Diet and Exercise in the Treatment of NIDDM

The need for early emphasis

R. James Barnard, PhD
Tiffany Jung, MPH
Stephen B. Inkeles, MD, MPH

OBJECTIVE — To investigate the effectiveness of an intensive diet and exercise program for controlling non-insulin-dependent diabetes mellitus (NIDDM) and reducing risk factors associated with macrovascular complications.

RESEARCH DESIGN AND METHODS — Medical charts obtained from 4,587 participants in a lifestyle modification program were screened for patients with NIDDM. A total of 652 patients was identified, and their responses to the 3-week program were analyzed.

RESULTS — Fasting glucose level was reduced from 10.0 to 8.45 mmol/l, and 71% of 197 subjects taking oral hypoglycemic agents and 39% of 212 taking insulin were able to discontinue their medication. Of the 243 not taking medication, 76% reduced their fasting glucose levels to ≤7.84 mmol/l. Blood pressure was significantly reduced, and of the 319 initially taking antihypertension drugs, 34% had their medication discontinued. Serum total and low-density lipoprotein cholesterol were reduced by 22% and triglycerides by 33%. The ratio of total to high-density lipoprotein cholesterol was reduced by 13%.

CONCLUSIONS — Lifestyle modification consisting of diet combined with aerobic exercise can be effective for controlling NIDDM and reducing risk factors associated with macrovascular complications in both men and women. The program was far more effective in controlling the disease in patients taking no medication or oral agents compared with patients taking insulin. These results stress the need for early emphasis on lifestyle modification in the treatment of NIDDM.

Non-insulin-dependent diabetes mellitus (NIDDM) is a catastrophic disease costing upwards of 20–30 billion dollars annually in the U.S. (1). A large part of the cost is the result of micro- and macrovascular complications resulting from diabetes. The microvascular complications and other problems, including neuropathy, are felt to be primarily the result of a lack of glycemic control. Macrovascular complications, however, are primarily the result of diabetes compounding the other common risk factors of hyperlipidemia, hypertension, and cigarette smoking. Several recent studies have shown that as serum cholesterol increases, so does coronary heart disease (CHD) mortality, and that in diabetic patients, the CHD mortality is 4–5 times greater at any level of cholesterol (2). Despite the fact that macrovascular complications are responsible for most of the premature mortality in diabetic patients, attention has generally been focused on controlling glycemia (3). Thus, it would appear that lifestyle modification to control diabetes and the other CHD risk factors may be effective in the long term for reducing the tremendous medical costs of the disease and its complications. In fact, lifestyle modification consisting of diet, regular aerobic exercise, and normalization of body weight has been emphasized by the American Diabetes Association and the National Institutes of Health as being important factors in the treatment of NIDDM (4,5).

The purpose of this study was to evaluate the response of a large number of NIDDM patients (n = 652) to an intensive diet and exercise program.

RESEARCH DESIGN AND METHODS — The subjects of this investigation were 652 participants (408 men and 244 women) ranging in age from 19 to 83 years, (mean = 59.4 ± 0.4 years) who attended the Pritikin Longevity Center 26-day residential program. The subjects studied were all the NIDDM patients in a group of 4,587 adults who attended the center between 1977 and 1988 (6). All participants signed a consent form permitting their data to be used for research purposes. The group included 212 patients taking insulin and 197 taking oral hypoglycemic agents. The remaining

From the Department of Physiological Science and Division of Clinical Nutrition, Department of Medicine, University of California, Los Angeles, California.

Address correspondence and reprint requests to R. James Barnard, PhD, Department of Physiological Science, 2322 Life Sciences, University of California, Los Angeles, 405 Hilgard Avenue, Los Angeles, CA 90024-1527.

Received for publication 18 January 1994 and accepted in revised form 30 June 1994.

