reproductive, medical and dietary characteristics according to categories of total fried food and specific fried foods. We grouped total fried food consumption into three categories (<1 serving/week; 1–3 servings/week and >3 servings/week). For individual fried foods, we defined three frequency categories as follows: none; 1 serving/month; >1 servings/month. We then assessed GDM risk in relation to total periconceptional fried food intake and also relative to intake of individual fried food items (i.e., fried fish, fried chicken, fried potatoes, snack chips and doughnuts). To estimate relative risks (RRs) and 95% confidence intervals (95% CIs) for GDM, we fitted generalized linear models with a log-link function, Poisson family (a “log Poisson” regression model), and robust standard errors. This model allows estimation of RRs for prospective studies with binary outcome data [27]. To assess confounding, we entered covariates into each model one at a time and compared adjusted and unadjusted RRs. Final models included covariates that altered unadjusted RRs by at least 10% and those that were identified *a priori* as potential confounders. The following covariates were *a priori* considered as potential confounders based on their hypothesized role in the relationships between GDM and fried food consumption: maternal age (continuous), race/ethnicity (Non-Hispanic White, African American, Asian, Other), multiparity (yes/no), gravidity, post high school education (yes/no), smoking during pregnancy (never smoker, former or current smoker), no exercise during pregnancy (yes/no), prenatal vitamin intake (yes/no), pre-pregnancy overweight status as defined using body mass index (BMI) (<25, 25 kg/m²) and dietary covariates. Two multivariable models were constructed. Model 1 included maternal age, race/ethnicity, educational attainment, cigarette smoking, physical activity, family history of diabetes, total energy intake, intake of alcohol, coffee, sugar-sweetened beverages,

red and processed meat, dietary magnesium and vitamin D. Model 2, included all the previously listed covariates and pre-pregnancy overweight status. In multivariable analyses, we evaluated linear trends for GDM risk by treating total or specific fried food intake as continuous variables after assigning a score to each category: none, 1 serving/month, >1 servings/month. All analyses were performed using Stata 12.0 statistical software (StataCorp, College Station, Texas, USA). All reported *p*-values are two-sided and deemed significant at $\alpha=0.05$.

Results

Selected sociodemographic and lifestyle characteristics of the study cohort are summarized in Table 1. The mean age of study participants was 33 years and mean gestational age at delivery was 39 weeks. The majority of women were married (92.5%), White (86.8%), never-smokers (73.62%), and stayed physically active during pregnancy (88.5%). Women who reported higher consumption of total fried foods were younger, less likely to be nulliparous and White, and more likely to be current smokers (Table 1). Additionally, they tended to be less educated and more likely to be physically inactive during pregnancy. Mean pre-pregnancy BMI increased across categories of total fried food consumption. Women with higher total fried food consumption were more likely to consume more red and processed meats, saturated fat and drink more sugar-sweetened beverages. We observed lower intake of fruit and vegetables and total protein with increasing total fried foods intake (Table 1). Among the various types of fried foods, fried potatoes were consumed more frequently (more than one serving per month in 68.3% of participants) followed by snack chips and doughnuts (more than one serving per month in 67.8% and 42.3%, respectively) (Table 1). Consumption of fried fish was reported more frequently (>1 servings/month) than fried chicken (14.5 % and 12.1 %, respectively) (Table 1).

A total of 169 incident GDM cases were identified in this cohort (4.96%). Total fried food intake (i.e., fried fish, fried chicken, fried potatoes, doughnuts and snack chips combined) was not statistically significantly associated with higher GDM risk. After adjustment, the RR of GDM were 1.0, 0.92 (95% CI 0.61, 1.38) and 1.26 (95% CI 0.79, 2.01) for <1 serving/week, 1–3 servings/week, >3 servings/week (*P* for trend=0.30) (Table 2). When we analyzed specific fried foods, the RR of GDM among women who consumed more than one serving per month of fried fish was 1.73 (95% CI 1.19, 2.52) compared with those who reported no consumption of fried fish (*P* for trend=0.013) after adjustment for age, race/ethnicity, educational attainment, smoking status, physical activity, family history of diabetes, alcohol, coffee, sugar-sweetened beverages, red and processed meat, total energy intake, and other dietary covariates (Table 3). Similarly, the adjusted RR of GDM among women who consumed fried chicken > 1 servings/month was 2.06 (95% CI 1.39, 3.05) compared with those who reported no fried chicken consumption (*P* for trend <0.001) (Table 3). These associations remained significant after additional adjustment for pre-pregnancy overweight status (Table 3). We assessed GDM risk in those women who consumed more than one serving per week of fried fish or fried chicken as compared with no intake of either type of fried food. The intake of fried fish [(RR and 95% CI: 2.66 (1.45, 4.87)] and fried chicken [(RR and 95% CI: 2.42 (1.14, 5.15)] was associated with increased GDM risk (Supplemental Table 1). Maternal consumption of fried potatoes, snack chips, and doughnuts did not appear

