Women Walking for Health and Fitness

How Much Is Enough?

John J. Duncan, PhD; Neil F. Gordon, MBCh, PhD; Chris B. Scott, MS

Objective.—We studied whether the quantity and quality of walking necessary to decrease the risk of cardiovascular disease among women differed substantially from that required to improve cardiorespiratory fitness.

Design.—A randomized, controlled, dose-response clinical trial with a follow-up of 24 weeks.

Setting.—A private, nonprofit biomedical research facility.

Participants.—One hundred two sedentary premenopausal women, 20 to 40 years of age, were randomized to one of four treatment groups; 59 completed the study (16 aerobic walkers [8.0-km/h group], 12 brisk walkers [6.4-km/h group], 18 strollers [4.8-km/h group], and 13 sedentary controls). Eighty-one percent were white, 17% black, and 2% Hispanic.

Intervention.—Intervention groups walked 4.8 km per day, 5 days per week at 8.0 km/h, 6.4 km/h, or 4.8 km/h on a tartan-surfaced, 1.6-km track for 24 weeks.

Main Outcome Measures.—Fitness (determined by maximal oxygen uptake) and cardiovascular risk factors (determined by resting blood pressure and serum lipids and lipoprotein levels).

Results.—As compared with controls, maximal oxygen uptake increased significantly (P < .0001) and in a dose-response manner (aerobic walkers=brisk walkers=strollers). In contrast, high-density lipoprotein cholesterol concentrations were not dose related and increased significantly (P < .05) and to the same extent among women who experienced considerable improvements in their physical fitness (8.0-km/h group, +0.08 mmol/L) and those who had only minimal improvements in fitness (4.8-km/h group, -0.08 mmol/L). High-density lipoprotein cholesterol also increased among the 6.4-km/h group, but did not attain statistical significance (+0.08 mmol/L; P = .06). Dietary patterns revealed no significant differences among groups.

Conclusion.—Thus, we conclude that vigorous exercise is not necessary for women to obtain meaningful improvements in their lipoprotein profile. Walking at intensities that do not have a major impact on cardiorespiratory fitness may nonetheless produce equally favorable changes in the cardiovascular risk profile. (JAMA. 1991;266:3296-3300)

FREQUENCY, intensity, and duration of exercise provide the framework for developing an exercise prescription. The interaction of these factors has been thoroughly examined only with regard to the proper dose of exercise required to increase cardiorespiratory fitness. Because cardiorespiratory fitness and cardiovascular health have been considered synonymous, it is not surprising that the guidelines published in 1978 by the American College of Sports Medicine describing the quality and quantity of exercise necessary to increase fitness are often extrapolated to the prescription of exercise to prevent cardiovascular disease. However, attainment of a fit state may not be necessary to modify specific cardiovascular disease risk factors favorably.

The latter hypothesis is supported by recent epidemiologic data that demonstrate that persons who participate in physical activities that are less intense and, therefore, unlikely to have a profound impact on cardiorespiratory fitness, do, nonetheless, derive cardiovascular health benefits. However, it is difficult to quantify the amount of physical activity required to provide an apparent protective effect against the development of cardiovascular disease from epidemiologic data alone. While ample support shows low-level activity may lead to a less atherogenic lipid profile among men, few studies have investigated this possibility among women. Therefore, to explore separately and independently the relationship between cardiorespiratory fitness and cardiovascular health among women, we designed a 24-week, dose-response, randomized clinical trial among sedentary, premenopausal women, in which walking exercise intensity varied across three treatment groups (strollers, brisk walkers, and aerobic walkers) while keeping the distance and frequency constant. Statistical analyses focused on whether changes in clinical cardiovascular risk factors paralleled changes in cardiorespiratory fitness across the three walking groups and the control group.

