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HIGHLIGHTED TOPIC | *Role of Exercise in Reducing the Risk of Diabetes and Obesity*

Inactivity, exercise, and visceral fat. STRRIDE: a randomized, controlled study of exercise intensity and amount

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Slentz, Cris A., Lori B. Aiken, Joseph A. Houmard, Connie W. Bales, Johanna L. Johnson, Charles J. Tanner, Brian D. Duscha, and William E. Kraus. Inactivity, exercise, and visceral fat. STRRIDE: a randomized, controlled study of exercise intensity and amount. *J Appl Physiol* 99: 1613–1618, 2005. First published July 7, 2005; doi:10.1152/japplphysiol.00124.2005.—Despite the importance of randomized, dose-response studies for proper evaluation of effective clinical interventions, there have been no dose-response studies on the effects of exercise amount on abdominal obesity, a major risk factor for metabolic syndrome, diabetes, and cardiovascular disease. One hundred seventy-five sedentary, overweight men and women with mild to moderate dyslipidemia were randomly assigned to participate for 6 mo in a control group or for ~8 mo in one of three exercise groups: *1*) low amount, moderate intensity, equivalent to walking 12 miles/wk (19.2 km) at 40–55% of peak oxygen consumption; *2*) low amount, vigorous intensity, equivalent to jogging 12 miles/wk at 65–80% of peak oxygen consumption; or *3*) high amount, vigorous intensity, equivalent to jogging 20 miles/wk (32.0 km). Computed tomography scans were analyzed for abdominal fat. Controls gained visceral fat (8.6 ± 17.2%; $P = 0.001$). The equivalent of 11 miles of exercise per week, at either intensity, prevented significant accumulation of visceral fat. The highest amount of exercise resulted in decreased visceral (−6.9 ± 20.8%; $P = 0.038$) and subcutaneous (−7.0 ± 10.8%; $P < 0.001$) abdominal fat. Significant gains in visceral fat over only 6 mo emphasize the high cost of continued inactivity. A modest exercise program, consistent with recommendations from the Centers for Disease Control/American College of Sports Medicine (CDC/ACSM), prevented significant increases in visceral fat. Importantly, a modest increase over the CDC/ACSM exercise recommendations resulted in significant decreases in visceral, subcutaneous, and total abdominal fat without changes in caloric intake.

Studies of Targeted Risk Reduction Interventions through Defined Exercise; exercise training; visceral fat; exercise amount

THE PREVALENCE OF OVERWEIGHT and obesity is high and continues to rise, presenting ever-increasing challenges for individuals and health professionals. Overweight individuals are at increased risk for cardiovascular disease, diabetes, and other health disorders (2, 5, 14, 21, 24). The location of the excess weight is of particular importance, because the strength of the relation between central obesity and disease risk is well documented (6, 13, 17, 30, 32), with visceral fat often considered the major culprit (3, 4, 12, 20, 22, 23). In addition, several studies have also shown a significant relationship between abdominal subcutaneous fat and metabolic risk factors (1, 8, 18–20). It is important for interventions designed to reduce abdominal obesity to monitor their effects on both visceral and subcutaneous fat.

Controversy exists regarding the minimal and/or optimal amount of exercise needed for health benefits. Interestingly, despite the importance of centrally located body fat, there are few if any prospective exercise training studies that compare the effects of different amounts and intensities of exercise on changes in parameters of central obesity. Studies of Targeted Risk Reduction Interventions through Defined Exercise, a randomized, controlled clinical trial, was prospectively designed to investigate, in an 8-mo training study, the separate effects of the amount of exercise and exercise intensity on cardiovascular risk factors in overweight men and women with mild to moderate dyslipidemia. This report summarizes the effects of exercise amount and intensity on visceral, subcutaneous, and total abdominal fat.

METHODS

A complete description of the Studies of Targeted Risk Reduction Interventions through Defined Exercise design, hypotheses, recruitment strategies, methods, and preliminary recruiting results are published elsewhere (16).

