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EFFECTS OF LOW CARBOHYDRATE DIETS IN INDIVIDUALSWITH TYPE 2 DIABETES MELITUS : THE SYSTEMATIC REVIEW

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Abstract

People who have type 2 diabetes have been known to use low carbohydrate dietary (LCDs) approaches for some time now, but the standard of care in many parts of the world has been centered on the utilization of a low fat, calorie controlled diet — in line with that which is typically recommended for the general population — for the better part of the last few decades. This has been the case even though it has been known for some timenow that people who have type 2 diabetes use low carbohydrate dietary (LC Patients who have been diagnosed with diabetes are often given the recommendation to participate in some kind of food intervention program; however, several authoritative organizations have varying recommendations in this area. Before insulin was discovered, the most common method for managing diabetes was via the use of restricted diets that focused onlimiting carbohydrate intake. However, in recent years, there has been a shift away from favoring these diets as an effective technique for managing diabetes. Because insulin resistance is a key underlying mechanism of type 2 diabetes, which is driven in part by chronic hyperglycemia, it has been suggested that lowering dietary intake of carbohydrates, the majority of which is absorbed as glucose or fructose, will improve blood glucose control and outcomes of type 2 diabetes. This is because chronic hyperglycemia is one of the factors that contributes to the development of insulin resistance. This article demonstrates that LCD may reliably reduce HbA1c levels; however, the reduction in HbA1c that takes place once a patient has a HbA1c level of less than 8 is not considered to be meaningful.

Keyword: *Blood Glucose; Carbohidrate; Diabetes Mellitus; Diets*

INTRODUCTION

Before the discovery of insulin in 1921, the standard therapy for diabetes was starvation diets that were low in carbohydrates. This was because diabetes was thought to be a condition related to the metabolism of carbohydrates for a long time.[1] From the 1930s through the 1960s, many specialists continued to recommend that individuals withdiabetes adopt a diet that was rich in fat and low in carbohydrates. As a direct result of this advice, the majority of people with diabetes followed such a diet.[2]

The dramatic increase in deaths from vascular disease in those whose lives were prolonged by insulin treatment led to a volte-face in the 1980s, with authorities now recommending low fat, high carbohydrate diets. Nevertheless, some early work in the 1920s and 1930s had suggested that high carbohydrate diets improved glucose tolerance.[3,4] There is a renewed interest in very low carbohydrate diets for the treatment of diabetes, with various physicians extolling the virtues of dietary carbohydrate restriction as the first approach in diabetes management, and some authorities recognizing low carbohydrate diets as an appropriate weight-loss strategy for those with type 2 diabetes.[5]

People who have type 2 diabetes have been known to make use of low carbohydrate dietary (LCDs) approaches for some time now, but the standard of care in many parts of the world has been centered on the utilization of a low fat, calorie controlleddiet — in line with that which is typically recommended for the general population — forthe better part of the last few decades. LCDs, on the other hand, have recently seen a surgein consumer demand. Because of this, there has been a rise in concern over their efficacy,safety, and effectiveness.[6]

Carbohydrate restriction in this group is still not without debate, despite the fact that such diets are being adopted by a growing number of persons who have type 2 diabetes. In point of fact, the discussion on this method has frequently been negative, withthe majority of the criticism being unwarranted and failing to objectively assess the material that is now available.[6] Therefore, the objective of this present narrative review is to take into account the previous body of research, as well as to investigate the common problems and practical factors connected with the implementation of this methodology.[7,8]This article investigates the possible connection between eating a diet low in carbohydrates and type 2 diabetes mellitus.

METHODS

Protocol

The Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 checklist served as the basis for the regulations that guided the executionof this systematic review.

Eligibility Criteria

This systematic review was developed to analyze papers on "carbohidrate diets" and "type 2 diabetes mellitus". These are the topics that were extensively covered in the study that was considered. In order for your work to be considered, the following conditions must be met: 1) Articles must be written in the English language. 2) Articles must have been published after 2017, but prior to the creation of this systematic review. Under no circumstances will the following types of textual contributions be considered for inclusion in the anthology: 1) Editorial letters, 2) submissions without a Digital Object Identifier (DOI), and 3) article reviews and submissions similar to those previously published in the journal.