SUBJECTS AND METHODS

More than 300 women who responded to various media sources calling for volunteers to participate in this study were interviewed by telephone. One hundred two adult, premenopausal, sedentary women who were 20 to 40 years of age were randomly selected from this pool of volunteers if they (1) did not smoke; (2) consumed fewer than three alcoholic drinks per day; (3) did not currently receive dietary interventions; (4) did not have cardiopulmonary and/or musculoskeletal diseases; and (5) did not regularly exercise more than 1 day per week for the previous 6 months. Women with resting blood pressures below 160/90 mm Hg, total serum cholesterol levels below 6.59 mmol/L, and serum triglyceride levels below 2.25 mmol/L who were willing to accept random assignment to any of the four treatment groups were entered into the study, randomized to intervention groups, and followed up for 24 weeks. Written informed consent was obtained from each participant prior to entry into the study. All groups were advised not to change dietary, exercise (other than prescribed), or other lifestyle habits. Three-day dietary food records and physical activity questionnaires were administered at baseline and again at the end of the 24-week intervention study to evaluate whether participants adhered to these recommendations.

Control subjects (n = 21) remained sedentary for the duration of the study and were not contacted except to schedule follow-up testing. Subjects assigned to the aerobic walkers (n = 29), brisk walkers (n = 26), or strollers (n = 26)
trained under the supervision of an exercise physiologist on a tartan-surfaced, 1.6-km track, 5 days per week, for 24 weeks.

The frequency, intensity, and duration of each exercise session was standardized across all three walking intervention groups. Initially, each group walked a distance of 2.4 km, 5 days per week. Thereafter, walking distance was gradually increased each week until all subjects reached a maintenance distance of 4.8 km by the seventh week of training. Similarly, walking intensity was standardized to maintain a relative level each week among each of the three walking groups. Initial exercise intensities were equivalent to 70% of group assignment (e.g., 70% of 8.0 km/h [aerobic walkers], 79% of 6.4 km/h [brisk walkers], and 70% of 4.8 km/h [strollers]) and subsequently increased to 100% of prescribed intensity by the 14th week of training and maintained until completion of the study (week 24).

Resting blood pressure, serum lipid and lipoprotein levels, maximal oxygen uptake (VO₂ max), nude body weight, and percentage of body fat were measured at baseline and after 24 weeks of intervention. Resting blood pressure was measured in triplicate in the seated position on three separate days according to recommendations of the American Heart Association. The same trained observer made all measurements with a mercury sphygmomanometer. The mean of the three readings on the third day was used as baseline and follow-up values. Systolic and diastolic blood pressures were recorded at the first and fifth phases of Korotkoff's sounds.

Blood specimens were collected in the morning after abstaining from all food, beverages (except water), and vigorous activity for 12 to 14 hours. Serum was separated from venous blood within 30 minutes by centrifugation at 3400 rpm for 10 minutes and stored at -80°C until analyses. Total serum cholesterol and triglyceride levels were measured using an Olympus AU 6000 (Olympus Corp, Lake Success, NY) according to enzymatic procedures. High-density lipoprotein (HDL) cholesterol levels were measured after very-low-density lipoprotein and low-density lipoprotein (LDL) cholesterol levels were precipitated using a modified phosphotungstic acid (0.55 mmol/L) and magnesium chloride (25 mmol/L) reagent. Four hundred microliters of phosphotungstic acid and magnesium chloride solution (6.4 g of phosphotungstic acid, 20 mL of magnesium chloride in 3 L of deionized water) were added to 200 μL of serum. After the solution was mixed, the suspension was centrifuged at 3200 rpm at 4°C and analyzed. This method, which has been demonstrated to be more stable than concentrated phosphotungstic acid reagents, markedly reduces the interference from high-serum triglyceride concentrations. The LDL cholesterol was calculated with the following equation: total cholesterol - (HDL cholesterol + [triglycerides/5]).