Subjects. Subjects ($n = 330$) from Durham, Greenville, and surrounding communities in North Carolina met inclusion criteria and were randomized into the study. Sixty-eight percent (225) completed the 8-mo study. Of the 225 completers, 78% ($n = 175$) had complete pre- and poststudy computed tomography (CT) scan data, and the data from these subjects were included in this analysis. There were no differences in any variables measured between those in the subgroup who had CT scans and those who did not have scans. Inclusion criteria were 40–65 yr of age, sedentary (exercise <2 times/wk), overweight or mildly obese (body mass index of 25–35 kg/m^2) with mild to moderate lipid abnormalities [either low-density lipoprotein (LDL) cholesterol of 130–190 mg/dl or high-density lipoprotein (HDL) cholesterol of <40 mg/dl for men or <45 mg/dl for women]. Women were postmenopausal. Exclusion criteria were diabetes, hypertension, other metabolic or musculoskeletal diseases, current use of or intent to

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diet, use of confounding medications, overt presence of coronary heart disease, or unwillingness to be randomized to any group. The study was approved by an Institutional Review Board. After written, informed consent was given, subjects were randomly assigned to one of three 8-mo exercise groups (~2 mo of ramp-up and 6 mo of steady-state training) or a 6-mo control group. Individuals initially assigned to the control group were promised to be randomized into an exercise group at the end of the control period. However, only their control data were used in any analyses. The research protocol was approved by the institutional review boards at Duke University and East Carolina University.

Exercise training protocols (dose, mode, and intensity). The exercise groups were *1*) high amount/vigorous intensity (equivalent to jogging 20 miles/wk), *2*) low amount/vigorous intensity (equivalent to jogging 12 miles/wk), and *3*) low amount/moderate intensity (equivalent to walking 12 miles/wk). Details are published elsewhere (15, 16). The actual exercise prescription was to expend 14 kcal·kg body wt^{-1}·wk^{-1} for the two low-amount groups (26) and to expend 23 kcal·kg body wt^{-1}·wk^{-1} for the high-amount group. Although the amount of exercise is expressed in terms of walking or jogging a certain distance to simplify the description of the exercise groups, the main exercise modalities were treadmills and elliptical trainers, with some use of cycle ergometers. Subjects could use any or all of these modalities. The specific exercise intensities were 65–80% of peak oxygen consumption for the two vigorous-intensity groups and 40–55% of peak oxygen consumption for the moderate-intensity group. The exercise capacity was determined via a graded maximal exercise test with continuous measurement of oxygen consumption. The actual work rate, correlating to the prescribed exercise intensity, was determined during a submaximal exercise test performed on a separate day during the first 2–3 wk of exercise training.

Exercise training (verification, adherence, initial ramp period, and training duration). All exercise sessions were verified by direct supervision or by use of a heart rate monitor that provides recorded data (Polar Electro, Woodbury, NY). Adherence was calculated weekly as a percent, equal to the actual number of exercise minutes completed each week at the appropriate intensity, divided by the total number of minutes prescribed. There was an initial ramp period of 2–3 mo where exercise duration and exercise intensity were gradually increased until the appropriate exercise prescription was obtained. This initial ramping period was followed by 6 additional mo of training at the appropriate exercise prescription.

Dietary evaluations and control of body weight. Nutrient intakes of each subject were determined via 3-day food records and 24-h recalls, before and after exercise training. To study the effects of exercise alone and eliminate the confounding effects of major weight loss, subjects were counseled not to diet or change their diet during this study.

Body weight and CT measures. Height (to the nearest 0.64 cm) and weight (to the nearest 0.1 kg) were measured in light clothing without shoes on a digital electronic scale (Scale-Tronix 5005, Wheaton, IL). All CT scans were performed on a General Electric CT/I (GE Medical Systems, Milwaukee, WI). After a digital frontal scout radiograph of the abdomen was obtained, a single 10-mm-thick axial section was performed at the level of the L-4 pedicle. CT scans were analyzed using Slice-O-matic software from TomoVision to determine surface area of the visceral and subcutaneous abdominal compartments.

Statistical methods for repeated measures and correlations. Baseline descriptive statistics include means and standard deviations (see Table 1). Paired *t*-tests (2-tailed) were used to determine whether a change within any specific group was significant (see Table 2). To determine whether there were significant differences between groups, data were analyzed using one-way ANOVA (Statview Software, SAS Institute, Cary, NC) (see Fig. 1). All ANOVA tests performed were found to be significant. Therefore, a Fishers paired least-significant difference post hoc test was performed to determine which groups were significantly different from the others. P values of <0.05 were considered significant.

