# Chapter 20
# Low-Carbohydrate Diet for the Treatment of Gestational Diabetes Mellitus

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## Key Points

- CHO are the main macronutrients influencing postprandial glucose levels, and thus, diets for GDM usually manipulate the amount, quality or distribution of CHO.
- A minimum daily intake of 175 g CHO for pregnant women is recommended, regardless of whether GDM is present; however, there is no consensus on the optimal amount of CHO in women with GDM.
- Although there is not enough evidence concerning a potential negative impact of mild ketosis on the foetus, ketonuria or ketonaemia are commonly monitored in pregnant women with GDM treated with low-CHO diets.
- By definition, in non-pregnant adults, low-CHO diets contain <130 grams of CHO per day or <26% of total daily energy; however, for the purpose of this review, a low-CHO diet in GDM is a diet with the lowest CHO content used in a given trial (usually between 35 and 45% of total energy).
- Although low-CHO diets for GDM are safe and, in some cases, have been more beneficial to pregnancy outcomes than higher CHO diets, there is not enough evidence favouring this choice over others as MNT for GDM.
- There are few clinical trials addressing the overall dietary CHO content.
- Practice guidelines for GDM tend to recommend low-CHO diets, despite the sparse evidence.

**Keywords** Carbohydrates · Diet · Gestational diabetes · Gestational diabetes mellitus · Medical nutrition therapy · Nutrition therapy

## Abbreviations

ADA American Diabetes Association
AUC Area under the curve

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|---|---|
| CHO | Carbohydrate/s |
| CI | Confidence interval |
| GDM | Gestational diabetes mellitus |
| GI | Glycaemic index |
| GL | Glycaemic load |
| MNT | Medical nutrition therapy |
| RCT(s) | Randomized controlled trial (trials, in plural) |
| RR | Relative risk |

## Introduction

The digestion and subsequent absorption of carbohydrates (CHO) cause a rise in blood glucose; thus, postprandial glycaemia is CHO-dependent. This phenomenon has also been confirmed for pregnant women with gestational diabetes mellitus (GDM) [1]. In GDM, glucose intolerance is induced by maternal insulin resistance, resulting in maternal hyperglycaemia, which leads to foetal hyperinsulinism because of increased substrate delivery to the foetus. This pathological state can have negative maternal and infant consequences during and beyond pregnancy. Regarding the newborn, GDM is associated with shoulder dystocia, bone fracture, nerve palsy, deaths [2], excessive foetal growth reaching macrosomia [2–4], neonatal hyperglycaemia [3, 4] erythrocytosis, hyperbilirubinemia and stillbirths [4]. With respect to mothers, there is a higher rate of preeclampsia, caesarean delivery and postpartum depression [2]. Further, pregnant women with GDM have a higher risk of impaired glucose tolerance, type 2 diabetes and cardiovascular disease later in life, and their offspring also have a higher risk of type 2 diabetes [5] and obesity [6].

The main objective of GDM treatment is to avoid all GDM-associated complications through optimal glycaemic control, i.e. achieving normoglycaemia, while preventing ketosis and covering the nutritional requirements of the mother and foetus [7]. These targets can usually be achieved with diet only for the majority of women with GDM (52–92%) [8, 9]. For this reason, medical nutrition therapy (MNT), accompanied by physical activity, is the first-line treatment for GDM. The control of CHO is the mainstay of dietary treatment. Research into GDM diets has primarily been centred on this macronutrient; however, there are still little available data. Because pregnant women with GDM have the same nutritional needs as non-diabetic pregnant women, the recommended dietary CHO intake is a minimum of 175 g per day [10].

## Different Approaches to Control Carbohydrates

The traditional approach consists of regulating the total amount, specifically by reducing intake. In type 1 and type 2 diabetes mellitus, it is well documented that restricting CHO produces immediate therapeutic benefits (reduction or elimination of medication in type 2 diabetes and lower insulin requirements in type 1 diabetes, among others) with a good adherence and no adverse effects comparable to the effects of pharmacological treatment [11].

Another approach is founded on the modification of the dietary glycaemic index (GI). First described in 1981 [12], the GI was used to estimate the in vivo blood glucose response to the intake of a given food item, relative to that of a CHO reference food. The GI has become the target of many studies, including clinical trials in GDM. However, another chapter in this book already presents an in-depth discussion of the use of low-GI diets for GDM treatment.

