Effects of exercise training on thermoregulatory responses and blood volume in older men

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Okazaki, Kazunobu, Yoshi-Ichiro Kamijo, Yoshiaki Takeno, Tadashi Okumoto, Shizue Masuki, and Hiroshi Nose. Effects of exercise training on thermoregulatory responses and blood volume in older men. J Appl Physiol 93: 1630–1637, 2002. —We assessed the effects of aerobic and/or resistance training on thermoregulatory responses in older men and analyzed the results in relation to the changes in peak oxygen consumption rate ($\dot{V}O_2$ peak) and blood volume (BV). Twenty-three older men [age, $64 \pm 1$ (SD) yr; $\dot{V}O_2$ peak, $32.7 \pm 1.1$ ml·kg$^{-1}$·min$^{-1}$] were divided into three training regimens for 18 wk: control (C; $n = 7$), aerobic training (AT; $n = 8$), and resistance training (RT; $n = 8$). Subjects in C were allowed to perform walking of 10,000 steps/day, 6–7 days/wk. Subjects in AT exercised on a cycle ergometer at 50–80% $\dot{V}O_2$ peak for 60 min/day, 3 days/wk, in addition to the walking. Subjects in RT performed a resistance exercise, including knee extension and flexion at 60–80% of one repetition maximum, two to three sets of eight repetitions per day, 3 days/wk, in addition to the walking. After 18 wk of training, $\dot{V}O_2$ peak increased by $5.2 \pm 3.4$% in C ($P = 0.07$), $20.0 \pm 2.5$% in AT ($P < 0.0001$), and $9.7 \pm 5.1$% in RT ($P < 0.0003$), but BV remained unchanged in all trials. In addition, the esophageal temperature ($T_{es}$) thresholds for forearm skin vasodilation and sweating, determined during 30-min exercise of 60% $\dot{V}O_2$ peak at $3^\circ$C, decreased in AT ($P < 0.02$) and RT ($P < 0.02$) but not in C ($P > 0.2$). In contrast, the slopes of forearm skin vascular conductance/$T_{es}$ and sweat rate/$T_{es}$ remained unchanged in all trials, but both increased in subjects with increased BV irrespective of trials with significant correlations between the changes in the slopes and BV ($P < 0.005$ and $P < 0.0005$, respectively). Thus aerobic and/or resistance training in older men increased $\dot{V}O_2$ peak and lowered $T_{es}$ thresholds for forearm skin vasodilation and sweating but did not increase BV. Furthermore, the sensitivity of the increase in skin vasodilation and sweating at a given increase in $T_{es}$ was more associated with BV than with $\dot{V}O_2$ peak.

aerobic training; resistance training; skin blood flow; sweating

THERMOREGULATORY RESPONSES have been known to deteriorate with aging, which is likely associated with the decrease in peak oxygen consumption rate ($\dot{V}O_2$ peak).

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AEROBIC CAPACITY AND THERMOREGULATION IN THE ELDERLY

1631

is yet unknown how the increased BV enhances the FBF response. Some studies have reported that aerobic training increased BV in older subjects (4, 24), but others did not (29, 30, 35). In addition, few of these studies reported change in the FBF response after training. One study by Ho et al. (13) suggested that the enhanced FBF response was caused by increased cardiac output due to increased plasma volume (PV) after a 4-wk aerobic training, but they found no significant increase in PV because of too small number of subjects.

In the present study, we examined the effect of 8- and 18-wk aerobic or resistance training on BV and FBF response in older subjects to elucidate the involvement of increased BV in the exercise-training-induced enhancement of FBF response in older men. The reason for adding a resistance training trial was that the training enabled us to distinguish the mere effect of increased VO2peak on FBF response from other effects induced by aerobic training, such as more prolonged cardiovascular and/or heat loading. In addition, we measured the changes in sweat rate (SR) response to increased Tce after exercise training because, to our knowledge, there have been no studies on the effects of exercise training on this factor in older men. In addition, we assessed Tce thresholds for forearm skin vascular conductance (FVC) and SR responses and slopes of the response-Tce relationships to examine how VO2peak and/or BV modify the responses to increased Tce.

METHODS

Subjects and Procedures

The procedures in this study were approved by the Review Board on Human Experiments, Shinshu University School of Medicine. After the experimental protocols were fully explained, 23 older (58–72 yr) healthy men gave their written informed consent before participating in this study. The subjects were relatively active but did not participate in any regular exercise training program. All subjects were nonsmokers and had no overt history of cardiovascular or pulmonary diseases or any orthopedic limitations to the exercising test and training. During the experiment, no subject was taking medication that had a potential to impact cardiovascular and thermoregulatory function or BV and constituents.

