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Time course and mechanisms of adaptations in cardiorespiratory fitness with endurance training in older and young men

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Murias JM, Kowalchuk JM, Paterson DH. Time course and mechanisms of adaptations in cardiorespiratory fitness with endurance training in older and young men. *J Appl Physiol* 108: 621–627, 2010. First published January 7, 2010; doi:10.1152/japplphysiol.01152.2009.—The time-course and mechanisms of adaptation of cardiorespiratory fitness were examined in 8 older (O) (68 ± 7 yr old) and 8 young (Y) (23 ± 5 yr old) men pretraining and at 3, 6, 9, and 12 wk of training. Training was performed on a cycle ergometer three times per week for 45 min at ~70% of maximal oxygen uptake ($\dot{V}O_{2\,max}$). $\dot{V}O_{2\,max}$ increased within 3 wk with further increases observed posttraining in both O (+31%) and Y (+18%), ($P < 0.05$). Maximal cardiac output ($\dot{Q}_{max}$, open-circuit acetylene) and stroke volume were higher in O and Y after 3 wk with further increases after 9 wk of training ($P < 0.05$). Maximal arterial-venous oxygen difference (a-vO_{2diff}) was higher at *weeks 3* and *6* and posttraining compared with pretraining in O and Y ($P < 0.05$). In O, ~69% of the increase in $\dot{V}O_{2\,max}$ from pre- to posttraining was explained by an increased $\dot{Q}_{max}$ with the remaining ~31% explained by a widened a-vO_{2diff}. This proportion of $\dot{Q}$ and a-vO_{2diff} contributions to the increase in $\dot{V}O_{2\,max}$ was consistent throughout testing in O. In Y, 56% of the pre- to posttraining increase in $\dot{V}O_{2\,max}$ was attributed to a greater $\dot{Q}_{max}$ and 44% to a widened a-vO_{2diff}. Early adaptations (first 3 wk) mainly relied on a widened maximal a-vO_{2diff} (~66%) whereas further increases in $\dot{V}O_{2\,max}$ were exclusively explained by a greater $\dot{Q}_{max}$. In conclusion, with short-term training O and Y significantly increased their $\dot{V}O_{2\,max}$; however, the proportion of $\dot{V}O_{2\,max}$ increase explained by $\dot{Q}_{max}$ and maximal a-vO_{2diff} throughout training showed a different pattern by age group.

aging; maximal oxygen uptake; cardiac output; arterial-venous O_2 difference

A DECLINE in aerobic performance with advancing age has been well documented (16, 23, 37, 38, 51, 54). This decline in aerobic fitness is associated with an age-related decrease in physical functional capacity and has been linked to reduced quality of life and loss of independence (36) as well as cognitive function (37). Additionally, maximal aerobic power [maximal oxygen uptake ($\dot{V}O_{2\,max}$)] has been shown to be an independent risk factor for all-cause and cardiovascular disease mortality (37). Taken together, these data suggest that maintaining a high maximal aerobic power is an important component in successful healthy aging.

Training studies in older adults lasting ~6–12 mo have yielded improvements in $\dot{V}O_{2\,max}$ ranging from 15 to 29% (4, 10, 15, 25, 47, 48, 52), and even shorter-term exercise training interventions of ~9–12 wk have produced increases in $\dot{V}O_{2\,max}$ of ~6–18% (6, 9, 17, 32, 33, 39). Although the percent increase in $\dot{V}O_{2\,max}$ in older adults has been reported to be similar to that observed in young individuals (17, 20, 32, 33, 44, 48), direct comparisons of the effects of endurance training between older and young adults within the same training program are limited. Further, information regarding the time course of training-induced adaptations in older compared with younger subjects is lacking. In only a few studies has short-term endurance training (9–12 wk) and time course of changes in $\dot{V}O_{2\,max}$ been studied in older adults (17, 20, 32), and in these studies only older adults were tested with no comparisons made to younger control training groups.

The interplay of the time course of central vs. peripheral mechanisms explaining the adaptations involving improvements in $\dot{V}O_{2\,max}$ during training in older compared with younger adults remain to be elucidated. Spina et al. (48) reported that improvements in cardiac output ($\dot{Q}$) and stroke volume (SV) contributed to the majority of the increase in $\dot{V}O_{2\,max}$ in older men after 9–12 mo of endurance training. Others (17, 32) have confirmed that improvements in maximal $\dot{Q}$ ($\dot{Q}_{max}$) in older adults occur even in response to shorter-term endurance training programs (10–12 wk); however, only pre- and posttraining measurements were taken at peak exercise. Thus to date little is known about the time course of central vs. peripheral adaptations underlying the large changes in $\dot{V}O_{2\,max}$ with short-term exercise training in older adults and whether the response differs from young.

The main goal of this study was to determine the time course and mechanisms of adaptation to a 12-wk endurance training program in older (O) and young (Y) male adults. We hypothesized that *1*) both O and Y would increase $\dot{V}O_{2\,max}$ to a similar extent and follow a similar time course during the duration of the exercise-training program; and *2*) in both O and Y groups, improvements in $\dot{Q}_{max}$ would explain the majority of the increase in $\dot{V}O_{2\,max}$ (approximately two-thirds of the change) whereas a widened arterial-venous oxygen difference (a-vO_{2diff}) would be responsible for a smaller portion of the change.

