Relationships of Heart Disease Risk Factors to Exercise Quantity and Intensity

Paul T. Williams, PhD

Objective: To assess the relationships of exercise amount and exercise intensity to coronary heart disease risk factors measured cross-sectionally in runners.

Methods: Physician-supplied medical data were compared with subjects' reported average running amount (kilometers run per week) and running intensity during their best recent 10-km race (kilometers per hour) in 7059 male and 1837 female recreational runners. Ten-kilometer race velocity (kilometers per hour) is known to be related to exercise intensity during training.

Results: Men and women who ran faster (ie, at greater intensity) had lower blood pressures; triglyceride levels; ratios of total cholesterol to high-density lipoprotein cholesterol; body mass indexes (calculated as the weight in kilograms divided by the square of the height in meters); and circumferences of the waist, hips, and chest when adjusted for weekly running distance, age, consumption of alcohol, and diet. Relative to the effect of running distance, running velocity had a 13.3 times greater calculated effect on systolic blood pressure, a 2.8 times greater calculated effect on diastolic blood pressure, and a 4.7 times greater calculated effect on waist circumference in men, and a 5.7 times greater calculated effect on systolic blood pressure in women when adjusted. In contrast, running distance had a more than 6-fold greater calculated effect on adjusted plasma high-density lipoprotein cholesterol levels than running velocity in both sexes.

Conclusions: Exercising more intensely could improve coronary heart disease risk factor level beyond that achieved by exercise amount alone; however, proof of causality remains to be determined from clinical trials. Exercise intensity and amount may target specific coronary heart disease risk factors.

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LONG-DISTANCE runners have higher concentrations of high-density lipoprotein (HDL) cholesterol and lower levels of low-density lipoprotein (LDL) cholesterol, triglycerides, blood pressure, and adiposity relative to sedentary individuals. Prior discussions of intensity have focused on comparing moderate (3-6 metabolic equivalents or 17-29 kJ/min) with vigorous physical activity (>6 metabolic equivalents or >29 kJ/min) while largely neglecting the health effects of vigorous activity performed at different intensities. Running is classified as a vigorous exercise performed at different intensities depending in part on speed.

Individual differences in 10-km and marathon (42 195 m) race performance times are principally caused by differences in the capacity to produce energy through oxidative pathways (maximum oxygen uptake) and the limitation in achieving this capacity due to the accumulation of metabolic by-products (blood lactate threshold). Accomplished runners can run 10-km race velocities requiring between 79% and 98% of maximum oxygen uptake and marathon race velocities requiring between 68% and 88% of maximum oxygen uptake. Nearly 30 years ago, running performance (velocity) was proposed as a field measurement of maximum oxygen uptake in epidemiological studies, yet its relation to coronary heart disease (CHD) risk factors has been scarcely reported and never for large samples.

Much use has been made of the concordance between physical fitness and amount of physical activity. Treadmill fitness parameters (maximum oxygen uptake or work capacity) have been used to validate differences in exercise amount in clinical trials. Validate physical activity questionnaires, purportedly provide a less-subjective measure of physical activity than self-reported amounts, and formulate government recommendations for exercise amount. Although related, physical fitness and physical activity amounts are different variables. Greater fitness often refers to a greater maximum oxygen uptake, which requires a greater efficiency of the oxygen transport system, whereas a higher weekly amount of physic-
SUBJECTS AND METHODS

The design and subject characteristics of the National Runners’ Health Study are described elsewhere.1,2,3 The cohort received a 2-page questionnaire that was distributed nationally at races and to subscribers of the nation’s largest running magazine (Runners’ World). This questionnaire solicited information on demographics (sex, age, race, and years of education); running history (age when the participant began running at least 19 km per week, average weekly running distance, and best marathon and 10-km race times during the prior 5 years); weight history (highest and current weight, weight when the participant started running, lowest weight as a runner, and circumferences of the chest, waist, and hips); diet (vegetarianism and the current weekly intakes of alcohol, red meat, fish, fruit, vitamin C, vitamin E, and aspirin); current and past cigarette use; history of heart attacks and cancer; and medications taken for disorders of blood pressure, thyroid gland, cholesterol, or diabetes. Average number of kilometers run per week was computed by averaging the reported yearly distances of the preceding 5 years. Body mass index (BMI) was calculated as the weight in kilograms divided by the height in meters squared.