Search Strategy

The search for studies to be included in the systematic review was carried out from December, 2nd 2022 using the PubMed and SagePub databases by inputting the words: "carbohydrate diets" and "type 2 diabetes mellitus". Where ("carbohydrate s"[All Fields] OR "carbohydrated"[All Fields] OR "carbohydrates"[MeSH Terms] OR "carbohydrates"[All Fields] OR "carbohydrate"[All Fields]) AND ("diet"[MeSH Terms] OR "diet"[All Fields] OR "diets"[All Fields] OR "diet s"[All Fields] OR "dieted"[All Fields] OR "dieting"[All Fields]) AND ("diabetes mellitus, type 2"[MeSH Terms] OR "type 2 diabetes mellitus"[All Fields]) is used as search keywords.

Data retrieval

After completing a literature analysis and reviewing the titles and abstracts of previously published studies, the author of the study amended the criteria for what shouldbe included in the study and what should not be included in the study. The new criteria may be found in the appendix of the study. This was done so that it could be determinedwhat aspects of the situation should be included in the study and what aspects should notbe included in the study. Following a study of previously conducted and published studies, the author came to the conclusion that these modifications were necessary.

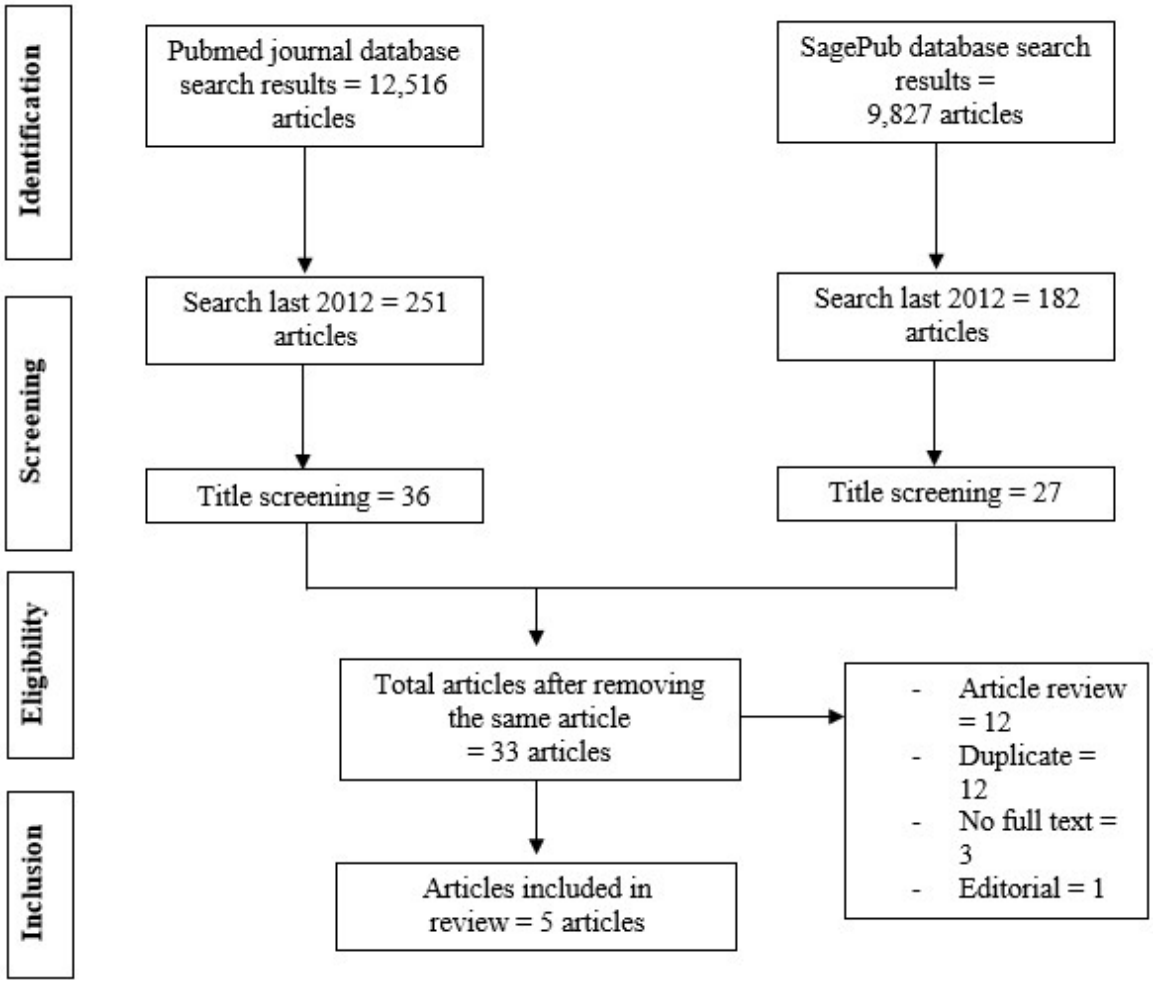

Figure 1. Article search flowchart

During the process of compiling the systematic review, it was determined that theonly research projects that were worthy of consideration were those that achieved successin achieving each and every one of the parameters. This was done in order to guarantee that the review is as comprehensive as it possibly can be. It is possible to gather information about each individual study, such as its title, author, publication date, origin of study location, research study design, and research variables. Some of the information that may be collected includes the following: This information may be provided in a variety of formats, depending on your preference.

Quality Assessment and Data Synthesis

The authors conducted their own independent reviews of a subset of the researchlisted in the titles and abstracts of the papers to determine which studies could be considered. Following this, the full texts of the studies that meet the inclusion criteria forthe systematic review will be read to determine which studies can be used as final inclusions for the purposes of the review. This will be done in order to respond to the question, "Which studies can we use for the review?"

RESULT

Dyson *et al* (2015) showed HbA1c fell in the LCD group only (LCD at 6 months
−4.8 ± 8.3 mmol/mol, p = 0.004, at 12 months −2.2 ± 7.7 mmol/mol, p = 0.12; LFD at 6 months −0.9 ± 8.8 mmol/mol, p = 0.56). At 6 months, HDL-cholesterol had increased with the LCD (from 1.13 ± 0.33 mmol/l to 1.25 ± 0.47 mmol/l, p = 0.018) while LDL- cholesterol did not differ between groups. Insulin doses were reduced in the LCD group (0 months, LCD 42 ± 65 E, LFD 39 ± 51 E; 6 months, LCD 30 ± 47 E, LFD 38 ± 48 E; p = 0.046 for between-group change).[8]

Table 1. The litelature include in this study

