# Beneficial Effects of Viscous Dietary Fiber From Konjac-Mannan in Subjects With the Insulin Resistance Syndrome

## Results of a controlled metabolic trial

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OBJECTIVE— Dietary fiber has recently received recognition for reducing the risk of developing diabetes and heart disease. The implication is that it may have therapeutic benefit in prediabetic metabolic conditions. To test this hypothesis, we investigated the effect of supplementing a high-carbohydrate diet with fiber from Konjac-mannan (KJM) on metabolic control in subjects with the insulin resistance syndrome.

RESEARCH DESIGN AND METHODS— We screened 278 free-living subjects between the ages of 45 and 65 years from the Canadian-Maltese Diabetes Study. A total of 11 (age 55 ± 4 years, BMI 28 ± 1.5 kg/m$^2$) were recruited who satisfied the inclusion criteria: impaired glucose tolerance, reduced HDL cholesterol, elevated serum triglycerides, and moderate hypertension. After an 8-week baseline, they were randomly assigned to take either KJM fiber–enriched test biscuits (0.5 g of glucomannan per 100 kcal of dietary intake or 8–13 g/day) or wheat bran fiber (WB) control biscuits for two 3-week treatment periods separated by a 2-week washout. The diets were isoenergetic, metabolically controlled, and conformed to National Cholesterol Education Program Step 2 guidelines. Serum lipids, glycemic control, and blood pressure were the outcome measures.

RESULTS— Decreases in serum cholesterol (total, 12.4 ± 3.1%, $P < 0.004$; LDL, 22 ± 3.9%, $P < 0.002$; total/HDL ratio, 15.2 ± 3.4%, $P < 0.003$; and LDL/HDL ratio, 22.2 ± 4.1%, $P < 0.002$), apolipoprotein (apo) B (15.1 ± 4.3%, $P < 0.0004$), apo B/A-1 ratio (13.1 ± 3.4%, $P < 0.0003$), and serum fructosamine (5.2 ± 1.4%, $P < 0.002$) were observed during KJM treatment compared with WB-control. Fasting blood glucose, insulin, triglycerides, HDL cholesterol, and body weight remained unchanged.

CONCLUSIONS— A diet rich in high-viscosity KJM improves glycemic control and lipid profile, suggesting a therapeutic potential in the treatment of the insulin resistance syndrome.

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Abbreviations: apo, apolipoprotein; CHD, coronary heart disease; IGT, impaired glucose tolerance; KJM, Konjac-mannan; NCEP, National Cholesterol Education Program; WB, wheat bran.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Abnormal glucose tolerance and insulin resistance are related to multiple cardiovascular risk factors, especially reduced HDL cholesterol, elevated serum triglycerides, and hypertension (1). When clustered, these abnormalities increase the risk of coronary heart disease (CHD) morbidity and mortality, an effect that is independent of other conventional risk factors (2). Co-ocurrence is usually present in insulin-insensitive individuals (3) and is often described in relation to visceral adiposity (4) and lack of physical activity (5). The estimated prevalence ranges from 3% (2) to ~30% (1,6) depending on how this insulin resistance syndrome is defined and in which population it is measured.

Although it has been extensively described (1–5), followed up (6), and had its prevalence determined (1,2), no specific recommendations for treatment of this syndrome have been proposed by health agencies. In practice, initial therapy of individual risk factors such as moderate dyslipidemia, hypertension, or hyperglycemia is nonpharmacological. Treatment will often include behavioral changes to reduce body weight, increase physical activity, and moderate alcohol consumption. To achieve nutritional goals, there are three main approaches: a high-carbohydrate/low-fat diet (7), sharing calories between monounsaturated fat and complex carbohydrate at the expense of saturated fat (8), or supplementing a high-carbohydrate/low-fat diet with exercise (9).

Evidence suggests that fiber may also be used in a therapeutic role. Recent epidemiological findings confirm the relationship between high dietary fiber intake and lower risk of developing both diabetes (10,11) and CHD (12). Soluble dietary fiber, in particular, has been shown clinically to reduce the need for insulin (13), improve glycemia (14), and reduce serum LDL cholesterol (15). Its viscosity is proposed as an important mechanistic factor (16). We recently demonstrated that a metabolically controlled low-fat diet supplemented with the high-viscosity Konjac-

mannan (KJM) simultaneously improved three major CHD risk factors in type 2 diabetic subjects (17).

