

Twenty-year follow-up of aerobic power and body composition of older track athletes

MICHAEL L. POLLOCK, LARRY J. MENGELKOCH, JAMES E. GRAVES, DAVID T. LOWENTHAL, MARIAN C. LIMACHER, CARL FOSTER, AND JACK H. WILMORE
Departments of Medicine and Exercise and Sport Sciences, Center for Exercise Science, University of Florida, and Geriatric Research, Education and Clinical Center, Veterans Affairs Medical Center, Gainesville, Florida 32610

Pollock, Michael L., Larry J. Mengelkoch, James E. Graves, David T. Lowenthal, Marian C. Limacher, Carl Foster, and Jack H. Wilmore. Twenty-year follow-up of aerobic power and body composition of older track athletes. *J. Appl. Physiol.* 82(5): 1508–1516, 1997.—The purpose was to determine the aerobic power (maximal oxygen uptake) and body composition of older track athletes after a 20-yr follow-up (T3). At 20 yr, 21 subjects [mean ages: 50.5 ± 8.5 yr at initial evaluation (T1), 60.2 ± 8.8 yr at 10-yr follow-up (T2), and 70.4 ± 8.8 yr at 20-yr follow-up (T3)] were divided into three intensity groups: high (H; remained elite; *n* = 9); moderate (M; continued frequent moderate-to-rigorous endurance training; *n* = 10); and low (L; greatly reduced training; *n* = 2). All groups decreased in maximal oxygen uptake at each testing point (H, 8 and 15%; M, 13 and 14%; and L, 18 and 34% from T1 to T2 and T2 to T3, respectively). Maximal heart rate showed a linear decrease of ~5–7 beats·min⁻¹·decade⁻¹ and was independent of training status. Body weight remained stable for the H and M groups and percent fat increased ~2–2.5%/decade. Although fat-free weight decreased at each testing point, there was a trend for those who began weight-training exercise to better maintain it. Cross-sectional analysis at T3 showed that leg strength and bone mineral density were generally maintained from age 60 to 89 yr. Those who performed weight training had a greater arm region bone mineral density than those who did not. These longitudinal data show that the physiological capacities of older athletes are reduced despite continued vigorous endurance exercise over a 20-yr period (~8–15%/decade). Changes in body composition appeared to be less than those shown for the healthy sedentary population and were related to changes in training habits.

aging; maximal oxygen uptake; weight training; bone mineral density

AFTER MATURITY, maximal oxygen uptake ($\dot{V}O_{2\max}$), pulmonary ventilation ($\dot{V}E_{\max}$), and heart rate (HR_{\max}) decline with age (2, 5, 9, 11, 16, 17, 19, 23, 25, 28, 29, 33). Fat-free weight (FFW) and muscular strength also decrease (7, 14, 23, 27, 30), whereas body fat increases significantly with age (23, 26). In a review of multiple studies, Heath et al. (9) determined that in healthy men $\dot{V}O_{2\max}$ declined ~9%/decade after the age of 25 yr. These same authors and others (5, 12, 17, 23, 29) have shown that if persons remain physically active and body weight remains fairly stable, a decline in $\dot{V}O_{2\max}$ with age will be 5%/decade.

Although it has been suggested that the slopes of the regression in $\dot{V}O_{2\max}$ with age may be similar for active and sedentary persons, this interpretation may have been a consequence of using cross-sectional data rather than the results from longitudinal studies (5). Longitudinal

studies conducted on subjects both below and above 50 yr of age show that decreases in $\dot{V}O_{2\max}$ are greatly affected by the initial level of aerobic capacity and change in activity status (5, 23, 26, 28).

A reduction in HR_{\max} with age has been shown to be linear, with an ~10 beat·min⁻¹·decade⁻¹ decline (16, 26). These findings seem to be supported more by cross-sectional data than by longitudinal studies. Longitudinal data suggest a 5–7 beat/min reduction in HR_{\max} with age, particularly in groups below 55 yr of age (2, 11, 17, 23, 29, 33). Longitudinal studies on older participants are more variable and generally lacking (11, 33).

Changes in body composition with age are also greatly affected by activity status, with more active individuals being less fat and able to maintain FFW (27). Although these findings are consistent in the literature for short-term longitudinal (~6 mo to 1 yr) and cross-sectional studies (26), few data exist for longer term studies.

Therefore, the purpose of this investigation was to determine the aerobic power and body composition of older track athletes after a 20-yr follow-up. This information is particularly important because most of the longitudinal studies have been conducted on persons <60 yr of age.

METHODS

Subjects. Twenty-seven male track athletes were recruited to participate in the initial evaluation (T1) (25). To qualify for the study, the athletes had to have trained regularly for at least the preceding 2 yr and placed first, second, or third in regional, national, or international competition in running (800 m or less, *n* = 6; 1,500 m or longer, *n* = 17) or race-walking (5,000 m or longer, *n* = 2) events within the preceding year. Twenty-five subjects returned for testing at the 10-yr follow-up [T2; 9.8 ± 1.4 (SD) yr]. One subject who was not in the original group at T1 was tested after the manuscript was accepted for publication at T2 (*n* = 24) (23). Two subjects had died (homicide; cancer), and one subject was not available for testing. Twenty-one of the original subjects returned for testing at the 20-yr follow-up (T3; 20.0 ± 1.3 yr). The four remaining subjects were unavailable for testing at T3: two had orthopedic conditions that prevented regular exercise training (severe hip arthritis; low back pain), one had Alzheimer's disease, and one could not be located.

At T3, the subjects ranged from 60 to 92 yr of age (70.4 ± 8.8 yr; 20 white, 1 black; 15 runners, 6 walkers). Results from activity questionnaires and personal interviews of the subject's training intensity and level of competition at T3 were used to determine the assignment of subjects into high (H; *n* = 9), moderate (M; *n* = 10), and low-intensity groups (L; *n* = 2). The H group included subjects whose usual

training intensity was ~60–85% HR_{\max} reserve. In addition, they performed an interval session or aerobic threshold training session ($\geq 85\%$ HR_{\max} reserve) ≥ 1 times per week, and they continued participation in high-level competition. They also maintained their elite athletic status by placing first, second, or third in national or international age-group championships the previous year. The M group included subjects whose usual training intensity was ~60–80% HR_{\max} reserve. These subjects only occasionally competed. The L group changed their mode of activity from running to walking. Their usual training intensity was $\leq 70\%$ HR_{\max} reserve, and they did not compete. The two subjects in the L group reduced their activity levels between T2 and T3 secondary to physical limitations (total hip replacement; knee meniscectomy and prostate surgery).

No subject was using medications at T1 or T2. At T3, one subject (*group L*, age 75 yr) was taking medication for treatment of hypertension [a lisinopril-hydrochlorothiazide (25 mg) combination four times a day] and another (*group M*, age 61 yr) was using flecainide acetate (125 mg twice a day) to control for paroxysmal atrial fibrillation.

The present protocol (T3) was approved by the Institutional Review Board of the University of Florida College of Medicine (Gainesville). All subjects provided informed consent.

The subjects were instructed not to engage in vigorous physical training, to abstain from drinking alcohol for a minimum of 24 h before testing, and to report to the laboratory around 8:00 AM at least 12 h postprandial. The subjects underwent a cardiopulmonary examination by a physician before testing. After 15 min of quiet sitting, resting blood pressure was determined by auscultation and resting HR was counted for 30 s.

Body composition. Body composition was determined from height, weight, circumference, and skinfold fat measurements. Anthropometric procedures followed the recommendations described by Pollock and Wilmore (26). Skinfold fat measurements were obtained from seven sites (chest, axilla, triceps, subscapula, abdomen, suprailiac, and anterior thigh). Circumferences were measured at the chest, waist, gluteal (hip), thigh, biceps, and wrist. Chest expansion was determined by calculating the difference in circumference between a full expiration and a full inspiration. Height and circumference measurements were taken to the nearest 0.1 cm, total body weight to the nearest 100 g, and skinfold fat to the nearest 0.5 mm. Anthropometric variables were taken by the same investigator at T1, T2, and T3. The calculation of body density, percent fat, and FFW has been previously described (23).

