Annals of Nutritional Disorders & Therapy

Open Access

Austin Publishing Group

Mini Review

Possible Benefits of A Mediterranean Diet During Pregnancy: Prevention and Treatment of Gestational Diabetes

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Received: April 22, 2015; **Accepted:** June 25, 2015; **Published:** July 01, 2015

Abstract

Gestational Diabetes Mellitus (GDM) is an important problem of public health worldwide since it compromises both the mother's and fetus's health, not only during pregnancy but also later in life. This has become a greater issue with the introduction of new diagnostic criteria (IADPSG), after which the prevalence has increased greatly in our country. Thus, it should become a major priority to dig deep towards the best method of prevention of this metabolic alteration. Several studies have stated the efficacy of a Mediterranean Diet (MedDiet) in regulating insulin resistance and glycaemia daily excursions as well as its potential effects in the treatment of type 2 diabetes. This report aims to state the reasons for which a MedDiet could offset and treat GDM and its complications, focusing our attention on the use of Extra Virgin Olive Oil (EVOO) and nuts as a tool to ensure adherence to this diet.

Keywords: Gestational diabetes mellitus; Mediterranean diet; Prevention; EVOO; Nuts

Introduction

Gestational Diabetes Mellitus (GDM) is a condition discovered back in 1828 by Heinrich Gottleib Bennewitz, although it was not until later on in 1957 that it became named as such by ER Carrington et al. [1]. Ever since, it has been recognized as a pathology that occurs during pregnancy where the woman exhibits glucose intolerance with onset or first recognition during pregnancy [2] and which can lead to adverse outcomes in both mother and baby [3].

The Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study revealed proof that maternal glycaemic control is essential and not having one could lead to severe outcomes in fetus [4]. Recently it has been observed that the prevalence of GDM is increasing [5,6]. This increase, aside from the fact that obesity and sedentary lifestyle have become more common [7], owes to the fact that diagnostic criteria have changed [3] and as a result of it GDM prevalence has been associated with a 3.5 fold increase in our studied sample of pregnant women in Madrid, Spain [6]. The HAPO study [4] triggered the emergence of new criteria (IADPSG) to diagnose GDM and this has been associated with an increase in its prevalence, from 8% (NDDG criteria) and 10, 6% (Carpenter and Coustan) to 35, 5% [6,8] in our area.

Women with GDM experience increased risk of gestational hypertension and, although generally GDM resolves after giving birth, these women have a 7 fold chance of suffering from type 2 diabetes mellitus (T2DM) in the future as well as hypertension and heart disorders [9-11]. Compared to those with normal glucose tolerance, women with GDM also tend to have pregnancies with more obstetric complications resulting in giving birth to macrosomic or Large-for Gestational Age (LGA) offspring [11,12] this being the main complication in women with GDM and which is responsible for a higher rate of admission to the neonatal intensive care unit [6,13]. Maternal hyperglycemia has other detrimental effects on the fetus that is found to suffer from hypoglycemia caused by fetal hyperinsulinemia, which elevates the chances for obesity and diabetes later on [14,15].

Established risk factors of GDM are advanced age, family history of Diabetes Mellitus (DM), both of which are non-modifiable and, regarding the modifiable risk factors, pregestational body weight, obesity and Gestational Weight Gain (GWG) [16,17] which are also the most strongly related to GDM incidence [18,19]. They also happen to be the factors that mostly determine the baby's weight and predispose them to being either macrosomic or LGA [19]. Unfortunately very little pregnancies are actually planned (less than 8% in our population) which makes it rather complicated to control the pregestational weight therefore it should be a main objective to focus on the control of gestational weight gain and obesity during the actual pregnancy. This aims to target a control of glycaemic levels in the mother, which have been seen to have a direct impact on pregnancy, especially in what relates to less cases of macrosomia [20]. As a matter of fact, intensive treatment of hyperglycemia in pregnancy reduces perinatal adverse outcomes [20,21]. Luckily all of these factors can be modified with an adequate nutritional intervention, and it goes without saying that this is the primary approach that should be assessed when looking to prevent and treat GDM.

Citation: Assaf-Balut C, Bordiú E, Rubio MA, Torrejon MJ, Familiar C, Durán A, et al. Possible Benefits of A Mediterranean Diet During Pregnancy: Prevention and Treatment of Gestational Diabetes. Ann Nutr Disord & Ther. 2015;2(2): 1025.

Influence of maternal diet on offspring

The nutritional environment to which the fetus is exposed during pregnancy will determine long-term outcomes [22]. Aside from the genetic heritage, the mother's lifestyle while pregnant has a direct impact on the health of the baby, tightly linked to components of the metabolic syndrome [23]. As stated previously, all this can lead to perinatal complications like, for instance, having macrosomic babies. However, it is not only this aspect that should worry us, but also the effect the diet has upon the adipose-tissue distribution [24] and the latter of it in postnatal growth and which is prolonged till childhood [25].