In the present study, we tested the hypothesis that the same intervention would improve the control of conventional and emerging CHD risk factors in prediabetic individuals with a full cluster of metabolic abnormalities that define the insulin resistance syndrome. KJM flour is obtained by grinding the tuber root of the *Amorphophallus Konjac C. Koch.* plant and is traditionally used as a food and remedy in the Far East. In addition to our previous findings (17), other findings have shown it to improve cholesterol levels (18,19), hypertension, and glycemia (20,21).

## RESEARCH DESIGN AND METHODS

### Subjects

We screened 278 free-living subjects from the Canadian-Maltese Diabetes Study between the ages of 45 and 65 years. This population is known to have one of the highest rates of diabetes (22). Of the subjects, 38 satisfied the initial inclusion criteria: impaired glucose tolerance (IGT) (23); clinical absence of CHD; BMI $<30$ kg/m$^2$; not taking medications for hyperglycemia, hyperlipidemia or hypertension; not smoking; nor consuming more than two alcoholic drinks per day. These subjects were further screened for the presence of the full insulin resistance syndrome (2). This included moderate hypertension ($>135/85$ and $<145/95$ mmHg), dyslipidemia (low HDL cholesterol [$<0.9$ mmol/l for men and $<1.2$ mmol/l for women], and elevated triglycerides [$>2.3$ mmol/l and $<4.5$ mmol/l]). Based on power analysis from the previous study (17), 11 subjects (5 men, 6 women) who qualified were recruited. In addition to meeting the above criteria, their fasting (98 ± 13 pmol/l) and 2-h postprandial (439 ± 68 pmol/l) plasma insulin levels were greater ($P < 0.05$) than 2 SDs of the initial screening pool (71 ± 8 and 316 ± 47 pmol/l, respectively). All 11 also had moderately high serum cholesterol (5.2–6.7 mmol/l) and were sedentary, with a mean (± SD) age of 55 ± 4 years (range: 46–61); a BMI of 28 ± 3 kg/m$^2$; a waist-to-hip ratio of 0.98 ± 0.2 (waist: 96 ± 12 cm) in men and 0.91 ± 0.4 (waist: 87 ± 19 cm) in women. They gave written informed consent to participate in the current study that was approved by the Human Ethics Committees of St. Michael's Hospital and the University of Toronto.

### Study design

The study employed a double-blind placebo-controlled crossover design identical to that used on our previous study (17). It began with an 8-week baseline period during which participants followed a National Cholesterol Education Program (NCEP) Step 2 (8) ad libitum diet, documented by three nonconsecutive days of food records every 2 weeks. This run-in phase was included to eliminate possible effects of dietary change on metabolic parameters. The experimental phase of the study followed. This phase consisted of two successive 3-week treatment periods, separated by a 2-week washout interval over which a Step 2 diet was followed and documented by another 3-day food record. During the first treatment period, subjects were randomly assigned to either the KJM (Step 2 metabolically controlled diet enriched with KJM fiber) or the control treatment (the same diet enriched with wheat bran [WB] fiber). For the second treatment period, the subjects were crossed over. Blood collection, weight, blood pressure, and waist and hip measurements were done at the beginning and end of each 3-week treatment period. The study began with five subjects taking the KJM treatment and six the control.

### Diet

Both treatments consisted of a 3-day rotating Step 2 diet with three meals per day provided under metabolic conditions. All foods were pre-weighed, packaged, and delivered by courier to participants for consumption at home or at work. The mean macronutrient profile closely conformed to a Step 2 diet ($<30\%$ of calories from total fat, $<7\%$ from saturated fat, and $<300$ mg/day cholesterol) (8). Energy intakes for weight maintenance were provided according to Lipid Research Clinics Tables with adjustment for physical activity (24). Total dietary fiber was administrated at 1.5 g/100 kcal, with a mean daily intake according to energy intake ranging from 24 g to a plateau of 40 g for those consuming $\geq$2,800 kcal per day. The actual diet consumed is presented in Table 1.