RESULTS

Baseline and exercise prescription data are presented in Table 1. There were no differences at baseline between groups for age, body mass index, caloric intake, or percentage of calories from macronutrients. The number of women and men was nearly equal in each group, and minorities made up 19.5% of the subject population. The total amount of exercise time for each group was approximately 3 h/wk for the low-amount/

Table 1. *Baseline characteristics and exercise prescription*

Variables	Total Group	Control	Low Amount, Moderate Intensity	Low Amount, Vigorous Intensity	High Amount, Vigorous Intensity
n	175	47	40	46	42
Age	52.7 (6.5)	52.3 (7.65)	54.0 (5.5)	53.0 (7.0)	51.5 (5.3)
BMI, kg/m^2	29.6 (3.0)	29.8 (3.0)	29.8 (3.2)	29.7 (3.1)	29.1 (2.4)
Race					
Caucasian	137 (80.6%)	36	32	36	37
African American	29 (17.1%)	11	8	8	3
Asian/Hispanic	4 (2.4%)	0	0	2	2
Women/Men	84/91	24/23	18/22	23/23	19/23
Food intake, kcal/day	2,079 (596)	2,047 (536)	2,075 (584)	2,079 (668)	2,072 (495)
CHO, %	48.7 (9.3)	51.3 (9.5)	48.6 (7.7)	47.8 (10.0)	46.8 (9.5)
Fat, %	33.7 (7.4)	31.6 (8.4)	34.2 (5.7)	34.7 (7.0)	34.5 (7.7)
Protein, %	15.8 (3.9)	15.6 (3.7)	15.8 (4.3)	15.5 (2.7)	16.3 (4.9)
Exercise prescription					
Intensity, % peak $\dot{V}O_2$)			40–55	65–80	65–80
Prescription amount, kcal·kg^{-1}·wk^{-1}*			14	14	23
Prescription time, min/wk			204 (43)	129 (29)	208 (37)
Adherence, %			88.5 (13.6)	93.5 (10.1)	83.7 (15.1)
Actual amount, miles/wk			10.5	11.2	16.9
Actual time, min/wk†			178 (37)	120 (27)	173 (41)
Frequency, sessions/wk			3.5 (0.6)	3.1 (0.5)	3.6 (0.8)

Values are means (SD). BMI, body mass index; CHO, carbohydrate; $\dot{V}O_2$, O_2 uptake. There were no significant baseline differences between groups. *Prescription amount (14 and 23 kcal·kg^{-1}·wk^{-1}) are approximately calorically equivalent to 12 and 20 miles of walking or jogging per week for the low-dose and high-dose groups, respectively. †Actual time = adherence × prescription time.

Table 2. *Baseline and change scores for visceral fat, subcutaneous abdominal fat, total abdominal fat, and body weight*

	Control (n = 47)			Low Amount/Moderate Intensity (n = 40)			Low Amount/Vigorous Intensity (n = 46)			High Amount/Vigorous Intensity (n = 42)		
Variable	Baseline	%Change	P value	Baseline	%Change	P value	Baseline	%Change	P value	Baseline	%Change	P value
Visceral fat	165 (68)	8.6 (17.2)	0.001*	173 (72)	1.7 (19.7)	0.58	154 (55)	2.5 (21.3)	0.43	168 (64)	−6.9 (20.8)	0.038*
Subcutaneous fat	313 (107)	1.1 (11.9)	0.53	287 (103)	−1.2 (11.8)	0.54	291 (97)	3.1 (18.7)	0.27	274 (78)	−7.0 (10.8)	0.000*
Total abdominal fat	477 (127)	3.9 (10.4)	0.015*	460 (132)	0.2 (10.6)	0.91	444 (114)	2.0 (15.5)	0.38	442 (102)	−6.8 (12.0)	0.001*
Body weight, kg	86.9 (14.2)	1.0 (2.7)	0.017*	88.0 (16.3)	−0.7 (2.1)	0.032*	85.0 (13.4)	−0.8 (2.3)	0.027*	85.7 (12.2)	−2.6 (3.3)	0.000*

Values are means (SD). There were no significant baseline differences between groups. Abdominal fat measures are from computed tomography in cm^2. *Significant change score (post compared with pre value, paired t-test, 2-tailed).

moderate-intensity and high-amount/vigorous-intensity groups and was 2 h/wk for the low-amount/vigorous-intensity group. Caloric intake, measured before and after exercise training, did not change significantly ($P > 0.20$) in any exercise group or in the control group (data not shown).

