Resistance and aerobic training in older men: effects on $V\dot{O}_2$peak and the capillary supply to skeletal muscle

HEPPLE, R. T., S. L. M. MACKINNON, J. M. GOODMAN, S. G. THOMAS, AND M. J. PLYLEY.
Department of Physiology, Graduate Department of Community Health, and Department of Physical Therapy, University of Toronto, Toronto, Ontario, Canada M5S 3J7

Resistance and aerobic training in older men: effects on $V\dot{O}_2$peak and the capillary supply to skeletal muscle. J. Appl. Physiol. 82(4):1305–1310, 1997.—Both aerobic training (AT) and resistance training (RT) may increase aerobic power ($V\dot{O}_2$peak) in the older population; however, the role of changes in the capillary supply in this response has not been evaluated. Twenty healthy men (age 65–74 yr) engaged in either 9 wk of lower body RT followed by 9 wk of AT on a cycle ergometer (RT—AT group) or 18 wk of AT on a cycle ergometer (AT—AT group). RT was performed three times per week and consisted of three sets of four exercises at 6–12 repetitions maximum. AT was performed three times per week for 30 min at 60–70% heart rate reserve. $V\dot{O}_2$peak was increased after both RT and AT ($P < 0.05$). Biopsies (vastus lateralis) revealed that the number of capillaries per fiber perimeter length was increased after both AT and RT ($P < 0.05$), paralleling the changes in $V\dot{O}_2$peak. However, capillary density was increased only after AT ($P < 0.01$). These results, and the finding of a significant correlation between the change in capillary supply and $V\dot{O}_2$peak ($r = 0.52$), suggest the possibility that similar mechanisms may be involved in the increase of $V\dot{O}_2$peak after high-intensity RT and AT in the older population.

capillaries; aerobic power; aging; oxygen flux

The hypothesis that the capillary supply to skeletal muscle may play a vital role in determining aerobic metabolic function is supported by work with animal models (22), young athletic humans (17), and investigations suggesting that muscle diffusing capacity limits maximal oxygen consumption ($V\dot{O}_2$) (27). In this respect, aging and inactivity have been found to result in a reduction of the capillary supply to skeletal muscle (3) and a reduction in whole body aerobic power ($V\dot{O}_2$peak) (31), whereas aerobic training (AT) of sedentary older persons is associated with an increase in both $V\dot{O}_2$peak and the capillary supply (4). Because high-intensity resistance training (RT) has also been suggested to increase the $V\dot{O}_2$peak in this population (8), an examination of the role of the capillary changes, relative to the increases in $V\dot{O}_2$peak observed with both types of training, would seem to be warranted. Consistent with this line of reasoning we recently demonstrated that RT in older men is associated not only with an increased $V\dot{O}_2$peak but also with an increase in the capillary supply to the skeletal muscle fibers (13). Furthermore, given the significance that has been ascribed to the changes in muscle function with aging, it is possible that there may be a synergistic effect of a combined RT and AT program. To examine these issues, we compared the effects of a sequential RT and AT program with those of an exclusively AT program in a group of healthy older men, focusing on the changes in $V\dot{O}_2$peak and changes in muscle capillary supply.

Methods

Subjects. Healthy older men (age 65–74 yr) were recruited through newspaper advertisement. All potential candidates were thoroughly screened by using a combination of the Physical Activity Readiness Questionnaire (the PARQ), resting and submaximal exercise electrocardiograph (ECG), and blood pressure measurements. Individuals demonstrating contraindications for exercise (i.e., positive PARQ, abnormal ECG or blood pressure response, or musculoskeletal impairment) were excluded from the study. From the screening we obtained a sample of 20 male subjects (age 68.3 ± 1.1 (SD) yr). All individuals were unmedicated, normotensive (systolic blood pressure ≤ 150 Torr; diastolic blood pressure ≤ 90 Torr), and had been nonsmokers for at least 10 yr before beginning the study. While none of the subjects participated in regimented physical training before starting the study, most engaged in periodic low-intensity physical activity consisting of golf, tennis, and/or walking two or fewer times per week. All subjects were informed of the procedures, risks, and benefits, and they gave written consent for participation in the study.

