The Finnish Diabetes Prevention Study (DPS)

Lifestyle intervention and 3-year results on diet and physical activity

JAANA LINDSTRÖM, MSC[1]
ANNE LOUHERANTA, PHD[2]
MARJO MANNELIN, MSC[3]
MERJA RASTAS, MSC[4]
VIRPI SALMINEN, MSC[5]

JOHAN ERIKSSON, MD, PHD[1]
MATTI UUSITUPA, MD, PHD[2]
JAAKKO TUOMILEHTO, MD, PHD[1,6]
FOR THE FINNISH DIABETES PREVENTION
  STUDY GROUP

OBJECTIVE — To describe the *1)* lifestyle intervention used in the Finnish Diabetes Prevention Study, *2)* short- and long-term changes in diet and exercise behavior, and *3)* effect of the intervention on glucose and lipid metabolism.

RESEARCH DESIGN AND METHODS — There were 522 middle-aged, overweight subjects with impaired glucose tolerance who were randomized to either a usual care control group or an intensive lifestyle intervention group. The control group received general dietary and exercise advice at baseline and had an annual physician's examination. The subjects in the intervention group received additional individualized dietary counseling from a nutritionist. They were also offered circuit-type resistance training sessions and advised to increase overall physical activity. The intervention was the most intensive during the first year, followed by a maintenance period. The intervention goals were to reduce body weight, reduce dietary and saturated fat, and increase physical activity and dietary fiber.

RESULTS — The intervention group showed significantly greater improvement in each intervention goal. After 1 and 3 years, weight reductions were 4.5 and 3.5 kg in the intervention group and 1.0 and 0.9 kg in the control group, respectively. Measures of glycemia and lipemia improved more in the intervention group.

CONCLUSIONS — The intensive lifestyle intervention produced long-term beneficial changes in diet, physical activity, and clinical and biochemical parameters and reduced diabetes risk. This type of intervention is a feasible option to prevent type 2 diabetes and should be implemented in the primary health care system.

Diabetes Care 26:3230–3236, 2003

T he Finnish Diabetes Prevention Study (DPS) was one of the first controlled, randomized studies to show that type 2 diabetes is preventable with lifestyle intervention (1). The risk of diabetes was reduced by 58% in the intensive lifestyle intervention group compared with the control group. These results have been reproduced by the Diabetes Prevention Program (DPP), in which lifestyle intervention, with a similar 58% risk reduction, was superior to the metformin treatment (2). The lifestyle intervention used in the DPP was not designed to be used in community settings (3), whereas one of the main objectives in the DPS was to test an intervention feasible in primary health care.

In this study, we describe the lifestyle intervention program used in the DPS, the changes in dietary habits and exercise behavior that were achieved during the first year and the maintenance of these changes after 3 years, and assess the efficacy of the intervention on body weight, plasma glucose, and lipids.

RESEARCH DESIGN AND METHODS

— The DPS was a multicenter study with five participating centers in Helsinki, Kuopio, Turku, Tampere, and Oulu. The study protocol was approved by the ethics committee of the National Public Health Institute in Helsinki, Finland. Each study center employed a physician, study nurse, and nutritionist (MSc in nutrition) on a part-time basis. Either an exercise instructor/physiotherapist was a member of the study team or these services were provided commercially.

Study subjects were recruited mainly by screening high-risk groups such as first-degree relatives of type 2 diabetes patients who voluntarily responded to local advertisements or were identified in earlier epidemiological surveys. The inclusion criteria were *1)* age 40–64 years at screening, *2)* BMI >25 kg/m^2 at screening, and *3)* the mean value of two 75-g oral glucose tolerance tests (OGTTs) in the impaired glucose tolerance range based on World Health Organization criteria (4). The randomization of participants ($n = 522$) started in 1993 and continued until 1998.

At the first study visit after the screening phase, the subjects were randomly allocated to the intervention group or the usual care control group. Randomization

was stratified by center, sex, and 2-h plasma glucose value (1,5).