The questionnaire also requested permission to obtain measures of participants’ height, weight, cholesterol, triglycerides, blood pressure, and resting heart rate from the runners’ physicians. On the rare occasion that data from more than 1 clinic visit were provided by the physician, we used the values from the most recent clinical visit in which HDL cholesterol was measured. High-density lipoprotein cholesterol values were obtained from the medical records of 7059 male and 1837 female nonvegetarian, nonsmoking runners who were without a history of heart disease or cancer and who were not using medications that might affect lipoprotein levels. The 1002 males taking medications were added to the 7059 males not taking medications to calculate the percentage of men with hypertension. Hypertension was defined as a systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher, or use of medication for blood pressure control (untreated hypertension may be overestimated because it is based on readings from a single clinic visit).

Average number of kilometers run per week was computed by averaging the reported yearly distances during the preceding 5 years. The test-retest correlations for self-reported running distance per week (r=0.89; unpublished data from 110 runners who completed duplicate questionnaires several months apart, 1993) compare favorably with those reported for the Minnesota leisure time physical activity questionnaire4 and others.5,6 There is also a significant inverse relationship between reported running distance and resting heart rate,5,6 a measure commonly used to validate physical activity assessment, since bradycardia is a cardiac manifestation of aerobic conditioning. Consumption of alcohol, red meat, fruit, and fish was validated by correlating reported usual weekly intakes from the questionnaires with recorded actual intakes from 4-day food records. The 2 methods corresponded most strongly for alcohol intake (r=0.65) and moderately for red meat (r=0.46) and fruit (r=0.38). The weak correlation for consumption of fish presumably relates in part to infrequent intake (r=0.19).

Two approaches were used to validate questions on anthropometric measurements from 110 men: test-retest correlations from duplicate questionnaires and correlations of clinical measurements of height, weight, and circumference measurements with their self-reported values. Self-reported height and weight showed strong agreement with the duplicate questionnaires (r=0.98 and r=0.97, respectively) and with the clinic measurements of these variables (r=0.96 for both). There were reasonable but somewhat weaker test-retest correlations for self-reported waist circumference (r=0.84), hip circumference (r=0.79), and chest circumference (r=0.93). Self-reported body circumferences also correlated reasonably with the clinic circumference measurements of the waist (r=0.68), hips (r=0.63), and chest (r=0.77).

We estimated that 15% to 19% of the subjects who received our questionnaire responded (uncertainty due to an unknown number of race participants who received the questionnaire and undeliverable addresses). Physician-supplied HDL concentrations were obtained for 26% of the respondents. The remainder either did not give permission to release medical data (27%), had no HDL cholesterol measurements in their medical record (22%), or their physicians did not respond (23%).

Multiple regression analyses were used to test for linear relationships between distances run and risk factor levels and to assess the significance of these relationships when adjusted for other variables. Linear trends for proportions were tested using the estimated trends and SEs for a linear contrast among categorical variables.27 Logistic regression analysis was used for modeling discrete dependent variables.

RESULTS

The best 10-km race times during the past 5 years were obtained for 5915 (83.8%) of the 7059 male runners and 1449 (78.9%) of the 1837 female runners. Compared with those lacking these data, men and women who provided their best race times ran significantly (P<.05) further each week (mean difference±SE, 11.7±0.6 and 11.6±1.0 km for men and women, respectively) and correspondingly had significantly higher HDL cholesterol concentrations (mean difference±SE, 0.07±0.01 mmol/L[2.6±0.4 mg/dL] and 0.06±0.02

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mmol/L [2.2±0.9 mg/dL, respectively] and lower pulse rates (mean difference [±SE], −2.4±0.4 and −3.1±0.8/min, respectively), diastolic blood pressures (mean difference [±SE], −0.6±0.3 and −1.3±0.6 mm Hg, respectively), and plasma triglyceride concentrations (mean difference [±SE], −0.10±0.03 mmol/L [−9.0±2.9 mg/dL] and −0.16±0.06 mmol/L [−14.0±5.1 mg/dL], respectively). There were no significant differences in education level; menstrual status; hormone use; or intake of alcohol, red meat, fish, fruit, aspirin, or vitamins between those having and those lacking these data. Men reporting race times were a mean (±SE) of 1.4±0.3 years younger than those who did not (P<.001), but among women age was not related to the presence of these data (P=.25). The best 10-km race times were converted to kilometers per hour in all analyses. The characteristics of the male and female runners who provided 10-km race times are displayed in Table 1.

