Relationship Between Physiological Loss, Performance Decrement, and Age in Master Athletes


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Background. The use of master athletes to describe an idealized rate of physiological loss associated with aging is quite common. The results of such studies suggest that older athletes may be able to reduce the rate of decline in functional loss. The findings of such studies have been questioned due to their limited sample size and the age range and gender of their subjects.

Methods. We examined a group of 146 male and 82 female master athletes over the age of 40 years. Physiological parameters included maximal oxygen uptake (VO2max), body composition, muscle strength, bone density, and blood chemistries. Medical histories and training records were obtained via questionnaire.

Results. Results demonstrated gender differences in body composition, blood chemistries, blood pressure, VO2max, muscle strength, bone density, and performance (p < .05). All metabolic parameters for men and most for women demonstrated significant losses across the age range (p < .05). In addition, strength and performance for men and women and bone density for women declined significantly with age (p < .05). The demonstrated loss rates did not differ by gender.

Conclusions. Although limited by the lack of a sedentary comparison group, these data suggest that age-related losses in VO2max may not be different from data previously reported for older sedentary adults and that loss in muscle strength and performance with aging is not linear.

The aging process in humans is marked by significant decreases in physiological function. However, the actual rate of loss associated with aging varies widely across organ systems and between individuals and is largely related to genetic profiles and lifestyle (1–3). Studies have reported that exercise attenuates losses in functional capacity with aging. Rogers and coworkers (4) noted a loss of only 5.5% per decade in maximal aerobic capacity in chronically trained master endurance athletes, half that suggested for sedentary adults. Pollock and colleagues (5,6), in one of the very few longitudinal studies on older subjects, reported a nonsignificant decline in maximal oxygen uptake (VO2max) over a 10-year period in master athletes aged 50 to 60 years; however, by the 20-year follow-up, the decline rates approximated 1% per year.

It is obvious from these and other studies that moderate-to high-intensity activity is a means by which the normal functional/physiological losses associated with aging may be altered. Further, studying older, highly active individuals (e.g., master athletes) produces physiological loss rates drastically different from what would be expected from the general, sedentary population. Unfortunately, most reported data utilizing master athletes are from a relatively small number of elite runners (4,7–10; n = 15, n = 11, n = 16, n = 44, and n = 8, respectively). Studies employing cross-sectional design strategies require relatively large samples, leaving open to question the value of the studies mentioned above. To address these issues, we have begun collecting data on a group of older, active master athletes who, for the most part, are not elite but do train regularly and compete at a wide range of performance levels. It is the purpose of this paper to describe this study population from the data obtained during their first visit and, in a cross-sectional design, to relate performance, training intensity, and frequency to laboratory tests of cardiovascular function, strength, bone status, and body composition.

METHODS

Design

This study evaluated 146 male and 82 female master athletes, ranging in age from 40 to 86 years. These highly active older adults are participants in a 20-year longitudinal study at the University of Southern California (USC). The USC Institutional Review Board approved the study. All subjects provided written informed consent upon entry into the study. These data represent each subject’s first visit over a 3- to 4-year span during which the study population was gathered. All subjects completed medical histories, and, after a brief physical examination, individuals with medical conditions precluding full participation in the study were excluded. The exclusion criteria included prior myocardial infarction, undi-
agnosed EKG arrhythmias, and/or hypertension. Subjects with previously diagnosed arrhythmias, hypertension, diabetes, or other metabolic disease who had received clearance from their personal physicians were included. All subjects were actively training and competing in their respective athletic events at the time of the baseline measurements. Whereas the majority of subjects competed in running events (87% of the men and 91% of the women), only approximately 40% were considered highly competitive (11) (Table 1).

**Body Composition Assessment**

Upon arrival in the laboratory, height was measured with a stadiometer, and weight was determined on a calibrated Homs beam-scale. Residual lung volume was assessed utilizing the oxygen dilution technique (12), and body composition was determined via hydrodensitometry. Corrections to body density were made for residual lung volume, and body composition was computed utilizing the Brozek equation (13).