to be associated with statistically significant increases in GDM risk (Table 3, Supplemental Table 1). We conducted a sensitivity analysis including vegetables and nuts intake in the logistic models, and these food items were not confounders in the association GDM risk and fried food intake (data not shown).

Discussion

In this large pregnancy cohort, we found that increasing periconceptional (i.e., pre-pregnancy and early pregnancy) fried fish and fried chicken consumption is associated with increased risk of GDM, even after adjustment for other GDM risk factors such as maternal age, pre-pregnancy overweight status, family history of diabetes, physical activity during pregnancy, and other dietary characteristics. However, total fried food consumption was not significantly associated with increased GDM risk.

To our knowledge, this is the first study that evaluated relationships between the types of fried food consumption in the periconceptional period and incident GDM risk. In a previous study examining the association of pre-pregnancy fried food consumption and risk of GDM, no information on the types of individual fried foods was available [7]. Among men and non-pregnant women, findings from studies of fish intake and type 2 diabetes had conflicting results that vary by geographic areas [28], type of fish consumption [29,30] and cooking method [30]. Our results are consistent with some, but not all, previous studies assessing type 2 diabetes and fried fish intake in men and non-pregnant women. For example, in a population-based cohort of Swedish men, fried fish intake (6 servings/month vs. 1 servings/month) was significantly associated with higher risk of type 2 diabetes, (hazard ratio=1.14 ; 95% CI: 1.03, 1.31) [31]. In another study, African American women who consumed fried fish 1 to 3 times per month had 21 % higher risk of type 2 diabetes compared to women that never consumed fried fish (IRR: 1.21; 95% CI: 1.07, 1.37) [8]. However, Patel and coworkers (2009), in the European Prospective Investigation of Cancer [EPIC]-Norfolk, found no increase in risk of type 2 diabetes among men and women who consumed fried fish [(1 vs. <1 portion/week) (OR=0.91; 95% CI: 0.75, 1.10)] after adjustment for confounders [30].

In our study cohort, fried chicken intake was associated with increased GDM risk even after we controlled for a number of confounders. Consistent with our results, previous studies have documented associations of fried chicken consumption with higher risk of type 2 diabetes in African American women [(2 times per week vs. none) (IRR=1.68; 95%CI: 1.36, 2.08)] [8]. Furthermore, Native Americans diagnosed with diabetes were more likely to report higher intake of fried chicken and fried fish in the Inter-Tribal Heart Project –a cross-sectional study [32].

Although biological mechanisms underlying the observed association of fried food consumption with GDM risk are unknown, some authors have hypothesized that increased formation of trans-fatty acids [33] and advanced glycoxidation end products (AGEs) during the frying process [34] may play a role [35,36]. This thesis is supported by evidence from experimental studies showing reduced insulin sensitivity with increased intake of trans fatty acids [37]. Furthermore, investigators have shown that AGEs contribute to cellular oxidative

damage and chronic systemic inflammation [38,39] which have been implicated in insulin resistance, pancreatic cell damage and diabetes [36,40].

Potential benefits of fish consumption in relation to insulin resistance and type 2 diabetes have been suggested to be mediated through long-chain n-3 fatty acids, which have inhibitory effects on inflammatory pathways and activation of peroxisome proliferator-activated receptors (PPARs) [41]. However, frying may lead to degradation of long-chain n-3 fatty acids and increase in other fatty acids including trans-fatty acids [42]. Of note, fried fish is usually lean fish (i.e. bass, cod, flounder), and contains lower amounts of n-3 fatty acids than oily fish (i.e. salmon, trout, and herring) [43]. Additionally, environmental contaminants such as polychlorinated biphenyls (PCBs) and methylmercury (MeHg) present in fish might increase the risk of diabetes [44, 45].

