Aerobic training and cutaneous vasodilation in young and older men

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Thomas, Carla M., Jane M. Pierzga, and W. Larry Kenney. Aerobic training and cutaneous vasodilation in young and older men. J. Appl. Physiol. 86(5): 1676-1686, 1999.—To determine the effect and underlying mechanisms of exercise training and the influence of age on the skin blood flow (SkBF) response to exercise in a hot environment, 22 young (Y; 18–30 yr) and 21 older (O; 61–78 yr) men were assigned to 16 wk of aerobic (A; YA, n = 8; OA, n = 11), resistance (R; YR, n = 7; OR, n = 3), or no training (C; YC, n = 7; OC, n = 7). Before and after treatment, subjects exercised at 60% of maximum oxygen consumption (VO2max) on a cycle ergometer for 60 min at 36°C. Cutaneous vascular conductance, defined as SkBF divided by mean arterial pressure, was measured at control (vasoconstriction intact) and bretylium-treated (vasoconstriction blocked) sites on the forearm using laser-Doppler flowmetry. Forearm vascular conductance was calculated as forearm blood flow (venous occlusion plethysmography) divided by mean arterial pressure. Esophageal and skin temperatures were recorded. Only aerobic training (functionally defined a priori as a 5% or greater increase in VO2max) produced a decrease in the mean body temperature threshold for increasing forearm vascular conductance (36.89 ± 0.08 to 36.63 ± 0.08°C, P < 0.003) and cutaneous vascular conductance (36.91 ± 0.08 to 36.65 ± 0.08°C, P < 0.004). Similar thresholds between control and bretylium-treated sites indicated that the decrease was mediated through the active vasodilator system. This shift was more pronounced in the older men who presented greater training-induced increases in VO2max than did the young men (22 and 9%, respectively). In summary, older men improved their SkBF response to exercise-heat stress through the effect of aerobic training on the cutaneous vasodilator system.

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response were mediated by the VC or VD system. We hypothesized that 1) endurance exercise training would enable a higher SkBF at a given Tp during exercise-heat stress, 2) a decrease in Tp threshold for vasodilation would facilitate these improvements, and 3) increased daily activity insufficient to raise VO\textsubscript{2max} (resistance training) would not alter SkBF responses.

**METHODS**

Subjects. This study was approved by the Institutional Review Board for the Protection of Human Subjects of the Pennsylvania State University. Subjects were given a detailed explanation of all experimental procedures, and informed consents were obtained before testing began. Twenty-two young (Y; 18–30 yr) and 21 older (O; 61–78 yr) men were recruited from central Pennsylvania to participate in this study. All subjects were screened by a physician, and body composition was estimated from skinfold thickness at seven sites (triceps, subscapular, pectoral, midaxillary, suprailiac, abdominal, and thigh). Subjects within each age group were assigned to 1) an aerobic training (A; YA, n = 8; OA, n = 11), 2) a resistance training (R; YR, n = 7; OR, n = 3), or 3) a no training (C; YC, n = 7; OC, n = 7) group. Aerobic training was functionally defined, a priori, as a 5% or greater increase in VO\textsubscript{2max} to establish the presence of a stimulus that could induce the changes in SkBF. At completion of data collection, seven of the subjects in the two A groups did not increase VO\textsubscript{2max}. Therefore, the A groups were subdivided: those that increased their VO\textsubscript{2max} (YA\textsubscript{5%}, n = 5; OA\textsubscript{5%}, n = 7) and those that did not (YA, n = 3; OA, n = 4) (Table 1).

For all subjects, two maximal graded exercise tests were performed before training, ensuring that the men were comfortable with the protocol, and an accurate measure of initial VO\textsubscript{2max} was obtained. The greater value for VO\textsubscript{2max} as measured during the graded treadmill tests by analysis of the subjects’ expired gases (Medical Gas Analyzer LB-2, Beckman; and S-3A Oxygen Analyzer, Applied Electrochemistry), was used. Young subjects chose a comfortable running pace that they maintained throughout the test as the grade of the motor-driven treadmill increased 2% every 2 min. A Polar monitor (Polar CIC, Port Washington, NY) measured heart rate (HR). Older subjects chose a comfortable walking pace that they maintained while the grade of the treadmill was increased at 2-min intervals by 2 or 2.5%, depending on the participant’s HR as recorded by electrocardiogram (Case 15, Marquette Electronics, Milwaukee, WI). Blood pressure was measured by brachial auscultation.