**Pre-exercise Physical Assessments**

Resting EKG was recorded on a Hewlett Packard 1500 Electrocardiograph (Palo Alto, CA). Blood pressure (BP) was measured with subjects in a supine position utilizing an auscultatory technique. After a brief physical examination, an indwelling angiocatheter was inserted into a forearm vein, and overnight fasting blood was drawn into prechilled EDTA Vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ) containing ethylenediaminetetraacetic acid (EDTA). After resting blood was drawn, a small volume of saline was injected into the vein to reduce catheter clotting prior to exercise. Samples were immediately centrifuged, pipetted into storage vials, and stored at −80°C until analysis.

**Fitness Assessment**

VO₂max was determined using a continuous, graded exercise test on a motor-driven treadmill. The test began at 2.5 mph and 0% grade and increased by 0.5 mph and 2%, respectively, every 2 minutes during exercise. Exercise continued until the subject terminated the test at a point of subjective exhaustion. The volume of expired air, O₂, and CO₂ content were determined on an Ametek metabolic measurement system (Pittsburgh, PA). O₂max was said to be achieved if the test met three of the following criteria: (i) respiratory exchange ratio (RER) value > 1.05, (ii) Heart rate (HR) ±5 beats per minute of the age-predicted maximum HR, (iii) a plateau (increase of <50 ml of O₂) in VO₂ with increasing workloads, and (iv) lactate concentrations >7 mmol (14). The EKG was monitored continuously during exercise and for the first 5 minutes of recovery. Heart rates were recorded at rest, at the end of each minute during exercise, at max, and for the first 5 minutes of recovery.

**Blood Sampling**

During exercise, blood was drawn from the indwelling catheter into prechilled EDTA Vacutainer tubes at the end of each minute of exercise, at peak, and at 2 and 5 minutes post exercise. Samples were analyzed for lactate and glucose concentrations using a 23L Lactate Analyzer (Yellow Springs Instruments, Yellow Springs, OH).

**Determination of Lactate Threshold**

Minute values for blood lactate concentration were plotted against time and the graphs visually inspected for the point at which a noticeable change in slope occurred. This point was determined to be the lactate threshold (LT). The LT was matched with the minute values of VO₂, and the percent of VO₂max at LT was determined.

**Bone Mineral Density Assessment**

Bone mineral density (BMD) and bone mineral content (BMC) were measured using the Hologic QDR 1500 DXA (v. 7.1; Bedford, MA). Whole-body, lumbar spine, and hip measurements were performed following standard procedures. Normalized values (T and Z scores) were generated from gender-matched, and in the case of hip scores ethnicity-matched, control subjects.

**Muscle Strength Assessment**

Isokinetic and isometric knee strength was assessed using a KinCom dynamometer (Chattecx Corp., Hixson, TN). Maximal isokinetic knee extension strength was measured concentrically and eccentrically at 60°/s between 15° and 80° of knee flexion. Subjects were provided a minimum of three efforts with 1-minute recovery between efforts. Strength was calculated as the peak torque achieved in Newton-meters (Nm). Maximal isometric knee extension strength was measured at 30°, 45°, and 60° of knee flexion, and isometric knee flexion strength was measured at 15°, 30°, and 45° of knee flexion. Subjects performed each exercise three times, and the best value at each angle from the three efforts was recorded as peak torque (in Nm). Only the results for isometric flexion at 15° and isometric extension 60° are reported in this paper.

**Blood Chemistry**

Glucose, triglyceride, cholesterol, and HDL-cholesterol concentrations were determined from the resting blood samples using a Kodak Ectachem DT60 II (Eastman Kodak Co., Rochester, NY). Measurement by reflectance spectrophotometry provides the basis for the determination of substrate concentrations.