Blinded control pools from the Centers for Disease Control, Atlanta, Ga, yielded values within 1.9% to 3.5% of Centers for Disease Control target values. The average day-to-day standard deviation (SD) and coefficient of variation (CV) of pooled control serum samples over the duration of the study were 2.5 SD, 1.5% CV for cholesterol; 1.1 SD, 2.8% CV for HDL cholesterol; and 5.0 SD, 3.5% CV for triglycerides. Calibrator samples (BMD Preciset, Boehringer Mannheim Diagnostics, Indianapolis, Ind) were run with each patient sample and checked for linearity. There was no evidence of drift within or between any of the analyses.

Baseline and posttraining serum lipid and lipoprotein levels were calculated using the mean of samples drawn on two or three different days (3 days if variation in duplicate samples exceeded 25% for HDL cholesterol). Baseline and posttraining lipid and lipoprotein samples were taken at the same phase of the menstrual cycle.

Body weight and height were measured anthropometrically with an Acme seated scale (model ASCMIN, Acme Scale Company, Oakland, Calif) and an anthropometer accurate to the nearest 0.01 kg and 0.1 cm, respectively. Body density was determined by hydrostatic weighing techniques, and percentage of body fat was calculated according to the Siri equation.

Maximal graded treadmill testing using automated cardiorespiratory monitoring techniques (MMC Horizon Systems Exercise Evaluation Cart, Sensormedics, Yorba Linda, Calif) was performed on all subjects using a modified Balke protocol. Gas and volume calibrations were performed before and after each test. Subjects exercised until volitional exhaustion, and the peak oxygen uptake and heart rate attained were taken as VO₂ max and maximal heart rate, respectively.

Statistical analyses were performed using the Statistical Analysis System. Power and sample-size determinations were calculated based on an effect size of 7 mL/kg per minute (VO₂ max), and an SD of 3 mL/kg per minute. An estimated sample size of 25 subjects per group at an α level of .05 yielded a β level of .99. Dropout during the study lowered the power to an approximate α level of .05 at a 1-β level of .94. Group randomization was performed by the principal investigator via a table of random numbers. Initially, 21 subjects were randomly assigned to each of the four groups. We continued random assignment to walking groups only until each of the three walking groups comprised 26 subjects. Three subjects were added to the aerobic walkers in anticipation of higher intensity-related dropout rates among the faster walkers. All baseline and posttest-dependent variables (except percentage of body fat) were measured by personnel who were blinded to individual participant group assignments.

Exact probability and analysis of variance were used to compare demographic data (age and race) and baseline dependent variables (VO₂ max, body composition, lipid levels, and resting blood pressure) for the four treatment groups. Changes in group scores in cardiorespiratory fitness, body composition, and cardiovascular health-related variables were analyzed for subjects who completed the study (aerobic walkers [n = 16], brisk walkers [n = 12], strollers [n = 18], and controls [n = 13]) by an analysis of variance model. Changes within the treatment groups were performed using paired t tests. Group contrasts were performed with a Scheffé test to guard against experimental error rate of unequal-sized samples. Statistical significance was established at P < .05. Data are presented as the mean ± SD.

RESULTS

Demographic and Compliance Data

Table 1 shows that cardiorespiratory and cardiovascular health-related variables were comparable at baseline across all four treatment groups. Eighty-one percent of the women who participated in the study were white, 17% black, and 2% Hispanic. Five women in the aerobic-walkers group, seven women in the brisk-walkers group, five women in the strollers group, and four women in the control group were taking birth control pills. Compliance to training (total number of sessions attended divided by total number of sessions possible) exceeded 85% for all three walking groups.

Dropout rates were as follows: four women in the aerobic-walkers group, one in the strollers group, and one in the control group became pregnant during the study and subsequently withdrew from further participation. In addition, nine aerobic walkers, 14 brisk walkers, seven strollers, and seven controls dropped out due to relocation, medical reasons unrelated to the study, or loss of interest. Finally, three aerobic walk-
ers (two receiving thyroid medication and one with endometriosis) and three women in the control group (two receiving thyroid medication and one receiving steroid hormone therapy after baseline testing) were excluded from lipid and/or blood pressure analyses.