METHODS

Subjects. Eight O (68 ± 7 yr old; mean ± SD) and 8 Y (23 ± 5 yr old) men volunteered and gave written consent to participate in the study. All procedures were approved by The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects. All subjects were nonobese (body mass index ≤30 kg/m^2), nonsmokers, and were physically active, but none had been involved in any type of endurance training program for at least 12 mo before the study. Additionally, no subjects were taking medications that would affect the cardiorespiratory or hemodynamic responses to exercise. Older subjects had no history of cardiovascular, respiratory, or musculoskeletal diseases, were medically screened by a physician, and underwent a maximal exercise stress test.

Protocol. Before training began, subjects performed a maximal cycle ergometer ramp test to exhaustion (O, 15–20 W/min; Y, 25

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W/min) (on a Lode Corival 400 cycle ergometer; Lode BV, Groningen, The Netherlands) for determination of $\dot{V}O_{2\,max}$ and estimation of the lactate threshold (θ_L). θ_L was defined as the $\dot{V}O_2$ at which CO_2 output ($\dot{V}CO_2$) began to increase out of proportion to $\dot{V}O_2$ along with a systematic rise in minute ventilation-to-$\dot{V}O_2$ ratio and end-tidal P_{O_2}, whereas minute ventilation-to-$\dot{V}CO_2$ ratio and end-tidal P_{CO_2} were stable. Approximately 1 min after the end of the ramp test a fingertip blood sample (~0.05 μl) was obtained to measure end-exercise blood lactate concentration using a portable device (Lactate Scout, Sports Resource Group, Hawthorne, NY). Within 5 min after completion of this test, subjects performed a constant-load cycling exercise to volitional fatigue at 85% of the peak power output (PO_{peak}) achieved during the ramp incremental test. This protocol (described in Ref. 42) was performed to assess the attainment of $\dot{V}O_{2\,max}$ and to allow determination of $\dot{Q}_{max}$. Subjects were instructed to indicate when they thought they were ~30 s from exhaustion. At that point, verbal encouragement increased and within ~15 s the measurement of $\dot{Q}$ began. $\dot{V}O_{2\,max}$ was defined as the highest $\dot{V}O_2$ observed for an average of 20 consecutive seconds during either the ramp test to exhaustion or the 2- to 3-min constant load at 85% of PO_{peak}. On a separate day, subjects were asked to cycle at a PO corresponding to ~90% of their pretraining θ_L, and when a steady state in gas exchange was achieved $\dot{Q}$ was measured. Similar procedures were repeated after 3, 6, 9, and 12 wk of training.

Blood tests. Before the start (pre-) and after 6 (mid-) and 12 wk (post-) of training blood samples were drawn from each subject's antecubital vein for determination of hematocrit (Hct) and hemoglobin (Hb) concentrations.

Training. The endurance training program consisted of three exercise sessions per week on a stationary cycle ergometer (Monark Ergomedic 874E; Monark Exercise AB, Varberg, Sweden) for a total duration of 12 wk. Training intensity was adjusted at 3-wk intervals to reflect changes in fitness level. During the first 10 wk, each session consisted of continuous training (CT) for 45 min at a PO that elicited ~70% of the $\dot{V}O_{2\,max}$ observed during the incremental ramp test. During the final 2 wk of training (6 exercise sessions), each individual in each group (O and Y) was assigned to one of two subgroups: *1*) CT as described above; or *2*) high-intensity interval training (HIT), performing 10–12 exercise bouts each lasting 1 min at 90–100% of the PO_{peak} achieved during the incremental ramp test, with 1 min rest separating bouts. Since $\dot{V}O_{2\,max}$ was likely to plateau after ~8 wk of CT (34), the HIT was used as a strategy for progressive and continued gains in the exercise program resulting in further increases in $\dot{V}O_{2\,max}$ favored by peripheral adaptations (12).

Measurements. Gas-exchange measurements were similar to those previously described (3). Briefly, inspired and expired flow rates were measured using a low-dead-space (90 ml) bidirectional turbine (Alpha Technologies VMM 110), which was calibrated before each test by using a syringe of known volume. Inspired and expired gases were sampled continuously (every 20 ms) at the mouth and analyzed for concentrations of O_2, CO_2, nitrogen (N_2), acetylene (C_2H_2), and helium (He) by mass spectrometry (Perkin Elmer MGA-1100) after calibration with precision-analyzed gas mixtures. Breath-by-breath alveolar gas exchange was calculated by using algorithms of Beaver et al. (5).

Heart rate (HR) was monitored continuously by electrocardiogram using PowerLab (ML132/ML880; ADInstruments, Colorado Springs, CO) with a three-lead arrangement. Data were recorded using LabChart v4.2 (ADInstruments, Colorado Springs, CO) on a separate computer.

