Maximal lactate steady state declines during the aging process

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Mattern, Craig O., Margaret J. Gutilla, Darrin L. Bright, Timothy E. Kirby, Kenneth W. Hinchcliff, and Steven T. Devor. Maximal lactate steady state declines during the aging process. J Appl Physiol 95: 2576–2582, 2003. First published September 5, 2003; 10.1152/japplphysiol.00298.2003.—Increased participation of aged individuals in athletics warrants basic research focused on delineating age-related changes in performance variables. On the basis of potential age-related declines in aerobic enzyme activities and a shift in the expression of myosin heavy chain (MHC) isoforms, we hypothesized that maximal lactate steady-state (MLSS) exercise intensity would be altered as a function of age. Three age groups [young athletes (YA), 25.9 ± 1.0 yr, middle-age athletes (MA), 43.2 ± 1.0 yr, and older athletes (OA), 64.6 ± 2.7 yr] of male, competitive cyclists and triathletes matched for training intensity and duration were studied. Subjects performed a maximal O2 consumption (VO2 max) test followed by a series of 30-min exercise trials to determine MLSS. A muscle biopsy of the vastus lateralis was procured during the last 20 min of a 30-min constant-workload test (3), is an increasingly popular performance variable that is frequently used to design training programs for competitive athletes of all ages, including masters athletes. However, we are unaware of any published investigations designed to determine whether the aging process alters the MLSS exercise intensity. On the basis of previous work with respect to the age-related adaptations of skeletal muscle and enzyme activity (9, 11, 37), it is plausible that the aging process may alter the exercise intensity associated with MLSS.

The aging process involves a shift in the expression of myosin heavy chain (MHC) isoforms in favor of type I fibers (9). This age-related MHC shift will likely negatively influence power output at MLSS by lowering the speed of contraction of the working muscle tissue. Considering that type II fibers are the primary producers of lactate and that type I fibers are the main lactate consumers (5–8), the MHC isoform shift may also have a profound influence on lactate metabolism.

In addition to age-related changes in MHC isoform profile, the aging process also has been shown to have an impact on the activity of enzymes involved in energy metabolism.
transduction. Studies of skeletal muscle aerobic enzyme activities suggest that there is an age-related decline in both sedentary (37) and athletic populations (11), whereas there is no concomitant decline in the activity of enzymes of anaerobic metabolism (37). Consequently, it is reasonable to expect that if an older person has a compromised ability to perform work aerobically, that individual will be forced to rely to a greater extent on anaerobic metabolism for energy, yielding a heightened rate of lactate production. Because MLSS represents the equilibrium between rate of appearance in the blood and disappearance from the blood, increased lactate production, without a concomitant increase in lactate clearance, could reduce the MLSS exercise intensity.

On the basis of age-related changes in skeletal muscle MHC isoform profile and aerobic enzyme activities, our hypothesis is that the age-related changes in skeletal muscle composition and enzyme activity will result in a lower power output at MLSS in masters athletes.

**MATERIALS AND METHODS**

**Experimental design.** A cross-sectional study design using groups of young (YA), middle-aged (MA), and older athletes (OA) matched for training intensity and duration was employed to study the effect of age on MLSS. Subjects were asked to report to the laboratory for testing on four to eight separate occasions. The first visit was to collect descriptive data, including height, weight, body composition via skinfold assessement, and maximal O2 consumption (VO2max). The next two to six visits were for the establishment of the MLSS exercise intensity. During the final visit, a biopsy of the vastus lateralis muscle was performed. The experimental design is summarized in the time line depicted in Fig. 1. Although the time line depicts four visits for the establishment of the MLSS exercise intensity, there was an intra-subject range of two to six visits to accurately establish MLSS intensity.

**Subjects.** All experimental protocols were approved by the The Ohio State University Institutional Review Board. Before enrollment, all participants completed informed consent. Three age groups (YA, MA, and OA) of male, competitive cyclists and triathletes were recruited from local cycling and triathlon teams to participate. Power calculations a priori revealed that a minimum of nine subjects per group were required to provide a statistical power of 0.80. Data were collected from 10 subjects per group. One subject in the YA group was determined to be a statistical outlier based on VO2max, so that group in the proposed design functioned as a control group.