The study was prematurely terminated in March 2000 by an independent end point committee, since the incidence of diabetes in the intervention group was highly significantly lower than in the control group (1). However, the follow-up according to the original protocol was continued until each subject's following annual visit. Therefore, the number of subjects who reached the 3-year visit in this report is somewhat higher (n = 434) than the number (n = 374) reported earlier (1).

Background for lifestyle intervention

The main goals of the lifestyle intervention were based upon available evidence on diabetes risk factors (6–9). They were weight reduction ≥5%, moderate intensity physical activity ≥30 min/day, dietary fat <30 proportion of total energy (E%), saturated fat <10 E%, and fiber ≥15 g/1,000 kcal.

The hypothesis of diabetes prevention by increased physical activity had already been tested in the Malmö feasibility study (10) with encouraging results. A nonrandomized pilot study testing the effect of aerobic and resistance training exercise programs on insulin resistance in subjects with impaired glucose tolerance was completed before the beginning of the DPS (11). On the basis of this pilot study, resistance training was offered to the participants of the DPS intervention.

To further increase the knowledge of a suitable diet for long-term intervention, a dietary pilot study was completed in a subgroup of subjects before the start of the DPS study (12). The results suggested that a monounsaturated fat–enriched diet improves glucose metabolism when consumed after a diet rich in saturated fat compared with a reduced-fat, polyunsaturated fat–enriched diet. Therefore, besides a moderate-fat (<30 E%), low–saturated fat (<10 E%) diet, a diet with somewhat more monounsaturated fat (total fat not exceeding 35 E%) was also considered acceptable.

At the nutritional counseling sessions, the intervention goals were translated into practice. General examples of this translation are presented in an online appendix (available at http://care.diabetes journals.org).

Intervention group
Dietary intervention. The participants had face-to-face consultation sessions (from 30 min to 1 h) with the study nutritionist at weeks 0, 1–2, and 5–6 and at months 3, 4, 6, and 9, i.e., altogether seven sessions during the first year and every 3 months thereafter. The first year sessions had a preplanned topic (e.g., diabetes risk factors, saturated fat, fiber, physical activity, and problem solving), but the discussions were individualized, focusing on specific individual problems. Printed material was used to illustrate the message and to serve as a reminder at home. In addition, there were voluntary group sessions, expert lectures, low-fat cooking lessons, visits to local supermarkets, and between-visit phone calls and letters. The goal was to equip the subjects with necessary knowledge and skills and to achieve gradual, permanent behavioral changes. A change in behavior was considered a process, as suggested by Prochaska (13). The dietary advice was based on 3-day food records, which were completed four times yearly. Nutrient intakes were calculated, and a summary of the results was given and explained to the subjects. Subjects were encouraged to make intermediate goals for themselves by thinking about practical things they could try to change (e.g., instead of an abstract goal such as "increase fiber intake," a practical goal would be "eat a slice of rye bread on every meal"). Weight was measured at every visit, and a weight chart was drawn. The participants were also encouraged to measure and record their weight at home on a regular basis. Recommended weight loss was not more than 0.5 to 1 kg per week. The spouse was invited to join the sessions, especially if he or she was the one responsible for shopping and cooking in the family. After 6 months, the use of a very-low-calorie diet (VLCD) for 2–5 weeks or as a substitute for one to two meals per day was considered, if preferred by the subject, to boost weight loss. Altogether, 48 subjects participated in the VLCD groups arranged as part of the intensive intervention.
Exercise intervention. The subjects were individually guided to increase their overall level of physical activity. This was done by the nutritionist during the dietary counseling sessions and highlighted by the study physicians at the annual visits. Endurance exercise was recommended to increase aerobic capacity and

cardiorespiratory fitness. Supervised, progressive, individually tailored circuit-type moderate intensity resistance training sessions to improve the functional capacity and strength of the large muscle groups of the upper and lower body were also offered free of charge. As a means for improving motivation, an "exercise competition" between the five study centers was organized twice during the study period. Voluntary group walking and hiking were also organized.

Education program for the control group
At baseline, the control group was given general information about lifestyle and diabetes risk. This was done either individually or in one group session (30 min to 1 h), and some printed material was delivered. The message to reduce weight, increase physical activity, and make qualitative changes in diet was the same as for the intervention group subjects, but counseling was not individualized.