Statistical analyses compared dropouts with subjects who completed the study. No differences were observed between dropouts and corresponding group members who completed the study regarding fitness (VO₂ max), body fat, or lipid level concentrations (P>0.05). Dropouts in the groups of brisk walkers, strollers, and controls did have significantly lower (P<0.001) blood pressure. Dropouts in the brisk-walkers group also had significantly higher (P<0.001) body weight compared with subjects who completed the study.

**Walking for Fitness**

Table 2 shows VO₂ max and body composition responses to 24 weeks of walking. A linear, dose-response gradient across all walking groups was observed for VO₂ max, with the aerobic walkers experiencing the greatest increase of VO₂ max (+5.0 mL/kg per minute), the brisk walkers experiencing a moderate rise of VO₂ max (+3.0 mL/kg per minute), and the strollers achieving a minimal increase in VO₂ max (+1.4 mL/kg per minute). Posttraining VO₂ max was not significantly changed among subjects in the control group. Analysis of group differences of VO₂ max yielded a significant overall F test (F = 16.23 [3, 51 df]; P<0.001). Group contrasts (Scheffe) revealed that VO₂ max differed significantly (P<0.05) between the control group and each of the walking groups and between the strollers and aerobic walkers.

Heart rates recorded during exercise training mirrored the dose-response VO₂ max gradient observed across all three walking groups. The aerobic walkers exercised at 86% (163 beats per minute) of their maximal heart rate, while the brisk walkers and strollers trained at 67% (126 beats per minute) and 56% (106 beats per minute) of their maximal heart rates.

**Walking for Health**

Table 3 shows changes in serum lipid and lipoprotein levels and blood pressure after 24 weeks of training. Changes in total cholesterol, LDL cholesterol, and triglycerides did not differ significantly between any of the treatment and control groups. However, mean serum concentrations of HDL cholesterol (Figure) increased significantly (P<0.05) among the aerobic walkers (+0.08 mmol/L) and strollers (+0.08 mmol/L) but not in the brisk walkers (+0.06 mmol/L; P=0.06) or controls (+0.02 mmol/L; P=0.77). The ratio of total cholesterol and HDL cholesterol was lowered significantly (P<0.01) within the strollers, but not in any of the other three treatment groups. Self-reported alcohol intake, calorie intake (assessed by 3-day dietary food records), dietary fat, carbohydrate, and protein intake, medication use, and smoking habits did not change significantly among any of the four study groups after 24 weeks.

The Figure illustrates the fitness and lipid responses to 24 weeks of training. Cardiorespiratory fitness increased in a dose-response manner with increasing levels of walking intensity while HDL cholesterol concentrations increased irrespective of the dose or walking intensity. Resting seated blood pressure did not change significantly within or between any of the walking groups after 24 weeks (Table 3). Similarly, no changes were observed among subjects in the control group.

**COMMENT**

We have attempted to quantify, in a linear, dose-response manner, the health and fitness implications of participating in a popular and frequently prescribed mode of exercise—walking. The VO₂ max data indicate that for those who are interested in obtaining meaningful improvements in cardiorespiratory fitness, walking at a brisk to aerobic pace provides the physiologic stimulus necessary to accomplish this goal. The recognized standard of cardiorespiratory fitness, VO₂ max, increased in a linear, dose-response manner from the strollers (+1.4 mL/kg per minute) to the brisk walkers (+3.0 mL/kg per minute).
Table 3. Changes (Mean ± SD) in Resting Blood Pressure, Lipids, and Lipoprotein Concentrations After 24 Weeks of Intervention