In the present study, the YA, MA, and OA groups were matched such that the coefficient of variation between the groups for both training duration (h/week) and intensity relative to maximal heart rate was ±20% (Table 2). To determine appropriate subject matches across the age groups, all subjects wore a downloadable heart rate monitor (model St70, Polar, Kempele, Finland) for all training during the week (days 2–8) between preliminary testing and experimental testing. Similar to Heath et al. (16), training intensity during this week was estimated by relating the heart rate of the subject during their training sessions to the heart rate and O2 consumption (VO2) obtained from the same subject during their VO2max test in the laboratory.

**Preliminary testing.** Body density was estimated via seven-site skinfold measures (24) by using Harpenden calipers, and percent fat was calculated via the Siri equation (32). VO2max testing was accomplished by attaching the rear wheel of each subject’s bicycle to an electronic resistance device (CompuTrainerPro PC1, RacerMate, Seattle, WA). The CompuTrainer provides a measurable, changeable resistance, and the accuracy of this resistance device has been previously confirmed (10). The workload of the VO2max test began at 150 W for the YA and MA groups, and the OA group began at 100 W. For all subjects, the workload increased by 20 W every minute. Electrocardiographic and gas-exchange data were monitored continuously via a metabolic cart-eclectrocardiograph (Medical Graphics, St. Paul, MN) and blood pressure was measured every 3 min to ensure that subjects’ responses to exercise were appropriate.

**MLSS.** To control for differences in nutritional intake between MLSS trials, subjects were required to eat a consistant diet between individual trials and record their diet for 2 days before each MLSS trial. Dietary data were collected before all trials so that dietary intake before the trial determined to be the MLSS exercise intensity could be compared among the three groups (Table 3). Furthermore, all subjects consumed an identical meal (11.0 kcal/kg, 1.76 g/kg of carbohydrate, 0.26 g/kg of fat, and 0.40 g/kg of protein) 6 h before each test day. Finally, subjects were asked to maintain and record their training intensity and duration throughout their involvement in the experiment (Table 4).

The protocol and criteria for achievement of MLSS were similar to those of Beneke (2). For the subjects in the YA and MA groups, the workload for the first test was 60% of the

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**Table 1.** Descriptive characteristics of the subjects

<table>
<thead>
<tr>
<th></th>
<th>YA (n = 9)</th>
<th>MA (n = 9)</th>
<th>OA (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>25.9 ± 1.0</td>
<td>43.2 ± 1.0</td>
<td>64.6 ± 2.7</td>
</tr>
<tr>
<td>Height, cm</td>
<td>178.7 ± 1.9</td>
<td>176.8 ± 2.1</td>
<td>175.7 ± 2.3</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.0 ± 1.8</td>
<td>81.4 ± 4.6</td>
<td>74.5 ± 2.5</td>
</tr>
<tr>
<td>Percent fat, %</td>
<td>8.1 ± 1.1</td>
<td>12.6 ± 1.8</td>
<td>16.5 ± 1.6</td>
</tr>
<tr>
<td>VO2max</td>
<td>67.7 ± 1.2†</td>
<td>56.0 ± 2.6†</td>
<td>47.0 ± 2.6†</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects; YA, young athletes; MA, middle-aged athletes; OA, older athletes; VO2max; maximal O2 consumption. †YA significantly different from MA and OA, P < 0.05. ††Significant differences among all 3 groups, P < 0.05.
exercise intensity

Diet variables before the MLSS

Table 3.

\[
\begin{array}{cccc}
\text{Kilocalories consumed, kcal/day} & 3,107 \pm 220 & 2,510 \pm 224 & 2,914 \pm 218 \\
\text{Carbohydrate consumed, } \% & 58.9 \pm 3.6 & 56.1 \pm 3.1 & 58.1 \pm 1.9 \\
\text{Protein consumed, } \% & 15.4 \pm 0.8 & 18.9 \pm 2.0 & 15.3 \pm 0.6 \\
\text{Fat consumed, } \% & 27.1 \pm 3.2 & 25.1 \pm 3.1 & 26.5 \pm 2.3 \\
\end{array}
\]