$\dot{Q}$ measurements. $\dot{Q}$ was measured using the acetylene (C_2H_2) open-circuit inert gas-washin method and analyzed using custom data-acquisition software. This technique was described and validated previously (24). Briefly, a pneumotachograph (Hans Rudolph model 3800, Kansas City, MO; transducer, Validyne MP45-871, Northridge, CA) was attached to a nonrebreathing Y-valve (Hans Rudolph 7900), which was connected to a manual valve that allowed switching inspired gases between room air and a bag containing a mixture of C_2H_2 (0.7%), O_2 (21%), He (9%), and balance N_2. Changes in gas concentrations were aligned with gas volumes by measuring the time delay for fractional changes of the gases to occur. Throughout the submaximal and peak exercise measurements of $\dot{Q}$ subjects were asked to continue their normal breathing pattern when the source of inspired air was switched to the bag containing the gas mixture, and after 10 breaths the protocol was terminated. Data analysis for the calculation of $\dot{Q}$ was performed immediately after each maneuver using equations reported previously (24). The a-vO_{2diff} was calculated from the Fick equation as: a-vO_{2diff} (ml O_2/100 ml blood) = $\dot{V}O_2$ (l/min)/$\dot{Q}$ (l/min) × 100. Stroke volume was calculated as: SV (ml/beat) = $\dot{Q}$ (ml/min)/HR (beats/min).

Statistics. Data are presented as means ± SD. Independent *t*-tests and repeated-measures ANOVA were used to determine statistical significance for the dependent variables. The ANOVA model was described as $S_{16} \times T_5 \times A_2$ such that subjects (*S*; number of subjects) are crossed with testing time (*T*; 5 testing times: pretraining, *week 3, week 6, week 9*, and posttraining), and age (A; older and young adults). A Tukey post hoc analysis was used when significant differences were found for the main effects of each dependent variable. The ANOVA was analyzed by SPSS version 12.0 (SPSS, Chicago, IL). Statistical significance was declared when $P < 0.05$.

RESULTS

Subject characteristics and resting Hct and Hb values are reported in Table 1. Adherence to the training program was 94 ± 1% (28/30 training sessions) and 95 ± 1% (29/30 training sessions) in O and Y, respectively. Each subject completed at least 90% of the programmed training sessions (range: O = 27–29 sessions; Y = 27–30 sessions). The average training intensity (PO) per session increased significantly after each testing session in both O (i.e., *weeks 1–3*, 95 ± 31 W; *weeks 4–6*, 107 ± 31 W; *weeks 7–9*, 116 ± 32 W;) and Y (e.g., *weeks 1–3*, 183 ± 31 W; *weeks 4–6*, 198 ± 36 W; *weeks 7–9*, 207 ± 36 W); training PO was always higher in Y than in O ($P < 0.05$). In the subgroup of O and Y subjects performing the CT during the last 2 wk of the training program ($n = 7$ including O and Y), a further increase in the average PO was observed compared with *weeks 7–9* [e.g., PO *weeks 7–9*, 152 ± 56 W (O, 92 ± 9; Y, 198 ± 48 W) vs. PO *weeks 10–12*, 159 ± 69 W (O, 96 ± 11; Y, 207 ± 45 W)]. The group performing the HIT ($n = 9$ including O and Y) exercised at a higher average PO compared

Table 1. *Subjects' characteristics and resting hematocrit and hemoglobin values*

			Body Weight, kg		Hct		Hb, g/dl	
	Age, yr	Height, m	Pre	Post	Pre	Post	Pre	Post
O	68 (7)*	1.77 (0.09)	81.6 (7.6)	81.2 (7.4)	0.43 (0.03)	0.43 (0.02)	14.8 (0.8)	14.9 (0.7)
Y	23 (5)	1.78 (0.05)	79.9 (8.1)	81.1 (8.1)	0.44 (0.02)	0.44 (0.02)	15.3 (0.7)	15.4 (0.7)

Values are means (SD). O, older adult men; Y, young adult men; Hct, hematocrit; Hb, hemoglobin; Pre, pretraining; Post, posttraining. *Significantly different from Y ($P < 0.05$).

with the previous testing measurement [i.e., PO *weeks 7–9* (continuous), 169 ± 53 W vs. *weeks 10–12* (HIT), 285 ± 88 W]; however, the estimated energy expenditure for an average of 11 ± 1 one-minute bouts of exercise was ~60% lower ($P < 0.05$) for HIT than for CT. Since training type (e.g., continuous vs. HIT) did not significantly affect any of the variables of interest (i.e., PO_{peak} and maximal and submaximal $\dot{V}O_2$, HR, $\dot{Q}$, SV, and a-vO_{2diff}) the group data are combined and compared over the time course of training.

Table 2 summarizes the changes in peak exercise values in response to training. PO_{peak} progressively increased from pre- to posttraining in both O and Y (Table 2). A higher $\dot{V}O_{2\,max}$ was observed within 3 wk of training in both O and Y, with further increases in $\dot{V}O_{2\,max}$ seen in both groups posttraining. No testing time-by-age interactions were detected reflecting a similar rate of adaptation of $\dot{V}O_{2\,max}$ in both O and Y and a maintained difference between age groups across time. The percent change in $\dot{V}O_{2\,max}$ from pretraining to posttraining was larger in O (31 ± 10%) compared with Y (18 ± 10%) adults ($P < 0.05$). The mean slope of the change in $\dot{V}O_{2\,max}$ was ~0.16 and ~0.13 l/min every third week in O and Y, respectively (Fig. 1). The $\dot{V}O_{2\,max}$ obtained during the ramp incremental test was similar to that observed during the 2- to 3-min constant-load test to exhaustion (which was also used to determine $\dot{Q}_{max}$) in both O and Y ($P > 0.05$). Pre- and posttraining values at the end of the ramp incremental test for lactate concentration (O pre, 9.3 ± 1.1 mmol/l; O post, 10.9 ± 2.9 mmol/l; Y pre, 10.8 ± 2.0 mmol/l; Y post, 13.1 ± 3.0 mmol/l), and respiratory exchange ratio (RER) (O pre, 1.20 ± 0.10; O post, 1.16 ± 0.09; Y pre, 1.24 ± 0.10; Y post, 1.23 ± 0.03) were unchanged.