The present study has several strengths and limitations. We used a large well-characterized cohort of pregnant women. Additionally, structured interviews, medical record abstractions, and the validated semi-quantitative FFQ provide high-quality data to evaluate the relationships and adjust for potential confounding factors. Estimating the risk of GDM in relation to specific types of fried foods commonly consumed during pregnancy is a key strength of our study, a consequence of the detailed dietary information we collected. Limitations of our study include the lack of information about serving size or frying methods (e.g., deep or pan frying; fresh or reused oil; duration and temperature, types of frying oil [i.e. olive, corn, vegetable, etc.]), and the intake of fried foods at home and away from home. Even though we adjusted our analysis for unhealthy lifestyle and dietary habits, we could not exclude the possibility of residual confounding as in all observational studies.

Conclusions

Our findings suggest that higher levels of maternal periconceptional fried fish and fried chicken, are associated with increased GDM risk. Additional studies are needed to confirm these findings and elucidate whether the association we observed between fried food consumption and risk of GDM are attributable to fried food intake away from home or unhealthy lifestyles.

Regardless, from a clinical and public health perspective, we have identified a risk factor for GDM that may be readily modifiable by lifestyle or cooking choices that lead to the consumption of less fried foods, especially fried chicken and fried fish.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by awards (R01HD-32562 and K01HL103174) from the National Institutes of Health (NIH). Dr. Osorio-Yáñez has been financially supported by Consejo Nacional de Ciencia y Tecnología (CONACYT-Mexico; 238130). The funders had no further role in study design; in the collection, analysis and interpretation of data; in drafting of the report; and in the decision to submit the paper for publication. The authors thank the staff of the Center for Perinatal Studies for their skillful technical assistance. The authors are indebted to the staff of the Center for Perinatal Studies for their expert technical assistance.

References

1. Bowman SA, Vinyard BT. Fast food consumption of U.S. adults: Impact on energy and nutrient intakes and overweight status. *J Am Col Nutr.* 2004; 23(2):163–8.
2. Paeratakul S, Ferdinand DP, Champagne CM, Ryan DH, Bray GA. Fast-food consumption among US adults and children: dietary and nutrient intake profile. *J Am Diet Assoc.* 2003; 103(10):1332–8. [PubMed: 14520253]
3. Freeman, A. Fast Food: Oppression through Poor Nutrition. *California Law Review*; 2007. p. 95 <http://scholarship.law.berkeley.edu/californialawreview/vol95/iss6/8> [accessed 11.05.16]
4. Elbe B, Gyamfi J, Kersh R. Child and adolescent fast-food choice and the influence of calorie labeling: a natural experiment. *Int J Obes (Lond).* 2011; 35(4):493–500. [PubMed: 21326209]
5. Guallar-Castillon P, Rodriguez-Artalejo F, Lopez-Garcia E, León Muñoz LM, Amiano P, Ardanaz E, et al. Consumption of fried foods and risk of coronary heart disease: Spanish cohort of the European Prospective Investigation into Cancer and Nutrition study. *BMJ.* 2012; 344:e363. [PubMed: 22275385]
6. Guallar-Castillon P, Rodriguez-Artalejo F, Fornes NS, Banegas JR, Etxezarreta PA, Ardanaz E, et al. Intake of fried foods is associated with obesity in the cohort of Spanish adults from the European Prospective Investigation into Cancer and Nutrition. *Am J Clin Nutr.* 2007; 86(1):198–205. [PubMed: 17616781]
7. Bao W, Tobias DK, Olsen SF, Zhang C. Pre-pregnancy fried food consumption and the risk of gestational diabetes mellitus: a prospective cohort study. *Diabetologia.* 2014; 57(12):2485–91. [PubMed: 25303998]
8. Krishnan S, Coogan PF, Boggs DA, Rosenberg L, Palmer JR. Consumption of restaurant foods and incidence of type 2 diabetes in African American women. *Am J Clin Nutr.* 2010; 91(2):465–71. [PubMed: 20016014]
9. Cahill LE, Pan A, Chiuve SE, Sun Q, Willet WC, Hu FB, Rimm EB. Fried-food consumption and risk of type 2 diabetes and coronary artery disease: a prospective study in 2 cohorts of US women and men. *Am J Clin Nutr.* 2014; 100(2):667–75. [PubMed: 24944061]
10. Sayon-Orea C, Martinez-Gonzalez MA, Gea A, Flores-Gomez E, Basterra-Gortari FJ, Bes-Rastrollo M. Consumption of fried foods and risk of metabolic syndrome: the SUN cohort study. *Clin Nutr.* 2014; 33(3):545–9. [PubMed: 23954218]
11. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. *Circulation.* 2008; 117(6):754–61. [PubMed: 18212291]
12. American Diabetes Association, ADA. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care.* 2013; 36(Suppl 1):S67–S74. [PubMed: 23264425]
13. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. *Lancet.* 2009; 373(9677):1773–9. [PubMed: 19465232]
14. Dabelea D, Mayer-Davis EJ, Lamichhane AP, D'Agostino RB Jr, Liese AD, Vehik KS, et al. Association of intrauterine exposure to maternal diabetes and obesity with type 2 diabetes in youth: the SEARCH Case-Control Study. *Diabetes Care.* 2008; 31(7):1422–26. [PubMed: 18375420]
15. King H. Epidemiology of glucose intolerance and gestational diabetes in women of childbearing age. *Diabetes Care.* 1998; 21(Suppl.2):B9–13. [PubMed: 9704221]
16. Solomon CG, Willett WC, Carey VJ, Rich-Edwards J, Hunter DJ, Colditz GA, et al. A prospective study of pregravid determinants of gestational diabetes mellitus. *JAMA.* 1997; 278(13):1078–83. [PubMed: 9315766]
17. Luque-Fernandez MA, Bain PA, Gelaye B, Redline S, Williams MA. Sleep-disordered breathing and gestational diabetes mellitus: a meta-analysis of 9,795 participants enrolled in epidemiological observational studies. *Diabetes Care.* 2013; 36(10):3353–60. [PubMed: 24065843]
18. Thompson ML, Ananth CV, Jaddoe VW, Miller RS, Williams MA. The association of maternal adult trajectory with preeclampsia and gestational diabetes mellitus. *Paediatr Perinat Epidemiol.* 2014; 28(4):287–96. [PubMed: 24842329]

