

Improvement of BMI, Body Composition, and Body Fat Distribution With Lifestyle Modification in Japanese Americans With Impaired Glucose Tolerance

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OBJECTIVE — To determine whether diet and endurance exercise improved adiposity-related measurements in Japanese Americans with impaired glucose tolerance (IGT).

RESEARCH DESIGN AND METHODS — This study compared the effects of an American Heart Association (AHA) step 2 diet (<30% of total calories as fat, <7% saturated fat, 55% carbohydrate, and <200 mg cholesterol daily) plus endurance exercise for 1 h three times a week (treatment group) with an AHA step 1 diet (30% of total calories as fat, 10% saturated fat, 50% carbohydrate, and <300 mg cholesterol) plus stretching exercise three times a week (control group) on BMI, body composition (% fat), and body fat distribution at 6 and 24 months of follow-up in 64 Japanese American men and women with IGT, 58 of whom completed the study.

RESULTS — At 6 months, the treatment group showed significantly greater reduction in percent, body fat (-1.4 ± 0.4 vs. $-0.3 \pm 0.3\%$); BMI (-1.1 ± 0.2 vs. -0.4 ± 0.1 kg/m²); subcutaneous fat by computed tomography at the abdomen (-29.3 ± 4.2 vs. -5.7 ± 5.9 cm²), thigh (-13.2 ± 3.6 vs. -3.6 ± 3.0 cm²), and thorax (-19.6 ± 3.6 vs. -8.9 ± 2.6 cm²); and skinfold thickness at the bicep (-2.0 ± 0.6 vs. 1.1 ± 0.6 mm) and tricep (-3.7 ± 0.8 vs. -0.9 ± 0.6 mm), which continued despite moving to home-based exercise for the last 18 months.

CONCLUSIONS — Diet and endurance exercise improved BMI, body composition, and body fat distribution and, thus, may delay or prevent type 2 diabetes in Japanese Americans with IGT.

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Type 2 diabetes has rapidly become a global health problem. Previously seen mostly in developed nations, this disease has become more common in developing countries where lifestyle practices have begun to emulate those of developed societies (1). Diabetes is an economically burdensome disease due to

the increased morbidity and mortality stemming from associated complications such as coronary artery, cerebrovascular, retinal, neurological, and renal diseases (2–4). The solution to this growing problem may lie in preventing or delaying the onset of disease in at-risk populations (5–10).

Type 2 diabetes is more prevalent in Japanese Americans than in the general U.S. population or in Japanese (11). Although generally not obese, Japanese Americans are nonetheless heavier than Japanese (12). This has been postulated to be due to differences between the Japanese and the American lifestyles with respect to diet and physical activity, with Japanese generally being more physically active and consuming a diet with fewer calories derived from fat, particularly saturated fat, compared with Japanese Americans (13–15).

Separate from increased body weight is the role of the distribution of body fat as a risk factor for diabetes. Thus, the central pattern of body fat distribution, and in particular an increased amount of intra-abdominal or visceral fat, has been associated in Japanese Americans with several traits found with the metabolic syndrome, including hyperinsulinemia, abnormal glucose tolerance, hypertension, dyslipidemia, and cardiovascular disease (16–21). Given the importance of body weight and body fat distribution as risk factors for diabetes, and the observation that visceral fat may be significantly reduced even if total weight loss is not great, studies designed to reduce adiposity, especially visceral adiposity, in individuals at risk for type 2 diabetes are of great interest (22–24).

Impaired glucose tolerance (IGT), a diagnosis made from results of a 2-h 75-g oral glucose tolerance test (OGTT), identifies individuals at high risk for diabetes (25). The rate of conversion from IGT to diabetes has been reported to be 3.6–8.7% annually, depending on the charac-

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Abbreviations: AHA, American Heart Association; CT, computed tomography; IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test.

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

See accompany editorial on p. 1650.

teristics of the group being studied (26). Thus individuals with IGT represent a high-risk target group for studying interventions to prevent type 2 diabetes.

Given this background, a lifestyle intervention directed toward reducing dietary saturated fat and increasing physical activity was selected to examine whether prescription of this combination improved adiposity and body fat distribution in Japanese Americans with IGT.

RESEARCH DESIGN AND METHODS

This was a randomized trial of lifestyle intervention in men and women of Japanese ancestry who had IGT. The protocol was reviewed and approved by the Human Subjects Review Committee at the University of Washington, and written informed consent was obtained from each participant.

