Physiological determinants of 10-km performance in highly trained female runners of different ages

SUSAN L. EVANS, KEVIN P. DAVY, EDITH T. STEVENSON, AND DOUGLAS R. SEALS
(With the Technical Assistance of Mary Jo Reiling and Heather Silverman)
Departments of Kinesiology and Medicine, University of Colorado, Boulder, Colorado 80309

Evans, Susan L., Kevin P. Davy, Edith T. Stevenson, and Douglas R. Seals. Physiological determinants of 10-km performance in highly trained female runners of different ages. J. Appl. Physiol. 78(5): 1931–1941, 1995.—Endurance exercise performance declines with age; however, there is little information on the mechanisms responsible, especially in women. We tested the hypothesis that this performance decline in women is associated with decreases in maximal 

maximal oxygen consumption; lactate threshold; running economy

AFTER APPROXIMATELY AGE 35, endurance exercise performance declines progressively with advancing age in both men and women (8, 16, 25). However, there is little information regarding the physiological mechanisms responsible for this decline, especially in women. On the basis of studies in young adults, three primary factors are thought to explain the differences in endurance performance among individual athletes (2, 10, 24). It is generally considered that maximal 

oxygen consumption (VO

max) is a primary determinant because it establishes the upper limit of the individual’s ability to produce energy through oxidative pathways. A second factor is the exercise intensity at which blood lactate concentration begins to rise exponentially, referred to as the blood lactate threshold (LT). Exercise intensities above the LT, whether due to an inability to sufficiently augment aerobic energy production or to remove metabolic by-products that negatively affect cellular functioning, are difficult to maintain for extended periods of time. Finally, exercise economy, or the 

O

2 cost of performing submaximal exercise, is thought to partially determine the exercise velocity at which the blood LT occurs.

The modest amount of data available regarding the respective roles of these factors in the age-related decline in endurance exercise performance is primarily on men. Several cross-sectional studies have shown that middle-aged and older male endurance athletes have lower levels of 

VO

2 max than do young adult athletes (19, 20, 22, 29). In one report (19), the rate of decline in endurance-running performance in male athletes aged 30–80 yr was similar to that observed for 

VO

2 max (i.e., both 6–7% per decade). It has also been reported that late middle-aged and older male 10 km runners have a similar LT expressed as % 

VO

2 max compared with highly trained and competitive young runners (2). Stevenson et al. (32) have recently reported that highly trained female distance runners aged 49–67 yr demonstrate levels of 

VO

2 max well above untrained age-matched control subjects but below that reported previously in young elite female runners (25). However, there is no information on the levels of 

VO

2 max in highly trained female runners of different ages who are competing at similar levels with regard to age-group ranking; such data are necessary to determine the potential contribution of this factor to the decline in endurance performance. Moreover, little or no information is available on possible age-related changes in the LT and running economy (RE) (33) in this population.

Accordingly, the purpose of the present study was to determine the respective contributions of 

VO

2 max, LT, and RE to the age-related decline in endurance exercise performance in women. To accomplish this, we measured 

VO

2 max and determined the LT and RE in highly trained adult female 10-km runners over a 33-yr age range. Importantly, although absolute levels of performance declined with age, the runners of different ages were of the same relative level of competitiveness, as documented by their performance in the same local 10 km road race. We hypothesized that the age-related decline in endurance performance in these women would be associated primarily with a reduction in 

VO

2 max and that the LT, expressed as % 

VO

2 max and RE would be well preserved with advancing age in this population.

METHODS

Subjects

Thirty-one female runners between the ages of 23 and 56 yr were studied. All had placed first, second, or third for their year of age in either the 1993 (n = 4) or 1994 (n = 27) Boulder Boulder, the second largest 10-km road race in the United States (35,000 runners). All testing was performed within 2
mo of the 1994 race. The 1993 times were used for subjects who either did not participate in (n = 3) or dropped out of (n = 1) the 1994 race for reasons unrelated to injury. Subjects were chosen from the citizens race winners, which excluded runners who ran in the elite race. All subjects ran within 11% of the average first place time for their year of age over a 3 yr period (1991–1993) of the race.

Subjects were apparently healthy and free of overt cardiovascular disease as assessed by medical history. On the basis of a self-report questionnaire 84% (n = 21) of the premenopausal runners were eumenorrheic (menstruating every 25–36 days). The remaining 16% (n = 4) were considered dysmenorrheic (menstruating less often than every 36 days). Six of the runners were peri- or postmenopausal.