19. Pedersen M, von Stedingk H, Botsivali M, Agramunt S, Alexander J, Brunborg G, et al. Birth weight, head circumference, and prenatal exposure to acrylamide from maternal diet: the European prospective mother-child study (New Generis). *Environ Health Perspect.* 2012; 120(12):1739–45. [PubMed: 23092936]
20. Martin CL, Sotres-Alvarez D, Siega-Ruiz AM. Maternal Dietary Patterns during the Second Trimester Are Associated with Preterm Birth. *J Nutr.* 2015; 145(8):1857–64. [PubMed: 26084362]
21. Enquobahrie DA, Williams MA, Qiu C, Luthy DA. Early pregnancy lipid concentrations and the risk of gestational diabetes mellitus. *Diabetes Res Clin Pract.* 2005; 70(2):134–42. [PubMed: 16188575]
22. Qiu C, Zhang C, Gelaye B, Enquobahrie DA, Frederick IO, Williams MA. Gestational diabetes mellitus in relation to maternal dietary heme iron and nonheme iron intake. *Diabetes Care.* 2011; 34(7):1564–9. [PubMed: 21709295]
23. Patterson RE, Kristal AR, Tinker LF, Carter RA, Bolton MP, Agurs-Collins T. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. *Ann Epidemiol.* 1999; 9(3):178–87. [PubMed: 10192650]
24. Willet, W., Sampson, L. *Nutritional Epidemiology* Third Edition. New York, NY: Oxford University Press; 2013.
25. Stott-Miller M, Neuhaus ML, Stanford JL. Consumption of deep-fried foods and risk of prostate cancer. *Prostate.* 2013; 73(9):960–9. [PubMed: 23335051]
26. American Diabetes Association, ADA. Gestational Diabetes Mellitus. *Diabetes Care.* 2004; 27(Suppl. 1):S88–S90. [PubMed: 14693936]
27. Zou G. A modified poisson regression approach to prospective studies with binary data. *Am J Epidemiol.* 2004; 159(7):702–6. [PubMed: 15033648]
28. Yanai H, Hamasaki H, Katsuyama H, Adachi H, Moriyama S, Sako A. Effects of intake of fish or fish oils on the development of diabetes. *J Clin Med Res.* 2015; 7(1):8–12. [PubMed: 25368695]
29. Zhang M, Picard-Deland E, Marette A. Fish and marine omega-3-polyunsaturated Fatty Acid consumption and incidence of type 2 diabetes: a systematic review and meta-analysis. *Int J Endocrinol.* 2013; 2013:501015. [PubMed: 24089611]
30. Patel PS, Sharp SJ, Luben RN, Khaw KT, Bingham SA, Wareham NJ, et al. Association between type of dietary fish and seafood intake and the risk of incident type 2 diabetes: the European prospective investigation of cancer (EPIC)-Norfolk cohort study. *Diabetes Care.* 2009; 32(10):1857–63. [PubMed: 19592633]
31. Wallin A, Di Giuseppe D, Orsini N, Akesson A, Forouhi NG, Wolk A. Fish consumption and frying of fish in relation to type 2 diabetes incidence: a prospective cohort study of Swedish men. *Eur J Nutr.* 2015; doi: 10.1007/s00394-015-1132-6
32. Archer SL, Greenlund KJ, Valdez R, Casper ML, Rith-Najarian S, Croft JB. Differences in food habits and cardiovascular disease risk factors among Native Americans with and without diabetes: the Inter-Tribal Health Project. *Public Health Nutr.* 2004; 7(8):1025–32. [PubMed: 15548340]
33. Li A, Ha Y, Wang F, Li W, Li Q. Determination of thermally induced trans-fatty acids in soybean oil by attenuated total reflectance fourier transform infrared spectroscopy and gas chromatography analysis. *J Agric Food Chem.* 2012; 60(42):10709–13. [PubMed: 23025355]
34. Goldberg T, Cai W, Peppas M, Dardaine V, Baliga BS, Uribarri J, et al. Advanced glycoxidation end products in commonly consumed foods. *J Am Diet Assoc.* 2004; 104(8):1287–91. [PubMed: 15281050]
35. Odegaard AO, Pereira MA. Trans fatty acids, insulin resistance, and type 2 diabetes. *Nutr Rev.* 2006; 64(8):364–72. [PubMed: 16958313]
36. Cai W, Ramdas M, Zhu L, Chen X, Striker GE, Vlassara H. Oral advanced glycation endproducts (AGEs) promote insulin resistance and diabetes by depleting the antioxidant defenses AGE receptor-1 and sirtuin 1. *Proc Natl Acad Sci USA.* 2012; 109(39):15888–93. [PubMed: 22908267]
37. Zhao X, Shen C, Zhu H, Wang C, Liu X, Sun X, et al. Trans-Fatty Acids Aggravate Obesity, Insulin Resistance and Hepatic Steatosis in C57BL/6, Possibly by Suppressing the IRS1 Dependent Pathway. *Molecules.* 2016; 21(6):E705. doi: 10.3390/molecules21060705 [PubMed: 27248994]

