Can J Diabetes xxx (2021) 1–5

Contents lists available at ScienceDirect

Canadian Journal of Diabetes

journal homepage: www.canadianjournalofdiabetes.com

Perspectives in Practice

# A Low-Carbohydrate, High-Fat Ketogenic Diet Program Implemented by an Interdisciplinary Primary Care Team Improves Markers of Cardiometabolic Health in Adults With Type 2 Diabetes: A Retrospective Secondary Analysis

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**Key Messages**

- A community-based ketogenic diet program improves glycated hemoglobin alongside reductions in use of glucose-lowering medication.
- A model for physician-led multidisciplinary diet programs shows promise in type 2 diabetes disease management.
- Future programs should focus on continuity of patient supports and education to maximize adherence to dietary changes.

ARTICLE INFO

*Article history:*
Received 17 May 2021
Received in revised form
30 July 2021
Accepted 1 September 2021

## Introduction

The prevalence of type 2 diabetes (T2D) is rising in Canada, with approximately 29% of Canadians living with diagnosed diabetes, undiagnosed T2D or prediabetes (1). Growing evidence suggests that dietary carbohydrate restriction (defined as daily intake <130 g or <26% energy from carbohydrates) can improve T2D outcomes (2). Compared with usual care, low-carbohydrate diets have a superior glycated hemoglobin (A1C)-lowering effect, and result in improved glucose stability despite simultaneous glucose-lowering medication de-prescription (2,3).

Family physicians are the primary access point for care for most Canadians living with T2D (4). Therefore, effective physician-led programs are attractive for improving T2D outcomes for patients. Diabetes Canada emphasizes the importance of a multidisciplinary team that focusses on communication and coordination between providers, patient education and increased availability of supports for patients (4). Indeed, Canadians with T2D enrolled in multidisciplinary primary care networks report better health outcomes than those receiving care exclusively from physicians (5,6). Diabetes Canada has recently acknowledged the efficacy of low-carbohydrate diets for T2D management (7), yet few practical examples have been provided on how to implement this effectively within primary care.

The purpose of this retrospective secondary analysis was to determine the impact of a low-carbohydrate, high-fat (LCHF) ketogenic diet program utilizing a physician-led, multidisciplinary care model in a rural community setting on clinical outcomes in patients with T2D.

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https://doi.org/10.1016/j.jcjd.2021.09.001

## Methods

### Ethics

A 3-month, medically supervised LCHF ketogenic diet program for patients with T2D was implemented with follow ups at 6 and 12 months. This program was delivered by an internal medicine specialist, a family physician, a registered dietitian and a registered nurse from a clinical practice in rural Nova Scotia, Canada. The program was not designed as a research study; however, after completion, ethical approval for the retrospective secondary analysis of the anonymized medical records was obtained from the clinical research ethics board of the University of British Columbia (H20-02228).

### Participant recruitment and eligibility

Patients accepted into the program were 18 to 75 years of age and had diagnosed T2D, blood pressure <150/90 mmHg, body mass index of >28 kg/m$^2$, estimated glomerular filtration rate of >45 mL/min per 1.73 m$^2$ and no history of recent stroke or heart attack. Eligible patients with internet access who were willing to engage in health-related group discussion about topics were referred to the program by local physicians. Of the 58 referrals received, 50 met the program criteria and were invited to enroll. Of these, 35 began the program (3 patients declined to participate, 9 did not attend both the mandatory consultation and information sessions and 3 had additional safety and medical concerns identified). Eight patients dropped out in the first 4 weeks, leaving 27 patients who completed the 3-month intensive program and presented at the 6-month follow up. Two patients were lost to follow up at 12 months.

### Program consultation and supports

Patients underwent a 45-minute comprehensive medical consultation with an internal medicine physician with the focus on optimization and guiding de-escalation of glucose-lowering medications. Patients had a short (5 to 10 minutes) consultation with the internist or family physician before the group meetings (held at 2, 4, 8 and 12 weeks) to focus on medication adjustments. Blood pressure, anthropometrics and laboratory data were recorded by a nurse at baseline and every 3 months at these consultations. Complex patients (e.g. multidose injections, complex antihypertensive regimens, congestive heart failure, frailty) were identified at the initial consultation and monitored closely during the first few weeks of intervention, often via brief but frequent phone or e-mail interactions, to assist with medication adjustments.