Values are means ± SE; n, no. of subjects.

maximal workload of the $\dot{V}O_2_{\text{max}}$ test, and for the OA group subjects the first workload represented 55% of the maximal workload of the $\dot{V}O_2_{\text{max}}$ test. For each subsequent MLSS test, the workload was increased by 5% above the initial value (i.e., 55% of maximal workload followed by 60% of maximal workload, and so forth) for all age groups until no steady state in lactate could be achieved. This technique allows for the detection of a workload that is at or slightly below the MLSS exercise intensity. During each trial, blood lactate was detected at the criterion of a workload that is at or slightly below the MLSS exercise intensity. During each trial, blood lactate was determined at rest and after every 5 min of exercise by using a lactate analyzer (Yellow Springs Instruments, Yellow Springs, OH). Gas-exchange data were collected at minutes 1–4, 14–18, and 26–30 of exercise. The electrocardiogram was monitored continuously. The criterion for achievement of MLSS was no change in lactate of >1 mmol/l in the last 20 min of the 30-min test (29, 33).

**Muscle biopsy.** A muscle biopsy was procured from the distal portion of the left vastus lateralis ~10 cm superior to the most proximal aspect of patella. An incision 1 cm in length was made after the area was anesthetized with 1% lidocaine containing epinephrine. With the knee in the extended position, a muscle biopsy needle 6 mm in diameter and containing epinephrine. The tissue was trimmed of adipose tissue, immediately frozen in liquid N$_2$, and transferred to a −80°C freezer for subsequent analysis. The incision was closed with sterile adhesive strips. Aside from temporary soreness, subjects reported no adverse effects as a result of the muscle biopsy procedure.

**Detection and quantification of isofoms of MHC.** Muscle homogenates from the vastus lateralis biopsy sample were prepared for sodium dodecyl sulfate-polyacrylamide gel electrophoresis by homogenizing −20 mg of muscle in a 1:100 dilution with Tris buffer (62.5 mM, pH 8.8). The total protein concentration of the homogenate was determined via a bicinchoninic acid protein assay such that −6 µg of total protein were loaded into each lane of the gel (13). The stacking gel contained 4% total acrylamide, and the separating gel contained 8%. Both the stacking gel and separating gel contained 0.1 M Tris buffer (0.92 ml), 22.5 mM 5,5'-dithiobis(2 nitrobenzoic acid) (36). Gels were run at 285 mV for 26 h by using a large (22 × 16.5 cm) gel system. After electrophoresis, proteins were fixed in the gel with 12.5% trichloroacetic acid, stained with rapid Cooamassie blue stain, and destained in a solution of 7.5% methanol and 5% acetic acid. Quantification of the relative MHC protein isoform density was determined via Bio-Rad Multi-Analyset version 1.0 software. The relative MHC isoform percentages were transformed by using an arcsine transformation to allow for statistical analysis.

**CS.** CS activity was measured by using previously established procedures (34). In brief, a 15- to 30-mg portion of each muscle biopsy was homogenized in a 5% wt/vol dilution with 0.1 M Tris buffer (0.1% wt/vol Trition X-100 pH 8.35). The homogenate was centrifuged, and 20 µl of the supernatant were combined with 0.1 M Tris buffer (0.92 ml), 22.5 mM acetyl-CoA (20 µl), 5 mM 5,5'-dithiobis(2 nitrobenzoic acid) (20 µl). The background activity of the solution was determined at 30°C via spectrophotometer. Then, 25 mM oxalo-