This being said, it becomes obvious the imperativeness of meticulously planning pregnant women's nutrition and beginning a nutritional intervention in this group as soon as possible, in order to achieve an adequate BMI and with the purpose of improving short and long term outcomes in health.

Nutritional intervention

Pregnancy exposes the mother to a diabetogenic state where they find themselves to be more prone to suffer from insulin resistance favored by a rise in human chorionic gonadotropin (HCG) and human placental lactogen (hPL) [26]. Regarding this aspect, it would be essential to follow an adequate diet.

Several studies have been conducted to assess whether or not a nutritional intervention is effective when treating women with GDM as well as reducing its associated complications, all mostly proving that indeed it does contribute to the lowering of adverse outcomes. Moreover, interventions seem to succeed more when counseling and nutritional education is included [21,27]. Additionally, other studies have proven that interventions related to physical activity or dietary counseling control GWG [28], reduces shoulder dystocia and fetal overgrowth risk (LGA and macrosomia) when treating mild gestational diabetes [29]. It also proves to reduce hypertensive disorders and preeclampsia in pregnancy [30].

Prevention of GDM and its adverse outcomes are possible without the need to resort to pharmacological treatment, in the same way metabolic disorders such as metabolic syndrome (MetS) or T2DM in particular can be managed solely with a change in lifestyle and diet [31], not only in its treatment but also in terms of its prevention [32,33]

It is important to take into consideration that it is not only important to do the intervention but actually at what point it should be done. An early intervention is essential given that this contributes to the reduction of both maternal and offspring complications [34-36] and as a matter of fact O'Sullivan and coworkers [37] stated the need for this intervention to be started instantly when the pregnant women turns for medical care.

Most studies focus on an intervention based on a low-glycaemic index diet and physical activity. Despite all the evidence there is supporting how a Mediterranean Diet (MedDiet) positively affects patients with T2DM, to date nobody has contemplated the effect this kind of nutrition will have on pregnant women, prevalence of gestational diabetes and perinatal outcomes.

Mediterranean diet intervention

So, why a MedDiet? It is a diet based in a high consumption of whole grains, vegetables, fruits, legumes, olive oil, fish, dietary fiber and nuts, moderate consumption of dairy products and a low one of red and processed meat [38-40]. This provides a high amount of fiber, unsaturated fats, vitamins and natural antioxidants, and small ones of trans and saturated fats. It is a healthy diet that lacks neither macronutrients nor micronutrients. As a matter of fact given its high quantity of vegetables, fruits and olive oils it is a rich source of carotenoids, vitamin C and E [39]. And it is in this aspect, the fact that it is such a complete diet, that it emerges as a potential protective diet pattern against chronic diseases, such as T2DM [37,41]. These favorable effects don't only come from the consumption of fruits and vegetables, or even dietary fiber, its most important feature is the high fat content (unsaturated) of the MedDiet. It enables gastric emptying to be delayed, having an effect on postprandial metabolism, proving to reduce postprandial glucose and enhance insulin levels, all together lowering the glycaemic response [38,42]. Furthermore, it's considered to be a key foundation/pillar in weight-loss strategies [43], with significant difference in comparison to other typically low-fat diets. This is what has raised the interest of investigators in its use in the prevention and treatment of T2DM.

MedDiet in T2DM

GDM is similar to T2DM given that in both cases there is a physiological mishandling of glycaemia levels caused by the insulin resistance to which the individual is exposed. In both groups the main approach is to avoid increases of glucose levels. Concerning GDM, it is rather interesting to comprehend and analyze how T2DM patients are treated, what kind of lifestyle they are lured into and why.

In the management of T2DM, the MedDiet has been suggested and subsequently proved to be an ideal therapy to patients suffering from this condition as a long-term exposition to this diet has shown a reduction in both insulin and postprandial glucose levels [44-46]. Recently, it has also shown effectiveness in its prevention. As a matter of fact, the adherence to a typical MedDiet pattern was associated with a 50% reduction in the risk of T2DM [47].

Given that the key approach to treating T2DM patients is the control of the glycaemic load, which is going to depend on the glycaemic index as well as the proportion of carbohydrates in a meal or food, all of these patients nutrition therapy is primarily focused in the carbohydrate content of the diet and the way it affects postprandial glycaemia. However, latest investigations have determined that in these subjects the importance of the glycaemic load is equal to that of the fat content of the diet, specifically the types of fats: monounsaturated (MUFA) and polyunsaturated (PUFA) [48-55].