In Table 2, the results from paired t-tests on the change scores for within-group comparisons are presented for each of the CT-derived abdominal fat compartments and for body weight. In the control group, visceral fat levels increased by 8.6%, which was statistically significant ($P = 0.001$). Visceral fat levels did not change significantly in either of the low-amount exercise groups. The high-amount exercise group experienced an average decrease in visceral fat of 6.9%, which was significant ($P = 0.038$). Only the high-amount exercise

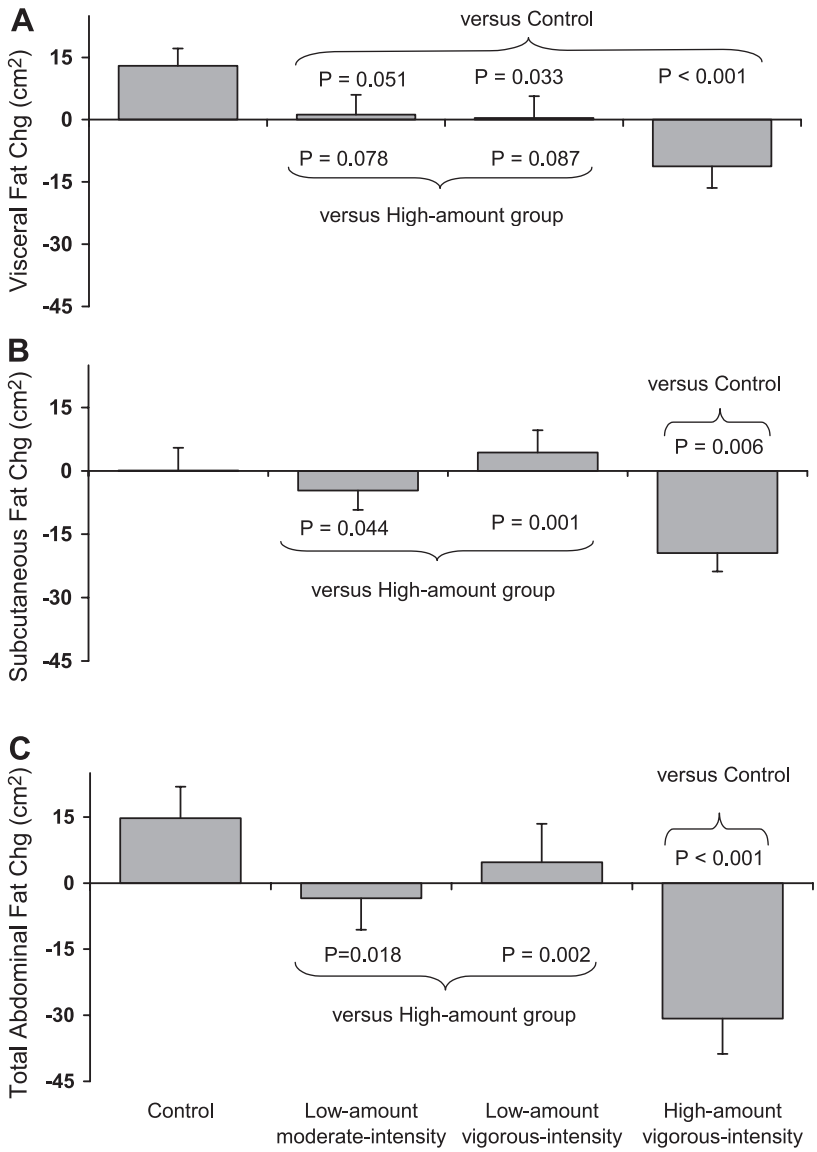

Fig. 1. Comparison of the effects of continued physical inactivity (controls) and 3 different exercise training programs on mean changes (Chg) in visceral abdominal fat (*A*), subcutaneous abdominal fat (*B*), and total abdominal fat (*C*). Subjects in the control group maintained their normal diet and level of physical activity for 6 mo. In the exercise groups, the amount and intensity of exercise were gradually increased to the prescribed level over the course of 1–3 mo, after which time exercise was maintained at the prescribed level of 6 mo. Low-amount, moderate-intensity exercise represents the caloric equivalent of walking ~12 miles/wk at 40–55% of peak oxygen consumption; low-amount, vigorous-intensity exercise represents the same amount of exercise at 65–80% of peak oxygen consumption. High-amount, vigorous-intensity exercise represents the caloric equivalent of jogging ~20 miles/wk at 65–80% of oxygen consumption. Values shown represent means of individual change scores. Error bars represent standard errors.

group had any change in subcutaneous abdominal fat amount, which decreased in this group by 7.0% ($P < 0.001$). The significant increase in total abdominal fat in the controls reflects the increase in visceral fat. Neither low-amount exercise group experienced significant change in subcutaneous or total abdominal fat. Body weight increased significantly in the control group (0.88 kg) and decreased significantly in all exercise groups (0.60 in both low-amount groups and 2.31 kg in the high-amount group) in a dose-response manner, i.e., greater weight loss with greater amounts of exercise (in kcal expended·kg^{-1}·wk^{-1}). For the purposes of defining dose-response effects, both dose and amount refer to the number of kilocalories expended via exercise per kilogram of body weight per week. Exercise intensity did not appear to have any effect on body weight or any of the abdominal fat compartments, as both low-amount groups experienced similar responses.