Baseline and annual measurements
All study subjects had an annual OGTT, a medical history, and a physical examination with measurements of height, weight, and waist circumference. These procedures have been described in detail previously (1,5). Plasma, serum, or capillary glucose was determined locally according to standard guidelines and corrected by linear regression equation using values measured in the National Public Health Institute central laboratory (60–80 duplicate plasma samples per center) as the dependent variable. The second screening OGTT was considered the glucose baseline. Diabetes diagnosis had to be confirmed by a second OGTT and induced termination of the study. Serum total cholesterol, HDL cholesterol, and triglycerides were determined using an enzymatic assay method. HbA_{1c} was analyzed using the Bayer DCA2000 Analyzer.

Those study subjects who terminated the follow-up prematurely were defined as dropouts. Nevertheless, data from their earlier visits were included in the analyses. There were 8 and 6 subjects who did not attend the 1-year annual visit and an additional 9 and 9 who did not attend the 3-year visit in the intervention and control groups, respectively.

Table 1—*Baseline characteristics*

	Intervention group	Control group
n (male/female)	265 (91/174)	257 (81/176)
Age (years)	55 ± 7	55 ± 7
First-degree relatives with diabetes (%)	66	61
Schooling in years (%)		
0–9	40	40
10–12	27	27
≥13	33	33
Type of work (%)		
Agricultural or industrial	9	10
Office work or student	42	44
Homemaker, retired, or unemployed	49	49
Weight (kg)	86.7 ± 14.0	85.5 ± 14.4
BMI (kg/m^2)	31.4 ± 4.5	31.1 ± 4.5
Waist circumference (cm)	102.0 ± 11.0	100.5 ± 10.9
Fasting plasma glucose (mmol/l)	6.1 ± 0.8	6.2 ± 0.7
2-h plasma glucose (mmol/l)	8.9 ± 1.5	8.9 ± 1.5
HbA$_{1c}$ (%)	5.7 ± 0.6	5.6 ± 0.6
Serum total cholesterol (mmol/l)	5.6 ± 1.0	5.6 ± 0.9
Serum HDL cholesterol (mmol/l)	1.2 ± 0.3	1.2 ± 0.3
Serum total cholesterol–to–HDL cholesterol ratio	4.9 ± 1.3	4.8 ± 1.2
Serum triglycerides (mmol/l)	1.7 ± 0.8	1.7 ± 0.8
Blood pressure–lowering medication (%)	29	31
Lipid-lowering medication (%)	5	7
Total LTPA (min/week)	339 (193–545)	329 (173–586)
Moderate-to-vigorous LTPA (min/week)	156 (62–288)	169 (65–352)
Energy (kcal/day)	1,771 ± 520	1,744 ± 527
Carbohydrates		
E%	43.6 ± 7.5	43.2 ± 6.7
g	190 ± 57	185 ± 54
Fat		
E%	36.0 ± 6.7	37.1 ± 6.5
g	72 ± 28	73 ± 29
Saturated fat (E%)*	16.2 ± 4.0	17.0 ± 4.3
Monounsaturated fat (E%)	12.9 ± 2.8	13.0 ± 2.9
Polyunsaturated fat (E%)	5.7 ± 1.7	5.8 ± 2.2
Alcohol		
E%	2.8 ± 5.1	2.1 ± 4.1
g	8 ± 17	6 ± 12
Cholesterol (mg)	312 ± 137	304 ± 130
Fiber		
g	20 ± 7	20 ± 8
g/1,000 kcal	11.7 ± 4.0	11.7 ± 3.9
Water soluble (g)	4.6 ± 1.7	4.6 ± 1.6
Water soluble (g/1,000 kcal)	2.7 ± 1.0	2.7 ± 0.9
Water insoluble (g)	14.4 ± 5.4	14.1 ± 5.9
Water insoluble (g/1,000 kcal)	8.4 ± 2.9	8.4 ± 2.9

Data are mean ± SD or median (interquartile range). *Difference between the groups (Student's unpaired *t* test), *P* = 0.0188.