At T3, skinfold percent fat was compared with percent fat data obtained from underwater weighing (UWW) and from dual-energy X-ray absorptiometry (DXA). The UWW procedure was previously described (23). Although only skinfold fat data were collected from all subjects at T1, UWW was determined at T2 and T3 and DXA at T3. The mean values were 14.8 vs. 13.2% fat for the skinfold and UWW techniques, respectively, at T2 (23). At T3, the mean values were 17.1, 18.4, and 16.8% fat for the skinfold, UWW, and DXA methods, respectively. Thus, in our opinion, the skinfold technique appears to be valid for use in this investigation.

Bone mineral density (BMD) was assessed noninvasively with DXA (DPX-L, Lunar Radiation, Madison, WI) only at T3. This information was used in a cross-sectional analysis to compare BMD to age, years, and type of training, including resistance (weight)-training activities. The sample for this analysis included two additional marathon runners (ages 92 and 94 yr) who had won medals in recent national marathon competition. Three scans were performed: anteroposterior total body, lateral spine (L_2 and L_3), and supine hip, providing

information on the right trochanter. The same radiology technician performed and analyzed all scans.

Pulmonary function. Pulmonary function measurements were determined by spirometry while the subjects were seated. The subjects performed at least three forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) maneuvers. Trials were repeated until two trials were in close agreement ($\leq 5\%$). Reported values for FEV_1 and FVC were selected with the "best test" method (single test that gives the largest sum of FEV_1 plus FVC) (1).

Aerobic capacity. The subjects performed maximal exercise on a motor-driven treadmill. At T1, all subjects performed maximal treadmill exercise using either a multistage running or walking protocol (depending on the subject's track specialty), which has been previously described (25). At T2, all subjects performed two maximal treadmill exercise tests over a 2-day period. Because of the subjects' increased age, it was felt that a diagnostic test using the standard Bruce protocol (26) was necessary to screen the subjects on *day 1* to rule out overt coronary heart disease (symptoms, arrhythmias, or ischemia). On *day 2*, the original running or walking protocol was utilized. The mean values between the two tests were similar (run protocol, 49.7 vs. Bruce, 48.2 $ml \cdot kg^{-1} \cdot min^{-1}$; $P \geq 0.05$), and the highest value attained for each subject was used to determine $\dot{V}O_{2\max}$ at T2. At T3, the Bruce protocol was utilized with the following exceptions: the modified Naughton protocol (2-min stages) (26) was used for two subjects who had orthopedic limitations.

At T1 and T3, $\dot{V}O_{2\max}$ was determined by the Douglas bag method. At T2, $\dot{V}O_{2\max}$ was determined by an automated system that has been previously described (23). Expiratory ventilation was measured with a Parkinson-Cowan dry-gas meter (model CD-4) at T1 and T2 and a 150-liter Tissot spirometer (Collins, Braintree, MA) at T3. Heart rate and electrocardiographic measurements were recorded continuously during exercise with multiple-lead electrocardiographic recordings and for 5–7 min of recovery. Blood pressure was measured as previously described (23). At T2 and T3, the rating of perceived exertion (RPE) was determined at the end of each minute and at peak exercise (4).

Strength testing (T3 only). Lumbar back strength was assessed by an isometric test of the lumbar extensor muscles at seven different angles (0, 12, 24, 36, 48, 60, and 72° of lumbar flexion) with the MedX (Ocala, FL) lumbar extension machine. One-repetition maximum chest press and leg press exercises were performed on Nautilus (Richmond, VA) chest press and leverage leg press machines. The subjects began the test by lifting a light weight. This was followed by incremental increases of 2.3–4.5 kg depending on the difficulty of the previous lift. A 1-min rest was allowed between trials. The subjects continued to increase the weight lifted until they reached the maximal amount of weight that could be lifted in one repetition. Generally, four to five trials were used to reach one repetition maximum (26).

Physical activity questionnaires and interview. T1, T2, and T3 included the same activity questionnaires that provided training information about the mode of exercise and quantity and quality of training used the year before testing. At T2 and T3, each subject had an extensive interview by the same investigator. The purpose of the interview was to verify the information provided on the questionnaires and to document on a year-to-year basis (T2, *years 0–10*; T3, *years 10–20*) the nature and extent of their training program and to determine their level of competitiveness. The subjects had to be participating in resistance training using both upper and lower extremity exercises a minimum of two times per week for a minimum of 2 yr before testing to be classified as a weight lifter.

Statistical analysis. Longitudinal training data for anthropometric and metabolic measurements and pulmonary function values were compared with a 3 (time) \times 3 (group) or 2 (time) \times 3 (group) repeated-measures analysis of variance. When means were significantly different, contrast analyses were performed to determine which individual treatment means were significantly different. Cross-sectional strength and BMD data compared by age groups at T3 were analyzed by one-way analysis of variance. When a significant *F* ratio was observed, Scheffé's post hoc analysis was used to determine which individual treatment means were significantly different. An independent *t*-test was used to compare differences among variables between subjects who included regular resistance training between T2 and T3. Simple and multiple regression techniques were used to analyze the relationships among variables. Statistical significance was accepted as $P \leq 0.05$.

RESULTS

Subjects. At T3, the H group averaged 70.4 ± 8.5 yr of age, the M group 69.8 ± 10.2 yr, and the L group 73.5 ± 2.1 yr ($P \geq 0.05$ among groups). The average years of follow-up were also not significant among groups ($P \geq 0.05$); H group, 19.3 ± 1.7 yr; M group, 20.4 ± 0.7 yr; and L group, 20.9 ± 0 yr. Although the miles trained per week were generally the same from T1 to T2 for the H and M groups, there was a significant reduction in mileage per week from T2 to T3. The total miles trained between the H and M groups at T3 were not significantly different. The major difference in training for

both the H and M groups was their significant reduction in pace from T2 to T3 (Table 1).

Maximal aerobic capacity and related variables. Table 1 shows data by groups for $\dot{V}O_{2\max}$ expressed in liters per minute and milliliters per kilogram of body weight or FFW per minute. The results showed that $\dot{V}O_{2\max}$ decreased significantly for all groups at each test period but was more pronounced from T2 to T3. The same was true whether the data were expressed in terms of liters per minute or milliliters per kilogram of body weight or FFW per minute except for the H group from T1 to T2 for $\dot{V}O_{2\max}$ expressed in milliliters per kilogram of FFW per minute. Figure 1 graphically displays the $\dot{V}O_{2\max}$ (in $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) for the H, M, and L groups compared with an estimated aging curve for athletes and sedentary persons (19). Figure 1 clearly shows that the L group decreased in $\dot{V}O_{2\max}$ at a greater rate from T2 to T3 than both the H and M groups.

In contrast to the $\dot{V}O_{2\max}$ data, O_2 pulse and $\dot{V}E_{\max}$ were maintained from T1 to T2 and then decreased significantly from T2 to T3 for all groups. HR_{\max} showed a consistent reduction ($P \leq 0.05$) from T1 to T2 and from T2 to T3 for both the H and M groups but was maintained by the L group. Resting HR increased but remained low and generally constant for all groups over the 20-yr follow-up (Table 1).

Two markers of relative effort on the treadmill test, the respiratory exchange ratio and RPE, are shown in Table 1. The respiratory exchange ratio from T1 to T2