38. Boyer F, Vidot JB, Dubourg AG, Rondeau P, Essop MF, Bourdon E. Oxidative stress and adipocyte biology: focus on the role of AGEs. *Oxid Med Cell Longev*. 2015; 2015:534873.doi: 10.1155/2015/534873 [PubMed: 25878764]
39. Adamopoulos C, Mihailidou C, Grivaki C, Papavassiliou KA, Kiaris H, Piperi C, et al. Systemic effects of AGEs in ER stress induction in vivo. *Glycoconj J*. 2016; 33(4):537–44. [PubMed: 27236787]
40. Coughlan MT, Yap FY, Tong DC, Andrikopoulos S, Gasser A, Thallas-Bonke V, et al. Advanced glycation end products are direct modulators of β -cell function. *Diabetes*. 2011; 60(10):2523–32. [PubMed: 21911745]
41. Deckelbaum RJ, Worgall TS, Seo T. n-3 fatty acids and gene expression. *Am J Clin Nutr*. 2006; 83(Suppl.6):1520S–1525S. [PubMed: 16841862]
42. Wagner KH, Elmadfa I. Chemical and biological modulations of food due to the frying process. *Int J Vitam Nutr Res*. 2012; 82(3):163–7. [PubMed: 23258396]
43. Kromhout D, Yasuda S, Geleijnse JM, Shimokawa H. Fish and omega-3 fatty acids in cardiovascular disease: do they really work? *Eur Heart J*. 2012; 33(4):436–43. [PubMed: 21933782]
44. Wu H, Bertrand KA, Choi AL, Hu FB, Laden F, Grandjean P, et al. Persistent organic pollutants and type 2 diabetes: a prospective analysis in the nurses' health study and meta-analysis. *Environ Health Perspect*. 2013; 121(2):153–61. [PubMed: 23131992]
45. He K, Xun P, Liu K, Morris S, Reis J, Guallar E. Mercury exposure in young adulthood and incidence of diabetes later in life: the CARDIA Trace Element Study. *Diabetes Care*. 2013; 36(6): 1584–9. [PubMed: 23423697]