Later, the glycaemic load (GL) concept was introduced [13]. The GL comprises the quantity of CHO consumed and their GI. The GL is a better predictor of the postprandial glycaemia associated with a food or diet [14].

In this chapter, we will review the effects of low-CHO diets in GDM. The general hypothesis is that these strategies can achieve glycaemic goals in GDM and prevent the incidence of poor perinatal outcomes. Some authors have suggested that the total amount of CHO has a stronger contribution to the GL than GI [15]; however, there are no published randomized controlled trials (RCTs) comparing different GL diets in GDM.

An alternative method would be to distribute CHO into several meals and snacks in order to control postprandial glycaemia, but there are no trials addressing this issue [16].

## Clinical Evidence of Low-Carbohydrate Diets for Gestational Diabetes Mellitus

### – What do we mean when we say low carbohydrate?

One of the fears of reducing the CHO content is the induction of maternal ketosis, which may adversely affect the foetus. For this reason, ketonuria is usually measured in studies. Nevertheless, there is not enough evidence for the real negative impact of mild ketonaemia on the foetus.

Recently, the American Diabetic Association (ADA) Standards of Medical Care in Diabetes, due to a lack of sufficient evidence for the ideal macronutrient distribution for diabetes, recommended, with the lowest level of evidence, the individualization of dietary treatment to achieve energy and metabolic targets [17]. In the absence of a specific recommended percentage or amount of CHO for pregnant women with or without GDM, it is difficult to define low-CHO diets in normal physiological and pathological situations. A recent critical review suggested some definitions based on the dietary CHO content. Diets with a total of less than 130 grams of CHO per day or less than 26% of total energy in the form of CHO are proposed to be considered as low-CHO diets. Additionally, diets with a percentage of total energy from CHO between 26 and 45% would be considered moderately CHO-restricted diets, while high-CHO diets would provide more than 45% energy as CHO [11]. In this chapter, we will consider studies comparing any two dietary interventions with different CHO content, and the one with the lowest CHO amount will be considered the low-CHO diet because the classification proposed by the mentioned authors did not consider the particularities of pregnancy. It must be noted that 130 g/d of CHO is the minimum recommended for adults and children. For pregnant women, this value is higher, i.e. 175 g/d [10].

### – Clinical trials with low-carbohydrate diets

Although modifying the quantity of CHO seems to be the first MNT approach for the treatment of GDM, there have been a few clinical trials comparing low-CHO with higher CHO diets. In all the diets tested in these trials, complex CHO prevail over simple sugars, but we will not expand on this matter because other chapter of this book addresses complex CHO diets for GDM. Table 20.1 summarizes the characteristics of the trials testing diets with different CHO content. We are, therefore, providing a detailed description of the few available studies.

In the early 1980s, Nolan et al. performed a small randomized crossover study comparing two diets. In one arm of the study, a low-CHO diet with 35% CHO (20% protein, 45% fat, 31 g fibre) was included. This was compared to a high-CHO diet, with 70% CHO (20% protein and 10% fat, 70 g fibre). Both diets were isocaloric and contained unrefined CHO. In both arms, the foods were provided by the investigators. Only five women participated in the study and adhered to each diet for a short period of 4 days. The researchers found that a low-CHO diet resulted in less favourable