Table 1. Training data and metabolic measurements

| | All Subjects | | | High-Intensity Group | | | Moderate-Intensity Group | | | Low-Intensity Group | | |
|--|----------------------|----------------------|-----------------------|----------------------|-----------------------|-----------------------|--------------------------|-----------------------|-----------------------|----------------------|-----------------------|-----------------------|
| | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 |
| <i>n</i> | 21 | 21 | 21 | 9 | 9 | 9 | 10 | 10 | 10 | 2 | 2 | 2 |
| Age, yr | 50.5 ± 8.5 | 60.2* ± 8.8 | 70.4† ± 8.8 | 51.2 ± 7.6 | 60.4* ± 8.5 | 70.4† ± 8.5 | 49.5 ± 10.3 | 59.5* ± 10.3 | 69.8† ± 10.2 | 52.5 ± 2.1 | 62.5* ± 2.1 | 73.5† ± 2.1 |
| Follow-up, yr | | 9.8 ± 1.4 | 20.0† ± 1.3 | | 9.2 ± 1.9 | 19.3† ± 1.7 | | 10.1 ± 0.7 | 20.4† ± 0.7 | | 10.6 ± 0.1 | 20.9† ± 0 |
| Mileage, miles/wk | 32.8 ± 23.8 | 27.1 ± 15.2 | 18.2* ± 11.7 | 38.3 ± 24.3 | 34.4 ± 18.9 | 21.9‡ ± 15.2 | 30.6 ± 25.5 | 23.5 ± 8.5 | 17.0* ± 7.6 | 18.8 ± 5.3 | 12.0 ± 4.2 | 7.5 ± 0.7 |
| Pace, min/miles | 7.9 ± 1.3 | 8.6 ± 1.6 | 10.4§ ± 3.2 | 7.9 ± 1.4 | 8.3 ± 1.5 | 9.5‡ ± 1.3 | 7.9 ± 1.4 | 8.7 ± 1.8 | 10.0‡ ± 3.4 | 7.4 ± 0.1 | 8.8 ± 0.4 | 16.3‡ ± 1.8 |
| HR_{rest} , beats/min | 49 ± 8 | 48 ± 8 | 52† ± 8 | 46 ± 7 | 47 ± 9 | 50 ± 10 | 52 ± 8 | 49 ± 7 | 54 ± 5 | 50 ± 3 | 48 ± 11 | 56 ± 8 |
| HR_{\max} , beats/min | 174 ± 10 | 167* ± 10 | 161* ± 9 | 173 ± 13 | 165* ± 10 | 160‡ ± 9 | 177 ± 8 | 171* ± 9 | 162‡ ± 9 | 166 ± 6 | 159 ± 8 | 163 ± 3 |
| $\dot{V}O_{2\max}$ l/min | 3.792 ± 0.615 | 3.465 ± 0.545 | 2.863§ ± 0.715 | 3.643 ± 0.470 | 3.390* ± 0.440 | 2.849‡ ± 0.526 | 3.930 ± 0.750 | 3.593* ± 0.663 | 3.009‡ ± 0.875 | 3.770 ± 0.580 | 3.165* ± 0.199 | 2.197‡ ± 0.008 |
| $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ | 54.3 ± 8.0 | 50.1* ± 7.0 | 40.5‡ ± 8.9 | 55.4 ± 8.7 | 52.1* ± 6.8 | 43.2‡ ± 6.3 | 54.2 ± 7.7 | 50.0* ± 6.9 | 40.8‡ ± 9.5 | 50.0 ± 8.7 | 42.3* ± 1.3 | 27.0‡ ± 0.1 |
| $\text{ml} \cdot \text{kg FFW}^{-1} \cdot \text{min}^{-1}$ | 61.7 ± 7.5 | 58.7 ± 6.8 | 48.7§ ± 9.8 | 61.6 ± 9.0 | 59.7 ± 7.3 | 51.1‡ ± 8.5 | 62.3 ± 6.4 | 59.2* ± 6.4 | 49.3‡ ± 10.0 | 59.0 ± 9.6 | 51.4* ± 1.7 | 34.5‡ ± 0.2 |
| O_2 pulse, ml/beat | 21.9 ± 3.7 | 20.8 ± 3.3 | 17.8§ ± 4.7 | 21.2 ± 3.0 | 20.5 ± 2.3 | 17.8‡ ± 3.2 | 22.3 ± 4.6 | 21.1 ± 4.4 | 18.6‡ ± 5.9 | 22.7 ± 2.7 | 20.0 ± 0.3 | 13.5‡ ± 0.3 |
| $\dot{V}E_{\max}$, l/min BTSP | 144.0 ± 23.4 | 151.4 ± 20.0 | 117.4‡ ± 24.7 | 143.0 ± 19.4 | 148.6 ± 18.3 | 117.5‡ ± 14.7 | 145.5 ± 29.8 | 154.6 ± 23.9 | 122.3‡ ± 31.1 | 142.0 ± 15.6 | 147.9 ± 2.7 | 92.2‡ ± 14.4 |
| RER | 1.11 ± 0.07 | 1.11 ± 0.05 | 1.13 ± 0.07 | 1.10 ± 0.08 | 1.11 ± 0.06 | 1.12 ± 0.05 | 1.11 ± 0.07 | 1.09 ± 0.05 | 1.14 ± 0.08 | 1.14 ± 0.01 | 1.14 ± 0.02 | 1.15 ± 0.06 |
| Perceived exertion | | 19.1 ± 0.8 | 19.2 ± 0.8 | | 19.3 ± 0.7 | 19.1 ± 0.3 | | 19.0 ± 0.9 | 19.2 ± 1.0 | | 18.5 ± 0.7 | 19.5 ± 0.7 |

Values are means \pm SD; *n*, no. of subjects except $n = 20$ for all subjects and 8 for high-intensity group initial evaluation (T1) respiratory exchange ratio (RER) and $n = 20$ for all subjects and 9 for moderate-intensity group T1 resting heart rate (HR_{rest}) and maximal ventilation ($\dot{V}E_{\max}$). T2, 10-yr follow-up; T3, 20-yr follow-up; HR_{\max} , maximal HR; $\dot{V}O_{2\max}$, maximal O_2 uptake; FFW, fat-free weight. Significant difference ($P \leq 0.05$): * from T1; † T2 vs. T3; ‡ T1 vs. T3 and T2 vs. T3. § Group by time interaction.

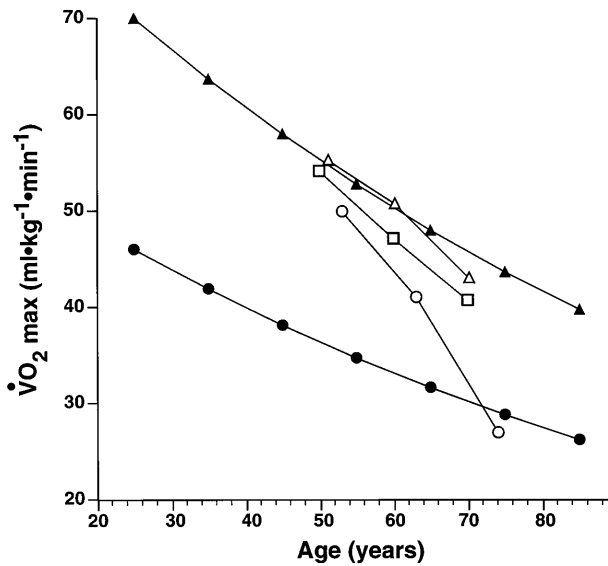


Fig. 1. Maximal oxygen uptake ($\dot{V}O_{2max}$) of older endurance athletes who continued to train at a high (Δ), moderate (\square), or low intensity (\circ) after 10- and 20-yr follow-ups (present study). Curves for athletes (\blacktriangle) and untrained healthy persons (\bullet) are cross-sectional norms from Heath et al. (9).

and from T2 to T3 and the RPE from T2 to T3 did not change significantly over time for all groups. These data were clearly in the range that is reflective of a maximal effort.

Factors associated with change in aerobic power. To gain insight on potential factors affecting the change in $\dot{V}O_{2max}$ ($\Delta\dot{V}O_{2max}$), Table 2 presents data on age and longitudinal differences (T1 to T2 and T2 to T3) of selected variables and their relationship to the $\Delta\dot{V}O_{2max}$ described by simple linear regression. To further assess these relationships, multiple regression analyses were performed, with all variables entered into the regression equation in one single block. In assessing the T1 to T2 $\Delta\dot{V}O_{2max}$, combining $\Delta\%fat$, $\Delta\dot{V}E_{max}$, and ΔO_2 pulse increased the correlation with $\Delta\dot{V}O_{2max}$ to $r = 0.93$ ($r^2 = 0.86$; $P \leq 0.05$). In assessing the T2 to T3 $\Delta\dot{V}O_{2max}$, combining age, $\Delta pace$, $\Delta\dot{V}E_{max}$, and ΔO_2 pulse increased the correlation with $\Delta\dot{V}O_{2max}$ to $r = 0.87$ ($r^2 = 0.75$; $P \leq 0.05$).

Body composition and selected anthropometric data. The data for body composition and other selected anthropometric measures are shown in Table 3. Standing height was maintained from T1 to T2 for all groups

and decreased slightly from T2 to T3 for the H and M groups. Total body weight remained constant over the 20-yr testing period for the H and M groups but increased significantly from T2 to T3 for the L group. Although the H and M groups remained lean over the 20-yr follow-up period, they significantly decreased their FFW from T1 to T2 and from T2 to T3 and increased their body fat over the same time periods. The FFW of the L group initially decreased from T1 to T2 but increased to their T1 values at T3. The apparent maintenance of FFW is deceptive in that the L group had increased their body weight by 6.3 kg and their body fat by 3.7% from T2 to T3.