The HR_{max} overall response from pre- to postintervention was unaffected by training (Table 2). $\dot{Q}_{max}$ was higher ($P < 0.05$) in O and Y after 3 wk of training. A further increase in $\dot{Q}_{max}$ occurred after 9 wk of training (Table 2). Maximal SV (SV_{max}) also increased significantly in both groups after 3 wk of training. Additional improvements in SV_{max} were observed at *week 9* (Table 2). Maximal a-vO_{2diff} was higher ($P < 0.05$) at *weeks 3* and *6* and posttraining compared with pretraining in O and Y (Table 2). No testing time-by-age interactions were observed for $\dot{Q}_{max}$, SV_{max}, and maximal a-vO_{2diff} revealing a similar rate of change in each group across time for these variables. In the O, 69% of the change in $\dot{V}O_{2\,max}$ from pre- to posttraining was explained by the increase in $\dot{Q}_{max}$ while the remaining 31% was explained by an improved a-vO_{2diff} [calculated as the percent change in $\dot{Q}$ (or a-vO_{2diff}) divided by the total percent change in $\dot{V}O_{2\,max}$]. In the O, approximately one-third of the increase in $\dot{V}O_{2\,max}$, $\dot{Q}_{max}$, and maximal a-vO_{2diff} occurred during the first 3 wk of training while the remaining approximately two-thirds took place between *week 3* and the end of the training program. The proportion of increase in $\dot{V}O_{2\,max}$ explained by $\dot{Q}_{max}$ (~2/3) and maximal a-vO_{2diff} (~1/3) was similar for each of these time periods (Fig. 2). In Y, 56% of the change in $\dot{V}O_{2\,max}$ was attributed to a higher $\dot{Q}_{max}$ and 44% to a widened a-vO_{2diff}. In contrast to O, approximately two-thirds of the increase in $\dot{V}O_{2\,max}$ in the Y occurred within the first 3 wk of training with the rest of the change taking place after *week 3* of the program. Interestingly, the early adaptations to training in this group relied on improvements in maximal a-vO_{2diff} (~66%) while increases in $\dot{Q}_{max}$ explained the increases in $\dot{V}O_{2\,max}$ from *week 3* to posttraining (Fig. 2).

Table 3 depicts the physiological responses to a constant-load submaximal exercise intensity corresponding to ~90% θ_L (O, 68 ± 15 W; Y, 128 ± 28 W). The steady-state $\dot{V}O_2$ ($\dot{V}O_{2sub}$) corresponding to these POs were not affected by training (O: pretraining, 2.27 ± 0.35 l/min; posttraining, 2.23 ± 0.35 l/min; Y: pretraining, 1.52 ± 0.15 l/min; posttraining, 1.49 ± 0.17 l/min). Compared with pretraining, submaximal HR was lower ($P < 0.05$) after *week 3* in O and Y, with no further

Table 2. *Maximal exercise responses for PO, $\dot{V}O_2$, HR, $\dot{Q}$, SV, and a-vO_{2diff} in O and Y from pretraining through posttraining*

	Pretraining	*Week 3*	*Week 6*	*Week 9*	Posttraining
PO_{peak}, W					
O[e]	188 (44)	201 (40)[a]	208 (44)[a,b]	215 (49)[a,b]	219 (49)[a,b,c,d]
Y	314 (41)	346 (47)[a]	359 (45)[a,b]	365 (57)[a,b]	377 (50)[a,b,c,d]
$\dot{V}O_{2max}$, l/min					
O[e]	2.29 (0.49)	2.48 (0.42)[a]	2.65 (0.58)[a]	2.77 (0.53)[a]	2.95 (0.48)[a,b,c,d]
Y	3.82 (0.47)	4.27 (0.52)[a]	4.22 (0.44)[a]	4.28 (0.49)[a]	4.47 (0.34)[a,b,c,d]
$\dot{V}O_{2max}$, ml·kg^{-1}·min^{-1}					
O[e]	28.3 (7.1)	30.7 (6.0)[a]	32.8 (7.6)[a]	34.5 (8.0)[a]	36.6 (6.5)[a,b,c,d]
Y	48.0 (6.1)	53.8 (7.6)[a]	52.5 (6.4)[a]	53.1 (6.5)[a]	55.4 (5.5)[a,b,c,d]
HR_{max}, beats/min					
O[e]	144 (22)	139 (23)[a]	141 (21)	142 (19)	145 (17)[b,d]
Y	189 (7)	185 (5)[a]	185 (5)	185 (6)	187 (7)[b,d]
$\dot{Q}_{max}$, l/min					
O[e]	16.8 (3.0)	18.0 (3.8)[a]	18.7 (4.2)[a]	19.8 (3.5)[a,b,c]	20.3 (3.7)[a,b,c]
Y	25.9 (2.8)	26.7 (2.2)[a]	27.3 (2.1)[a]	28.6 (1.6)[a,b,c]	28.4 (1.8)[a,b,c]
SV_{max}, ml/beat					
O	122.1 (21.7)	130.4 (19.4)[a]	133.2 (22.0)[a]	140.6 (21.5)[a,b,c]	140.2 (21.3)[a,b]
Y	137.3 (17.2)	144.7 (12.6)[a]	148.2 (15.2)[a]	154.6 (10.6)[a,b,c]	152.3 (12.6)[a,b]
Maximal a-vO_{2diff}, ml O_2/100 ml blood					
O	13.5 (2.2)	14.0 (2.2)[a]	14.2 (1.7)[a]	14.0 (1.9)	14.7 (2.1)[a]
Y	14.7 (0.9)	15.8 (1.2)[a]	15.4 (1.3)[a]	14.8 (1.4)	15.7 (0.9)[a]

