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

KAZUNOBU OKAZAKI, YOSHI-ICHIRO KAMIJO, YOSHIAKI TAKENO, TADASHI OKUMOTO, SHIZUE MASUKI, AND HIROSHI NOSE
Department of Sports Medicine, Research Center on Aging and Adaptation, Shinshu University School of Medicine, Matsumoto 390-8621, Japan

Received 14 March 2002; accepted in final form 6 July 2002

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. First published July 12, 2002; 10.1152/japplphysiol.00222.2002.—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 (V\text{\textsubscript{O}}\text{\textsubscript{2 peak}}) and blood volume (BV). Twenty-three older men (age, 64 ± 1 (SE) yr; V\text{\textsubscript{O}}\text{\textsubscript{2 peak}}, 32.7 ± 1.1 ml·kg\textsuperscript{-1}·min\textsuperscript{-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% V\text{\textsubscript{O}}\text{\textsubscript{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, V\text{\textsubscript{O}}\text{\textsubscript{2 peak}} increased by 5.2 ± 3.4% in C (P < 0.07), 20.0 ± 2.5% in AT (P < 0.0001), and 9.7 ± 5.1% in RT (P < 0.003), but BV remained unchanged in all trials. In addition, the esophageal temperature (T\text{\textsubscript{es}}) thresholds for forearm skin vasodilation and sweating, determined during 30-min exercise of 60% V\text{\textsubscript{O}}\text{\textsubscript{2 peak}} at 30°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\text{\textsubscript{es}} and sweat rate/T\text{\textsubscript{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 V\text{\textsubscript{O}}\text{\textsubscript{2 peak}} and lowered T\text{\textsubscript{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\text{\textsubscript{es}} was more associated with BV than with V\text{\textsubscript{O}}\text{\textsubscript{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 (V\text{\textsubscript{O}}\text{\textsubscript{2 peak}}).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
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 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 FBF response 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 FBF response 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 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 VO2 peak 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 T es 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 FBF response after exercise training because, to our knowledge, there have been no studies on the effects of exercise training on this factor in older men.

Subjects and Procedures

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, 20 healthy men (58–72 yr) who had no history of cardiovascular or pulmonary diseases or other 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 three training trials for 8 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. Average 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.

Measurements

VO2 peak and VT. VO2 peak 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 outputting. 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). VO2 peak was determined after the three largest consecutive values at the end of exercise were averaged. The criteria for determining VO2 peak 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>Measure</th>
<th>C (n = 7) Before</th>
<th>8 wk</th>
<th>18 wk</th>
<th>C (n = 7) Before</th>
<th>8 wk</th>
<th>18 wk</th>
<th>AT (n = 8) Before</th>
<th>8 wk</th>
<th>18 wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>65 ± 2</td>
<td>64 ± 1</td>
<td>64 ± 2</td>
<td>65 ± 2</td>
<td>65 ± 2</td>
<td>64 ± 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>165 ± 2</td>
<td>161 ± 2</td>
<td>165 ± 2</td>
<td>165 ± 2</td>
<td>165 ± 2</td>
<td>165 ± 2</td>
<td></td>
<td></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>
<td>64.6 ± 2.8</td>
<td></td>
<td></td>
<td></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 ± 2.8†</td>
<td>36.2 ± 3.1†</td>
<td>32.9 ± 0.7</td>
<td>37.8 ± 1.37</td>
<td></td>
<td></td>
<td></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>
<td>25.3 ± 1.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRmax, beats/min</td>
<td>168 ± 2</td>
<td>165 ± 4</td>
<td>165 ± 7</td>
<td>165 ± 3</td>
<td>159 ± 3</td>
<td>163 ± 4</td>
<td></td>
<td></td>
<td></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>
<td>202 ± 18†</td>
<td></td>
<td></td>
<td></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>
<td>70.1 ± 1.1</td>
<td></td>
<td></td>
<td></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>
<td>43.8 ± 0.9</td>
<td></td>
<td></td>
<td></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>
<td>4.3 ± 0.1</td>
<td></td>
<td></td>
<td></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 Systems, 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, intermitted 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.