The waist circumference followed the same pattern of increase as found with body fat values and was particularly evident from T2 to T3 in the L group. In contrast, the hip, thigh, and biceps circumferences were generally maintained for all groups except for an increase in hip girth from T2 to T3 for the L group. Chest expansion was generally maintained by all groups over the 20-yr follow-up except for a significant decrease from T2 to T3 for the H group. Because there was no significant difference in chest expansion from T1 to T3 for the H group, the above-mentioned decrease from T2 to T3 was most likely reflective of the small increase found between T1 and T2.

Pulmonary function. Spirometry results (FEV_1 , FVC, and FEV_1 -to-FVC ratio) were available for 20 of 21 subjects from T1, T2, and T3. Mean values remained similar for all subjects and all groups from T1 to T2 (Table 4). At T3, a significant decline was observed in FEV_1 and FVC. The greater decline in FVC compared with FEV_1 resulted in a significant increase in the FEV_1 -to-FVC ratio at T3 compared with T2. Residual volume (RV), total lung capacity (TLC), and the RV-to-TLC ratio were determined in all subjects at T2 and T3. At T3, RV remained similar to T2 values, whereas TLC significantly declined. The decline in TLC resulted in a significant increase in the RV-to-TLC ratio.

Cross-sectional data on strength and BMD at T3. The data for strength and BMD by age are shown in Table 5. The results show that leg strength was well maintained up to 79–89 yr and significantly reduced by age 90+. In contrast, lumbar extension peak torque was significantly lower at 79+ yr. There was a general trend for chest press strength to decline with each decade of age and particularly after 79 yr, but it was only significant

Table 2. Longitudinal changes among all subjects of selected variables and simple linear regression model of relationship with $\Delta\dot{V}O_{2max}$

| | Age, yr | $\Delta\dot{V}O_{2max}$, ml·kg ⁻¹ ·min ⁻¹ | $\Delta\%Fat$, skinfolds | ΔFFW , kg | $\Delta Mileage$, miles/wk | $\Delta Pace$, min/miles | $\Delta\dot{V}E_{max}$, l/min BTSP | ΔHR_{max} , beats/min | ΔO_2 Pulse, ml/beats |
|--|----------------|---|------------------------------|----------------------|--------------------------------|------------------------------|--|----------------------------------|---------------------------------|
| <i>$\Delta T1$ to $T2$</i> | | | | | | | | | |
| Mean \pm SD | 60.2 \pm 8.8 | -4.2 \pm 4.5 | 2.5 \pm 1.6 | -2.4 \pm 1.2 | -5.7 \pm 0.4 | 0.68 \pm 0.93 | 8.4 \pm 14.4 | -7 \pm 6 | -1.1 \pm 1.6 |
| r | 0.07 | 1.0 | 0.52* | 0.16 | 0.02 | 0.32 | 0.50* | 0.15 | 0.88* |
| <i>$\Delta T2$ to $T3$</i> | | | | | | | | | |
| Mean \pm SD | 70.4 \pm 8.8 | -9.6 \pm 4.6 | 2.4 \pm 2.2 | -0.5 \pm 1.9 | -8.9 \pm 10.0 | 1.82 \pm 2.30 | -34.0 \pm 14.9 | -6 \pm 7 | -3.0 \pm 2.3 |
| r | 0.45* | 1.0 | 0.29 | 0.01 | 0.08 | 0.56* | 0.56* | 0.08 | 0.85* |

$n = 21$ subjects except $n = 20$ subjects for longitudinal change (Δ) from T1 to T2 $\Delta\dot{V}E_{max}$. Ages are for subjects at T2 and T3. * $P \leq 0.05$.

Table 3. *Anthropometric measurements*

| | All Subjects | | | High-Intensity Group | | | Moderate-Intensity Group | | | Low-Intensity Group | | |
|---------------------------|----------------|-----------------|-----------------|----------------------|----------------|-----------------|--------------------------|-----------------|-----------------|---------------------|----------------|-----------------|
| | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 |
| <i>n</i> | 21 | 21 | 21 | 9 | 9 | 9 | 10 | 10 | 10 | 2 | 2 | 2 |
| Height, cm | 177.4 ± 5.9 | 176.6* ± 6.2 | 175.8‡ ± 6.5 | 175.6 ± 5.6 | 175.5 ± 6.0 | 174.5‡ ± 6.6 | 179.6 ± 6.2 | 178.2* ± 6.9 | 177.6* ± 7.0 | 174.3 ± 2.2 | 173.4 ± 1.6 | 173.1 ± 0.5 |
| Weight, kg | 70.1 ± 7.8 | 69.4 ± 8.2 | 70.8‡ ± 8.8 | 66.3 ± 5.7 | 65.3 ± 5.9 | 65.8 ± 5.2 | 72.3 ± 8.9 | 71.9 ± 9.3 | 73.1 ± 9.6 | 75.9 ± 1.7 | 75.2 ± 3.0 | 81.5‡ ± 0.1 |
| FFW, kg | 61.4 ± 6.2 | 59.1 ± 6.2 | 58.6§ ± 6.5 | 59.5 ± 5.1 | 57.0* ± 5.0 | 55.7‡ ± 4.7 | 62.7 ± 7.5 | 60.4* ± 7.4 | 60.0* ± 7.6 | 63.9 ± 0.6 | 61.6* ± 1.9 | 63.7‡ ± 0.6 |
| Σ7 Skinfolds, mm | 66.9 ± 25.8 | 75.2* ± 25.6 | 82.6‡ ± 26.3 | 53.7 ± 12.7 | 61.8 ± 15.8 | 70.6* ± 19.9 | 74.5 ± 31.4 | 83.3 ± 29.8 | 87.7* ± 28.7 | 88.0 ± 5.7 | 95.0 ± 7.1 | 111.5* ± 6.4 |
| %Fat, skinfolds | 12.2 ± 4.2 | 14.6* ± 4.2 | 17.1‡ ± 4.2 | 10.2 ± 2.1 | 12.7* ± 2.6 | 15.3‡ ± 2.9 | 13.2 ± 5.3 | 15.8* ± 5.0 | 17.7‡ ± 4.8 | 15.7 ± 1.1 | 18.1 ± 0.8 | 21.8‡ ± 0.6 |
| Waist girth, cm | 80.0 ± 6.0 | 82.4* ± 6.5 | 85.4‡ ± 7.3 | 76.3 ± 3.9 | 79.3* ± 5.0 | 81.2* ± 5.5 | 83.1 ± 6.5 | 84.3* ± 7.4 | 87.4‡ ± 7.1 | 82.7 ± 0.7 | 86.6 ± 0.2 | 94.6‡ ± 3.3 |
| Hip girth, cm | 93.6 ± 3.9 | 92.1* ± 4.0 | 92.9‡ ± 3.8 | 91.7 ± 3.5 | 90.5 ± 3.6 | 90.9 ± 2.2 | 94.6 ± 3.7 | 92.9 ± 4.0 | 93.6 ± 3.8 | 97.8 ± 2.2 | 95.8 ± 1.4 | 98.6‡ ± 2.8 |
| Thigh girth, cm | 54.1 ± 3.8 | 54.3 ± 3.1 | 53.5 ± 3.3 | 54.1 ± 3.8 | 54.1 ± 2.9 | 52.3 ± 2.4 | 55.0 ± 4.6 | 54.4 ± 3.6 | 53.8 ± 3.6 | 57.5 ± 1.8 | 54.9 ± 1.8 | 57.8 ± 0.8 |
| Biceps girth, cm | 30.5 ± 2.2 | 30.5 ± 2.3 | 30.7 ± 2.4 | 29.2 ± 1.7 | 29.3 ± 1.5 | 29.2 ± 1.5 | 31.2 ± 1.9 | 31.0 ± 2.4 | 31.3 ± 2.2 | 33.9 ± 0.2 | 33.2 ± 1.0 | 34.8 ± 0.8 |
| Chest expansion girth, cm | 7.3 ± 1.8 | 7.8 ± 1.4 | 7.1‡ ± 1.8 | 7.0 ± 1.5 | 7.8 ± 1.2 | 6.7‡ ± 1.5 | 7.2 ± 1.7 | 7.5 ± 1.3 | 7.2 ± 1.9 | 9.1 ± 3.8 | 9.7 ± 1.3 | 7.9 ± 3.1 |

Values are means ± SD; *n*, no. of subjects except *n* = 20 for all subjects and 9 for moderate-intensity group T1 waist girth and hip girth, *n* = 19 for all subjects and 8 for moderate-intensity group T1 thigh girth and biceps girth, and *n* = 18 for all subjects and 8 each for high- and moderate-intensity group T1 chest expansion girth. Significant difference ($P \leq 0.05$): * from T1; † T2 vs. T3; ‡ T1 vs. T3 and T2 vs. T3. § Group by time interaction.