Screening of participants

All participants were of full Japanese ancestry. After an initial telephone screening, participants underwent a medical history and physical examination, electrocardiogram, and blood tests. Participants were excluded if they had a history or evidence of significant coronary artery disease; valvular heart disease; hypertension (blood pressure $>160/90$ mmHg); arthritis; pulmonary, neurologic, or psychiatric disease or dementia that hindered their ability to participate; unusual dietary restrictions (e.g., strict vegetarian); current use of lipid-lowering drugs; or tobacco use. Participants were also excluded if laboratory tests showed evidence for liver or kidney disease or anemia (hematocrit $<38\%$ for men, $<36\%$ for women) or if triglyceride levels were >300 mg/dl. Subjects who did not meet any exclusion criteria underwent an OGTT performed in the morning following an overnight fast of 8–10 h. Only those found to have IGT on two separate occasions using the World Health Organization criteria for a 75-g OGTT were eligible for further screening, which consisted of a maximal Bruce protocol treadmill test (27).

Randomization and interventions

Upon enrollment, each participant was assigned to the treatment or control group using adaptive randomization (28).

Treatment group

The treatment group received endurance exercise training and a dietary prescription. For the first 6 months, exercise sessions were directed by an exercise physiologist. Subjects performed endurance exercise (walk/jog) on a treadmill three times a week for 1 h at each session. Exercise began with a 10-min warm-up period and ended with a 10-min cool-down period. Initially, exercise was designed to attain 50% of heart rate reserve [$0.5 \times (\text{maximum heart rate} - \text{resting heart rate}) + \text{resting heart rate}$]. Heart rate reserve was estimated from the maximum and resting heart rates observed at the baseline $\text{VO}_{2\text{max}}$ for each individual. The exercise was gradually increased at 2-week intervals over a period of 3 months until subjects were exercising at a goal of 70% of heart rate reserve. Pulse rates were electronically monitored during exercise. The treatment group was also prescribed an isocaloric American Heart Association (AHA) step 2 diet comprising $<30\%$ of total calories as fat ($<7\%$ as saturated fat), 55% as carbohydrate, the balance as protein, and <200 mg cholesterol daily.

Control group

Members of the control group performed stretching exercises three times a week for 1 h (each session under staff supervision) and were prescribed an isocaloric AHA step 1 diet comprising 30% of total calories as fat (10% as saturated fat), 50% as carbohydrate, 20% as protein, and <300 mg cholesterol daily.

Treatment and control groups

Based on 3-day food records, each participant's baseline diet was analyzed; this information was used by a dietitian to instruct participants on their prescribed diet. At visits where participants met with the dietitian, food records were used as a tool to show how well they were meeting the prescribed diet. After the first 6 months, exercise in both groups was home-based without supervision by investigators. All participants were simply instructed to continue their prescribed diet and exercise for an additional 18 months and were reminded about this at 12 months. Participants were instructed to keep records of their exercise, and heart rate monitors were available. The exercise information was not analyzed but was used to encourage adherence.

Weight reduction was not a goal for either group.

Follow-up

In addition to their baseline examination, participants were examined at 6, 12, and 24 months. At baseline and 6 and 24 months, each participant provided a medical history, underwent a exercise treadmill test to measure $\text{VO}_{2\text{max}}$, had body composition and anthropometry assessed, and underwent an OGTT and a computed tomography (CT) scan. An OGTT was also performed at 12 months.

Branching treadmill tests were performed under physician supervision to determine $\text{VO}_{2\text{max}}$. Respiratory quotients (VCO_2/VO_2) ≥ 1.12 were the goal during testing. Anthropometry included height (cm), weight (kg), waist girth (cm), and skinfold thickness (mm) at the forearm, biceps, triceps, subscapula, chest (mid-axilla), abdomen (midpoint between the umbilicus and the most lateral point of the abdomen), suprailiac, and anterior thigh. Measurements were performed by the same investigator using a standard method (29). BMI was calculated as weight (kg) divided by height (m) squared.

Body composition for percent body fat and total body fat was determined using standard underwater weighing techniques according to Goldman (30). Density corrections for residual lung volume were made using helium dilution.

CT scans to assess subcutaneous and intra-abdominal fat areas were performed at the University of Washington Medical Center Department of Radiology as described previously (31). Single scans were done at the thorax, abdomen, and thigh to measure subcutaneous and visceral fat areas.