NIDDM, non-insulin-dependent diabetes mellitus; CHD, coronary heart disease; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

Table 1—*Effects of diet and exercise on CHD risk factors in NIDDM patients according to medication status*

	Insulin (n = 212)		Oral medication (n = 197)		No medication (n = 243)	
	Before	After	Before	After	Before	After
Fasting glucose (mmol/l)	10.9 ± 0.3	10.2 ± 0.3	10.3 ± 0.3	8.5 ± 0.2	9.1 ± 0.2	6.9 ± 0.2
Patients taking medication	212	129	197	57	0	5
Total cholesterol (mmol/l)	6.19 ± 0.10	4.77 ± 0.88	6.14 ± 0.10	4.86 ± 0.08	6.19 ± 0.09	4.82 ± 0.07
LDL cholesterol (mmol/l)	4.15 ± 0.13	3.08 ± 0.11	4.05 ± 0.13	3.26 ± 0.10	4.17 ± 0.14	3.31 ± 0.10
HDL cholesterol (mmol/l)	1.06 ± 0.03	0.91 ± 0.02	1.01 ± 0.03	0.90 ± 0.02	1.04 ± 0.02	0.92 ± 0.02
Total cholesterol: HDL cholesterol	6.51 ± 0.26	5.64 ± 0.17	6.44 ± 0.22	5.70 ± 0.16	6.48 ± 0.22	5.57 ± 0.16
Triglycerides (mmol/l)	2.82 ± 0.16	1.98 ± 0.07	2.84 ± 0.15	1.96 ± 0.06	2.95 ± 0.11	1.84 ± 0.06
Systolic blood pressure (mmHg)	131.7 ± 1.4	122.0 ± 1.3	130.7 ± 1.4	122.3 ± 1.3	132.9 ± 1.2	125.0 ± 1.2
Diastolic blood pressure (mmHg)	77.3 ± 0.7	72.2 ± 0.6	78.1 ± 0.8	73.9 ± 0.8	82.1 ± 0.6	76.0 ± 0.6
Body weight (kg)	86.2 ± 1.4	81.9 ± 1.3	87.3 ± 1.4	82.8 ± 1.2	89.1 ± 1.3	84.6 ± 1.2

Data are means ± SE. Before, values before the program; after, values after the program. All values before the program were significantly different from values after it ($P < 0.001$).

243 were taking no diabetic medication but had a fasting glucose level ≥7.84 mmol/l (140 mg/dl).

The participants first registered at the center on a Sunday afternoon. After dinner, they attended an orientation in which they were given information about the program and instructions for a 12-h fast. On Monday morning, blood samples were obtained and analyzed for total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, and glucose. Low-density lipoprotein (LDL) cholesterol was calculated as follows: LDL cholesterol = total cholesterol − (HDL cholesterol + triglycerides/5). LDL cholesterol was not calculated when triglycerides were >4.52 mmol/l (400 mg/dl). Additional blood samples were drawn each subsequent Monday morning for the duration of each participant's stay at the center, and finger-stick glucose measurements were taken more frequently.

During the 26-day program, the participants were involved in daily aerobic exercise, primarily walking, and were placed on a high–complex-carbohydrate, high-fiber, low-fat, low-cholesterol, and low-salt diet. Of dietary calories, <10% were obtained from fat, 15% from protein, and the remainder from carbohydrate. The diet contained 35–40 g of dietary fiber per 420 kJ (1,000 kcal) and a total daily intake of no more than 4 g of sodium chloride and <25 mg of cholesterol. Caloric restriction is not emphasized but is a natural consequence of eating a high–complex-carbohydrate, high-fiber diet. Details of the exercise program and the diet, including computer analyses, as well as the educational program have been published previously (6–9).

To assess the effectiveness of the overall program, pre- and postprogram data were analyzed for significant differences within each subgroup using a repeated measures paired Student's t test (BMDP statistical software: program 3D). Additionally, the subgroups were checked for statistical differences before the start of the program and once again at the end using analysis of variance (BMDP statistical software: program 1V). Pearson correlation coefficients were determined for the changes in all parameters. Values are expressed as means ± SE, and P values <0.05 were considered statistically significant.