To gain additional insight into the pattern of age-related changes in endurance performance and its physiological determinants, the women also were divided into three groups: group I [30 ± 1 (SE) yr, range 23–35 yr; n = 10], group II (42 ± 1 yr, range 37–47 yr; n = 11), and group III (52 ± 1 yr, range 49–56 yr; n = 10). To establish that the groups were similar with regard to relative competitiveness, the race times of the subjects were compared with age-based 10-km times for both the average (1991–1993) first-place Bolder Boulder and world best (1) 10-km times. The race times were 101 ± 1, 101 + 2, and 101 + 2% of the average first-place Bolder Boulder times for groups I, II, and III, respectively, and 123 + 2, 124 + 2, and 125 + 5% of world best times. The percentage of world best race times was higher for group III than for groups I and II (P < 0.05). The higher than average percentage of our runners compared with world best likely reflect both their noninternational elite status and the fact that the race was performed at an altitude of 1,600 m.

The menstrual status composition of the subgroups was 90 (n = 9) and 82% (n = 9) eumenorrheic in groups I and II, respectively, with the remaining portions dysmenorrheic. In group III, 60% (n = 6) of the runners were either peri- or postmenopausal, with the remaining 40% (n = 4) eumenorrheic. Three of the 21 group I and II runners were taking oral contraceptives, whereas 2 of the 6 perimenopausal or postmenopausal women in group III were receiving hormone replacement therapy.

The nature, purpose, and risks of the study were explained to each subject before written informed consent was obtained. The experimental protocol was approved by the Human Research Committee at the University of Colorado at Boulder.

Measurements

Ten-kilometer performance. Ten-kilometer performance was the time to complete the 1993 or 1994 Bolder Boulder 10-km road race. The 10-km times were converted to running velocity in meters per minute.

Body mass and composition. Total body mass was measured to the nearest 0.1 kg on a physician’s balance scale (Detecto, Webb City, MO). Total body density was determined by hydrodensitometry (6), with residual volume measured by a nitrogen dilution technique (34). Body fat percentage was calculated using the equation of Brozek et al. (6).

VO2 max. On-line computer-assisted open-circuit spirometry was used to determine O2 consumption (VO2), CO2 production, and respiratory exchange ratio. Expired O2 and CO2 gas fractions were measured using a Perkin-Elmer MGA-1100 mass spectrometer (Pomona, CA) calibrated with known gas concentrations. Expired volume was determined using a turbine (VMM-2, Interface Associates, Laguna Niguel, CA) calibrated with a 3 liter syringe before and after each session. Heart rate was determined from an electrocardiographic tracing using a CM5 lead configuration. VO2 max was measured during a continuous incremental protocol on a motorized treadmill (Quinton Instruments, Seattle, WA). Each subject ran at a speed that elicited ~80% of age-predicted maximal heart rate after a 6-min warm-up period. Treadmill grade was increased 2.5% every 2 min until volitional exhaustion. To ensure the attainment of a valid VO2 max, at least three of following four criteria were met by each subject: 1) a plateau in VO2 (<100 ml) with increasing exercise intensity, 2) a respiratory exchange ratio at the end of exercise of at least 1.15, 3) achievement of age-predicted maximal heart rate, and 4) achievement of a rating of perceived exertion of at least 18 (Borg scale, 6–20 units; Ref. 4). Twenty-nine of the 31 runners achieved a plateau in VO2 (criterion 1). Blood was drawn by venipuncture 3 min after cessation of exercise for subsequent measurement of maximal blood lactate concentration.

Because fat-free weight (FFW) can decrease with age at the same absolute level of body weight (18) and has been shown to contribute significantly to age-related declines in VO2 max (18, 26), VO2 max is expressed in both milliliters per kilogram per minute and milliliters per kilogram FFW per minute.

LT. A seven-stage discontinuous treadmill protocol was used for determination of LT. Subjects ran for 7 min, were given 5 min of rest, and then performed 10 min of low-intensity cycle ergometry to facilitate clearance of blood lactate between stages. Running velocity during stage 1 was chosen to elicit ~50% of each subject’s VO2 max. Stages 2 through 7 elicited ~58, 65, 72, 80, 87, and 95% of VO2 max. Treadmill velocity was confirmed for each subject and stage with the use of a calibrated tachometer (Biddle, Plymouth Meeting, PA). Heart rate was monitored with a Polar Vantage XL monitor (Polar CIC, Port Washington, NY). Immediately before stage 1 and at the end of each of the seven stages, a venous blood sample was obtained from an antecubital catheter for subsequent determination of blood lactate concentration. Fifty microliters of whole blood were immediately placed in chilled tubes containing 100 μl of glucose-lactate buffer (model 2357, Yellow Springs Instruments, Yellow Springs, OH), 0.22% Triton X (Sigma Chemical, St. Louis, MO), and 5.0 g/l 100 ml of sodium fluoride (Sigma Chemical). Blood samples were stored at 5°C and later analyzed with a lactate analyzer (model 2300, Yellow Springs Instruments).

LT for each subject was determined by a method that was previously utilized by Coyle et al. (11). The exercise intensity associated with LT was designated in three ways: %VO2 max [as percentage of VO2 max (LTsmax)], running velocity at which LT occurred (LTv), and submaximal VO2 at which LT occurred (LTVO2).