Assessment of dietary intake
Study subjects were asked to complete a 3-day food record at baseline (before the randomization visit) and before every annual visit. A picture booklet of portion sizes of typical foods was used to estimate the amount of food consumed (14). The nutrient intakes were calculated using a dietary analysis program developed in the National Public Health Institute (15). The program allows modification of database recipes, so that the subjects could account for changes made to traditional recipes (e.g., the use of skim instead of whole milk). For this report, the most recent nutrient content dataset was used, and therefore the nutrient intake results are slightly different from those reported previously (1).

Assessment of physical activity
The study subjects completed the validated Kuopio Ischaemic Heart Disease Risk Factor Study 12-month Leisure-Time Physical Activity (LTPA) questionnaire (16,17) at baseline and at every annual visit. In this detailed quantitative questionnaire, subjects estimated the frequency (times per month), duration (minutes), and intensity (0, recreational and outdoor activities; 1, conditioning exercise; 2, brisk conditioning exercise; and 3, competitive strenuous exercise) of their most common lifestyle and structured LTPA over the previous 12 months. Common moderate- or high-intensity physical activity included brisk walking, skiing, jogging, swimming, bicycling, gymnastics, resistance training, and ball games (if reported intensity ≥1); gardening and snow shoveling, hunting, picking berries, and gathering mushrooms (if reported intensity ≥2); fishing, hobby crafts, and repairs and house work (if reported intensity = 3); and rowing, forest work, and wood cutting (if reported intensity ≥1). The duration (minutes per week) of total physical activity and moderate- and high-intensity LTPA were calculated.

Statistical analysis
Differences between the intensive intervention and control group were tested for statistical significance with Student's *t* test (approximately normally distributed variables), Mann-Whitney nonparametric test, χ^2 test, or ANCOVA adjusting for baseline value, using SAS software (SAS Institute, Cary, NC) version 8.2.

RESULTS

Baseline results
At baseline, the study groups were similar in terms of characteristics reflecting increased diabetes risk (Table 1). Of all study subjects, 55% had a BMI >30 kg/m^2. The median amount of at least moderate-intensity LTPA was 160 min/week. In their spare time, 34% of the study subjects reported that they mostly read, watched TV, and spent time in other ways

Table 2—*Changes in physical activity and nutrient intakes from baseline to years 1 and 3*

	From baseline to year 1			From baseline to year 3		
	Intervention group	Control group	P^*	Intervention group	Control group	P^*
n	256	250		231	203	
Total LTPA (min/week)	16 (−126 to 115)	21 (−133 to 138)	0.9045	50 (−126 to 115)	23 (−142 to 171)	0.2415
Moderate-to-vigorous LTPA (min/week)	49 (−41 to 140)	14 (−47 to 90)	**0.0073**	61 (−33 to 168)	6 (−91 to 104)	**0.0057**
Energy (kcal/day)	−247 ± 438	−108 ± 464	**0.0001**	−204 ± 489	−97 ± 458	**0.0067**
Carbohydrates						
E%	3.3 ± 8.1	1.7 ± 7.3	**0.0023**	3.3 ± 8.0	2.0 ± 7.6	**0.0070**
g	−13 ± 52	−4 ± 48	0.0590	−11 ± 57	−1 ± 50	0.1042
Fat						
E%	−3.4 ± 8.2	−2.1 ± 7.6	**0.0002**	−4.7 ± 7.7	−3.2 ± 7.5	**<0.0001**
g	−16 ± 25	−8 ± 28	**<0.0001**	−16 ± 27	−11 ± 27	**0.0003**
Saturated fat (E%)	−2.7 ± 4.6	−1.2 ± 5.1	**<0.0001**	−3.2 ± 4.5	−1.9 ± 4.9	**<0.0001**
Monounsaturated fat (E%)	−0.8 ± 3.8	−0.4 ± 3.4	**0.0257**	−1.0 ± 3.6	−0.6 ± 3.5	**0.0453**
Polyunsaturated fat (E%)	−0.0 ± 2.1	−0.2 ± 2.5	0.5020	0.0 ± 2.4	−0.4 ± 2.3	0.0872
Alcohol						
E%	−1.1 ± 4.4	−0.3 ± 4.6	0.1452	−0.2 ± 4.1	−0.2 ± 4.3	0.6836
g	−4 ± 15	−1 ± 13	0.1168	−0 ± 14	−0 ± 14	0.8580
Cholesterol (mg)	−69 ± 138	−28 ± 148	**0.0005**	−63 ± 167	−31 ± 155	0.0586
Fiber						
g	1 ± 7	0 ± 7	0.1146	1 ± 8	1 ± 7	0.4393
g/1,000 kcal	2.5 ± 4.6	0.7 ± 4.1	**<0.0001**	2.4 ± 4.7	1.1 ± 4.1	**0.0013**
Water soluble (g)	0.2 ± 1.7	0.5 ± 1.5	0.2218	0.1 ± 1.7	0.1 ± 1.5	0.7275
Water soluble (g/1,000 kcal)	0.5 ± 1.1	0.2 ± 1.0	**<0.0001**	0.5 ± 1.1	0.2 ± 1.0	**0.0050**
Water insoluble (g)	0.6 ± 5.5	0.0 ± 5.5	0.1136	0.7 ± 5.9	0.4 ± 5.7	0.5608
Water insoluble (g/1,000 kcal)	1.7 ± 3.3	0.5 ± 3.0	**<0.0001**	1.7 ± 3.4	0.9 ± 3.1	**0.0034**