Table 2. Comparative data on matched subjects

<table>
<thead>
<tr>
<th>Duration, min/wk</th>
<th>YA (n = 9)</th>
<th>MA (n = 9)</th>
<th>OA (n = 9)</th>
<th>Means ± SE</th>
<th>CV, %</th>
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</thead>
<tbody>
<tr>
<td>Intensity, %MHR</td>
<td>74</td>
<td>89</td>
<td>76</td>
<td>79.7 ± 4.7</td>
<td>10.2</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>397</td>
<td>375</td>
<td>303</td>
<td>358.3 ± 28.4</td>
<td>13.7</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>86</td>
<td>71</td>
<td>77</td>
<td>78.0 ± 4.3</td>
<td>9.7</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>540</td>
<td>383</td>
<td>405</td>
<td>442.7 ± 49.1</td>
<td>19.2</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>72</td>
<td>76</td>
<td>75</td>
<td>74.3 ± 1.2</td>
<td>2.8</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>552</td>
<td>399</td>
<td>412</td>
<td>454.3 ± 49.0</td>
<td>18.7</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>73</td>
<td>79</td>
<td>81</td>
<td>77.7 ± 2.4</td>
<td>5.4</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>568</td>
<td>483</td>
<td>485</td>
<td>512.0 ± 28.0</td>
<td>9.5</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>73</td>
<td>76</td>
<td>82</td>
<td>77.0 ± 2.7</td>
<td>6.0</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>641</td>
<td>713</td>
<td>690</td>
<td>681.3 ± 21.3</td>
<td>5.4</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>71</td>
<td>75</td>
<td>76</td>
<td>74.0 ± 1.5</td>
<td>3.6</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>644</td>
<td>697</td>
<td>726</td>
<td>689.0 ± 24.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>63</td>
<td>70</td>
<td>77</td>
<td>70.0 ± 4.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>649</td>
<td>897</td>
<td>748</td>
<td>764.7 ± 72.1</td>
<td>16.3</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>75</td>
<td>72</td>
<td>71</td>
<td>72.7 ± 1.2</td>
<td>2.9</td>
</tr>
<tr>
<td>Intensity, %MHR</td>
<td>900</td>
<td>973</td>
<td>837</td>
<td>903.3 ± 39.3</td>
<td>7.5</td>
</tr>
<tr>
<td>Group duration, min/week</td>
<td>572.1 ± 59.4</td>
<td>581.7 ± 81.6</td>
<td>544.7 ± 68.9</td>
<td></td>
<td></td>
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</table>

% MHR, percentage of maximal heart rate; CV, coefficient of variation.

Table 3. Diet variables before the MLSS exercise intensity

Table 4. Training variables for first and last weeks of participation

<table>
<thead>
<tr>
<th>First Week (n = 27)</th>
<th>Last Week (n = 27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Training volume, min/wk</td>
<td>472.8 ± 53.9</td>
</tr>
<tr>
<td>Training intensity, RPE</td>
<td>13.4 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. RPE, rating of perceived exertion.
Acetate (20 µl) was added, and a kinetic assay was performed with data taken every 2.5 s for a total of four cycles.

Statistical analyses. Comparisons of the dependent variables among age groups were made by using an ANOVA. When differences were detected, a Newman-Keuls post hoc test was used to determine where the differences occurred. In addition, multiple regression analysis was performed to determine the ability to predict MLSS exercise intensity. The α-level was set a priori at \( P < 0.05 \).

RESULTS

Descriptive variables. Descriptive data are presented in Table 1. \( V_\text{O}_2 \text{max} \) declined with increasing age such that there were significant \( (P < 0.05) \) differences among all three age groups. The YA group had significantly less body fat than the MA and OA groups.

Measurements at MLSS exercise intensity. Dietary data before the MLSS exercise intensity are presented in Table 3. There were no significant \( (P > 0.05) \) differences in kilocalories per day, percent carbohydrates, percent protein, or percent fat consumed among age groups. In addition, there were no significant \( (P > 0.05) \) differences in training volume or training intensity between the first week of involvement in the study and the last week of participation (Table 4).

Power output data at MLSS exercise intensity are presented in Fig. 2. Absolute power output at MLSS exercise intensity of the OA group was significantly \( (P < 0.05) \) lower than the YA and MA groups. When data were normalized for body mass, all three groups were significantly \( (P < 0.05) \) different from each other.

\( V_\text{O}_2 \) data at MLSS exercise intensity are provided in Fig. 3. There was an age-related decrease such that significant \( (P < 0.05) \) differences were evident among all three age groups. However, this age-related change could, in part, be driven by the age-related decline in \( V_\text{O}_2 \text{max} \). Consequently, to determine whether there were any age-related differences in MLSS exercise intensity exclusive from changes in \( V_\text{O}_2 \text{max} \), comparisons were made for the percentage of \( V_\text{O}_2 \text{max} \) at MLSS exercise intensity, which also revealed significant \( (P < 0.05) \) differences among all three groups. The decline in MLSS exercise intensity relative to \( V_\text{O}_2 \text{max} \) occurred at a rate of ~3.3% per decade (Fig. 4). There were no differences among age groups for blood lactate concentrations at MLSS exercise intensity (Fig. 5).