Three investigators, blinded to subject status, determined the LT for five of the subjects, and the principal investigator determined the LT twice per subject. Determinations of LT made by the primary investigator were correlated to those made by each of the secondary investigators, and the mean of these correlation coefficients was calculated for interinvestigator reliability (r = 0.91). To determine intrainvestigator reliability, a correlational analysis was also performed on the two determinations by the primary investigator (r = 0.99). In addition, as determined by analysis of variance (ANOVA), there were no significant differences among any of the determinations of LT (P = 0.93).

RE. RE was determined from the mean of the four 30-s VO2 values obtained in minutes 6 and 7. Two different measures of RE were used: VO2 in milliliters per kilogram per kilometer at each subject’s 10 km race pace (REave), and VO2 in milliliters per kilogram per minute at a common running velocity of 10 km/h (6.2 mph) (REave). Similar approaches have been used previously (2, 7, 9, 12–14, 21).
RESULTS

Physiological Correlates of 10-km Performance in the Overall Study Population

Univariate correlations. Univariate correlation analyses were performed to determine which of the three physiological determinants were most closely associated with 10-km performance (Fig. 1). The variables that were strongly related to 10-km performance were VO\(_{2}\)\(_{\text{max}}\), LT\(_{\text{vel}}\), and LT\(_{\text{Vo2}}\). In contrast, LT\(_{\%\text{max}}\) and RE were not significantly correlated with performance.

Stepwise regression analysis. Stepwise regression analysis was performed to establish which of the physiological determinants accounted for the variance in 10-km performance among the individual runners in the overall study population. LT\(_{\text{vel}}\) appeared first in the analysis and explained most (79%) of the variability in 10-km performance. VO\(_{2}\)\(_{\text{max}}\) (in ml · kg\(^{-1}\) · min\(^{-1}\)) appeared next and explained an additional ~6% of the variability. Thus, together these two variables explained a total of 85% of the variability in 10-km performance in the overall study population.

Changes in 10-km Performance and Its Physiological Determinants with Age in the Overall Study Population

To establish the relationships between age and 10-km performance and its physiological determinants, univariate correlation analyses were performed on the overall study population (Fig. 2). Race performance was strongly inversely related to age (r = −0.83). Of the physiological determinants of performance, VO\(_{2}\)\(_{\text{max}}\), LT\(_{\text{vel}}\), and LT\(_{\text{Vo2}}\) were highly associated with age. LT\(_{\%\text{max}}\) and RE were not significantly related to age.

Relationships Between 10-km Performance and Its Physiological Determinants Across Three Distinct Age Groups

Physical characteristics (Table 1). Mean age differences were 12 and 10 yr between groups I and II, and groups II and III, respectively (all P < 0.05). There were no significant differences, however, among the groups for body weight, height, or percent body fat.

Endurance exercise training (Table 2). There were no significant differences among the three groups for total years of training or weekly training frequency. Moreover, the runners in all three groups reported completing approximately one “speed” or “interval” workout each week. In contrast, compared with group I, weekly training distance tended to be (P = 0.09) or was (P < 0.05) lower in groups II and III, respectively; there was no difference between the latter two groups. Training velocity was not different between groups I and II, but group III trained at a slower pace than either of the younger groups (P < 0.05). When training velocity was expressed as a percentage of 10-km race pace or percentage of LT\(_{\text{vel}}\), however, there were no significant differences among the three groups. Furthermore, the percent of VO\(_{2}\)\(_{\text{max}}\) at which the subjects trained actually increased from group I to group III (P < 0.05).

Ten-kilometer performance and its physiological determinants (Fig. 3). Although there was some overlap between successive groups, average 10-km performance declined progressively from group I to II to III (all P < 0.05). The mean differences were 8 and 12% between groups I and II and groups II and III, respectively.

There was substantial overlap in VO\(_{2}\)\(_{\text{max}}\) between groups I and II; as such, the mean difference between these two groups of runners (5–6% for the two expressions) was only marginally significant (P = 0.05). In contrast to the marked overlap and relatively small mean differences between groups I and II, VO\(_{2}\)\(_{\text{max}}\) was substantially lower in group III compared with the two younger groups. In particular, VO\(_{2}\)\(_{\text{max}}\) (in ml · kg\(^{-1}\) · min\(^{-1}\) and ml · kg FFW\(^{-1}\) · min\(^{-1}\)) was 15–17% lower on average in group III than in group II, and only two subjects in the former group had a value that overlapped with those in the latter group.

Similar to performance, LT\(_{\text{vel}}\) decreased progressively from group I to II to III. The mean differences were 9 and 18% between groups I and II and groups II and III, respectively. In contrast, LT\(_{\text{Vo2}}\) was not different between groups I and II but was lower in group III compared with the two younger groups. LT\(_{\%\text{max}}\) was not different among the three groups; group I, in particular, demonstrated marked interindividual variability in this variable.