Author	Origin	Method	Sample Size	Result
Dyson, 2015[8]	UK	Prospective randomised parallel trial	61	HbA1c fell in the LCD group only (LCD at 6 months −4.8 ± 8.3 mmol/mol, p = 0.004, at 12 months −2.2 ± 7.7 mmol/mol, p = 0.12; LFD at 6 months −0.9 ± 8.8 mmol/mol, p = 0.56). At 6 months, HDL-cholesterol had increased with the LCD (from 1.13 ± 0.33 mmol/l to 1.25 ± 0.47 mmol/l, p = 0.018) while LDL-cholesterol did not differ between groups. Insulin doses were reduced in the LCD group (0 months, LCD 42 ± 65 E, LFD 39 ± 51 E; 6 months, LCD 30 ± 47 E, LFD 38 ± 48 E; p = 0.046 for between-group change).
Krebs, 2013[9]	New Zealand	Clinical trial	40	Glycemic control significantly improved (HbA1c −1.1 ± 0.25%) with reductions in hypoglycemic medication. Fasting glucose, homeostasis model assessment (HOMA), and area under the curve (AUC) glucose (intravenous glucose tolerance test [IVGTT]) were significantly reduced by week 12 (p < 0.05). There were nonsignificant improvements in insulin sensitivity (SI) at week 12 (p = 0.19) and week 24 (p = 0.31). Systolic blood pressure was reduced (mean −10.0 mmHg between weeks 0 and 24, p = 0.13). Mean high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol all increased. The ratio of total: HDL cholesterol and triglycerides was reduced.
Guldbrand, 2012[10]	Sweden	Clinical trial	61	At 24 months, patients on the LFD had lost −2.97 ± 4.9 kg and those on LCD −2.34 ± 5.1 kg compared with baseline (p = 0.002 and p = 0.020 within groups, respectively). HbA1c fell in the LCD group only (LCD at 6 months −4.8 ± 8.3 mmol/mol, p = 0.004, at 12 months −2.2 ± 7.7 mmol/mol, p = 0.12; LFD at 6 months −0.9 ± 8.8 mmol/mol, p = 0.56). At 6 months, HDL-cholesterol had increased with the LCD (from 1.13 ± 0.33 mmol/l to 1.25 ± 0.47 mmol/l, p = 0.018) while LDL-cholesterol did not differ between groups. Insulin doses were reduced in the LCD group (0 months, LCD 42 ± 65 E, LFD 39 ± 51 E; 6 months, LCD 30 ± 47 E, LFD 38 ± 48 E; p = 0.046 for between-group change).
Mayer, 2014[11]	USA	RCT	46	At baseline, mean body mass index (BMI) was 39.5 kg/m2 (s.d. 6.5) and haemoglobin A1c (HbA1c) 7.6% (s.d. 1.3). Although the interventions reduced BMI similarly (LCD −2.4 kg/m2; LFD + O −2.7 kg/m2, p = 0.7), LCD led to a relative improvement in HbA1c: −0.7% in LCD versus +0.2% in LFD + O [difference −0.8%, 95% confidence interval (CI) = −1.6, −0.02; p = 0.045]. LCD also led to a greater reduction in antiglycaemic medications using a novel medication effect score (MES) based on medication potency and total daily dose; 70.6% of LCD versus 30.4% LFD + O decreased their MES by ≥50% (p = 0.01).
Yamada, 2014[12]	Japan	RCT	24	The HbA1c levels decreased significantly from baseline to six months in the low-carbohydrate diet group (baseline 7.6±0.4%, six months 7.0±0.7%, p=0.03) but not in the calorie-restricted group (baseline 7.7±0.6%, six months 7.5±1.0%, n.s.), (between-group comparison, p=0.03). The patients in the former group also experienced improvements in their triglyceride levels, without experiencing any major adverse effects or a decline in the quality of life.