In Fig. 1, comparisons between the four groups illustrate the effects of continued physical inactivity (controls) and different amounts and intensities of exercise training on visceral, subcutaneous, and total abdominal fat. In Fig. 1*A*, the effect of exercise amount on visceral fat reveals a dose-response relationship, where the control group gained visceral fat relative to all three exercise groups (albeit, the low-amount/moderate-intensity group was at the margin of statistical significance, compared with the controls). And the high-amount group lost more visceral fat than the low-dose groups, although these differences did not quite achieve statistical significance ($P > 0.07$ and <0.09). In Fig. 1*B*, the data show that little or no change in subcutaneous abdominal fat for the controls and both low-dose exercise groups, whereas the high-amount exercise group lost a significant amount of subcutaneous abdominal fat compared with the other three groups. Figure 3*C* illustrates the additive effects of both the subcutaneous and visceral changes where the high-amount exercise group lost significantly more total abdominal fat compared with the other three groups. When gender was added to the ANOVA model, no significant gender effects were observed ($P > 0.20$).

The correlations between abdominal fat depots (subcutaneous and visceral abdominal fat) and variables of metabolic risk (lipid and carbohydrate variables) are shown in Table 3. Both pretreatment (baseline) correlations and change score correlations are shown. Baseline subcutaneous abdominal fat was not significantly related to any baseline metabolic risk variable, whereas baseline visceral fat was highly significantly related to all baseline metabolic risk variables tested (and presented) ($P < 0.0001$). All posttreatment correlations were essentially the same as pretreatment variables (data not shown). Correlations between change in abdominal fat depots (both subcutaneous and visceral) and changes in the metabolic risk variables also are shown in Table 3. The change in subcutaneous abdominal fat was significantly correlated only to the change in HDL size ($P < 0.05$). The change in visceral fat was significantly related to change in LDL particle number and change in insulin sensitivity index. Correlations between change in visceral fat and changes in the other metabolic variables (HDL size, LDL size, and triglyceride) were just on the border of statistical significance ($P < 0.10$ and >0.05).

Table 3. *Correlations between change in abdominal fat and change in variables of metabolic risk at baseline*

Variable	Subcutaneous Abdominal Fat		Visceral Fat	
	Correlation	*P* value	Correlation	*P* value
HDL size	−0.18	0.025*	−0.15	0.057
LDL size	−0.08	NS	−0.16	0.052
LDL particle no.	0.109	NS	0.22	0.006*
Triglyceride	0.10	NS	0.14	0.080
Si	−0.11	NS	−0.19	0.018*

HDL, high-density lipoprotein; LDL, low-density lipoprotein; NS, nonsignificant, $P > 0.10$; Si, insulin sensitivity as derived from minimal model analysis of the frequently sampled intravenous glucose tolerance test. *Statistically significant.

