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 (VO2max). VO2max increased within 3 wk with further increases observed posttraining in both O (+31%) and Y (+18%), (P < 0.05). Maximal cardiac output (Qmax, 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 arteriovenous oxygen difference (a-vO2diff) 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 VO2max from pre to posttraining was explained by an increased Qmax with the remaining ~31% explained by a widened a-vO2diff. This proportion of Q and a-vO2diff contributions to the increase in VO2max was consistent throughout testing in O. In Y, 56% of the pre- to posttraining increase in VO2max was attributed to a greater Qmax and 44% to a widened a-vO2diff. Early adaptations (first 3 wk) mainly relied on a widened maximal a-vO2diff (~66%) whereas further increases in VO2max were exclusively explained by a greater Qmax. In conclusion, with short-term training O and Y significantly increased their VO2max; however, the proportion of VO2max increase explained by Qmax and maximal a-vO2diff throughout training showed a different pattern by age group.

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 (VO2max)] 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 VO2max 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 VO2max of ~6–18% (6, 9, 17, 32, 33, 39). Although the percent increase in VO2max 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 VO2max 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 VO2max during training in older compared with younger adults remain to be elucidated. Spina et al. (48) reported that improvements in cardiac output (Q) and stroke volume (SV) contributed to the majority of the increase in VO2max in older men after 9–12 mo of endurance training. Others (17, 32) have confirmed that improvements in maximal Q (Qmax) 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 VO2max 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 VO2max 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 Qmax would explain the majority of the increase in VO2max (approximately two-thirds of the change) whereas a widened arteriovenous oxygen difference (a-vO2diff) 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/m2), 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
Table 1. Subjects' characteristics and resting hematocrit and hemoglobin values

<table>
<thead>
<tr>
<th></th>
<th>Age, yr</th>
<th>Height, m</th>
<th>Body Weight, kg</th>
<th>Hct</th>
<th>Hb, g/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>O</td>
<td>68 (7)*</td>
<td>1.77 (0.09)</td>
<td>81.6 (7.6)</td>
<td>81.2 (7.4)</td>
<td>0.43 (0.03)</td>
</tr>
<tr>
<td>Y</td>
<td>23 (5)</td>
<td>1.78 (0.05)</td>
<td>79.9 (8.1)</td>
<td>81.1 (8.1)</td>
<td>0.44 (0.02)</td>
</tr>
</tbody>
</table>

Values are means (SD). O, older adult men; Y, young adult men; Hct, hematocrit; Hb, hemoglobin; Pre, pretraining; Post, postraining. *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., POpeak and maximal and submaximal VO2, HR, Q, SV, and a-vO2diff) 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. POpeak progressively increased from pre- to posttraining in both O and Y (Table 2). A higher VO2max was observed within 3 wk of training in both O and Y, with further increases in VO2max seen in both groups posttraining. No testing time–age interactions were detected reflecting a similar rate of adaptation of V˙O2 max in both O and Y and a maintained difference between age groups across time. The percent change in VO2max 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 VO2max was ~0.16 and ~0.13 l/min every third week in O and Y, respectively (Fig. 1). The VO2max 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 Qmax) in both O and Y (> 0.05). Pre- and posttraining values at the end of the ramp incremental test for lactate concentration (O pre, 9.3 ± 0.17 mmol/l; Y pre, 10.8 ± 2.9 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 HRmax overall response from pre- to postintervention was unaffected by training (Table 2). Qmax was higher (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., POpeak and maximal and submaximal VO2, HR, Q, SV, and a-vO2diff) the group data are combined and compared over the time course of training.

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The HRmax overall response from pre- to postintervention was unaffected by training (Table 2). Qmax was higher (P < 0.05) in O and Y after 3 wk of training. A further increase in Qmax occurred after 9 wk of training (Table 2). Maximal SV (SVmax) also increased significantly in both groups after 3 wk of training. Additional improvements in SVmax were observed at week 9 (Table 2). Maximal a-vO2diff was higher (P < 0.05) at weeks 3 and 6 and posttraining compared with pretraining in O and Y (Table 2). No testing time–age interactions were observed for Qmax, SVmax, and maximal a-vO2diff revealing a similar rate of change in each group across time for these variables. In the O, 69% of the change in VO2max from pre- to posttraining was explained by the increase in Qmax while the remaining 31% was explained by an improved a-vO2diff calculated as the percent change in Q (or a-vO2diff) divided by the total percent change in VO2max. In the O, approximately one-third of the increase in VO2max, Qmax, and maximal a-vO2diff 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 VO2max explained by Qmax (~2/3) and maximal a-vO2diff (~1/3) was similar for each of these time periods (Fig. 2). In Y, 56% of the change in VO2max was attributed to a higher Qmax and 44% to a widened a-vO2diff. In contrast to O, approximately two-thirds of the increase in VO2max 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-vO2diff (~66%) while increases in Qmax explained the increases in VO2max from week 3 to posttraining (Fig. 2).