Table 4. *Changes in pulmonary function*

| | All Subjects | | | High-Intensity Group | | | Moderate-Intensity Group | | | Low-Intensity Group | | |
|---------------------------|------------------|------------------|-------------------|----------------------|------------------|-------------------|--------------------------|------------------|-------------------|---------------------|------------------|------------------|
| | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 | T1 | T2 | T3 |
| <i>n</i> | 20 | 20 | 20 | 9 | 9 | 9 | 10 | 10 | 10 | 1 | 1 | 1 |
| FEV ₁ , liters | 3.906 ± 0.559 | 3.840 ± 0.619 | 3.392‡ ± 0.627 | 3.742 ± 0.467 | 3.656 ± 0.499 | 3.282‡ ± 0.498 | 4.013 ± 0.640 | 3.971 ± 0.721 | 3.469‡ ± 0.764 | 4.313 ± 0 | 4.190 ± 0 | 3.620* ± 0 |
| FVC, liters | 5.187 ± 0.848 | 5.248 ± 0.742 | 4.376‡ ± 0.692 | 4.980 ± 0.459 | 5.009 ± 0.579 | 4.211‡ ± 0.591 | 5.445 ± 1.080 | 5.447 ± 0.872 | 4.527‡ ± 0.806 | 5.289 ± 0 | 5.420 ± 0 | 4.340‡ ± 0 |
| FEV ₁ /FVC, % | 76 ± 6 | 73 ± 7 | 78‡ ± 6 | 77 ± 6 | 73 ± 7 | 78‡ ± 6 | 74 ± 5 | 73 ± 7 | 76‡ ± 7 | 82 ± 0 | 77 ± 0 | 83 ± 0 |
| RV, liters | | 2.514 ± 0.426 | 2.530 ± 0.469 | | 2.365 ± 0.384 | 2.463 ± 0.403 | | 2.507 ± 0.347 | 2.416 ± 0.326 | | 3.217 ± 0.428 | 3.403 ± 0.675 |
| TLC, liters | | 7.712 ± 0.974 | 6.838‡ ± 0.885 | | 7.374 ± 0.778 | 6.674‡ ± 0.750 | | 7.954 ± 1.115 | 6.943‡ ± 1.047 | | 8.335 ± 0 | 7.265‡ ± 0 |
| RV/TLC, % | | 32 ± 3 | 36‡ ± 4 | | 32 ± 4 | 37‡ ± 3 | | 32 ± 3 | 35‡ ± 3 | | 35 ± 0 | 40 ± 0 |

Values are means ± SD; *n*, no. of subjects except *n* = 21 for all subjects and 2 for low-intensity group residual volume (RV). FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity; TLC, total lung capacity. Significant difference ($P \leq 0.05$): * from T1; † T2 vs. T3; ‡ T1 vs. T3 and T2 vs. T3.

Table 5. *Cross-sectional strength and bone mineral density data by age at T3*

| | Total Group | <i>n</i> | 60–69 yr | <i>n</i> | 70–78 yr | <i>n</i> | 79–89 yr | <i>n</i> | 90+ yr | <i>n</i> |
|---|---------------|----------|---------------|----------|---------------|----------|---------------|----------|----------------|----------|
| Leg press 1 RM, kg | 120.7 ± 32.9 | 21 | 134.4 ± 29.3 | 9 | 126.4 ± 9.7 | 5 | 126.7 ± 21.9 | 4 | 62.1 ± 17.2‡ | 3 |
| Chest press 1 RM, kg | 54.9 ± 17.1 | 22 | 65.4 ± 13.5 | 9 | 57.6 ± 14.3 | 6 | 44.9 ± 12.4 | 4 | 31.0 ± 5.7* | 3 |
| Lumbar peak torque, N·m | 292.2 ± 58.0 | 22 | 326.4 ± 50.0‡ | 10 | 297.8 ± 50.0 | 5 | 234.6 ± 28.1 | 4 | 245.4 ± 45.4 | 3 |
| Bone mineral density, g/cm ² | | | | | | | | | | |
| L ₂ | 1.126 ± 0.119 | 23 | 1.163 ± 0.134 | 10 | 1.082 ± 0.131 | 6 | 1.098 ± 0.066 | 4 | 1.128 ± 0.115 | 3 |
| L ₃ | 1.181 ± 0.125 | 23 | 1.177 ± 0.132 | 10 | 1.186 ± 0.147 | 6 | 1.190 ± 0.120 | 4 | 1.174 ± 0.126 | 3 |
| L ₁₋₄ | 1.148 ± 0.112 | 23 | 1.157 ± 0.132 | 10 | 1.124 ± 0.127 | 6 | 1.154 ± 0.085 | 4 | 1.157 ± 0.075 | 3 |
| Total body | 1.185 ± 0.050 | 23 | 1.203 ± 0.057 | 10 | 1.185 ± 0.022 | 6 | 1.182 ± 0.046 | 4 | 1.129 ± 0.041* | 3 |
| Trochanter | 0.859 ± 0.103 | 23 | 0.888 ± 0.117 | 10 | 0.839 ± 0.105 | 6 | 0.834 ± 0.051 | 4 | 0.837 ± 0.133 | 3 |

Values are means ± SD; *n*, no. of subjects. 1 RM, 1 repetition maximum. Of 23 subjects evaluated for bone mineral density, two 90+ yr subjects were evaluated only at T3. Significant difference ($P \leq 0.05$): * 90+ yr vs. 60–69 yr; † 90+ yr vs. 69–69 yr, 70–78, and 79–89 yr; ‡ 60–69 yr vs. 79–89 and 90+ yr.

at 90+ yr. All BMD measurements were generally maintained for all age groups 60–90+ yr except for the total body BMD value for 60–69 vs. 90+ yr.

Resistance training. The subjects who reported participating regularly in weight training between T2 and T3 ($n = 16$) were compared at T3 with those who did not ($n = 5$). The data in Table 6 represent the changes in scores from T2 to T3 in selected variables. There was no significant difference between the groups in any of the selected variables.

DISCUSSION

Aerobic capacity. Previous studies (2, 5, 9, 28, 29, 33) have shown a 5–15% reduction/decade in $\dot{V}O_{2\max}$ for men 20–75 yr of age. Some have suggested that highly trained endurance athletes and average-trained fitness participants may have less than a 5% decline/decade in $\dot{V}O_{2\max}$ if they continue high-level training. In contrast, highly trained endurance athletes who become sedentary have a greater than average reduction in $\dot{V}O_{2\max}$ with age (5, 28, 33). Part of the disparity in reports of the change and/or decline in $\dot{V}O_{2\max}$ with age is related to whether the data were reported from cross-sectional or longitudinal studies. Dehn and Bruce (5) stated that the information reported from longitudinal studies is more accurate. Cross-sectional studies may represent a bias sample, generally including more fit persons who are willing to volunteer and provide a maximal effort during exercise testing (5).

Lifestyle or change in activity habits can significantly affect the rate of decline in $\dot{V}O_{2\max}$ with age (10, 11, 23). Jackson et al. (10), in a cross-sectional analysis of 1,499 healthy men 25–70 yr of age, found that $\dot{V}O_{2\max}$ was lower in older groups and that age accounted for 50% of the variation associated with self-reported physical activity index and percent fat values. This was also confirmed in a 4.1-yr follow-up on a subsample of 156 men. Even so, the lack of reported information concerning activity status of the various studies often makes longitudinal data difficult to interpret.