DISCUSSION

The important relationships between disease risk and central obesity in general and visceral obesity in particular are well described (2–4, 6, 12, 13, 17, 20, 22, 23, 30, 32). In this study, we present data from the first prospective, randomized, controlled study on the effects of different amounts of exercise on visceral, subcutaneous, and total abdominal fat. The data support several key findings. First, in sedentary, overweight adults assigned to the control group, a relatively short period of continued physical inactivity resulted in a sizeable and significant increase in visceral abdominal fat. This finding emphasizes the high cost of continuing to choose a sedentary lifestyle for overweight, middle-aged adults. A second key finding was that in both low-amount groups, no significant increase in visceral fat was observed, suggesting that this amount of exercise was adequate for preventing the deterioration seen in the inactive controls. This is an important observation, because this amount of exercise is similar to that recommended by the Center for Disease Control/American College Sports Medicine (27) and because the importance of prevention (14, 21) is highlighted by the high rate of weight-loss recidivism. Until we are able prevent weight regain after short-term dieting success, a greater emphasis toward prevention should be a major goal in the US (14, 21). Third, the observation that the high amount of exercise (approximately equivalent to 17 miles/wk of vigorous exercise) not only prevented increases in visceral fat but actually resulted in sizable and significant decreases in visceral fat, as well as in subcutaneous and total abdominal fat, suggests an exercise prescription for reversing metabolic disease. That this amount of exercise can reverse metabolic disease is supported by the present data and by previously published findings from this cohort that showed improvements in lipids and lipoproteins (15), insulin sensitivity (10), and body mass and fat mass loss (31).

Taken together, the data suggest a clear dose-response relationship between exercise amount and changes in visceral fat. Our interpretation of the data as indicating a dose-response relationship between exercise amount (in kcal·kg body wt^{-1}·wk^{-1}) is based on two points. First, there are multiple possible responses that can be characterized as a dose-response relationship, as illustrated by Haskell (9). All fulfill the basic concept that, with greater amounts of exercise, greater biological benefits accrue. With some health benefits, the response relationship might be curvilinear, whereas with others the response may be linear. Second, if we fit a linear curve to the mean visceral fat response (as seen in Fig. 1*A*) vs. the mean exercise dose for each group (calculated as amount of exercise

prescribed times adherence), we observe a relationship with a r^2 of 0.96. Fitting a best-fit curvilinear "trend line" results in a r^2 of 0.99. Although in our study all intergroup comparisons were not significantly different, a higher powered study might have found a difference. Although analysis of variance revealed a highly significant group effect, there were no significant gender effects ($P > 0.20$) or group × gender interactions ($P > 0.20$).

The importance of visceral fat and its associations with risk factors for coronary heart disease and Type 2 diabetes have been well established. Although overall obesity is clearly related to increased risk for these diseases, the greater importance of the location of adipose tissue is illustrated by the finding that, compared with total body fat, visceral fat is a significantly higher correlate of insulin response to a glucose challenge, fasting triglycerides, both systolic and diastolic blood pressure, and for HDL-to-total cholesterol ratio. In fact, visceral fat explains approximately twice the amount of variance in these variables compared with total body fat (13, 28). Our data support these observations. In a recent study comparing lean insulin-sensitive subjects to lean insulin-resistant subjects and obese insulin-resistant subjects, the data revealed that differences in visceral fat explain much of the atherogenic lipoprotein profile that is associated with obesity and insulin resistance (23). Whether visceral obesity is a major contributor to disease risk or simply a covariate of other causative factors is controversial (7, 30). Either way, the consistent, significant associations between visceral fat and risk factors for coronary heart disease and Type 2 diabetes suggests that it is, at the very least, a good marker of increased risk for these diseases.