The two treatments differed only in the type of fiber. On the KJM treatment, participants received KJM-enriched test biscuits, whereas on the WB-control treatment they received an equal quantity of WB control biscuits. Subjects were instructed to eat an equal amount of biscuits together with an 8-oz. beverage three times daily as a snack, including once at bedtime. Both were provided by Dicofarm S.p.A. (Rome). The biscuits had similar nutrient profiles and were indistinguishable in taste and appearance. KJM biscuits contained ~10% KJM flour, of which 69% was the active high-viscosity glucomannan, 15% other polysaccharides, and 16% excipients by weight (17). Because KJM flour comprised half (0.75 g/100 kcal) of the total fiber on the KJM treatment, ~0.5 g/100 kcal (8–13 g/day) was glucomannan. WB biscuits, in contrast, had a lower proportion of total dietary fiber than KJM biscuits. Therefore, ~11 g/day of wheat bran fiber derived from standardized American Association of Cereal Chemists hard red wheat bran was, therefore, added to the WB-control diet to compensate for these fiber differences. Subjects were instructed to sprinkle the additional fiber on cereal, yogurt, and/or other compatible foods to improve the fiber's palatability.

Any foods from the metabolic diet together with study biscuits not consumed during the study were returned to the clinic for weighing to measure compliance. Dietary changes found to occur during the first 3-week treatment period were duplicated in the diets for the second treatment period for each participant.

### Laboratory methods

Laboratory methods were identical to those used in our previous study (17). In brief, blood samples were separated immediately and stored as serum in four aliquots at −70°C after collection. They were thawed at the end of the study for analysis of total cholesterol, HDL cholesterol, and triglycerides measured enzymatically. LDL cholesterol content was estimated by the formula of Friedewald et al. Apolipoprotein (apo) A1 and B were determined by rocket immunoelectrophoresis. Fasting blood glucose was analyzed by a hexokinase method using a Cobas Mira Autoanalyzer (Roche Diagnostic, Mississauga, Canada). Serum fructosamine was analyzed in triplicate using Cobas Fara II and plasma insulin in duplicate by radioimmunoassay with reagent from ICN Biomedicals (Horsham, PA). C-peptide was determined by radioimmunoassay.

### Statistical analyses

Results are expressed as means ± SEM, except for age, anthropometric measurements, and nutrient intake (means ± SD). Data were analyzed by the Statistical Analysis System (SAS Institute, Cary, NC). Differences between the diets were assessed by

two-tailed Student's *t* test for paired data (univariate procedure). This same statistic also assessed differences in serum lipids, apolipoproteins, glycemia, blood pressure, and body weight between the beginning (week 0) and end (week 3) of each treatment (WB-control and KJM). Analysis of covariance (ANCOVA) with general linear model (GLM) procedure was used to test for differences in these same parameters between the two treatments. Control of individual variation from the repeated-measures aspect of the design was addressed by incorporating the random subject effect as well as the starting measurement. Diet, sex, and phase effects were also incorporated in this model. Adjustment for multiple comparisons was made by the Bonferroni-Hochberg procedure (25). *P* values for each end point were ordered sequentially and contrasted with the corresponding adjusted comparisonwise critical alpha ($\alpha$) levels. The null hypotheses were rejected only if *P* values were less than their corresponding $\alpha$ values.