Patients attended group information sessions at 2, 4, 8 and 12 weeks at a local community health centre. These sessions were led by a registered dietitian and supported by a nurse, internal medicine specialist and family physician (see Supplementary Table 1 for topics covered). Sessions were designed to be informative, build community within the group and facilitate peer support for patients. Additional supports included a printed guide, a private social media page and relevant online resources. Patients had the option to attend a salad-making class and a grocery store tour with the program dietitian to further assist in following and sustaining the diet.

### LCHF diet

The LCHF ketogenic diet program focussed on consuming whole foods, ≤20 g/day of net carbohydrates, achieving a moderate protein intake and consuming fats to satiety. Counting of carbohydrates (rather than calories) was encouraged by using a traffic-light categorization system of “foods to enjoy freely” (e.g. green/fibrous vegetables, fish, unprocessed meat), “foods to enjoy in moderation” (e.g. cheese, processed meats, nuts/seeds, berries) and “foods to avoid” (e.g. grains, starchy vegetables). A sample 2-day menu is provided in Supplementary Table 2.

### Medication de-prescription protocol

De-prescription of glucose-lowering and antihypertensive medications was monitored closely and adapted to each patient’s medical context, as described in detail in the Supplementary Appendix.

### Clinical measures

Clinical improvement of T2D was assessed by A1C and glucose-lowering medication use as quantified by the medication effect score (MES). MES reflects the overall intensity of a diabetes medication regimen and is based on medication doses and their efficacy in reducing blood glucose (8). The percent of patients achieving an A1C of <6.5% and taking no glucose-lowering medications or metformin only was calculated as an exploratory outcome (9). Improvements in cardiovascular risk factors were assessed by improvement of dyslipidemia and blood pressure. Markers of potential hepatic steatosis were assessed by aspartate aminotransferase and alanine transaminase, and the hepatic steatosis index was calculated (10). Kidney function was assessed by serum creatinine and estimated glomerular filtration rate.

### Statistical analysis

All analyses were performed using R software (R Foundation for Statistical Computing, Vienna, Austria) (11). Continuous outcomes were analyzed using mixed-effects linear models (12) with fixed effects for time point (baseline, 3, 6 or 12 months) and insulin use at baseline (yes or no) and a random effect for participant. Model specification was assessed visually using normal probability plots and residuals vs fitted values plots. When the behaviour of the model residuals warranted a log transformation, effect estimates and 95% confidence intervals (CIs) were backtransformed to ratio (percentage) differences using the emmeans package (13). When a log transformation could not be used, nonparametric bootstrap analyses were performed with 2,000 resamples with replacement and bias corrected, and accelerated 95% CIs were calculated (14). The difference in the proportion of participants achieving the exploratory outcome of A1C <6.5% and no glucose-lowering medication use (except for metformin) and the associated 95% CIs between the baseline and 3-, 6- and 12-month time points were compared using exact McNemar’s tests (15,16).

## Results

Patients’ baseline characteristics are reported in Table 1.

### Markers of T2D outcomes

A1C was reduced from baseline at 3 months (−0.8%; 95% CI, −1.1 to −0.5; p<0.0001), 6 months (−0.8%; 95% CI, −1.1 to −0.4; p<0.0001) and 12 months (−0.4%; 95% CI, −0.7 to 0.0; p=0.042; Supplementary Figure 1). Glucose-lowering medication changes at 3, 6 and 12 months compared with baseline are presented in Supplementary Figure 2A–C, and glucose-lowering medication changes across time are presented in Supplementary Figure 2D. MES was consistently reduced at all time points, and the proportion of those with an A1C of <6.5% and no glucose-lowering