Fats and glycaemic control: The quality of fats have been an object of study by many, where unsaturated fats (MUFA and PUFA) have shown to have a desirable effect on insulin resistance, glycaemic control and has been in fact associated with a regression in DM [56] . Therefore despite the fact that fats are energy-dense nutrients, their inclusion in a diet are essential especially regarding their effect on metabolic control. In healthy subjects, MUFA are the ones that are mostly related to a positive response in insulin secretion [48,49]. However, the consumption of both MUFA and PUFA is important

in the management of patients with DM, as well as their ratios to saturated fatty acids (SFAs), being able to control glycaemic levels and ameliorate diabetic complications [50].

Olive Oil: It is the main source of MUFAs in this diet [39]. Consumption of olive oil, a rich source of MUFA, demonstrated to improve insulin response [51] and sensitivity [52]. It also exhibited beneficial effects in terms of impaired glucose intolerance [53], as well as inducing postprandial incretion-responses [54, 55]. Moreover, the incorporation of this product is more beneficial when its virgin variety (EVOO) is consumed because it is richer in polyphenol content [57], which favors a lower risk of T2DM because of its anti-inflammatory properties.

Nuts: Aside from EVOO, there are other sources from which we can benefit from these effects in terms of glycaemic control: nuts. These are also high in fats, but mostly a mixture of unsaturated fats such as MUFAs and PUFAs [58]. Like olive oil, nuts have also been related to promoting an adequate glycaemic control, where intervention and observational studies regarding nut consumption have shown benefits on glucose metabolism [59], a decrease in hyperglycemia [59,60] and insulin resistance [61] and control of body weight gain [62,63]. This effect has also been observed when meals are enriched with nuts resulting in a reduction of its acute postprandial glycaemic impact [63].

Almonds have proven beneficial in optimizing postprandial insulin responses [64], not only as snacks but incorporating them in mealtimes, especially before starchy meals, as this significantly reduces postprandial glycaemia [65,66]. Studies have shown that even when consuming high-glycaemic index meals, if nuts such as pistachios, peanuts or almonds are added, the postprandial glucose response is ameliorated by a 30-50% probably because digestion is slowed down [65]. Additionally, the chronic consumption of almonds, 5 times a week during a period of 12-weeks also significantly reduced HbA1c [66]. This provides further proof of to what extent the incorporation of nuts can determine a convenient glycaemic regulation.

Walnuts are also related to diabetes prevention and a significantly lower risk of T2DM [66-67]. They appear to exert a favorable influence in insulin resistance [67-68]. Walnuts differ from other nuts because of their richness in PUFAs content, which is uniquely high in comparison to other nuts [58]. This motivates an elevation of circulating concentrations of PUFAs, hence improving insulin resistance [69].

Along with almonds and walnuts, pistachios have also shown to improve these physiological effects on glucose response [70,71]. In fact, it is emerging as a nutritional strategy for T2DM prevention beyond the use of almonds and walnuts, despite the fact that these last ones contain a higher proportion of PUFAs [58]. The consumption of pistachios can improve glucose and insulin metabolism, alleviating postprandial glucose levels. This effect is seen mostly when they are consumed with carbohydrates [71]. Their beneficial effect is such that incorporating them chronically to our diet can even reverse risks associated with pre-diabetes [72], lower fasting glucose [72,73] and enhance insulin resistance [72]. Furthermore, a regular consumption of pistachio nuts in subjects with MetS have shown to significantly reduce glycaemia 2 hours after 75g glucose challenge [74] playing an important role in the improving risk factors of MetS.

In order to prevent and treat this condition incorporating these types of fats to a diet is as important as for what foods they are substituting. There is evidence that suggests that consuming MUFA and PUFA in substitution to saturated and trans fats [53,69] or carbohydrate foods [75] has beneficial effects on insulin sensitivity and is likely to reduce risk of T2DM.

Oily fish: No doubt oily fish is one of the main food groups, along with others such as whole grains, EVOO or nuts, that confer MedDiet its outstanding reputation. Its importance is also highlighted, and the reason behind it is its high content of unsaturated fats [38]. Indeed, oily fish is a rich source of omega-3 fatty acids, which, like EVOO and nuts, have also proved to have a marked effect on insulin resistance, glycaemia levels, and others metabolic markers [76]. These types of fats help reduce MetS prevalence [77]. Not only do they help manage weight and obesity [78], but they also have favorable effects in terms of glucose homeostasis [79] and glucose tolerance [80]. Moreover, pregnant women with GDM benefited greatly from incorporating oily fish to their diet as the omega-3 fatty acids managed to have favorable effects on insulin metabolism, specifically on insulin resistance [81].

Ongoing study

Will all of this evidence and information stating how a MedDiet rich in unsaturated fats is linked to a risk reduction and successful treatment of T2DM, we cannot help but hope it can also work for GDM. This leads to the proposal of the MedDiet as a possible prevention therapy of this condition.