**Performance**

Subjects self-reported training and performance data via questionnaire. Subjects were asked to report years of train-
ing, distance/time trained per week, days trained per week, best performances (5 km, 10 km, marathon [26.2 mi]) for each year they had been competing and best performances within 2 months of the testing date.

**Statistical Methods**

Data were entered into a computer and stored using Microsoft Excel. All statistics were performed using SPSS (v. 9.0). Standard descriptive statistics were used to generate means and standard deviations. Simple t tests were utilized to examine mean and slope differences between men and women. Linear regression was used to provide relationships, and when data did not conform to linear analysis, the data were fit using either second or third degree polynomial equations. When relating bone density to training and performance variables, partial order correlation was used controlling for body weight. For all statistical treatments, p was considered significant at the .05 confidence level.

**RESULTS**

The subjects’ physical characteristics are presented in Table 2. Gender differences were observed for height (t = 13.81, p < .00), weight (t = 14.91, p < .00), percent fat (t = 8.39, p < .00), and lean body mass (LBM) (t = 20.1, p < .00). Body weight did not change with age in either gender. LBM was inversely related to age (r = −0.226 for men and r = −0.210 for women, p < .05) with predicted loss rates of 0.15 kg per year independent of gender. BMI was not different between male and female athletes and was not related to age. No age-related changes in body fat were observed for men; however, a significant, positive correlation was found (r = .237, p < .05) in women.

When examining variables related to cardiovascular risk, we observed gender differences for fasting glucose (t = 4.92, p < .00), triglycerides (t = 2.38, p < .02), HDL-C (t = 6.05, p < .00), systolic BP (t = 4.73, p < .00) and diastolic BP (t = 3.82, p < .00). Total cholesterol was not related to

| Table 2. Physical Characteristics and Coronary Risk Profile of Subjects |
|-----------------------------|-----------------------------|-----------------------------|
|                              | Men (n = 139)               | Women (n = 82)               |
| Parameter                    | Mean (±SD)                  | Range                       | Mean (±SD)                  | Range |
| Age, y                       | 53.8 ± 9.9                  | 39–87                       | 49.4 ± 7.7†                  | 40–77 |
| Weight, kg                   | 73.7 ± 8.9                  | 49–106.1                    | 56.5 ± 7.2†                  | 40.6–80.0 |
| % Fat                        | 16.0 ± 4.4                   | 5.8–26.5                    | 21.5 ± 5.7†                  | 5.1–37.0 |
| LBM, kg                      | 61.76 ± 6.8                 | 45.8–84.1                   | 43.9 ± 5.6†                  | 25.9–58.5 |
| Height, m                    | 1.77 ± .066                 | 1.60–1.93                   | 1.64 ± .071†                 | 1.49–1.82 |
| BMI                          | 23.4 ± 2.3                  | 17.8–32.2                   | 22.3 ± 1.84                  | 18.8–27.5 |
| Glucose, mg/dl               | 99.2 ± 12.8                 | 69–152                      | 91.1 ± 10.6†                 | 66–125 |
| Triglycerides, mg/dl         | 81.5 ± 39.5                 | 33–222                      | 69.0 ± 32.4†                 | 21–210 |
| Cholesterol, mg/dl           | 195.1 ± 36.9                | 89–293                      | 197.0 ± 37.5                 | 94–298 |
| HDL cholesterol, mg/dl       | 64.3 ± 17.8                 | 26–106                      | 79.1 ± 17.64†                | 38–108 |
| Systolic BP, mm Hg           | 126.8 ± 16.4                | 102–170                     | 116.9 ± 11.9†                | 92–158 |
| Diastolic BP, mm Hg          | 77.1 ± 7.9                  | 60–96                       | 72.9 ± 7.9†                  | 50–90 |
| Resting HR, bpm              | 56.8 ± 10.9                 | 36–92                       | 56.3 ± 10.1                  | 30–79 |