Criteria for exclusion from the study included 1) an abnormal electrocardiogram during the graded exercise test, 2) hypertension (resting systolic pressure >140 mmHg or diastolic pressure >90 mmHg), 3) smoking, 4) diagnosed metabolic or cardiovascular diseases, or 5) the taking of medication that may interfere with or influence thermoregulatory or cardiovascular variables of interest.

Experimental timeline. This study ran over the course of 1 yr with subjects from both age groups entering at random times to minimize seasonal effects on thermoregulation. After the initial screening and two graded exercise tests, subjects performed an exercise-heat-stress protocol (described in Exercise-heat-stress protocol). After the exercise-heat-stress protocol, the A and R groups were trained in a supervised setting for 16 wk. The C group did not train and was instructed to maintain their present level of daily activity. After 16 wk, a final graded exercise test and exercise-heat-stress protocol were performed.

Exercise-heat-stress protocol. To promote euhydration, subjects were instructed to refrain from consuming any alcohol or caffeine during the evening preceding the exercise-heat-stress protocol. When subjects arrived at the laboratory, bretylium tosylate iontophoresis was administered locally to block VC activity at two sites on the right forearm (13), with the second site providing an alternate if the VC blockade at the first was incomplete. Bretylium tosylate (100 mM) was dissolved in doubly distilled (18.3 MΩ·cm) water (NANOpure, Barnstead, Dubuque, IA) and iontophoresed for 40 min

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**Table 1. Subject characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
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<tr>
<td></td>
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<td>YR (n = 7)</td>
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<td>94 ± 5</td>
<td>91 ± 4</td>
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<td>VO\textsubscript{2max}, ml·kg\textsuperscript{-1}·min\textsuperscript{-1} Pre</td>
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<td>45.7 ± 3.0</td>
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Values are means ± SE; n, no. of subjects. Young aerobic group of subjects who demonstrated at least a 5% increase in maximum oxygen consumption (VO\textsubscript{2max}) (YA\textsubscript{5%}), young aerobic with <5% increase in VO\textsubscript{2max} (YA), young control group (YC), older aerobic group of subjects who demonstrated a 5% increase in VO\textsubscript{2max} (OA\textsubscript{5%}), older aerobic with <5% increase in VO\textsubscript{2max} (OA), older resistance training group (OR), and older control group (OC). MAP, mean arterial pressure. Pre and post, before and after training, respectively. †Differences at P < 0.05 between older group and all young groups. ‡Difference at P < 0.05 between pretraining value and postraining value within a group.
over a 3-cm² area of skin by using alternating current (Lectro Patch, General Medical, Los Angeles, CA). A third site on the same forearm (3 cm²) was iontophoresed with doubly distilled water to serve as a control with intact VC and VD systems.

After 1 h, blockade of VC was verified by whole body cooling with the use of a water-perfused suit. Subjects were instrumented with laser-Doppler flowmetry probes (DTR4 Laser Doppler Perfusion and Temperature Monitor, Moor Instruments), which measure an index of SkBF (flux) at a bretylium site and at the control site. Laser-Doppler flux (LDF) readings reflect only SkBF and are not influenced by underlying muscle blood flow (28). After a 5- to 10-min baseline of steady LDF readings, ice water was circulated through the suit for 3 min as LDF, HR, and mean arterial pressure (MAP) were monitored. VC blockade was considered successful if LDF decreased at the control site and remained stable or increased at the bretylium site. If the blockade was incomplete, the second bretylium site was tested in a similar fashion. The experiment continued only if the VC blockade was successful.

After successful VC blockade, the subjects removed the water-perfused suit, voided, and provided an initial nude body weight (±10 g). To measure an index of core temperature ($T_c$), a thermistor was inserted in the esophagus to 25% of the subject's height (22), a depth corresponding to the level of the right atrium. At this time, subjects drank 5 ml water/kg body wt —30 min before exercise to promote adequate hydration. No additional fluids were allowed until the exercise-heat stress protocol was completed. At the end of the experiment, subjects provided a final nude weight. The difference between pre- and postexperiment weights, corrected for fluid ingestion, was reported as an estimate of sweat loss.

Subjects entered a preconditioned environmental chamber (36°C dry bulb; 24°C wet bulb, 40% relative humidity) and rested in a seated position on a cycle ergometer while the blood pressure monitor, plethysmograph, and four T-type skin thermocouples (calf, thigh, chest, and arm) were applied (15 min). After instrumentation, resting baseline measurements were recorded for 10 min. MAP, HR, $T_{es}$, weighted mean skin temperature ($T_{sk}$), and LDF were monitored continuously throughout the resting baseline and exercise periods. In addition, forearm blood flow (FBF) was recorded at 2-min intervals during baseline and exercise (33) (see Measurements).