**Table 20.1** Clinical evidence of low-carbohydrate diets

| Reference | Type of clinical trial | *N* | BMI | Ethnicity | Duration of intervention | Low-CHO diet % CHO (%) | High-CHO diet %CHO (%) | Main results of low-CHO diet |
|---|---|---|---|---|---|---|---|---|
| Nolan [18] | Randomized crossover | 5 | 26.9 ± 8 | NR | 4 days each diet | 35 | 70 | Worse results in fasting plasma cholesterol ($P < 0.01$), FFA ($P < 0.02$) and glucose response to glucose load ($P < 0.05$)<br>No differences in: plasma glucose, urinary glucose output, fasting plasma triglycerides and insulin response to glucose load |
| Major [19] | Non-randomized | 42 | NR | Hispanic | Until delivery | <42 | 45–50 | Lower postprandial glucose values ($P < 0.04$), insulin need ($P < 0.048$), prevalence of births large for gestational age ($P < 0.035$), caesarean and macrosomia ($P < 0.037$)<br>No differences but better HbA1c, no shoulder dystocia or birth trauma<br>Ketonuria in two women |
| Cypryk [21] | Randomized | 30 | NR | Caucasian | 14 days | 45 | Over 60 | Low-CHO greater decrease glycemia after breakfast ($P < 0.05$)<br>No difference in glucose fasting values, ketonuria or obstetric outcomes |
| Moreno-Castilla [22] | Randomized | 152 | 26 ± 5 | Caucasian | Until delivery | 40 | 55 | No differences in the rate of insulin or in pregnancy outcomes |
| Hernandez [24] | Randomized crossover | 16 | 33.6 ± 1 | Caucasian | 12 days | 40 | 60 | Lower: postprandial glycemia, daytime mean glucose and lower AUC of postprandial glycemia and of 24 h total glucose values ($P = 0.02$)<br>Higher FFA AUC |
| Hernandez [25] | Randomized crossover | 12 | Low-CHO: 33.4 ± 1.4<br>High CHO: 34.3 ± 1.6 | Hispanic (high-CHO)/ Caucasian (low-CHO) | Until delivery | 40 | 60 | No benefits in terms of maternal insulin resistance and offspring adiposity |

*Abbreviations* NR: not reported, FFA: free fatty acids, AUC: area under the curve

outcomes over high CHO with the following results: higher fasting plasma cholesterol (6.3 ± 1.1 mmol/L vs. 5.9 ± 1.1 mmol/L, $P < 0.01$), free fatty acids (FFA) (690 ± 270 µmol/L vs. 590 ± 270 µmol/L, $P < 0.02$) and glucose response to glucose load ($P < 0.05$). Nevertheless, there were no statistically significant differences in plasma glucose (fasting and postprandial), urinary glucose excretion, fasting plasma triglycerides or insulin response to a glucose load [18].

In 1998, Major et al. conducted a non-randomized non-controlled study of 42 women with gestational diabetes who were predominantly Hispanic. Participants were assigned to a low-CHO diet with less than 42% of total CHO content (25% protein, 35% fat) or a high content-CHO diet with 45–50% CHO. In a univariate analysis, the researchers found lower postprandial glucose values (110 ± 18 mg/dL versus 132 ± 19 mg/dL, $P < 0.04$) and daily insulin need (relative risk (RR) 0.14; 95% confidence interval (CI) 0.02, 1.0; $P < 0.048$) in the lower CHO group. The low-CHO diet also resulted in a larger reduction in HbA1c, although this finding was non-significant. Additionally, women in the low-CHO diet group had a lower frequency of large for gestational age newborns (RR 0.22; 95% CI 0.05, 0.091; $P < 0.035$), caesarean sections and macrosomia (RR 0.15; 95% CI 0.04, 0.94; $P < 0.037$). The only potential adverse effect of the low-CHO diet was the appearance of ketonuria in two women, which was managed by increasing the amount of CHO [19].

Conversely, in 2001, Romon et al. in a prospective observational nutrition survey sub-study developed in France, found that infant birth weight was negatively correlated with CHO intake ($r$ −0.27, $P < 0.05$), a negative correlation that persisted after a forward-stepwise regression analysis [20].

In 2007, Cypryk et al. did not find any difference regarding fasting glucose values, ketonuria or obstetric outcomes in a randomized trial with 30 Caucasian women with GDM allocated to diets with either a 45% or 65% CHO content (25% protein and 30% fat or 25% protein and 15% fat, respectively), both with a total of 1800 kcal per day. Notwithstanding, only a low-CHO diet resulted in a significant reduction in postprandial breakfast glycaemia (102 ± 16 mg/dL before intervention vs 94 ± 11 mg/dL after intervention, $P < 0.021$) [21].