Values are means (SD). PO_{peak}, peak power output; $\dot{V}O_{2max}$, maximal O_2 uptake; HR_{max}, maximal heart rate; $\dot{Q}_{max}$, maximal cardiac output; SV_{max}, maximal stroke volume; maximal a-vO_{2diff}, maximal arterial-venous O_2 difference. [a]Significantly different from pretraining values ($P < 0.05$). [b]Significantly different from *week 3* ($P < 0.05$). [c]Significantly different from *week 6*. [d]Significantly different from *week 9*. [e]Significantly different from Y ($P < 0.05$).

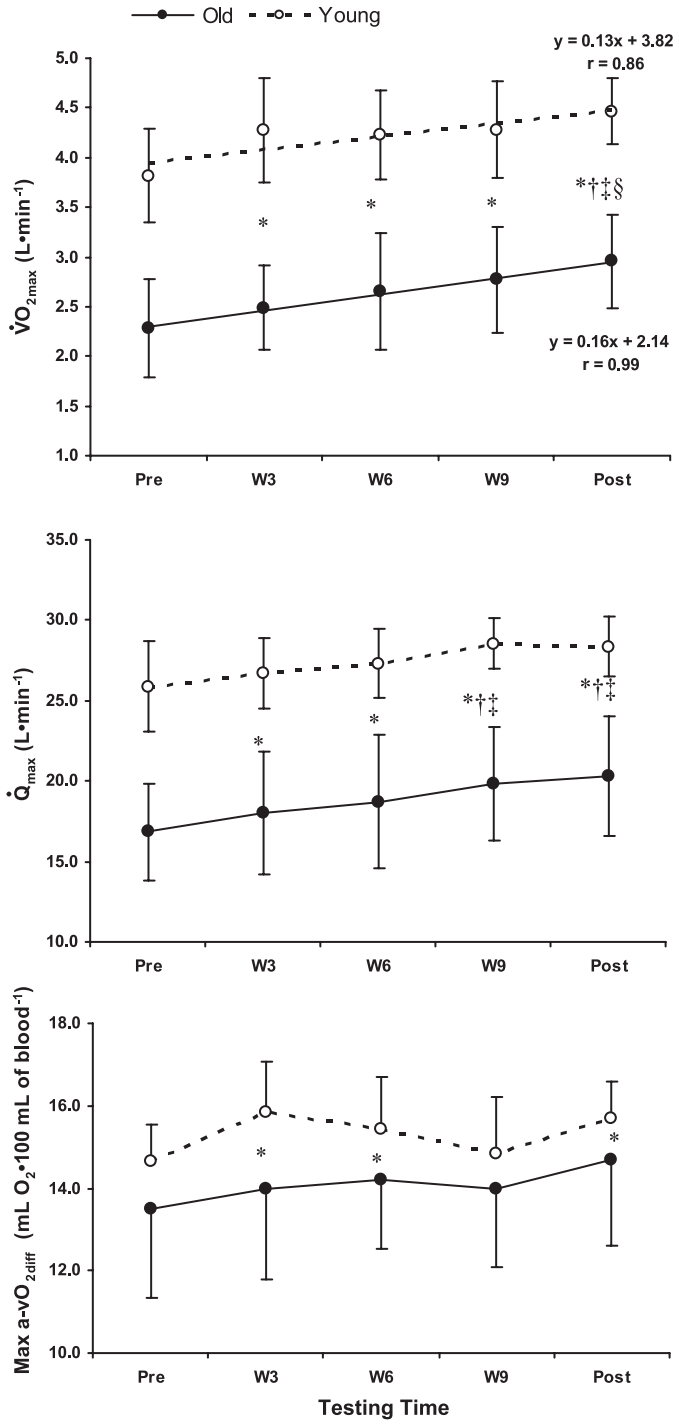

Fig. 1. Changes in maximal oxygen uptake ($\dot{V}O_{2\,max}$), maximal cardiac output ($\dot{Q}_{max}$), and maximal arterial-venous oxygen difference (Max a-vO_{2diff}) in response to training in older (O) and young (Y) male adults. Slopes are not calculated for $\dot{Q}_{max}$ and maximal a-vO_{2diff} because of the nonlinear nature of the response in Y; however, the coefficient of correlation was $r = 0.96$ and 0.99 for $\dot{Q}_{max}$ and $r = 0.88$ and 0.32 for maximal a-vO_{2diff} in O and Y, respectively. *Significantly different from pretraining values ($P < 0.05$). †Significantly different from *week 3* (W3) ($P < 0.05$). ‡Significantly different from *week 6* (W6). §Significantly different from *week 9* (W9).