Data are means ± SD or median (interquartile range). *ANCOVA adjusting for baseline value or Mann-Whitney test for two unpaired groups. Values in bold are statistically significant.

that are not physically demanding; these individuals were considered sedentary (not shown). Dietary intake of both macro- and micronutrients were similar between the groups except for the energy proportion of saturated fat, which was slightly higher in the control group.

1- and 3-year results
Physical activity. The proportion of sedentary individuals was 14 and 30% at year 1 (*P* < 0.0001 for difference between groups) and 17 and 29% at year 3 (*P* = 0.0028) in the intervention and control groups, respectively. However, the total amount of reported time spent physically active did not change, but moderate-to-vigorous LTPA increased in the intervention group compared with the control group at years 1 and 3 (Table 2).
Dietary intake. The E% of carbohydrates increased; the E% of fat, saturated fat, and also monounsaturated fat decreased; the fiber density increased; and cholesterol intake decreased more in the

intervention group compared with the control group during the first year (Table 2). The absolute amounts of fat decreased, and energy intake decreased in the intervention group more than in the control group. At 3 years the reductions in energy, fat (E% and g), saturated fat (E%), and monounsaturated fat (E%), and the increase in carbohydrates (E%) and fiber density were still statistically significantly greater in the intervention group.

The specific goals of the intervention were more often reached by the intervention group than the control group subjects. Of the intervention and control group subjects, 46 and 14% managed to lose ≥5% of weight during the first year (*P* < 0.0001), the fat intake goal was reached by 37 and 20% (*P* < 0.0001), the saturated fat intake goal was reached by 21 and 9% (*P* < 0.0001), and the fiber density goal was reached by 37 and 23% (*P* < 0.0006), respectively. In general, women tended to change their diet more than men, but there was no interaction

between group assignment and sex (not shown).
Clinical and metabolic parameters. Several beneficial changes in clinical and metabolic parameters were observed in the intervention group compared with the control group at year 1 and still at the year 3 examination (Table 3). Mean weight reduction was 4.5 kg in the intervention group and 1.0 kg in the control group at 1 year. Some regain of weight appeared during the following 2 years. After exclusion of those intervention group subjects who took part in the VLCD group (whose weight reduction was 6.2 ± 7.0 kg at year 1 and 4.8 ± 7.2 kg at year 3), the mean weight reduction was 4.1 ± 4.3 kg at year 1 and 3.2 ± 4.5 kg at year 3 (*P* < 0.0001 compared with the control group).