Table 1

Participant characteristics according to reported fried food consumption in early pregnancy, Seattle and Tacoma, WA, Omega Cohort Study

	Total Fried Food				Fried Fish				Fried Chicken				Fried Potatoes				Stuck Chips				Doughnuts			
	<1 servings/ week	1-3 servings/ week	>3 servings/ week	P for trend	None	1 serving/ month	>1 servings/ month	P for trend	None	1 serving/ month	>1 servings/ month	P for trend	None	1 serving/ month	>1 servings/ month	P Value	None	1 serving/ month	>1 servings/ month	P Value	None	1 serving/ month	>1 servings/ month	P Value
Subjects																								
N	793	1572	1049		2,212	708	494		2,348	653	413		438	646	2,330		727	374	2,313		1,288	682	1,444	
%	23.2	46.1	30.7		64.8	20.7	14.47		68.78	19.13	12.10		12.83	18.92	68.25		21.3	11.0	67.8		37.7	20.0	42.3	
Maternal Age ^{a)}	33.4±4.0	32.9±4.2	32.2±4.6	<0.001	32.9±4.3	32.8±4.5	32.5±4.3	0.085	33.1±4.13	32.5±4.42	31.8±5.0	<0.001	33.7±4.41	33.3±4.22	32.5±4.3	<0.001	33.2±4.11	33.0±4.44	32.7±4.36	0.001	33.0±4.3	32.9±4.2	32.6±4.4	0.042
Pre-pregnancy BMI (kg/m ²) ²	22.8±4.1	23.5±4.7	23.9±4.8	<0.001	23.3±4.4	23.6±4.7	24.2±5.1	<0.001	23.1±4.23	23.8±4.74	25.0±5.92	<0.001	22.9±4.5	22.9±4.0	23.7±4.8	<0.001	23.0±4.3	23.6±4.61	23.6±4.70	0.014	23.1±4.3	23.4±4.3	23.8±4.9	<0.001
Gestational Age at delivery (w)	39.0±1.8	38.9±2.0	38.8±2.0	0.112	38.9±1.96	38.9±1.90	38.8±1.96	0.478	38.9±2.0	38.8±1.60	38.6±2.19	0.005	39.0±1.78	38.7±2.29	38.9±1.88	0.795	38.9±1.72	39.0±1.89	38.9±2.03	0.467	38.9±1.91	38.8±2.15	38.8±1.90	0.210
White (%)	88.9	86.8	85.1	0.013	89.7	84.9	76.5	<0.001	91.23	80.9	70.7	<0.001	88.81	88.24	86.0	0.062	82.94	88.5	87.7	0.009	88.7	86.7	85.11	0.090
Post - High School Education (%)	98.1	97.3	95.7	0.010	96.8	97.1	97.5	0.719	97.7	97.1	92.42	<0.001	97.9	97.31	96.7	0.378	97.9	98.6	96.4	0.021	97.6	97.7	96.01	0.025
Married (%)	92.2	93.9	90.6	0.006	93.04	92.5	89.9	0.055	93.5	92.04	87.41	<0.001	92.7	93.34	92.2	0.693	92.2	92.0	92.7	0.844	92.24	94.0	92.0	0.236
Nulliparous (%)	66.1	61.4	61.0	0.047	62.84	59.8	64.0	0.244	65.7	56.51	52.8	<0.001	71.23	61.8	60.9	<0.001	62.60	63.90	62.04	0.780	65.7	61.88	59.63	0.005
Smoked during pregnancy (%)	3.53	5.12	7.67	0.002	4.66	7.31	6.95	0.039	4.78	5.88	9.39	0.001	4.09	3.33	6.42	0.014	4.86	5.03	5.80	0.504	4.41	3.64	7.44	<0.001
Physically activity (%)	90.4	89.2	86.1	0.011	88.3	89.53	88.2	0.652	89.4	87.1	85.9	0.069	89.7	89.13	88.12	0.575	89.55	89.9	87.96	0.370	89.11	89.2	87.7	0.427
Family history of diabetes (%)	10.8	13.7	15.3	0.022	12.97	12.57	17.41	0.024	11.88	18.22	15.50	<0.001	11.2	11.61	14.51	0.050	12.93	14.44	13.58	0.782	12.66	14.1	14.1	0.507