**Table 1**
Patients' characteristics at baseline (N=27)

| Characteristic | Value |
|---|---|
| Age, years | 61 (8) |
| Males/females | 14/13 |
| Body mass index, kg/m$^2$ | 36.5 (6.2) |
| Waist circumference, cm | 118.4 (12.2) |
| Type 2 diabetes duration, years | 12 (8) |
| Glucose-lowering medications | 2.6 (1.2) |
| Number on insulin therapy | 9 |
| Duration of insulin therapy, years | 11 (3) |
| Systolic blood pressure, mmHg | 139 (18) |
| Diastolic blood pressure, mmHg | 78 (9) |
| Hemoglobin A1C, % | 7.4 (1.1) |
| Total cholesterol, mmol/L | 4.29 (0.91) |
| HDL-C, mmol/L | 1.20 (0.36) |
| LDL-C, mmol/L | 2.25 (0.76) |
| Triglycerides, mmol/L | 1.82 (0.71) |
| Proportion with macrovascular complications, % | 22 |
| Proportion with microvascular complications, % | |
| ≥1 microvascular complication | 33 |
| Nephropathy | 33 |
| Retinopathy | 0 |
| Neuropathy | 7 |

*A1C*, glycated hemoglobin; *HDL-C*, high-density lipoprotein cholesterol; *LDL-C*, low-density lipoprotein cholesterol.
*Note:* Data expressed as mean (standard deviation) or as number, unless noted otherwise.

medications or metformin only compared with baseline was significantly increased at 6 months only (Table 2).

### *Weight loss*

All measures of weight loss are reported in Table 2. Body mass index and waist circumference were significantly reduced at 3, 6 and 12 months (all $p<0.05$). A significant percentage of initial body mass was also lost at all time points (all $p<0.05$).

### *Blood pressure, blood lipids and liver function*

All measures of blood pressure, blood lipids and liver function are reported in Table 2. Systolic blood pressure was significantly reduced at 3, 6 and 12 months (all $p<0.05$), whereas diastolic blood pressure was only significantly reduced at 6 months ($p<0.05$). Total cholesterol and low-density lipoprotein cholesterol were unchanged throughout the 12 months; however, high-density lipoprotein cholesterol was significantly increased at 3, 6 and 12 months, whereas triglycerides were significantly decreased at all time points (all $p<0.05$). Antihypertensive and lipid-lowering medications are reported in Supplementary Table 3. Liver function, as assessed by the hepatic steatosis index, improved significantly at each of the time points (all $p<0.05$).

## Discussion

The utility of carbohydrate restriction for improving glycemic control has been documented in experimental trials (2), but examples of successful implementation options for physician-led programs in primary care are limited. Herein we have reported a small, retrospective medical chart analysis of outcomes from an LCHF ketogenic diet program employing a multidisciplinary care model in a community setting in Canada. Glycemic control improved alongside robust reductions in glycemic-lowering medications. Furthermore, clinically relevant improvements in body weight, blood pressure, high-density lipoprotein cholesterol and liver function were seen up to 12 months.

Two key markers of T2D management, A1C and MES, improved significantly across all time points compared with baseline. In addition, the proportion of patients with an A1C of <6.5% and no glucose-lowering medications or metformin only improved significantly at 6 months. A reduction in A1C of >0.5% is considered clinically significant (17), and the reduction in A1C at 3 and 6 months exceeded this target (Supplementary Figure 1). These reductions were comparable with those seen in the ongoing Virta