After screening, the subjects were randomly allocated to one of two training groups (10 subjects per group). The first group (RT—AT) underwent 9 wk of RT designed to increase the muscle strength of the lower body, followed by 9 wk of AT (programs described in RT and AT). The second group (AT—AT) underwent two consecutive 9-wk periods of AT (i.e. 18 wk total).

RT. Each subject in the RT—AT group participated in a RT program three times per week for 9 wk. Each training session consisted of a warm-up and cool-down series of stretching exercises and three sets of four resistance exercises, performed on each leg separately and at an intensity regularly adjusted to elicit fatigue within 6–12 repetitions (i.e., 6–12 repetitions maximum) on Universal weight machines. On the basis of the reported effect of circuit-types of RT as an AT stimulus in a young adult population (25), a minimum of 2 min of recovery were taken between each exercise to prevent a circuit-training benefit.

AT. The subjects participated in AT on a cycle ergometer, for 30 min, three times per week, for either 9 (RT—AT group) or 18 wk (AT—AT group). The exercise intensity was determined by using the modified Karvonen formula (18). Heart rate was measured at 5-min intervals with the aid of heart rate monitors (Polar), and logs were maintained to monitor subject compliance and progress.

$V\dot{O}_2$peak. The $V\dot{O}_2$peak was assessed before training was begun (T1), after 9 wk of training (T2), and after 18 wk of training (T3) on an electrically braked cycle ergometer (Collins Pedalmate) fitted with toe clips, by using an incremental protocol to voluntary exhaustion under the supervision of a physician. Subjects were given a 5-min warm-up at 50 W, followed by 5 min at 100 W. After a brief recovery of 5 min, individuals were brought to maximum effort by using step increases in power output, beginning at 75 W (2 min) and progressing to 100 W (2 min), with subsequent power output...
increments of 16.7 W/min to fatigue. The criteria used for acceptance of VO_{2peak} values as a maximum included two or more of 1) a heart rate greater than or equal to age-predicted maximum (≥10 beats/min), 2) a respiratory exchange ratio (RER = CO_{2} production/VO_{2}) ≥1.10, or 3) a change in VO_{2} ≤0.5 ml with an increase in power output (i.e., one-half of the predicted rise in VO_{2} with an increase in power output). Heart rate was monitored continuously from a V5 tracing displayed on a built-in oscilloscope of the ECG-defibrillator (Physiocontrol, Lifepak 9P). Blood pressure was monitored by using an automated inflation system (Dynamap vital signs monitor, model 1846 SX), and gas analysis was performed with a metabolic cart (Morgan) on-line with a microcomputer. Ventilatory volumes were assessed with a ventilation monitor (Morgan Ventilometer Mark 2) connected to a pneumotachograph on the inspiratory arm of the mouthpiece.Expired gases were sampled and analyzed via an infrared CO_{2} monitor (Jaeger CO_{2} Test) and an O_{2} analyzer (Ametek S3-A), which utilized a stabilized zirconia cell heated to 750°C.

Muscle biopsy. Percutaneous needle biopsies of the vastus lateralis of the dominant leg were performed before the training program was begun according to the method of Bergstrom (2), as adapted by Mubarak et al. (24). Samples were obtained midway between the iliac crest and the upper border of the patella, at a depth of 2–3 cm. The subsequent samples (i.e., after training: T2 and T3) were taken at a distance of 2 cm from the original incision and at the same depth to minimize variation due to muscle inhomogeneities (13). Local anesthetic (2% Xylocaine) was administered to the subject before the incision. Muscle samples were mounted in cross section in an embedding medium (OCT), immersed in liquid isopentane cooled in liquid nitrogen, and stored at −80°C for subsequent histochemical analysis.