A recent meta-analysis was performed to determine whether or not a nutritional approach to preventing GDM is effective and concluded it was not [82]. However, the fact that to date none of the previous interventions used the MedDiet as a possible solution should not be overlooked.

Another meta-analysis about the possible lifestyle interventions during early pregnancy, between the 8-10 and 24-28 weeks of gestation [83,84], suggests that all interventions done between these periods of time are effective in terms of GDM prevention. A study conducted by Ruiz-Gracia et al [85] not only identified a low-risk nutritional pattern associated to the prevention of GDM, but also proved how there's an association between lifestyle change during early pregnancy and prevention of GDM. This nutritional pattern was identified as a weekly consumption of >3 nuts, < 4 juices, cookies and pastries and ≤ 1 refined cereals. These results are in concordance with those obtained in other studies where decreasing sugary or sweetened drinks [86,87] alongside with other aspects like increasing physical activity or decreasing the consumption of red and processed meat are also associated to a lower GDM risk. Hence, doing the opposite can be identified as risk factors for developing GDM.

In addition, and in agreement with other studies proving the beneficial effect of MedDiet in T2DM [37,38,41,42,44-46], these results can also be extrapolated to women in their post gestational period, whom also benefited from the adherence to MedDiet as it prevented glucose homeostasis impairment [88].

For this reason, we propose that a lifestyle intervention based on MedDiet and physical activity, starting on the first gestational visits (8-12 weeks) and being maintained during the whole of the pregnancy, in women with normal fasting glucose levels (92 mg/dl),

can reduce the incidence of GDM between the 24-28 weeks, as well as reducing the morbidity in mother, gestation and newborns.

In order to facilitate the access to this kind of diet, the intervention group will be provided with EVOO and pistachio nuts of the Mediterranean region of Spain, which will both help mimic a typical Spanish diet and also serve as a tool to ensure maximum adherence to the MedDiet intervention.

We should hope that all the evidence examined in this report, combined with the fact that generally pregnant women are receptive to interventions that might improve the health of their offspring, might be enough to prove our hypothesis right.

Acknowledgement

Funding: This research is endorsed by grants from Fundación para Estudios Endocrinometabolicos, and IdISSC Hospital Clinico San Carlos, Madrid, and the Instituto de Salud Carlos III of Spain [PI14/01563].

