

# Leisure-Time Physical Activity and the Metabolic Syndrome in the Finnish Diabetes Prevention Study

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 PREVENTION STUDY GROUP

**OBJECTIVE** — To assess the effects of leisure-time physical activity (LTPA) and resistance training on metabolic syndrome (MetS) and its components in a post hoc analysis of the Finnish Diabetes Prevention Study, a randomized controlled lifestyle counseling trial.

**RESEARCH DESIGN AND METHODS** — A cohort of 486 middle-aged overweight men and women with impaired glucose tolerance were followed for an average of 4.1 years. The intervention and control groups were combined in the analyses. LTPA was assessed by questionnaires, dietary intake by food records, and features of the MetS by anthropometric and biochemical measures annually. Resistance training sessions were documented for 137 participants.

**RESULTS** — Increased moderate-to-vigorous LTPA, even after adjustments for changes in dietary intakes of total and saturated fat, fiber, and energy, and change in BMI was associated with a greater likelihood for resolution (29.7 vs. 19.1%;  $P = 0.004$  in the upper versus lower third of change) and a lesser likelihood for development (23.5 vs. 44.7%;  $P = 0.041$ ) of the MetS. Of the components of the MetS, the increase in moderate-to-vigorous LTPA was associated most strongly with improvement of glycemia. Among the 137 participants who participated in resistance training, MetS components were favorable in individuals who were in the upper third of participation rate (median 51 times/year) compared with individuals in the lowest third (median 8.5 times/year).

**CONCLUSIONS** — Increased moderate-to-vigorous LTPA was associated with a decreased likelihood of developing the MetS and an increased likelihood of its resolution in individuals at high risk for type 2 diabetes.

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The metabolic syndrome (MetS) is a constellation of interrelated metabolic risk factors, including abdominal obesity, insulin resistance, hyperglycemia, dyslipidemia, and elevated blood pressure, often accompanied by a prothrombotic and proinflammatory state (1,2). The underlying pathophysiology of the MetS is unclear, but both insulin resistance and abdominal obesity are considered main components (1,2). The MetS increases the risk of both type 2 diabetes (3) and cardiovascular disease (4,5).

Recent recommendations for the prevention and treatment of the MetS and its components promote increased physical activity (including aerobic and resistance exercise), a healthy diet, and weight loss (2,6–8). In lifestyle interventions trials, the incidence of type 2 diabetes has been reduced by more than half in individuals with impaired glucose tolerance, and the prevalence of the MetS has also been decreased (9,10). In the Finnish Diabetes Prevention Study (DPS), increased moderate-to-vigorous leisure-time physical activity (LTPA) was strongly associated with a lower risk of type 2 diabetes, independently of dietary changes and weight loss (11).

Some prospective epidemiological studies and uncontrolled trials have suggested that increased moderate-to-vigorous exercise decreases the incidence or prevalence of the MetS (8,12,13). However, data on the role of changes in LTPA in the prevention and treatment of the MetS in long-term studies are limited. Therefore, we conducted a post hoc analysis of the Finnish DPS. Our hypothesis was that the change in LTPA and participation in resistance training would be associated with the change in the MetS and its components.

## RESEARCH DESIGN AND METHODS

**RESEARCH DESIGN AND METHODS** — A detailed description of the design, subjects, and methods applied in the DPS has been reported previously (14). In brief, the DPS was a randomized lifestyle intervention study in 522 middle-aged overweight participants with impaired glucose tolerance, aimed at

the prevention of type 2 diabetes. In the present study, we included those 486 participants (249 in the intervention and 237 in the control group) who had completed a questionnaire quantifying LTPA at baseline and during yearly follow-up visits (11). A subgroup of 137 participants was taking part in supervised resistance training sessions. The study protocol was approved by the ethics committee of the National Public Health Institute in Helsinki, and all subjects gave written informed consent.

### Intervention

The aim of the intervention was to encourage people to make healthy lifestyle choices. The participants in the intervention group were given detailed and individualized dietary and exercise counseling as described elsewhere (15). Endurance exercise was recommended to increase aerobic capacity and cardiorespiratory fitness. Session for supervised and individually tailored progressive circuit-type resistance training with moderate intensity were recommended twice a week and offered free of charge in three of the five study centers.

The participants in the control group were given general information about healthy food choices, physical activity, and weight loss at baseline, but no individualized counseling was offered.