Histochemical analysis. Specimens were sectioned to a thickness of 10 µm on a cryostat, mounted on albumin-coated slides, and kept at −20°C until fixation. Histochemical processing was done within 1 wk of sectioning. The sections were first fixed for 5 min in a Guth and Samaha (10) fixative at room temperature and then incubated for 1 h at 36°C in a Pb-adenosinetriphosphatase staining medium to simultaneously stain for both fiber types and capillaries (28). No subtypes of the type II fiber population are revealed with this method in human tissue.

Morphometry. Muscle sections were viewed under a light microscope, on-line with a microcomputer and an image-analysis system (Mocha, Jandel Scientific). Capillaries were quantified manually from the microscope on each fiber to estimate the following indexes: 1) the number of capillaries around a fiber (capillary contacts (CC)), 2) the capillary-to-fiber ratio on an individual-fiber basis (C/F_{i}), and 3) the number of fibers sharing each capillary (sharing factor (SF); Ref. 26; Fig. 1). Quantitation of the capillary supply was performed on 25 fibers of each type by randomly selecting a fiber in an artifact-free region (free of connective tissue and demonstrating a uniform staining intensity among fibers of a given type) and counting the closest 25 neighboring fibers of each type (4). Fiber area (FA) and perimeter (P) were measured with the image-analysis system and commercial software (Mocha), calibrated to transform the number of pixels (viewed on a computer monitor) into micrometers. Fiber type distributions were determined by counting all of the fibers in a section [315 ± 26 (SE) muscle fibers; range 220–504 fibers]. All quantitative analyses were performed blind by a single observer.

To examine the potential for blood-tissue exchange, the capillary density (CD) and the capillary-to-fiber perimeter exchange (CFPE) index (12) were calculated. The CD was calculated by using the fiber as the reference space, as described previously (6). The CFPE index was used to obtain an index of the size of the capillary-to-fiber interface and was determined from the following equation

\[ CFPE = \frac{C/F}{P} \]

By relating the capillary supply to the fiber P (which is proportional to the 3-dimensional surface area of the fiber) rather than to the FA (which is proportional to the volume of the fiber), the CFPE index allows quantitation of the capillary supply relative to the region of greatest resistance to oxygen flux, namely the capillary-to-fiber surface. The effects of capillary tortuosity and sarcomere length on the CFPE index are considered elsewhere (12).

Data analysis. Global measures for FA, P, CC, SF, C/F_{i}, CD, and the CFPE index were determined from the mean values of the fiber type specific data from each individual by multiplying the values for type I and type II fibers by their respective fiber type proportion and adding the results together to produce the global measure for that individual as demonstrated by the following expression for FA: [\text{FA}_{\text{type I}} \times \text{FA}_{\text{type II}}] + [\text{FA}_{\text{type I}} \times \text{FA}_{\text{type II}}] = \text{global FA}, where \text{FA}_{\text{type I}} and \text{FA}_{\text{type II}} are the FAs of type I and II fibers, respectively. The day-to-day variability in the FA and P measurements was <0.05% (coefficient of variation), while the variability in the capillary measures (i.e., CC, SF, C/F_{i}, CD, and CFPE index) ranged from 4.8 to 5.4% in this study.

Data are expressed as group means ± SE. Statistical comparisons were performed by analysis of variance for repeated measures and by linear and multiple-regression analyses, by using the methods of Donner and Cunningham (5) to adjust the SE of the slope parameter for regression analyses on repeated measures designs. The P value chosen to determine significance was set at 0.05.