Fig. 2. Power output at maximal lactate steady-state intensity. A: absolute power output. B: power output normalized for body mass. YA, young athletes; MA, middle-age athletes; OA, older athletes. Values are means ± SE; \( n = 27 \) subjects. *OA significantly different from YA and MA, \( P < 0.05 \). #Significant differences among all 3 groups, \( P < 0.05 \).

Fig. 3. Oxygen consumption (\( V_\text{O}_2 \)) at maximal lactate steady-state exercise intensity (A) and maximal lactate steady-state exercise intensity expressed as a percentage of \( V_\text{O}_2 \text{max} \) (B). Values are means ± SE; \( n = 27 \) subjects. #Significant differences among all 3 groups, \( P < 0.05 \).

Fig. 4. Relationship between age and maximal lactate steady-state exercise intensity. \( n = 27 \) Subjects.

\( r = -0.68 \) 
\( P<0.05 \)
Skeletal muscle properties. There were no differences detected in citrate synthase activity among any of the three age groups (Fig. 6).

There were no differences in the percentage of type I, type IIA, or type IIX MHC isoforms among any of the three age groups (Fig. 7). However, multiple regression analysis revealed that the percent of type I MHC isoform significantly contributed to the model used to predict MLSS exercise intensity expressed as a percentage of \( V\dot{O}_2 \text{max} \), such that age and percent type I MHC isoform combined accounted for 58% of the variance (\( r = 0.76 \)).

DISCUSSION

MLSS is the highest maintainable blood lactate concentration during constant workload, and it represents the equilibrium between lactate production and lactate clearance (3). The results of this investigation establish that both absolute and relative MLSS exercise intensity decrease with age in highly trained endurance athletes, as evidenced by an age-related reduction in both absolute power output and \( V\dot{O}_2 \) at MLSS intensity as well as a decrease in the \( V\dot{O}_2 \) relative to \( V\dot{O}_2 \text{max} \) at MLSS intensity. The age-related decrease in \( V\dot{O}_2 \text{max} \) observed in this investigation has been corroborated by a multitude of studies (1, 16, 22, 23, 28, 30). Therefore, it is not unexpected that as athletes age they are capable of producing less absolute power and consequently experience a compromised exercise performance for a given training load. However, our data suggest that the reduction in performance is not solely due to a decrease in \( V\dot{O}_2 \text{max} \) but that it is also a consequence of a compromised MLSS exercise intensity.

MLSS data from the YA group are comparable to the previous literature. For example, in young male cyclists, MLSS occurred at 80.2% of \( V\dot{O}_2 \text{max} \) (35) and at a lactate concentration of \( \sim 5.5 \) mmol/l (3, 35), and our values of 80.8% and 4.1 mmol/l, respectively, are in good agreement. In addition, in young well-trained male subjects, power output at MLSS has been shown to be \( \sim 240 \) W (12), which is similar to our finding of 253.4 W. There are no previous data related to MLSS in older individuals to allow for comparisons. However, data are available regarding age-related changes in lactate threshold.

When studying the aging process of athletes in a cross-sectional design, careful and deliberate attention is required when matching subjects in the various age groups. To study the effects of aging per se on performance utilizing a cross-sectional matching design, young athletes should be compared with their older counterparts, with careful attention paid to the performance measurements of the older subjects when they were young (14). Logistically, however, this type of
cross-sectional matching paradigm is not feasible, because of the athletes’ reliance on their recollection of performance results from several decades prior.

Matching for current training intensity and duration across age ranges yields a practical experimental design that allows for systematic control of training intensity and duration. Therefore, the results garnered from this type of design can be interpreted to reflect true age-related adaptations. Without control of training regimens, experimental differences between age groups could be the result of the aging process and/or variation in training practices.