References

1. McIntyre HD, Colagiuri S, Roglic G, Hod M. Diagnosis of GDM: a suggested consensus. Best Pract Res Clin Obstet Gynaecol. 2015; 29: 194-205.
2. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2012; 35: S64–S71.
3. Metzger BE, Gabbe SG, Persson B, Buchanan TA, Catalano PA, Damm P, et al. International Association of Diabetes and Pregnancy Study Groups recommendations on the diagnosis and classification of hyperglycemia in pregnancy. Diabetes Care. 2010; 33: 676–682.
4. HAPO Study Cooperative Research Group, Metzger BE, Lowe LP, Dyer AR, Trimble ER, Chaovarindr U, Coustan DR. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med. 2008; 358: 1991-2002.
5. ACOG Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin. Clinical management guidelines for obstetricians-gynecologists. Obstet Gynecol. 2004; 104: 639-646.
6. Duran A, Sáenz S, Torrejón MJ, Bordiú E, Del Valle L, Galindo M, et al. The introduction of IADPSG criteria for the screnning and diagnosis of Gestational Diabetes Mellitus results in improved pregnancy outcomes at a lower cost in a large cohort of pregnant women: The St Carlos Gestational Study. Diabetes Care. 2014; 37: 2442-2450.
7. Veeraswamy S, Vijayam B, Gupta VK, Kapur A. Gestational diabetes: the public health relevance and approach. Diabetes Res Clin Pract. 2012; 97: 350-358.
8. Ramos-Leví AM, Pérez-Ferre N, Fernández MD, Laura del Valle L, Bordiu E, Bedia AR, et al. Risk Factors for Gestational Diabetes Mellitus in a Large Population of Women Living in Spain: Implications for Preventative Strategies. Int J Endocrinol. 2012; 2012: 312529.
9. Retnakaran R, Qi Y, Connelly PW, Sermer M, Zinman B, Hanley AJ. Glucose intolerance in pregnancy and postpartum risk of metabolic syndrome in young women. J Clin Endocrinol Metab. 2010; 95: 670-677.
10. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet. 2009; 373: 1773-1779.
11. Lapolla A, Bonomo M, Dalfrà MG, Parretti E, Mannino D, Mello G, et al. GISOGD. Prepregnancy BMI influences maternal and fetal outcomes in women with isolated gestational hyperglycaemia: a multicentre study. Diabetes Metab. 2010; 36: 265-270.
12. Sacks DA. Fetal macrosomia and gestational diabetes: what's the problem? Obstet Gynecol. 1993; 81: 775-781.
13. Hillier TA, Pedula KL, Schmidt MM, Mullen JA, Charles MA, Pettitt DJ. Childhood obesity and metabolic imprinting: the ongoing effects of maternal hyperglycemia. Diabetes Care. 2007; 30: 2287-2292.
14. Dabelea D. The predisposition to obesity and diabetes in offspring of diabetic mothers. Diabetes Care. 2007; 30: S169-S174.
15. Griffin ME, Coffey M, Johnson H, Scanlon P, Foley M, Stronge J, et al. Universal vs. risk factor-based screening for gestational diabetes mellitus: detection rates, gestation at diagnosis and outcome. Diabet Med. 2000; 17: 26-32.
16. Xiong X, Saunders LD, Wang FL, Demianczuk NN. Gestational diabetes mellitus: prevalence, risk factors, maternal and infant outcomes. Int J Gynaecol Obstet. 2001; 75: 221-228.
17. Hedderson MM, Williams MA, Holt VL, Weiss NS, Ferrara A. Body mass index and weight gain prior to pregnancy and risk of gestational diabetes mellitus. Am J Obstet Gynecol. 2008; 198: 409.
18. Sugiyama T, Metoki H, Hamada H, Nishigori H, Saito M, Yaegashi N, et al. A retrospective multi-institutional study of treatment for mild gestational diabetes in Japan. Diabetes Res Clin Pract. 2014; 103: 412-418.
19. Black MH, Sacks DA, Xiang AH, Lawrence JM. The relative contribution of prepregnancy overweight and obesity, gestational weight gain, and IADPSG-defined gestational diabetes mellitus to fetal overgrowth. Diabetes Care. 2013; 36: 56-62.
20. Flores Le-Roux JA, Benaiges Boix D, Pedro-Botet J. [Gestational diabetes mellitus: importance of blood glucose monitoring]. Clin Investig Arterioscler. 2013; 25: 175-181.
21. Carolan-Olah MC. Educational and intervention programmes for Gestational Diabetes Mellitus (GDM) management: An integrative review. Collegian. 2015.
22. Ojha S, Robinson L, Symonds ME, Budge H. Suboptimal maternal nutrition affects offspring health in adult life. Early Hum Dev. 2013; 89: 909-913.
23. Koletzko B, Brands B, Poston L, Godfrey K, Demmelmair H. Early Nutrition Project. Early nutrition programming of long-term health. Proc Nutr Soc. 2012; 71: 371-378.
24. Symonds ME, Pope M, Sharkey D, Budge H. Adipose tissue and fetal programming. Diabetologia. 2012; 55: 1597-1606.
25. Sébert SP, Hyatt MA, Chan LL, Patel N, Bell RC, Keisler D, et al. Maternal nutrient restriction between early and midgestation and its impact upon appetite regulation after juvenile obesity. Endocrinology. 2009; 150: 634-641.
26. Kühl C. Etiology of gestational diabetes. Baillieres Clin Obstet Gynaecol. 1991; 5: 279-292.
27. Hu G, Tian H, Zhang F, Liu H, Zhang C, Zhang S, et al. Tianjin Gestational Diabetes Mellitus Prevention Program: study design, methods, and 1-year interim report on the feasibility of lifestyle intervention program. Diabetes Res Clin Pract. 2012; 98: 508-517.
28. Streuling I, Beyerlein A, von Kries R. Can gestational weight gain be modified by increasing physical activity and diet counseling? A meta-analysis of interventional trials. Am J Clin Nutr. 2010; 92: 678-687.
29. Landon MB, Spong CY, Thom E, Carpenter MW, Ramin SM, Casey B, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. N Engl J Med. 2009; 361: 1339-1348.
30. Falavigna M, Schmidt MI, Trujillo J, Alves LF, Wendland ER, Torloni MR, et al. Effectiveness of gestational diabetes treatment: a systematic review with quality of evidence assessment. Diabetes Res Clin Pract. 2012; 98: 396-405.
31. Potenza MV, Mechanick JI. The metabolic syndrome: definition, global impact, and pathophysiology. Nutr Clin Pract. 2009; 24: 560-577.
32. Umpierre D, Ribeiro PA, Kramer CK, Leitão CB, Zucatti AT, Azevedo MJ, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis. JAMA. 2011; 305: 1790-1799.
33. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA,et al. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002; 346: 393-403.

34. Hoppichler F, Lechleitner M. Counseling programs and the outcome of gestational diabetes in Austrian and Mediterranean Turkish women. Patient Educ Couns. 2001; 45: 271-274.

35. Perichart-Perera O, Balas-Nakash M, Parra-Covarrubias A, Rodriguez-Cano A, Ramirez-Torres A, Ortega-González C, et al. A medical nutrition therapy program improves perinatal outcomes in Mexican pregnant women with gestational diabetes and type 2 diabetes mellitus. Diabetes Educ. 2009; 35: 1004-1013.