RESULTS

\[ \dot{V}O_{2peak} \]. \dot{V}O_{2peak} data are based on 10 subjects in the RT → AT group at each time point while the AT → AT group \dot{V}O_{2peak} data are based on 10 subjects at each of T1 and T2 and on 9 subjects at T3 (1 subject dropped out after the T2 assessment). The RT → AT group demonstrated significant increases in the \dot{V}O_{2peak} after both RT and AT (P < 0.05; Table 1). Body mass did not
biopsy procedures at all three time points in the
RT→AT group, the data in the AT→AT group are based
on seven subjects at the T1 assessment, five at T2 (2
samples had insufficient tissue for analysis), and seven
at T3. The fiber type distribution did not differ signifi-
cantly across time points, exhibiting a coefficient of
variation of 9.1 ± 2.2% from T1 to T3. The percentage
of type I fibers was 59 ± 2% for the RT→AT group and
57 ± 6% for the AT→AT group (average of all 3 time
points).

The T1 to T2 data for the RT→AT group have been
presented previously (13) and are included here for com-
parison. Briefly, there were significant increases in
both the cross-sectional area and P (P < 0.05) of the
muscle fibers after RT (i.e., T1 to T2). These altera-
tions in fiber size were accompanied by a significant in-
crease in CC (P < 0.05) and the C/F, (P < 0.05) and no change
in the SF. Whereas there was no change in CD after the
RT period, the CFPE index was significantly increased
(indicative of a larger capillary-to-fiber surface; P < 0.05).

After AT in the RT→AT group (i.e., from T2 to T3),
there were significant reductions in both the cross-
sectional area (P < 0.05) and P (P < 0.05) of the muscle
fibers, which returned to values that were not signifi-
cantly different from those at T1. The reduction of the
fiber size after AT was associated with no further
change in CC or in the C/F, beyond the values that
were observed after RT. As a result, there was a
change throughout the study in this group, and thus
the pattern of the alterations in mass-specific VO₂peak
did not differ from that observed for the absolute
measures. There was also an increase in the peak
power output after both phases of training in the
RT→AT group (P < 0.05), but neither peak heart rate
nor RER measured at exhaustion was found to change
with training.

The AT→AT group also demonstrated a significant in-
crease in the VO₂peak after the first 9 wk of AT (P < 0.05)
and a small, not statistically significant, increase in the
second 9-wk period of AT. However, because of the
decrease of body mass from T1 to T3 in this group,
the mass-specific VO₂peak was significantly increased
after both 9 and 18 wk of training (P < 0.05) in this
group. There was also a significant increase in the peak
workload attained at exhaustion after the first 9 wk of
AT (P < 0.01). No further increase in the peak workload
was observed in the second 9 wk of training, consistent
with the data for absolute VO₂peak. As was also observed
in the RT→AT group, there were no significant differ-
ces in the peak heart rate or the peak RER attained
at exhaustion. Due, in part, to considerable interindi-
vidual variation in the training response, the magni-
tude of the improvement in absolute VO₂peak was not
significantly different between groups at either T2 or T3.

Muscle structure. The morphometric data are pre-
sented in Table 2. Whereas 9 subjects consented to the

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### Table 1. Cycle aerobic power assessment