Three-day food records were obtained from each participant at baseline and at 3, 6, 9, 12, and 24 months. Average portion sizes were estimated by the participant using food models and common household measuring utensils. Total caloric intake and absolute and proportional intakes of fat, carbohydrate, protein, saturated fat, monounsaturated fat, polyunsaturated fat, alcohol, and total cholesterol were measured. Nutrient calculations were performed using the Nutrition Data System for Research (NDS-R) software version 4.02, developed by the Nutrition Coordinating Center, University of Minnesota. Asian food composition

Table 1—Baseline characteristics of the treatment and control groups

	Treatment	Control	P (t test or χ^2 test)	P (ANCOVA)*
n	32	32		
Age (years)	55.8 \pm 1.8	52.2 \pm 1.8	0.6	
Female [% (n)]	63 (20)	47 (15)	0.3	
Adiposity variables				
Weight (kg)	66.1 \pm 2.9	69.7 \pm 2.6	0.4	0.8
BMI (kg/m ²)	25.6 \pm 0.8	26.6 \pm 0.8	0.4	0.6
Percent body fat	30.1 \pm 1.5	31.2 \pm 1.4	0.6	0.12
CT fat area (cm ²)				
Intra-abdominal	86.3 \pm 8.1	112.3 \pm 9.9	0.038	0.11
Abdomen subcutaneous	225.5 \pm 24.3	209.3 \pm 17.4	0.6	0.7
Thigh subcutaneous	79.7 \pm 7.5	70.6 \pm 7.2	0.4	0.9
Thorax subcutaneous	147.8 \pm 14.8	164.1 \pm 17.9	0.5	0.2
Skinfold thickness (mm)				
Subscapular	26.9 \pm 1.9	26.6 \pm 1.6	0.9	0.9
Bicep	14.9 \pm 1.1	14.7 \pm 1.1	0.9	0.6
Tricep	21.0 \pm 1.1	19.0 \pm 1.3	0.3	0.7
Forearm	6.5 \pm 0.5	7.3 \pm 0.6	0.3	0.10
Suprailiac	29.6 \pm 2.0	28.0 \pm 1.8	0.5	0.4
Waist circumference (cm)	80.9 \pm 2.0	87.2 \pm 2.2	0.04	0.12
Physical fitness				
VO _{2max} (ml \cdot kg ⁻¹ \cdot min ⁻¹)	28.8 \pm 1.3	29.0 \pm 1.3	0.6	0.8
Diet				
Kilocalories per day	1,822 \pm 71	1,795 \pm 83	0.2	—
Fat (%)	29.7 \pm 1.7	29.8 \pm 1.7	0.7	—
Saturated fat (%)	8.9 \pm 0.7	9.1 \pm 0.8	1.0	—
Protein (%)	16.5 \pm 0.5	17.5 \pm 0.7	0.2	—
Carbohydrate (%)	53.8 \pm 1.6	52.7 \pm 2.0	0.5	—
Cholesterol (mg)	210 \pm 20	236 \pm 35	0.7	—

*Data are means \pm SE. Adjusted for sex, where applicable.

tables and Japanese recipe books were used, and Japanese American dietitians were consulted before Japanese foods were entered using food components already in the database with similar nutrient values. If similar foods could not be identified, nutritional content was determined and the new food was programmed into the database.

Assessment of adherence

Maximal aerobic capacity (VO_{2max}) at 6 and 24 months compared with baseline was used to estimate adherence to the exercise protocol. Three-day food records at 3, 6, 9, 12, and 24 months were used to determine adherence to the prescribed diet. Participants were given printouts of their food record results compared with their prescribed AHA diet.

Statistical analysis

Results are shown as means \pm SE. Significance of differences between group means was tested by Student's *t* test.

Where differences might have been attributable to differences in sex or baseline values, ANCOVA was used, adjusting for sex and for baseline values. Significance of differences in frequency was tested by χ^2 test. Significance was set at *P* < 0.05.