**BFB and SR measurements.** 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 turbidity 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 reported 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]p; colorimetry). Total albumin content in plasma ([Alb]tot) was determined as a product of PV and [Alb]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 regimen 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 Ta 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 VO2 peak for 20 min without fan cooling. Tsk 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 (Tsk) was determined as Tsk = 0.25-Tfa + 0.43-Tch + 0.32-Tair (25), where Tfa, Tch, and Tair, are skin surface temperature at the forearm, chest, and thigh measured with the thermocouples, respectively. SR was determined by capacitance plethysmography, calculated from the relative humidity and temperature of the air (THP-B3T, Shinee, Tokyo, Japan) flowing out of a 12.56-cm2 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 VO2 peak 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 mmHg−1·min−1·100 mmHg−1). Tsk, Tch, 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 Tsk thresholds for increasing SR (THSR) and increasing FVC (THFVC) were determined on each subject as the Tsk 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/Tsk) and FVC (FVC/Tsk) at a given increase in Tsk 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% VO2 peak 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 VO2 peak 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% VO2 peak for the 1st, 2nd, and 3rd wk, respectively; 70% VO2 peak for the 4th to 8th wk, 75% VO2 peak for the 9th to 10th wk; and 80% VO2 peak 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 Tsk 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, THSR, THFVC, SR/Tsk, and FVC/Tsk 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></th>
<th>RT ($n = 8$)</th>
<th></th>
<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>HR, beats/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>65 ± 4</td>
<td>67 ± 5†</td>
<td>63 ± 3††</td>
<td>68 ± 3</td>
<td>66 ± 4†</td>
<td>63 ± 4††</td>
</tr>
<tr>
<td>Ex5</td>
<td>116 ± 4</td>
<td>121 ± 5‡</td>
<td>119 ± 4†‡</td>
<td>115 ± 4</td>
<td>113 ± 4</td>
<td>117 ± 5‡</td>
</tr>
<tr>
<td>Ex20</td>
<td>133 ± 5</td>
<td>136 ± 6</td>
<td>136 ± 4†‡</td>
<td>127 ± 7</td>
<td>131 ± 7</td>
<td>131 ± 10</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>94 ± 5</td>
<td>87 ± 4‡</td>
<td>88 ± 4‡</td>
<td>98 ± 3</td>
<td>92 ± 4‡</td>
<td>91 ± 3‡</td>
</tr>
<tr>
<td>Ex5</td>
<td>141 ± 10</td>
<td>139 ± 10</td>
<td>142 ± 13</td>
<td>134 ± 5</td>
<td>125 ± 5‡</td>
<td>132 ± 10†</td>
</tr>
<tr>
<td>Ex20</td>
<td>145 ± 8</td>
<td>142 ± 10†</td>
<td>140 ± 16‡</td>
<td>124 ± 6</td>
<td>126 ± 7‡</td>
<td>127 ± 11</td>
</tr>
<tr>
<td>$T_{es}$, °C</td>
<td></td>
<td></td>
<td></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>
<td>37.2 ± 0.1</td>
<td>37.0 ± 0.2†</td>
<td>37.0 ± 0.2†</td>
</tr>
<tr>
<td>Ex5</td>
<td>37.4 ± 0.1</td>
<td>37.2 ± 0.1†</td>
<td>37.1 ± 0.1†‡</td>
<td>37.3 ± 0.2</td>
<td>37.1 ± 0.1‡</td>
<td>37.1 ± 0.2‡</td>
</tr>
<tr>
<td>Ex20</td>
<td>38.1 ± 0.1</td>
<td>38.2 ± 0.1</td>
<td>38.2 ± 0.1</td>
<td>37.9 ± 0.2</td>
<td>37.8 ± 0.1‡</td>
<td>37.8 ± 0.3‡</td>
</tr>
<tr>
<td>$T_{sk}$, °C</td>
<td></td>
<td></td>
<td></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>34.3 ± 0.1</td>
<td>33.9 ± 0.3</td>
<td>33.9 ± 0.2</td>
<td>33.9 ± 0.2</td>
</tr>
<tr>
<td>Ex5</td>
<td>33.6 ± 0.3</td>
<td>34.1 ± 0.2†</td>
<td>33.9 ± 0.2†</td>
<td>33.3 ± 0.4</td>
<td>33.2 ± 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>
<td>33.8 ± 0.3</td>
<td>33.7 ± 0.2</td>
<td>33.8 ± 0.5</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, $P < 0.01$.

RESULTS

Table 1 shows the physical characteristics, BV, and PV before and after training. After 8-wk training, $\dot{V}O_{2peak}$ 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]$_p$ 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 $\dot{V}O_{2peak}$ after training was weakly but significantly correlated with those in $TH_{SR}$ ($\Delta TH_{SR}$: $r = 0.30, P < 0.05$) and $TH_{FVC}$ ($\Delta TH_{FVC}$: $r = 0.34, P < 0.03$) but not with those in SR/$T_{es}$ ($\Delta (SR/T_{es})$; $P > 0.05$) or FVC/$T_{es}$ ($\Delta (FVC/T_{es})$; $P > 0.4$). In contrast, the change in BV (ABV) after training was significantly correlated with $\Delta (SR/T_{es})$ ($r = 0.51, P < 0.0005$) and $\Delta (FVC/T_{es})$ ($r = 0.45, P < 0.005$) (Fig. 2, A and B) but not with $\Delta TH_{SR}$ ($P > 0.1$) or $\Delta TH_{FVC}$ ($P > 0.3$). As shown in Fig. 3 (A and B), $\Delta TH_{SR}$ was significantly correlated with $\Delta TH_{FVC}$ ($r = 0.79, P < 0.0001$), and $\Delta (SR/T_{es})$ was significantly correlated with $\Delta (FVC/T_{es})$ ($r = 0.63, P < 0.0001$). As shown in Fig. 4, the change in Alb$_{tot}$ 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 TH FVC and THSR decreased with the increase in \( \dot{V}_O^2 \) peak, whereas FVC/Tes and SR/Tes 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 \( \dot{V}_O^2 \) peak than with increased BV, whereas changes in FVC/Tes and SR/Tes were more associated with that in BV than \( \dot{V}_O^2 \) peak.