### Table 1. Characteristics of Male and Female Runners Who Provided Their Best 10-km Performance Times*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>45.66±9.47</td>
<td>40.56±9.19</td>
</tr>
<tr>
<td>Education, y</td>
<td>16.72±2.41</td>
<td>15.97±2.39</td>
</tr>
<tr>
<td>10-km race velocity, km/h</td>
<td>14.01±1.81</td>
<td>12.47±1.75</td>
</tr>
<tr>
<td>Distance run, km/wk</td>
<td>39.94±19.94</td>
<td>38.36±17.95</td>
</tr>
<tr>
<td>Resting heart rate, beats/min</td>
<td>61.88±10.55</td>
<td>64.66±10.15</td>
</tr>
<tr>
<td>Body mass index, kg/m²†</td>
<td>23.67±2.40</td>
<td>21.15±2.34</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>84.67±5.87</td>
<td>68.18±6.66</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td>94.97±6.93</td>
<td>91.54±6.24</td>
</tr>
<tr>
<td>Chest circumference, cm</td>
<td>101.40±6.89</td>
<td>87.72±5.28</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>121.58±13.27</td>
<td>112.35±12.67</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>76.95±8.74</td>
<td>71.35±9.09</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L (mg/dL)</td>
<td>1.35±0.35 (52.19±13.57)</td>
<td>1.66±0.40 (64.94±15.51)</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L (mg/dL)</td>
<td>3.22±0.81 (124.38±32.26)</td>
<td>2.78±0.76 (107.53±29.21)</td>
</tr>
<tr>
<td>Triglycerides, mmol/L (mg/dL)</td>
<td>1.14±0.70 (101.33±61.93)</td>
<td>0.89±0.44 (78.57±38.85)</td>
</tr>
<tr>
<td>Ratio of total-HDL cholesterol</td>
<td>3.99±1.23</td>
<td>3.04±0.80</td>
</tr>
</tbody>
</table>

*All data presented as mean (±SD). BP indicates blood pressure; HDL, high-density lipoprotein; and LDL, low-density lipoprotein.
†Body mass index is calculated as the weight in kilograms divided by the square of the height in meters.

### Figure 1. Average race speed during best 10-km and marathon races for the preceding 5 years in men. Race times were reported for 10-km races by 5915 men and for marathons by 4014 men.

### Figure 2. Average race speed during best 10-km and marathon races during a 5-year period in women. Race times were reported for 10-km races by 1449 women and for marathons by 613 women.

**AGE AND RUNNING DISTANCE**

In both men and women, age and weekly running distance accounted for 40% of the variance in best running velocity during a 10-km race. The effects on velocity are illustrated in Figure 1 and Figure 2. The age-related decline in running velocity was significant at all training distances and became more pronounced (P<.01) at higher weekly distances. In men, the annual decline in velocity averaged (regression coefficient [±SE]) −0.055±0.005 km/h for distances of less than 24 km/wk, −0.067±0.003 km/h for distances between
and older (for all, 60 years (P < .001), 0.06±0.01 (P < .001), 0.04±0.01 (P < .001), and 0.01±0.01 km/h per kilometer run per week (P = .22).