Subjects were randomly divided into the three training trials for 18 wk [control (C; n = 7), resistance training (RT; n = 8), and aerobic training (AT; n = 8); Table 1] to avoid differences in physical characteristics among the trials before training.

In the C trial, subjects were not engaged in a specific training program except for walking of 9,465 ± 1,954 steps/day, 6.7 ± 0.2 days/wk. Subjects in the RT and AT trials trained under our supervision in addition to the walking of 10,353 ± 2,336 and 8,749 ± 490 steps/day, respectively. The training was performed between September and April to avoid any effect of heat acclimatization during the summer season. Averaged ambient temperature (Ta) in the city was 19°C in September, –1°C in January, and 4°C in April. Relative humidity (RH) was ~70% throughout the period.

VO2peak, ventilation threshold (VT), BV, and constituents, and muscle strength for isometric knee extension were measured in all subjects before and after 8-wk and 18-wk training. FBF and SR were also measured during exercise in a hot environment.

Measurements

VO2peak and VT. VO2peak was measured while the subjects were in an upright position with the use of a cycle ergometer at Ta of 25.0 ± 0.1°C and RH of 46 ± 1% (means ± range). After baseline measurements at rest were taken for 3 min, the subjects started pedaling bicycles at 60 cycles/min without loading. Exercise intensity was increased by 30 W every 3 min until 120 W, and, above this intensity, it was increased by 15 W every 2 min until subjects could not maintain the rhythm. Oxygen consumption rate (VO2) was determined every 15 s from the oxygen and carbon dioxide fractions in expired gas and the expired ventilatory volume (Aeromonitor AE260, Minato, Tokyo, Japan). Heart rate (HR) was recorded every 1 min from the trace of an electrocardiogram (Life Scope 8, Nihon Kohden, Tokyo, Japan). VO2peak was determined after the three largest consecutive values at the end of exercise were averaged. The criteria for determining VO2peak were that the respiratory exchange ratio was >1.1, VO2 leveled off despite increasing workload, and HR reached the age-predicted maximal value. VT was determined by the V-slope method and presented as VO2 at VT (2).

Table 1. Physical characteristics and blood volumes and constituents before and after 8-wk and 18-wk training

<table>
<thead>
<tr>
<th></th>
<th>C (n = 7)</th>
<th></th>
<th>RT (n = 8)</th>
<th></th>
<th>AT (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>65 ± 2</td>
<td>64 ± 1</td>
<td>65 ± 2</td>
<td>65 ± 1</td>
<td>64 ± 1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>165 ± 2</td>
<td>161 ± 2</td>
<td>165 ± 2</td>
<td>165 ± 2</td>
<td></td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>59.2 ± 3.6</td>
<td>61.6 ± 1.2</td>
<td>62.1 ± 1.4</td>
<td>63.4 ± 1.6</td>
<td>65.3 ± 3.1</td>
</tr>
<tr>
<td>VO2peak, ml·kg–1·min–1</td>
<td>32.6 ± 1.0</td>
<td>32.6 ± 3.0</td>
<td>35.0 ± 2.87</td>
<td>39.2 ± 3.1</td>
<td>32.9 ± 0.7</td>
</tr>
<tr>
<td>VT, ml·kg–1·min–1</td>
<td>22.1 ± 1.9</td>
<td>22.1 ± 2.0</td>
<td>23.5 ± 2.2</td>
<td>26.8 ± 3.0</td>
<td>20.4 ± 0.9</td>
</tr>
<tr>
<td>HRmax, beats/min</td>
<td>168 ± 2</td>
<td>165 ± 4</td>
<td>165 ± 4</td>
<td>165 ± 7</td>
<td>165 ± 3</td>
</tr>
<tr>
<td>Isometric knee extension, N·m</td>
<td>184 ± 21</td>
<td>186 ± 15</td>
<td>207 ± 15</td>
<td>215 ± 22</td>
<td>190 ± 18</td>
</tr>
<tr>
<td>BV, ml/kg</td>
<td>71.0 ± 2.1</td>
<td>70.7 ± 2.4</td>
<td>69.4 ± 2.3</td>
<td>71.6 ± 2.9</td>
<td>69.9 ± 1.2</td>
</tr>
<tr>
<td>PV, ml/kg</td>
<td>43.0 ± 1.8</td>
<td>43.0 ± 1.7</td>
<td>42.3 ± 1.3</td>
<td>44.0 ± 1.8</td>
<td>43.3 ± 0.9</td>
</tr>
<tr>
<td>[Alb]p, g/dl</td>
<td>4.3 ± 0.1</td>
<td>4.3 ± 0.1</td>
<td>4.3 ± 0.1</td>
<td>4.1 ± 0.0</td>
<td>4.4 ± 0.1</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. C, control; RT, resistance training; AT, aerobic training; VO2peak, peak oxygen consumption rate; VT, oxygen consumption rate at the ventilatory threshold; HRmax, maximal heart rate; BV, blood volume; PV, plasma volume; [Alb]p, plasma albumin concentration. Values in RT after 18-wk training are for 6 subjects. Blood volumes and constituents in AT are for 7 subjects.