Our present 20-yr follow-up data show average declines from T1 to T2 and from T2 to T3 of 8 and 15%, respectively, for the H group, 13 and 14%, respectively, for the M group, and 18 and 34%, respectively, for the L group (Fig. 1). This may appear contradictory to the results of the 10-yr follow-up by Pollock et al. (23) in which the H group did not change in $\dot{V}O_{2\max}$. The earlier H group did not change its intensity and volume of exercise from T1 to T2. The H group at T3 were different individuals who all had significantly reduced their training intensity and mileage. Heath et al. (9) averaged the $\dot{V}O_{2\max}$ values from nine different studies

that included 563 sedentary healthy men and found a decline in $\dot{V}O_{2\max}$ of 9%/decade. On the basis of the assumptions of Heath et al., aging curves for active and sedentary men were developed and plotted in Figs. 1 and 2. It is obvious from Fig. 1 that our H and M groups followed a similar pattern of decline in $\dot{V}O_{2\max}$ over the 20-yr period of follow-up and had a slightly greater rate of decline than the active persons' curve, particularly from T2 to T3 for the H group. Even so, both the H and M groups were ranked above the 95th percentile norm values compared with their age group. [Norms were based on data from the Cooper Aerobic Center Longitudinal Study (CACLS) and published by Pollock and Wilmore (26). Additional norms were provided by Dr. Stephen Blair and Mark Harris of the CACLS for persons over 80 yr of age (personal communication)]. In addition, the H group was able to maintain its elite status in national and international competition in aerobic endurance events. It is clear that the accelerated rate of decline in $\dot{V}O_{2\max}$ from T2 to T3 (34%) for the L group was caused by a dramatic reduction in exercise volume and training intensity.

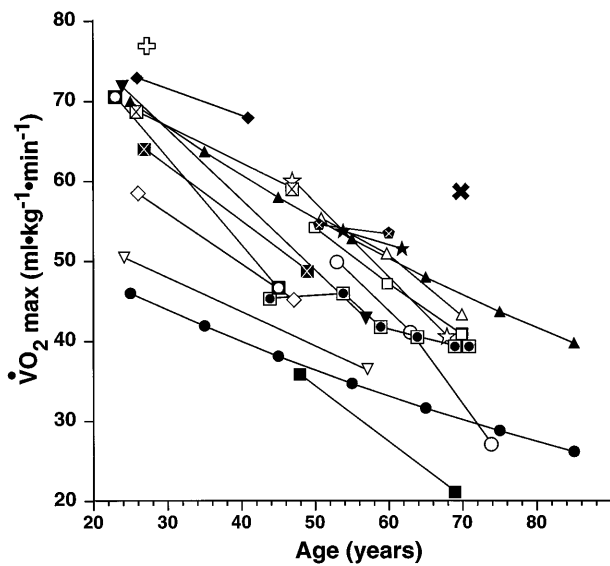
Figure 2 summarizes the $\dot{V}O_{2\max}$ values for various groups of endurance athletes, nonathletic aerobic-fitness participants, and sedentary men reported from various longitudinal studies (2, 9, 11, 12, 17, 23, 28, 29, 33). Also plotted in Fig. 2 are cross-sectional data points for elite young distance runners (22) and a 70-yr-old world recordholder in the marathon (18). Several important points can be gleaned from Fig. 2: 1) the rate of decline in $\dot{V}O_{2\max}$ is not the same for each study; 2) the rate of decline in $\dot{V}O_{2\max}$ is related to the initial level of aerobic power and a reduction in the activity level of the active groups; 3) $\dot{V}O_{2\max}$ remains relatively constant over time if training status does not change; and 4) in most studies in which active participants reduced their training level but still remained quite active, $\dot{V}O_{2\max}$ declined at a rate of 5–10%/decade.

In the 10-yr follow-up studies by Pollock et al. (23) on highly trained endurance athletes and Kasch and Wallace (12) on average aerobic-fitness-trained participants, no significant changes in $\dot{V}O_{2\max}$ were observed. These were the only studies listed in which training volume and intensity did not change. It is quite evident from the present study and the 22-yr follow-up by Trappe et al. (33) on endurance athletes and by Kasch et al. (11) and Trappe et al. (33) on aerobic-fitness participants that maintaining the volume and intensity of training over periods of time longer than 10 yr is difficult and has not been reported. Difficulty in maintaining a high level of training intensity appears to be the case with both younger and older participants. It

Table 6. Comparison of subjects who reported regular resistance exercise training at T3 with longitudinal changes from T2 to T3

| | <i>n</i> | Age, yr | Δ Weight, kg | Δ FFW, kg | $\Delta\%$ Fat, skinfolds | Δ Chest Expansion Girth, cm | Δ Biceps Girth, cm | Δ Thigh Girth, cm | $\Delta\dot{V}O_{2\max}$, ml·kg ⁻¹ ·min ⁻¹ |
|------------------------|----------|------------|---------------------|------------------|---------------------------|------------------------------------|---------------------------|--------------------------|---|
| Resistance and aerobic | 16 | 70.3 ± 9.6 | 0.6 ± 3.9 | -0.4 ± 2.1 | 2.3 ± 2.3 | -0.6 ± 1.2 | 0.4 ± 1.1 | -0.06 ± 3.0 | -9.9 ± 5.1 |
| Aerobic | 5 | 71.0 ± 6.4 | 0.9 ± 3.1 | -1.0 ± 1.0 | 2.9 ± 2.0 | -1.3 ± 0.8 | -0.2 ± 0.9 | -1.4 ± 1.6 | -8.9 ± 2.9 |

Values are means ± SD; *n*, no. of subjects. No significant differences were found between groups.



- ◇ Astrand et al., 1973, Athletes-Untrained (n=31)
- ◻ Trappe et al., 1996, Highly Trained-Untrained (n=15)
- ⊠ Trappe et al., 1996, Fitness Trained (n=18)
- ⊞ Trappe et al., 1996, Highly Trained (n=10)
- ☆ Trappe et al., 1996, Older Fitness Trained (n=10)
- ◼ Kasch et al. 1995, Fitness Trained (n=12)
- Kasch et al. 1995, Untrained (n=12)
- ◆ Marti et al., 1991, Athletes-Trained (n=27)
- ▲ Heath et al., 1981, Athlete, Norms
- Heath et al., 1981, Untrained, Norms
- ⊠ Maud et al., 1981, Elite Older Athlete (n=1)
- ⊞ Pollock et al., 1977, Elite Young Athletes (n=20)
- ⊞ Pollock et al., 1987, Highly Trained (n=11)
- ▼ Robinson et al., 1976, Athletes-Untrained (n=13)
- ▽ Robinson et al., 1976, Untrained (n=9)
- ★ Rogers et al., 1990, Highly Trained (n=15)
- Pollock et al., present, Low intensity (n=2)
- Pollock et al., present, Moderate Intensity (n=10)
- △ Pollock et al., present, High Intensity (n=9)
- ⊞ Cooper, 1990, Norms

Fig. 2. $\dot{V}O_{2\max}$ of endurance athletes and nonathletes of various ages. Data are from studies listed (bottom).

appears from one study that increasing the volume of training may offset the change in training intensity and lessen the decline in $\dot{V}O_{2\max}$ (11). Whether maintaining both the volume and intensity of training for 15 or more yr would enable $\dot{V}O_{2\max}$ to be maintained is not known.

Factors associated with change in aerobic power. It is important to note that age and measures of training intensity (pace and miles per week) were not well correlated to the $\Delta\dot{V}O_{2\max}$ during the first 10-yr period. Table 1 shows that the mean ages (60 yr) of the groups were similar and training was relatively similar within groups during the first 10-yr period. These data suggest that the age-related decrement in $\dot{V}O_{2\max}$ can be attenu-

ated through middle age (i.e., to age 60 yr) if subjects maintain similar levels of training activity (23). During the second 10-yr follow-up period, however, age and pace were significantly correlated to the decline in $\dot{V}O_{2\max}$. These data thus suggest that a critical interaction among age, level of physical activity, and cardiopulmonary function occurs near age 65–70 yr, resulting in a nonlinear change in aerobic power.

Changes in body composition appear to have important effects on the age-associated decline in $\dot{V}O_{2\max}$. Jackson et al. (10) reported that decreasing levels of physical activity and increasing levels of percent fat accounted for nearly 50% of the age-associated difference in $\dot{V}O_{2\max}$ in men age 25–70 yr. However, the mean age of the subjects at a 4.1-yr follow-up was 50 yr, so it is difficult to interpret how these effects might differ in older participants. Fleg and Lakatta (6) reported that a large portion (at least 50%) of the age-associated decline in $\dot{V}O_{2\max}$ can be attributed to the loss in muscle mass in untrained men. In the present study, changes in percent fat had a moderate correlation with $\Delta\dot{V}O_{2\max}$ during the first 10-yr period, but changes in body composition variables had weaker correlations during the second 10-yr follow-up period (Table 2). These data suggest that body composition changes may be less influential after age 65–70 yr than the cardiopulmonary factors associated with reduced training in the reduction in $\dot{V}O_{2\max}$ in endurance-trained subjects.