Krebs, 2013 showed a reduction glycemic control with significantly improved (HbA1c −1.1 ± 0.25%) in hypoglycemic medication group. Fasting glucose, homeostasis model assessment (HOMA), and area under the curve (AUC) glucose (intravenous glucose tolerance test [IVGTT]) were significantly reduced by week 12 (p < 0.05). There were nonsignificant improvements in insulin sensitivity (SI) at week 12 (p = 0.19) and week 24 (p = 0.31). Systolic blood pressure was reduced (mean −10.0 mmHg between weeks 0 and 24, p = 0.13). Mean high-density lipoprotein (HDL), low-density lipoprotein(LDL), and total cholesterol all increased. The ratio of total: HDL cholesterol and triglycerides was reduced.[9]

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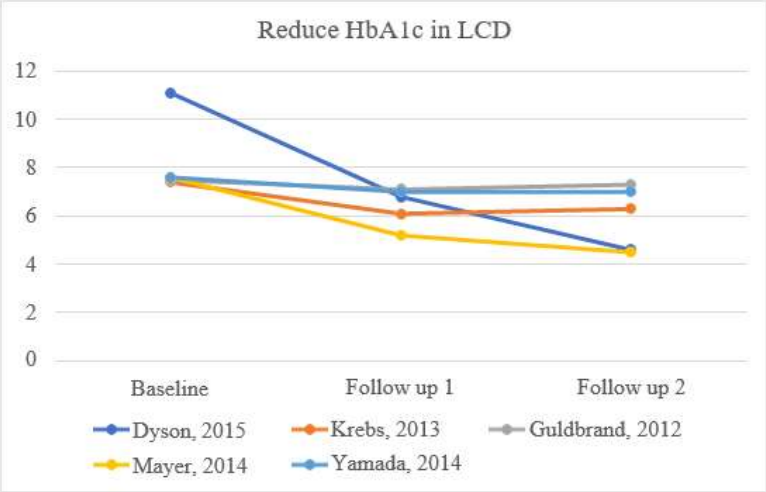

Figure 2. Reduce HbA1c in study

Yamada conducted a study in Japan and they showed HbA1c levels decreased significantly from baseline to six months in the low-carbohydrate diet group (baseline 7.6±0.4%, six months 7.0±0.7%, p=0.03) but not in the calorie-restricted group (baseline 7.7±0.6%, six months 7.5±1.0%, n.s.), (between-group comparison, p=0.03). The patients in the former group also experienced improvements in their triglyceride levels, without experiencing any major adverse effects or a decline in the quality of life.[12]