### Assessment of physical activity

The validated Kuopio Ischemic Heart Disease risk factor study questionnaire (11,12) was used for the assessment of physical activity. The participants estimated the frequency, average duration, and intensity of different forms of exercise for individual months during the past 12 months. Based on the reported intensity of different activities and their corresponding metabolic equivalent (MET) values, the total LTPA was divided to low-intensity and to moderate-to-vigorous intensity LTPA (13). Low-intensity LTPA (<3.5 METs) included activities such as gardening, picking berries, casual walking, and bicycling at recreational intensity. Moderate-to-vigorous LTPA ( $\geq 3.5$  METs) included activities such as brisk walking, jogging, skiing, swimming, rowing, forest work, gymnastics, resistance training, ball games, snow shoveling, and heavy housework.

The duration of total LTPA and its components were calculated as hours/week from the baseline to the end of the follow-up. The changes were calculated

by subtracting averaged follow-up value from the corresponding baseline value (11). The participation in resistance training was recorded electronically when the participants visited the resistance training facilities and was analyzed as sessions/year.

### Other measurements

Medical history and 3-day food records were collected at baseline and at each annual visit. Average intakes of energy (kcal/day), carbohydrates (E%), total fat (E%), saturated fat (E%), and dietary fiber (g/1,000 kcal) were calculated. The average values from years 1–3 were used to measure dietary intakes during follow-up (11).

Anthropometry and blood pressure were assessed as described previously. Plasma glucose was determined locally according to standard guidelines. Serum total and HDL cholesterol and triglyceride levels were determined by enzymatic methods (Boehringer Mannheim, Germany).

For the definition of the MetS, we used the National Cholesterol Education Program 2005 criteria (6).

### Statistical analysis

The data were analyzed using SPSS statistical software (version 11.5; SPSS, Chicago, IL). The baseline values are given as mean  $\pm$  SD, as median with 0.25–0.75 interquartile range, or as percentages. The Student two-tailed *t* test, Mann-Whitney *U* test (fasting and 2-h serum insulin, triglycerides, and LTPA), and  $\chi^2$  test were applied to compare the differences at baseline and during the follow-up. For participants who dropped out or developed diabetes during the study, the measurements at the last observation year was used as the end value.

The primary outcome measure was the change in the MetS status in the combined intervention and control group from baseline to the end, i.e., resolution of the MetS from baseline, development of MetS, or no change with LTPA changes as explanatory variables. Secondary outcome measures were the changes of the MetS components. The change of different LTPA was categorized into thirds. The association with the change in MetS status and its components was analyzed with multinomial regression. The models were adjusted for age, sex, intervention group, and DPS study years (model 1) with further adjustments for changes in diet (intake of total fat, saturated fat, fiber,

and energy) (model 2) and BMI (model 3). The change in low-intensity LTPA was also adjusted for the change in moderate-to-vigorous LTPA and vice versa. *P* values <0.05 were considered statistically significant.

## RESULTS

### Baseline clinical and metabolic characteristics

In the combined study cohort, 74.3% had the MetS at baseline. The participants with the MetS at baseline had significantly higher BMI, waist circumference, blood pressure, fasting and 2-h glucose, fasting and 2-h insulin, and serum triglyceride levels and lower serum HDL cholesterol levels (Table 1).

In general, men exercised more than women. Women without the MetS reported significantly more hours per week spent on total and low-intensity LTPA during the previous 12 months than women with the MetS.

### Changes in LTPA during the follow-up

The median for total LTPA increased from 7.2 (3.6–10.8) at baseline to an average of 7.7 (4.8–11.7) hours per week (*P* = 0.061) in men and from 5.3 (2.8–8.6) to 5.8 (3.2–9.0) hours per week (*P* = 0.016) in women during the follow-up. The median for moderate to vigorous LTPA increased from 2.3 (0.9–4.8) to 3.1 (1.8–4.9) (*P*  $\leq$  0.001) hours per week in men and from 1.4 (0.3–3.5) to 2.5 (1.1–4.1) (*P*  $\leq$  0.001) hours per week in women. There was no significant change in low-intensity LTPA.

### LTPA changes and the incidence for resolution and development of the MetS

Of the 361 participants meeting the MetS criteria at baseline, 20.8% (*n* = 75; 26.6% in the intervention and 14.7% in the control group; *P* = 0.005) showed resolution during the follow-up. Of the 126 participants not meeting the MetS criteria at baseline, 31.2% (*n* = 39; 30.8% in the intervention and 31.7% in the control group; *P* = 0.95) developed MetS during the follow-up. The development of the MetS was associated with weight gain and less LTPA in both groups.