36. Maher N, McAuliffe F, Foley M. The benefit of early treatment without rescreening in women with a history of gestational diabetes. J Matern Fetal Neonatal Med. 2013; 26: 318-320.

37. O'Sullivan EP, Avalos G, O'Reilly M, Dennedy MC, Gaffney G, Dunne F. Atlantic DIP collaborators. Atlantic Diabetes in Pregnancy (DIP): the prevalence and outcomes of gestational diabetes mellitus using new diagnostic criteria. Diabetologia. 2011; 54: 1670-1675.

38. Detopoulou P, Demopoulos CA, Karantonis HC, Antonopoulou S. Mediterranean Diet and Its Protective Mechanisms against Cardiovascular Disease: An Insight into Platelet Activating Factor (PAF) and Diet Interplay. Ann Nutr Disord Ther. 2015; 2: 1016.

39. Trichopoulou A. The Mediterranean diet: definition. Epidemiological aspects and current patterns. Matalas A, Stavrinos V, Wolinsky I, editors. In: Mediterranean Diet: Constituents and Health Promotion. Washington: CRC Press. 2001.

40. Simopoulos AP. The Mediterranean diets: What is so special about the diet of Greece? The scientific evidence. J Nutr. 2001; 131: 3065S-3073S.

41. Martínez-González MA, Sánchez-Villegas A. The emerging role of Mediterranean diets in cardiovascular epidemiology: monounsaturated fats, olive oil, red wine or the whole pattern? Eur J Epidemiol. 2004; 19: 9-13.

42. O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. J Am Coll Cardiol. 2008; 51: 249-255.

43. García de la Torre N, del Valle L, Durán A, Rubio MA, Fuentes M, Galindo M, et al. Dietary Patterns and Weight Loss in New-onset Type 2 Diabetes Mellitus: A Sub-analysis of the St Carlos Study: A 3-year, Randomized, Clinic-based, Interventional Study. British Journal of Medicine & Medical Research. 2014; 4: 5667-5677.

44. Huang CL, Sumpio BE. Olive oil, the mediterranean diet, and cardiovascular health. J Am Coll Surg. 2008; 207: 407-416.

45. Sloth B, Due A, Larsen TM, Holst JJ, Heding A, Astrup A. The effect of a high-MUFA, low-glycaemic index diet and a low-fat diet on appetite and glucose metabolism during a 6-month weight maintenance period. Br J Nutr. 2009; 101: 1846-1858.

46. Paniagua JA, de la Sacristana AG, Sánchez E, Romero I, Vidal-Puig A, Berral FJ, et al. A MUFA-rich diet improves posprandial glucose, lipid and GLP-1 responses in insulin-resistant subjects. J Am Coll Nutr. 2007; 26: 434-444.

47. Martin Rojas-Marcos P, Del Valle L, Fuentes Ferrer M, Runkle I, Duran A, Perez-Ferre N, et al. The lifestyle patterns in a Mediterranean population and its association with diabetes mellitus. MOPOR Case Control Study. Obesity and Metabolism. 2010;6: 69-75.

48. Salas-Salvadó J, Fernández-Ballart J, Ros E, Martínez-González MA, Fitó M, Estruch R, et al. Effect of a Mediterranean diet supplemented with nuts on metabolic syndrome status: one-year results of the PREDIMED randomized trial. Arch Intern Med. 2008; 168: 2449-2458.

49. Beysen C, Karpe F, Fielding BA, Clark A, Levy JC, Frayn KN. Interaction between specific fatty acids, GLP-1 and insulin secretion in humans. Diabetologia. 2002; 45: 1533-1541.

50. Diabetes and Nutrition Study Group of the Spanish Diabetes Association (GSEDNu). Diabetes Nutrition and Complications Trial: adherence to the ADA nutritional recommendations, targets of metabolic control, and onset of diabetes complications. A 7-year, prospective, population-based, observational multicenter study. J Diabetes Complications. 2006; 20: 361-366.

51. Rojo-Martínez G, Esteva I, Ruiz de Adana MS, García-Almeida JM, Tinahones F, Cardona F, et al. Dietary fatty acids and insulin secretion: a population-based study. Eur J Clin Nutr. 2006; 60: 1195-1200.

52. López S, Bermúdez B, Pacheco YM, Villar J, Abia R, Muriana FJ. Distinctive postprandial modulation of beta cell function and insulin sensitivity by dietary fats: monounsaturated compared with saturated fatty acids. Am J Clin Nutr. 2008; 88: 638-644.

