Eating Patterns and Health Outcomes in Patients With Type 2 Diabetes

Roberta Aguiar Sarmento,1,2 Juliana Peçanha Antonio,1,2 Ingrid Lamas de Miranda,1,2 Bruna Bellicanta Nicoletto,1 and Jussara Carnevale de Almeida1,2,3

1Endocrinology Division, Hospital de Clínicas de Porto Alegre, Universidade Federal do Rio Grande do Sul, 90035-003 Porto Alegre, Rio Grande do Sul, Brazil; 2Food and Nutrition Research Center, Hospital de Clínicas de Porto Alegre, Universidade Federal do Rio Grande do Sul, 90035-003 Porto Alegre, Rio Grande do Sul, Brazil; and 3Department of Nutrition, Faculdade de Medicina, Universidade Federal do Rio Grande do Sul, 90035-003 Porto Alegre, Rio Grande do Sul, Brazil

Purpose: To evaluate the relationship between eating patterns and therapeutic target’s achieving in patients with type 2 diabetes.

Methods: In this cross-sectional study, patients underwent clinical, laboratory, and nutritional evaluations. Dietary intake was assessed by a quantitative food frequency questionnaire and eating patterns identified by cluster analysis. The therapeutic targets were as follows: blood pressure, <140/90 mm Hg; BMI, <25 kg/m² (<27 kg/m² for elderly); waist circumference, <94 cm for men and <80 cm for women; fasting plasma glucose, <130 mg/dL; HbA1c, <7%; triglycerides, <150 mg/dL; HDL-cholesterol, >40 mg/dL for men and >50 mg/dL for women; LDL-cholesterol, <100 mg/dL.

Results: One hundred ninety seven patients were studied. We identified two eating patterns: “unhealthy” (n = 100)—high consumption of refined carbohydrates, ultra-processed foods, sweets and desserts (P < 0.05); and “healthy” (n = 97)—high intake of whole carbohydrates, dairy, white meat, fish, fruits and vegetables (P < 0.05). The healthy group more frequently achieved therapeutic targets for fasting plasma glucose, HbA1c, and LDL-cholesterol than the unhealthy group. Poisson regression confirmed the association of healthy eating pattern with attaining the therapeutic target for fasting plasma glucose [PR, 1.59 (95% CI, 1.01 to 2.34); P = 0.018], HbA1c [PR, 2.09 (95% CI, 1.17 to 3.74); P = 0.013], and LDL-cholesterol [PR, 1.37 (95% CI, 1.01 to 1.86); P = 0.042].

Conclusions: A healthy eating pattern, including the frequent intake of whole carbohydrates, dairy, white meat, fish, fruits, and vegetables, is associated with reduced fasting plasma glucose, HbA1c, and LDL cholesterol levels in patients with type 2 diabetes.

Copyright © 2018 Endocrine Society

This article has been published under the terms of the Creative Commons Attribution Non-Commercial, No-Derivatives License (CC BY-NC-ND; https://creativecommons.org/licenses/by-nc-nd/4.0/).

Freeform/Key Words: eating patterns, glycemic profile, lipid profile, metabolic control, type 2 diabetes

Medical nutrition therapy is one of the cornerstones of diabetes management [1]. Evidence from prospective cohort studies and clinical trials has shown the importance of individual nutrients and foods for diabetes prevention and management [2, 3], but the overall effect of diet in achieving the recommended therapeutic targets has not been fully elucidated [1].

Eating patterns are defined as the quantities, proportions, variety, or combinations of different foods and beverages in diets, and the frequency with which they are habitually

Abbreviations: BMI, body mass index; CI, confidence interval; FFQ, food frequency questionnaire; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PR, prevalence ratio; UAE, urinary albumin excretion.
 consumed [4]. The identification of eating patterns can be useful to investigate the relationship between diet and disease, especially when more than one dietary component (nutrients or foods) seem to be involved, as in diabetes [5]. This evaluation can be analyzed in two ways: a priori, eating patterns are defined based on guidelines and nutritional recommendations, or a posteriori, when data from dietary surveys are aggregated through specific statistical analysis [6, 7].

Several eating patterns defined a priori such as Mediterranean, low glycemic index, moderately low carbohydrate, or vegetarian diets have been recommended for the management of weight and glucose control in diabetes [1, 8]. However, recently the American Diabetes Association stated that there is no single ideal dietary distribution of calories from carbohydrates, fats, and protein for diabetes patients [1]. In this context, the choice of eating pattern should be individualized, taking into account the patient’s current consumption preferences and the goal of metabolic targets [1, 9].

The aim of this cross-sectional study was to evaluate the relationship between eating patterns defined a posteriori and achieving recommended therapeutic targets (blood pressure, body weight, glycemic control, and lipid profile) in patients with type 2 diabetes in Southern Brazil.

1. Materials and Methods

A. Patients

The current study was conducted in patients with type 2 diabetes, defined as individuals >30 years of age at onset of diabetes, with no previous episode of ketoacidosis or documented ketonuria and who had not been using insulin in the 5 years since the diabetes was diagnosed [10]. The study recruited outpatients who consecutively attended the Endocrinology Division of the Hospital de Clínicas de Porto Alegre, Brazil.

The inclusion criteria were: age, <80 years; serum creatinine, <2.0 mg/dL; and body mass index (BMI), <40 kg/m². Patients on corticosteroid treatment or who had orthostatic hypotension or gastrointestinal symptoms suggestive of autonomic neuropathy were excluded. This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving patients were approved by the Ethics Committee of the Hospital de Clínicas de Porto Alegre, Brazil. Written informed consent was obtained from all patients.