Significantly greater improvements were seen at year 1 in fasting plasma glucose (−0.2 vs. 0.0 mmol/l), 2-h plasma glucose (−0.9 vs. −0.3 mmol/l), HbA$_{1c}$ (−0.1 vs. 0.1%), serum total cholesterol–

Table 3—*Changes in clinical and metabolic characteristics from baseline to years 1 and 3*

	From baseline to year 1			From baseline to year 3		
	Intervention group	Control group	P*	Intervention group	Control group	P*
n	256	250		231	203	
Weight						
kg	−4.5 ± 5.0	−1.0 ± 3.7	**<0.0001**	−3.5 ± 5.1	−0.9 ± 5.4	**<0.0001**
%	−5.1 ± 5.3	−1.1 ± 4.2	**<0.0001**	−4.0 ± 5.8	−1.1 ± 6.2	**<0.0001**
BMI (kg/m^2)	−1.6 ± 1.8	−0.4 ± 1.3	**<0.0001**	−1.3 ± 1.9	−0.3 ± 2.0	**<0.0001**
Waist circumference (cm)	−4.4 ± 5.2	−1.3 ± 4.8	**<0.0001**	−3.3 ± 5.7	−1.2 ± 5.9	**0.0005**
Fasting plasma glucose (mmol/l)	−0.2 ± 0.7	0.0 ± 0.7	**<0.0001**	−0.0 ± 0.7	0.1 ± 0.7	0.0664
2-h plasma glucose (mmol/l)	−0.9 ± 1.9	−0.3 ± 2.2	**0.001**	−0.5 ± 2.4	−0.1 ± 2.2	0.0664
HbA$_{1c}$ (%)	−0.1 ± 0.7	0.1 ± 0.6	**0.0003**	−0.2 ± 0.6	0.0 ± 0.6	**0.002**
Serum total cholesterol (mmol/l)	−0.1 ± 0.7	−0.1 ± 0.7	0.5097	−0.1 ± 0.9	0.1 ± 0.8	0.0712
Serum HDL cholesterol (mmol/l)	0.05 ± 0.19	0.02 ± 0.17	0.0681	0.14 ± 0.20	0.11 ± 0.19	0.1354
Serum total cholesterol–to–HDL cholesterol ratio	−0.4 ± 0.8	−0.1 ± 0.8	**0.0011**	−0.6 ± 0.9	−0.3 ± 0.8	**0.0009**
Serum triglycerides (mmol/l)	−0.2 ± 0.6	−0.0 ± 0.7	**<0.0001**	−0.1 ± 0.6	−0.0 ± 0.8	**0.024**

Data are means ± SD. *ANCOVA adjusting for baseline value. Values in bold are statistically significant.

to–HDL cholesterol ratio (−0.4 vs. −0.1), and serum triglycerides (−0.2 vs. −0.0 mmol/l) in the intervention group compared with the control group. During the first 3 years of the study, 22 subjects (9%) in the intervention group and 51 (20%) in the control group developed diabetes ($P = 0.0001$, χ^2 test). The use of VLCD preparations did not affect diabetes risk; 8% of the users developed diabetes in 3 years.

CONCLUSIONS — It is evident that lifestyle intervention can prevent or at least postpone type 2 diabetes (1,2,18) and should therefore be implemented in primary health care. However, the lifestyle interventions used have been either insufficiently described (18) or not intended as conventional treatment of high-risk individuals (3). In this study we describe the intervention used in the DPS in detail in order to enable utilization of our experience by others.

The intensive lifestyle intervention induced several beneficial changes in diet, physical activity, blood glucose, and lipid concentrations and a highly significant reduction in diabetes incidence. The intervention program was most intensive during the first year, and consequently the changes in clinical characteristics were most prominent after the first year. The effect of intervention, e.g., the differences between the intensive intervention and the control groups, was somewhat attenuated at 3 years, but this result may be

biased due to the study design. Those subjects who developed diabetes during the first 2 years of the trial did not have a 3-year examination; the majority belonged to the control group. Furthermore, the control group subjects were actually given a "mini-intervention" and therefore were not a true nontreatment group.

The weight reduction during the study was modest but comparable with that of other studies (10,19–21) on subjects with the metabolic syndrome, impaired glucose tolerance, or type 2 diabetes and using lifestyle approach to weight reduction. In the DPP study, a highly intensive and therefore expensive lifestyle intervention (3,22) produced a 5.6-kg weight reduction during the first year of intervention, with slight, gradual regain at the end of the study, i.e., 4 years (2).