RESULTS

Recruitment and retention of participants

After telephone screening of 340 individuals, 270 underwent further screening as described above. Of those, 117 had a normal OGTT and 51 had results indicating diabetes. Twenty-eight individuals had IGT but were not randomized because of medical information received at their screening examination or because they decided not to participate. Seventy-four subjects with IGT were randomized, 36 to the treatment group and 38 to the control group. Of these 74, 3 could not participate because of poor venous access, and 1 had an abnormal Bruce protocol treadmill

test and should not have been randomized. Of the remaining subjects, nine dropped out: four from the treatment group (two for time commitment, one for arthritis symptoms, and one for back pain) and five from the control group (four for time commitment and one for thrombocytosis). Two subjects developed diabetes at 6 months (one from each group) and one at 12 months (from the control group). Twenty-nine of the treatment group (18 women and 11 men), average age 55.4 \pm 1.9 years, and 29 of the control group (12 women and 17 men), average age 56.9 \pm 1.9 years, completed the protocol.

Baseline characteristics

Baseline characteristics of those randomized who completed baseline studies are shown in Table 1. Although the two groups were not significantly different when sex was taken into consideration in the analysis, the treatment group had more women and consequently a signifi-

cantly lower amount of intra-abdominal fat in the univariate analysis. Similarly, waist girth was significantly lower in the treatment group in the univariate analysis.

Dietary and exercise adherence

At 3, 6, 9, 12, and 24 months of follow-up, the treatment group consumed 22.0–23.3% of calories as fat and 5.8–6.6% of calories as saturated fat. Thus, on average, the treatment group met the dietary goals of <30% of calories from fat and 7% from saturated fat. At these same time points, the percentage of treatment group participants who consumed <7% of calories as saturated fat ranged between 55 and 70%. Furthermore, 79–88% of treatment participants consumed <30% of calories from fat and 66–97% consumed <200 mg of cholesterol at the same time points.

For control participants at 3, 6, 9, 12, and 24 months of follow-up, the mean percentage of calories from fat was 24.6–29.7%, and from saturated fat, 7.1–8.5%; thus, participants on average met the dietary goals for this group of 30% of calories from fat and 10% from saturated fat. At the same time points, 77–88% of control group participants consumed <10% of calories as saturated fat; 59–79% consumed <30% of calories from fat; and 74–89% consumed <300 mg of cholesterol.

Change in $\text{VO}_{2\text{max}}$ was used to assess change in physical fitness. At the end of the 6 months of supervised exercise, the treatment group achieved a highly significant $3.3 \pm 0.8 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ improvement in $\text{VO}_{2\text{max}}$ ($P < 0.0001$) compared with the control group ($-0.6 \pm 0.6 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). At 24 months, the treatment group still had a significantly greater improvement over baseline ($2.6 \pm 0.7 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) compared with the control group ($-0.7 \pm 0.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$; $P = 0.0002$). The increases in the treatment group are about twice the improvement expected from weight loss alone (2.7 kg at 6 months and 2.8 kg at 24 months) and thus are at least partly due to improved physical fitness. An improvement of $1.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ was arbitrarily selected as indicative of an increase in physical fitness and as an estimate of adherence to treatment. This value represented one standard deviation less than the mean improvement of $3.3 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ observed at 6 months in the treatment group. The percentage of participants

showing a change in $\text{VO}_{2\text{max}}$ of $\geq 1.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ was 51.6% for the treatment group and 15.6% for the control group at 6 months ($P = 0.006$), and 59.3% and 13.8%, respectively, at 24 months ($P = 0.001$).

Adiposity at 6 and 24 months

Change at 6 and 24 months from baseline measurements of BMI, body composition, CT fat areas, and skinfolds are shown by group in Table 2. In general, except for intra-abdominal fat area, waist girth, and subscapular and forearm skinfold thickness, the treatment group showed a significantly greater reduction in all of the measured adiposity variables. Insofar as intra-abdominal fat area was concerned, there was no significant difference at 6 months between the two groups. Both groups together showed a mean reduction of $15 \pm 3 \text{ cm}^2$, a 15% decrease from their baseline value. At 24 months, the reduction in intra-abdominal fat area from baseline tended to be greater in the treatment group, although the difference was not significant. The difference in reduction in waist girth between the groups at 6 months was of borderline significance when adjustments were made for baseline and sex but was significantly different at 24 months. For subscapular skinfold thickness, the difference in reduction was of borderline significance between the groups at 6 months when adjustments were made for baseline and sex and significantly greater in the treatment group at 24 months. In general, the treatment group participants were able to maintain their reductions in adiposity variables at 24 months, although the reductions were somewhat smaller than at 6 months. Although the control group participants showed significant reductions in some adipose variables at 6 months—namely BMI, intra-abdominal and thorax subcutaneous fat areas, and subscapular, forearm, and suprailiac skinfold thickness—these changes were not maintained at 24 months.