Table 1—*Average intake of energy and nutrients before and during study periods in 11 subjects*

| Parameters | Baseline | KJM | WB |
|---|---|---|---|
| Total energy (kcal/day) | 2,070 ± 700 | 2,579 ± 628 | 2,355 ± 420 |
| Total fat (% of energy) | 30.5 ± 4.3 | 29.3 ± 3.2 | 28.7 ± 2.4 |
| Saturated fat (% of energy) | 7.2 ± 4.7 | 6.7 ± 0.8 | 6.4 ± 0.7 |
| Monounsaturated fat (% of energy) | 10.3 ± 5.1 | 12.7 ± 2.1 | 12.2 ± 2.6 |
| Polyunsaturated fat (% of energy) | 13.0 ± 5.7 | 9.9 ± 1.8 | 10.1 ± 0.9 |
| Cholesterol (mg/day) | 328 ± 102 | 219 ± 48 | 236 ± 77 |
| Total protein (% of energy) | 14.6 ± 8.2 | 16.2 ± 2.7 | 15.6 ± 3.2 |
| Available carbohydrate (% of energy) | 54.9 ± 21 | 54.5 ± 9.4 | 55.7 ± 7.3 |
| Sugars (% of energy) | 13.3 ± 3.6 | 11.2 ± 0.9 | 9.2 ± 1.4 |
| Fiber (g/day) | | | |
| Total | 24.2 ± 11 | 34.7 ± 8.4 | 33.4 ± 9.6 |
| Water-soluble | 6.9 ± 3.2 | 23.4 ± 1.7 | 9.9 ± 3.2* |
| Water-insoluble | 17.3 ± 7.3 | 11.2 ± 3.8 | 23.1 ± 2.6* |
| Sodium (mg) | 5,810 ± 2,384 | 3,162 ± 648 | 3,380 ± 647 |
| Potassium (mg) | 3,882 ± 713 | 4,530 ± 611 | 4,840 ± 872 |
| Calcium (mg) | 1,366 ± 193 | 1,260 ± 238 | 1,487 ± 446 |

Data are means ± SD. KJM and WB-control diets are based on actual intake. Baseline values are based on the mean of four 3-day food records. *$P < 0.001$ for differences between KJM and WB-control treatments (Student's *t* test for paired data).

RESULTS — All participants followed the experimental protocol with little difficulty. Returned food from metabolic diets indicated that subjects consumed an average of 96% and 95% of diet calories prescribed on the KJM and WB-control treatments, respectively. Returned biscuits indicated they consumed 81% (97 g/day) of KJM and 86% (103 g/day) of WB-control biscuits. Consumption patterns translated into an insignificant decrease in body weight during both treatment periods with no difference between treatments (Table 2). The only side effect experienced was a transient complaint of flatulence and soft stools reported by three and two of the participants during the KJM and the WB-control treatments, respectively, but none chose to discontinue the study.

### Serum lipids

Blood lipids improved during KJM compared with WB-control treatment (Table2). Total and LDL cholesterol fell significantly by 19 ± 2.7% ($P < 0.0001$) and 29 ± 3.4% ($P < 0.0001$) during KJM treatment compared with 6.3 ± 3.4% ($P = 0.088$) and 6.6 ± 5.0% ($P = 0.231$) on control treatment. The between-treatment differences were 12.4 ± 3.1% ($P < 0.005$) and 22 ± 3.9% ($P < 0.003$), respectively. The combined fall in total cholesterol from 6.2 ± 0.3 to 5.0 ± 0.2 mmol/l and LDL from 3.9 ± 0.2 to 2.8 ± 0.2 mmol/l on KJM treatment indicated reclassification of the lipid status of the group (8 of 11 subjects) from elevated to normal cholesterolemia (7). Similar results were observed for apo B. During KJM treatment, apo B fell significantly by 19 ± 2.8% ($P < 0.0004$) compared to 4.5 ± 4.5% ($P = 0.34$) on control, for a significant difference of 15.1 ± 4.3% ($P < 0.0004$) between the treatments.

In contrast, such effects were not seen in apo A-1 or triglycerides. During KJM and control treatments, HDL cholesterol decreased significantly on both treatments: 8.5 ± 2.2%, $P < 0.04$ on KJM diet and 9.6 ± 2.2%, $P < 0.003$ on WB-control, with an insignificant between-treatment change ($P = 0.98$). Similarly, during both treatments, triglycerides increased insignificantly, with no significant difference between treatments.