B. Clinical and Laboratory Evaluation

Patients were submitted to clinical, laboratory, and lifestyle evaluation. Information about clinical data (comorbidities associated with diabetes and medication use) was collected from the patient’s most recent medical records. Blood pressure was measured twice (Omron HEM-705CP) according to international recommendations [11]. Increased urinary albumin excretion (UAE) was considered in the presence of UAE ≥ 14 mg/L in a random spot urine collection, or ≥30 mg in 24-hour collection, and the diagnosis was always confirmed [1, 12]. Patients were classified as current smokers or not (former and nonsmokers) and self-identified as white or nonwhite. Economic status was evaluated by a standardized Brazilian questionnaire [13], and physical activity level was classified according to the short version of the International Physical Activity Questionnaire [14] culturally adapted to the Brazilian population [15]. Physical activity was graded at three levels, that is, low, moderate, and high, according to activities during a typical week [14].

Blood samples were obtained after a 12-hour fast. Serum creatinine level was determined by a Jaffe reaction and estimated glomerular filtration rate by the Study Group and the Chronic Kidney Disease Epidemiology Collaboration Calculator. Plasma glucose was measured by a glucose oxidase method, hemoglobin A1c (HbA1c) was assessed by high-performance liquid chromatography (Tosoh 2.2 Plus HbA1c; Tosoh Corporation, Tokyo, Japan; reference values 4.8% to 6%), total cholesterol and triglycerides were measured by enzymatic-colorimetric
methods, and high-density lipoprotein (HDL) cholesterol was measured by a homogeneous direct method. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald’s formula, that is, LDL cholesterol = total cholesterol – HDL cholesterol – (triglycerides/5) [16] only for patients with triglyceride values < 400 mg/dL. UAE was measured by immunoturbidimetry [MicroAlb Sera-Pak immuno microalbuminuria (Bayer, Tarrytown, NY) on Cobas Mira Plus (Roche)].

C. Nutritional Evaluation: Anthropometric and Dietary Assessments

The body weight and height of patients (light clothing and without shoes) were obtained with measurements recorded to the nearest 100 g for weight and to the nearest 0.1 cm for height. BMI was then calculated. Waist circumference was measured at the midpoint between the iliac crest and the last floating rib. A flexible and nonstretch fiberglass tape was used for this measurement.

Information on food intake was collected from a quantitative food frequency questionnaire (FFQ) previously constructed [17] and validated [18] in patients from Southern Brazil. The FFQ consist of 98 food items and covered the past 12 months of food intake. Also, a portfolio with photographs of each food item and its portion sizes was used to support patients in identifying the consumed portion.

The intake report obtained by the FFQ was converted into daily consumption to estimate the nutritional composition [19–21]. The glycemic index and load were obtained from the international table [22]. When the glycemic index of foods present in the instruments was not found, we used data from food with a similar composition. Calculations were performed using the syntax of the SPSS version 20.0 program (SPSS, Chicago, IL).

D. Therapeutic Target Definitions

Patients were considered to be within the therapeutic target according to the following criteria: systolic/diastolic blood pressure, <140/90 mm Hg; BMI, <25 kg/m² or <27 kg/m² for the elderly [1, 23]; waist circumference, <94 cm for men and <80 cm for women [24]; fasting plasma glucose, <130 mg/dL; HbA1c values < 7%; serum triglycerides < 150 mg/dL; HDL cholesterol, >40 mg/dL for men and >50 mg/dL for women; and LDL cholesterol, <100 mg/ dL [1].

E. Statistical Analysis

The FFQ foods were aggregated into 18 groups and the amount consumed from each food group was converted into a percentage of total daily caloric intake. We performed a cluster analysis based on food groups to derive two nonoverlapping groups (eating patterns) using the K-means method. Median and interquartile range were calculated for each of the 18 food groups and compared by a Mann–Whitney U test for independent samples.

We examined the assumption of normality for all evaluated variables by a Kolmogorov–Smirnov test. A $\chi^2$ test, Student t test, and Mann–Whitney test for independent samples were used to test differences across the eating patterns. Energy and nutrient intake data were adjusted before analyses for energy intake according to the residual method [5].

To investigate the associations between eating patterns and achieve therapeutic targets we used Poisson regression with robust variance analysis. As the first step of the analysis, we estimated the effect of eating patterns on each therapeutic target (dependent variable). The second analysis (model 1) was adjusted for sex, age, economic status, current smoking, and diabetes duration. The third analysis (model 2) was additionally adjusted for diabetes treatment, physical activity, BMI, and energy intake. BMI was not included as covariate in the analysis of body weight and waist circumference targets (model 3).

Analyses were performed using SPSS version 20.0 (SPSS) and the type I error rate was fixed at $P \leq 0.05$ (2-tailed).
2. Results

Our sample consisted of 197 patients with type 2 diabetes: women, 63.5%; white, 70.6%; age, 62.5 ± 9.1 years; diabetes duration, 10 (5 to 19) years; BMI, 30.9 ± 4.3 kg/m²; presence of hypertension, 89.8%; presence of increased UAE, 39.1%; HbA1c, 8.5% ± 2.0%; and fasting plasma glucose, 164.7 ± 68.2 mg/dL.

We identified two eating patterns regarding quality of food groups consumed based on cluster analysis. The first cluster, defined as a “healthy” eating pattern (n = 97), had a high intake of whole carbohydrates, dairy, white meat, fish, fruits, and vegetables (P ≤ 0.05). The second cluster identified was defined as an “unhealthy” eating pattern (n = 100) and was characterized by high consumption of refined carbohydrates, ultraprocessed foods, sweets, and desserts (P < 0.05).