<table>
<thead>
<tr>
<th></th>
<th>RT→AT Group</th>
<th>AT→AT Group</th>
</tr>
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<tbody>
<tr>
<td>Age, yr</td>
<td>68.3 ± 1.1</td>
<td>68.3 ± 1.0</td>
</tr>
<tr>
<td>Mass, kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>77.1 ± 3.1</td>
<td>85.1 ± 3.9</td>
</tr>
<tr>
<td>T2</td>
<td>77.0 ± 3.1</td>
<td>84.6 ± 3.7</td>
</tr>
<tr>
<td>T3</td>
<td>76.2 ± 2.8</td>
<td>81.0 ± 2.6*</td>
</tr>
<tr>
<td>VO₂peak, l/min</td>
<td></td>
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</tr>
<tr>
<td>T1</td>
<td>2.13 ± 0.13</td>
<td>2.15 ± 0.14</td>
</tr>
<tr>
<td>T2</td>
<td>2.31 ± 0.12*</td>
<td>2.46 ± 0.12‡</td>
</tr>
<tr>
<td>T3</td>
<td>2.48 ± 0.13‡</td>
<td>2.51 ± 0.10‡</td>
</tr>
<tr>
<td>VO₂peak, ml·min⁻¹·kg⁻¹</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>27.7 ± 1.4</td>
<td>25.3 ± 1.3</td>
</tr>
<tr>
<td>T2</td>
<td>30.1 ± 1.2*</td>
<td>29.3 ± 1.2‡</td>
</tr>
<tr>
<td>T3</td>
<td>32.6 ± 1.4‡</td>
<td>31.0 ± 1.1‡</td>
</tr>
<tr>
<td>WLpeak, W</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>163 ± 7</td>
<td>167 ± 9</td>
</tr>
<tr>
<td>T2</td>
<td>174 ± 10*</td>
<td>186 ± 11†</td>
</tr>
<tr>
<td>T3</td>
<td>189 ± 9†</td>
<td>189 ± 12‡</td>
</tr>
<tr>
<td>HRpeak, beats/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>152 ± 3</td>
<td>154 ± 3</td>
</tr>
<tr>
<td>T2</td>
<td>150 ± 4</td>
<td>153 ± 3</td>
</tr>
<tr>
<td>T3</td>
<td>156 ± 3</td>
<td>153 ± 3</td>
</tr>
<tr>
<td>RER</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>1.13 ± 0.02</td>
<td>1.12 ± 0.02</td>
</tr>
<tr>
<td>T2</td>
<td>1.11 ± 0.04</td>
<td>1.15 ± 0.02</td>
</tr>
<tr>
<td>T3</td>
<td>1.13 ± 0.02</td>
<td>1.15 ± 0.02</td>
</tr>
</tbody>
</table>

Values are means ± SE. T1, before beginning of training; T2, after
9 wk of training; T3, after 18 wk of training; VO₂peak, aerobic power;
WLpeak, peak power output; HRpeak, peak heart rate; RER, respira-
tory exchange ratio. RT→AT group performed 9 weeks of resistance
training (RT) from T1 to T2 and 9 wk of aerobic training (AT) from T2
to T3 while AT→AT group performed 2 consecutive 9-wk periods of
AT (18 wk total from T1 to T3). *P < 0.05 vs. T1 measurements. †P <
0.05 vs. T2 measurements. ‡P < 0.01 vs. T1 measurements.