Total energy intake (kcal)	1484±505	1675±528	1980±548	<0.001	1678±541	1776±577	1856±593	<0.001	1706±545	1704±573	1856±612	<0.001	1557±512	1617±529	1785±587	<0.001	1506±573	1657±543	1784±549	<0.001	1590±543	1677±584	1874±564	<0.001
% calories from total fat	27.5±6.80	30.5±5.53	31.2±5.4	<0.001	29.8±6.3	31.6±5.7	32.8±5.56	<0.001	27.0±2.6	60.2±23.5	68.1±25.7	<0.001	47.6±19.5	52.7±20.8	62.8±23.7	<0.001	49.4±22.5	54.8±20.7	62.6±23.2	<0.001	51.6±21.8	57.0±20.4	66.4±23.9	<0.001
% calories from saturated fat	10.0±3.14	11.2±2.7	12.0±2.6	<0.001	10.9±2.94	11.5±2.79	11.6±2.64	<0.001	10.9±2.94	11.51±2.8	11.7±2.47	<0.001	10.0±3.06	10.7±2.95	11.4±2.8	<0.001	10.3±3.32	10.9±2.79	11.4±2.75	<0.001	10.6±3.09	11.1±2.70	11.7±2.67	<0.001
% calories from protein	17.8±3.20	17.5±2.70	16.7±2.5	<0.001	17.5±2.95	17.3±2.40	17.4±2.53	0.203	17.3±2.8	17.32±2.7	17.40±2.6	0.843	17.91±3.3	17.51±2.87	17.2±2.65	<0.001	17.8±3.16	17.84±2.7	17.1±2.65	<0.001	17.3±3.02	17.3±2.71	17.0±2.56	<0.001
% calories from carbohydrates	56.2±8.5	53.1±6.90	51.0±5.36	<0.001	54.14±7.5	52.1±6.62	50.6±6.8	<0.001	54.0±7.39	52.0±7.27	50.5±6.6	<0.001	56.0±8.60	54.6±7.55	52.3±6.90	<0.001	55.3±8.61	53.4±7.40	52.5±6.81	<0.001	54.2±8.12	53.2±7.4	52.3±6.51	<0.001
Alcohol (g/day) ^{a)}	0.60±2.8	0.82±3.83	0.87±3.5	0.123	0.73±3.68	0.79±3.0	1.02±3.48	0.125	0.86±3.86	0.71±2.75	0.48±2.27	0.036	0.65±2.52	0.61±3.32	0.86±3.71	0.118	0.77±3.86	0.78±3.3	0.79±3.43	0.882	0.74±3.75	0.87±3.56	0.78±3.25	0.790
Total fiber (g/day) ^{a)}	18.1±2.77	18.3±7.50	19.1±7.01	0.004	18.7±7.44	18.3±7.31	18.2±7.4	0.111	19.5±7.47	17.5±7.08	17.3±7.2	<0.001	19.01±7.8	18.4±7.20	18.5±7.40	0.280	18.2±7.95	18.6±7.65	18.6±7.2	0.186	18.01±7.7	18.5±7.36	19.0±7.12	0.001
Red and Processed meat (serv/day)	0.44±0.40	0.62±0.43	0.81±0.53	>0.001	0.59±0.47	0.71±0.46	0.77±0.52	>0.001	0.37±0.32	0.48±0.34	0.57±0.39	>0.001	0.46±0.44	0.51±0.42	0.71±0.49	<0.001	0.53±0.46	0.59±0.46	0.68±0.48	>0.001	0.55±0.45	0.62±0.46	0.73±0.50	<0.001
Fruits and Vegetables (serv/day)	4.61±2.33	4.32±2.28	4.07±2.07	<0.001	4.39±2.26	4.18±2.19	4.13±2.18	0.005	4.46±2.24	4.0±2.14	4.0±2.32	<0.001	4.90±2.42	4.50±2.24	4.15±2.18	<0.001	4.30±2.48	4.46±2.03	4.23±2.19	0.003	4.43±2.41	4.32±2.10	4.20±2.15	0.005
Sugar Sweetened Beverages (serv/day)	0.07±0.26	0.11±0.25	0.23±0.44	<0.001	0.13±0.34	0.15±0.31	0.18±0.30	0.001	0.12±0.32	0.15±0.31	0.22±0.41	<0.001	0.07±0.33	0.09±0.26	0.17±0.34	<0.001	0.09±0.29	0.10±0.23	0.16±0.35	>0.001	0.11±0.28	0.13±0.40	0.17±0.33	<0.001

Data are Means ± SD or Percentage.