After baseline, subjects exercised on a cycle ergometer for 60 min at 60% of their VO2max using workloads calculated from the results of their previous graded exercise test. Each subject began cycling at the same resistance (0.5 kp), which was subsequently increased 0.5 kp every 2 min until the workload corresponding to 60% of VO2max was attained. After the exercise period, the resistance was decreased, and subjects continued cycling slowly to maintain MAP for 10–15 min. During the cool down, local heating was performed by thermostatically controlled heated probe holders at the laser-Doppler sites on the forearm. Local $T_{sk}$ was increased to 42.5–43°C and maintained for 30 min to obtain a site-specific maximal LDF that was verified by subsequent occlusion of arm blood flow for 5 min and was monitored for resulting reactive hyperemia (18).

Measurements. $T_{sk}$ was recorded as the minute average of readings collected at 300 per minute throughout baseline and exercise from a thermistor sealed in an infant feeding tube. $T_{sk}$ was similarly averaged over every minute as the weighted sum of four uncovered sites, chest ($T_ch$), upper arm ($T_a$), thigh ($T_th$), and calf ($T_leg$), such that $T_{sk} = 0.3·T_{ch} + 0.2·T_a + 0.3·T_th + 0.2·T_leg$ (25). $T_{es}$ was calculated as $T_{es} = 0.9·T_{es} + 0.1·T_{sk}$ (30). HR and MAP were continuously monitored by using a Finapres blood pressure cuff (Finapres BP Monitor 2300, Ohmeda, Louisville, CO) placed on the middle finger of the right hand. LDF (DTR4 Laser Doppler Perfusion and Temperature Monitor, Moor Instruments) was recorded as an average of 60 readings every minute and is expressed as cutaneous vascular conductance (CVC) (CVC = LDF/MAP). In addition, CVC data obtained during exercise were expressed as a percentage of the maximum CVC (%CVCmax) determined during postexercise local heating. Measured variables were recorded and stored on a computer (Macintosh Quadra 650, Apple Computer, Cupertino, CA) by using SuperScope II (GW Instruments, Somerville, MA) data-acquisition system.

FBF was measured by using venous occlusion plethysmography with a mercury-in-Silastic strain gauge (EC4 Plethysmograph, Hokanson, Bellvue, WA) (33) placed around the middle of the subject’s left forearm. During dynamic leg exercise, a rise in FBF reflected an increase in cutaneous blood flow, as the perfusion of underlying resting muscle does not change (11). A set of four FBF measurements was recorded over 60 s at 2-min intervals during baseline. At the onset of exercise, three readings over 45 s were recorded at 1-min intervals until the subject’s final workload was reached. At this point, four FBF measurements were performed at 2-min intervals for the duration of the exercise period. During the readings, a blood pressure cuff around the left wrist inflated to a supra-arterial pressure (~200 mmHg), preventing blood flow to the hand, whereas a second blood pressure cuff around the upper arm cycled between inflation (~50 mmHg, 10 s) and deflation (5 s) (E20 Rapid Cuff Inflator, Hokanson). Forearm vascular conductance (FVC) was calculated as FBF/MAP and reported in units of milliliters per 100 milliliters per minute per 100 mmHg.

Exercise training. Subjects trained four times per week for 16 wk (3 supervised and 1 unsupervised session). The group achieved 60–80% of their maximal HR for 30–60 min per session as monitored by the subjects with regular checks by the supervisor. Training programs were reevaluated through examination of daily training records, and workloads were adjusted every 1–2 wk to ensure that subjects maintained their target HR while exercising. During workouts, subjects used treadmills (Precor Commercial Treadmill, Precor), cycle ergometers (Spinnaker Systems, StairMaster Sports/Med Products), rowers (Concept II, Morrisville, VT), and stair climbers (Climbing Systems, StairMaster Sports/Med Products).

The R group performed one-repetition maximums (1 RMs) at the beginning of their programs. 1 RM was defined as the maximal amount of weight that a subject could move through his full range of motion. Subjects trained at 60% of their 1 RM during workouts that consisted of leg extension and flexion, chest press, upper back extension (Keiser K 300s, Keiser Health and Fitness, Fresno, CA), and assisted pull-ups and dips (Gravitron 2000, StairMaster Sports/Med Products) with training sessions lasting ~30 min. Also a 5-min warm-up and cooldown on the cycle ergometers was included in each session. 1-RM measurements were taken at 3- to 4-wk intervals, and the subjects’ programs were adjusted so that they continued working at 60% of their 1 RMs. Overall body strength was calculated as the average of 1 RMs from each of the different workout components (leg extension and flexion, chest press, upper back extension, and assisted pull-ups and dips).