**Table 2**
Weight loss, blood pressure, blood lipids and markers of kidney and liver function

| | Effect estimate (change from baseline with 95% CI) | | | p Value (vs baseline) | | |
|---|---|---|---|---|---|---|
| | 3 months | 6 months | 12 months | 3 months | 6 months | 12 months |
| Weight, kg | −8.7 (−11.4 to −6.0) | −10.7 (−13.3 to −8.0) | −9.5 (−12.3 to −6.7) | <0.0001 | <0.0001 | <0.0001 |
| Body mass index, kg/m$^2$ | −3.2 (−5.0 to −1.3) | −3.9 (−5.8 to −2.0) | −4.7 (−6.7 to −2.7) | 0.0017 | 0.00013 | <0.0001 |
| Weight loss, % | −8.0 (−10.1 to −5.8) | −9.7 (−11.8 to −7.6) | −8.3 (−10.5 to −6.0) | <0.0001 | <0.0001 | <0.0001 |
| Waist circumference, cm | −8.8 (−12.9 to −4.7) | −12.8 (−16.9 to −8.7) | −11.9 (−16.7 to −7.2) | <0.0001 | <0.0001 | <0.0001 |
| Hip circumference, cm | −7.0 (−11.0 to −3.0) | −9.5 (−13.5 to −5.5) | −10.0 (−14.7 to −5.3) | 0.0011 | <0.0001 | 0.0001 |
| Medication effect score, au * | −1.7 (−3.0 to −1.0) | −1.8 (−3.2 to −1.1) | −1.7 (−3.3 to −1.0) | NA | NA | NA |
| Percentage with A1C <6.5% and no glucose-lowering medications or metformin only | 14.8 (−3.3 to 33.7) | 22.2 (1.4 to 42.3) | 3.7 (−14.3 to 22.0) | 0.13 | 0.03 | >0.999 |
| Systolic blood pressure, mmHg | −9 (−14 to −3) | −9 (−14 to −3) | −8 (−13 to −2) | 0.0021 | 0.0028 | 0.0092 |
| Diastolic blood pressure, mmHg | 0 (−4 to 3) | −4 (−8 to 0) | −4 (−8 to 0) | 0.81 | 0.035 | 0.051 |
| Total cholesterol, mmol/L | −2.64 (−9.71 to 4.98)% † | -0.03 (−7.29 to 7.79)% † | 3.58 (−4.23 to 12.04)% † | 0.48 | 0.99 | 0.37 |
| HDL-C, mmol/L | 0.13 (0.04 to 0.21) | 0.13 (0.05 to 0.21) | 0.18 (0.09 to 0.27) | 0.0043 | 0.0036 | 0.00018 |
| LDL-C, mmol/L | −0.05 (−0.36 to 0.27) | 0.14 (−0.18 to 0.45) | 0.20 (−0.13 to 0.53) | 0.78 | 0.41 | 0.24 |
| Triglycerides, mmol/L | −19.32 (−29.17 to −8.11)% † | −24.49 (−33.71 to −14.00)% † | −24.04 (−33.67 to −13.02)% † | 0.0016 | <0.0001 | 0.00013 |
| Creatinine, g/dL | −3.04 (−6.51 to 0.43) | −2.31 (−5.83 to 1.20) | −0.12 (−3.73 to 3.49) | 0.093 | 0.2 | 0.95 |
| eGFR, mL/min per 1.73 m$^2$ † | 2.30 (−0.28 to 4.94) | 0.30 (−2.82 to 3.35) | 0.19 (−2.80 to 2.82) | NA | NA | NA |
| White blood cell count, 10$^9$ cells/L | −8.84 (−16.46 to −0.52)% † | 15.06 (−0.53 to 33.09)% † | −7.83 (−15.83 to 0.92)% † | 0.038 | 0.059 | 0.077 |
| Aspartate aminotransferase, U/L | −5.70 (−18.34 to 8.91)% † | −18.31 (−34.37 to 1.68)% † | −15.66 (−27.42 to −1.99)% † | 0.42 | 0.069 | 0.027 |
| Alanine transaminase, U/L | −7.14 (−22.59 to 11.40)% * | −23.50 (−43.39 to 3.37)% † | −17.82 (−32.19 to −0.40)% † | 0.42 | 0.08 | 0.046 |
| Hepatic steatosis index, au | −2.7 (−5 to −0.5) | −3.9 (−7.5 to −0.3) | −4.8 (−7.1 to −2.4) | 0.02 | 0.04 | 0.00025 |

*A1C*, glycated hemoglobin; *au*, arbitrary units; *BCa*, Bias-corrected and accelerated; *CI*, confidence interval; *eGFR*, estimated glomerular filtration rate; *HDL-C*, high-density lipoprotein cholesterol; *LDL-C*, low-density lipoprotein cholesterol; *NA*, not available—a precise p value cannot be obtained for a BCa bootstrap analysis.
*Note:* Data show effect estimates relative to baseline from linear mixed effects model (95% confidence interval) (N=27).
* Bias-corrected and accelerated confidence intervals derived from nonparametric bootstrap analysis.
† Effect estimate expressed as percentage difference (ratio of geometric means) from log-transformed analyses (baseline vs respective time point) obtained from a linear mixed-effects model.