In 2007, the Fifth International Workshop Conference on GDM issued nutrition recommendations for women with GDM, including gestational weight gain, calorie intake, and macronutrient composition and distribution. These recommendations were based on the current limited available scientific evidence, while encouraging the performance of further controlled clinical trials comparing intensive dietary strategies, mainly focused on the amount, type and distribution of CHO [16]. Following these recommendations, our group designed and performed the first RCT comparing a low-CHO diet with 40% of the total energy content from CHO with a control diet with 55% of the total energy content from CHO. This trial was conducted in 152, mainly Caucasian, pregnant women with GDM [22]. The study hypothesis stated that a low-CHO diet would lead to less dietary treatment failure (main study outcome: the need for insulin treatment) with similar obstetric and perinatal outcomes. Both diets had a minimum of 1800 kcal per day. The protein content was 20% of the total calorie amount, and fat represented 40% in the low-CHO diet and 25% in the control diet. The difference in fat intake was achieved mainly by increasing the olive oil content. CHO were distributed into three main meals and three snacks, and these amounts and distribution were fixed unless the women reached the main study outcome (need of insulin treatment). The assessment of the CHO intake was made by estimated 3-day food records, and it reflected that the intake of total CHO was different between the groups, although most participants reported eating less CHO than prescribed (202.7 g/day and 177.1 g/day in the high and low-CHO groups, respectively, $P = 0.0001$). This difference in the CHO content was mainly due to an increase in the intake of starch in the high-CHO diet, as the intake of sugars was equivalent in both study groups. A total of 130 participants completed the trial, and the intention-to-treat analysis showed that there were no significant differences between both groups in the rate of insulin treatment (included insulin dose and time to insulin initiate) (54.7%) or in pregnancy outcomes.

It should be noted that when a lower CHO diet is prescribed to GDM patients, there should be a parallel increase in the percentage of fat to maintain the total energy supply, as the proportion of protein usually remains stable at 15–20% of total daily calories. Outside of pregnancy, it is well known that a high-fat diet increases serum FFA, promoting insulin resistance; this effect appears to be more deleterious for saturated fats [23]. Hernandez et al. [24] compared the glycaemic profiles and postprandial lipids of a conventional lower CHO/high-fat diet (40% CHO, 45% fat, 15% protein) versus the consumption of a higher complex CHO/lower fat CHOICE (Choosing Healthy Options in Carbohydrate Energy) diet (60% CHO, 25% fat, 15% protein). They designed a controlled randomized crossover trial, in which they provided two alternative regimens of 3-day diets to 16 pregnant women with overweight/obesity and GDM. The patients wore a continuous glucose monitor sensor (blinded), and, on the fourth day of each diet, they measured the postprandial (5 h) glucose, insulin, triglycerides and FFA after a breakfast meal test. Both diets comprised a minimum of 1800 kcal per day with the same distribution in each meal (three main meals and two snacks). Both diets provided similar amounts of fibre, serving foods with low-moderate GI, and simple sugars represented less than 30% of total CHO and less than 18% of total daily calories. The fatty acid content, mostly monounsaturated, was also similar. Women were blinded to the continuous glucose monitoring system values. The results showed that the low-CHO diet led to a lower overall 1 h postprandial glucose across three meals (107 ± 3 mg/dL vs. 115 ± 2 mg/dL, $P \leq 0.01$) and 2 h postprandial glucose across three meals (97 ± 3 mg/dL vs. 106 ± 3 mg/dL, $P = 0.001$). Specific, meal-related glycaemic profiles showed that a low-CHO diet led to a significantly lower 1 h postprandial glycaemia after lunch (101 ± 3 mg/dL vs. 115 ± 3 mg/dL, $P \leq 0.001$) and 2 h postprandial glycaemia after breakfast (99 ± 3 mg/dL vs. 111 ± 4 mg/dL, $P \leq 0.01$) and lunch (93 ± 3 mg/dL vs. 104 ± 3 mg/dL, $P \leq 0.001$). The mean daytime glucose was also lower in the low-CHO diet group (93 ± 3 mg/dL vs. 98 ± 2 mg/dL, $P = 0.03$), and the daytime glucose area under the curve (AUC) was also lower (93,663 ± 2630 mg·min/dL vs. 99,493 ± 2136 mg·min/dL, $P = 0.01$). The primary study outcome showed that the low-CHO diet resulted in lower 24-h total glucose values (128,653 ± 3810 mg·min/dL vs. 136,730 ± 2980 mg·min/dL, $P = 0.02$). Additionally, FFA were lower at the 5 h postprandial measurement of controlled breakfast test in the high-CHO diet, which led to a significantly lower FFA AUC. As higher FFA levels may induce excess foetal growth, the authors suggested that a diet liberalizing complex CHO and limiting fat (CHOICE) could be a strategy to prevent macrosomia.