The medians and interquartile ranges of food group consumption (converted into a percentages of daily caloric intake) according to eating patterns are described in Table 1. We observed a median of 0 in two food groups, that is, alcoholic beverages and fish, although the fish consumption was different between groups: 67% of patients reported no consumption of fish in the unhealthy cluster and 49.5% in the healthy cluster (P = 0.01). Regarding consumption of alcoholic beverages, 59% of patients in the unhealthy cluster and 67% of patients in the healthy cluster reported no consumption (P = 0.24).

The nutrient intake according to eating pattern is shown in Table 2. Differences in nutrient intake were in accordance with results of cluster analyses. Patients from the healthy eating pattern had significantly lower energy, trans-unsaturated fatty acid, and sodium intakes than did those in the unhealthy eating pattern (P < 0.05). The healthy group consumed a diet with a lower index and glycemic load (P < 0.05) than did the unhealthy group. The intake of protein, total, soluble, and insoluble fiber, omega-3 fatty acid, calcium, magnesium, iron, potassium, and vitamin C were highest in patients from the healthy eating pattern (P < 0.05).

Clinical and laboratory characteristics of the eating patterns are shown in Table 3. Most clinical and laboratory features did not differ between groups, but there were more women in the healthy group (71.1% vs 56.0%; P = 0.038), were older (63.9 ± 9.1 years vs 61.1 ± 9.0 years; P = 0.028), and had lower fasting plasma glucose (150.2 ± 61.5 mg/dL vs 179.1 ± 71.1 mg/dL).

| Food Groups (% of Total Caloric Intake) | Eating Patterns |
|--------------------------------------|-----------------|
|                                      | Unhealthy (n = 100) | Healthy (n = 97) | P Value |
| Whole carbohydrates                  | 0.0 (0.0–2.4)     | 10.1 (3.5–17.5)  | 0.001   |
| Refined carbohydrates                | 32.3 (27.6–38.5)  | 14.9 (10.7–18.4) | 0.001   |
| Fried foods                          | 1.5 (0.1–5.2)     | 0.9 (0.0–4.3)    | 0.450   |
| Ultraprocessed foods                 | 2.7 (1.0–4.5)     | 1.4 (0.2–2.6)    | 0.001   |
| Dairy                                | 8.0 (3.9–11.7)    | 11.0 (7.4–16.1)  | 0.001   |
| Light and diet foods                 | 0.0 (0.0–1.3)     | 0.4 (0.0–3.1)    | 0.198   |
| Caffeinated beverages                | 0.9 (0.4–1.8)     | 1.0 (0.5–1.7)    | 0.919   |
| Alcoholic beverages                  | 0.0 (0.0–0.3)     | 0.0 (0.0–0.4)    | 0.633   |
| Sweets and desserts                  | 3.2 (0.5–7.2)     | 2.1 (0.3–4.7)    | 0.032   |
| Red meat                             | 10.0 (6.1–13.6)   | 11.4 (6.1–14.8)  | 0.217   |
| White meat                           | 4.3 (2.5–6.9)     | 5.6 (3.4–8.2)    | 0.009   |
| Fish                                 | 0.0 (0.0–0.1)     | 0.0 (0.0–1.4)    | 0.035   |
| Fruits                               | 12.4 (7.7–16.3)   | 16.7 (12.5–21.6) | 0.001   |
| Vegetables                           | 2.3 (1.5–3.6)     | 3.5 (2.5–5.7)    | 0.001   |
| Beans                                | 3.3 (1.7–4.9)     | 3.2 (1.5–4.5)    | 0.919   |
| Natural juices                       | 0.1 (0.0–1.4)     | 0.2 (0.0–1.5)    | 0.612   |
| Solid fats                           | 0.4 (0.0–1.5)     | 0.8 (0.0–1.5)    | 0.497   |
| Vegetable oils                       | 2.2 (1.3–4.9)     | 2.5 (0.6–4.6)    | 0.218   |

Data are expressed as median (interquartile range). P values were determined by a Mann–Whitney U test.
than did the unhealthy group. Men from the healthy group have a smaller waist circumference (102.7 ± 9.3 cm vs 107.9 ± 11.4 cm; P = 0.048) than do men from the unhealthy group.

Results comparing the proportion of patients who achieved therapeutic targets in healthy and unhealthy groups are depicted in Table 4. A larger proportion of patients who maintained a healthy eating pattern achieved fasting plasma glucose values < 130 mg/dL (47.4% vs 31.3%; P = 0.028), HbA1c < 7% (33.0% vs 17.0%; P = 0.013), and LDL cholesterol < 100 mg/dL (63.2% vs 46.6%; P = 0.034). There were no differences between groups in the evaluation of other therapeutic targets (blood pressure, BMI, waist circumference, HDL cholesterol, and triglycerides).

In the crude analysis of Poisson regression, it was observed that the healthy eating pattern was associated with achieving the therapeutic targets for fasting plasma glucose [prevalence ratio (PR), 1.51; 95% CI, 1.06 to 2.17], HbA1c (PR, 1.94; 95% CI, 1.16 to 3.26), and LDL cholesterol (PR, 1.36; 95% CI, 1.03 to 1.79). In model 1, these associations were confirmed for fasting plasma glucose (PR, 1.59; 95% CI, 1.01 to 2.34), and HbA1c (PR, 2.61; 95% CI, 1.51 to 4.53). In models 2 and 3, HbA1c (PR, 2.09; 95% CI, 1.17 to 3.74), and LDL cholesterol (PR, 1.37; 95% CI, 1.01 to 1.86) were the target variables associated with a healthy eating pattern (Table 4).