Table 3 depicts the physiological responses to a constant-load submaximal exercise intensity corresponding to ~90% θL (O, 68 ± 15 W; Y, 128 ± 28 W). The steady-state V˙O2 (V˙O2sub) 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, VO2, HR, Q, SV, and a-vO2diff in O and Y from pretraining through posttraining

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Week 3</th>
<th>Week 6</th>
<th>Week 9</th>
<th>Posttraining</th>
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</thead>
<tbody>
<tr>
<td>POpeak, W</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>188 (44)</td>
<td>201 (40)a</td>
<td>208 (44)b</td>
<td>215 (49)b</td>
<td>219 (49)b,c,d</td>
</tr>
<tr>
<td>Y</td>
<td>314 (41)</td>
<td>346 (47)a</td>
<td>359 (45)b</td>
<td>365 (57)b</td>
<td>377 (50)b,c,d</td>
</tr>
<tr>
<td>VO2max, l/min</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>2.29 (0.49)</td>
<td>2.48 (0.42)a</td>
<td>2.65 (0.58)a</td>
<td>2.77 (0.53)p</td>
<td>2.95 (0.48)b,c,d</td>
</tr>
<tr>
<td>Y</td>
<td>3.82 (0.47)</td>
<td>4.27 (0.52)a</td>
<td>4.22 (0.44)a</td>
<td>4.28 (0.49)p</td>
<td>4.47 (0.34)b,c,d</td>
</tr>
<tr>
<td>V˙O2max, ml·kg⁻¹·min⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>28.3 (7.1)</td>
<td>30.7 (6.0)a</td>
<td>32.8 (7.6)a</td>
<td>34.5 (8.0)b</td>
<td>36.6 (6.5)b,c,d</td>
</tr>
<tr>
<td>Y</td>
<td>48.0 (6.1)</td>
<td>53.8 (7.6)a</td>
<td>52.5 (6.4)b</td>
<td>53.1 (6.5)b</td>
<td>55.4 (5.5)b,c,d</td>
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<tr>
<td>HRmax, beats/min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>144 (22)</td>
<td>139 (23)a</td>
<td>141 (21)</td>
<td>142 (19)</td>
<td>145 (17)b,d</td>
</tr>
<tr>
<td>Y</td>
<td>189 (7)</td>
<td>185 (5)a</td>
<td>185 (5)</td>
<td>185 (6)</td>
<td>187 (7)b,d</td>
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<tr>
<td>Qmax, l/min</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>16.8 (3.0)</td>
<td>18.0 (3.8)a</td>
<td>18.7 (4.2)a</td>
<td>19.8 (3.5)b,c</td>
<td>20.3 (3.7)b,c,d</td>
</tr>
<tr>
<td>Y</td>
<td>25.9 (2.8)</td>
<td>26.7 (2.2)a</td>
<td>27.3 (2.1)a</td>
<td>28.6 (1.6)b,c</td>
<td>28.4 (1.8)b,c,d</td>
</tr>
<tr>
<td>S Vmax, ml/beat</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>122.1 (21.7)</td>
<td>130.4 (19.4)a</td>
<td>133.2 (22.0)a</td>
<td>140.6 (21.5)b,c</td>
<td>140.2 (21.3)b</td>
</tr>
<tr>
<td>Y</td>
<td>137.3 (17.2)</td>
<td>144.7 (12.6)a</td>
<td>148.2 (15.2)a</td>
<td>154.6 (10.6)b,c</td>
<td>152.3 (12.6)b</td>
</tr>
<tr>
<td>Maximal a-vO2diff, ml O2/100 ml blood</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>13.5 (2.2)</td>
<td>14.0 (2.2)a</td>
<td>14.2 (1.7)a</td>
<td>14.0 (1.9)</td>
<td>14.7 (2.1)a</td>
</tr>
<tr>
<td>Y</td>
<td>14.7 (0.9)</td>
<td>15.8 (1.2)a</td>
<td>15.4 (1.3)a</td>
<td>14.8 (1.4)</td>
<td>15.7 (0.9)a</td>
</tr>
</tbody>
</table>