The C group did not participate in any training regimen. They were instructed to maintain their present level of daily activity throughout the 16-wk period.

Statistical analysis. Data are presented as means ± SE. Descriptive plots of $T_{sk}$, HR, MAP, and $T_{es}$ vs. exercise time...
were analyzed as follows. The independent variable (time) was partitioned into seven bins of 10 min. For each subject, the variables were averaged within each 10-min bin, and a repeated-measures analysis of variance model was fit to the data. Group was the between-subject factor, and binned time was the within-subject factor.

The descriptive plots of FVC vs. Tb and %CVCmax vs. Tb showed a functional response that exhibited consistent features between curves. Four independent raters identified the four features of each blinded plot: baseline, threshold, slope, and plateau (Fig. 1). The average of the estimates from each rater for FVC had interrater reliabilities of 1.0, 0.97, 0.94, and 1.0, and for %CVCmax had reliabilities of 0.99, 0.97, 0.96, and 1.0 for the baseline, threshold, slope, and plateau, respectively. The average estimate of each feature from the four raters was used as the dependent variable in the repeated-measures analysis of variance to examine the effect of group and treatment in the FVC model with condition being added as a third parameter in the model for %CVCmax.

To describe subject characteristics, paired t-tests were performed on VO2max 1 RM of each workout component and overall strength (R groups only), and body composition variables pre- to posttreatment. A one-way analysis of variance was performed to determine differences between the VO2max values of Y and O groups, 1 RM of each workout component and overall strength (R groups only), body composition, age, and height. For all analyses, an α-level of 0.05 was used as the criterion for statistical significance of factors and their interactions. A one-tailed test was performed on the data for threshold because only a leftward shift in the value from pre- to posttreatment was of interest.

RESULTS

As shown in Table 1, the older subjects had a significantly (P < 0.05) lower VO2max and a higher percent body fat compared with the young subjects. The only significant age difference pretraining between the YR and OR groups was in the leg extension exercise (YR, 23 ± 4 kg and OR, 13 ± 3 kg, P < 0.05). There were no changes in percent body fat from pre- to postraining in any of the groups, although some groups did show changes in total body weight. The YR group increased total body weight pre- to postraining (79.2 ± 7.1 to 80.2 ± 7.2 kg, P < 0.05), and the YC group increased fat-free weight (FFW) pre- to postraining (68.5 ± 3.1 to 69.1 ± 3.0 kg, P < 0.05; Table 1). In contrast, both the OA5% and OA groups decreased total body weight from pre- to postraining (OA5%: 80.6 ± 3.0 to 78.2 ± 2.8 kg, OA: 81.2 ± 6.4 to 80.2 ± 6.1 kg, P < 0.05; Table 1).

Aerobic training increased VO2max by 9% in the YA5% group (45.4 ± 1.4 to 49.3 ± 1.8 ml·kg⁻¹·min⁻¹, P < 0.05) and by 22% in the OA5% group (24.8 ± 1.3 to 30.2 ± 1.2 ml·kg⁻¹·min⁻¹, P < 0.05). As expected, no change in VO2max occurred pre- to postraining for any other group (Table 1). Resistance training significantly (P < 0.05) increased overall strength pre- to postraining by 41%, independent of age. The YR group increased (P < 0.05) overall strength by 31%, and the OR group increased (P < 0.05) overall strength by 57%.

After aerobic training, Tb was lower during baseline and throughout the exercise-heat stress, regardless of age. However, whereas Tb in the OA5% group was also lower throughout the entire exercise-heat-stress protocol after training, Tb remained unchanged in the YA5% group (Fig. 2). There were no differences in HR, MAP, or Tsk in any of the groups pre- to postraining.

During the pretreatment exercise-heat stress, the plateau FVC values were significantly lower in the O than in the Y group (Y: 18.3 ± 1.1 FVC units, O: 12.3 ± 1.1 FVC units, P < 0.05; Fig. 3A). Also, the slope of the FVC-Tb relationship was also lower in the O than in the Y group (Y: 21 ± 2 ΔFVC unit/Δ°C, O: 12 ± 1 ΔFVC unit/Δ°C, where Δ is change, P < 0.05; Fig. 3A). There was no difference in threshold between the Y and O groups before treatment (Y: 36.78 ± 0.04°C, O: 36.77 ± 0.07°C; Fig. 3A).

