# Effects of a Diet Higher in Carbohydrate/Lower in Fat Versus Lower in Carbohydrate/Higher in Monounsaturated Fat on Postmeal Triglyceride Concentrations and Other Cardiovascular Risk Factors in Type 1 Diabetes

Irene Strychar, edd, rd[1,2,3,4]
Jeffrey S. Cohn, phd[5]
Geneviève Renier, md, phd[1,2,3,4]
Michèle Rivard, phd[6]
Nahla Aris-Jilwan, md[3]
Hugues Beauregard, md[3]
Sara Meltzer, md[7]
André Bélanger, md[8]
Richard Dumas, md[8]
Alain Ishac, msc[1]
Farouk Radwan, md[9]
Jean-François Yale, md[7]

**OBJECTIVE** — To compare the effects of a eucaloric diet higher in carbohydrate/lower in fat versus lower in carbohydrate/higher in monounsaturated fat on postmeal triglyceride (TG) concentrations and other cardiovascular disease risk factors in nonobese subjects with type 1 diabetes and in good glycemic control.

**RESEARCH DESIGN AND METHODS** — In a parallel group design study, 30 subjects were randomly assigned and completed one of the two eucaloric diets. Assessments included: BMI, blood pressure, A1C, plasma lipids, and markers of oxidation, thrombosis, and inflammation. At 6 months, subjects were hospitalized for 24 h to measure plasma TG excursions.

**RESULTS** — There were no significant differences between groups other than decreased plasminogen activator inhibitor 1 (PAI-1) levels and weight gain in the lower-carbohydrate/higher–monounsaturated fat group. During the 24-h testing, the lower-carbohydrate/higher–monounsaturated fat group had a lower plasma TG profile.

**CONCLUSIONS** — A diet lower in carbohydrate/higher in monounsaturated fat could offer an appropriate choice for nonobese type 1 diabetic individuals with good metabolic and weight control.

*Diabetes Care* **32:1597–1599, 2009**

The optimal macronutrient composition of the diet in diabetes has not yet been established. Dietary recommendations for carbohydrates range from 45–60% of energy intake and 25–35% for fat (1–2); the amount of carbohydrates is usually inversely associated with amount of fat. On the one hand, higher amounts of carbohydrates are directly associated with increases in plasma triglyceride (TG) levels and postprandial glycemic levels (3), risk factors in the development of micro- and macrovascular complications in diabetes (4–5). On the other hand, higher-fat diets have the potential to increase fasting and postprandial TG levels, independent risk factors for macroangiopathy (5). Thus, nutrition therapy needs to address the effects of diet on both fasting and postprandial TG and glycemic levels.

In type 2 diabetes, replacing carbohydrates with monounsaturated fats in eucaloric diets was associated with lower TG levels and improved glycemic control (6–7); however, results are contradictory in type 1 diabetes (8–9). Therefore, our objective was to compare the effects of a eucaloric diet, higher in carbohydrate/lower in fat versus lower in carbohydrate/higher in monounsaturated fat, on 24-h TG and glycemic excursions and other cardiovascular risk factors in type 1 diabetes. We expected that: *1*) the lower carbohydrate content of the higher–monounsaturated fat diet would reduce VLDL-TG production, resulting in lower fasting TG levels and 24-h TG area under the curve, in line with type 2 diabetes studies (6–7); and, *2*) the higher monounsaturated fat intake would positively affect LDL oxidation, adhesion molecules, and markers of thrombosis and inflammation (10–12).

## RESEARCH DESIGN AND METHODS

— Adults with type 1 diabetes on intensive insulin therapy were recruited. Exclusion criteria included BMI $\geq$30 kg/m$^2$, A1C >8.4%, and major diabetes complications. The project received ethics approval (2003–2007).