The change in total LTPA was associated with the change in MetS status (resolution, no change, development) after adjustment for age, sex, intervention

Table 1—Baseline characteristics of the participants according the absence (MetS<sup>-</sup>) or presence (MetS<sup>+</sup>) of the MetS

	All	MetS <sup>-</sup>	MetS <sup>+</sup>	P
n	486	125	361	—
Group allocation				0.843
Intervention	249	65	184	
Control	237	60	177	
Sex				0.003
Male [n (%)]	162 (33.3)	55 (34.0)	107 (66.0)	
Female [n (%)]	324 (66.7)	70 (21.6)	254 (78.4)	
Age (years)	55.4 ± 7.0	55.8 ± 7.1	55.3 ± 7.0	0.476
40–49 (%)	27.0	27.2	26.9	
50–59 (%)	33.1	28.8	34.6	
≥60 (%)	39.9	44.0	38.0	
Weight (kg)	86.3 ± 14.3	80.3 ± 10.0	88.4 ± 14.9	<0.001
BMI (kg/m <sup>2</sup> )	31.2 ± 4.5	28.8 ± 3.4	32.1 ± 4.6	<0.001
Waist (cm) (all)	101.2 ± 11.0	94.7 ± 8.1	103.5 ± 11.0	<0.001
Men	104.2 ± 9.7	97.3 ± 6.0	107.7 ± 9.4	
Women	99.8 ± 11.4	92.7 ± 9.0	101.7 ± 11.2	
Fasting glucose (mmol/l)	6.1 ± 0.7	5.8 ± 0.7	6.3 ± 0.7	<0.001
2-h glucose (mmol/l)	8.9 ± 1.5	8.6 ± 1.4	9.0 ± 1.5	0.014
Fasting insulin (mU/l)	13 (10–18)	10 (8–13)	14 (11–19)	<0.001
2-h insulin (mU/l)	79 (54–120)	60 (38–77)	89 (63–134)	<0.001
Serum total cholesterol (mmol/l)	5.6 ± 0.9	5.7 ± 0.8	5.6 ± 0.9	0.064
Serum HDL cholesterol (mmol/l)	1.21 ± 0.29	1.39 ± 0.24	1.15 ± 0.28	<0.001
Serum triglycerides (mmol/l)	1.56 (1.18–2.09)	1.20 (0.96–1.43)	1.72 (1.34–2.29)	<0.001
Lipid-lowering medication (%)	5.4	3.2	6.2	0.576
Systolic blood pressure (mmHg)	138 ± 18	133 ± 20	140 ± 16	<0.001
Diastolic blood pressure (mmHg)	86 ± 10	82 ± 11	87 ± 9	<0.001
Antihypertensive medication (%)	35.3	16.8	41.7	<0.001
Total LTPA (all) (h/week)	5.7 (3.1–9.3)	6.9 (4.3–10.1)	5.1 (2.8–9.1)	0.001
Men	7.2 (3.6–10.8)	7.5 (3.8–10.4)	6.9 (3.4–10.9)	0.503
Women	5.3 (2.8–8.6)	6.7 (4.3–9.8)	4.9 (2.6–8.2)	0.002
Moderate-to-vigorous LTPA (all)	1.7 (0.5–4.0)	1.9 (0.6–4.4)	1.6 (0.4–3.8)	0.165
Men	2.3 (0.9–4.8)	2.1 (1.1–4.7)	2.4 (0.7–4.9)	0.725
Women	1.4 (0.3–3.5)	1.7 (0.4–4.2)	1.3 (0.3–3.5)	0.481
Low-intensity LTPA	3.0 (1.2–5.9)	4.1 (1.9–7.2)	2.9 (1.1–5.3)	0.004
Men	3.2 (1.4–6.9)	3.7 (1.9–7.2)	3.0 (1.2–6.9)	0.268
Women	2.9 (1.2–5.6)	4.4 (1.5–7.2)	2.8 (1.1–4.6)	0.011

Data are means ± SD for normally distributed or medians (interquartile ranges) for skewed parameters or percentages.

group, and DPS study years (model 1, Fig. 1A) and dietary intakes (model 2, Fig. 1A). The change in moderate-to-vigorous LTPA was even more strongly associated with the change in MetS status in analyses adjusting for the variables in model 1 and changes in low-intensity LTPA. The resolution of the MetS was seen in 29.7 versus 19.1% ( $P = 0.004$ ), and the development of MetS was seen in 23.5 versus 44.7% ( $P = 0.041$ ) in the upper versus lower third of change in moderate-to-vigorous LTPA. The associations remained significant after further adjustments for changes in diet (model 2) and BMI (model 3) (Fig. 1B). Changes in low-intensity LTPA were not associated with the change in MetS status (Fig. 1C).