RE\(_{\text{RPE}}\) was not different among the groups, although there was considerable interindividual variability, particularly within group III. RE\(_{\text{com}}\) was not different between groups I and II or groups II and III but was lower (i.e., VO\(_{2}\) was higher) in group III than in group I.

Stepwise regression among subgroups. Because both VO\(_{2}\)\(_{\text{max}}\) and LT\(_{\text{vel}}\) were progressively lower from group I to II to III, it was not possible, on the basis of the ANOVA results alone, to assess the relative importance of these two determinants to the declines in 10-km performance across the three age groups. To do so, we performed separate stepwise regression analyses on subjects pooled from groups I and II and from groups II and III. This allowed us to isolate the contributions of the determinants of endurance performance throughout the age range of our sample.

The analysis of the subjects in groups I and II revealed that LT\(_{\text{vel}}\) explained 60% of the variance in 10-km performance, with VO\(_{2}\)\(_{\text{max}}\) (in ml · kg FFW\(^{-1}\) · min\(^{-1}\))...
FIG. 1. Relationship between performance expressed as 10-km race pace and its physiological determinants: maximal O₂ consumption (VO₂ max, A and B), submaximal O₂ consumption (VO₂; running economy: race pace (C) and 10 km/h (D)), and lactate threshold (E–G). FFW, fat-free weight.
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Aging and endurance performance in women

A 10 km Race Pace

\[ r = -0.83 \]
\[ r^2 = 0.69 \]
\[ p = 0.0001 \]

B Lactate Threshold

\[ r = 0.78 \]
\[ r^2 = 0.60 \]
\[ p = 0.0001 \]

C Lactate Threshold

\[ r = -0.33 \]
\[ r^2 = 0.11 \]
\[ p = 0.0688 \]

D Lactate Threshold

\[ r = 0.33 \]
\[ r^2 = 0.11 \]
\[ p = 0.0653 \]

E \( \dot{V}O_2 \text{max} \)

\[ r = -0.74 \]
\[ r^2 = 0.55 \]
\[ p = 0.0001 \]

F \( \dot{V}O_2 \text{max} \)

\[ r = -0.70 \]
\[ r^2 = 0.48 \]
\[ p = 0.0001 \]

G Running Economy: Race Pace

\[ r = 0.10 \]
\[ r^2 = 0.01 \]
\[ p = 0.5911 \]

H Running Economy: 10 km \( \cdot \) hr\(^{-1} \)

\[ r = 0.45 \]
\[ r^2 = 0.20 \]
\[ p = 0.0119 \]

FIG. 2. Relationship between age and 10-km race pace (A) and its physiological determinants: lactate threshold (B–D), \( \dot{V}O_2 \text{max} \) (E and F), and running economy [race pace (G) and 10 km/h (H)]. Note different units of measurement on y-axes.

Contributing an additional 12%, for a total explained variance of 72%. In contrast, \( \dot{V}O_2 \text{max} \) (in ml \( \cdot \) kg\(^{-1} \) \cdot \) min\(^{-1} \)) explained most (74%) of the variance in 10-km performance among the runners in groups II and III, with \( \dot{V}O_2 \text{vel} \) contributing 4%, to explain a total of 78% of the variability.

Physiological responses at \( \dot{V}O_2 \text{max} \), LT, and 10-km race pace. The exercise responses at \( \dot{V}O_2 \text{max} \) for the three subgroups are shown in Table 3. Of particular note, maximal heart rate was on average 5 beats/min lower in group II than in group I, although this was not statistically significant. In contrast, maximal heart
rate was markedly lower (average 13 beats/min; P < 0.05) in group III than in group II. There were no significant differences in respiratory exchange ratio, ratings of perceived exertion, or blood lactate concentrations at \( V_{O2,\text{max}} \) among the three groups.

The responses of the subjects in the three subgroups at the running velocity associated with LT for the three subgroups are shown in Table 4. Of interest, absolute heart rate was progressively lower from group I to II to III. In contrast, there were no significant differences in respiratory exchange ratio, ratings of perceived exertion, blood lactate concentrations, or heart rate expressed as a percentage of maximal heart rate among the three groups.

The responses of the subjects in the three subgroups at the running velocity associated with 10-km race pace are shown in Table 5. Respiratory exchange ratio, ratings of perceived exertion, blood lactate concentrations, percent \( V_{O2,\text{max}} \), and the \( V_O2 \) expressed as percent LT were not significantly different among the three groups. Absolute levels of \( V_O2 \) and heart rate were not different in groups I and II but were significantly lower in group III than in the younger groups. Heart rate expressed as percentage of maximal heart rate was not different from group I to II to III.