3. Discussion

In this cross-sectional study, we obtained data from 197 patients with type 2 diabetes and identified two eating patterns by cluster analysis. The healthy eating pattern, characterized

| Table 2. Daily Energy Intake, Macronutrients and Micronutrients, Fiber, Glycemic Index, and Glycemic Load in Patients With Type 2 Diabetes According to Eating Patterns |
|---------------------------------|-----------------|-----------------|--------|
| Nutrients                       | Unhealthy (n = 100) | Healthy (n = 100) | P Value |
| Energy, kcal                    | 2005.6 ± 788.5    | 1757.0 ± 649.3  | 0.017*
| Protein, g                      | 84.3 ± 19.0       | 94.1 ± 15.3     | 0.001*
| Carbohydrate, g                 | 268.8 ± 42.4      | 257.7 ± 42.8    | 0.068*
| Total fiber, g                  | 25.0 ± 7.1        | 30.7 ± 9.9      | 0.001*
| Soluble fiber, g                | 7.0 ± 2.1         | 8.4 ± 2.8       | 0.001*
| Insoluble fiber, g              | 16.8 ± 5.3        | 20.4 ± 7.5      | 0.001*
| Total lipids, g                 | 53.8 ± 13.4       | 56.4 ± 13.2     | 0.178*
| Saturated fatty acid, g         | 19.0 ± 5.7        | 20.2 ± 5.6      | 0.157*
| Monounsaturated fatty acid (g)  | 17.4 ± 4.9        | 18.3 ± 5.3      | 0.247*
| Polyunsaturated fatty acid, g   | 8.8 ± 3.5         | 9.4 ± 3.2       | 0.241*
| Omega-3 fatty acid, g           | 0.7 ± 0.3         | 0.8 ± 0.3       | 0.006*
| Omega-6 fatty acid, g           | 6.9 ± 3.1         | 7.4 ± 2.8       | 0.282*
| Trans-unsaturated fatty acid, g | 1.6 (1.1–2.4)     | 1.3 (1.0–1.7)   | 0.001*
| Cholesterol, mg                 | 248.5 ± 97.4      | 271.7 ± 88.4    | 0.082*
| Calcium, mg                     | 751.9 ± 302.7     | 992.8 ± 358.5   | 0.001*
| Magnesium, mg                   | 268.5 ± 54.9      | 333.3 ± 67.7    | 0.001*
| Iron, mg                        | 9.0 ± 2.2         | 10.2 ± 2.0      | 0.001*
| Sodium, mg                      | 1584.2 ± 472.4    | 1356.5 ± 341.1  | 0.001*
| Potassium, mg                   | 3124.9 ± 710.0    | 3738.3 ± 608.0  | 0.001*
| Vitamin C, mg                   | 190.6 (124.8–297.7) | 250.4 (195.3–350.7) | 0.001*
| Glycemic index, %               | 50.0 ± 5.6        | 43.7 ± 5.0      | 0.001*
| Glycemic load, g                | 134.3 ± 32.2      | 113.1 ± 24.7    | 0.001*

Data are expressed as means ± standard deviation or median (interquartile range).
*Student t-test for independent samples.
**Data adjusted for energy intake according to the residuals method.
***Mann–Whitney U test.
by high consumption of whole carbohydrates, dairy, white meat, fish, fruits, and vegetables, was associated with better glycemic and lipid control than the unhealthy eating pattern. Patients in the healthy eating pattern had lower fasting plasma glucose, HbA1c, and LDL cholesterol and most frequently reached the recommended therapeutic targets for these parameters as compared with patients from the unhealthy eating pattern. As expected, patients in the healthy group had a higher intake of protein, total, soluble, and insoluble fiber, omega-3 fatty acid, calcium, magnesium, iron, potassium, and vitamin C. Moreover, the association between the healthy eating pattern and achieving the therapeutic targets for fasting plasma glucose, HbA1c, and LDL cholesterol remained, even when potential confounding factors were taken into account as demonstrated by regression analyses.

It is known that carbohydrates are the nutrients that most affect blood glucose levels. However, up to now there is no consensus evidence about the ideal amount of carbohydrate

Table 3. Clinical and Laboratory Characteristics of Patients With Type 2 Diabetes According to Eating Patterns