Like that of the FVC-Tb relationship, the slopes of %CVCmax-Tb at both the control (Fig. 3B and Table 2) and bretylium sites before training were significantly
lower in the combined older subjects than in the combined younger subjects (control: Y: 120 ± 10 Δ%CVC\textsubscript{max}/Δ°C; O: 79 ± 9 Δ%CVC\textsubscript{max}/Δ°C; bretylium: Y: 115 ± 12 Δ%CVC\textsubscript{max}/Δ°C; O: 70 ± 7 Δ%CVC\textsubscript{max}/Δ°C; P < 0.05). However, unlike that of FVC, the baseline of %CVC\textsubscript{max} was higher in the combined group of O than in the combined group of Y (Y: 17.3 ± 1.8 %CVC\textsubscript{max}; O: 23.1 ± 1.8 %CVC\textsubscript{max}, P, 0.05; Fig. 3B and Table 2).

Aerobic training produced a leftward shift in the \(T_b\) threshold for vasodilation for the FVC-\(T_b\) relationship in the combined YA5% and OA5% groups (36.89 ± 0.08 to 36.63 ± 0.06°C, P < 0.003; Fig. 4A). This leftward shift in threshold was also significant in the %CVC\textsubscript{max}-\(T_b\) relationship for the combined YA5% and OA5% group (36.91 ± 0.08 to 36.65 ± 0.08°C, P < 0.004; Fig. 4B). Unlike that of FVC, the %CVC\textsubscript{max} plateau was significantly higher after than before aerobic training in the combined YA5% and OA5% group (66.3 ± 3.8 to 73.0 ± 3.4 %CVC\textsubscript{max}, P < 0.05; Fig. 4B).

Furthermore, the OA5% group's FVC-\(T_b\) threshold shifted leftward after aerobic training (36.92 ± 0.12 to 36.60 ± 0.08°C, P < 0.001; Fig. 5), whereas that of the YA5% group tended toward a similar decrease in threshold (36.86 ± 0.12 to 36.67 ± 0.11°C, P < 0.07; Fig. 5). There were, however, no other significant differences between pre- and posttraining for the FVC-\(T_b\) relationship in any of the other groups.

The leftward shift in the %CVC\textsubscript{max}-\(T_b\) threshold after aerobic training was also significant for both control and bretylium-treated sites in the OA5% group (control: 36.94 ± 0.13 to 36.59 ± 0.08°C, bretylium: 37.04 ± 0.12 to 36.77 ± 0.16°C, P < 0.001; Fig. 6 and Table 2). Aerobic training also increased the %CVC\textsubscript{max} plateau at the control site in the OA5% group (68.7 ± 3.9 to 77.7 ± 2.2 %CVC\textsubscript{max}, P < 0.05; Fig. 6 and Table 2).

Before training and independent of treatment, the younger subjects' %CVC\textsubscript{max} plateau at the bretylium-treated site was significantly lower than that of the corresponding control site (control: 68.1 ± 2.9 %CVC\textsubscript{max}, bretylium: 58.7 ± 3.0 %CVC\textsubscript{max}, P < 0.05; Table 2). In older subjects before training, the \(T_b\) threshold at the bretylium-treated site was higher than that of the control site (control: 36.75 ± 0.08°C, bretylium: 36.89 ± 0.08°C, P < 0.05; Table 2).

In the pretrained OC group, the baseline at the bretylium-treated site was lower than the baseline at...
the control site (control site: 25.1 ± 3.7 %CVC\textsubscript{max}, bretylium site: 18.1 ± 2.4 %CVC\textsubscript{max}, P < 0.05; Table 2). The plateaus in the YA5%, YR, and OC groups at the bretylium-treated sites were lower than those of the corresponding control sites (YA5% control: 63.0 ± 7.6 %CVC\textsubscript{max}, YA5% bretylium: 58.0 ± 13.1 %CVC\textsubscript{max}, YR control: 68.5 ± 4.8 %CVC\textsubscript{max}, YR bretylium: 58.1 ± 3.6 %CVC\textsubscript{max}, OC control: 79.5 ± 4.0 %CVC\textsubscript{max}, OC bretylium: 67.9 ± 4.0 %CVC\textsubscript{max}, P < 0.05). In post-trained OA5% and OA groups, the plateaus of the bretylium-treated sites were lower than the respective plateau values of the control sites (OA5% control: 77.7 ± 2.2 %CVC\textsubscript{max}, bretylium: 61.5 ± 5.1 %CVC\textsubscript{max}, OA control: 68.0 ± 7.5 %CVC\textsubscript{max}, bretylium: 57.5 ± 10.6 %CVC\textsubscript{max}, P < 0.05).