### Table 2. Muscle morphometric data obtained from the vastus lateralis

<table>
<thead>
<tr>
<th></th>
<th>RT→AT Group</th>
<th>AT→AT Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fiber area, µm²</td>
<td>3.874 ± 314</td>
<td>4.932 ± 434</td>
</tr>
<tr>
<td>T1</td>
<td>4.916 ± 309*</td>
<td>4.653 ± 733</td>
</tr>
<tr>
<td>T2</td>
<td>4.158 ± 513†</td>
<td>4.705 ± 572</td>
</tr>
<tr>
<td>Fiber perimeter, µm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>262 ± 11</td>
<td>290 ± 16</td>
</tr>
<tr>
<td>T2</td>
<td>296 ± 11*</td>
<td>276 ± 28</td>
</tr>
<tr>
<td>T3</td>
<td>260 ± 17†</td>
<td>298 ± 17</td>
</tr>
<tr>
<td>Capillary contacts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>3.67 ± 0.22</td>
<td>3.54 ± 0.16</td>
</tr>
<tr>
<td>T2</td>
<td>4.39 ± 0.27*</td>
<td>3.99 ± 0.38</td>
</tr>
<tr>
<td>T3</td>
<td>4.40 ± 0.34*</td>
<td>4.50 ± 0.30*</td>
</tr>
<tr>
<td>Individual capillary-to-fiber ratio</td>
<td></td>
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</tr>
<tr>
<td>T1</td>
<td>1.37 ± 0.11</td>
<td>1.29 ± 0.07</td>
</tr>
<tr>
<td>T2</td>
<td>1.61 ± 0.13*</td>
<td>1.48 ± 0.16</td>
</tr>
<tr>
<td>T3</td>
<td>1.62 ± 0.14*</td>
<td>1.68 ± 0.13*</td>
</tr>
<tr>
<td>Sharing factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>2.82 ± 0.07</td>
<td>2.77 ± 0.05</td>
</tr>
<tr>
<td>T2</td>
<td>2.73 ± 0.07</td>
<td>2.73 ± 0.07</td>
</tr>
<tr>
<td>T3</td>
<td>2.72 ± 0.05</td>
<td>2.70 ± 0.05</td>
</tr>
<tr>
<td>Capillary density, capillaries/mm²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>343 ± 28</td>
<td>270 ± 23</td>
</tr>
<tr>
<td>T2</td>
<td>336 ± 31</td>
<td>367 ± 34‡</td>
</tr>
<tr>
<td>T3</td>
<td>423 ± 48‡</td>
<td>376 ± 29‡</td>
</tr>
<tr>
<td>CFPE index, capillaries/1,000 µm</td>
<td></td>
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<tr>
<td>T1</td>
<td>4.25 ± 0.30</td>
<td>3.80 ± 0.28</td>
</tr>
<tr>
<td>T2</td>
<td>4.84 ± 0.39*</td>
<td>4.81 ± 0.33*</td>
</tr>
<tr>
<td>T3</td>
<td>5.48 ± 0.49‡</td>
<td>5.16 ± 0.20‡</td>
</tr>
</tbody>
</table>

Values are means ± SE. CFPE, capillary-to-fiber perimeter ex-
change. *P < 0.05 vs. T1 measurements. †P < 0.05 vs. T2 measure-
ments. ‡P < 0.01 vs. T1 measurements.
significant increase in the CD (P < 0.01) and a further increase in the CFPE index (P < 0.05) after AT in this group.

The AT → AT group demonstrated no significant alterations in either the cross-sectional area or P of the muscle fibers after 9 or 18 wk of AT. In contrast, there were significant increases in CC (P < 0.05) and the C/Fi (P < 0.05) and no significant change in the SF, after 18 wk of AT. After 9 wk of AT, both the CD and the CFPE index increased (P < 0.01), with no further changes in the second 9-wk phase of AT.

DISCUSSION

We found that in a population of older men, 9 wk of high-intensity RT of the legs followed by 9 wk of AT on a cycle ergometer resulted in similar changes in both the VO₂peak and the size of the capillary-fiber interface as did 18 wk of AT on a cycle ergometer. Both RT and AT resulted in significant increases in the VO₂peak and the capillary supply to the skeletal muscle, suggesting similarities in the mechanism of these training responses. In addition, it is noteworthy that the changes in the CFPE index (related to the capillary-to-fiber surface) paralleled the changes in VO₂peak as a consequence of both RT and AT, whereas CD (related to diffusion distances) was altered only after the AT.

Critique of study design. In this study, a cycle ergometer was utilized to determine an estimate of the maximal aerobic power. Given that the cycle was chosen as the AT modality and that the muscle sampled for determining the changes in muscle structure was the vastus lateralis, a muscle highly recruited for cycling activity (9), the use of the cycle for quantitating the effects of the training on VO₂peak was reasonable. In addition, it is noteworthy that there were no differences between groups with respect to the number of subjects attaining the criteria for determination of VO₂peak, nor were there differences between testing periods (11), indicating that similar efforts were put forth by the subjects at each testing period.