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Trained</th>
<th>Change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seated blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>106/74 ± 7/6</td>
<td>110/75 ± 7/7</td>
<td>+2/1</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>107/72 ± 6/9</td>
<td>108/73 ± 11/6</td>
<td>-3/0</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>109/74 ± 9/8</td>
<td>113/73 ± 10/6</td>
<td>+1/1</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>107/70 ± 6/7</td>
<td>107/70 ± 7/8</td>
<td>0/0</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>4.44 ± 0.83</td>
<td>4.55 ± 0.70</td>
<td>+2/6</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>4.62 ± 0.65</td>
<td>4.73 ± 0.64</td>
<td>-2/6</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>4.63 ± 0.67</td>
<td>4.75 ± 0.61</td>
<td>-4/6</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>4.62 ± 0.76</td>
<td>4.63 ± 0.79</td>
<td>+5/6</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>2.63 ± 0.57</td>
<td>2.76 ± 0.54</td>
<td>+4/1</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>2.98 ± 0.79</td>
<td>2.86 ± 0.77</td>
<td>-4/6</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>2.99 ± 0.69</td>
<td>2.74 ± 0.63</td>
<td>-8/7</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>2.81 ± 0.59</td>
<td>2.90 ± 0.63</td>
<td>+3/7</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>1.43 ± 0.33</td>
<td>1.45 ± 0.30</td>
<td>+1/6</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>1.34 ± 0.36</td>
<td>1.42 ± 0.41</td>
<td>+6/6</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>1.53 ± 0.36</td>
<td>1.59 ± 0.34</td>
<td>+4/6</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>1.39 ± 0.31</td>
<td>1.47 ± 0.31</td>
<td>+6/6</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>0.65 ± 0.45</td>
<td>0.70 ± 0.39</td>
<td>-18/5</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>1.10 ± 0.51</td>
<td>1.01 ± 0.57</td>
<td>-8/5</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>0.92 ± 0.32</td>
<td>0.92 ± 0.40</td>
<td>0/5</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>0.84 ± 0.38</td>
<td>1.05 ± 0.52</td>
<td>+12/5</td>
</tr>
<tr>
<td>Cholesterol and HDL ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group (n = 10)</td>
<td>3.21 ± 0.05</td>
<td>3.20 ± 0.06</td>
<td>+2/5</td>
</tr>
<tr>
<td>Strollers (n = 18)</td>
<td>3.79 ± 1.05</td>
<td>3.54 ± 0.87</td>
<td>-7/5</td>
</tr>
<tr>
<td>Brisk walkers (n = 12)</td>
<td>3.37 ± 0.78</td>
<td>3.13 ± 0.91</td>
<td>-7/5</td>
</tr>
<tr>
<td>Aerobic walkers (n = 13)</td>
<td>3.52 ± 0.96</td>
<td>3.45 ± 0.81</td>
<td>-2/5</td>
</tr>
</tbody>
</table>

*Strollers travel 4.8 km/h; brisk walkers, 6.4 km/h; and aerobic walkers, 8 km/h. LDL indicates low-density lipoprotein; HDL, high-density lipoprotein.
†Difference from baseline (P < .05).

Effect of walking intensity on changes from baseline in maximal oxygen uptake (ΔVO₂ max) and high-density lipoprotein cholesterol (Δ HDL cholesterol) after 24 weeks of exercise training.

In particular, women who walked 4.8 km, 6 days per week, at low intensity for a long duration (4.8 km/h) achieved only a minimal increase in fitness, but nonetheless experienced the same significant increase in HDL cholesterol concentration (+0.08 mmol/L; P < .05) as women who followed a similar program but walked vigorously enough to achieve an aerobically trained state (8.0 km/h; +0.08 mmol/L; P < .05). The direction and magnitude of change in HDL cholesterol among the brisk walkers (+0.06 mmol/L; P < .05) were similar to the other two walking groups, but did not reach statistical significance, possibly due to the smaller sample size. Clearly, the trend was toward an increase in HDL cholesterol among all three walking groups. Thus, our study is the first to show that among a group of healthy women the rise in HDL cholesterol, unlike the rise in fitness, is not related to intensity of exercise.