Despite this lack of effect of KJM treatment on HDL cholesterol, apo A-1, or triglycerides, the decreases in total cholesterol and apo B were sufficient to improve lipid ratios. During KJM treatment, total/HDL, LDL/HDL, and apo B/A-1 ratios decreased by 11 ± 3.0% ($P < 0.005$), 22 ± 3.7% ($P < 0.0002$), and 13 ± 3.0% ($P < 0.003$), respectively. This compares to an insignificant increase of 4.1 ± 4.1% in total/HDL ratio, 0.2 ± 6.3% in LDL/HDL ratio, and 0.7 ± 3.6 % in apo B/A-1 on WB-control. The resulting between-treatment differences were 15.2 ± 3.4% ($P < 0.003$) for total/HDL cholesterol, 22.2 ± 4.1% ($P < 0.002$) for LDL/HDL cholesterol, and 13.1 ± 3.4% ($P < 0.0003$) for apo B/A-1.

### Glycemic control

An improvement in glycemic control was observed on the KJM compared with WB-control treatment (Table 2). Serum fructosamine was reduced during the KJM treatment by 5.6 ± 1.5% ($P < 0.003$), compared with 0.39 ± 1.3% ($P = 0.77$) on control treatment, with a between-treatment difference of 5.2 ± 1.4% ($P < 0.002$). No significant between-treatment differences were seen for insulin or glucose concentrations. On KJM, however, fasting glycemia fell by 13 ± 2.5% ($P < 0.0001$) compared with 9.6 ± 4.3% ($P < 0.05$) on control.

### Blood pressure

No change in systolic or diastolic blood pressure was observed on either treatment or between treatments (Table 2).

All above results remained unchanged after adjustment for multiple comparisons by the Bonferroni-Hochberg procedure.

CONCLUSIONS — This preliminary study demonstrated that the addition of 0.5 g/100 kcal (8–13 g/day) of high-viscosity glucomannan in biscuit form to a high-carbohydrate/low-saturated fat NCEP Step 2 diet improved metabolic control beyond diet alone in individuals with the insulin resistance syndrome. We observed significant reductions in hyperglycemia as

Table 2—*Changes in serum lipids, glycemia, blood pressure, and body weight during and between the KJM and WB-control study periods in 11 subjects*