<table>
<thead>
<tr>
<th>Distance</th>
<th>Age, y</th>
<th>Male</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>&lt;40</td>
<td>40-50</td>
</tr>
<tr>
<td>0-23.9 km/wk</td>
<td>0.67±0.09‡</td>
<td>−0.44±0.08‡</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>352</td>
<td>481</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.68±0.09‡</td>
<td>−0.46±0.08‡</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.64±0.08‡</td>
<td>−0.48±0.08‡</td>
</tr>
<tr>
<td>24-47.9 km/wk</td>
<td>0.65±0.05‡</td>
<td>−0.53±0.04‡</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>800</td>
<td>1232</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.65±0.05‡</td>
<td>−0.55±0.04‡</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.60±0.05‡</td>
<td>−0.56±0.04‡</td>
</tr>
<tr>
<td>48-71.9 km/wk</td>
<td>0.35±0.07‡</td>
<td>−0.73±0.06‡</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>358</td>
<td>572</td>
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<tr>
<td>Adjusted*</td>
<td>0.30±0.07§</td>
<td>−0.73±0.06‡</td>
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<tr>
<td>No. of subjects</td>
<td>0.28±0.07</td>
<td>−0.74±0.06‡</td>
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<tr>
<td>≥72 km/wk</td>
<td>0.70±0.12‡</td>
<td>−0.61±0.11‡</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>196</td>
<td>163</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.60±0.12‡</td>
<td>−0.32±0.12‡</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.57±0.12‡</td>
<td>−0.32±0.12‡</td>
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</table>

<table>
<thead>
<tr>
<th>Distance</th>
<th>Age, y</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;40</td>
<td>40-50</td>
</tr>
<tr>
<td>0-23.9 km/wk</td>
<td>0.66±0.15‡</td>
<td>−0.68±0.21§</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>166</td>
<td>100</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.60±0.15‡</td>
<td>−0.77±0.21§</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.55±0.15‡</td>
<td>−0.77±0.21§</td>
</tr>
<tr>
<td>24-47.9 km/wk</td>
<td>0.42±0.07‡</td>
<td>−0.40±0.10‡</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>379</td>
<td>256</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.40±0.07‡</td>
<td>−0.40±0.11‡</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.39±0.07§</td>
<td>−0.40±0.11‡</td>
</tr>
<tr>
<td>48-71.9 km/wk</td>
<td>0.47±0.08‡</td>
<td>−0.36±0.12‡</td>
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<tr>
<td>Unadjusted</td>
<td>203</td>
<td>127</td>
</tr>
<tr>
<td>Adjusted*</td>
<td>0.35±0.08‡</td>
<td>−0.30±0.12‡</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>0.34±0.08‡</td>
<td>−0.30±0.12‡</td>
</tr>
</tbody>
</table>

*Adjusted for age and weekly running distance within each cell.
†The women’s 50-60 y and ≥60 y age groups are combined for women to increase the sample size within the smallest cells.
‡P < .001.
§P < .01
‖P < .05

24 and 48 km/wk; −0.075±0.004 km/h for distances between 48 and 72 km/wk; and −0.099±0.008 km/h for distances exceeding 72 km/wk (for all, P < .001). The corresponding declines in women were −0.05±0.01; −0.06±0.01; −0.08±0.01; and −0.11±0.02 km/h per year, respectively (for all, P < .001). The figures also show that running velocity increased with weekly running distance regardless of age. Per kilometer run per week, running velocity increased (regression coefficient [±SE] 0.049±0.002 km/h for men younger than 40 years (P < .001), 0.049±0.001 km/h for men between 40 and 50 years (P < .001), 0.042±0.002 km/h for men between 50 and 60 years (P < .001); and 0.039±0.003 km/h for men 60 years and older (for all, P < .001). The corresponding increases for women were 0.05±0.01 (P < .001), 0.06±0.00 (P < .001), 0.04±0.01 (P < .001), and 0.01±0.01 km/h per kilometer run per week (P = .22).

**BODY MASS INDEX**

Body mass index accounted for an additional 6% of the variance in running velocity in men and 4% in women. Table 2 shows that lower BMIs (the dependent variable) were associated with significantly faster running velocities in both men and women, independent of age and weekly running distance. The relationship between running velocity and BMI may be due to low body weight facilitating faster running velocity, as opposed to faster velocity promoting weight loss. Specifically, change in BMI since the onset of running was related principally to average kilometers run per week (regression coefficient [±SE] −0.032±0.002 kg/m² reduction in BMI per kilometer, P < .001 for men; −0.019±0.003 kg/m² reduction in BMI per kilometer, P < .001 for women) but not to race velocity (P = .10 and P = .46 for males and females, respectively) during the best 10-km race when adjusted for age, years spent running, years of education, menstrual status, hormone use, and dietary variables.