| Characteristics                     | Unhealthy (n = 100) | Healthy (n = 97) | P Value |
|-------------------------------------|---------------------|------------------|---------|
| Females                             | 56 (56.0)           | 69 (71.1)        | 0.038a  |
| Age, y                              | 61.1 ± 9.0          | 63.9 ± 9.1       | 0.028b  |
| Whites                              | 65 (65.0)           | 74 (76.3)        | 0.088a  |
| Years of study                      | 6.5 (4.0–11.0)      | 6.0 (4.0–11.0)   | 0.729a  |
| Economic status: middle class       | 43 (45.3)           | 48 (51.7)        | 0.449a  |
| Current smoking                     | 20 (20.0)           | 8 (8.2)          | 0.060a  |
| Physical activity: low level        | 59 (60.8)           | 61 (64.9)        | 0.568a  |
| Diabetes duration, y                | 10.0 (4.0–17.7)     | 10.0 (5.0–19.5)  | 0.635b  |
| Hypertension                        | 89 (89.0)           | 88 (90.7)        | 0.815a  |
| Systolic blood pressure, mmHg       | 143.3 ± 26.2        | 140.3 ± 17.8     | 0.351b  |
| Diastolic blood pressure, mmHg      | 78.3 ± 13.1         | 76.9 ± 10.0      | 0.403b  |
| Increased UAE                       | 41 (41.0)           | 31 (31.9)        | 0.228b  |
| Diabetes treatment                  |                     |                  |         |
| Diet                                | 1 (1.0)             | 4 (4.1)          | 0.350c  |
| Oral hypoglycemic drugs             | 42 (42.0)           | 46 (47.4)        |         |
| Insulin and oral hypoglycemic drugs | 50 (50.0)           | 43 (44.3)        |         |
| Antihypertensive drugs, number      | 2.0 (1.0–4.0)       | 2.0 (2.0–3.0)    | 0.892c  |
| Use of ACE inhibitor                | 68 (68.0)           | 56 (57.7)        | 0.143d  |
| Use of lipid-lowering drugs         | 71 (71.0)           | 64 (66.0)        | 0.448d  |
| Previous cardiovascular event       | 31 (31.0)           | 25 (25.8)        | 0.384d  |
| BMI, kg/m²                          | 31.4 ± 4.6          | 30.4 ± 3.9       | 0.098b  |
| Waist circumference, cm             |                     |                  |         |
| Male                                | 107.9 ± 11.4        | 102.7 ± 9.3      | 0.048b  |
| Female                              | 103.6 ± 11.1        | 102.2 ± 8.6      | 0.442b  |
| Fasting plasma glucose, mg/dL       | 179.0 ± 71.1        | 150.2 ± 61.5     | 0.003c  |
| HbA1c, %                            | 8.7 ± 2.0           | 8.3 ± 2.0        | 0.230b  |
| Total cholesterol, mg/dL            | 179.1 ± 37.1        | 171.4 ± 41.8     | 0.195b  |
| HDL cholesterol, mg/dL              |                     |                  |         |
| Male                                | 40.3 ± 11.1         | 39.5 ± 9.6       | 0.786b  |
| Female                              | 43.6 ± 9.0          | 43.8 ± 12.0      | 0.907b  |
| LDL cholesterol, mg/dL              | 105.0 ± 32.9        | 97.3 ± 34.4      | 0.131b  |
| Triglyceride, mg/dL                 | 150.0 (106.0–198.5) | 131.0 (98.0–197.0)| 0.405b |
| Serum creatinine, mg/dL             | 0.9 ± 0.3           | 0.8 ± 0.3        | 0.806b  |
| GFR, mL/min/1.73 m²                 | 84.9 ± 18.5         | 81.5 ± 21.4      | 0.247b  |
| UAE, mg/dL                          | 11.1 (3.8–48.1)     | 5.6 (3.0–27.0)   | 0.070b  |

Data are expressed as means ± standard deviation, median (interquartile range), or number of patients with the analyzed characteristic (%).

Abbreviation: GFR, glomerular filtration rate.

aX² Test.
bStudent t test.
cMann–Whitney U test.
intake for people with diabetes [1, 9]. In fact, in the present study, the carbohydrate consumption did not differ between the unhealthy and healthy group. The association between healthy eating pattern and glycemic control could be better explained by the quality of carbohydrate intake than the amount of this macronutrient. In agreement with this, we demonstrated a higher consumption of whole carbohydrates, fruits, and vegetables in this group of patients. As a consequence, these patients consumed diets with a lower glycemic index and glycemic load values as compared with patients in the unhealthy eating pattern. Currently, diets with a low glycemic index have been associated with improved glycemic control [25].

Another nutrient probably related to the best observed glycemic control in our study is dietary fiber. Accordingly, in our patients in the healthy eating pattern, a higher total,

### Table 4. Proportion of Patients With Type 2 Diabetes Who Achieve Therapeutic Targets According to Eating Patterns

| Therapeutic Targets                  | Unhealthy (n = 100) | Healthy (n = 97) | P value |
|--------------------------------------|---------------------|-----------------|---------|
| Blood pressure, n (%)                | 50 (50.5)           | 49 (52.1)       | 0.822*  |
| PR (95% CI)                          | 1                   | 1.03 (0.78–1.36) | 0.822   |
| PR adjusted^b (95% CI)               | 1                   | 1.08 (0.80–1.45) | 0.628   |
| PR adjusted^c (95% CI)               | 1                   | 1.07 (0.78–1.47) | 0.663   |
| BMI, n (%)                           | 16 (16.0)           | 14 (14.4)       | 0.844^a |
| PR (95% CI)                          | 1                   | 0.90 (0.47–1.75) | 0.760   |
| PR adjusted^b (95% CI)               | 1                   | 1.08 (0.48–2.44) | 0.844   |
| PR adjusted^c (95% CI)               | 1                   | 1.07 (0.49–2.36) | 0.859   |
| Waist circumference, n (%)           | 8 (8.0)             | 6 (6.2)         | 0.783^a |
| PR (95% CI)                          | 1                   | 0.77 (0.28–2.15) | 0.621   |
| PR adjusted^b (95% CI)               | 1                   | 1.40 (0.47–4.17) | 0.551   |
| PR adjusted^c (95% CI)               | 1                   | 0.93 (0.26–3.27) | 0.905   |
| Fasting plasma glucose, n (%)        | 31 (31.3)           | 46 (47.4)       | 0.028*  |
| PR (95% CI)                          | 1                   | 1.51 (1.06–2.17) | 0.024   |
| PR adjusted^b (95% CI)               | 1                   | 1.59 (1.01–2.34) | 0.018   |
| PR adjusted^c (95% CI)               | 1                   | 1.47 (0.98–2.19) | 0.060   |
| HbA1c, n (%)                         | 17 (17.0)           | 32 (33.0)       | 0.013^a |
| PR (95% CI)                          | 1                   | 1.94 (1.16–3.26) | 0.012   |
| PR adjusted^b (95% CI)               | 1                   | 2.61 (1.51–4.53) | 0.001   |
| PR adjusted^c (95% CI)               | 1                   | 2.09 (1.17–3.74) | 0.013   |
| Triglycerides, n (%)                 | 43 (48.3)           | 48 (55.2)       | 0.371^a |
| PR (95% CI)                          | 1                   | 1.14 (0.86–1.52) | 0.364   |
| PR adjusted^b (95% CI)               | 1                   | 1.15 (0.86–1.53) | 0.344   |
| PR adjusted^c (95% CI)               | 1                   | 1.11 (0.82–1.50) | 0.501   |
| HDL cholesterol, n (%)               | 27 (30.3)           | 25 (28.4)       | 0.869^a |
| PR (95% CI)                          | 1                   | 0.94 (0.59–1.48) | 0.778   |
| PR adjusted^b (95% CI)               | 1                   | 0.91 (0.59–1.40) | 0.663   |
| PR adjusted^c (95% CI)               | 1                   | 0.81 (0.51–1.30) | 0.387   |
| LDL cholesterol, n (%)               | 41 (46.6)           | 55 (65.2)       | 0.034^a |
| PR (95% CI)                          | 1                   | 1.36 (1.03–1.79) | 0.030   |
| PR adjusted^b (95% CI)               | 1                   | 1.32 (1.00–1.74) | 0.052   |
| PR adjusted^c (95% CI)               | 1                   | 1.37 (1.01–1.86) | 0.042   |