Health trial, which uses a technology platform providing remote continuous care (9). Interestingly, a meta-analysis of the effect of weight loss on A1C showed that trials that achieved <5% weight loss demonstrated modest reductions in A1C of 0.2% to 0.3%, whereas trials that achieved >5% weight loss observed more robust reductions of 0.6% to 1.0% (18). In the present program, weight losses of >5% were observed at every time point (Table 2), likely contributing to a more pronounced reduction in A1C. Notably, reductions in A1C were achieved alongside robust reductions of certain classes of glucose-lowering medications, particularly sulfonylureas, sodium-glucose cotransporter-2 inhibitors and insulin (Supplementary Figure 2). This was reflected in the consistently reduced MES at all time points. The reduction in use of insulin and sulfonylureas is clinically important due to their known risk of hypoglycemia as well as questionable safety in patients with high cardiovascular risk (19,20). Thus, the program was effective at improving A1C in the presence of significant reductions in glucose-lowering medications.

Despite robust weight loss, there was a tendency for A1C to trend toward baseline by the 12-month follow up. The program did not formally assess dietary adherence and patients were not excluded if they stopped following a low-carbohydrate diet. This observation highlights the importance of consistent access to professional and peer supports to foster patient success, as well as the benefit of close follow up or potential for remote monitoring to support adherence or inform therapy adjustments. Indeed, the 2 most impactful factors on adherence to weight-loss interventions are continued supervision and social supports (21). Thus, provision of ongoing supports, likely in an individualized fashion, is advisable to sustain continued A1C reductions over the course of 1 year or longer.

### *Strengths and limitations*

A group visit model is more cost-effective (vs one-on-one visits with a dietitian), allowing the physicians and dietitians to offer education at a more scalable level. However, the program described still required consultations with an internist or family physician at multiple visits across 12 months. These program costs would be balanced against the lower cost of medications (or visits to the diabetes centre for insulin management) and expected long-term cost savings of lower vascular risk based on improvements in A1C, blood pressure and blood lipids. The cost-effectiveness of such an intervention is of great interest and requires further study.

Generalized conclusions from this program are limited given its single-arm, nonrandomized design. Patients were referred and self-selected for participation and thus not necessarily a representative sample of the general patient population, particularly because the majority of patients had relatively well-controlled diabetes at baseline. Furthermore, despite high initial interest in the program, the dropout rate in the first month suggests that such an intervention is not for everyone. In addition, dietary adherence was not assessed quantitatively.

This retrospective analysis is also limited by a lack of qualitative data regarding the feasibility and sustainability of the program, along with barriers and facilitators to success. These topics were frequently discussed informally with team members and patients, but not formally assessed. We were also unable to quantify use of all different components of the intervention or decipher which ones were most important for success.

### *Conclusions and future directions*

A physician-supported program utilizing an LCHF ketogenic diet appears effective for improving clinical outcomes in patients with T2D in a community setting when delivered by a multidisciplinary team. Key considerations include frequent safety monitoring when de-prescribing glucose-lowering medications in concert with carbohydrate restriction, and access to ongoing patient supports. This program may represent one effective model for implementing low-carbohydrate dietary interventions in primary care in Canada. Future programs should aim to quantify the potential long-term cost savings to the Canadian health-care system. In addition, future programs should explore the sustainability of such a program within a physician's practice and for the team members involved, with the aim of optimizing therapeutic nutrition interventions that can be implemented as an option for standard diabetes care.

## Supplementary Material

To access the supplementary material accompanying this article, visit the online version of the *Canadian Journal of Diabetes* at www.canadianjournalofdiabetes.com.

## Acknowledgments

The authors thank Rebecca Green-Lapierre MSc, PDt, and Ciara Stephens BScN, RN, for their valuable leadership, program development and patient support. We also thank Mardi Burton, NSHA health services manager, Primary Health Care, for her support and Amrit Sampalli, MD candidate, for support with chart review and data collection. Grants were provided by the Valley Regional Hospital Foundation and the Valley Regional Internal Medicine Fund for the creation of structured supports to assist patients in engaging in nutritional ketosis with medical supervision. Support was also provided by a Health Research Scholar Award (16890) from the Michael Smith Foundation (to J.P.L.). De-identified data can be made available for academic purposes from the corresponding author upon reasonable request.