| | KJM | | | WB-control | | | Between treatments | |
|---|---|---|---|---|---|---|---|---|
| Risk factor | Week 0 | Week 3 | Change (%) | Week 0 | Week 3 | Change (%) | Change (%) | *P* |
| Cholesterol (mmol/l) | | | | | | | | |
| Total | 6.2 ± 0.3 | 5.0 ± 0.2 | −19 ± 2.69* | 6.0 ± 0.2 | 5.6 ± 0.2 | −6.3 ± 3.36 | −12.4 ± 3.1 | 0.0038* |
| LDL | 3.9 ± 0.2 | 2.8 ± 0.2 | −29 ± 3.37* | 3.8 ± 0.2 | 3.5 ± 0.2 | −6.6 ± 5.04 | −22.3 ± 3.9 | 0.0017* |
| HDL | 1.0 ± 0.1 | 0.9 ± 0.1 | −8.5 ± 2.19* | 1.0 ± 0.1 | 0.9 ± 0.1 | −9.6 ± 2.24* | 1.2 ± 2.2 | 0.9812 |
| Triglyceride (mmol/l) | 2.8 ± 0.2 | 3.0 ± 0.2 | 10.1 ± 9.92 | 2.9 ± 0.4 | 3.0 ± 0.3 | 12.1 ± 14 | −1.6 ± 10 | 0.7317 |
| Apolipoprotein (g/l) | | | | | | | | |
| Apo A-1 | 1.4 ± 0.1 | 1.4 ± 0.1 | −6.5 ± 2.46* | 1.5 ± 0.1 | 1.4 ± 0.1 | −4.8 ± 3.38 | −1.8 ± 3.1 | 0.3622 |
| Apo B | 1.6 ± 0.1 | 1.3 ± 0.1 | −19 ± 2.78* | 1.6 ± 0.1 | 1.5 ± 0.1 | −4.5 ± 4.47 | −15.1 ± 4.3 | 0.0003* |
| Lipid ratios | | | | | | | | |
| Total/HDL cholesterol | 6.5 ± 0.5 | 5.7 ± 0.4 | −11 ± 3.02* | 6.2 ± 0.4 | 6.4 ± 0.5 | 4.14 ± 4.16 | −15.2 ± 3.4 | 0.0023* |
| Apo B/apo A-1 | 1.1 ± 0.1 | 1.0 ± 0.1 | −13 ± 3.02* | 1.1 ± 0.1 | 1.1 ± 0.1 | 0.72 ± 3.61 | −13.1 ± 3.4 | 0.0002* |
| LDL/HDL | 4.2 ± 0.4 | 3.2 ± 0.3 | −22 ± 3.72* | 3.9 ± 0.3 | 3.9 ± 0.4 | 0.22 ± 6.27 | −22.2 ± 4.1 | 0.0012* |
| Glycemic control | | | | | | | | |
| Glucose (mmol/l) | 6.8 ± 0.5 | 5.9 ± 0.3 | −13 ± 2.48* | 6.6 ± 0.3 | 5.9 ± 0.4 | −9.6 ± 4.27 | −3.8 ± 3.6 | 0.7653 |
| Fructosamine (mmol/l) | 286 ± 13.6 | 269 ± 11.9 | −5.6 ± 1.46* | 279 ± 11.7 | 278 ± 12.6 | −0.39 ± 1.3 | −5.2 ± 1.4 | 0.0013* |
| Insulin (pmol/l) | 94.8 ± 16.6 | 91.1 ± 16.5 | 0.91 ± 8.88 | 99.2 ± 16.5 | 88.5 ± 11.4 | −3.0 ± 9.67 | 3.9 ± 8.9 | 0.9683 |
| Blood pressure (mmHg) | | | | | | | | |
| Systolic | 139 ± 2.0 | 135 ± 3.6 | −2.9 ± 1.88 | 135 ± 2.6 | 138 ± 3.7 | 2.2 ± 2.5 | −5.1 ± 2.2 | 0.448 |
| Diastolic | 85.4 ± 1.8 | 84.8 ± 1.5 | −0.26 ± 2.55 | 85.5 ± 1.7 | 86.5 ± 1.5 | 1.33 ± 1.49 | −1.4 ± 2.1 | 0.2647 |
| Body weight (kg) | 80.7 ± 5.1 | 80.6 ± 5 | −0.17 ± 0.14 | 81 ± 5.3 | 80.6 ± 5.1 | −0.29 ± 0.35 | 0.1 ± 0.2 | 0.5303 |

Data are means ± SEM, except for body weight, which is mean ± SD. Within-treatment differences (week 0 vs. week 3) were assessed by paired Student's *t* test and between-treatment differences by ANCOVA (general linear model procedure). *Significant after adjustment of $\alpha$ level by the Bonferroni-Hochberg procedure. Null-hypotheses were rejected only if the *P* values were less than their corresponding adjusted $\alpha$ level.

measured by the short-term marker of glycemic control, fructosamine, although the clinical significance of the observed changes remains to be demonstrated. We also observed significant reductions in hyperlipidemia as measured by total, LDL, LDL/HDL, and total/HDL cholesterol, and apo B and apo B/A-1, relative to a matched WB-control treatment. These findings represent the first to demonstrate such improvements using soluble fiber in subjects with this particular cluster of risk factors that also includes the intermediate diabetic classification, IGT.

Because of the strong implications of insulin resistance syndrome, a more aggressive approach has been suggested to achieve similar reductions. Diabetes and heart disease share common precursors for the development of atherosclerosis that often co-occur. Long before diabetes becomes manifest, the clustering of metabolic abnormalities exerts a synergistic effect on the atherosclerotic process (26). Based on findings from Trevisea and colleagues, cardiovascular disease (CVD) risk appears to increase linearly with an increase in the number of these risk factors. It is recommended therefore that insulin-resistant patients have their CHD risk factors managed as if they have established CHD (27).