Our findings agree with cross-sectional, epidemiologic data reported by LaPorte et al.²⁴ and more recently by Hartung et al.²⁵ showing HDL cholesterol increases across a spectrum of exercise intensity—from exercise for quadruplegia to marathon training. Their data suggest that both low- and high-intensity activity increases HDL cholesterol concentrations by the same relative levels. However, in contrast to epidemiologic data, inconsistent results have been reported from the few prospective clinical studies that have investigated the lipid response to training among women. Rotkis et al.²⁶ observed a 0.13 mmol/L increase in the HDL cholesterol levels of 22 women who increased their training distance from 26.8 to 74.2 km per week. In contrast, other studies¹⁷-²⁰ failed to demonstrate a significant rise in HDL cholesterol concentrations after training. Inconsistent results in a rise in HDL cholesterol through exercise have also been reported.²¹-²⁶ Goldberg and Elliott²¹ and Wood et al.²² point out that most studies investigating the lipid response to training among women have been short term and may not have allowed enough time for significant alterations of lipids and lipoproteins to occur. Furthermore, Wood et al.²² have reported that HDL cholesterol is more likely to increase significantly if the length of training exceeds 12 weeks (six of eight longitudinal exercise studies reviewed that were 13 weeks or longer were associated with a significant increase in HDL cholesterol concentrations) and to remain unchanged if the length of training is 12 weeks or less (HDL cholesterol was unchanged in 12 of 14 longitudinal exercise studies that were 12 weeks or less). These observations may at least
partly explain the apparent discrepancy between our study and previous training studies of shorter durations.

Upon initial review of our data, the magnitude of change in HDL cholesterol appears modest. However, from a public health perspective, even small improvements in coronary risk factors, if established on a population basis, could lower cardiovascular-related mortality. Low to moderate physical activities have grown to compliance rates that are much lower than in HDL cholesterol. The mechanisms whereby walking confers its HDL cholesterol effects are unclear.

Clinical data indicate every 1% rise in HDL cholesterol lowers the risk of coronary disease by as much as 3%. Thus, an important public health impact may be obtained simply by persuading the majority of the population who are least active to become just a little more active. In fact, this hypothesis is supported by a recent epidemiologic report that suggests that women who regularly participate in physical activities, even at low levels, may experience lower all-cause mortality rates compared with a cohort of sedentary women. The randomized design of our study, collection of multiple blood samples, and control for the potential influence of the menstrual cycle by collecting blood samples at the same phase in the menstrual cycle at baseline and at the 24-week follow-up period, strengthen our findings. Similarly, the fact that changes in HDL cholesterol concentrations occurred without significant concomitant changes in dietary intake, medication use, or smoking habit provides additional evidence that the observed HDL cholesterol responses were associated with walking rather than other factors that are known to influence lipoprotein concentrations. Although the mechanisms whereby walking confers its HDL cholesterol effects are unclear, previous research has shown that fat loss, whether induced by dieting or exercise, produces favorable changes in plasma lipoprotein levels. We found an association in HDL cholesterol concentration to be associated with significant fat loss among the strollers, not among the aerobic walkers.

It remains to be determined whether the changes in HDL cholesterol concentrations that were observed in the present study are applicable to populations that are of a different age, sex, or ethnicity than those in the present study. Similarly, it is unknown whether individuals who are at a high risk of developing cardiovascular disease or those who have a cardiovascular disease will respond in a similar manner as observed among the healthy women who participated in our study. Finally, it is uncertain whether health or fitness outcomes noted in our study would be observed if other aspects of the exercise prescription (ie, frequency and/or duration of exercise) were manipulated while holding intensity constant. Further research is needed to clarify these issues.

**References**