Despite its importance, there are few randomized, controlled studies of the effects of exercise on visceral fat. In a 12-wk study in overweight men, Ross et al. (29) reported that an exercise program designed to increase energy expenditure by 700 kcal/day for 12 wk resulted in a weight loss of 7.5 kg and a decrease in visceral fat of 52 cm^2 (reported as the cross-sectional area of fat on a single CT scan), corresponding to a decrease of 6.9 cm^2 visceral fat per kilogram of weight loss. The men in their diet-only group (700 kcal deficit) had a similar decrease of 5.9 cm^2 per kilogram of weight loss. In the present study, the men in the high-amount exercise group experienced a reduction of 5.6 cm^2 per kilogram of weight loss. Irwin et al. (11) studied the effects of a 12-mo exercise program in overweight postmenopausal women and found a decrease of 8.5 cm^2 of visceral fat and 1.3 kg of body weight, corresponding to a ratio of 6.5 cm^2 per kilogram of weight change. In the high-amount exercise group from the present study, the women lost 6.9 cm^2 of visceral fat per kilogram of body weight.

In cross-sectional studies, visceral fat is often found to be significantly correlated with metabolic risk factors. Statistically significant correlation coefficients ranging from 0.30 to 0.60 are often reported for the relation between visceral fat and numerous lipid and carbohydrate risk factors in these studies (12, 22, 25). Our cross-sectional data reveal similar magnitude coefficients when baseline metabolic risk variables are correlated to baseline visceral fat ($P < 0.0001$; coefficients range from 0.27 to 0.44 for the variables reported in Table 3; data not shown). However, as can be seen in Table 3, when we correlate change in visceral fat levels with change in these metabolic variables, the magnitude of the coefficients is much lower, although the relations are significant (for LDL particle number and insulin sensitivity) or just on the border of statistical significance. Although Jansen et al. (12) reported baseline correlations ranging from 0.32 to 0.51 (all $P < 0.05$), they found no significant correlations between change in visceral fat levels and change in metabolic variables after weight loss from diet only or diet plus exercise.

It is important to remember that most if not all of the data linking visceral fat with metabolic variables are associative, not causative. There is much controversy as to whether visceral fat is a major health culprit or simply a marker of obesity-related health problems (30). Ravussin and Smith make a compelling case that failure to develop adequate fat cell mass in the face of excess energy intake may be the primary culprit, which then leads to ectopic fat deposition and in this way link visceral fat and adiposity to disease (28a). Either way, it seems clear that visceral fat, whether causative or simply a more specific marker of disease risk than general obesity, is an important health parameter.

Major strengths of this study include *1*) the randomized, controlled design; *2*) the dose-response testing; *3*) the direct verification of time and intensity and, therefore, exposure for nearly all exercise training sessions; *4*) the carefully defined and controlled exercise amounts and intensities; *5*) a significant proportion of women and minorities in the study population; *6*) an exercise stimulus that is identical for men and women (defined by kilocalories of energy expenditure per kilogram of body weight per week rather than the same number of minutes per week) that allows for better comparisons of exercise effects between genders; and finally *7*) a large number of subjects in each group yielding good statistical power to detect important exercise exposure effects. One important limitation should be noted. Due to practical reasons, we could not compare the effects of a training regimen with high weekly exercise amount at the lower intensity. We believed that the large amount of time necessary for a high-amount/moderate-intensity group (up to 8 h/wk for low fitness subjects) coupled with the fact that volunteer participants had to be willing to be randomly assigned to any group, would seriously limit recruiting ability and thus generalizability of the findings. Instead, our study was designed to look at the effects of exercise amount separately (by looking at the 2 groups that exercised at the same intensity but completed different amounts of total exercise) and exercise intensity separately (by comparing the 2 low-amount exercise groups that exercised at different intensities).

In conclusion, continued physical inactivity in a sedentary middle-aged overweight population led to significant gains in visceral abdominal fat over a relatively short period of time (6 mo). This finding emphasizes the high cost of continued physical inactivity for sedentary, overweight adults. Even a relatively modest exercise program, consistent with the activity recommendations from Centers for Disease Control and American College of Sports Medicine prevented significant increases in visceral abdominal fat. In view of the high rate of recidivism with weight-loss programs, the importance of prevention cannot be overemphasized. However, a modest increase in weekly caloric expenditure over Centers for Disease Control and American College of Sports Medicine recommendations resulted in significant decreases in visceral, subcutaneous, and total abdominal fat without significant changes in caloric intake. Both the detrimental effects seen in the inactive

control group and the beneficial effects of the high-amount exercise were observed in men and women.

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