changes observed thereafter. Submaximal $\dot{Q}$ ($\dot{Q}_{sub}$) remained unchanged in both groups throughout the training. The $\dot{Q}_{sub}$/$\dot{V}O_{2sub}$ was similar in O and Y and was not affected by training (O: pretraining, 7.7 ± 1.0 l/min; posttraining, 7.9 ± 1.2 l/min; Y: pretraining, 7.5 ± 0.6 l/min; posttraining, 7.4 ± 0.4 l/min). SV_{sub} was higher ($P < 0.05$) by *week 3* compared with pretraining, with no further changes during the training program. Submaximal a-vO_{2diff} (a-$vO_{2diffsub}$) in O and Y was not affected by training.

The absolute $\dot{V}O_2$ corresponding to θ_L (l/min) significantly increased after 3 wk of training in both O and Y. A further increase in θ_L (l/min) was observed at *week 6* and again posttraining (Table 3) such that the pre- to posttraining change was 32 ± 20% in O and 17 ± 10% in Y. There was no testing time-by-age interaction suggesting a similar rate of improvement in θ_L in both age groups.

DISCUSSION

This study examined the time course and mechanisms of adaptation to a 12-wk endurance training program in older and young male adults. The main findings were as follows: *1*) the time course of changes in $\dot{V}O_{2\,max}$ was similar in O and Y; *2*) the percent increase in $\dot{V}O_{2\,max}$ was significantly larger in O (31 ±

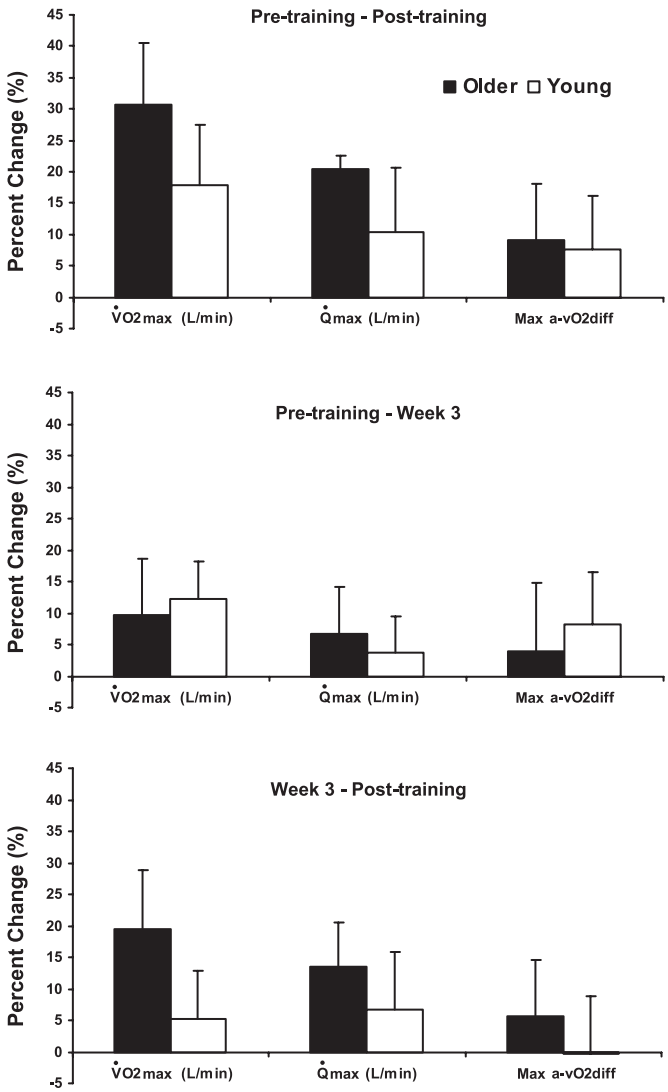

Fig. 2. Percent changes in $\dot{V}O_{2\,max}$, $\dot{Q}_{max}$, and maximal a-vO_{2diff} from pretraining to posttraining (*top*), pretraining to *week 3* (*middle*), and *week 3* to posttraining (*bottom*) in O and Y adults

Table 3. *Submaximal exercise responses for HR, $\dot{Q}$, SV, and a-vO_{2diff} in O and Y from pretraining through posttraining*