Data are expressed as number of patients with analyzed characteristic (%) and as the PR (95% CI). Therapeutic target definitions are: blood pressure, <140/90 mm Hg; BMI, <25 kg/m² or <27 kg/m² for the elderly; waist circumference, <94 cm for men and <80 cm for women; fasting plasma glucose, <130 mg/dL; HbA1c, <7%; serum triglycerides, <150 mg/dL; HDL cholesterol, >40 mg/dL for men and >50 mg/dL for women; LDL cholesterol, <100 mg/dL.

^aX² Test.

^bModel 1: adjusted for sex, age, economic status, current smoking, and diabetes duration.

^cModel 3: adjusted for sex, age, economic status, current smoking, diabetes duration, diabetes treatment, physical activity, and energy intake.

^dModel 2: adjusted for sex, age, economic status, current smoking, diabetes duration, diabetes treatment, physical activity, BMI, and energy intake.
soluble, and insoluble fiber consumption was observed. It has already been demonstrated that a high fiber intake was associated with better glycemic control in patients with diabetes [26, 27]. However, up to now, the beneficial effects of fiber intake, especially soluble fibers, could not be isolated from the effects of glycemic index and glycemic load because most foods that have a low glycemic index also have a high fiber content [8].

Alternatively, the better lipid profile observed in patients in the healthy eating pattern, as compared with the unhealthy eating pattern, was, at least partially, due to dietary fiber content. A beneficial fiber effect on the lipid profile [28] with reduction of total and LDL cholesterol and triglycerides [29, 30] had already been previously established. In our study, a higher proportion of patients in the healthy group (rich in fibers) had LDL cholesterol <100 mg/dL as compared with patients in the unhealthy group. This result could not be explained by lipid-lowering drugs because the frequency of drug users was not different in healthy and unhealthy groups, nor were BMI and the level of physical activity.

Fat consumption, along with fiber intake, could have influenced the improvement of LDL cholesterol in a healthy eating pattern. The dietary cholesterol and the saturated fatty acid intake did not differ between healthy and unhealthy groups. However, the trans-unsaturated fatty acids intake was lower in patients in the healthy group. In fact, this dietary component was already associated with high LDL cholesterol levels [31].

Although our study has a cross-section design that allowed us to describe only possible associations, it is worthwhile observing that the healthy eating pattern identified in the present study presents similarities with the Dietary Approaches to Stop Hypertension diet, which is an a priori eating pattern characterized by high consumption of vegetables, fruits, low-fat dairy products, whole grains, poultry, and fish and is low in sweets and desserts [32, 33]. In fact, the beneficial effect of this dietary pattern has already been demonstrated in short-term trials in patients with diabetes [34–36].

The association between eating patterns defined a posteriori and health outcomes in individuals with diabetes has been studied more recently in different countries. However, most of these studies, different from ours, used factor analysis to determine eating patterns [37–44]. To our knowledge, our study was the first to use cluster analysis, a method that creates patterns that are mutually exclusive (i.e., categorical variables) and that are defined by maximizing differences in mean intake of food groups [6]. Cluster analysis findings are easier to interpret because an individual is in one cluster only, outcomes are specific to individuals within each cluster, and each cluster has a specific food and nutrient composition [45].

Moreover, in our study, some methodological precautions were also taken into account. We used a food frequency questionnaire previously constructed [17] and validated [18] in patients from Southern Brazil, and the macronutrient and micronutrient data were adjusted for energy using the residual method [5]. Also, the sample size we used to analyze a food consumption tool was appropriately calculated [46]. We included 10 individuals for each food group studied (18 food groups studied and 180 subjects).

A possible limitation of our study was the absence of an actual sodium intake estimate. We used the intrinsic sodium of foods derived from a table [19] instead of measurements of 24-hour urinary sodium, a more accurate evaluation of salt consumption [47]. Finally, as expected, the adopted cross-sectional design hinders any causal inferences. The associations of healthy eating patterns as described in our study should be evaluated in different samples of patients with diabetes, in long-term cohorts, and, ideally, in randomized clinical trials. The recommendation of a healthy eating pattern, instead of prescribing allowed or forbidden foods, should be tested as a useful dietary strategy for patients with diabetes.