DISCUSSION

Treating type 2 diabetes is difficult, since it involves managing glycemia, CVD risk factors, obesity, and other co-morbidities using a mix of lifestyle changes (diet and physical activity), behavioral and psychiatric therapies, pharmacological therapy, and bariatric surgery.[13] One of the problems associated with the phrase "low carbohydrate" is the lack of clarity on what this implies in terms of the amount of carbohydrates that are consumed. Very low carbohydrate ketogenic diets (VLCKD) tend to have more significant effects than other, less limited carbohydrate diets.[14]

Ketosis is easily induced when carbohydrate consumption is less than 50 g per day, and it is shown that these diets have a greater impact on the body. In a recent work, the taxonomy for diets that contain different quantities of dietary carbohydrates was provided. Other diets, such as the Zone Diet and the South Beach Diet, promote a moderate carbohydrate restriction together with high protein and low fat intakes. In practice, the majority of diets that are modeled after the Atkins diet are designed to have very few carbohydrates (less than 20 g per day initially) and a high protein and fat content.In contrast, the Atkins Diet is designed to have a very low carbohydrate intake.[6,15]

It is possible to argue, from a physiological point of view, that carbohydrates should be avoided in order to achieve good glycaemic control in type 2 diabetes. This is due to the fact that a typical feature of type 2 diabetes is the combination of reduced insulin sensitivity and the failure of beta cells to provide adequate amounts of insulin to manage glucose that is derived from carbohydrates in the diet. However, when the macronutrient composition of a diet is altered by decreasing the amount of carbohydrates consumed, the energy from this source is primarily replaced by the energy obtained from fat.[16,17]

This is because it is difficult to maintain a high energy intake from protein over the course of a diet. Therefore, a diet that is low in carbohydrates, also known as a low- carbohydrate diet (LCD), is quite comparable to a diet that has a high consumption of fat, which has historically been related with an elevated risk for arteriosclerosis, especially when substantial amounts of saturated fat are taken. However, new evidence has called into question the idea of the dangers associated with a diet heavy in fat. According to thefindings of an observational research conducted in Sweden including 28,000 participants in the middle years of their lives, neither a high overall fat consumption nor an intake of substantial levels of saturated fat (22%) was associated with an elevated risk for cardiovascular disease.[16,17]

Patients diagnosed with diabetes are often given advice to follow a structured diet intervention, however different authoritative organizations have different guidelines in this regard. Diets that emphasize carbohydrate restriction were widely utilized for the control of diabetes before to the discovery of insulin. However, in recent years, these diets have gone out of favor as an effective method of diabetes management. Because insulin resistance is a key underlying mechanism of type 2 diabetes, which is driven in part by chronic hyperglycemia, it has been suggested that lowering dietary intake of carbohydrate, the majority of which is absorbed as glucose or fructose, will improve bloodglucose control and outcomes of type 2 diabetes.[18,19]

In the scientific literature, structured diets with carbohydrate restriction have beencharacterized in a variety of ways, but these diets are often categorized into one of three categories: 20-50 grams of carbohydrates per day, or less than 10% of a diet consisting of 2000 calories per day, is generally sufficient to induce ketosis. Less than 130 grams ofcarbohydrates per day, or less than 26% of a diet consisting of 2000 calories per day, and less than 45% of a diet consisting of 2000 calories per day are also sufficient. For the sakeof this analysis, we will refer to diets as low carbohydrate if they contain

fewer than 130 g of carbs per day or less than 26% of their daily caloric intake (based on 2000 kcal) comes from carbohydrates (LCD).[20,21]

According to the diet records kept during the trial period of twenty-four months, patients who followed the LCD had an increase in the proportion of their daily energy intake coming from both total fat and saturated fat. This was in accordance with the protocol for the research. Only the LCD group had changes in blood lipid levels, which manifested as a rise in HDL-cholesterol around the 6-month mark, when the weight loss was at its most prominent. The fact that there were also changes in lipid-lowering treatment throughout the course of the trial renders these results equivocal with respect tothe question of whether or not they were entirely dependent on dietary shifts.[22]

CONCLUSION

This article shows that LCD can consistently suppress HbA1c levels, even so thedecrease in HbA1c that occurs after a patient has an HbA1c level of less than 8 is not significant.

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