RESULTS— The pre- and postprogram data for the entire group of 652 participants were first analyzed, and all of the postprogram values were found to be significantly lower than the preprogram values ($P < 0.001$). After the analysis of the entire group, the participants were subdivided according to diabetic medication status at entry for further analyses (Table 1). The preprogram values were similar for all three groups except for fasting glucose and diastolic blood pressure, which were significantly elevated in the no medication group. After 3 weeks of the intervention, fasting glucose levels had been significantly reduced in all three groups ($P < 0.001$). However, the insulin group had the smallest drop. Initially, 212 patients were taking insulin, and at the time of discharge, insulin therapy had been stopped in 83 (39%) of the patients. Insulin therapy was stopped in a greater percentage of women (45%) compared with men (36%). Therapy was switched from insulin to oral agents in three patients. Initially, 197 patients (121 men and 76 women) were taking oral agents, and at discharge, 140 (71%) had their medication discontinued. Men responded a little better than women, in that in 75% vs. 64%, oral agent therapy was discontinued. Therapy in two patients was switched from oral agents to insulin because their glucose levels were out of control and did not respond to the intervention. Of the 243 patients not taking medication upon entry into the program, in 185 (76%) fasting glucose level dropped to <7.84 mmol/l by discharge. In five of the patients, oral agent therapy was started because their fasting glucose levels were >11.2 mmol/l at discharge. Those whose fasting glucose levels were still >7.84 mmol/l were encouraged to continue the program at home but to immediately check with their personal physicians.

Serum lipid levels were significantly reduced for all three groups. Total cholesterol reductions ranged from 21% for the oral medication group to 23% for the insulin group. LDL cholesterol showed similar reductions ranging from 20% for the oral medication group to 26% for the insulin group. HDL cholesterol showed small reductions ranging from 11 to 14%, with the biggest drop in the insulin group. Despite the drop in HDL cholesterol, the ratio of total cholesterol to HDL cholesterol was reduced in all three groups. Triglycerides showed major reductions, ranging from 30% for the insulin group to 38% for the no medication group.

Blood pressure, both systolic and diastolic, was reduced in all three groups. In addition to the drop in blood pressure, in 25 of 91 (27%) in the insulin group, 43 of 99 (43%) in the oral medication group, and 39 of 129 (30%) in the no diabetic medication group, antihypertensive drug therapy was discontinued. Body weight was reduced by similar amounts in three groups ranging from 4.3 to 4.5 kg. For the entire group, the change in body weight was not correlated with the change in fasting glucose ($r = 0.03$), total cholesterol ($r = 0.04$), or LDL cholesterol ($r = 0.05$). Low but significant ($P = 0.05$) correlations were found for the change in body weight and the change in triglycerides ($r = 0.16$), HDL cholesterol ($r = 0.13$), and systolic ($r = 0.13$) and diastolic ($r = 0.11$) blood pressure.

CONCLUSIONS — The results of this study show that a very low-fat, high–complex-carbohydrate diet combined with aerobic exercise, primarily walking, can be effective for controlling diabetes and reducing serum lipid levels and other CHD risk factors. The ability of this lifestyle modification program to control diabetes was far more effective in patients either taking no medication or oral hypoglycemic agents compared with those taking insulin. These results are similar to those reported by Nagulesparan et al. (10) using caloric restriction. Although caloric restriction does work, it is difficult to sustain by simply reducing the total amount of food consumed. However, the very low-fat, high–complex-carbohydrate diet leads to a natural caloric restriction because of the bulk of the low-calorie foods. This has been documented in native Hawaiians with obesity and diabetes when they were switched from the typical U.S. high-fat, refined sugar diet to a traditional native Hawaiian diet low in fat and high in complex carbohydrates (11). Several other small studies have also documented the value of a very low-fat, high–complex-carbohydrate diet for controlling diabetes and eliminating the need for medication (12). Our 2- to 3-year follow-up study of some of the patients in this study showed that a majority of patients continued the program and kept their diabetes under control (7).