**Table 3. Maximal Exercise, Performance, and Training Characteristics of Subjects**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Men (n = 139)</th>
<th>Range</th>
<th>Women (n = 82)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2max, l/min</td>
<td>3.78 ± .01</td>
<td>1.43–5.58</td>
<td>2.55 ± 0.42a</td>
<td>1.67–3.53</td>
</tr>
<tr>
<td>VO2max, ml/kg/min</td>
<td>51.63 ± 11.0</td>
<td>20.0–79.2</td>
<td>45.68 ± 8.1a</td>
<td>28.1–64.1</td>
</tr>
<tr>
<td>VEmax, l/min BTPS</td>
<td>123.66 ± 27.0</td>
<td>39.7–183.5</td>
<td>84.15 ± 13.8a</td>
<td>43.4–106.6</td>
</tr>
<tr>
<td>HRmax</td>
<td>172.0 ± 12.8</td>
<td>135–203</td>
<td>173.8 ± 11.6</td>
<td>144–203</td>
</tr>
<tr>
<td>LAmax, mM</td>
<td>11.0 ± 3.8</td>
<td>3.8–20.1</td>
<td>8.6 ± 2.9a</td>
<td>2.5–14.5</td>
</tr>
<tr>
<td>LT, %</td>
<td>66.1 ± 16.0</td>
<td>27–97</td>
<td>64.9 ± 12.8</td>
<td>30.4–87.8</td>
</tr>
<tr>
<td>Glucose max, mM</td>
<td>7.45 ± 2.3</td>
<td>3.4–10.7</td>
<td>7.82 ± 1.5</td>
<td>4.9–12.8</td>
</tr>
<tr>
<td>RER</td>
<td>1.12 ± 0.11</td>
<td>0.84–1.33</td>
<td>1.09 ± 0.11</td>
<td>0.92–1.30</td>
</tr>
<tr>
<td>Years training</td>
<td>15.2 ± 9.7</td>
<td>2.5–62.0</td>
<td>10.4 ± 5.1 (76)</td>
<td>1.0–26.0</td>
</tr>
<tr>
<td>Miles/wk</td>
<td>33.5 ± 19.3</td>
<td>2.0–99.0</td>
<td>33.3 ± 14.2 (72)</td>
<td>5.0–70.0</td>
</tr>
<tr>
<td>Days trained/wk</td>
<td>5.35 ± 1.3</td>
<td>3–7</td>
<td>5.48 ± 1.13 (76)</td>
<td>2–7</td>
</tr>
<tr>
<td>5 km, min</td>
<td>20.7 ± 3.9 (42)</td>
<td>15.36–39.0</td>
<td>23.54 ± 3.7 (42)a</td>
<td>17.5–34.0</td>
</tr>
<tr>
<td>10 km, min</td>
<td>43.3 ± 7.2 (74)</td>
<td>33.3–79.0</td>
<td>48.8 ± 7.16 (56)a</td>
<td>36.4–70.0</td>
</tr>
<tr>
<td>Marathon</td>
<td>38.2 ± 42.0 (46)</td>
<td>158.1–315.0</td>
<td>246.2 ± 45.2 (40)a</td>
<td>167.2–390.0</td>
</tr>
</tbody>
</table>

**Notes:** VO2max = maximal oxygen uptake; VEmax = maximal ventilation; HRmax = maximal heart rate; LAmax = maximal lactic acid; LT = lactate threshold; RER = respiratory exchange ratio. Number of subjects is given in parentheses.

*Significant difference between men and women; p < .05.
age in men, whereas in women cholesterol increased approximately 1.5 mg/dl per year ($p < .05$). HDL-C did not change with age for men or women. Systolic BP increased significantly with age ($r = .359$ for men and $r = .292$ for women, $p < .05$), approximately 0.5 mm Hg/yr for both genders. Diastolic BP and resting HR were stable across age in this sample irrespective of gender.