**DISCUSSION**

The major findings of the present study were 1) aerobic training shifted the \( T_a \) threshold for cutaneous vasodilation (leftward to a lower \( T_a \)), 2) this leftward shift in threshold was due to adaptations in the active VD system, 3) an aerobic training stimulus sufficient to increase \( VO_2\text{max} \) appeared to be necessary for the shift to occur, and 4) older (>60 yr) subjects were able to improve their SkBF response to an exercise-heat stress through a 4-mo aerobic training program.

Exercise training. The aerobic training program was effective in increasing the \( VO_2\text{max} \) of the YA5% and OA5% groups by 9 and 22%, respectively (Table 1). The increase in \( VO_2\text{max} \) of the YA5% group reported here is
slightly less than what Roberts et al. (26) observed in young men after 10 days of aerobic training (13%). When comparing data from these two studies, the majority of our YA5% group began with a higher VO$_2$max than that of the subjects in the Roberts et al. project; therefore, for a given absolute increase in VO$_2$max, a greater percent change in VO$_2$max occurred in the previous study (26). In short, the posttraining VO$_2$max of our subjects was similar to the individual pretraining VO$_2$max of their subjects.

The YR and OR groups did not exhibit a change in VO$_2$max after training, but strength increased significantly from pre- to posttraining, indicating that an increase in daily activity occurred. This allowed for comparison with the A groups (YA5% and OA5%) to determine whether an aerobic training stimulus was necessary for improvements in SkBF response to an exercise-heat stress.

Although the YR group's weight increased pre- to posttraining, high variability in individual measurements prevented a similar increase in FFW from reaching statistical significance. In contrast, the YC group increased FFW from before to after the 4-mo period with no change in body weight or percent body fat. Because determination of FFW depended on skinfold analysis, the variability in skinfold measurements made it difficult to interpret these results accurately. Furthermore, the OA5% and OA groups decreased body weight after aerobic training but presented no other significant differences between pre- and posttraining values. This would suggest either that the aerobic training decreased fat weight and FFW in the same proportion or that the skinfold technique was not sensitive enough to detect small changes in body composition.

Aging and SkBF. As in previous experiments in our laboratory (8, 15, 16), older subjects exhibited a diminished rise in FVC (Fig. 3A), in %CVC$_{max}$ at control sites (see Fig. 3B and Table 2), and in %CVC$_{max}$ at bretylium-treated sites (Table 2) for a given increase in T$_b$ during the pretraining exercise-heat stress compared with the younger subjects (Fig. 3). In one of the earlier studies, Kenney et al. (16) blocked VC using bretylium tosylate in both young and older men who underwent an
exercise-heat stress similar to that employed in the present experiments. If the VC system were responsible for the diminished rise in SkBF, then the slope of the line for the %CVC\textsubscript{max}-\(T_b\) relationship at the bretylium-treated site would be steeper, indicating an increased sensitivity to the rise in \(T_b\) in the absence of VC. This was not the case in either the research by Kenney et al. (16) or our present study; therefore, an attenuation in the VD system rather than enhanced VC was responsible for this diminished increase in SkBF in aged skin. In addition, older subjects achieved a lower absolute SkBF, which is probably a result of structural changes in the microcirculation of the skin (18).

Interestingly, older and young men achieved a similar percentage of their respective maximal conductances at both bretylium and control skin sites (Fig. 3B and Table 2) during the plateau phase of the %CVC\textsubscript{max} vs. \(T_b\) curve. However, the absolute SkBF attained during the exercise-heat stress was 30% lower in O than in Y groups (Fig 3A) (30). This suggests that, although older subjects have decreased maximal skin vasodilation (18), they are able to achieve the same percentage of maximum SkBF. Observations that bretylium treatment did not abolish the plateau suggested that control of this phase is mediated through active VD (12). Although the mechanism of plateau-phase initiation through active VD is unknown, some suggest that low-pressure baroreceptors may promote reduced vasodilation (3, 12) to preserve MAP.

In summary, older subjects had a reduced absolute increase in SkBF (i.e., lower FVC plateau values) than that of young subjects during an exercise-heat stress, although the older subjects achieved the same percentage of maximal vasodilatory capacity as the young subjects. This attenuated rise in SkBF seen in older subjects was due to decreased active VD sensitivity to changes in \(T_b\), rather than increased VC activity.