Values are means (SD). POpeak, peak power output; VO2max, maximal O2 uptake; HRmax, maximal heart rate; Qmax, maximal cardiac output; SVmax, maximal stroke volume; maximal a-vO2diff, maximal arterial-venous O2 difference. *Significantly different from pretraining values (P < 0.05). †Significantly different from week 3 (P < 0.05). ‡Significantly different from week 6. §Significantly different from week 9. ¶Significantly different from Y (P < 0.05).
changes observed thereafter. Submaximal $Q (Q_{\text{sub}})$ remained unchanged in both groups throughout the training. The $Q_{\text{sub}}/V_{\text{O2sub}}$ was similar in O and Y and was not affected by training.

The absolute $V_{\text{O2}}$ corresponding to $\theta_1$ ($l/min$) significantly increased after 3 wk of training in both O and Y. A further increase in $\theta_1$ ($l/min$) 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 $\theta_1$ 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 $V_{\text{O2max}}$ was similar in O and Y; 2) the percent increase in $V_{\text{O2max}}$ was significantly larger in O (31 ± Y: pretraining, 7.5 ± 0.6 l/min; posttraining, 7.4 ± 0.4 l/min).

SV$_{\text{sub}}$ was higher ($P < 0.05$) by week 3 compared with pretraining, with no further changes during the training program. Submaximal a-v$O_2$diff (a-v$O_2$diff$_{\text{sub}}$) in O and Y was not affected by training.

The absolute $V_{\text{O2}}$ corresponding to $\theta_1$ ($l/min$) significantly increased after 3 wk of training in both O and Y. A further increase in $\theta_1$ ($l/min$) 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 $\theta_1$ in both age groups.
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\(_{max}\) (see RESULTS) were important to maximize 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 increases in SV\(_{max}\) in O have been reported 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 \( \dot{V}O_2 \) 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 (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...
decrease in catecholamine sensitivity and loss of efficiency of postsynaptic β-adrenergic signaling (26, 27).

A training-induced increase in a-V̇O₂diff also provided a significant contribution to the increase in V̇O₂max 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) O₂ 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 V̇O₂max from pre- to post-training was attributed to a greater Q̇max, similar to that observed in O (i.e., ~66%). However, in Y the early increase in V̇O₂max (i.e., during the first 3 wk of training) was a consequence of a greater a-V̇O₂diff. Unlike O who showed a more steady response during the 12 wk of training, in Y, V̇O₂max remained unchanged between weeks 3 and 9, followed by an increase between weeks 9 and 12, a consequence of a greater Qmax. It is unclear why increases in V̇O₂max in Y men relied more on a-V̇O₂diff during the first weeks of training, but it is possible that a more effective distribution of Q in the periphery may have resulted in a better matching of O2 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 HRmax did not change from pre- to posttraining, the improvements in Qmax that explained the further increase in V̇O₂max with training were solely explained by a higher SVmax. Similarly, an improved SV was also observed at submaximal intensities as previously reported (30, 43, 49).

A training-induced increase in the V̇O₂ corresponding to the θbar was observed after weeks 3, 6, and 12 in both O and Y. Similar increases in θbar 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 θbar 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 V̇O₂max. 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 V̇O₂max remains sufficient to produce increments in V̇O₂max 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 V̇O₂max 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 V̇O₂max 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 V̇O₂max in both older and young men. Increments in V̇O₂max from pre- to posttraining in O were achieved through changes in Qmax (~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-V̇O₂diff (first 3 wk) with further increases in aerobic power being explained exclusively by a larger Qmax whereas O showed consistent improvements in Qmax (~2/3 increase) throughout the training program.

ACKNOWLEDGMENTS

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GRANTS

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DISCLOSURES

No conflicts of interest are declared by the authors.

REFERENCES

CARDIORESPIRATORY FITNESS ADAPTATIONS IN OLDER AND YOUNG MEN