In a more recent study using a less stringent fat-modified, high-fiber diet, Hanefeld et al. (13) reported that in patients with newly diagnosed NIDDM, 72% were able to control the disease over a 5-year period, but 47% of the control group required antidiabetic drugs. Despite the fact that diabetes was controlled in many patients with the fat-modified diet, serum lipid levels were unchanged, and there was no difference in the incidence of myocardial infarction between the two groups. Conversely, in this study, major reductions were observed in serum lipid levels. Believing that such severe fat restriction as used in this study is not practical, Garg (14) has recommended the use of a diet high in monounsaturated fat. Although replacing saturated fat with monosaturated fat will reduce total cholesterol and especially LDL cholesterol, the effect of these diets on glycemic control has not been adequately evaluated. Most of the studies reviewed by Garg held calories constant, which is inappropriate because it has been well documented that eating high–complex-carbohydrate, high-fiber diets ad libitum will result in a lower calorie intake. This is especially beneficial in the long term for weight loss, which is one of the primary goals for treating the majority of patients with NIDDM. In addition, there is evidence indicating that high–monounsaturated fat diets increase platelet aggregation, but high–complex-carbohydrate diets have the opposite effect (9,15).

Even though the optimal diet has not been determined, the results of this study indicate a real need for strong emphasis on lifestyle modification consisting of both diet and exercise in the treatment of NIDDM.

References

1. Elixhauser A: The cost-effectiveness of preventive care for diabetes mellitus. *Diabetes Spectrum* 2:349–353, 1989
2. Bierman EL: Atherogenesis in diabetes. *Arterioscler Thromb* 12:647–656, 1992
3. Stern MP, Patterson JK, Haffner SM, Hazuda HP, Mitchell BD: Lack of awareness and treatment of hyperlipidemia in type II diabetes in a community survey. *JAMA* 262:360–364, 1989
4. American Diabetes Association: Clinical practice recommendations. *Diabetes Care* 14 (Suppl. 2), 1991
5. National Institutes of Health: Consensus Development Conference on Diet and Exercise in Non-Insulin-Dependent Diabetes Mellitus. *Diabetes Care* 10:639–644, 1987
6. Barnard RJ: Effects of life-style modification on serum lipids. *Arch Intern Med* 151: 1389–1394, 1991
7. Barnard RJ, Massey MR, Cherny S, O'Brien LT, Pritikin N: Long-term use of a high-complex-carbohydrate, high-fiber, low-fat diet and exercise in the treatment of NIDDM patients. *Diabetes Care* 6:268–273, 1983
8. Barnard RJ, Ugianskis EJ, Martin DA, Inkeles SB: Role of diet and exercise in the management of hyperinsulinemia and as-

sociated atherosclerotic risk factors. *Am J Cardiol* 69:440–444, 1992
9. Barnard RJ, Hall JA, Chaudhari A, Miller JE, Kirscheubaum MA: Effects of a low-fat, low-cholesterol diet on serum lipids, platelet aggregation and thromboxane formation. *Prostaglandins Leukotrienes Med* 26:241–252, 1987
10. Nagulesparan M, Savage PJ, Bennion LJ, Unger RH, Bennett PH: Diminished effect of caloric restriction on control of hyperglycemia with increasing duration of type II diabetes mellitus. *J Clin Endocrinol Metab* 53:560–568, 1981
11. Shintani TT, Hughes CK, Beckman S, O'Connor HK: Obesity and cardiovascular risk intervention through the ad libitum feeding of traditional Hawaiian diet. *Am J Clin Nutr* 53 (Suppl 6):16475–16515, 1991
12. Anderson JW, Gustafson NJ, Bryant CA, Tietyen-Clark J: Dietary fiber and diabetes: a comprehensive review and practical application. *J Am Diet Assoc* 87:1189–1197, 1987
13. Hanefeld M, Fischer S, Schnechel H, Rothe G, Schulze J, Dude H, Schwanebeck U, Julius U: Diabetes intervention study: multi-intervention trial in newly diagnosed NIDDM. *Diabetes Care* 14: 308–317, 1991
14. Garg A: High-monounsaturated fat diet for diabetic patients. *Diabetes Care* 17: 242–246, 1994
15. Schick PK, Wojenski CM, Walker J: The effect of olive oil, hydrogenated palm oil, and ω-3 fatty acid-enriched diets on megakaryocytes and platelets. *Arterioscler Thromb* 13:84–89, 1993