Glucose tolerance

One participant in the treatment group and two in the control group developed diabetes. It should be noted, however, that this study was not designed to demonstrate prevention of diabetes. The proportion of participants showing normal glucose tolerance at least once during their 24 months of follow-up was significantly

cantly greater in the treatment group (67% vs. 30%; $P = 0.010$).

CONCLUSIONS— Previous epidemiologic studies in Japanese Americans have suggested that lifestyle factors may be responsible for their higher rates of type 2 diabetes compared with Japanese. More specifically, both lower levels of physical activity and higher intake of saturated fat were implicated (13–15). Furthermore, although Japanese Americans are not generally obese, they are nonetheless heavier than Japanese (11,12). Thus Japanese Americans with IGT, a powerful risk factor for type 2 diabetes (26), were selected to participate in this randomized clinical trial of endurance exercise and reduction of dietary saturated fat as interventions to improve adiposity variables associated with type 2 diabetes.

The benefits of increased physical activity and reduced dietary intake of saturated fat to reduce diabetes incidence have been confirmed in the Diabetes Prevention Program, the Da Qing IGT and Diabetes Study in China, and the Finnish Diabetes Prevention Study (6,8,9). Only some of the participants in the Da Qing Study were overweight, with $\text{BMI} \geq 25 \text{ kg/m}^2$, whereas all of those in the Finnish Study had to be overweight ($\text{BMI} \geq 25 \text{ kg/m}^2$). In the Diabetes Prevention Program, most of the participants were overweight and all had to have $\text{BMI} \geq 24 \text{ kg/m}^2$, except for Asians, who had to have $\text{BMI} \geq 22 \text{ kg/m}^2$. The Da Qing Study reported only change in BMI, while the Diabetes Prevention Program included change in weight and the Finnish Study included change in weight and waist circumference. None of the studies examined the full range of adiposity variables used in our study.

The participants in our study had an average BMI of 26 kg/m^2 , thus considered to be overweight but not obese. Although weight loss was not an intended outcome in either group, reductions in adiposity variables were nonetheless seen in both groups. At 6 months, the treatment group showed greater reductions in all adiposity variables, except for intra-abdominal fat, waist girth, and subscapular and forearm skinfolds, compared with the control group. Furthermore, although reductions were seen in some variables in the control group at 6 months, these were not maintained at 24 months. On the other hand, the treatment group continued to show

Table 2—Change in adipose variables (compared to baseline) at 6 and 24 months of follow-up

	Treatment	Control	P (t test)	P (ANCOVA)*
Weight (kg)				
6 months	−2.7 ± 0.4	−0.9 ± 0.3	0.0007	0.0003
24 months	−1.8 ± 0.5	0.7 ± 0.6	0.0022	0.0043
BMI (kg/m ²)				
6 months	−1.1 ± 0.2	−0.4 ± 0.1	0.0006	0.0003
24 months	−0.7 ± 0.2	0.2 ± 0.2	0.0023	0.0022
Percent body fat				
6 months	−1.4 ± 0.4	−0.3 ± 0.3	0.023	0.029
24 months	−0.8 ± 0.3	0.7 ± 0.5	0.014	0.0048
CT fat area (cm ²)				
Intra-abdominal				
6 months	−16.1 ± 3.2	−14.5 ± 4.6	0.8	0.2
24 months	−10.6 ± 3.5	−2.5 ± 7.1	0.3	0.10
Abdomen subcutaneous				
6 months	−29.3 ± 4.2	−5.7 ± 5.9	0.0018	0.0026
24 months	−15.5 ± 4.8	18.2 ± 6.8	0.0002	0.0002
Thigh subcutaneous				
6 months	−13.2 ± 3.6	−3.6 ± 3.0	0.048	0.037
24 months	−7.2 ± 3.0	1.3 ± 3.1	0.052	0.02719
Thorax subcutaneous				
6 months	−19.6 ± 3.6	−8.9 ± 2.6	0.017	0.0050
24 months	−13.9 ± 5.3	−3.0 ± 4.2	0.015	0.0065
Skinfold thickness (mm)				
Subscapular				
6 months	−3.8 ± 1.0	−1.8 ± 0.7	0.11	0.057
24 months	−4.6 ± 0.8	−2.9 ± 0.8	0.2	0.042
Bicep				
6 months	−2.0 ± 0.6	1.1 ± 0.6	0.0008	0.0002
24 months	0.6 ± 0.5	2.7 ± 0.8	0.035	0.015
Tricep				
6 months	−3.7 ± 0.8	−0.9 ± 0.6	0.0059	0.0031
24 months	−3.3 ± 0.8	−0.3 ± 0.7	0.0040	0.0002
Forearm				
6 months	−0.6 ± 0.2	−0.6 ± 0.2	1.0	0.5
24 months	−0.5 ± 0.2	−0.1 ± 0.5	0.4	0.3
Suprailiac				
6 months	−5.4 ± 1.4	−3.0 ± 0.9	0.14	0.061
24 months	−7.5 ± 1.4	−3.6 ± 1.6	0.072	0.037
Waist circumference (cm)				
6 months	−2.7 ± 0.6	−1.4 ± 0.8	0.19	0.085
24 months	−1.2 ± 0.9	0.9 ± 0.9	0.096	0.035