*Significantly different from before training, P < 0.05. †Significantly different from before training, P < 0.01.
Muscle strength for isometric knee extension. Muscle strength for isometric extension was measured in each side of the knee with a dynamometer (Biodex 3, Biodex Medical System, Shirley, NY). After regular warming-up and familiarization protocols, the anatomic axis of the knee joint was aligned with the mechanical axis of the dynamometer arm to adjust the angle between the lower and upper legs to 105°. Then, three 3-s maximum voluntary contractions, interleaved by a 30-s recovery, were conducted. The peak torque averaged for three trials was adopted for the value for one side of the knee, and it is given as an averaged value of both sides of the knee in Table 1.

BV and constituents. On the day of the measurement, subjects reported to the laboratory at 7:00 AM normally hydrated but without having taken any food for 8 h before the experiment. PV was determined by the Evans blue dye-dilution method (7). The background absorbance due to turbiditiy was corrected by using a regression equation on the relationship between 620 and 740 nm, previously determined on 64 control plasma samples in 22 subjects according to the method applied elsewhere (5, 30). BV was calculated from PV and hematocrit (Hct) values after correction for plasma trapped among the red blood cells in the Hct tube (0.96) and an F-cell ratio (0.91) (8). The measurement error of BV was 2.1 ± 1.8% (means ± SD) (n = 4), which was obtained by measuring BV twice in the same subjects with a BV of 64.9–93.5 ml/kg after 2- to 3-wk intervals. The residues of blood samples drawn before the injection of the dye were used to determine the Hct (microcentrifuge method) and plasma albumin concentrations (Alb\textsubscript{p}, colorimetry). Total albumin content in plasma (Alb\textsubscript{tot}) was determined as a product of PV and Alb\textsubscript{p}. The BV measurement was not performed in one of eight subjects in the AT trial who showed an allergic reaction to the patch test of the dye performed 24 h before the measurement on every subject.

FBF and SR measurements. Subjects reported to the laboratory normally hydrated but having fasted for at least 2 h before the measurement, at the same time of day before and after the training regimens to avoid any effect of circadian rhythm. Clad in shorts and shoes, subjects emptied their bladders, entered the chamber controlled at 30.0 ± 0.1°C of T\textsubscript{s} and 50 ± 1% of RH (means ± range), and sat in the contour chair of the cycle ergometer in a semirecumbent position for 45 min while all measurement devices were applied. After baseline measurements were taken at rest for 10 min, the subjects exercised in a semirecumbent position at 60% of their pretraining VO\textsubscript{2peak} for 20 min without fan cooling.