**INDEPENDENT ASSOCIATION OF 10-km RUNNING VELOCITY WITH CHD RISK FACTORS**

Table 3 and Table 4 present the regression coefficients for the independent effects of running velocity and weekly running distance as predictors of CHD risk factors (ie, since both variables are included simultaneously in the regression model, the coefficient for running velocity is adjusted for weekly distance). The analyses suggest that running at faster
speeds, independent of weekly running distance, was associated with significantly smaller circumferences of the waist, hips, and chest, significantly lower systolic and diastolic blood pressure, and significantly lower plasma concentrations of triglycerides and ratios of total cholesterol to HDL cholesterol in both men and women. Faster running speeds were also associated with significantly higher HDL cholesterol and significantly lower LDL cholesterol concentrations in men when adjusted for weekly running distance.

Adjustment for BMI eliminates the significance of the effects of running velocity on the ratio of total-HDL cholesterol in both men and women, plasma HDL and LDL cholesterol concentrations in men, and waist and chest circumferences, diastolic blood pressure, and plasma triglyceride concentrations in women. The relationships of running velocity with men's body circumferences, blood pressures, and triglyceride concentrations remain significant when adjusted, as does the relationship between velocity and women's systolic blood pressure.

**INDEPENDENT ASSOCIATIONS OF WEEKLY RUNNING DISTANCE ON CHD RISK FACTORS**

Men and women who ran longer weekly distances also had significantly higher plasma HDL cholesterol concentrations and narrower waists and hips, regardless of how fast they ran during their best 10-km race. In addition, running distance was associated with lower plasma concentrations of triglycerides and the ratio of total to HDL cholesterol in men, and narrower chest circumferences in women when adjusted for 10-km velocity. Adjustment for BMI produced only modest reductions in the coefficient relating running distance to CHD risk factors.

**RELATIVE CALCULATED INFLUENCES OF 10-km RUNNING VELOCITY AND WEEKLY DISTANCE**

Standardized regression coefficients provide one method for comparing the relative influences of variables measured on different scales, e.g., running velocity measured in kilometers per hour and running amount measured in kilometers per week. The standardized coefficient is the predicted change in the dependent variable (ie, CHD risk factor) corresponding to a difference of 1 SD of the independent variable. Tables 3 and 4 present the standardized coefficients to compare the relative apparent effects of running distance and running velocity on CHD risk factors. For example, men's waist circumference is calculated to decrease by 0.4 cm per 1 SD increase in running velocity when running amount is held constant, and to decrease by 0.05 cm per 1 SD increase in weekly distance when velocity is constant. Tables 3 and 4 also present the ratio (in absolute value) of the standard-
For both sexes, running velocity had a greater calculated effect than running distance; ratios less than 1 show the opposite. All coefficients are adjusted for age; years of education; menstrual status; hormone use; and intakes of alcohol, red meat, fish, fruit, and alcohol. Adjustment for BMI was included as indicated.

For both sexes, running velocity had a greater calculated effect than running distance; ratios less than 1 show the opposite. All coefficients are adjusted for age; years of education; menstrual status; hormone use; and intakes of alcohol, red meat, fish, fruit, and alcohol. Adjustment for BMI was included as indicated.

**Figure 3** displays the relative risk of hypertension in men by age-specific quintiles of weekly running distance and 10-km race velocity. Relative to the lowest quintile, the relative risk reduction is greater when the men were partitioned by running velocity than by weekly running distance. Multiple logistic regression analyses (not displayed) with hypertension as the dependent variable and age, BMI, 10-km race velocity, and weekly running distance as the independent variables showed 10-km race velocity to be significant (*P* < .001) and weekly running distance as nonsignificant (*P* = .48) when adjusted for the other variables in the model.