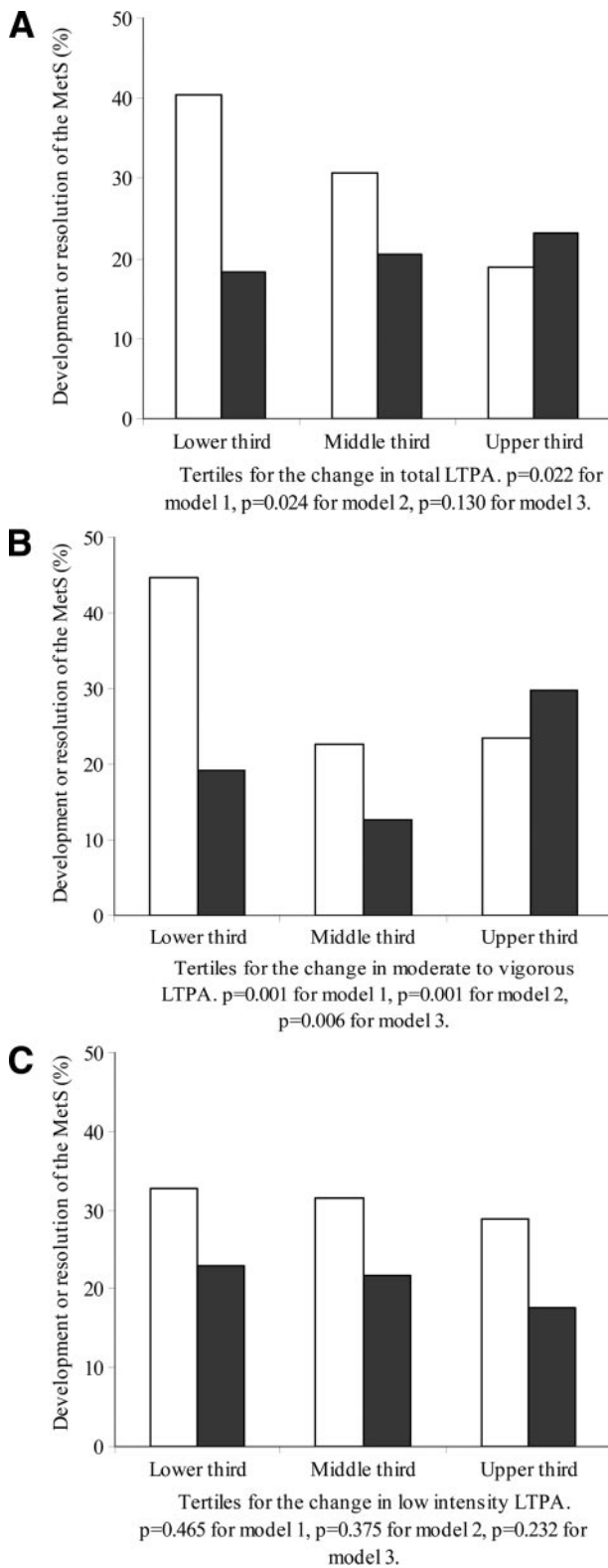
### LTPA changes and the components of the MetS

The increase in total LTPA was associated with a decrease in the prevalence of hyperglycemia ( $P = 0.020$ – $0.053$ ), low HDL cholesterol ( $P = 0.018$ – $0.057$ ), and hypertriglyceridemia ( $P = 0.002$ – $0.003$ ) (Table 2). Increased moderate-to-vigorous LTPA decreased the prevalence of elevated fasting glucose ( $P = 0.003$ – $0.018$ ), but no association with abdominal obesity ( $P = 0.065$ – $0.181$ ), low HDL cholesterol ( $P = 0.098$ – $0.232$ ), and high blood pressure ( $P = 0.068$ – $0.151$ ) was found. In contrast, an increase in low-intensity LTPA was associated with an improvement in hypertriglyceridemia ( $P = 0.006$ – $0.004$ ), but not any of the other components of the MetS.