## Author Disclosures

J.P.L. is the chief scientific officer for the not-for-profit Institute for Personalized Therapeutic Nutrition and holds shares in Metabolic Insights, Inc, a for-profit company developing noninvasive metabolic monitoring devices. M.M. is an expert advisor for the Institute for Personalized Therapeutic Nutrition, a member of Diet Doctor's medical review board, receives consultation fees to review guides and reports consulting fees from Novo Nordisk.

The remaining authors report no potential conflicts of interest.

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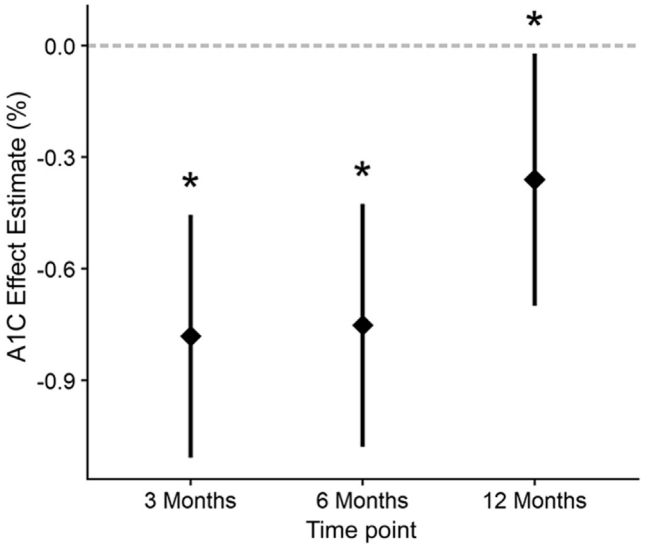
## Supplementary Appendix