Maintenance of the weight reduction after the 1-year intensive intervention period was satisfactory. It has been speculated that greater initial weight loss achieved with the use of VLCD preparations or with drugs might predict better long-term results (23). In our study, those not satisfied with the weight reduction achieved with the core program benefited from supplementary VLCD treatment, and their diabetes incidence was similar to those not using VLCD.

Independent of weight loss, calorie restriction results in improved glucose metabolism in subjects with type 2 diabe-

tes (24,25). It can be speculated that small negative energy balance sustained for a lengthy time period could be more advantageous to glucose tolerance than similar weight loss achieved with strict, short-term energy restriction. Therefore, in the long run, a lifestyle-intervention approach to weight control rather than a weight-reduction diet might be a more cost-efficient way to manage overweight in individuals at high risk for diabetes.

We used one-to-one intervention to individualize the intervention and because the recruitment and screening of study subjects was gradual. Group care may have some benefits in the form of social support and would probably be more cost-efficient in the clinical setting, where there is no need for accurate collection of food intake data.

The habitual nutrient intakes of the study subjects were estimated using 3-day food records, which is a reliable method in analyzing dietary intakes of groups. A well-known phenomenon in dietary analyses is underreporting, which is even more prominent in overweight individuals (26). The relatively low energy intakes indicate that some underreporting has taken place in our study. On the other hand, our analyses are based on differences between the two groups, and underreporting is presumably similar in both groups. Moreover, under- and overreporting has been shown to be subject specific (27), and as we measure changes

in dietary intake within a person, the bias may be attenuated.

The frequency, duration, and intensity of leisure time and lifestyle physical activity during the preceding 12 months were estimated by the study subjects at each annual visit. It was not straightforward and may have been incomplete due to difficulty recollecting, especially occasional activities. The majority of the study subjects considered themselves at least moderately active (≥4 h/week of some kind of physical activity), but based on the LTPA questionnaire most of their activity was low intensity. The reported time spent on moderate- and high-intensity LTPA increased slightly in the intervention group compared with the control group. If other types of supervised exercise in addition to the circuit-type resistance training sessions had been offered, then the increment might have been higher.

The main finding in the DPS was that nonpharmacologic lifestyle intervention of people at high risk for diabetes does prevent or at least postpone the onset of type 2 diabetes. The observed difference in incidence between the intensive intervention and the usual care control group indicates that the intervention needs to be individualized and continuing and performed by skilled professionals in order to be effective. The lifestyle intervention program used in the DPS is practical and can be implemented in primary health care systems.

References

1. Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, Keinänen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001

2. The Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002

3. The Diabetes Prevention Program Research Group: The Diabetes Prevention Program (DPP): description of lifestyle intervention. *Diabetes Care* 25:2165–2171, 2002

4. World Health Organization: *Diabetes Mellitus: Report of a WHO Study Group*. Geneva, World Health Org., 1985 (Tech. Rep. Ser., no. 727)

5. Eriksson J, Lindström J, Valle T, Aunola S, Hämäläinen H, Ilanne-Parikka P, Keinänen-Kiukaanniemi S, Laakso M, Laukkonen M, Lehto P, Lehtonen A, Louheranta A, Mannelin M, Martikkala V, Rastas M, Sundvall J, Turpeinen A, Viljanen T, Uusitupa M, Tuomilehto J: Prevention of type II diabetes in subjects with impaired glucose tolerance: the Diabetes Prevention Study (DPS) in Finland: study design and 1-year interim report on the feasibility of the lifestyle intervention programme. *Diabetologia* 42:793–801, 1999

6. Ohlson LO, Larsson B, Svardsudd K, Welin L, Eriksson H, Wilhelmsen L, Björntorp P, Tibblin G: The influence of body fat distribution on the incidence of diabetes mellitus: 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* 34:1055–1058, 1985

7. Ohlson LO, Larsson B, Björntorp P, Eriksson H, Svardsudd K, Welin L, Tibblin G, Wilhelmsen L: Risk factors for type 2 (non-insulin-dependent) diabetes mellitus: thirteen and one-half years of follow-up of the participants in a study of Swedish men born in 1913. *Diabetologia* 31:798–805, 1988