**Marathon Participation**

Forty-nine percent of the 5915 men and 40% of the 1449 women participated in at least 1 marathon during the previous 5 years. Male marathon runners had higher concentrations of HDL cholesterol (mean difference ± SE, 0.024 ± 0.009 mmol/L [0.92 ± 0.36 mg/dL] higher; *P* < .01) and female marathon runners had lower ratios of total cholesterol to HDL cholesterol (−0.10 ± 0.04; *P* < .03) than nonmarathon runners when adjusted for 10-km race velocity, running distance, years of education, age, hormone use, and diet. Both male and female marathon runners had heavier BMIs than nonmarathon runners.
Relative risk of hypertension in men by age-specific quintiles of weekly running distance and 10-km race velocity. Hypertension was defined as systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher, or use of medication for blood pressure control (untreated hypertension may be overestimated because it is based on readings from a single clinic visit). One asterisk indicates $P = .17$; 2 asterisks, $P = .003$; 3 asterisks, $P = .001$; 4 asterisks, $P = .001$; and 5 asterisks, $P = .001$. Adjusted for BMI did not eliminate the significantly higher levels of HDL cholesterol (adjusted difference [±SE], 0.026±0.009 mmol/L [1.01±0.36 mg/dL]; $P = .005$) and the significantly lower ratio of total-to-HDL cholesterol (−0.13±0.05; $P < .006$) of the male and female marathon runners, respectively.

MARATHON RACE VELOCITY

Velocities at marathons, although slower than at 10-km races, exhibited the same relationships to age and weekly training distance (Figures 1 and 2). In men, but not in women, the addition of marathon race velocity to the models of Tables 3 and 4 was significant for BMI (regression coefficient [±SE], −0.21±0.04 kg/m² calculated reduction per kilometers per hour; $P < .001$); waist and chest circumferences (regression coefficient [±SE], −0.67±0.09 and −0.72±0.13 cm calculated reductions per kilometer per hour, respectively; $P < .001$); concentrations of HDL cholesterol (regression coefficient [±SE], 0.025±0.006 mmol/L [0.95±0.22 mg/dL] calculated increase per kilometer per hour; $P < .001$); and ratio of total-HDL cholesterol (regression coefficient [±SE], −0.06±0.02 calculated reduction per kilometer per hour; $P < .004$). Men's and women's hip circumferences, blood pressures, and levels of LDL cholesterol and triglycerides were unrelated to best marathon velocity during the previous 5 years when adjusted for the aforementioned covariates. Adjusting for BMI did not eliminate the significant relationship between the men's marathon race velocities and their waist circumferences (regression coefficient [±SE], −0.33±0.07 cm reduction per kilometer per hour; $P < .001$); levels of HDL cholesterol (regression coefficient [±SE], 0.021±0.006 mmol/L [0.82±0.22 mg/dL] increase per kilometer per hour; $P < .001$); and ratio of total-HDL cholesterol (regression coefficient [±SE], −0.04±0.02 reduction per kilometer per hour; $P = .04$).

COMMENT

The relationship of running performance with maximum oxygen uptake and blood lactate threshold has been demonstrated in both sexes and in trained and untrained individuals. Ten-kilometer race performance times are correlated strongly with maximum oxygen uptake and blood lactate threshold. Marathon performance time is also related to maximum oxygen uptake. During a marathon, faster runners work at an intensity requiring 75% of their maximum oxygen uptake, whereas the intensity of slower runners is at only 60%. Other less significant factors affecting performance include oxygen consumption at submaximal speeds (ie, running economy), muscle fiber composition, and anaerobic capacity.

Figures 1 and 2 show that those who ran for longer distances had greater age-related declines in running velocity than those who ran shorter distances in both sexes. The decline in running speed with age is probably caused by decreases in maximum oxygen uptake concomitant to decreases in maximum heart rate and stroke volume. Highly trained younger and older runners use approximately the same percentage of their maximum oxygen uptake during 10-km races. The running velocity at which blood lactate begins to accumulate declines with age but purportedly remains at approximately the same percentage of maximum oxygen uptake as in comparably trained younger runners. A portion of the decline in maximum oxygen uptake with age may be due to loss of muscle mass.