Results from the treadmill evaluation of $\text{VO}_2\text{max}$ are presented in Table 3. As expected, significant gender differences were observed for both absolute ($t = 12.93, p < .00$) and relative ($t = 4.16, p < .00$) $\text{VO}_2\text{max}$, maximal ventilation ($t = 12.44, p < .00$), and lactic acid at maximal exercise ($t = 4.33, p < .00$). The treadmill data are expressed graphically in Figure 1. All indices of aerobic capacity declined with age in men except LT, which increased significantly ($r = .323, p < .05$). In women, only LT ($r = .05; \text{NS}$) and maximal RER ($r = .08; \text{NS}$) did not change significantly with age. Regression equations are presented in Figure 1 only when a significant correlation with age was observed. The rate of decline in slope of relative $\text{VO}_2\text{max}$ was 1.2% per year for men and 0.8% per year for women; these rates were not statistically different ($t = .04; \text{NS}$).

Training histories and performance values are also provided in Table 3. Years training, distance trained, and frequency were similar between men and women and did not change as a function of age. As expected, women were statistically slower in all running events ($t = 3.05$ to $4.35, p < .00$). Figure 2 presents the running times for the various distances. The age-group world record for each 5-year age interval is also plotted to provide a comparison between the running times of the sample and elite performance norms. The increase in running time associated with age was simi-
lar between gender groups except for marathon times, although the slope of change was not different \((t = .002–.12; NS)\). The male subjects’ increase of 12 to 15 seconds per year indicated by the regression equation \((y = 0.197 \cdot \text{age} + 112.1 \text{m})\) is not consistent with other studies nor with the world record times. The value for women of 1.93 minutes per year \((y = 1.93 \cdot \text{age} + 151.3 \text{m})\) is similar in magnitude to the increase in established world record times.

Strength characteristics of the subjects are presented in Table 4. Significant gender differences were observed for all strength measures \((t = 6.44–8.94, p < .00)\). Both isometric and isokinetic strength significantly declined with age for men and women \((p < .05)\), approximating 1% per year for the entire sample. However, it was apparent by visualization that the relationship between strength loss and age was not linear. Therefore, we recalculated this relationship by applying a polynomial curve fitting (Figure 3). Using a third-degree polynomial equation improved the explained variance from 12.5% to 26.5% for isometric flexion and from 12.25% to 16.6% in isometric extension for men, but did not alter the prediction for isokinetic extension strength for men or any of the strength predictions for women. Comparing the slope differences between men and women in the rate of strength decline, no significant differences were observed \((t = < .003–.005; NS)\).

BMD and BMC data are also presented in Table 4 and Figure 3. For all bone measures, the values for men were significantly higher than those for women \((t = 3.11–10.16; p < .00)\). In these women, for all skeletal sites, significant inverse correlations were observed relating age to BMD and BMC.
(r = −.317, −.376, and −.276 for whole body, hip, and spine, respectively; p < .05). No age relationships were observed in men for BMD. The mean T scores for the sample were −.478 ± .88 and −.765 ± 1.06 (hip); −.462 ± 1.4, and −.811 ± 1.42 (spine) for men and women, respectively.

In relating running performance to laboratory assessment of physiological function, the results indicated a significant influence of body composition and weight on performance, particularly for female runners. In women, percent fat was significantly related to all running distances reported (r = .481, .495, and .620 for the 5 km, 10 km, and marathon, respectively; p < .05). For men, percent fat was not significantly related to running performance at any distance. BMI was significantly correlated with marathon running times for both men (r = .366, p < .05) and women (r = .478, p < .05). LBM was not related to run times in these subjects.