### Resistance training and the components of the MetS

In the subgroup of 137 individuals taking part in supervised resistance training, the median attendance rate was 27.0 (13.4–42.9) sessions/year during the entire study. Of the MetS status components, the resistance training attendance rate was associated, even after adjustment for dietary and BMI changes, with improvements in hyperglycemia ( $P = 0.127$ – $0.029$ ), hypertriglyceridemia ( $P = 0.046$ – $0.081$ ), and low HDL cholesterol ( $P < 0.001$ – $0.002$ ), but not with elevated blood pressure or abdominal obesity (Table 3).

**CONCLUSIONS**— Increased moderate-to-vigorous LTPA during the 4.1-



**Figure 1**—Incidences (%) for the development (for individuals without MetS at baseline,  $n = 125$ ) (□) and the resolution (for individuals with MetS at baseline,  $n = 361$ ) (■) of the MetS according to LTPA change tertiles for total LTPA (A), moderate-to-vigorous LTPA (B), and low-intensity LTPA (C). Model 1: adjustments for age, sex, intervention group, and DPS study years. The change in low-intensity LTPA was also adjusted for change in moderate-to-vigorous LTPA and vice versa. Model 2: model 1 and adjustments for change in dietary intakes of total fat, saturated fat, fiber, and energy. Model 3: model 2 and change in BMI.

year follow-up increased the likelihood for the MetS to resolve and decreased the likelihood for the MetS to develop, independently of changes in diet and body weight. Moreover, increased moderate-to-vigorous LTPA decreased the prevalence of hyperglycemia. Improvements in fasting plasma glucose, serum triglycerides, and HDL cholesterol, independently of changes in diet, lifestyle LTPA, and other types of LTPA, were associated with participation in resistance training.

Overall, strong and mostly linear dose-response associations of the change in total LTPA with the development and resolution of the MetS were seen. When breaking down physical activity into moderate-to-vigorous LTPA and low-intensity LTPA, it seems evident that most of the benefit was from moderate-to-vigorous-intensity LTPA. Changes in moderate-to-vigorous LTPA were associated with the change in metabolic status, even independently of the changes in BMI, but the association was not linear across categories. Changes in low-intensity LTPA were not associated with the development or resolution of the MetS. Why the dose-response association was not apparent for moderate-to-vigorous LTPA is unclear, but it may be related to the difficulty in the precise assessment of LTPA. Overall, however, our findings support efforts to increase or at least maintain LTPA, especially moderate-to-vigorous LTPA, in the prevention and treatment of the MetS.

In this analysis of the DPS, the intervention and control groups were combined. In separate analysis, there was a significant difference between the groups in the resolution of the MetS. However, there was no difference in the development of MetS between groups; ~30% of those without MetS at baseline developed MetS in both groups during the follow-up. This may be due the selection of the participants. They were individuals at high risk for type 2 diabetes and for the MetS. During the follow-up, 22% developed type 2 diabetes (11). While the development of MetS was associated with weight gain and less LTPA, those subjects who developed the MetS appeared to not adhere with our intervention. The apparent favorable effects of moderate-to-vigorous LTPA on resolution and development of the MetS are consistent with the results of the uncontrolled Heritage Family Study (16) and some prospective cohort studies showing that increased moder-



**Table 2—Incidences (%) for development and resolution of the MetS components according to LTPA change tertiles for total, low-intensity, and moderate-to-vigorous LTPA during the follow-up**

	Incidence (%)	Tertiles for total LTPA change median (0.25–0.75 interquartile range) (h/week)			P for trend
		Lower –3.2 (–5.5 to –1.6)	Middle 0.6 (0.1 to 1.2)	Upper 3.8 (2.4 to 5.8)	
Abdominal obesity					0.119*
Development	4.3%	7.4	2.5	3.1	0.184†
Resolution	11.1%	8.6	12.3	12.3	0.408‡
Elevated fasting glucose					0.020*
Development	12.4%	17.4	13.0	6.8	0.033†
Resolution	8.9%	8.1	10.5	8.0	0.053‡
Elevated triglycerides					0.002*
Development	9.3%	11.1	6.8	9.9	0.002†
Resolution	14.4%	6.2	20.4	16.7	0.003‡
Low HDL cholesterol					0.018*
Development	7.8%	12.3	6.8	4.3	0.013†
Resolution	16.5%	13.6	13.6	22.6	0.057‡
Elevated blood pressure					0.661*
Development	4.7%	5.6	5.6	3.1	0.643†
Resolution	9.7%	8.0	9.3	11.8	0.800‡