Tables 2 through 4 show that when adjusted for weekly distance and age, the velocity of male and female runners correlates significantly with lower BMI and circumferences of the waist, hips, and chest. Others have noted that faster 10-km velocities are associated with lower BMI, sum of skinfold thicknesses, and leaner body composition. Champion athletes have lower body weights and less fat than even well-trained athletes of similar age. Marti and Howald report that running pace predicted changes in truncal fat for a 15-year period in former elite male athletes. In our study, race performance (velocity) was, however, unrelated to weight loss since onset of running, suggesting that lower BMI is a cause rather than the consequence of faster race times. This interpretation is consistent with reports that body weight does not change when training intensity is increased, provided that weekly mileage remains the same, and total energy expended during running is primarily related to distance rather than speed.

Faster runners had lower blood pressure even when adjusted for BMI (Tables 3 and 4). Data pooled from 22 controlled training studies show an inverse association between changes in diastolic blood pressure and changes in exercise capacity. Blair et al report a threshold for the hypotensive effects of fitness, the effect being limited to higher fitness levels. Our cross-sectional data do not prove that increasing exercise intensity will reduce blood pressure. For example, inherited variations in maximum oxygen uptake could contribute in part to the ability to run quickly.

In contrast to blood pressure, plasma HDL cholesterol concentrations were more strongly related to weekly run-
High lipoprotein lipase activity may accelerate very muscle lipoprotein lipase may be stimulated by the depletion of intramuscular substrates, and at lower exercise levels than those required to increase concentrations of HDL cholesterol.

We found that runners who had run marathons had higher HDL cholesterol concentrations than those who had not. The ability to finish a marathon requires prolonged running that may have its own physiological effects, including increased use of blood-borne substrates for muscle metabolism relative to that of intramuscular substrates, increased use of lipids for muscle metabolism compared with carbohydrates, and acute increases in muscle lipoprotein lipase and the fractional removal rate of plasma triglycerides. Increases in muscle lipoprotein lipase may be stimulated by the depletion of intramuscular stores following prolonged running. High lipoprotein lipase activity may accelerate very low-density lipoprotein and chylomicron catabolism, which may cause HDL levels to increase because of the more rapid transfer of free cholesterol and phospholipids to HDL. It may also limit the availability of triglyceride-rich lipoprotein particles for cholesterol-ester triglyceride exchange with HDL, causing HDL cholesteryl ester to accumulate in plasma.

CAVEATS AND LIMITATIONS

Although these cross-sectional associations do not prove that running faster and greater distances causes these improvements in CHD risk factors, other well-designed, randomized, controlled intervention studies prove causality between running and changes in CHD risk factors. Self-selection may also contribute partially to the association between running level and HDL cholesterol. Men with higher levels of HDL cholesterol will tend to run more when undertaking an exercise program has been demonstrated in 2 studies. Higher HDL cholesterol levels at baseline may be a marker for men genetically endowed with muscle fiber types that make running easier, thereby facilitating weight loss.

Compared with runners identified from a random geographically defined sample, our sample tended to be leaner (−0.8 kg/m²) and have higher concentrations of HDL cholesterol (0.10 mmol/L [3.86 mg/dL] higher, data not shown), presumably because they tended to be more avid runners and somewhat healthier. An unbiased estimate of the dose-response relationship between CHD risk factors and running distance does not require that the runners be selected at random with respect to running mileage. However, volunteers are expected to differ from nonvolunteers and therefore caution is warranted in extrapolating our estimates to all runners. Only 56,000 of the approximately 290,000 subjects who were mailed questionnaires responded, and of these 9898 are used in the analysis.

CONCLUSIONS

Our data suggest that the benefits of physical activity depend on both intensity and amount. Reductions in CHD risk associated with being more physically fit and more active may be mediated through different risk factors. Whereas blood pressure was more strongly related to race performance than running amount, the opposite was true for HDL cholesterol. The variables that correlate strongly with race performance in our cross-sectional surveys (eg, diastolic blood pressure [Tables 3 and 4]) tend to be those that change in association with maximum oxygen uptake in longitudinal trials. Conversely, the variable that correlated most weakly with performance cross-sectionally (HDL cholesterol [Tables 3 and 4]) tends to be unrelated to changes in maximum oxygen uptake when studied longitudinally. Both lower blood pressure and higher levels of HDL cholesterol are expected to contribute independently to lower CHD risks. Follow-up studies of this cohort will test whether intensity and duration of exercise both contribute independently to reduced risk of CHD, and whether the additional benefits of running faster outweigh the risks of injury.

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