T\textsubscript{es} was monitored with a thermocouple in a polyethylene tubing (PE-90). The tip of the tube was advanced at a distance of one-fourth of the subject’s standing height from the external nares. Mean skin temperature (T\textsubscript{sk}) was determined as T\textsubscript{sk} = 0.25T\textsubscript{ta} + 0.43T\textsubscript{ch} + 0.32T\textsubscript{th} (25), where T\textsubscript{es}, T\textsubscript{ta}, and T\textsubscript{ch}, and T\textsubscript{th} are skin surface temperature at the forearm, chest, and thigh measured with the thermocouples, respectively. SR was determined by capacitance bygrometry, calculated from the relative humidity and temperature of the air (THP-B3T, Shinee, Tokyo, Japan) flowing out of a 12.56-cm\textsuperscript{2} capsule at the rate of 1.5 l/min on the chest at 5 cm below the left clavicle. FBF was measured by venous occlusion plethysmography with a mercury-in-Silastic tube strain gauge placed around the upper side of the subject’s left forearm positioned above the heart level, with the hand eliminated from the circulation by inflation of an occlusion cuff to a supra-arterial pressure (~280 mmHg) (34). HR was recorded every 1 min as described in VO\textsubscript{2peak} and VT. Systolic (SAP) and diastolic arterial blood pressures (DAP) were measured every 1 min from the right upper arm at the heart level by inflation of the cuff with a sonometric pickup of Korotkoff’s sound (STPB-780, Colin, Komaki, Japan). Mean arterial blood pressure (MAP) was calculated as DAP + (SAP – DAP)/3. FVC was calculated as FBF/MAP (reported in units of ml-100 mm\textsuperscript{-1} min\textsuperscript{-1} 100 mmHg\textsuperscript{-1}). T\textsubscript{es}, T\textsubscript{sk}, and SR were recorded every 5 s, and FBF was measured twice every 1 min at rest and during exercise and presented every 1 min as an average.

The T\textsubscript{es} thresholds for increasing SR (TH\textsubscript{SR}) and increasing FVC (TH\textsubscript{FVC}) were determined on each subject as the T\textsubscript{es} at 2–5 min after the start of exercise where SR or FVC increased above the baselines. The slopes of an increase in SR (SR/T\textsubscript{es}) and FVC (FVC/T\textsubscript{es}) at a given increase in T\textsubscript{es} were determined on each subject from a linear regression equation on the measurements recorded at 5–20 min of exercise.

Exercise Training Regimen

Subjects in the RT and AT trials trained for 18 wk according to the protocol recommended by the American College of Sports Medicine (1). As warming-up and cooling-down protocols, subjects in the AT and RT trials performed a 5-min stretch exercise and a 5-min cycle ergometer exercise at 50% VO\textsubscript{2peak} before and after the main exercise.

Subjects in the AT trial performed an exercise protocol, consisting of a knee extension and flexion, chest press, pull-up, and arm curl with weight resistance machines (Athlete, Mizuno, Tokyo, Japan) at 60–80% of one repetition maximum (1 RM), two to three sets of eight repetitions per day, 3 days/wk. The exercise intensity was increased with the training days: two sets of each exercise at 60, 70, and 75% 1 RM in the 1st, 2nd, and 3rd wk, respectively, and three sets at 80% 1 RM after the 4th wk. In addition to the exercise, supportive upper back extension, pelvic rise, and crunch without weight loading were performed throughout the training period.

Subjects in the AT trial performed a cycle ergometer exercise at 50–80% of VO\textsubscript{2peak} for 60 min/day, consisting of four sets of 15-min exercise followed by a 5-min rest. The exercise intensity was increased with the training days: 50, 60, and 65% VO\textsubscript{2peak} for the 1st, 2nd, and 3rd wk, respectively; 70% VO\textsubscript{2peak} for the 4th to 8th wk, 75% VO\textsubscript{2peak} for the 9th to 10th wk; and 80% VO\textsubscript{2peak} after the 11th wk. HR was continuously monitored and recorded every 5 min during exercise. The exercise intensity was readjusted every 1 wk so that HR at 5 min of exercise was equivalent to the target exercise intensity.

The environmental condition for the training room was controlled at T\textsubscript{es} of ~20°C and RH of ~50% without any significant differences between the RT and AT trials. During exercise, the subjects were allowed access to water ad libitum, and the amount was monitored. Subjects were weighed before and after the training regimen each day to estimate sweat loss. Body weight loss after training per day was 4–6 ml/kg body wt for the RT trial and 8–10 ml/kg body wt for the AT trial.

Statistics

The effects of training on physical characteristics, BV, blood constituents, TH\textsubscript{SR}, TH\textsubscript{FVC}, SR/T\textsubscript{es}, and FVC/T\textsubscript{es} within each trial were tested by a 3 (C, RT, AT) × 3 (before, 8 wk, and 18 wk) ANOVA for repeated measures (Table 1 and see Table 3). The effects of training on cardiovascular and thermoregulatory responses in a hot environment within each trial were tested by three-way ANOVA for repeated measures (Table 2). Subsequent post hoc tests to
Table 2. HR, MAP, T_{es} and T_{sk} during exercise in a hot environment before and after 8-wk and 18-wk training