**DISCUSSION**

In this sample of highly trained and competitive female 10-km runners aged 23–56 yr, \( V_{O2,\text{max}} \), \( LT_{\text{vel}} \), and \( LT_{V_O2} \) were all strongly related to performance. In turn, advancing age was strongly related with declines in 10-km performance, \( V_{O2,\text{max}} \), and \( LT_{\text{vel}} \) in the overall population. When the subjects were separated into distinct groups, however, the physiological determinants associated with declines in performance with age differed. Specifically, a reduction in \( LT_{\text{vel}} \) appeared to contribute most to the decline in performance from an average age of 30–42 yr, whereas reductions in \( V_{O2,\text{max}} \) appeared to contribute most to the further decline in performance from an average age of 42–52 yr. Neither \( LT_{\%\text{max}} \) nor \( \%LT \) was strongly associated with either 10-km performance or age in this population of female runners.

**Experimental Approach**

In the present study we used a unique experimental design to examine the relationships between aging and endurance exercise performance and its physiological determinants in women. Specifically, we attempted to study runners of increasing age and decreasing absolute 10-km performance times but of the same relative level of competitiveness to minimize, as much as possible, differences in constitutional predisposition for endurance performance. An alternative approach would have been to study young and older runners matched for 10-km performance, as described previously (2). However, with the latter design, the older runners likely would have a superior predisposition for endurance running and would be performing at a relatively higher level than would the young runners (15).

Several lines of evidence indicate that we were successful in executing our experimental plan. First, as expected, absolute levels of performance declined (10-km run time increased) progressively with age in our study population \( (r = 0.83) \). Second, there was a trend for a slightly greater percent decline in performance from group II to III \((-12\%) \) than from group I to II \((-8\%) \), consistent with the progressively greater rate of increase in 10-km race time after age 35 in this population \((8, 16, 25) \). Third, all of the runners finished either first, second, or third in their age year in the nonelite division of the same large national road race; thus, interpretation of performances was not confounded by comparisons of 10-km times from races performed on different courses and geographic locations. Fourth, all subjects' 10-km race times fell within \(-10\% \) of the average first place values for their year of age over the previous 3 yr of the race.

\( V_{O2,\text{max}} \)

Traditionally, \( V_{O2,\text{max}} \) has been considered an important determinant of endurance exercise performance \((3, 9–11, 17, 21) \). It has also been established that \( V_{O2,\text{max}} \) and endurance performance are more highly correlated in populations heterogeneous with respect to \( V_{O2,\text{max}} \) \((9, 25) \).
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FIG. 3. Individual (●) and mean (±SE; ○) age group values for 10-km race pace (A) and its physiological determinants: lactate threshold (B–D), \( \dot{V}O_{2\text{max}} \) (E and F), and running economy [race pace (G) and 10 km/h (H)]. Note different units of measurement on y-axes. *\( P < 0.05 \) vs. group I. **\( P < 0.05 \) vs. group II.

17, 25). The latter likely explains, at least in part, the strong relationship between \( \dot{V}O_{2\text{max}} \) and 10-km performance in the present study population (\( r = 0.81-0.89; \) Fig. 1, A and B).

With regard to its role as a potential mechanism for explaining the decline in endurance performance with aging, there was a tight inverse correlation between \( \dot{V}O_{2\text{max}} \) and age in our overall population of female run-
nners, based on univariate regression analysis ($r = -0.70 - 0.74$; Fig. 2, E and F). Further analysis of the three age groups provided critical insight into the issue of whether the decreases in $\dot{V}O_2\max$ contributed similarly to the declines in performance over specific age intervals within the overall population. At least three lines of evidence indicate that the contributions of decreases in $\dot{V}O_2\max$ to age-related declines in performance were not uniform. First, the magnitude of the difference in $\dot{V}O_2\max$ between groups II and III ($\sim 15\%$) was much greater than that observed between groups I and II ($\sim 5\%$), despite differences in performance that were 12 and 8\%, respectively. Second, close inspection of the individual data in Fig. 2, E and F, indicate little or no change in $\dot{V}O_2\max$ until $\sim 40$ yr of age. Third, stepwise regression analysis indicated that $\dot{V}O_2\max$ explained less of the variability in performance between groups I and II than did $\dot{V}O_2\max$, whereas it explained the greatest amount of the variability in performance between groups II and III. These findings, taken together, suggest that decreases in $\dot{V}O_2\max$ contributed relatively little to the decline in 10-km performance in our female runners from the mid-20s to approximately age 40 but substantially to the decline in performance from approximately age 40 to the mid-50s.

In the present study, the age-associated decline in $\dot{V}O_2\max$ in the overall population was directly related to the decline in maximal heart rate ($r = 0.59$). Moreover, the 5 and 13 beats/min differences in mean levels of maximal heart rate between groups I and II and groups II and III, respectively, correspond roughly to the magnitude of differences in $\dot{V}O_2\max$. As such, the present data suggest that, in this population of female endurance athletes, $\dot{V}O_2\max$ may be limited, at least in part, by the maximal achievable heart rate and, presumably, maximal cardiac output. This idea is consistent with the results of both cross-sectional and longitudinal studies in male endurance athletes (2, 20, 26, 29, 31) showing a close association between age-related differences in the two variables. It should be noted, however, that the present results differ from those of a recent study that reported no differences in maximal heart rate despite age-related reductions in $\dot{V}O_2\max$ in female runners aged 35–70 yr (33).