In this 6-month parallel group design study, subjects were randomly assigned to follow a eucaloric diet higher in carbohydrate/lower in fat or lower in carbohydrate/higher in monounsaturated fat: 54–57% and 43–46% carbohydrates, 27–30% and 37–40% total fat (10 and 20% monounsaturated), respectively. Saturated fat (<10%) and fiber (25 g/day)

From the [1]Research Center of the University of Montreal Hospital Center (CRCHUM), Montreal, Quebec, Canada; the [2]Montreal Diabetes Research Center of CRCHUM, Montreal, Quebec, Canada; the [3]Service of Endocrinology, Notre-Dame Hospital of the University of Montreal Hospital Center (CHUM), Montreal, Quebec, Canada; the [4]Department of Nutrition, Faculty of Medicine, University of Montreal, Montreal, Quebec, Canada; the [5]Heart Research Institute, Sydney, Australia; the [6]Department of Social and Preventive Medicine, University of Montreal, Montreal, Quebec, Canada; the [7]Nutrition and Food Science Centre, Royal Victoria Hospital, McGill University Health Center, Montreal, Quebec, Canada; the [8]Laval Clinic Research Center, Montreal, Quebec, Canada; and the [9]Department of Biochemistry, Notre-Dame Hospital of CHUM, Montreal, Quebec, Canada.
Corresponding author: Irene Strychar, irene.strychar@umontreal.ca.
Received 29 December 2008 and accepted 4 June 2009.
Published ahead of print at http://care.diabetesjournals.org on 18 June 2009. DOI: 10.2337/dc08-2322.

**Table 1—*Glycemic, anthropometric, blood pressure, and fasting plasma lipid measurements and oxidative, thrombotic, and inflammatory markers throughout the dietary trial for the higher-carbohydrate/lower-fat diet group* (n = 15) *and lower-carbohydrate/higher–monounsaturated fat diet group* (n = 15)**

| | Higher-carbohydrate/lower-fat diet | | | Lower-carbohydrate/higher–monounsaturated fat diet | | |
|---|---|---|---|---|---|---|
| | Baseline* | 6 Months† | Absolute change | Baseline* | 6 Months† | Absolute change |
| A1C (%) | 7.3 ± 0.7 | 7.2 ± 0.7 | −0.2 ± 0.5 | 7.0 ± 0.7 | 7.1 ± 0.9 | +0.1 ± 0.6 |
| BMI (kg/m$^2$) | 24.3 ± 2.6 | 24.1 ± 2.6 | −0.24 ± 1.0‡ | 24.3 ± 2.7 | 24.8 ± 2.7 | +0.56 ± 0.6‡ |
| Weight (kg) | 71.1 ± 13.7 | 70.3 ± 13.1 | −0.83 ± 3.0‡ | 71.8 ± 13.4 | 73.4 ± 13.6 | +1.6 ± 1.8‡ |
| Body fat (%) | 25.3 ± 7.6 | 23.8 ± 7.0 | −1.6 ± 3.8‡ | 23.9 ± 7.6 | 25.3 ± 7.4 | +1.4 ± 2.1‡ |
| Fat-free mass (%) | 36.6 ± 5.5 | 37.6 ± 5.3 | +1.0 ± 2.4 | 36.3 ± 4.7 | 36.5 ± 5.6 | +0.24 ± 2.2 |
| Systolic blood pressure (mmHg) | 116 ± 15 | 120 ± 10 | +3.9 ± 14.4 | 122 ± 15 | 122 ± 16 | −0.2 ± 21.1 |
| Diastolic blood pressure (mmHg) | 72 ± 7 | 77 ± 8 | +4.7 ± 11.0 | 78 ± 7 | 75 ± 8 | −2.6 ± 8.9 |
| TG (mmol/l) | 0.72 ± 0.30 | 0.86 ± 0.55 | +0.14 ± 0.46 | 0.68 ± 0.34 | 0.65 ± 0.20 | −0.03 ± 0.22 |
| Total cholesterol (mmol/l) | 4.40 ± 0.66 | 4.28 ± 0.78 | −0.12 ± 0.66 | 4.48 ± 0.88 | 4.25 ± 0.64 | −0.24 ± 0.66 |
| HDL cholesterol (mmol/l) | 1.47 ± 0.35 | 1.53 ± 0.35 | +0.06 ± 0.27 | 1.64 ± 0.33 | 1.63 ± 0.38 | −0.01 ± 0.22 |
| Total cholesterol–to–HDL cholesterol ratio | 3.12 ± 0.77 | 2.90 ± 0.78 | −0.22 ± 0.55 | 2.80 ± 0.67 | 2.67 ± 0.48 | −0.13 ± 0.37 |
| LDL cholesterol (mmol/l) | 2.60 ± 0.63 | 2.35 ± 0.75 | −0.25 ± 0.70 | 2.53 ± 0.73 | 2.31 ± 0.46 | −0.21 ± 0.57 |
| ApoB (g/l) | 0.78 ± 0.17 | 0.76 ± 0.22 | −0.01 ± 0.12 | 0.75 ± 0.12 | 0.72 ± 0.11 | −0.04 ± 0.07 |
| 8-iso PGF (pg/ml) | 239 ± 147 | 267 ± 167 | +27.9 ± 132.6 | 243 ± 132 | 210 ± 138 | −32.1 ± 68.3 |
| PAI-1 (ng/ml) | 55.0 ± 23.2 | 69.2 ± 23.1 | +14.2 ± 24.5‡ | 78.5 ± 37.7 | 65.7 ± 28.7 | −12.8 ± 27.0‡ |
| E-selectin (ng/ml) | 22.5 ± 14.7 | 21.7 ± 13.9 | −0.8 ± 3.5 | 23.8 ± 15.5 | 25.0 ± 14.5 | +1.2 ± 3.0 |
| sICAM-1 (ng/ml) | 314 ± 59 | 315 ± 58 | +0.2 ± 37 | 326 ± 84 | 328 ± 77 | +2.0 ± 46 |
| hsCRP-1 (μg/ml) | 7.6 ± 8.2 | 9.9 ± 12.3 | +2.3 ± 10.9 | 6.8 ± 5.4 | 5.1 ± 5.9 | −1.7 ± 4.8 |
| TNF-α (pg/ml) | 1.2 ± 0.5 | 1.5 ± 0.8 | +0.3 ± 0.8 | 1.4 ± 0.5 | 1.4 ± 0.9 | −0.1 ± 0.8 |