<table>
<thead>
<tr>
<th></th>
<th>C (n = 7)</th>
<th>RT (n = 8)</th>
<th>AT (n = 8)</th>
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<tbody>
<tr>
<td></td>
<td>Before 8 wk 18 wk</td>
<td>Before 8 wk 18 wk</td>
<td>Before 8 wk 18 wk</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>65 ± 4</td>
<td>67 ± 5†</td>
<td>63 ± 3†‡</td>
</tr>
<tr>
<td>Ex5</td>
<td>116 ± 4</td>
<td>121 ± 5‡</td>
<td>119 ± 4†</td>
</tr>
<tr>
<td>Ex20</td>
<td>133 ± 5</td>
<td>136 ± 6†</td>
<td>136 ± 4†</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>37.2 ± 0.1°</td>
<td>37.1 ± 0.1†</td>
<td>37.0 ± 0.1†</td>
</tr>
<tr>
<td>Ex5</td>
<td>37.4 ± 0.3°</td>
<td>37.2 ± 0.1</td>
<td>37.0 ± 0.2†</td>
</tr>
<tr>
<td>Ex20</td>
<td>38.1 ± 0.1°</td>
<td>38.2 ± 0.1</td>
<td>37.9 ± 0.2</td>
</tr>
<tr>
<td>T_{es}, °C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>34.0 ± 0.3°</td>
<td>34.3 ± 0.1°</td>
<td>33.9 ± 0.3°</td>
</tr>
<tr>
<td>Ex5</td>
<td>33.6 ± 0.3°</td>
<td>34.1 ± 0.2°</td>
<td>33.3 ± 0.4°</td>
</tr>
<tr>
<td>Ex20</td>
<td>34.2 ± 0.3°</td>
<td>34.1 ± 0.2°</td>
<td>33.9 ± 0.4°</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. HR, heart rate; MAP, mean arterial blood pressure; T_{es}, esophageal temperature; T_{sk}, mean skin temperature; Ex5, 5 min after exercise; Ex20, 20 min after exercise. Values in RT after 18-wk training are for 6 subjects. †Significantly different from before training, P < 0.05. ‡Significantly different from before training, P < 0.01. §Significantly different from after 8 wk training, P < 0.01.

determine significant differences in the various pairwise comparisons were performed by using Scheffé’s test. The null hypothesis was rejected when there were values of P < 0.05. Regression analyses were performed by Bracey’s methods (3). Because two of eight subjects in the RT trial quit the last 10-wk training regimen, the comparison between 8- and 18-wk training was performed on only six subjects. Values are expressed as means ± SE for seven subjects in the C trial and for eight subjects in the RT and AT trials, except as noted.

RESULTS

Table 1 shows the physical characteristics, BV, and PV before and after training. After 8-wk training, V_{O2peak} increased by 8.4 ± 2.9% (P < 0.01) in the RT trial and by 13.2 ± 2.4% in the AT trial (P < 0.0001) with respect to the pretraining values. After 18-wk training, it further increased by 9.7 ± 5.1% in the RT trial (P < 0.003) and by 20.0 ± 2.5% in the AT trial (P < 0.0001), whereas it remained unchanged in the C trial. There were no significant changes in body weight, maximal heart rate (HR_{max}), BV, PV, and [Alb]_{pl} after 8- and 18-wk training.

Table 2 shows HR, MAP, T_{es}, and T_{sk} during exercise in a hot environment before and after 8- and 18-wk training in three trials. Only the values at rest and at 5 and 20 min after the start of exercise are presented in the table to simplify. After 8- and 18-wk training, HR at rest decreased in the RT and AT trials but not in the C trial. The increase in HR during exercise was reduced in the AT trial but was enhanced in the RT and C trials. MAP at rest decreased significantly in all trials. The increase in MAP during exercise was reduced at 5 and 20 min in the AT trial, at 5 min in the RT trial, and at 20 min in the C trial, but it was enhanced at 20 min in the RT trial. T_{es} at rest decreased significantly in all trials. The increase in T_{es} during exercise was attenuated at 5 and 20 min in the RT and AT trials and at 5 min in the C trial. T_{sk} at rest was not altered in any trials. The increase in T_{sk} during exercise was reduced at 5 and 20 min in the AT trial and at 20 min in the C trial, but it increased at 5 min in the C trial.