Recently, Joyner (25) stated that “good (but not world class) runners can run at speeds that require between 79–98\% of $\dot{V}O_2\max$ in 10-km races.” In agreement with this, the female runners in the present study ran 10 km at speeds that elicited 91, 91, and 93\% of $\dot{V}O_2\max$ for groups I, II, and III, respectively. This is also consistent with the data from Allen et al. (2) that showed no differences in the $\%\dot{V}O_2\max$ associated with 10-km race pace in highly trained male young (89\%) and masters (92\%) runners.

### LT

The LT has been shown to be a critical determinant of endurance exercise performance in men (2, 10, 17, 21, 24, 25) and is highly predictive of the $\%\dot{V}O_2\max$ that can be sustained during endurance events (25). Surprisingly, as emphasized recently by Joyner (25), despite the importance of this factor in determining endurance performance in men, there are no previous data on LT in female endurance athletes of any age.

We found a strong direct relationship between both LT$_{vel}$ ($r = 0.89$; Fig. 1E) and LT$_{\dot{V}O_2}$ ($r = 0.87$; Fig. 1F) and 10-km performance in the overall study population. In turn, LT$_{vel}$ was also strongly inversely related to age in this population ($r = -0.78$; Fig. 2B), although

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### TABLE 3. Physiological responses at maximal exercise

<table>
<thead>
<tr>
<th></th>
<th>$\dot{V}O_2$</th>
<th>$\dot{V}O_2$, l/min</th>
<th>$\dot{V}O_2$, ml·kg$^{-1}$·min$^{-1}$</th>
<th>$\dot{H}R$, beats/min</th>
<th>RER</th>
<th>RPE</th>
<th>Lactate, mM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>$57.3 \pm 0.9$</td>
<td>$3.1 \pm 0.1$</td>
<td>$67.9 \pm 1.5$</td>
<td>$183 \pm 4$</td>
<td>$1.22 \pm 0.02$</td>
<td>$19 \pm 1$</td>
<td>$10.1 \pm 0.8$</td>
</tr>
<tr>
<td>(n = 10)</td>
<td>(53.1–61.3)</td>
<td>(2.6–3.7)</td>
<td>(60.6–75.8)</td>
<td>(160–200)</td>
<td>(1.15–1.30)</td>
<td>(17–20)</td>
<td>(6.8–14.7)</td>
</tr>
<tr>
<td>Group II</td>
<td>$54.1 \pm 1.0$</td>
<td>$2.8 \pm 0.1^{*}$</td>
<td>$64.6 \pm 1.3$</td>
<td>$175 \pm 2$</td>
<td>$1.17 \pm 0.01^{*}$</td>
<td>$19 \pm 1$</td>
<td>$8.5 \pm 0.4$</td>
</tr>
<tr>
<td>(n = 11)</td>
<td>(49.7–60.8)</td>
<td>(2.5–3.5)</td>
<td>(58.8–70.4)</td>
<td>(172–187)</td>
<td>(1.12–1.23)</td>
<td>(17–20)</td>
<td>(6.7–10.1)</td>
</tr>
<tr>
<td>Group III</td>
<td>$45.0 \pm 1.4^{++}$</td>
<td>$2.5 \pm 0.1^{++}$</td>
<td>$54.7 \pm 1.8^{++}$</td>
<td>$165 \pm 5^{++}$</td>
<td>$1.19 \pm 0.02$</td>
<td>$19 \pm 1$</td>
<td>$8.6 \pm 0.5$</td>
</tr>
<tr>
<td>(n = 10)</td>
<td>(38.5–51.5)</td>
<td>(2.2–2.6)</td>
<td>(48.5–65.4)</td>
<td>(142–183)</td>
<td>(1.10–1.27)</td>
<td>(17–19)</td>
<td>(6.0–10.5)</td>
</tr>
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</table>

Values are means ± SE; n, no. of women; nos. in parentheses, range. $\dot{V}O_2$, O$_2$ consumption; FFW, estimated fat-free weight; VE, minute ventilation; HR, heart rate; RER, respiratory exchange ratio; RPE, rating of perceived exertion. *P < 0.05 vs. group I. †P < 0.05 vs. group II.