In conclusion, in patients with type 2 diabetes a healthy eating pattern including the frequent intake of whole carbohydrates, dairy, white meat, fish, fruits and vegetables was associated with lower fasting plasma glucose, HbA1c, and LDL cholesterol levels as compared with an eating pattern with high consumption of refined carbohydrates, ultraprocessed foods, sweets, and desserts.
Acknowledgments

R.A.S., J.P.A., I.L.M., B.B.N., and J.C.A. are deeply grateful to the mentorship of Dr. Mirela Jobim de Azevedo, who died on 15 May 2017. She will be sadly missed, but her enduring and substantial legacy will remain intensely alive.

**Financial Support:** This study was partially supported by the Fundo de Incentivo à Pesquisa e Eventos—Hospital de Clinicas de Porto Alegre. R.A.S., J.P.A., and B.B.N were recipients of scholarships from the Fundação de Aperfeiçoamento de Pessoal de Nível Superior, and I.L.M. received a scholarship from the Programa Institucional de Bolsas de Iniciação Científica–Conselho Nacional de Desenvolvimento Científico e Tecnológico.

**Correspondence:** Jussara Carnevale de Almeida, Serviço de Endocrinologia do Hospital de Clinicas de Porto Alegre, Rua Ramiro Barcelos 2350, Prédio 12, 4º Andar, 90035-003 Porto Alegre, Rio Grande do Sul, Brazil. E-mail: jcalmeida@hcpa.edu.br.

**Disclosure Summary:** The authors have nothing to disclose.

References and Notes

1. American Diabetes Association. Standards of medical care in diabetes—2017. Diabetes Care. 2017; 40(Suppl 1):S4–S5.
2. Wheeler ML, Dunbar SA, Jaacks LM, Karmally W, Mayer-Davis EJ, Wylie-Rosett J, Yancy WS, Jr. Macronutrients, food groups, and eating patterns in the management of diabetes: a systematic review of the literature. 2010. Diabetes Care. 2012;35(2):434–445.
3. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr. 2013;97(3):505–516.
4. United States Department of Agriculture. Scientific report of the Dietary Guidelines Advisory Committee. Available at: https://health.gov/dietaryguidelines/2015-scientific-report/PDFs/Scientific-Report-of-the-2015-Dietary-Guidelines-Advisory-Committee.pdf. Accessed 22 November 2016.
5. Willett WC. *Nutritional Epidemiology*. Oxford, U.K.: Oxford University Press; 1998.
6. Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. Nutr Rev. 2004;62(5):197–203.
7. Ocké MC. Evaluation of methodologies for assessing the overall diet: dietary quality scores and dietary pattern analysis. Proc Nutr Soc. 2013;72(2):191–199.
8. Ley SH, Hamdy O, Mohan V, Hu FB. Prevention and management of type 2 diabetes: dietary components and nutritional strategies. Lancet. 2014;383(9933):1999–2007.
9. Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, Neumiller JJ, Nwankwo R, Verdi CL, Urbanski P, Yancy WS, Jr; American Diabetes Association. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care. 2013;36(11):3821–3842.
10. World Health Organization. *Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications. Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva, Switzerland: World Health Organization; 2003.
11. O’Brien E, Waebber B, Parati G, Staessen J, Myers MG. Blood pressure measuring devices: recommendations of the European Society of Hypertension. BMJ. 2001;322(7285):531–536.
12. Viana LV, Gross JL, Camargo JL, Zelmanovitz T, da Costa Rocha EP, Azevedo MJ. Prediction of cardiovascular events, diabetic nephropathy, and mortality by albumin concentration in a spot urine sample in patients with type 2 diabetes. J Diabetes Complications. 2012;26(5):407–412.
13. Associação Brasileira das Empresas de Pesquisa. Available at: www.abep.org. Accessed 1 March 2009.
14. International Physical Activity Questionnaire. Available at: https://sites.google.com/site/theipaq/scoring-protocol. Accessed 1 October 2009.
15. Hallal PC, Matsudo SM, Matsudo VKR, Araújo TL, Andrade DR, Bertoldi AD. Physical activity in adults from two Brazilian areas: similarities and differences. Cad Saude Publica. 2005;21(2):573–580.
16. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499–502.
17. Sarmento RA, Riboldi BP, da Costa Rodrigues T, de Azevedo MJ, de Almeida JC. Development of a quantitative food frequency questionnaire for Brazilian patients with type 2 diabetes. BMC Public Health. 2013;13:740.
18. Sarmento RA, Antonio JP, Riboldi BP, Montenegro KR, Friedman R, Azevedo MJ, Almeida JC. Reproducibility and validity of a quantitative FFQ designed for patients with type 2 diabetes from Southern Brazil. Public Health Nutr. 2014;17(10):2237–2245.
19. Lima DM. *Tabela de Composição dos Alimentos—TACO. Versão II, 2a. Edição*. Campinas, São Paulo, Brazil: NEPA–UNICAMP; 2006.

20. Philippi ST. *Tabela de Composição de Alimentos: Suporte para Decisão Nutricional*. São Paulo, Brazil: Universidade de São Paulo; 2002.

21. Rizek RL, Hepburn FN, Perloff BP. *Composition of Foods: Agriculture Handbook no 8*. Washington, DC: United States Department of Agriculture; 2006.

22. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. *Diabetes Care*. 2008;31(12):2281–2283.

23. Lipschitz DA. Screening for nutritional status in the elderly. *Prim Care*. 1994;21(1):55–67.

24. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, Fruchart JC, James WP, Loria CM, Smith SC, Jr; International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; International Association for the Study of Obesity. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*. 2009;120(16):1640–1645.

25. Wang Q, Xia W, Zhao Z, Zhang H. Effects comparison between low glycemic index diets and high glycemic index diets on HbA1c and fructosamine for patients with diabetes: a systematic review and meta-analysis. *Prim Care Diabetes*. 2015;9(5):362–369.