The SR and FVC responses to increased T_{es} during exercise in a hot environment are shown in Fig. 1. TH_{SR}, TH_{FVC}, SR/T_{es}, and FVC/T_{es}, are summarized in Table 3. TH_{SR} decreased by 0.22 and 0.28°C in the RT trial and by 0.15 and 0.17°C in the AT trial, after 8-wk and 18-wk training, respectively, but it did not change significantly in the C trial. Similarly, TH_{FVC} decreased by 0.27 and 0.32°C in the RT trial and by 0.15 and 0.29°C in the AT trial, after 8- and 18-wk training, respectively, but it did not change significantly in the C trial. There were no significant changes in SR/T_{es} and FVC/T_{es} before and after training in any trials.

When the data from all the trials were pooled, the change in V_{O2peak} after training was weakly but significantly correlated with those in TH_{SR} (r = 0.30, P < 0.05) and TH_{FVC} (r = 0.34, P < 0.03) but not with those in SR/T_{es} (r = 0.05) and FVC/T_{es} (r = 0.04). In contrast, the change in BV (ΔBV) after training was significantly correlated with Δ(T_{es}) (r = 0.51, P < 0.0005) and Δ(FVC/T_{es}) (r = 0.45, P < 0.005) (Fig. 2, A and B) but not with ΔTH_{SR} (P > 0.1) or ΔTH_{FVC} (P > 0.3). As shown in Fig. 3 (A and B), ΔTH_{SR} was significantly correlated with Δ(T_{es}) (r = 0.79, P < 0.0001), and Δ(SRT_{es}) was significantly correlated with Δ(FVC/T_{es}) (r = 0.63, P < 0.0001). As shown in Fig. 4, the change in Alb_{pl} after 8- and 18-wk training was significantly correlated with that in PV (r = 0.68, P < 0.0001).
DISCUSSION

In the present study, we verified the results previously reported in older men that BV did not increase after aerobic training (29, 30, 35) and that THFVC and THSR decreased with the increase in V\textsubscript{O2 peak}, whereas FVC/T\textsubscript{es} and SR/T\textsubscript{es} remained unchanged (33). Moreover, we confirmed the results not only after aerobic but also after resistance training. In addition, we clarified that the reductions in THFVC and THSR were more associated with increased V\textsubscript{O2 peak} than with increased BV, whereas changes in FVC/T\textsubscript{es} and SR/T\textsubscript{es} were more associated with that in BV than V\textsubscript{O2 peak}.

**THFVC and THSR After Training**

As shown in Table 1, V\textsubscript{O2 peak} in the RT and AT trials increased after 8- or 18-wk training. The reductions in THFVC and THSR in the RT and AT trials were weakly but significantly correlated with the increase in V\textsubscript{O2 peak}. THFVC or THSR at a given absolute exercise intensity has been reported to decrease after aerobic training not only in younger (18, 25) but also in older subjects (33). Smolander et al. (28) demonstrated that THFVC increased with relative exercise intensity in individual younger subjects. Thomas et al. (33) reported that 16-wk aerobic training decreased THFVC in subjects who increased V\textsubscript{O2 peak} by 5%. Moreover, Ho et al. (13) suggested that, in older subjects, THFVC was not altered after a 4-wk training even when absolute exercise intensity was increased from 60 to 70% of pretraining V\textsubscript{O2 peak}, equivalent to 60% of posttraining V\textsubscript{O2 peak}. These results suggest that the reduction in THFVC and/or THSR after training was associated with reduced relative exercise intensity due to increased V\textsubscript{O2 peak}.

**Table 3. THSR, THFVC, SR/T\textsubscript{es}, and FVC/T\textsubscript{es} during exercise in a hot environment before and after 8-wk and 18-wk training**