	Pretraining	*Week 3*	*Week 6*	*Week 9*	Posttraining
HR_{sub}, beats/min					
O[e]	94 (14)	89 (12)[a]	90 (15)[a]	86 (9)[a]	88 (9)[a]
Y	129 (15)	120 (15)[a]	116 (11)[a]	120 (13)[a]	117 (13)[a]
$\dot{Q}_{sub}$, l/min					
O[e]	11.7 (2.0)	11.3 (1.5)	11.8 (2.4)	12.1 (2.0)	11.8 (2.3)
Y	17.0 (2.5)	17.0 (2.1)	16.5 (2.4)	16.3 (2.0)	16.5 (2.6)
SV_{sub}, ml//beat					
O	125.3 (19.4)	128.9 (17.3)[a]	132.4 (23.2)[a]	140.7 (24.5)[a]	134.6 (25.4)[a]
Y	132.6 (18.2)	143.6 (24.9)[a]	143.2 (25.3)[a]	136.7 (21.5)[a]	142.8 (25.0)[a]
a-$vO_{2diffsub}$, ml O_2/100 ml blood					
O	13.2 (1.8)	12.6 (1.2)	12.0 (1.6)	12.4 (1.7)	12.9 (2.0)
Y	13.3 (1.0)	13.3 (1.0)	13.2 (1.0)	13.6 (0.9)	13.5 (0.7)
θ_L, l/min					
O[e]	1.48 (0.20)	1.70 (0.29)[a]	1.81 (0.29)[a,b]	1.88 (0.34)[a,b]	1.96 (0.40)[a,b,c,d]
Y	2.44 (0.52)	2.58 (0.50)[a]	2.66 (0.44)[a,b]	2.72 (0.49)[a,b]	2.83 (0.49)[a,b,c,d]

Values are means (SD). PO = 68 ± 15 W (O), 128 ± 28 W (Y). HR_{sub}, submaximal heart rate; $\dot{Q}_{sub}$, submaximal cardiac output; SV_{sub}, submaximal stroke volume; a-$vO_{2diffsub}$, submaximal arterial-venous O_2 difference; θ_L, estimated lactate threshold. [a]Significantly different from pretraining values ($P < 0.05$). [b]Significantly different from *week 3* ($P < 0.05$). [c]Significantly different from *week 6*. [d]Significantly different from *week 9*. [e]Significantly different from Y ($P < 0.05$).

10%) than in Y (18 ± 10%); *3*) the mechanisms explaining the time course of increase in $\dot{V}O_{2\,max}$ were different in O compared with Y.

Measurements of $\dot{V}O_{2\,max}$ in this study were rigorous. We confirmed no further increments in $\dot{V}O_2$ (suggesting that a true $\dot{V}O_{2\,max}$ was attained) by comparing the data obtained during the ramp test with those observed during the 2- to 3-min constant-load protocol as previously described (42). Additionally, secondary criteria for determination of $\dot{V}O_{2\,max}$ such as pre- and posttraining end-exercise lactate concentration and RER (see RESULTS) as well as HR_{max} (~95% of the estimated HR_{max} for each age group) (Table 2) suggest a maximal effort was achieved.

Our finding that $\dot{V}O_{2\,max}$ increased by 31% from pre- to posttraining in O is similar to the increases reported previously in response to long-term endurance training (10, 25, 44) but higher than the increases reported in other long-term (3, 15, 47, 48, 52) and short-term (6, 9, 17, 20, 32, 39) aerobic training studies in older men. The larger increase in $\dot{V}O_{2\,max}$ in the present study may be explained by the relatively high training intensity used (~70% of $\dot{V}O_{2\,max}$) and by the frequent progression in training intensity (PO adjusted every 3 wk). It was proposed that higher training intensities [75–80% of HR reserve (HRR)] are important to maximize increases in $\dot{V}O_{2\,max}$ in older adults (15, 44). Similarly, Makrides et al. (30) reported a 38% increase in $\dot{V}O_{2\,max}$ in older subjects in response to a 12-wk interval training regime where the training intensity was adjusted to ~85% of the initial $\dot{V}O_{2\,max}$ by the third week of the program. However, Gass et al. (17) proposed that the total amount of work, rather than training intensity, determined the increase in $\dot{V}O_{2\,max}$. Considering that in the present study the total amount of work was increased by increasing the training PO, it is likely that both training intensity and total amount of work played a role in modulating the increases in $\dot{V}O_{2\,max}$. Although in the present study the absolute increase in $\dot{V}O_{2\,max}$ was similar in both O and Y, the percent increase was larger in O (O, 31%; Y, 18%), reflecting the lower absolute pretraining $\dot{V}O_{2\,max}$ in O. It is unlikely that the higher percent increase in $\dot{V}O_{2\,max}$ in O reflected a relatively lower initial level of fitness compared with Y because the participants for both age groups in this study at baseline were above the mean $\dot{V}O_{2\,max}$ predicted for age-matched populations (1, 35).

Approximately two-thirds of the pre- to posttraining increase in $\dot{V}O_{2\,max}$ in O was explained by an increase in $\dot{Q}_{max}$ with a larger posttraining maximal a-vO_{2diff} accounting for the remaining approximately one-third of the change. A similar pattern of adaptation, with $\dot{Q}_{max}$ being the primary mechanism for the increase in $\dot{V}O_{2\,max}$, has been previously reported in response endurance training programs in older men (32, 48). What is novel about the present study is that the time course of central and peripheral changes were tracked at 3-wk intervals. Interestingly, the relative contribution from $\dot{Q}_{max}$ and maximal a-vO_{2diff} in explaining the larger $\dot{V}O_{2\,max}$ in O remained the same from pretraining to *week 3* and from *week 3* to posttraining (the testing times at which $\dot{V}O_{2\,max}$ was significantly increased), suggesting that central adaptations are important in establishing increases in $\dot{V}O_{2\,max}$ in O and also that these central adaptations occur rapidly (within the first 3 wk of starting training). Considering that the overall HR_{max} response was unchanged pre- to posttraining, the greater $\dot{Q}_{max}$ observed in O posttraining was a consequence of a larger SV_{max} (pre- to posttraining increase 16 ± 11% and 12 ± 10% in O and Y, respectively) (Table 2). Similarly, 3 wk of training in O resulted in a reduction in HR_{sub} and an increase in SV_{sub}. Training-induced increases in SV_{sub} in O have been reported previously (17, 32, 48).