53. Soriguer F, Rojo-Martínez G, Goday A, Bosch-Comas A, Bordiú E, Caballero-Díaz F, et al. Olive oil has a beneficial effect on impaired glucose regulation and other cardiometabolic risk factors. Di@bet.es study. Eur J Clin Nutr. 2013; 67: 911-916.

54. Vessby B, Uusitupa M, Hermansen K, Riccardi G, Rivellese AA, Tapsell LC, et al. Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. Diabetologia. 2001; 44: 312-319.

55. Thomsen C, Storm H, Holst JJ, Hermansen K. Differential effects of saturated and monounsaturated fats on postprandial lipemia and glucagon-like peptide 1 responses in patients with type 2 diabetes. Am J Clin Nutr. 2003; 77: 605-611.

56. García de la Torre N, Durán A, Del Valle L, Fuentes M, Barca I, Martín P, et al. Early management of type 2 diabetes based on a SMBG strategy: the way to diabetes regression--the St Carlos study: a 3-year, prospective, randomized, clinic-based, interventional study with parallel groups. Acta Diabetol. 2013; 50: 607-614.

57. Kecely T, Gordon MH. The antioxidant activity and stability of the phenolic fraction of green olives and extra virgin olive oil. J Agric Food Chem 2001; 81: 1391-1396.

58. Phillips KM, Patterson KY, Rasor AS, Exler J, Haytowitz DB, Holden JM, et al. Quality-control materials in the USDA National Food and Nutrient Analysis Program (NFNAP). Anal Bioanal Chem. 2006; 384: 1341-1355.

59. Ros E. Health benefits of nut consumption. Nutrients. 2010; 2: 652-682.

60. Jenkins DJ, Kendall CW, Banach MS, Srichaikul K, Vidgen E, Mitchell S, et al. Nuts as a replacement for carbohydrates in the diabetic diet. Diabetes Care. 2011; 34: 1706-1711.

61. O'Neil CE, Keast DR, Nicklas TA, Fulgoni VL 3rd. Nut consumption is associated with decreased health risk factors for cardiovascular disease and metabolic syndrome in U.S. adults: NHANES 1999-2004. J Am Coll Nutr. 2011; 30: 502-510.

62. Casas-Agustench P, López-Uriarte P, Bulló M, Ros E, Cabré-Vila JJ, Salas-Salvadó J. Effects of one serving of mixed nuts on serum lipids, insulin resistance and inflammatory markers in patients with the metabolic syndrome. Nutr Metab Cardiovasc Dis. 2011; 21: 126-135.

63. Bes-Rastrollo M, Wedick NM, Martinez-Gonzalez MA, Li TY, Sampson L, Hu FB. Prospective study of nut consumption, long-term weight change, and obesity risk in women. Am J Clin Nutr. 2009; 89: 1913-1919.

64. Kendall CW, Esfahani A, Josse AR, Augustin LS, Vidgen E, Jenkins DJ. The glycemic effect of nut-enriched meals in healthy and diabetic subjects. Nutr Metab Cardiovasc Dis. 2011; 21: S34-S39.

65. Li SC, Liu YH, Liu JF, Chang WH, Chen CM, Chen CY. Almond consumption improved glycemic control and lipid profiles in patients with type 2 diabetes mellitus. Metabolism. 2011; 60: 474-479.

66. Josse AR, Kendall CW, Augustin LS, Ellis PR, Jenkins DJ. Almonds and postprandial glycemia--a dose-response study. Metabolism. 2007; 56: 400-404.

67. Cohen AE, Johnston CS. Almond ingestion at mealtime reduces postprandial glycemia and chronic ingestion reduces hemoglobin A(1c) in individuals with well-controlled type 2 diabetes mellitus. Metabolism. 2011; 60: 1312-1317.

68. Pan A, Sun Q, Manson JE, Willett WC, Hu FB. Walnut consumption is associated with lower risk of type 2 diabetes in women. J Nutr. 2013; 143: 512-518.

69. Risérus U, Willett WC, Hu FB. Dietary fats and prevention of type 2 diabetes. Prog Lipid Res. 2009; 48: 44-51.

70. Risérus U. Fatty acids and insulin sensitivity. Curr Opin Clin Nutr Metab Care. 2008; 11: 100-105.

71. Kendall CW, West SG, Augustin LS, Esfahani A, Vidgen E, Bashyam B, et al. Acute effects of pistachio consumption on glucose and insulin, satiety hormones and endothelial function in the metabolic syndrome. Eur J Clin Nutr. 2014; 68: 370-375.

72. Kendall CW, Josse AR, Esfahani A, Jenkins DJ. The impact of pistachio intake alone or in combination with high-carbohydrate foods on post-prandial glycemia. Eur J Clin Nutr. 2011; 65: 696-702.