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### TABLE 4. Physiological responses at lactate threshold

<table>
<thead>
<tr>
<th></th>
<th>Velocity, m/min</th>
<th>$\dot{V}O_2$, ml·kg$^{-1}$·min$^{-1}$</th>
<th>$%\dot{V}O_2\max$</th>
<th>$\dot{H}R$, beats/min</th>
<th>$%HR_{max}$</th>
<th>RER</th>
<th>RPE</th>
<th>Lactate, mM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>$241 \pm 6$</td>
<td>$47.6 \pm 1.9$</td>
<td>$85 \pm 9$</td>
<td>$166 \pm 3$</td>
<td>$90 \pm 1$</td>
<td>$0.96 \pm 0.01$</td>
<td>$15 \pm 1$</td>
<td>$2.0 \pm 0.1$</td>
</tr>
<tr>
<td>(n = 10)</td>
<td>(220–290)</td>
<td>(42.7–54.5)</td>
<td>(74–91)</td>
<td>(147–180)</td>
<td>(84–93)</td>
<td>(0.92–1.07)</td>
<td>(13–17)</td>
<td>(1.5–3.1)</td>
</tr>
<tr>
<td>Group II</td>
<td>$220 \pm 4^{*}$</td>
<td>$45.2 \pm 0.6$</td>
<td>$84 \pm 1$</td>
<td>$166 \pm 2^{*}$</td>
<td>$87 \pm 1$</td>
<td>$0.95 \pm 0.01$</td>
<td>$14 \pm 1$</td>
<td>$1.9 \pm 0.1$</td>
</tr>
<tr>
<td>(n = 11)</td>
<td>(202–244)</td>
<td>(41.9–48.3)</td>
<td>(79–89)</td>
<td>(143–163)</td>
<td>(80–93)</td>
<td>(0.93–1.00)</td>
<td>(12–16)</td>
<td>(1.6–2.4)</td>
</tr>
<tr>
<td>Group III</td>
<td>$186 \pm 4^{++}$</td>
<td>$39.0 \pm 1.0^{++}$</td>
<td>$87 \pm 1$</td>
<td>$148 \pm 4^{++}$</td>
<td>$90 \pm 1$</td>
<td>$0.94 \pm 0.01$</td>
<td>$14 \pm 1$</td>
<td>$2.3 \pm 0.2$</td>
</tr>
<tr>
<td>(n = 10)</td>
<td>(171–205)</td>
<td>(35.0–45.1)</td>
<td>(80–93)</td>
<td>(131–163)</td>
<td>(86–95)</td>
<td>(0.89–0.98)</td>
<td>(10–18)</td>
<td>(1.6–3.6)</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of women; nos. in parentheses, range. $\%HR_{max}$, percent maximal HR. *P < 0.05 vs. group I. †P < 0.05 vs. group II.
the correlation with LT\(_{\text{Vo}_2}\) was only modest. When the subjects were divided into distinct age groups, LT\(_{\text{vel}}\) was progressively and significantly lower from group I to II to III (Fig. 3B). Stepwise regression analysis revealed that a decrease in LT\(_{\text{vel}}\) was the primary contributor to the differences in performance among subjects in groups I and II but played a secondary role to a decline in LT\(_{\text{Vo}_2}\) max in explaining the lower performance in the group III compared with the group II female runners. Thus, as stated above for \(V_{O_2}\) max,, it appears that the relative contributions of decreases in LT\(_{\text{vel}}\) to reductions in endurance performance can vary among different periods of aging in female endurance athletes.

Consistent with our results, Allen et al. (2) found that both 10-km performance and LT\(_{\text{vel}}\) were ~20% lower in male masters distance runners (mean age 55 yr, range 50–66 yr) compared with top local young male runners (25 yr, range 21–28 yr). The correlation coefficient from linear regression analysis indicated that differences in LT\(_{\text{vel}}\) accounted for >80% of the variance in 10-km performance. Taken together, the present results and those of Allen et al. indicate that a decrease in LT\(_{\text{vel}}\) is an important factor in the observed decline in 10-km performance between the ages of ~20 and 65 yr in highly trained male and female endurance athletes.

In young male endurance athletes homogeneous with respect to \(V_{O_2}\) max,, LT\(_{\text{smax}}\) is highly predictive of endurance performance (10, 17, 21, 25). No obvious differences have been observed in RE between men and women at the same absolute velocities, but there are only limited data available (25).

In the present study population, RE explained little, if any, additional variance in 10-km performance after differences in LT and \(V_{O_2}\) max were considered. Moreover, there was no relationship between \(R_{\text{Eadj}}\), i.e., our measure of RE adjusted for performance differences, and age (Figs. 2G and 3G). However, \(R_{\text{Ecom}}\) tended to decrease with age (Figs. 2H and 3H). These results suggest that reductions in RE did not play a major role in the age-related decline in 10-km performance in the present population of highly trained and competitive female runners, although the \(O_2\) cost of running at the same absolute submaximal speed may increase slightly with age in female distance runners.