VO2max (l/min) was related to LBM (r = .336, p < .05) and muscle strength (r = .268, .322, and .386 for isometric flexion, isometric extension, and isokinetic extension, respectively, p < .05) in men. In women, VO2max was negatively related to percent fat (r = −.529, p < .05) and positively related to LBM (r = .575, p < .05), isometric flexion strength (r = .483, p < .05), and isometric extension strength (r = .491, p < .05). When VO2max was expressed relative to body weight, significant correlations were observed with percent fat (r = −.303, p < .05), BMI (r = −.263, p < .05), glucose (r = −.212, p < .05), triglycerides (r = −.244, p < .05), systolic BP (r = −.244, p < .05), resting HR (r = −.188, p < .05), systolic BP (r = .221, p < .05), isokinetic extension strength (r = .252, p < .05), and whole-body BMC (r = −.218, p < .05) in men. Significant correlations were also observed for percent fat (r = −.316, p < .05), BMI (r = −.491, p < .05), triglycerides (r = −.256, p < .05), systolic BP (r = −.219, p < .05), resting HR (r = −.274, p < .05), and isometric extension strength (r = .311, p < .05) in women. No significant relationships were observed between aerobic fitness and BMD or BMC in the female athletes.

With regard to training histories, there were no significant correlations between years of training or days per week training and any physiological variable. Miles run per week, on the other hand, was significantly related to percent fat (r = −.272, p < .05), LBM (r = −.202, p < .05), BMI (r = −.223, p < .05), triglycerides (r = −.227, p < .05), HDL-C (r = −.267, p < .05), systolic BP (r = −.189, p < .05), resting HR (r = −.266, p < .05), whole-body BMC (r = −.266, p < .05), and spine BMD (r = −.304, p < .05) in men. For women, mileage was significantly related to percent fat (r = −.316, p < .05) and hip BMD (r = .327, p < .05).

**DISCUSSION**

These data are not presented as hypotheses-driven research. Rather, this is an initial description of a relatively large sample of master athletes involved in a longitudinal study designed to determine the influence of chronic exercise on multiple physiologic parameters. Although we recognize the design limitations introduced by the lack of research hypotheses and age-matched controls, we believe the data, as presented, are valuable for several reasons. First, the sample size is significantly larger with greater age and performance variance than previous studies utilizing master athletes, particularly with regard to women. From a statistical standpoint, when describing trends and relationships, larger sample sizes provide greater power to note meaningful results. Second, unlike previous master athlete studies, these athletes do not represent only the physiologic elite. More than one half of the subjects for each gender are considered local or regional standard competitors, perhaps enhancing the generalizability of these data.

There are three specific findings from this study that are unique and require discussion. First, these data suggest that the rate of decline in aerobic capacity is greater than previously reported in master athletes. Second, these data suggest that several of the functional and performance variables used in describing master athletes do not decline in a linear manner. Nonlinear statistical analysis indicates that the conclusions we draw about rates of decline may be dependent upon the age range of the sample we employ. Third, selection bias may be introduced into studies utilizing master athletes to develop an “optimal” aging rate for a given physiological parameter in that the athletes used are homogeneous for body size and type, and their results may not be relevant for inference to the general population.
With regard to aerobic power, these data suggest that active older men will lose maximal aerobic power at a rate of 1.2% per year between the ages of 40 and 80 years. For women, the percentage loss (0.8% per year after the age of 40) is somewhat less. These results differ from those of Proctor and Joyner (15) who compared young (19–31 years) and older athletes (50–70 years) and reported only an approximate 0.55% per year loss for men, less than half the loss rate in the current study. The loss rate they reported for women, 0.74% per year, was similar to our results. Similarly, both Heath and colleagues (8) and Fuchi and colleagues (9) reported declines of 0.48% and 0.59% per year decline in VO2max with age in male master athletes. In support of our findings, Rivera and colleagues (7) suggested a 1.0% per year loss in VO2max between 11 young (mean age 32 years) and 11 old (mean age 66 years) male master runners.