THFVC and THSR After Training

As shown in Table 1, \( \dot{V}_O^2 \) 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 \( \dot{V}_O^2 \) peak. THFVC or THSR at a given absolute exercise intensity has been reported to decrease 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 \( \dot{V}_O^2 \) 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 \( \dot{V}_O^2 \) peak, equivalent to 60% of posttraining \( \dot{V}_O^2 \) peak. These results suggest that the reduction in THFVC and/or THSR after training was associated with reduced relative exercise intensity due to increased \( \dot{V}_O^2 \) peak.

![Fig. 1. Sweat rate (SR) and forearm skin vascular conductance (FVC) responses to increased esophageal temperature (Tes) during exercise in a warm environment with ambient temperature of 30°C and relative humidity of 50% at the intensity of 60% of pretraining peak oxygen consumption rate. A: control (C; circles). B: resistance training (RT; diamonds). C: aerobic training (AT; squares). Open symbols, before training; gray symbols, 8-wk training; solid symbols, 18-wk training. Bars indicate means ± SE. Regression analyses were performed on the measurements from 5 to 20 min after the start of exercise.](#)

Table 3. THSR, THFVC, SR/Tes, and FVC/Tes 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>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>8 wk</td>
<td>18 wk</td>
</tr>
<tr>
<td>THes, °C</td>
<td>37.34 ± 0.08</td>
<td>37.33 ± 0.04</td>
<td>37.22 ± 0.10</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>
</tr>
<tr>
<td>SR/Tes, mg·cm⁻²·min⁻¹·°C⁻¹</td>
<td>0.9 ± 0.2</td>
<td>0.7 ± 0.3</td>
<td>0.6 ± 0.2</td>
</tr>
<tr>
<td>FVC/Tes, units/°C</td>
<td>7.0 ± 2.0</td>
<td>6.5 ± 2.0</td>
<td>5.5 ± 1.7</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. THes, Tes threshold for increasing sweat rate (SR); THFVC, Tes threshold for increasing forearm skin vascular conductance (FVC); SR/Tes, slope of an increase in SR at a given increase in Tes; FVC/Tes, slope of an increase in FVC at a given increase in Tes. Values in RT after 18 wk training are for 6 subjects. *Significantly different from before training, \( P < 0.05 \). †Significantly different from before training, \( P < 0.01 \).
Vo2 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, Takanata 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% Vo2 peak per day for 6 days at 36°C of Tan 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

---

**Fig. 2.** Relationship between the change in slope of an increase in SR at a given increase in Tan [Δ(SR/Tan)] and change in blood volume (ΔBV) (A) and relationship between the change in slope of an increase in FVC at a given increase in Tan [Δ(FVC/Tan)] and ΔBV (B) after training. Open symbols, changes between before training and 8-wk training; solid symbols, changes between 8- and 18-wk training. ΔBV was significantly correlated with Δ(SR/Tan) (r = 0.51, P < 0.0005; y = 0.23x – 0.12) and also with Δ(FVC/Tan) (r = 0.45, P < 0.005; y = 2.44x – 0.80).

**Fig. 3.** Relationship between the change in Tan threshold for increasing FVC (ΔTHFVC) and Tan threshold for increasing SR (ΔTHSR) (A) and relationship between the change in Δ(SR/Tan) and Δ(FVC/Tan) (B) after training. Open symbols, changes between before training and 8-wk training; solid symbols, changes between 8-wk and 18-wk training. ΔTHSR was significantly correlated with Δ(SR/Tan) (r = 0.79, P < 0.0001; y = 0.86x – 0.00). Δ(SR/Tan) was significantly correlated with Δ(FVC/Tan) (r = 0.63, P < 0.0001; y = 0.10x – 0.04).
Albtot for older men (Table 1 and Fig. 4). The exercise training-induced hypervolemia has been suggested to be dependent on an increase in Albtot, 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 Albtot (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 Albtot. They suggested that the failure to increase Albtot 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 Albtot for the present study may be related to factors other than the active exercise training regimens, protein in diet (14), or heat acclimatization (27).

As shown in Table 1, the increased V̇O₂peak 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 V̇O₂peak 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 V̇O₂peak 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 V̇O₂peak 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, ΔBV was positively correlated with Δ(FVC/Tes) and Δ(SR/Tes). 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 [saline infusion (22), head-out water immersion (21), 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 V̇O₂peak 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 V̇O₂peak in older men.

The significant correlations between ΔTHFVC and ΔTHSR (Fig. 3A) and between Δ(FVC/Tes) and Δ(SR/Tes) (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 THFVC and THSR rather than by their increased slopes of FVC/Tes and SR/Tes, which was associated more with the increased V̇O₂peak 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 V̇O₂peak after training in older men.

We thank the volunteer subjects for participating in this study. We also thank Drs. A. Takamata, Y. Yanagidaira, A. Sakai, and H. Endoh for helpful comments and discussion on this study.

This study was supported in part by grants from the Ministry of Education, Science, Sports and Culture of Japan and Japan Space Forum.

**REFERENCES**