\*Model 1: adjustments for age, sex, intervention group, and DPS study years. The change in low-intensity LTPA was also adjusted for change in moderate-to-vigorous LTPA and vice versa. †Model 2: model 1 and adjustments for change in dietary intakes of total fat, saturated fat, fiber, and energy. ‡Model 3: model 2 and change in BMI.

ate-to-vigorous LTPA was associated with a lower incidence of the MetS during follow-up (8,12,17). In the DPS cohort, increased moderate-to-vigorous LTPA seemed to protect against developing the MetS in both men and women. Higher cardiorespiratory fitness, which partly reflects higher levels of moderate-to-vigorous LTPA, has predicted a lower prevalence of the MetS independently of major confounding variables also in women in the Aerobics Center Longitudinal Study (18) and in the Dose-Responses to Exercise Training Study (19). Changes in low-intensity LTPA were not associated with changes in the MetS status. These findings are consistent with the results of the Kuopio Ischemic Heart Disease study, in which moderate-to-vigorous, but not low-intensity, LTPA was associated with development of the MetS (12). When examining specific components of the MetS, increased moderate-to-vigorous LTPA had the greatest effect on impaired fasting glucose, whereas the benefit on abdominal obesity, low HDL cholesterol, and high blood pressure was not significant. In contrast, changes in low-intensity LTPA had benefits on hypertriglyceridemia, but not on the other components of the MetS, and changes in total LTPA improved impaired fasting glucose and dyslipidemia.

In intervention trials, low-intensity LTPA has less consistently improved metabolic outcomes than more intense LTPA (8). However, we have previously reported that increased low-intensity and moderate-to-vigorous LTPA were similarly associated with a lower risk of type 2 diabetes in the Finnish DPS, suggesting that total energy expenditure on LTPA was more important than intensity (11). In line with that finding, the accumulated daily physical activity as measured with an accelerometer was a major determinant of insulin sensitivity, and time spent on moderate-to-vigorous physical activity did not affect insulin sensitivity independently of total activity in the European Relationship between Insulin Sensitivity and Cardiovascular Risk Study (20). The differences may be explained by differences in study populations and specific metabolic outcomes. More information on the long-term metabolic benefits of low-intensity LTPA in different age-groups and risk groups is nonetheless needed.

Regular participation in resistance training predicted favorable changes in MetS components. We found that a higher participation rate in resistance training was associated with benefits on impaired fasting glucose, hypertriglyceridemia, and low HDL cholesterol, but not abdominal obesity or blood pres-

sure. In 3- to 6-month trials, resistance training has variably increased muscle mass, decreased fat mass and abdominal obesity, and improved insulin sensitivity in obese adults, hypertensive patients, older men, and older type 2 diabetic patients (8,21,22). In individuals with type 2 diabetes, resistance training resulted in similar improvements of glycemic control as aerobic exercise (23), although the effect on glucose tolerance in impaired glucose tolerance has been less clear (8). Improvements in insulin sensitivity and metabolic risk factors may be mediated in part by changes in body composition, but strength training may also independently affect steps in insulin signaling and glucose transport (24). Based on meta-analyses of trials, resistance training may decrease blood pressure (25), but effects on dyslipidemia have been variable (8).

Our findings suggest that there is a graded benefit in the frequency of resistance training in the prevention or treatment of the MetS components, with rather substantial benefits for individuals engaging in resistance training on median once a week compared with individuals engaging in resistance training on median less than once a month. In the above-mentioned studies showing an improvement in insulin sensitivity in individuals