Aerobic training and SkBF. Previous studies have demonstrated that trained individuals exhibited a greater SkBF response to an exertional heat stress but
explained this increased response through diverse mechanisms (8, 26, 32) such as alterations in \( T_b \) threshold for vasodilation (8, 26) or changes in the slope of SkBF-\( T_b \) relationship (32). For example, a cross-sectional study undertaken by Tankersley et al. (32) found no differences between sedentary and highly fit older men in the \( T_b \) threshold for vasodilation during an exercise-heat stress. However, the slope of the FBF-\( T_b \) relationship was steeper in the highly fit group, although not statistically different. The researchers suggested that enhanced sensitivity of the SkBF-\( T_b \) relationship was responsible for the greater increases seen in SkBF for a given change in \( T_b \) in trained individuals (32). In another cross-sectional study, Ho et al. (8) compared young trained and sedentary men during an exercise-heat stress. The \( T_b \) threshold for vasodilation was shifted leftward in the trained group, but no differences in slope (sensitivity) occurred. However, when they aerobically trained a small group of older subjects for 4 wk, they observed an increase in SkBF because of a greater slope of the FBF-\( T_{es} \) relationship (8), thus adding to the controversy. In the longitudinal study by Roberts et al. (26), training produced an approximately \( -0.20^\circ C \) leftward shift in the \( T_{es} \) threshold for vasodilation. Similarly, in our present study, 4 mo of training produced an approximately \(-13\%\) increase in \( V_{O2max} \) and an approximately \(-0.30^\circ C \) leftward shift in threshold, independent of age. Therefore, our results suggest that aerobic training shifts the \( T_b \) threshold to a higher internal \( T_b \) for an exercise-heat stress observed in aerobically trained men is due to a decrease in \( T_b \) threshold for vasodilation rather than to an increase in the sensitivity (slope) of the SkBF-\( T_b \) relationship.

We explored the mechanism underlying the leftward shift in \( T_b \) threshold and enhanced SkBF response observed after aerobic training by utilizing bretylium tosylate iontophoresis to inhibit the local release of norepinephrine, thereby blocking VC activity at one site on the skin of the forearm while preserving normal VC function at an adjacent control site. The \( T_b \) threshold shifted leftward at both the control and bretylium-treated sites; therefore, we concluded that the shift was mediated through the active VC system. A lower \( T_b \) during baseline and a decreased \( T_b \) threshold for vasodilation after aerobic training may impart a thermoregulatory advantage during the exercise-heat stress through reduced \( T_b \) and greater SkBF for any given \( T_b \). The magnitude of the change in \( T_b \) from baseline necessary to elicit vasodilation in response to exercise-heat stress remained approximately the same after training so that the relative range in which \( T_b \) was regulated was unchanged. This resulted in a lower threshold for vasodilation. Furthermore, the increase in \( T_b \) from baseline to 60 min of exercise-heat stress was the same pre- and posttraining, indicating that thermoregulatory drive for vasodilation remained constant.

Heat acclimation has been shown to lower \( T_b \) at rest and during an exercise-heat stress (7, 21, 24, 26), and exercise training has been hypothesized to produce a state of partial heat acclimation (1, 2, 24). When Piwonka et al. (24) examined the responses of runners who had been training throughout the winter and compared their responses with a control group, the group of runners responded to an exercise-heat stress as though they were partially acclimated (i.e., lower HR, lower \( T_b \), and greater sweat production). Interestingly, a swimmer participating in the same study responded similarly to the untrained group even though he was considered to be a trained subject. This supported the suggestion that training that failed to increase \( T_b \) [i.e., in water (1) or in a cold environment (7)] did not result in the same thermoregulatory adaptations as training that increased \( T_b \). Our study illustrates that exercise training resulted in partial heat acclimation because aerobic training decreased \( T_b \) throughout the exercise-heat-stress protocol as well as for the 10-min baseline. Because heat acclimation due to seasonal changes could confound our results, subjects entered the 16-wk training program at random times throughout the year so that environmental effects on thermoregulation were minimized and the thermoregulatory adaptations observed in the study were due to exercise training.

Aerobic training sufficient to raise \( V_{O2max} \) increased the plateau \( %CVC_{max} \) at control skin sites during the exercise-heat stress (Fig. 4B), independent of age, without increases in the maximal absolute SkBF (FVC) achieved during the exercise-heat stress. This suggested the unlikely conclusion that the maximal diameter of the skin's vessels decreased after aerobic training. However, no data available in the literature supports a decrease in vessel diameter with aerobic training. More likely, variability in the data may have masked any small changes in the FVC values that occurred during the plateau phase.