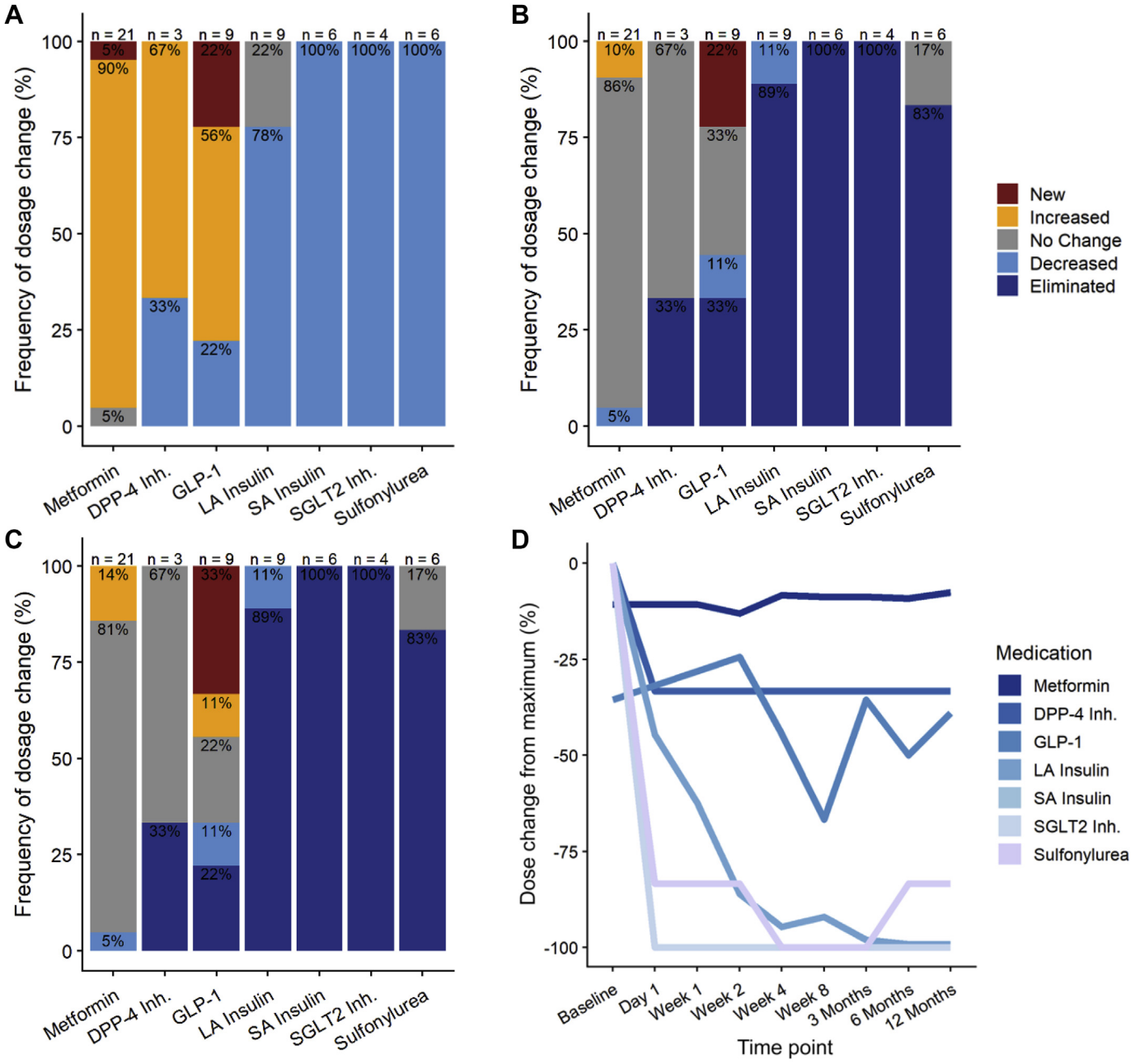
*Medication de-prescription protocol*

*Glucose-lowering medications:* Due to the rapid glucose-lowering effect of carbohydrate restriction, de-prescription of glucose-lowering medications was monitored closely and adapted to each patient's medical context. During the initial de-prescription of sulfonylureas and insulin, patients checked glucose at least twice per day with a target glucose of 7 to 10 mmol/L. Generally, sulfonylureas and basal insulin were reduced by 50% the day before starting the low-fat, high-carbohydrate ketogenic diet. For each subsequent day in the next 2 weeks, insulin was reduced by another 25% to 50% when glucose was within target and withheld if glucose was <5 mmol/L. Sulfonylureas were typically discontinued when glucose reached target. Routine use of rapid insulin was discontinued before meals, but a correction dose was offered on an individualized basis, as needed, to address postprandial hyperglycemia. Sodium-glucose cotransporter-2 inhibitors were discontinued 3 days before the start of the low-fat, high-carbohydrate ketogenic diet to lower the potential risk of euglycemic diabetic ketoacidosis. Metformin therapy was typically continued or increased based on guideline recommendations. Glucagon-like peptide-1 agonists were adjusted based on shared decision-making, cost and patient preference.

*Antihypertensive and lipid-lowering medications:* Blood pressure targets were set at <130/80 mmHg, according to Diabetes Canada guidelines. Typically, thiazide and loop diuretics were reduced or discontinued before initiation of the ketogenic diet due to the diet's initial diuretic effect. Home blood pressure monitoring was encouraged and antihypertensive therapies were de-escalated as needed. Angiotensin-converting enzyme inhibition and angiotensin-receptor blockers were optimized based on guideline therapies. Lipid-lowering medications were adjusted to achieve evidence-based low-density lipoprotein targets appropriate to each patient's condition.




**Supplementary Figure 1.** Effect estimates of A1C from a linear mixed-effects model plotted with 95% confidence interval. The dashed grey line represents baseline. * $p<0.05$ vs baseline. *A1C*, glycated hemoglobin.




**Supplementary Figure 2.** Dose changes in glucose-lowering medication use vs baseline at (A) 3 months, (B) 6 months and (C) 12 months. Change expressed as a percentage of the number of patients taking each medication class at baseline. (D) Average percent dose change in prescription of glucose-lowering medications across time. Dose change expressed as a percentage of maximum dose prescribed throughout the 12 months, including new prescriptions. *DPP-4 Inh.*, dipeptidyl peptidase-4 inhibitor; *GLP-1*, glucagon-like peptide-1; *LA*, long-acting; *SA*, short-acting; *SGLT2 Inh.*, sodium-glucose cotransporter-2 inhibitor.