26. Post RE, Mainous AG III, King DE, Simpson KN. Dietary fiber for the treatment of type 2 diabetes mellitus: a meta-analysis. *J Am Board Fam Med*. 2012;25(1):16–23.

27. Silva FM, Kramer CK, de Almeida JC, Steemeburgo T, Gross JL, Azevedo MJ. Fiber intake and glycemic control in patients with type 2 diabetes mellitus: a systematic review with meta-analysis of randomized controlled trials. *Nutr Rev*. 2013;71(12):790–801.

28. Venn BJ, Mann JI. Cereal grains, legumes and diabetes. *Eur J Clin Nutr*. 2004;58(11):1443–1461.

29. Brown L, Rosner B, Willett WW, Sacks FM. Cholesterol-lowering effects of dietary fiber: a meta-analysis. *Am J Clin Nutr*. 1999;69(1):30–42.

30. Anderson JW, Randles KM, Kendall CW, Jenkins DJ. Carbohydrate and fiber recommendations for individuals with diabetes: a quantitative assessment and meta-analysis of the evidence. *J Am Coll Nutr*. 2004;23(5):5–17.

31. Garshick M, Molchari-Greenberger H, Mosca L. Reduction in dietary trans fat intake is associated with decreased LDL particle number in a primary prevention population. *Nutr Metab Cardiovasc Dis*. 2014;24(1):100–106.

32. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH, Karanja N; DASH Collaborative Research Group. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997;336(16):1117–1124.

33. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER III, Simons-Morton DG, Karanja N, Lin PH; DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med*. 2001;344(1):3–10.

34. Azadbakht L, Fard NR, Karimi M, Baghaei MH, Surkan PJ, Rahimi M, EsmailiZadeh A, Willett WC. Effects of the Dietary Approaches to Stop Hypertension (DASH) eating plan on cardiovascular risks among type 2 diabetic patients: a randomized crossover clinical trial. *Diabetes Care*. 2011;34(1):55–57.

35. Azadbakht L, Surkan PJ, Esmailizadeh A, Willett WC. The Dietary Approaches to Stop Hypertension eating plan affects C-reactive protein, coagulation abnormalities, and hepatic function tests among type 2 diabetic patients. *J Nutr*. 2011;141(6):1083–1088.

36. Paula TP, Viana LV, Neto AT, Leitão CB, Gross JL, Azevedo MJ. Effects of the DASH diet and walking on blood pressure in patients with type 2 diabetes and uncontrolled hypertension: a randomized controlled trial. *J Clin Hypertens (Greenwich)*. 2015;17(11):895–901.

37. Lim JH, Lee YS, Chang HC, Moon MK, Song Y. Association between dietary patterns and blood lipid profiles in Korean adults with type 2 diabetes. *J Korean Med Sci*. 2011;26(9):1201–1208.

38. Iimuro S, Yoshimura Y, Umegaki H, Sakurai T, Araki A, Ohashi Y, Iijima K, Ito H; Japanese Elderly Diabetes Intervention Trial Study Group. Dietary pattern and mortality in Japanese elderly patients with type 2 diabetes mellitus: does a vegetable- and fish-rich diet improve mortality? An explanatory study. *Geriatr Gerontol Int*. 2012;12(Suppl 1):59–67.

39. Hsu CC, Jhang HR, Chang WT, Lin CH, Shin SJ, Hwang SJ, Huang MC. Associations between dietary patterns and kidney function indicators in type 2 diabetes. *Clin Nutr*. 2014;33(1):98–105.
40. Darani Zad N, Mohd Yusof R, Esmaili H, Jamaluddin R, Mohseni F. Association of dietary pattern with biochemical blood profiles and bodyweight among adults with type 2 diabetes mellitus in Tehran, Iran. J Diabetes Metab Disord. 2015;14(1):28.

41. Ghane Basiri M, Sotoudeh G, Djalali M, Reza Eshraghian M, Noorshahi N, Rafiee M, Nikbazm R, Karimi Z, Koohdani F. Association of major dietary patterns with general and abdominal obesity in Iranian patients with type 2 diabetes mellitus. Int J Vitam Nutr Res. 2015;85(3–4):145–155.

42. Shi Z, Zhen S, Zimmet PZ, Zhou Y, Zhou Y, Magliano DJ, Taylor AW. Association of impaired fasting glucose, diabetes and dietary patterns with mortality: a 10-year follow-up cohort in Eastern China. Acta Diabetol. 2016;53(5):799–806.

43. Osonoi Y, Mita T, Osonoi T, Suito M, Tamasawa A, Nakayama S, Someya Y, Ishida H, Kanazawa A, Gosho M, Fujitani Y, Watada H. Relationship between dietary patterns and risk factors for cardiovascular disease in patients with type 2 diabetes mellitus: a cross-sectional study. Nutr J. 2016;15(1):15.

44. Mathe N, Pisa PT, Johnson JA, Johnson ST. Dietary patterns in adults with type 2 diabetes predict cardiometabolic risk factors. Can J Diabetes. 2016;40(4):296–303.

45. Newby PK, Muller D, Tucker KL. Associations of empirically derived eating patterns with plasma lipid biomarkers: a comparison of factor and cluster analysis methods. Am J Clin Nutr. 2004;80(3):759–767.

46. Kac G, Sichieri R, Gigante DP. Epidemiologia Nutricional. Rio de Janeiro, Brazil: Fiocruz e Atheneu; 2007.

47. World Health Organization. The SHAKE technical package for salt reduction. Available at: http://apps.who.int/iris/bitstream/10665/250135/1/9789241511346-eng.pdf?ua=1. Accessed 1 November 2016.