Data are means ± SD. *n* = 32 at 6 months and 29 at 24 months. *Adjusted for baseline value and for sex, where applicable.

reductions in adiposity measurements at 24 months, albeit somewhat less than at 6 months.

Of interest is the similar reduction of intra-abdominal fat in the two groups at 6 months. Intra-abdominal fat, although comprising only a small portion of total body fat stores, has been described to show rapid turnover. Prior studies have shown that during weight loss attributable to diet and endurance exercise, there

is a proportionately greater reduction of intra-abdominal fat than of total body fat stores (22–24). Although the treatment group showed a greater reduction of BMI, the reduction of intra-abdominal fat in the control group was nonetheless similar to that found in the treatment group. This suggests that small changes in overall adiposity as measured by BMI can be associated with disproportionately larger changes in intra-abdominal fat. Hence it is

important to consider that intra-abdominal fat may undergo large reductions despite modest reductions in BMI.

The improvement in adiposity variables seen at 6 months in the control group can probably be attributed to the effect of participation in a clinical trial. In the U.K. Prospective Diabetes Study, for example, glycemic control as estimated by HbA_{1c} improved in the control group (32). The control group participants

probably paid greater attention to diet, activity, and medication as a result of greater contact with diabetes care providers and specialists. This improvement in glucose control disappeared after the first year of the trial when they did not receive regular reinforcement and coaching to follow intensive diabetes control. Probably for similar reasons, the initial improvement of adiposity variables was not maintained in our control group.

Although weight loss was not a goal, the treatment group experienced and maintained a significant reduction in BMI at 6 and 24 months. The concomitant reductions in percent body fat, CT fat areas, and skinfolds suggest that fat mass was reduced over lean mass.

Other studies that have successfully demonstrated prevention of type 2 diabetes through diet and exercise interventions have examined a limited number of adipose variables. In the Da Qing Study, using diet and exercise separately or in combination, weight loss was a goal in those participants with BMI ≥ 25 kg/m² (8). At 6 years of follow-up, reduction in BMI was very similar to that observed in our participants at 24 months. The Finnish Study, using a combination of diet and exercise and with a treatment goal of losing at least 5% of weight, found that intervention group participants lost weight and reduced their waist circumference, and the changes were significant compared with the control group at 1 year (9). The Diabetes Prevention Program also found that a combination of diet and exercise, with a treatment goal of at least 7% weight loss, resulted in a significant reduction in weight compared with the control group over an average 2.8 years of follow-up (6).

Many of our participants were already consuming an AHA step 1 diet at baseline, a diet very low in fat by U.S. standards. Furthermore, the amount of dietary fat was lower than what we had previously reported in our epidemiologic studies (14). Thus the participants in this study may not be representative of all Japanese Americans with respect to their usual diet. We do not have a ready explanation for this. Food records over follow-up confirmed that on average the control group adhered to the AHA step 1 diet, whereas the treatment group achieved and adhered to the AHA step 2 diet.