<table>
<thead>
<tr>
<th></th>
<th>C (n = 7)</th>
<th></th>
<th>RT (n = 8)</th>
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<th>AT (n = 8)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>8 wk</td>
<td>18 wk</td>
<td>Before</td>
<td>8 wk</td>
<td>18 wk</td>
</tr>
<tr>
<td>TH\textsubscript{es}, °C</td>
<td>37.34 ± 0.08</td>
<td>37.33 ± 0.04</td>
<td>37.22 ± 0.10</td>
<td>37.27 ± 0.16</td>
<td>37.05 ± 0.11</td>
<td>36.99 ± 0.16†</td>
</tr>
<tr>
<td>THFVC, °C</td>
<td>37.38 ± 0.09</td>
<td>37.39 ± 0.06</td>
<td>37.44 ± 0.07</td>
<td>37.44 ± 0.17</td>
<td>37.17 ± 0.11</td>
<td>37.12 ± 0.18*</td>
</tr>
<tr>
<td>SR/T\textsubscript{es}, mg·cm\textsuperscript{-2}·min\textsuperscript{-1}·°C\textsuperscript{-1}</td>
<td>0.9 ± 0.2</td>
<td>0.7 ± 0.3</td>
<td>0.6 ± 0.2</td>
<td>1.0 ± 0.3</td>
<td>0.9 ± 0.3</td>
<td>0.9 ± 0.3</td>
</tr>
<tr>
<td>FVC/T\textsubscript{es}, units/°C</td>
<td>7.0 ± 2.0</td>
<td>6.5 ± 2.0</td>
<td>5.5 ± 1.7</td>
<td>10.5 ± 3.3</td>
<td>7.6 ± 2.7</td>
<td>9.7 ± 1.8</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. TH\textsubscript{es}, T\textsubscript{es} threshold for increasing sweat rate (SR); THFVC, T\textsubscript{es} threshold for increasing forearm skin vascular conductance (FVC); SR/T\textsubscript{es}, slope of an increase in SR at a given increase in T\textsubscript{es}; FVC/T\textsubscript{es}, slope of an increase in FVC at a given increase in T\textsubscript{es}. Values in RT after 18-wk training are for 6 subjects. *Significantly different from before training, P < 0.05. †Significantly different from before training, P < 0.01.
$\dot{V}O_2$ peak and BV After Training

BV in the AT trial did not increase, as shown in Table 1. It has been reported that the response of BV to aerobic training was lower in older subjects than in younger subjects (29, 30, 35). This may be caused by reduced fluid intake after thermal dehydration (17, 30) or water deprivation (23) in older men. Recently, Taka-mata et al. (30) studied the changes in body fluid response to dehydration before and after an exercise heat acclimatization regimen (80-min bicycle exercise at 40% $\dot{V}O_2$ peak per day for 6 days at 36°C of $T_a$ and RH of 40%) and compared the results between older and younger men. They suggested that BV remained unchanged in older subjects, whereas it increased by ~5% in younger men. They also suggested that recovery from body fluid loss during 2-h rehydration was twofold higher in younger men than that in older subjects and that the recovery was augmented after heat acclimatization in younger men but not in older men. They ascribed the results to the attenuated water intake and the reduced release of body fluid-retention hormones during rehydration in older men. Although in the present study, aerobic training was performed in a cooler environment and body fluid loss was less than in previous studies (30), the blunted body fluid conservation mechanisms in older men may be involved in no increase in BV for the AT trial.

Another possible explanation for no increase in BV for the AT trial may be associated with no increase in

![Graph A](image1.png)

![Graph B](image2.png)

**Fig. 2.** Relationship between the change in slope of an increase in SR at a given increase in $T_a$ ($\Delta(SR/T_a)$) and change in blood volume ($\DeltaBV$) (A) and relationship between the change in slope of an increase in FVC at a given increase in $T_a$ ($\Delta(FVC/T_a)$) and $\DeltaBV$ (B) after training. Open symbols, changes between before training and 8-wk training; solid symbols, changes between 8- and 18-wk training. $\DeltaBV$ was significantly correlated with $\Delta(SR/T_a)$ ($r = 0.51, P < 0.0005; y = 0.23x - 0.12$) and also with $\Delta(FVC/T_a)$ ($r = 0.45, P < 0.0005; y = 2.44x - 0.80$).

![Graph C](image3.png)

![Graph D](image4.png)

**Fig. 3.** Relationship between the change in $T_a$ threshold for increasing FVC ($\Delta TH_{FVC}$) and $T_a$ threshold for increasing SR ($\Delta TH_{SR}$) (A) and relationship between the change in $\Delta(SR/T_a)$ and $\Delta(FVC/T_a)$ (B) after training. Open symbols, changes between before training and 8-wk training; solid symbols, changes between 8-wk and 18-wk training. $\DeltaBV$ was significantly correlated with $\Delta TH_{SR}$ ($r = 0.79, P < 0.0001; y = 0.86x - 0.00$). $\Delta(SR/T_a)$ was significantly correlated with $\Delta(FVC/T_a)$ ($r = 0.63, P < 0.0001; y = 0.10x - 0.04$).
were significantly correlated \((r = 0.68, P < 0.0001; y = 21.1x + 0.48)\).