It is apparent from Tables 1 and 2 that there was a slight (nonsignificant) difference in muscle fiber size and body mass between groups before training, which, in theory, could have had an impact on the adaptation to training. However, because the pattern of change in fiber size and capillary number (i.e., whether there was an increase, decrease, or maintenance with training) was not related to the initial fiber size or body mass (unpublished observations), it seems unlikely that these differences between groups had any bearing on the training response.

VO₂peak changes after RT and/or AT. The changes in absolute VO₂peak (l/min) represented 17 and 16% of the initial values in the RT → AT and AT → AT groups, respectively, comparable with recent studies of AT in this population, where 7–38% increases in VO₂peak have been observed (1, 19, 21). In a comparison of the responses between the two groups, the similarity of training response suggests prior RT does not have a synergistic effect on subsequent AT. It is also clear that RT alone provided a reasonably effective training stimulus for improving VO₂peak, in agreement with the results reported previously for high-intensity RT in this population (8). While the RT → AT group performed only one-half of the AT → AT group, it is not apparent what the nature and amount of stimulus delivered at the muscle tissue level might be. It is possible that, despite probable differences in motor unit recruitment between RT and AT (although we found no differences in either the pattern or magnitude of adaptation between type I and type II muscle fibers between resistance and AT (11)), RT may have provided an "aerobic-like" stimulus to adaptation in these older adults with respect to the adaptive process at the muscle level. This possibility will be evaluated in light of the changes in capillary supply that were observed.

Alterations in muscle morphometry: implications for blood-tissue exchange. Utilization of capillary measurements related to FA and P on transverse sections of muscle tissue provides a means of examining the significance of alterations in the morphometric profile on the capacity for the different processes that capillaries serve in blood-tissue exchange (12). Specifically, there is evidence to suggest that the FA-based measurements of the capillary supply (which relate to diffusion distances), such as the CD, may relate better to the delivery of fuel substrates (7) to muscle fibers and to the removal of waste products (30) from muscle fibers (i.e., processes that rely primarily on passive diffusion). In contrast, indexes of the capillary-to-fiber surface, such as the capillary-to-fiber P ratio (23) and the CFPE index (12), may provide more relevant information regarding the capacity for oxygen flux and the transport of substances from blood that rely on carrier- or receptor-mediated processes at the muscle fiber membrane (i.e., hormones, glucose, etc.).

As we have reported previously (13), the AT period resulted in a maintenance of the CD but significantly increased the CFPE index, indicating a larger surface area was available for exchange between the capillaries and muscle fibers after the RT (i.e., T1) and suggesting there may be an increased capacity for oxygen flux after the RT. In the subsequent period of AT, the increases in the CD and CFPE index were due exclusively to a reduction in the fiber size rather than an increase in capillary number from T2 to T3 in the RT → AT group, indicating no new capillary development occurred in this period. This response may indicate that the muscle fiber hypertrophy evident after the RT was not necessary for the AT period (because it was not maintained) and that, as a consequence of the reduction in fiber size and the resulting increase in capillary supply, there was no need for a further increase in the number of capillaries.

In contrast, the AT → AT group, after two consecutive 9-wk periods of AT on a cycle ergometer, demonstrated...
a significant increase in capillary number at T3 (i.e., CC and C/Fi) with no change in fiber size, resulting in an increased CD and an increased CFPE index at both T2 and T3. The net result, however, is that both RT and AT were associated with adaptations in the capillary supply that indicate the potential for an increased capacity for oxygen flux (i.e., an increased size of the capillary-to-fiber surface), and this similarity may help explain the means by which V̇O₂peak is augmented through these training paradigms in this population. Specifically, we observed that the CFPE index was increased after both resistance and AT, paralleling the increase in V̇O₂peak, whereas the CD was increased only after AT. It is also relevant that the magnitude of changes in capillary supply did not differ between groups, again suggesting prior RT did not potentiate but rather complemented the subsequent adaptation to AT in these subjects.