Physical fitness was assessed by measuring $\text{VO}_{2\text{max}}$. Baseline physical fitness

was similar in the two groups and was as expected for individuals in the age range represented by our participants. Although $\text{VO}_{2\text{max}}$ is not a direct measure of whether the participants actually performed exercise as prescribed, an increase in $\text{VO}_{2\text{max}}$ is a reliable indicator of an improvement in fitness. The increases in $\text{VO}_{2\text{max}}$ observed in the treatment group were greater than could be attributed to weight loss. Thus the increase in $\text{VO}_{2\text{max}}$ observed in the treatment group at 6 months suggests an improvement in physical fitness as a consequence of exercise training during this initial period. Most of the improvement in $\text{VO}_{2\text{max}}$ seen at 6 months was still present at 24 months. The control group was not instructed to perform aerobic exercise and had no change in $\text{VO}_{2\text{max}}$. We therefore conclude that the treatment group exercised more than the control group.

One of the limitations of this study is the small sample size, which may limit the ability to detect clinically meaningful differences owing to insufficient statistical power. For example, this may be an explanation for the lack of difference in change of intra-abdominal fat. Nonetheless, the results obtained suggest that in studies that have reported the prevention of type 2 diabetes through lifestyle modification, the benefits are probably mediated through not only overall weight or fat loss but also through improvement in distribution of body fat.

Thus this study has demonstrated that regular participation in endurance exercise and adherence to a diet that is reduced in saturated fat not only reduces overall adiposity but also improves body fat distribution in a nonobese group of Japanese Americans with IGT. The ability of our subjects to adhere to an intervention without close supervision is of practical importance, suggesting that the prescribed lifestyle changes may not be difficult to follow. The fact that most of our participants were already consuming a diet low in saturated fat at baseline suggests that they might already have been more conscious of healthy lifestyle practices, and furthermore that low levels of physical activity might have played an important etiologic role in the pathogenesis of IGT in this otherwise healthy group.

In conclusion, the results of this study suggest that lifestyle modification consisting of a reduction of dietary fat intake, particularly saturated fat, and regular par-

ticipation in endurance exercise improves BMI, body composition, and body fat distribution in Japanese Americans with IGT, and thus may be effective in delaying or preventing type 2 diabetes. Moreover, the results of this study complement three recent and large studies that have shown significant reduction of diabetes incidence through lifestyle change.