8. Colditz GA, Willett WC, Stampfer MJ, Manson JE, Hennekens CH, Arky RA, Speizer FE: Weight as a risk factor for clinical diabetes in women. *Am J Epidemiol* 132:501–513, 1990

9. Cassano PA, Rosner B, Vokonas PS, Weiss ST: Obesity and body fat distribution in relation to the incidence of non-insulin-dependent diabetes mellitus: a prospective cohort study of men in the normative aging study. *Am J Epidemiol* 136:1474–1486, 1992

10. Eriksson KF, Lindgärde F: Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise: the 6-year Malmo feasibility study. *Diabetologia* 34:891–898, 1991

11. Eriksson J, Tuominen J, Valle T, Sundberg S, Sovijärvi A, Lindholm H, Tuomilehto J, Koivisto V: Aerobic endurance exercise or circuit-type resistance training for individuals with impaired glucose tolerance? *Horm Metab Res* 30:37–41, 1998

12. Sarkkinen E, Schwab U, Niskanen L, Hannuksela M, Savolainen M, Kervinen K, Kesäniemi A, Uusitupa MI: The effects of monounsaturated-fat enriched diet and polyunsaturated-fat enriched diet on lipid and glucose metabolism in subjects with impaired glucose tolerance. *Eur J Clin Nutr* 50:592–598, 1996

13. Prochaska JO, DiClemente CC, Norcross JC: In search of how people change: applications to addictive behaviors. *Am Psychol* 47:1102–1114, 1992

14. Haapa E, Toponen T, Pietinen P, Räsänen L: *Annoskuvakirja*. Helsinki, Finland, Painokaari Oy, 1985

15. Ovaskainen M-L, Valsta L, Lauronen J: The compilation of food analysis values as a database for dietary studies: the Finnish experience. *Food Chemistry* 57:133–136, 1996

16. Lakka TA, Venäläinen JM, Rauramaa R, Salonen R, Tuomilehto J, Salonen JT: Relation of leisure-time physical activity and cardiorespiratory fitness to the risk of acute myocardial infarction. *N Engl J Med* 330:1549–1554, 1994

17. Lakka TA, Salonen JT: Intra-person variability of various physical activity assessments in the Kuopio Ischaemic Heart Disease Risk Factor Study. *Int J Epidemiol* 21:467–472, 1992

18. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV: Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. *Diabetes Care* 20:537–544, 1997

19. Swinburn BA, Metcalf PA, Ley SJ: Long-term (5-year) effects of a reduced-fat diet intervention in individuals with glucose intolerance. *Diabetes Care* 24:619–624, 2001

20. Poppitt SD, Keogh GF, Prentice AM, Williams DE, Sonnemans HM, Valk EE, Robinson E, Wareham NJ: Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. *Am J Clin Nutr* 75:11–20, 2002

21. Mensink M, Feskens EJ, Saris WH, De Bruin TW, Blaak EE: Study on Lifestyle Intervention and Impaired Glucose Tolerance Maastricht (SLIM): preliminary results after one year. *Int J Obes Relat Metab Disord* 27:377–384, 2003

22. The Diabetes Prevention Program Research Group: Costs associated with the primary prevention of type 2 diabetes mellitus in the diabetes prevention program. *Diabetes Care* 26:36–47, 2003

23. Astrup A, Rössner S: Lessons from obesity management programmes: greater initial weight loss improves long-term mainte-

nance. *Obes Rev* 1:17–19, 2000
24. Kelley DE, Wing R, Buonocore C, Sturis J, Polonsky K, Fitzsimmons M: Relative effects of calorie restriction and weight loss in noninsulin-dependent diabetes mellitus. *J Endocrinol Metab* 77:1287–1293, 1993
25. Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN: Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. *Diabetes Care* 17: 30–36, 1994
26. Livingstone MBE, Black AE: Markers of the validity of reported energy intake (Review). *J Nutr* 133 (Suppl. 3):895S–920S, 2003
27. Black AE, Cole TJ: Biased over- or underreporting is characteristic of individuals whether over time or by different assessment methods. *J Am Diet Assoc* 101:70–80, 2001