O_2 pulse [an estimate of stroke volume (3)] and $\dot{V}E_{\max}$ were highly correlated to $\Delta\dot{V}O_{2\max}$, whereas ΔHR_{\max} was not. The HR_{\max} declined $\sim 5\text{--}7$ beats $\cdot \text{min}^{-1} \cdot \text{decade}^{-1}$, and these data provide strong evidence that habitual physical training does not maintain HR_{\max} with increasing age. However, these data also indicate that the effect of ΔHR_{\max} on $\dot{V}O_{2\max}$ may be highly variable. The maintenance of O_2 pulse at T2, followed by a significant decline at T3 (Table 1), and the high correlation between ΔO_2 pulse and $\Delta\dot{V}O_{2\max}$ suggest that moderate- to high-intensity physical training may prevent an age-related decline in maximal stroke volume at least to approximately age 60 yr. This effect (and/or the muscle's ability to extract O_2 , which was not assessed in this study) might then attenuate the effect of decrements in HR_{\max} on $\Delta\dot{V}O_{2\max}$.

Similar to O_2 pulse, $\dot{V}E_{\max}$ was maintained at T2 but significantly declined at T3 (Table 1). These data and the moderate correlations observed between $\Delta\dot{V}E_{\max}$ and $\Delta\dot{V}O_{2\max}$ suggest that moderate- to high-intensity physical training may prevent an age-related decline in $\dot{V}E_{\max}$ at least to approximately age 60 yr. The large decline in $\dot{V}E_{\max}$ at T3 averaged -19 to -22% and was similar to or less than the decline reported by Trappe et al. (33) for highly trained younger subjects (-20%) and fitness-trained older runners (>50 yr, -30%).

Little information is available concerning the role of pulmonary function changes in the decline of $\dot{V}O_{2\max}$ in endurance-trained athletes. Studies report that most age-associated physiological changes in resting lung function can be attributed to the mechanics of ventilation, i.e., a decrease in chest wall and pulmonary compliance and a decrease in respiratory muscle strength (19). We attempted to determine whether

changes in resting lung function and an indirect measurement of chest wall and pulmonary compliance (circumference measurement of chest expansion) were correlated to $\Delta V_{E_{\max}}$. As shown in Tables 3 and 4, significant changes in chest expansion and resting lung function were not observed until T3. These data suggest that moderate- to high-intensity physical training may prevent an age-related decline in resting lung function at least to approximately age 60 yr. These data are in agreement with McClaran et al. (19), who suggested that nonlinear changes in resting lung function in fit elderly adults likely occur near age 65–70 yr. With the use of simple regression analyses, the $\Delta V_{E_{\max}}$ (T2 to T3) was only significantly correlated with the ΔTLC ($r = 0.44$) and Δpace ($r = 0.50$) but not with the $\Delta \text{chest expansion}$ or the changes in other lung function measurements. Because hyperpnea is an effective training method for respiratory muscle endurance training (15), it is interesting to speculate that changes in pace and its association with hyperpnea and respiratory muscle endurance, as well as changes in lung volume, may have important effects on age-related changes in $V_{E_{\max}}$.

HR_{max}. The data for HR_{max} show a consistent 5–7 beats·min⁻¹·decade⁻¹ decline over the 20-yr follow-up (Table 1). The longitudinal data for the various studies reported in Fig. 3 show a consistent 4–7 beats·min⁻¹·decade⁻¹ decline in HR_{max} for both the active and sedentary groups up to age 55 yr except for the athletes studied by Robinson et al. (Ref. 28; ~2 beats·min⁻¹·decade⁻¹). The longitudinal data approximate the cross-sectional CACLS norms up to age 45 yr, when the norms then diverge to 10–12 beats·min⁻¹·decade⁻¹ up to age 75 yr. These greater declines in HR_{max} found in clinical populations (16, 26) may result from a reluctance to push older populations who are unaccustomed to exercise to a true maximal effort.

The longitudinal data for HR_{max} for athletes and sedentary persons over 55 yr show a more varied pattern (Fig. 3). Kasch et al. (11) found a 7 beat·min⁻¹·decade⁻¹ decline in their active group, and Rogers et al.

(29), in an 8-yr follow-up, showed no decline in HR_{max}, whereas Trappe et al. (33) found a 10 beat·min⁻¹·decade⁻¹ decrease in older fitness-trained subjects.

Body composition variables. In comparison with elite young runners (24), the percent fat of runners 40–75 yr has been shown to be 5–10% greater (~5 vs. 10–15%) (9, 18, 23, 25, 33). The master athletes in this study compare favorably to other investigations (8, 26) on master endurance athletes, are lower than or comparable to average young men, and are significantly lower than age-matched average men (average young men, 14–17% fat; 40–79 yr, 20–24% fat). Although total body weight remained stable for the H and M groups, they each increased percent fat ~2–2.5%/decade. This is in contrast to the L group, who showed the same pattern of body composition change from T1 to T2 as the H and M groups but dramatically increased their body weight (6.3 kg) and percent fat (3.7%) from T2 to T3. These results clearly show that the significant reduction in physical activity for the L group helped account for its change in body composition. These results are consistent with those of Trappe et al. (33), who also showed greater increases in percent fat for their younger group who became inactive (~6.1%/decade) compared with the group who remained highly active (~2.5%) after a 22-yr follow-up (age ~24.5–46.5 yr). Their older fitness group who remained active showed no change in body weight but increased percent fat ~3%/decade.

In the present group, 16 subjects were also weight training regularly and 5 were not. In reviewing Table 6, although no significant differences were found from T2 to T3 between the two groups, there was a consistent trend for the training group to better maintain FFW, chest expansion, and both biceps and thigh circumferences. The small sample size makes interpretation difficult, and further research is necessary before a more definitive statement can be made.

BMD and strength. Although cross-sectional data and correlation analysis are limiting in determining long-term age effects and cause-and-effect relationships among variables, some of the findings may be important. Usually BMD declines in men after 50–60 yr but at a much slower rate than found in women (20). It has been shown that exercise training usually makes a small increase in, maintains, or attenuates the loss of BMD (20, 31). Also, cross-sectional studies comparing athletes and nonathletes or the dominant (exercise) arm with the nondominant arm showed greater BMD in the active groups or exercised limbs (21, 32). Therefore, it appears from Table 5 that the subjects tested at T3 generally maintained a satisfactory level of BMD. Possibly the better maintenance of BMD at the lumbar and trochanter sites compared with the total body values was a result of the specificity of training (21) and that high-impact activities such as running may have a greater effect in areas that have a larger concentration of trabecular bone (13).

Because of the lack of normative data, it is difficult to provide inference on the strength data. But it is well established that strength declines with age, particularly after 50 yr (8, 30). In the present study, it appears that leg press strength was well maintained through

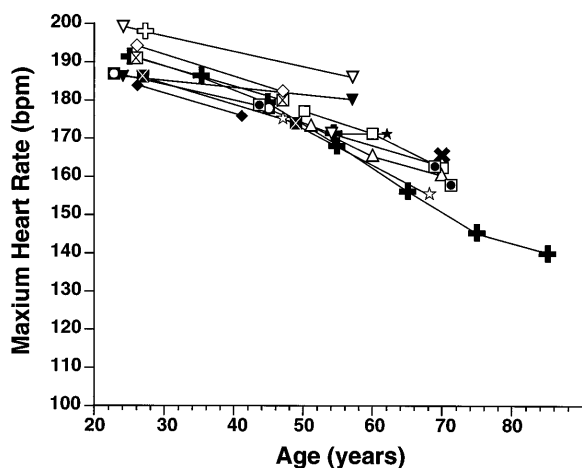


Fig. 3. Maximal heart rate of endurance athletes and nonathletes of various ages. Normative cross-sectional data are from Cooper Aerobics Center Longitudinal Study and published by Pollock and Wilmore (26). See Fig. 2 for legend identifying each study. bpm, Beats per minute.

age 85 yr and chest press and lumbar strength through age 78 yr. In a comparison of weight trainers and non-weight trainers, the weight trainers had a significantly higher arm region BMD (0.931 vs. 0.872 g/cm²) and the relationship between arm region BMD and chest press strength was significant ($r = 0.55$). Thus it appears from this study that BMD, strength, and FFW were better maintained compared with aged-matched norms of nonexercising healthy men (7, 30). Also, it appears that the addition of weight training to the exercise regimen may assist in maintaining upper body BMD, strength, and FFW. Again, these trends and relationships can only be confirmed with longitudinal data with a larger sample size.