After these results, these authors performed a pilot RCT comparing the effects of these two diets in terms of maternal insulin resistance, adipose tissue lipolysis and foetal adiposity in 12 overweight/obese pregnant women with GDM [25]. The low-CHO diet resulted in no benefits over the higher CHO diet. Furthermore, patients allocated to the CHOICE diet had a lower fasting glucose and FFA. An adipose tissue biopsy was performed, and studies showed greater insulin sensitivity and lower proinflammatory gene expression in those women that followed the CHOICE diet. The researchers also identified a trend towards lower infant adiposity from those pregnancies under the CHOICE diet, which was correlated with maternal fasting insulin and HOMA-IR. Finally, the authors attributed these effects to dietary fat.

## Recommendations or Guidelines

In Table 20.2, we include a brief summary of the recommendations of the principal guidelines concerning MNT for GDM. Despite the poor evidence and the lack of benefit for low-CHO diets for GDM, most guidelines recommend a low intake of CHO during GDM (between 35 and 50% of total energy from CHO), always covering the DRI for pregnant women of 175 g of CHO per day [7, 27]. The American Academy of Nutrition and Dietetics [27] recommends that CHO should represent less

**Table 20.2** Summary of more recent recommendations of different organizations about carbohydrates for gestational diabetes mellitus[b]

| Organization | Year | CHO amount recommended |
|---|---|---|
| AND [27] | 2008 | DRI A minimum of 175 g/day <45%[a] |
| ADA [7] | 2007 | DRI A minimum of 175 g/day |
| Endocrine Society [28] | 2013 | 35–45%[a] |
| CDA [29] | 2013 | 40–50%[a] |
| DDG-DGGG [30] | 2014 | 40–50%[a] 15–30 g for breakfast |

*Abbreviations* AND: Academy of Nutrition and Dietetics, ADA: American Diabetes Association, CDA: Canadian Diabetes Association, DGG-DGGG: German Diabetes Association and German Association for Gynaecology and Obstetrics, CHO: carbohydrates, DRI: Dietary reference intakes, NR: not reported

[a]% of total daily calories

[b]Derived from Moreno-Castilla et al. [26]

than 45% of total daily calories, while the Endocrine Society [28] advocates for a CHO content of 35–45% of the total calories. The Canadian Diabetes Association [29] and the German Diabetes Association and German Association for Gynaecology and Obstetrics [30] established energy from CHO in an interval that ranges from 40 to 50%. Thus, the range of recommended energy from CHO is wide (from 35 to 50%), depending on the guidelines. Nevertheless, as some authors concluded [31], evidence could not be extended to Asian countries because the studies were primarily from Western countries.

## Conclusions

There is not enough evidence to recommend a particular amount of CHO neither for the general pregnant population nor for pregnant women with GDM. However, Feinman et al. [11], in a critical review, supported a low-CHO diet as the first treatment strategy for type 2 diabetes and the most effective MNT, together with pharmacological treatment for type 1 diabetes.

Nevertheless, maternal glycaemia is not only affected by the CHO content. Furthermore, the GI alone does not reflect the real impact of CHO consumed. We do not eat only CHO with a specific GI, and the context has to be considered. We eat real foods in which the CHO content and the GI, among other nutritional factors, determine the maternal glucose response and, thus, perinatal outcomes. The control of GL would be a better approach to measuring the real impact of maternal diet on GDM; therefore, the RCT testing of low-GL diets are needed. In addition, it is important to consider CHO distribution throughout the day.

Furthermore, there is a possibility that other dietary factors, such as fat or protein, can play a part in the metabolic control of GDM, foetal growth and obstetric results. Therefore, these RCTs should include these outcomes.

When insulin therapy has to be introduced in GDM, it is difficult to determine whether the diet failed or the pregnant woman did not correctly follow it. Thus, it is important to not only elucidate the proper diet for GDM but also to check that it is being fully applied. It is for this reason qualified dietitians are needed. The ADA recommends again, with the highest level of evidence, that registered dietitians should be the healthcare professionals providing MNT to individuals with type 1 and type 2 diabetes [17]; however, their expertise is also important in GDM, as some authors have reported [32].

The knowledge of the best dietary GL and CHO distribution for GDM would result in cost savings, given the potential prevention of pharmacological treatment.

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