73. Hernández-Alonso P, Salas-Salvadó J, Baldrich-Mora M, Juanola-Falgarona M, Bulló M. Beneficial effect of pistachio consumption on glucose metabolism, insulin resistance, inflammation, and related metabolic risk markers: a randomized clinical trial. Diabetes Care. 2014; 37: 3098-3105.

74. Gulati S, Misra A, Pandey RM, Bhatt SP, Saluja S. Effects of pistachio nuts on body composition, metabolic, inflammatory and oxidative stress parameters in Asian Indians with metabolic syndrome: a 24-wk, randomized control trial. Nutrition. 2014; 30: 192-197.

75. Jenkins DJ, Kendall CW, Banach MS, Srichaikul K, Vidgen E, Mitchell S, et al. Nuts as a replacement for carbohydrates in the diabetic diet. Diabetes Care. 2011; 34: 1706-1711.

76. Samane S, Christon R, Dombrowski L, Turcotte S, Charrouf Z, Lavigne C, et al. Fish oil and argan oil intake differently modulate insulin resistance and glucose intolerance in a rat model of dietary-induced obesity. Metabolism. 2009; 58: 909-919.

77. De Camargo Talon L, Prado de Oliveira E, Moreto F, Portero-McLellan KC, Burini RC. Omega-3 fatty acids supplementation decreases metabolic syndrome prevalence after lifestyle modification program. Journal of Functional Foods. 2015.

78. Buckley JD, Howe PR. Long-chain omega-3 polyunsaturated fatty acids may be beneficial for reducing obesity-a review. Nutrients. 2010; 2: 1212-1230.

79. Gomes PM, Hollanda-Miranda WR, Beraldo RA, Castro AV, Geloneze B, Foss MC, et alSupplementation of α-linolenic acid improves serum adiponectin levels and insulin sensitivity in patients with type 2 diabetes. Nutrition. 2015; 31: 853-857.

80. Jelinek D, Castillo JJ, Arora SL, Richardson LM, Garver WS. A high-fat diet supplemented with fish oil improves metabolic features associated with type 2 diabetes. Nutrition. 2013; 29: 1159-1165.

81. Samimi M, Jamilian M, Asemi Z, Esmaillzadeh A. Effects of omega-3 fatty acid supplementation on insulin metabolism and lipid profiles in gestational diabetes: Randomized, double-blind, placebo-controlled trial. Clin Nutr. 2015; 34: 388-393.

82. Rogozińska E, Chamillard M, Hitman GA, Khan KS, Thangaratinam S. Nutritional manipulation for the primary prevention of gestational diabetes mellitus: a meta-analysis of randomised studies. PLoS One. 2015; 10: e0115526.

83. Radesky JS, Oken E, Rifas-Shiman SL, Kleinman KP, Rich-Edwards JW, Gillman MW. Diet during early pregnancy and development of gestational diabetes. Paediatr Perinat Epidemiol. 2008; 22: 47-59.

84. Agha M, Agha RA, Sandell J. Interventions to reduce and prevent obesity in pre-conceptual and pregnant women: a systematic review and meta-analysis. PLoS One. 2014; 9: e95132.

85. Ruiz-Gracia T, Duran A, Fuentes M, Rubio MA, Runkle I, Carrera EF, et al. Lifestyle patterns in early pregnancy linked to gestational diabetes mellitus diagnoses according to applying IADPSG criteria. The St. Carlos Gestational Study. Clinical Nutrition. 2015. http://dx.doi.org/10.1016/j.clnu.2015.04.017.

86. Ramos-Leví AM, Pérez-Ferre N, Fernández MD, Del Valle L, Bordiu E, Bedia AR, et al. Risk factors for gestational diabetes mellitus in a large population of women living in Spain: implications for preventative strategies. Int J Endocrinol. 2012; 2012: 312529.

87. Pérez-Ferre N, Fernández D, Torrejón MJ, Del Prado N, Runkle I, Rubio MA, et al. Effect of lifestyle on the risk of gestational diabetes and obstetric outcomes in immigrant Hispanic women living in Spain. J Diabetes. 2012; 4: 432-438.

88. Pérez-Ferre N, Del Valle L, Torrejón MJ, Barca I, Calvo MI, Matía P, et al. Diabetes mellitus and abnormal glucose tolerance development after gestational diabetes: A three-year, prospective, randomized, clinical-based, Mediterranean lifestyle interventional study with parallel groups. Clin Nutr. 2015; 34: 579-585.

Ann Nutr Disord & Ther - Volume 2 Issue 2 - 2015
ISSN : 2381-8891 | www.austinpublishinggroup.com

Citation: Assaf-Balut C, Bordiú E, Rubio MA, Torrejon MJ, Familiar C, Durán A, et al. Possible Benefits of A Mediterranean Diet During Pregnancy: Prevention and Treatment of Gestational Diabetes. Ann Nutr Disord & Ther. 2015;2(2): 1025.