With aerobic training, other adaptations occur that affect thermoregulatory function. Increases in plasma volume (4, 23), stroke volume (29, 31), and Qc (8, 31) occur as a result of exercise training, and all impact thermoregulatory function. In states of hypohydration (20) and hyperosmolality (5, 6), the \( T_b \) threshold for vasodilation is shifted to a higher internal \( T_b \). Exercise training that increases plasma volume (4, 23) could possibly decrease the \( T_b \) threshold by making more blood volume available for redistribution to the skin at any given \( T_b \). In a study by Ho et al. (8), redistribution of Qc was measured after aerobic training of four sedentary older subjects. Training produced an increase in plasma volume, resulting in a rise in stroke volume, which, in turn, enhanced Qc. The increased Qc was deemed responsible for the higher SkBF observed during posttraining assessment because no change in renal or splanchnic blood flow occurred.

An aerobic training stimulus was necessary to improve the SkBF response to an exercise-heat stress in our study. To ensure the reliability of the \( V_{O2max} \) determinations, the men performed two graded exercise treadmill tests before the 16-wk treatments. The inclusion of a resistance-trained, active control group that increased their daily activity in parallel with the aerobic group but did not increase their \( V_{O2max} \) and did not experience a leftward shift in the threshold for
vasodilation showed that simply increasing daily activity was insufficient to illicit these adaptations. Furthermore, the subsets of YA and OA that failed to increase \( V_{O2\text{max}} \) by more than 5% did not demonstrate a leftward shift in the threshold for vasodilation. Although no direct measure of \( T_b \) was obtained during exercise sessions, it is possible these men neither increased their \( T_b \) daily nor stressed their cardiorespiratory system to the same extent as those whose \( V_{O2\text{max}} \) did increase. Because increased \( T_b \), which probably accompanies aerobic exercise (1, 2, 7), and increased cardiorespiratory performance are two important components in improving thermoregulatory function during an exercise-heat stress, those subjects who did not stress these systems during the 16-wk training may not have induced the changes in these components necessary to enhance thermoregulatory capability.

Aging, training, and SkBF. Previous studies have not addressed the question of whether older subjects’ SkBF response can adapt to the same extent as that of younger subjects. The present study answers that question and provides insight into the mechanism by which it is accomplished. When age was included as an independent factor in the analysis of the data, only the OA5% group showed statistically significant (P < 0.05) improvements in the SkBF response to the exercise-heat stress (Figs. 5 and 6). The OA5% group, which achieved a 22% increase in \( V_{O2\text{max}} \), demonstrated a leftward shift (P < 0.001) of ~0.30°C in the \( T_b \) threshold for vasodilation after training. This shift in threshold was similar to that of the young subjects (~13% increase in \( V_{O2\text{max}} \) whose threshold decreased by ~0.20°C in the study by Roberts et al. (26). Although our YA5% group did not decrease their vasodilation threshold significantly, this may not indicate that older subjects are better at improving thermoregulatory responses but rather that a greater change in \( V_{O2\text{max}} \) may produce a greater leftward shift in threshold. Furthermore, in the OA5% group, as in the combined YA and OA group, the \( T_b \) shifted leftward at both control (Fig. 6 and Table 2) and bretylium-treated sites (Table 2), again confirming our hypothesis that adaptations that occur after training are mediated through the active VD system in older subjects.

The OA5% group’s \( T_b \) was significantly lower throughout the exercise-heat-stress protocol after training (Fig. 2B). This, in combination with the lower \( T_b \) threshold for vasodilation, decreased the final \( T_b \) at the end of the exercise-heat-stress protocol. Once again, this did not occur in the YA5% group, indicating that a minimal aerobic stimulus may be necessary to sufficiently stress the thermoregulatory and cardiorespiratory systems to see improvements in SkBF response to an exercise-heat stress.

Bretyllium-treated site parameters (baseline, threshold, slope, and plateau) differed from control site parameters within different subject groups (Table 2). These differences cannot be explained at this time, and a pattern of differences does not appear to be evident.

In summary, the leftward shift in \( T_b \) threshold for vasodilation to a lower value after aerobic training was mediated through the active VD system as \( T_b \) was centrally regulated at a lower temperature in the hypothalamus. For this leftward shift in threshold to occur, an aerobic training stimulus necessary to increase \( V_{O2\text{max}} \) by 5% or greater appeared to be necessary. This observation supported the proposal by others that exercise training that increased \( T_b \) daily may be a form of partial heat acclimation. Finally, older men (61–78 yr) improved their SkBF response to an exercise-heat stress through a leftward shift in \( T_b \) threshold for vasodilation after a 4-mo aerobic training program.

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