Data are means ± SD. *No significant differences were observed between diet groups at baseline ($P > 0.05$). †No significant differences were observed between diet groups at 6 months ($P > 0.05$). ‡Significantly different between diet groups for absolute change between baseline and 6 months ($P < 0.05$). hsCRP, high-sensitivity C-reactive protein; PGF, prostaglandins-F2-α; sICAM, soluable intracellular adhesion molecule; TNF, tumor necrosis factor.

contents were similar. The lower-carbohydrate/higher-fat diet had fewer starch and more fat choices in the form of olive oil.

On a weekly basis a dietitian monitored subjects dietary intake (24-h telephone food recall), premeal glycemia, dietary carbohydrate meal content, and insulin doses administered. Subjects recorded daily glycemic levels, insulin doses, grams of mealtime carbohydrates, and hypoglycemic events.

Baseline and 6-month measurements included BMI, blood pressure, A1C, plasma lipids, adhesion molecules, and markers of oxidation, thrombosis, and inflammation. At 6 months, subjects were hospitalized for 24 h to monitor TG and glycemic excursions. Meals were standardized according to diet assignment: breakfast and lunch were similar for the two groups, and differences in carbohydrate/fat contents were achieved at the supper meal. Usual prescribed insulin doses were given and took into account premeal glycemia and carbohydrate meal content.

Mann-Whitney tests were used to compare values between the two diets, and Wilcoxon's signed-rank tests were used to compare values within each diet group, since many variables did not meet the normality assumption of the *t* test. Significance was set as $P \leq 0.05$.