In conclusion, the results showed that the physiological capacities of older endurance athletes declined after a 20-yr follow-up, even when the intensity of training was continued at a high or moderate level. A small subgroup who greatly reduced their intensity of training made substantially larger declines in physiological capacities and body composition. Body composition changes were related to aging and/or the type of training performed. The inclusion of weight training may be helpful in maintaining FFW and upper body strength and BMD with age, but the data from this study are limiting and do not allow a strong statement concerning this issue.

The authors acknowledge Linda Martin for typing skills and Linda Garzarella for technical assistance in preparing the tables and figures for this manuscript.

This project was partially funded by the American Heart Association, Florida Affiliate, and National Institutes of Health National Center for Research Resources Grant RR-00082.

Present addresses: L. J. Mengelkoch, Physical Therapy Division, Ohio State Univ., 1583 Perry St., Columbus, OH 43210; J. E. Graves, Dept. of Health and Physical Education, Syracuse Univ., Syracuse, NY 13244-5040; C. Foster, Milwaukee Heart Institute, 960 N. 12th St., Milwaukee, WI 53201; J. H. Wilmore, Dept. of Kinesiology and Health Education, Univ. of Texas, Belmont Hall 222, Austin, TX 70712.

Address for reprint requests: M. L. Pollock, Univ. of Florida, Dept. of Medicine, PO Box 100277, Gainesville, FL 32610.

Received 25 June 1996; accepted in final form 23 December 1996.

REFERENCES

- American Thoracic Society.** Standardization of spirometry—1987 update. *Am. Rev. Respir. Dis.* 136: 1285–1298, 1987.
- Astrand, I., P. O. Astrand, I. Hallback, and A. Kilbom.** Reduction in maximal oxygen uptake with age. *J. Appl. Physiol.* 35: 649–654, 1973.
- Bhambhani, Y., S. Norris, and G. Bell.** Prediction of stroke volume from oxygen pulse measurements in untrained and trained men. *Can. J. Appl. Physiol.* 19: 49–59, 1994.
- Borg, G., and H. Linderholm.** Perceived exertion and pulse rate during graded exercise in various age groups. *Acta Med. Scand.* 472: 194–206, 1967.
- Dehn, M. M., and R. A. Bruce.** Longitudinal variations in maximal oxygen intake with age and activity. *J. Appl. Physiol.* 33: 805–807, 1972.
- Fleg, J. L., and E. G. Lakatta.** Role of muscle loss in the age-associated reduction in $\dot{V}O_{2\max}$. *J. Appl. Physiol.* 65: 1147–1151, 1988.
- Forbes, G. B.** The adult decline in lean body mass. *Hum. Biol.* 48: 161–173, 1976.
- Golding, L. A., C. R. Myers, and W. E. Sinning.** *Y's Way to Physical Fitness* (3rd ed.). Champaign, IL: Human Kinetics, 1989, p. 125–130.
- Heath, G. W., J. M. Hamberg, A. A. Ehsani, and J. O. Holloszy.** A physiological comparison of young and older endurance athletes. *J. Appl. Physiol.* 51: 634–640, 1981.
- Jackson, A. S., E. F. Beard, L. T. Wier, R. M. Ross, J. E. Stuteville, and S. N. Blair.** Changes in aerobic power of men, ages 25–70 yr. *Med. Sci. Sports Exercise* 27: 113–120, 1995.
- Kasch, F. W., J. L. Boyer, S. VanCamp, F. Nettl, L. S. Verity, and J. R. Wallace.** Cardiovascular changes with age and exercise: a 28-year longitudinal study. *Scand. J. Med. Sci. Sports* 5: 147–151, 1995.
- Kasch, F., and J. P. Wallace.** Physiological variables during 10 years of endurance exercise. *Med. Sci. Sports* 8: 5–8, 1976.
- Lane, N. E., D. A. Bloch, H. H. Jones, W. H. Marshall, P. D. Wood, and J. F. Fries.** Long-distance running, bone density, and osteoarthritis. *JAMA* 255: 1147–1151, 1986.
- Larsson, L., G. Grimby, and J. Karlsson.** Muscle strength and speed of movement in relation to age and muscle morphology. *J. Appl. Physiol.* 46: 451–456, 1979.
- Leith, D. E., and M. Bradley.** Ventilatory muscle strength and endurance training. *J. Appl. Physiol.* 41: 508–516, 1976.
- Londeree, B. R., and M. L. Moeschberger.** Effect of age and other factors on maximal heart rate. *Res. Q. Exercise Sport* 53: 297–304, 1982.
- Marti, B., and H. Howland.** Long-term effects of physical training on aerobic capacity: controlled study of former elite athletes. *J. Appl. Physiol.* 69: 1451–1459, 1990.
- Maud, P. J., M. L. Pollock, C. Foster, J. Anholm, G. Guten, M. Al-Nouri, C. Hellman, and D. H. Schmidt.** Fifty years of training and competition in the marathon: Wally Hayward aged 70—a physiological profile. *S. Afr. Med. J.* 59: 153–157, 1981.
- McClaran, S. R., M. A. Babcock, D. F. Pegelow, W. G. Reddan, and J. A. Dempsey.** Longitudinal effects of aging on lung function at rest and exercise in healthy active fit elderly adults. *J. Appl. Physiol.* 78: 1957–1968, 1995.
- Montoye, H. J.** The 1987 C. H. McCloy Research Lecture Better Bones and Biodynamics. *Res. Q. Exercise Sport* 58: 334–348, 1987.
- Montoye, H. J., E. L. Smith, D. F. Fardon, and E. T. Howley.** Bone mineral in senior tennis players. *Scand. J. Sports Sci.* 2: 26–32, 1980.
- Pollock, M. L.** Submaximal and maximal working capacity of elite distance runners. *Ann. NY Acad. Sci.* 301: 310–322, 1977.
- Pollock, M. L., C. Foster, D. Knapp, J. L. Rod, and D. H. Schmidt.** Effect of age and training on aerobic capacity and body composition of master athletes. *J. Appl. Physiol.* 62: 725–731, 1987.
- Pollock, M. L., L. R. Gettman, A. Jackson, J. Ayres, A. Ward, and A. C. Linnerud.** Body composition of elite class distance runners. *Ann. NY Acad. Sci.* 301: 361–370, 1977.
- Pollock, M. L., H. S. Miller, and J. H. Wilmore.** Physiological characteristics of champion American track athletes 40–75 years of age. *J. Gerontol.* 29: 645–649, 1974.
- Pollock, M. L., and J. H. Wilmore.** *Exercise in Health and Disease: Evaluation and Prescription for Prevention and Rehabilitation* (2nd ed.). Philadelphia, PA: Saunders, 1990, p. 128–138, 319–348, 660–665.
- Porter, M. M., A. A. Vandervoort, and J. Lexell.** Aging of human muscle: structure, function and adaptability. *Scand. J. Med. Sci. Sports* 5: 129–142, 1995.
- Robinson, S., D. B. Dill, R. D. Robinson, S. P. Tzankoff, and J. A. Wagner.** Physiological aging of champion runners. *J. Appl. Physiol.* 41: 46–51, 1976.
- Rogers, M. A., J. M. Hagberg, W. H. Martin, A. A. Ehsani, and J. O. Holloszy.** Decline in $\dot{V}O_{2\max}$ with aging in master athletes and sedentary men. *J. Appl. Physiol.* 68: 2195–2199, 1990.
- Sandler, R. B., R. Burdett, M. Zaleskiewicz, C. Sprowls-Repcheck, and M. Harwell.** Muscle strength as an indicator of the habitual level of activity. *Med. Sci. Sports Exercise* 23: 1375–1381, 1991.
- Suominen, H.** Bone mineral density and long term exercise: an overview of cross-sectional athlete studies. *Sports Med.* 16: 316–330, 1993.
- Suominen, H., and P. Rahkila.** Bone mineral density of the calcaneus in 70- to 81-yr-old male athletes and population sample. *Med. Sci. Sports Exercise* 23: 1227–1233, 1991.
- Trappe, A. W., D. L. Costill, M. D. Vukovich, J. Jones, and T. Melhom.** Aging among elite distance runners: a 22-yr longitudinal study. *J. Appl. Physiol.* 80: 285–290, 1996.