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References

- King H, Herman WH: Global burden of diabetes 1995–2025. *Diabetes Care* 21: 1414–1431, 1998
- Kannel WB, McGee DL: Diabetes and cardiovascular risk factors: the Framingham study. *Circulation* 59:8–13, 1979
- Herman WH, Eastman RC: The effects of treatment on the direct costs of diabetes. *Diabetes Care* 21 (Suppl. 3):C19–C24, 1998
- Amos AF, McCarty DJ, Zimmet P: The rising global burden of diabetes and its complications: estimates and projections to the year 2010. *Diabet Med* 14:S1–85, 1997
- The Diabetes Prevention Program: Design and methods for a clinical trial in the prevention of type 2 diabetes. *Diabetes Care* 22:623–634, 1999
- 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
- Uusitupa M, Louheranta A, Lindstrom J, Valle T, Sundvall J, Eriksson J, Tuomilehto J: The Finnish Diabetes Prevention Study. *Br J Nutr* 83 (Suppl. 1):S137–S142, 2000
- Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB: 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
- Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukkaanniemi S, Laakso M, Louheranta A, Rastas 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
10. Eriksson KF, Lindgarde F: Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmö feasibility study. *Diabetologia* 34:891–898, 1991
11. Fujimoto WY, Bergstrom RW, Boyko EJ, Kinyoun JL, Leonetti DL, Newell-Morris LL, Robinson LR, Shuman WP, Stolov WC, Tsunehara CH: Diabetes and diabetes risk factors in second- and third-generation Japanese Americans in Seattle, Washington. *Diabetes Res Clin Pract* 24 (Suppl.):S43–S52, 1994
12. Fujimoto WY, Akanuma Y, Kanazawa Y, Mashiko S, Leonetti D, Wahl P: Plasma insulin levels in Japanese and Japanese-American men with type 2 diabetes may be related to the occurrence of cardiovascular disease. *Diabetes Res Clin Pract* 6:121–127, 1989
13. Fujimoto WY, Bergstrom RW, Newell-Morris L, Leonetti DL: Nature and nurture in the etiology of type 2 diabetes mellitus in Japanese Americans. *Diabetes Metab Rev* 5:607–625, 1989
14. Tsunehara CH, Leonetti DL, Fujimoto WY: Diet of second-generation Japanese-American men and non-insulin-dependent diabetes. *Am J Clin Nutr* 52:731–738, 1990
15. Leonetti DL, Tsunehara CH, Wahl PW, Fujimoto WY: Baseline dietary intake and physical activity in Japanese American men in relation to glucose tolerance at 5-year follow-up. *Am J Hum Biol* 8:55–67, 1996
16. Bergstrom RW, Leonetti DL, Newell-Morris LL, Shuman WP, Wahl PW, Fujimoto WY: Association of plasma triglyceride and C-peptide with coronary heart disease in Japanese-American men with a high prevalence of glucose intolerance. *Diabetologia* 33:489–496, 1990
17. Fujimoto WY, Kahn SE, Hokanson JE, Brunzell JD: The visceral adiposity syndrome in Japanese-American men. *Obes Res* 2:364–371, 1994
18. Boyko EJ, Leonetti DL, Bergstrom RW, Newell-Morris L, Fujimoto WY: Visceral adiposity, fasting plasma insulin, and blood pressure in Japanese Americans. *Diabetes Care* 18:174–181, 1995
19. Boyko EJ, Leonetti DL, Bergstrom RW, Newell-Morris L, Fujimoto WY: Visceral adiposity, fasting plasma insulin, and lipid and lipoprotein levels in Japanese Americans. *Int J Obes* 20:801–808, 1996
20. Fujimoto WY, Bergstrom RW, Boyko EJ, Chen KW, Leonetti DL, Newell-Morris L, Shofer JB, Wahl PW: Visceral adiposity and incident coronary heart disease in Japanese-American men: the 10-year follow-up results of the Seattle Japanese-American Community Diabetes Study. *Diabetes Care* 22:1808–1812, 1999
21. Boyko EJ, Fujimoto WY, Leonetti DL, Newell-Morris L: Visceral adiposity and risk of type 2 diabetes mellitus: a prospective study among Japanese Americans. *Diabetes Care* 23:465–471, 2000
22. Ross R, Hudson R: Sensitivity associated with the identification of visceral adipose tissue levels using waist circumference in men and women: effects of weight loss. *Internat J Obes Relat Metab Disord* 20:533–538, 1996
23. Colman E, Katzell LI, Rogus E, Coon P, Muller D, Goldberg AP: Weight loss reduces abdominal fat and improves insulin action in middle-aged and older men with impaired glucose tolerance. *Metabolism* 44:1502–1508, 1995
24. Mourier A, Gautier JF, De Kerviler E, Bigard AX, Villette JM, Garnier JP, Duvallet A, Guezennec CY, Cathelineau G: Mobilization of visceral adipose tissue related to the improvement in insulin sensitivity in response to physical training in NIDDM: effects of branched-chain amino acid supplements. *Diabetes Care* 20:385–391, 1997
25. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Mott DM, Bennett PH: The natural history of impaired glucose tolerance in the Pima Indians. *N Engl J Med* 319:1500–1506, 1988
26. Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, Haffner SM, Pettitt DJ, Sorkin JD, Muller DC: Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. *Diabetes* 46:701–710, 1997
27. Alberti KG, Zimmet PZ: Definition, diagnosis and classification of diabetes mellitus and its complications. I. Diagnosis and classification of diabetes mellitus: provisional report of a WHO consultation. *Diabet Med* 15:539–553, 1998
28. Freidman LM, Furberg CD, DeMets DL (Eds): *Fundamentals of Clinical Trials*, 2nd ed. Littleton, MA, PSG Publishing, 1985, p. 59–62
29. Lohman TG, Roach AF, Martorell R (Eds): *Anthropometric Standardization Reference Manual*. Champaign, IL, Human Kinetics, 1988
30. Goldman RF: A method for underwater weighing and the determination of body density. In *Techniques for Measuring Body Composition*. Brozek HA, Ed. Washington, DC, National Academy of Sciences, 1961, p. 78–79
31. Shuman WP, Morris LL, Leonetti DL, Wahl PW, Moceri VM, Moss AA, Fujimoto WY: Abnormal body fat distribution detected by computed tomography in diabetic men. *Invest Radiol* 21:483–487, 1986
32. United Kingdom Prospective Diabetes Study (UKPDS). 13: Relative efficacy of randomly allocated diet, sulphonylurea, insulin, or metformin in patients with newly diagnosed non-insulin dependent diabetes followed for three years. *BMJ* 310:83–88, 1995