To generate a point of comparison with our \(R_{\text{Eadj}}\) data, we calculated RE in milliliters per kilogram per kilometer from the mean values for \(V_{O_2}\) (in ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\)) and 10-km speed presented in Table 4 of the Allen et al. (2) paper. The mean values for \(R_{\text{E}}\) in their masters and top local young male runners were 214 and 206 ml \cdot kg\(^{-1}\) \cdot km\(^{-1}\), respectively, i.e., a difference of only 3–4%. These results support our conclusion that the \(R_{\text{Eadj}}\) does not change with age in highly trained endurance runners and, therefore, is unlikely to contribute to the age-associated declines in performance.

With regard to \(R_{\text{Ecom}}\), Wells et al. (33) reported no differences in \(V_{O_2}\) (in ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\)) in female runners aged 35–70 yr at a common treadmill running speed of 5.4 km/h and 8% grade. Thus, in their population,
tion of female runners less highly trained and running at a slower speed (but with grade) than in the present study, no trend for an increased O$_2$ cost of running with age was observed.

Last, as pointed out recently by Joyner (25), the RE data from the present and past investigations may or may not accurately reflect the O$_2$ cost of running under actual road racing conditions. Furthermore, it is not known whether the RE data obtained during brief submaximal running trials are representative of those occurring during prolonged exercise under actual racing conditions.

**Endurance Training**

In the present study sample, weekly training distance decreased progressively from the youngest to the oldest groups (Table 2). The absolute running velocity during training was not different between the two youngest groups but was lower in the oldest runners (group III). These data are consistent with previous reports in young groups but was lower in the oldest runners (group III). The absolute running velocity during training was not different between the two youngest groups (Table 2). The absolute running velocity during training decreased progressively from the youngest to the oldest when the subjects' training velocities were expressed relative to their training capacities (Table 2), the intensity of training either was not different among the three age groups (percent 10-km race pace and percent LT$_{vel}$) or was actually greater with increasing age (%Vo$_{2\max}$ max). One interpretation of these data on relative training velocities is that the reductions in absolute training levels are due to the effects of the aging process itself and that the older runners are training at the highest possible percentage of their physical capacity.

**Limitations**

**Cross-sectional design.** The cross-sectional study design employed in the present study may or may not reflect the pattern or magnitude of the changes in 10-km performance and its physiological determinants with advancing age that occurs over time in individual athletes who remain highly trained and competitive. This would obviously necessitate longitudinal study designs. Even though there are such data on Vo$_{2\max}$, there is no information on LT or RE. It is interesting to note, however, that, using a design similar to that employed in the present study, Saltin and Astrand (31) found the same declines in Vo$_{2\max}$ with increasing age in elite orienteers whether studied cross-sectionally or longitudinally.

**Genetic attributes.** Bouchard et al. (5) have shown that genetic differences can contribute significantly to variations in Vo$_{2\max}$ and other factors that influence endurance exercise performance among individuals. Despite our attempt to minimize such potential differences by studying women of the same relative level of competitive performance, we have no way of knowing, for example, whether the youngest and oldest runners in the present study were of similar genetic constitution. Thus, it is possible that some portion of the age-related differences in performance could be explained by such effects. Relatedly, when the overall study population was divided into the three groups, there was a relatively small number of subjects per group. Thus, we cannot discount the possibility of sampling biases or of type II errors under these conditions.

**Motivation.** Our focus on the physiological determinants of performance assumes that other influences such as motivation and voluntary effort during actual competition do not change on average with advancing age. However, age-related decreases in such factors could act to reduce endurance performance without being manifest in our physiological measurements.

"Vo$_2$ steal." One potential contributor to the age-related decline in performance not measured in the present study is an age-associated increase in the work of breathing during running. This increased work of breathing would require the use of a greater percentage of the whole body Vo$_2$ to support oxidative metabolism in the respiratory muscles (23, 27). This Vo$_2$ steal (23) would not affect Vo$_{2\max}$ but could reduce submaximal endurance performance by reducing the portion of the Vo$_2$ that is available for oxidative metabolism in the locomotor muscles. This has been shown to occur in male runners with an average age of ~70 yr (23). However, such age-related changes in respiratory muscle function appear to occur to a lesser extent in women (15) and may or may not have been a factor in the middle-aged female runners studied in the present investigation.

**Altitude.** Because the performance data and physiological measurements were obtained at moderate altitude (~1,600 m or ~5,300 ft), the present results may have been influenced by the effects of the correspondingly reduced partial pressure of O$_2$. However, because all of the subjects lived, trained, and competed at this altitude, it is unlikely that this factor affected the relationships between age and 10-km performance and its physiological determinants.
Conclusions

In conclusion, the findings of the present investigation indicate that decreases in VO2max and the running velocity at LT are the two physiological phenomena most closely associated with decreases in 10-km performance with age in highly trained and competitive female distance runners. However, our results also suggest that the relative contributions of different physiological mechanisms to declines in endurance exercise performance are not necessarily uniform over a wide age range in this population.

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Address for reprint requests: S. L. Evans, Univ. of Colorado, Dept. of Kinesiology, Campus Box 354, Boulder, CO 80309.

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REFERENCES