Longitudinal studies also suggest slower declines in VO2max compared with these data. Rogers and colleagues (4) reported a loss rate of 0.55% per year between the ages of 60 and 68 in male master athletes. Similarly, Pollock and colleagues (5) reported no change in VO2max over 10 years in a small group of master athletes who maintained training, although by the 20-year follow-up, loss rates of 1.0% per year in VO2max were reported (6). The minimal functional decline in master athletes as reported by these cross-sectional and longitudinal studies might be related to the limited age ranges employed. For example, these current data would support previous findings of minimal decreases in performance if we examined only the 50-to-70–year cohort. These results may provide support for a statement by Buskirk and Hodgson (2) suggesting that loss rates in sedentary and active persons may be similar due to a curvilinear loss rate in the former. It was suggested that a rapid loss of VO2max occurs during the 20s and 30s followed by a slower rate of decline with advancing age.

Unfortunately there are fewer studies for comparison of female subjects. However, these data suggest that declines in VO2max in female athletes may be lower than in male athletes, approximating 0.8% per year. Interestingly, our re-
The second area of discussion is related to the finding that several of the physiological changes associated with aging are nonlinear. We have reported in this paper that neither muscle strength nor running performance declines linearly with age. The kinetics of strength change that we report are similar to the findings of Metter and colleagues (18) who used quadratic regression to express age and strength changes, whereas the nonlinear change in performance is similar to world record data recorded by the National Masters Track and Field Association. Given this statistical confound, trying to compare the results of older subjects at different age status (old vs oldest old) could be misleading. Furthermore, there is the possibility that age-related loss in other physiologic parameters may not be linear, as suggested by cross-sectional data.

Another important finding of this study relates to athletic somatotype. Jackson and colleagues (19) suggested that changes in aerobic power with age were due to changes in the percentage of fat. Our athletes had very low body fat (particularly the female athletes), low BMI, and reasonably low LBM. This finding may indicate a flaw in trying to use data on athletic populations to describe an ideal aging rate. This body type confound is an extremely important concern when discussing the relationship between exercise capacity and the blood lipid variables (20). Our subjects had exceptionally high levels of high density lipoprotein (HDL) cholesterol (HDL-C), implying a cardio-protective role of exercise. These findings are in agreement with previous results (21,22). However, in that we did not have a comparison group of sedentary, age/weight-matched subjects, it would be difficult to suggest that competitive exercise is more important than heredity and/or body weight in explaining the increased HDL-C. The magnitude of the HDL-C values in the present study, however, makes it difficult to believe that exercise did not have a significant influence.

This athletic somatotype may also confound the relationship between exercise and BMD in master athletes. Several studies have reported that master athletes do not have greater BMD than expected in sedentary age- and height-matched subjects (23,24). Although the present data do not include a sedentary comparison group, they support the previous findings based upon T and Z scores. It is difficult to explain the slight or non-existent benefit of aerobic training on BMD. The most plausible explanation is that most of these athletes did not begin competition until later in life, after the period of peak bone mass attainment. That we do not have exercise histories for these subjects during the time period in which skeletal modification is known to relate to peak bone mass accretion is a limitation of the study.

In conclusion, these data are presented as a cross-sectional comparison of a large group of male and female master athletes. Although limited by lack of a sedentary comparison group, these data suggest that age-related losses in VO\textsubscript{2}max may not be different from data previously reported for older sedentary adults and that loss in muscle strength and performance with aging is not linear. These data highlight the need for longitudinal information to further identify rates of loss with age in physical function and to determine if these loss rates accelerate at advanced ages.

Acknowledgments
This research was supported by the Wadt Memorial Research Fund and the Pickford Foundation. Currently Nora Constantino is at the Department of Health Ecology, University of Nevada, Reno; Kyle Tarpenning is at the Department of Kinesiology, Charles Sturt University, Bathurst, Australia; and Taylor Marcell is at the Department of Internal Medicine, University of Texas, Galveston.

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Received June 30, 2000
Accepted August 3, 2000
Decision Editor: John E. Morley, MB, BCh

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