Low-fat/high-carbohydrate diets may still have promise as a therapeutic approach. Although there has been a shift away from their advocacy in favor of those rich in monounsaturated fat (8), these diets supplemented with fiber may have similar metabolic advantages. Guar gum, pectin, oat products, and psyllium added to high-carbohydrate diets have been shown to improve total and LDL cholesterol significantly, with no improvement to triglycerides and slight or no adverse effects on HDL (16). Both guar (14) and KJM (17,18) supplementation have also been shown to improve other risk factors, including glycemia and blood pressure. This has led to support for the use of guar in the treatment of the insulin resistance syndrome (13). Evidence further suggests that supplementation with these soluble fibers may augment concurrent drug therapy. Improvements in these assorted risk factors following supplementation have been noticed beyond what was achieved by drugs alone in subjects receiving hypolipidemic (14,17,28), hypoglycemic (14,17,29), and hypotensive (17,30) medications.

The ability of soluble fiber to improve a high-carbohydrate/low-fat diet is supported by the findings of the present study. Total and LDL cholesterol were decreased and glycemic control was improved significantly. Also, although HDL, apo A-1, and triglycerides were unaffected, as has been noticed with other fibers, this was balanced by the significant improvements in the other lipid end points, leading to significant reductions in all three lipid ratios: total/HDL, LDL/HDL, and apo B/A-1. Similar improvements in these ratios have rarely been reported using dietary interventions (28,29). Overall, the suggestion is that an NCEP Step 2 diet supplemented with KJM may confer additional benefits over the Step 2 diet alone, benefits that may be comparable to strategies using monounsaturated fat.

KJM may be better suited than the other major soluble fibers to improving outcomes with high-carbohydrate/low-fat diets. Although meta-analyses use variance-adjusted values that tend to underestimate effectiveness, KJM can be compared to other soluble fibers in terms of its lipid-lowering ability per gram of fiber, using recent meta-analytical data (15). Daily intake of glu-

commanan from KJM on this and our previous study (17) produced an average net change in total and LDL cholesterol of −0.084 and −0.119 mmol/l per gram of fiber, respectively. These reductions represent approximately triple the lipid-lowering capacity of psyllium (−0.028 and −0.029 mmol/l, respectively), oat products (−0.037 and −0.032 mmol/l), and guar gum (−0.037 and −0.033 mmol/l) (15). In the case of pectin, they represent comparable total cholesterol-lowering capacity (−0.070 mmol/l) and approximately twice the LDL-lowering capacity (−0.055 mmol/l) (15). The very high viscosity of KJM may explain these differences. It has been shown to be approximately five times higher than that of guar gum (31) and beta-glucan (32), and considerably more than that of pectin (19).

Contributions made by KJM's rheological properties may offer insight into the proposed mechanism by which the KJM-supplemented biscuits had their beneficial effects. Possibilities for its lipid-lowering action may include an inhibition of cholesterol absorption in the jejunum (19) and bile acid absorption in the ileum (33) mediated by viscosity or less postprandial stimulation of 3-hydroxy 3-methylglutaryl CoA reductase (34). Other options include the generation of short-chain fatty acids, predominantly propionate, by colonic microflora that may decrease hepatic cholesterol synthesis (35). The improvement in glycemic control may be attributable to an effect of the gel-forming KJM on rate of digestion. It has been suggested that decreases in glucose and insulin levels after the consumption of water-soluble fibers are related to slower rates of food absorption in the small intestine associated with increased viscosity (16). This mechanism may explain why we observed a reduction in serum fructosamine, but did not observe concomitant reductions in fasting glycemia and insulinemia: KJM may be exerting its effect mainly postprandially.

In conclusion, the results from the current study support the role of KJM and other viscous dietary fiber as a means for improving high-carbohydrate diets in the amelioration of the insulin resistance syndrome. Improved metabolic control resulted in the correction of several risk factors that characterize the syndrome and figure prominently in the etiology of atherosclerotic CHD. Before the therapeutic potential of KJM can be fully realized in this or other situations, however, further controlled studies with larger sample sizes and of longer duration are required; these requirements are both major caveats of the present study. Determinations of the optimal fiber dose in different categories of subjects, of the rheological-biological relationship of KJM, and of KJM's effects on insulin sensitivity and thrombotic factors are also warranted.

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