Role of the capillary supply in whole body V̇O₂peak. There is mounting evidence that the capillary supply plays an important role in determining the maximal rate of oxygen flux at the muscle-capillary interface (15, 16, 22). A large number of studies have suggested that the maximal V̇O₂ is limited by diffusive processes between the blood and tissues (14, 27), consistent with an important role for the capillary supply to skeletal muscle in this response. Of course, if there is an optimization of structure and function, as proposed by others (20), we would expect the capillary supply to be matched to the V̇O₂peak without necessarily implying a limitation per se.

Stepwise multiple-regression analysis revealed that the CFPE index explained 40% of the variance in V̇O₂peak and that addition of the CD to the regression equation did not significantly improve the prediction. Figure 2 illustrates the relationship between the capillary supply, as defined by the CD and the CFPE index, and V̇O₂peak across all time points for all subjects. The training did not appear to alter the nature of the relationships that were observed (i.e., the slopes of the regressions were similar before and after training). In the consideration of Fig. 2, it is important to recognize that there is considerable evidence that strongly suggests it is not the diffusion distance per se that is the most crucial aspect of the capillary supply with respect to oxygen flux but rather the size of the capillary-to-fiber surface (15, 22, 29). Thus the observation that the CFPE index (an index of the capillary-to-fiber surface) explained a larger proportion of the variance in V̇O₂peak than did the CD (a determinant of diffusion distance) would tend to support this hypothesis.

A relationship between the capillary supply and V̇O₂peak has been documented previously in young adult humans (17); however, our investigation is the first to describe a relationship between the capillary supply and V̇O₂peak in the older population. While it is clear that other factors (e.g., metabolic adjustments, cardiac output, etc.) must account for a large portion of the variance in V̇O₂peak in this population, our findings suggest that the role of the capillary supply to muscle fibers is also important to whole body aerobic function in the older population. In this respect, it is most pertinent that when the change in V̇O₂peak (ml/min) was plotted as a function of the change in capillary supply (i.e., CFPE index) for all subjects, a moderate relationship was found (r = 0.52, P < 0.01), indicating that the individuals with the greatest increase in the capillary supply tended also to have the greatest increase in V̇O₂peak. Comparison of the slopes of these responses between groups revealed that the relationship between the capillary supply and V̇O₂peak was the same, irrespective of training modality. In contrast, the plot of the change in V̇O₂peak vs. the change in CD was not significant (P = 0.07).

Conclusions. We observed that a program of 9 wk of RT followed by 9 wk of AT produced a similar increase

Figure 2. A: relationship between capillary-to-fiber perimeter exchange index [CFPE index = (C/Fi)/P, where P is perimeter] and aerobic power (V̇O₂peak) when all data points are combined [V̇O₂peak = 15.7 + (2.7558 × CFPE index); r = 0.63, P < 0.01]. B: relationship between capillary density and V̇O₂peak when all data points are combined [V̇O₂peak = 22.6 + (0.0178 × capillary density); r = 0.44, P < 0.01].
in $V_O^{2\text{peak}}$ (l/min) as did 18 wk of AT in a population of older men. In conjunction with the changes in $V_O^{2\text{peak}}$, we observed significant increases in the capillary-to-fiber surface interface (as reflected in an increased CFPE index) after both RT and AT, whereas the CD was significantly increased only after AT. When the $V_O^{2\text{peak}}$ was regressed as a function of the capillary supply, the CFPE index was found to explain a greater proportion of the variance in $V_O^{2\text{peak}}$ than did the other indexes of the capillary supply. These observations support the utility of the CFPE index in providing an indication of the capacity for oxygen flux between the capillaries and muscle fibers and support an important role for the capillaries in the $V_O^{2\text{peak}}$ response in the older population. They also suggest the possibility that high-intensity RT and AT, by increasing the capillary supply to the skeletal muscle fibers, may operate through similar mechanisms to increase the $V_O^{2\text{peak}}$ in the older population.

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