Alb
total for older men (Table 1 and Fig. 4). The exercise training-induced hypervolemia has been suggested to be dependent on an increase in Alb
total, causing a fluid shift from the interstitial to intravascular fluid space according to the colloid osmotic pressure gradient between the spaces \((10, 19, 27)\). In younger subjects, exercise training-induced hypervolemia has been reported to be typically accompanied by an increase in Alb
total \((10, 19, 27)\). On the other hand, Zappe et al. \((35)\) reported that, in older men, PV did not increase after 4 days of repeated exercise with a cycle ergometer because of attenuated increases in Alb
total. They suggested that the failure to increase Alb
total in older men after exercise was caused by the lower ability to synthesize \((19)\) or translocate protein into the intravascular space than that reported in younger men \((11)\). The interindividual variation in the increase in Alb
total for the present study may be related to factors other than the active exercise training regimen, protein in diet \((14)\), or heat acclimatization \((27)\).

As shown in Table 1, the increased \(\dot{V}_{O_2}\) peak in the RT and AT trials was not accompanied by hypervolemia in older subjects. However, in younger subjects, it has been suggested that hypervolemia after aerobic training increased \(\dot{V}_{O_2}\) peak by increasing venous return to the heart and maximal cardiac stroke volume \((26, 31)\). Frontera et al. \((6)\) reported that, in older subjects, 12-wk strength training induced a 6% increase in \(\dot{V}_{O_2}\) peak and a 107% increase in 1 RM of the knee extensor, but they found no increase in BV. Recently, Jubrius et al. \((15)\) studied the cellular energetic adaptation to 6-mo aerobic or resistance training in older subjects and reported that oxidative capacity increased by 31 and 57% after aerobic and resistance training, respectively. Because muscle strength for knee extensor in the AT trial increased by the same degree as that in the RT trial (Table 1), the increase in \(\dot{V}_{O_2}\) peak for the AT trial was caused by the increased oxidative capacity or oxygen extraction rate in the lower leg muscles.

**FVC/Tes and SR/Tes and BV**

As shown in Fig. 2, \(\Delta BV\) was positively correlated with \(\Delta(FVC/T_{es})\) and \(\Delta(SR/T_{es})\). To our knowledge, there have been no studies showing the effects of exercise training-induced hypervolemia on the slopes in older subjects. In younger subjects, the maneuvers to increase the venous return to the heart \([\text{saline infusion (22)}, \text{head-out water immersion (21)}, \text{or continuous negative pressure breathing (20)]}\) increase FVC/Tes during exercise. These results suggest that increased BV enhances the FBF response by increasing cardiac output and/or by suppressing baroreflex-induced attenuation of skin vasodilation by increasing venous return to the heart in older subjects. Ho et al. \((13)\) reported that a 4-wk aerobic training enhanced the FBF response during exercise of 60% of \(\dot{V}_{O_2}\) peak in a hot environment. They ascribed this to increased cardiac output by PV expansion, although they found no significant increase in PV before and after training as a result of the small number of subjects. Coupled with the results of the present study, it is suggested that the slopes were increased by hypervolemia, irrespective of the increase in \(\dot{V}_{O_2}\) peak in older men.

The significant correlations between \(\Delta TH_{FVC}\) and \(\Delta TH_{SR}\) (Fig. 3A) and between \(\Delta(FVC/T_{es})\) and \(\Delta(SR/T_{es})\) (Fig. 3B) suggested the close association of the active vasodilator and sudomotor systems \((16)\). Mack et al. \((16)\) demonstrated in young subjects that reduction of central venous pressure by lower body negative pressure decreased not only FVC/Tes but also SR/Tes during exercise, suggesting that the reductions were caused by suppression of the sudomotor and active vasodilator systems by unloading cardiopulmonary baroreceptors. Thus the sudomotor and active vasodilator systems are closely associated during dynamic exercise. We confirmed this in older men after exercise training.

Summarizing these results, aerobic and/or resistance training in older men improved FVC and SR responses by the downward shift of TH\(FVC\) and TH\(SR\) rather than by their increased slopes of FVC/Tes and SR/Tes, which was associated more with the increased \(\dot{V}_{O_2}\) peak than with BV regardless of trials. In contrast, the change in the slopes was associated more with the change in BV, which was not necessarily accompanied by increased \(\dot{V}_{O_2}\) peak after training in older men.

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