The larger SV_{max} could be related to an enhanced left ventricular (LV) filling, increased LV contractility, or a combination of these factors. It has been proposed the most of the increases in $\dot{Q}_{max}$ are related to an increased diastolic filling because of a more compliant left ventricle (29), which could lead to an increased SV via the Frank-Starling mechanism (27). In regard to an increased LV contractile function, it has been proposed that an enlargement of the LV mass could be one of the mechanisms responsible for this adaptation (15, 45); however, it is likely this is a longer-term adaptation. Although no measures of catecholamines were obtained in this study, greater ventricular contractility following training in older adults could be related to increased sensitivity to these hormones (50), which would counteract the reported age-related

decrease in catecholamine sensitivity and loss of efficiency of postsynaptic β-adrenergic signaling (26, 27).

A training-induced increase in a-vO_{2diff} also provided a significant contribution to the increase in $\dot{V}O_{2\,max}$ in O. Even though no direct measures of peripheral adaptations are provided in the present study, previous reports have shown that a greater whole body (i.e., muscle) O_2 extraction following training in older adults could be related to improvements in capillarization and augmented number of type IIa muscle fibers (9, 10, 22), mitochondrial enzymes activity (7, 9, 10), and/or microvascular blood flow distribution (31, 46). Taken together, these data suggest that in older adults, both cardiac and skeletal muscle can adapt to training, and given an adequate training stimulus, this adaptation occurs relatively quickly (within 3 wk of training) and can continue for at least 12 wk of training.

In Y, ~60% of the increase in $\dot{V}O_{2\,max}$ from pre- to posttraining was attributed to a greater $\dot{Q}_{max}$, similar to that observed in O (i.e., ~66%). However, in Y the early increase in $\dot{V}O_{2\,max}$ (i.e., during the first 3 wk of training) was a consequence of a greater a-vO_{2diff}. Unlike O who showed a more steady response during the 12 wk of training, in Y, $\dot{V}O_{2\,max}$ remained unchanged between *weeks 3* and *9*, followed by an increase between *weeks 9* and *12*, a consequence of a greater $\dot{Q}_{max}$. It is unclear why increases in $\dot{V}O_{2\,max}$ in Y men relied more on a-vO_{2diff} during the first weeks of training, but it is possible that a more effective distribution of $\dot{Q}$ in the periphery may have resulted in a better matching of O_2 delivery and utilization. Previous training studies have reported peripheral adaptations early in training in young men that would support this contention (2, 11, 14, 21). Since the overall HR_{max} did not change from pre- to posttraining, the improvements in $\dot{Q}_{max}$ that explained the further increase in $\dot{V}O_{2\,max}$ with training were solely explained by a higher SV_{max}. Similarly, an improved SV was also observed at submaximal intensities as previously reported (30, 43, 49).

A training-induced increase in the $\dot{V}O_2$ corresponding to the θ_L was observed after *weeks 3, 6,* and *12* in both O and Y. Similar increases in θ_L following training have been reported previously for both older (39, 53) and young adults (13, 41). This improved response to submaximal exercise may be especially important in older men where certain activities of daily living may be performed above θ_L and thus qualify as "heavy" intensity and fatiguing (37).

Based on the work of O'Donovan et al. (34), we contemplated the possibility that only those men being part of the HIT group would further increase their $\dot{V}O_{2\,max}$. However, both training groups (CT and HIT) showed similar improvements in response to training. This suggests that *1*) when the training intensity is adjusted to reflect changes in aerobic performance, CT at an intensity of 70% of $\dot{V}O_{2\,max}$ remains sufficient to produce increments in $\dot{V}O_{2\,max}$ even after 10 wk of performing a similar exercise training protocol; and *2*) HIT may be a valid alternative to a chronic endurance training program even in older populations. Importantly, although a plateau response in $\dot{V}O_{2\,max}$ was not observed in this short-term training program, a "ceiling effect" would be expected with further endurance training.

In conclusion, we demonstrated that the time course of adaptations in $\dot{V}O_{2\,max}$ was similar in O and Y men with improvements occurring as early as 3 wk into training and continuing to the end of the program. Thus a short-term training program yielded substantial increases in $\dot{V}O_{2\,max}$ in both older and young men. Increments in $\dot{V}O_{2\,max}$ from pre- to posttraining in O were achieved through changes in $\dot{Q}_{max}$ (~2/3 of the change) as in Y. The time course of adaptation was age dependent in that Y initially relied on increases in maximal a-vO_{2diff} (first 3 wk) with further increases in aerobic power being explained exclusively by a larger $\dot{Q}_{max}$ whereas O showed consistent improvements in $\dot{Q}_{max}$ (~2/3 increase) throughout the training program.

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DISCLOSURES

No conflicts of interest are declared by the authors.

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