**Supplementary Table 1**
Description of topics covered in informational sessions throughout the program

| Topics covered |
|---|
| Rationale and evidence supporting carbohydrate restriction for T2D |
| Reading "Nutrition Facts" labels |
| Meal and snack ideas |
| Foods to include or avoid (green, yellow, red categorization method) |
| Identifying sources of carbohydrates |
| Identifying healthy sources of fat |
| Assuring adequate protein intake |
| How to know if you are in ketosis |
| The role of artificial sweeteners |
| Risks, benefits, alternatives and importance of medication adjustments |
| Goals for blood sugar during insulin de-escalation |
| Concepts of diabetes remission vs diabetes reversal |
| Intermittent fasting |
| Physical activity |
| 24-hour dietary recall |
| Mindfulness and emotional strategies |

*T2D*, type 2 diabetes.

**Supplementary Table 2**
Sample 2-day LCHF ketogenic diet menu plan

| Meal plan day 1 | Carbohydrates (g) | Meal plan day 2 | Carbohydrates (g) |
|---|---|---|---|
| Breakfast | | | |
| ½ cup full-fat cottage cheese | 4 | 2 eggs | 0 |
| ½ cup frozen mixed berries, thawed | 6 | 2 cups spinach, 4 sliced cherry tomatoes, 4 sliced white mushrooms, all fried in butter | 2.5 |
| Coffee with coconut oil or heavy cream | 0 | Coffee with coconut oil or heavy cream | 0 |
| Lunch | | | |
| ½ can light tuna and 2 tbsp mayonnaise | 2 | 2 cups ready-made coleslaw | 4 |
| 4 dill pickle slices and 4 romaine lettuce leaves | 0.5 | ¼ cup tamari almonds (fried in butter with Bragg's soy sauce) | 2 |
| 2 cups beef broth | 0 | 2 cups chicken broth | 0 |
| Coconut bombs (coconut oil, shredded coconut, sweetener) | 1.1 | | |
| Snack | | | |
| 2 devilled eggs | 0 | 30 g (1¼ inch) cubed cheddar cheese | 1 |
| | | 4 green olives | 0 |
| Dinner | | | |
| 3 oz. chicken breast | 0 | 4 oz salmon steak topped with heavy cream and dill | 0 |
| 1 cup roasted cauliflower with olive oil | 4.5 | 1 cup roasted broccoli with coconut oil | 4 |
| 2 cups mesclun mix salad topped with sliced cucumbers, red peppers and red cabbage | 3 | ½ cup sliced red pepper, ½ cup snow peas (all sautéed in butter) | 5.5 |
| Olive oil and lemon juice dressing | 0 | | |
| Net daily carbohydrate intake | 21.1 | | 17 |

*LCHF*, low-carbohydrate, high-fat; *tbsp*, tablespoon.

**Supplementary Table 3**
Frequency of direction of change in antihypertensive and lipid-lowering medication dose at 12 months relative to baseline

| | New (%) | Increased (%) | No change (%) | Decreased (%) | Eliminated (%) |
|---|---|---|---|---|---|
| Alpha-1 antagonists (n=1) | 0 | 0 | 100 | 0 | 0 |
| ACE inhibitors (n=11) | 0 | 14 | 57 | 29 | 0 |
| Aldosterone antagonists (n=1) | 0 | 0 | 0 | 0 | 100 |
| Angiotensin II receptor blockers (n=9) | 0 | 14 | 43 | 29 | 14 |
| Acetylsalicylic acid (n=9) | 0 | 0 | 100 | 0 | 0 |
| Calcium channel blockers (n=6) | 60 | 0 | 20 | 20 | 0 |
| Cholesterol absorption inhibitors (n=1) | 100 | 0 | 0 | 0 | 0 |
| Lipase inhibitors (n=1) | 0 | 0 | 0 | 0 | 100 |
| Platelet inhibitors (n=1) | 0 | 0 | 100 | 0 | 0 |
| Statins (n=20) | 18 | 29 | 29 | 12 | 12 |

*ACE*, angiotensin-converting enzyme.
Data expressed as percentage of total number of patients taking each class of antihypertensive and lipid-lowering medications at baseline.