Table 2—Continued

Teriles for low LTPA change median (0.25–0.75 interquartile range) (h/week)				Teriles for moderate-to-vigorous LTPA change median (0.25–0.75 interquartile range) (h/week)			
Lower –3.2 (–5.6 to –1.7)	Middle 0.1 (–0.4 to 0.5)	Upper 3.1 (1.8 to 5.1)	P for trend	Lower –1.5 (–3.1 to –0.5)	Middle 0.5 (0.2 to 0.8)	Upper 2.6 (1.8 to 3.8)	P for trend
4.9	4.9	3.0.1	0.718*	6.2	2.5	4.3	0.065*
12.3	10.5	10.5	0.753†	9.3	6.8	4.3	0.083†
11.8	12.3	13.0	0.941*	19.1	11.7	6.2	0.011†
7.5	9.3	9.9	0.928†	8.6	8.0	9.9	0.018†
9.9	6.2	11.7	0.006*	12.3	8.0	7.4	0.491*
8.0	19.3	16.0	0.005†	14.9	12.4	16.0	0.526†
7.4	9.3	6.8	0.004†	11.7	6.2	5.6	0.672†
16.7	14.2	18.5	0.762*	14.8	13.6	21.3	0.098*
4.9	4.3	5.0	0.668†	7.4	4.3	2.5	0.086†
9.9	11.1	8.1	0.807†	6.8	8.0	14.3	0.232†
			0.921*				0.068*
			0.883†				0.066†
			0.824†				0.151†

at risk for type 2 diabetes and in glycemic control in patients with type 2 diabetes, training frequency was generally two to three times per week. The metabolic benefits of resistance training at a lower frequency may become apparent only after much longer periods of training than in previously published trials, which have usually lasted 3–6 months. However, longer-term trials are needed to test this hypothesis.

Strengths of the DPS include its repeated assessments of LTPA and dietary intake. However, the present analyses are post hoc. Furthermore, the intervention had several components. Detailed assessment of the individual lifestyle components allows statistical disentanglement of their individual effects, but residual confounding is possible. Moreover, we did not objectively measure physical activity. Decreases in LTPA may have been related

to factors that themselves may be related to the development of the MetS. Adherence to resistance training was on average poor. When this study was conducted in the early 1990s, it was uncommon for middle-aged and overweight individuals to attend resistance training facilities, where most of the clientele were young and fit. Some also encountered difficulties with transportation and time schedules.

In conclusion, increased participa-

Table 3—The average resistance training attendance rate per year and the change (development and resolution) in the MetS components among a subgroup of 137 participants

	Incidence (%) (n = 137)	Teriles for average yearly attendance rate for resistance training median (0.25–0.75 interquartile range)			P
		Lower 8.5 (5.5–13.4)	Middle 27.0 (21.9–32.7)	Upper 50.7 (42.3–67.2)	
Abdominal obesity					0.438*
Development	0.0	0.0	0.0	0.0	0.537†
Resolution	10.2	4.4	13.0	13.0	0.549†
Elevated fasting glucose					0.127*
Development	16.8	13.3	26.1	10.9	0.157†
Resolution	5.1	6.7	2.2	6.5	0.029†
Elevated triglycerides					0.046*
Development	10.2	11.1	17.4	2.2	0.067†
Resolution	20.4	11.1	21.7	28.3	0.081†
Low HDL cholesterol					0.000*
Development	10.9	15.6	17.4	2.2	0.001†
Resolution	18.2	6.7	26.1	21.7	0.002†
Elevated blood pressure					0.982*
Development	3.6	4.4	2.2	4.3	0.967†
Resolution	10.2	8.9	10.9	10.9	0.957†

\*Model 1: adjustments for age, sex, group, DPS study years, averaged low-intensity LTPA, and LTPA other than gymnastics and calisthenics. †Model 2: model 1 and adjustments for change in dietary intakes of total fat, saturated fat, fiber, and energy. ‡Model 3: adjustment for model 2 and change in BMI.

tion in moderate-to-vigorous physical activity and regular long-term participation in resistance training improved the MetS status among men and women with impaired glucose tolerance in the Finnish DPS. Physical activity and resistance training also more specifically had benefits with respect to hyperglycemia and dyslipidemia, but improvements in abdominal obesity were not clearly seen. Resolution or prevention of the MetS and related features might contribute to the protective effect of physical activity on type 2 diabetes.

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P.I.-P. designed the study, researched the data, and wrote the manuscript. D.E.L. designed the study, researched the data, and contributed to writing the manuscript. J.G.E. and T.A.L. contributed to study design and edited the manuscript. J.L., M.P., S.A., and S.K.-K. contributed to data collection and coordination and reviewed the manuscript. M.U. and J.T. are the principal investigators of the DPS study and participated in reviewing/editing the manuscript.

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