^{a)} Energy adjusted (2,000 kcal/day). For continuous variables, P-value was calculated using the one-way ANOVA; for categorical variables, P-value was calculated using the Chi-square test.

Table 2

Relative risks (RR) and 95% confidence intervals (95% CI) for developing GDM according to total fried food consumption, Seattle and Tacoma, WA, Omega Cohort Study

	<1 serving/week	1–3 servings/week	>3 servings/week	P for trend
Total Fried Food consumption				
N	793	1572	1049	
Cases (%)	38 (4.79)	71 (4.52)	60 (5.72)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	0.97 (0.66, 1.40)	1.30 (0.85, 1.99)	0.209
¹ Adjusted RR (95% CI)	1.00 (Ref)	0.96 (0.64, 1.44)	1.32 (0.83, 2.11)	0.228
² Adjusted RR (95% CI)	1.00 (Ref)	0.92 (0.61, 1.38)	1.26 (0.79, 2.01)	0.300

¹ Adjusted for daily energy intake, maternal age, race/ethnicity, educational attainment, family history of diabetes, physical activity, alcohol, coffee, sugar-sweetened beverages, red and processed meats, calcium, dietary magnesium and vitamin D intake.

² Further adjusted for pre-pregnancy overweight status.

Table 3

Relative risks (RR) and 95% confidence intervals (95% CI) for developing GDM according to type of deep-fried foods, Seattle and Tacoma, WA, Omega Cohort Study

	None	1 serving/month	>1 servings/month	P - trend
Fried fish				
N	2,212	708	494	
Cases (%)	97 (4.39)	32 (4.52)	40 (8.10)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	1.05 (0.71, 1.55)	1.89 (1.32, 2.71)	0.003
¹ Adjusted RR (95% CI)	1.00 (Ref)	1.01 (0.67, 1.54)	1.73 (1.19, 2.52)	0.013
² Adjusted RR (95% CI)	1.00 (Ref)	0.99 (0.66, 1.49)	1.68 (1.16, 2.45)	0.019
Fried chicken				
N	2,348	653	413	
Cases (%)	94 (4.0)	40 (6.13)	35 (8.5)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	1.53 (1.07, 2.19)	2.16 (1.48, 3.14)	<0.001
¹ Adjusted RR (95% CI)	1.00 (Ref)	1.48 (1.02, 2.16)	2.06 (1.39, 3.05)	<0.001
² Adjusted RR (95% CI)	1.00 (Ref)	1.44 (0.98, 2.09)	1.81 (1.22, 2.70)	0.002
Fried potatoes				
N	438	646	2330	
Cases (%)	24 (5.48)	32 (4.95)	113 (4.85)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	0.91 (0.54, 1.52)	0.90 (0.59, 1.39)	0.683
¹ Adjusted RR (95% CI)	1.00 (Ref)	0.97 (0.56, 1.67)	0.93 (0.58, 1.47)	0.710
² Adjusted RR (95% CI)	1.00 (Ref)	1.02 (0.59, 1.75)	0.93 (0.58, 1.47)	0.662
Snack chips				
N	727	374	2313	
Cases (%)	38 (5.23)	22 (5.88)	109 (4.71)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	1.13 (0.68, 1.90)	0.92 (0.63, 1.33)	0.548
¹ Adjusted RR (95% CI)	1.00 (Ref)	1.16 (0.67, 2.01)	1.04 (0.71, 1.54)	0.897
² Adjusted RR (95% CI)	1.00 (Ref)	1.08 (0.62, 1.87)	1.03 (0.70, 1.52)	0.894
Doughnuts				
N	1,288	682	1444	
Cases (%)	64 (4.97)	37 (5.43)	68 (4.71)	
Energy Adjusted RR (95% CI)	1.00 (Ref)	1.10 (0.74, 1.64)	0.97 (0.69, 1.37)	0.868
¹ Adjusted RR (95% CI)	1.00 (Ref)	1.06 (0.70, 1.56)	0.85 (0.59, 1.22)	0.363
² Adjusted RR (95% CI)	1.00 (Ref)	1.04 (0.69, 1.55)	0.83 (0.58, 1.19)	0.306

¹ Adjusted for daily energy intake, maternal age, race/ethnicity, educational attainment, family history of diabetes, physical activity, alcohol, coffee, sugar-sweetened beverages, red and processed meats, total fiber intake, fatty fish, calcium, dietary magnesium and vitamin D intake.

²Further adjusted for pre-pregnancy overweight status.

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