In the present study, the YA, MA, and OA groups were matched for both training duration and intensity. However, when older and younger runners were matched for performance, training intensity, and training duration, the older runners demonstrated an identical V̇O₂ at lactate threshold and a higher lactate threshold relative to their V̇O₂ max (1). Although not directly comparable because of methodological differences between measurement of lactate threshold and MLSS, our data do not agree with these previous findings. This may be because the average age of the older subjects in the study of Allen et al. (1) was 56.0 yr, whereas it was 64.4 yr for the OA group in our investigation. In addition, our subjects were not matched for performance but rather only training intensity and duration such that aging per se could be studied.

Iwaoka et al. (23) matched subjects for training distance and percent body fat and determined that the V̇O₂ at the onset of blood lactate accumulation of the older athletes was lower than that of young athletes and that there were no differences relative to V̇O₂ max. The discrepancy with regard to measures relative to V̇O₂ max between the findings of Iwaoka et al. and the present data is likely attributable to the lack of matching of subjects for training intensity in the investigation performed by Iwaoka et al. Consequently if their older subjects were training at a higher intensity than their younger counterparts, an age-related change in lactate threshold could have been masked.

We hypothesized that MLSS would be different in our three different age groups of athletes based on 1) a previously observed reduction in citrate synthase activity (11, 37) and 2) an age-related MHC isoform shift in favor of type I fibers (9). However, in our highly aerobically trained subjects, we were unable to detect an age-related decline in citrate synthase activity or an alteration in MHC isoform profile. It is possible that the aerobic conditioning of these subjects afforded them protection against these previously observed age-related changes.

We did observe that age and type I MHC isoform combined were able to account for 58% of the variance in MLSS exercise intensity expressed as a percentage of V̇O₂ max. Therefore, type I MHC isoform is helpful in predicting MLSS intensity; however, because it was unaffected by age, it is not the mechanism responsible for the observed age-related decline in MLSS intensity.

In this investigation, we were unable to identify the mechanism(s) responsible for the reduced MLSS intensity of the older subjects. Therefore, future investigations on this topic are necessary to elucidate the mechanism(s). Because MLSS represents an equilibrium between lactate production and lactate clearance, any possible age-related reduction in lactate clearance could explain a reduced MLSS exercise intensity. Two potential sites for an age-related reduction in lactate clearance are the gluconeogenic pathway and lactate transporters. In animals, age is inversely correlated with the rate of gluconeogenesis (4, 21, 25–27). In addition, when older animals are trained, it appears that some of the loss in gluconeogenic function is recovered; however, training does not afford complete recovery to the level of function of a young animal. Age-related compromises in the ability of the liver to clear lactate via gluconeogenesis could be partially responsible for the observed reduction in MLSS exercise intensity.

Investigations of lactate transporters are in their infancy, and potential effects of age on these monocarboxylate transporters have yet to be studied. However, if age attenuates the function of monocarboxylate transporters, then transport of lactate out of working skeletal muscle and into nonworking skeletal muscle and heart tissue could be compromised, thereby reducing lactate clearance.

It has also been suggested that lactate threshold, and consequently MLSS intensity, is determined by peripheral factors such as mitochondrial oxidative capacity (18), capilirization (1) and oxidative enzyme activities (1). Of particular importance is the observation that capillary measurements based on muscle fiber perimeter is thought to be a better index of O₂ flux as opposed to measurements in reference to fiber area (19, 20). We are unaware of any investigations that have measured the capillary-to-fiber perimeter exchange index in active individuals of varying age.

Therefore, it is important to establish the relationship of capilirization and mitochondrial capacity to MLSS intensity. If the mechanism responsible for the age-related decline in MLSS can be isolated, then it is conceivable that training-oriented countermeasures can be developed such that masters athletes may attenuate the decline observed in MLSS.

In conclusion, we have demonstrated that MLSS exercise intensity decreases with age and that this reduction is independent of the previously observed reduction in V̇O₂ max. Type I MHC isoform is helpful in predicting MLSS exercise intensity; however, it is not the parameter responsible for the age-related decline. The mechanism(s) responsible for the observed age-related decline in MLSS is unknown.

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**DISCLOSURES**

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REFERENCES