**RESULTS** — Thirty individuals completed the study, and baseline characteristics were similar between the two study groups: mean age 37.9 ± 8.1 years, years since diagnosis 16.5 ± 10.6, BMI 24.3 ± 2.6 kg/m$^2$, A1C 7.2 ± 0.7%, dietary carbohydrates 45.5 ± 10.7%, total fat 36.9 ± 10.5%, monounsaturated fat 15.5 ± 5.7%, and saturated fat 11.7 ± 4.3%. The carbohydrate intake of our subjects was similar to that of the subjects in the Diabetes Control and Complications Trial (DCCT) intensive-treated group (13). During the 6-month trial, subjects followed their diet prescriptions (based on 24-h recalls) and appropriately assessed carbohydrate intake at meals (± 10%) for insulin dose adjustments. There were no differences between groups for insulin doses, glycemic levels, and hypo- and hyperglycemic episodes.

Clinical and biochemical results (Table 1) indicate no significant differences between the two groups at baseline and at 6 months, except for absolute change scores where subjects on the lower-carbohydrate/higher–monounsaturated fat diet had a 2% increase in weight and a 16% decrease in plasminogen activator inhibitor 1 (PAI-1) levels.

During the 24-h testing, TG profiles tended to be lower in the lower-carbohydrate/higher–monounsaturated fat group (see supplementary Fig. 1*A* and *B* in the online appendix available at http://care.diabetesjournals.org/cgi/content/full/dc08-2322/DC1), while glycemic levels after supper were higher in the higher-carbohydrate/lower-fat group. Since weight change affects TG levels, analyses were repeated with subjects maintaining stable weight (±2 kg) throughout the study (see supplementary Fig. 2*A* and *B* in the online appendix). Many significantly lower TG levels were observed in the lower-carbohydrate/higher–monounsaturated fat group, resulting in a smaller TG area under the curve ($P = 0.05$).

**CONCLUSIONS** — In a sample of nonobese adults with well-controlled, uncomplicated type 1 diabetes on inten-

sive insulin therapy, the lower-carbohydrate/higher–monounsaturated fat diet was found to have a positive effect on thrombotic factor PAI-1 and no adverse effect on lipid control. A small increase in BMI in the lower-carbohydrate/higher–monounsaturated fat group may be due to a lower meal-induced thermogenesis because of its higher fat content (14); longer-term studies are needed.

In the higher-carbohydrate/lower-fat group, where individuals had increased carbohydrate intake by ~20%, the higher fasting TG levels are in keeping with an associated increase of VLDL-TG production, as shown in type 2 diabetes studies (6–7). In the lower-carbohydrate/higher–monounsaturated fat group, fasting TG levels remained steady, despite a small weight gain. Insulin doses were unlikely to have determined fasting TG levels, given that the doses administered were comparable in the two groups and appropriately adjusted to carbohydrate intake in order to maintain A1C levels close to the recommended range.

During the 24-h test period, higher TG levels were observed in the higher-carbohydrate/lower-fat group. The finding that "carbohydrate-induced lipemia" rather than "fat-induced lipemia" (3) may have a dominant influence on triglyceridemia in the diabetic state in day-to-day conditions has clinical relevance. Our subjects had normal TG values; further research is warranted with individuals in poor lipid control.

Higher glycemic levels after the higher-carbohydrate/lower-fat supper were observed during the 24-h testing period, despite appropriate adjustment of insulin doses to carbohydrate meal content. Lower glycemic levels after the higher–monounsaturated fat supper meal may be due to slower gastric emptying in the presence of olive oil (15).

Limitations include small sample size and short study duration. Our results are in keeping with the American Diabetes Association statement that it is unlikely that an optimal mix of macronutrients exists for the diabetic state (1).

In conclusion, a diet lower in carbohydrate/higher in monounsaturated fat is an appropriate nutrition therapy for type 1 diabetic individuals with good metabolic and weight control.

**Acknowledgments**— This research project was funded by the Canadian Institutes of Health Research, Institute of Nutrition and Metabolism, Ottawa, Canada (MOP 62854).

Lifescan Canada supplied patients with UltraSmart glucose meters plus strips for daily blood glucose testing during the study period.

No potential conflicts of interest relevant to this article were reported.

We give special thanks to the patients who participated in this study. We acknowledge the valuable assistance of doctors, nurses, and personnel of Notre-Dame Hospital of the University of Montreal Hospital Center (CHUM), the McGill University Health Center, and the Laval Clinic Research Center.

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