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 (V\textsubscript{O\textsubscript{2 max}}). V\textsubscript{O\textsubscript{2 max}} increased within 3 wk with further increases observed posttraining in both O (+31%) and Y (+18%), (P < 0.05). Maximal cardiac output (Q\textsubscript{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-v\textsubscript{O\textsubscript{2 diff}}) 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 (V\textsubscript{O\textsubscript{2 max}})) has been shown to be an important component of successful healthy aging.

Training studies in older adults lasting ~6–12 mo have yielded improvements in V\textsubscript{O\textsubscript{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 V\textsubscript{O\textsubscript{2 max}} of ~6–18% (6, 9, 17, 32, 33, 39). Although the percent increase in V\textsubscript{O\textsubscript{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 V\textsubscript{O\textsubscript{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 V\textsubscript{O\textsubscript{2 max}} 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 V\textsubscript{O\textsubscript{2 max}} in older men after 9–12 mo of endurance training. Others (17, 32) have confirmed that improvements in maximal Q (Q\textsubscript{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 V\textsubscript{O\textsubscript{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 V\textsubscript{O\textsubscript{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 Q\textsubscript{max} would explain the majority of the increase in V\textsubscript{O\textsubscript{2 max}} (approximately two-thirds of the change) whereas a widened arterial-venous oxygen difference (a-v\textsubscript{O\textsubscript{2 diff}}) 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\textsuperscript{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
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></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</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, 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., 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 preto 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-by-age interactions were detected reflecting a similar rate of adaptation of VO2max 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 (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 and 1.16 mmol/l), and respiratory exchange ratio (RER) (O pre, 1.20 vs. post, 1.16 mmol/l), and respiratory exchange ratio (RER) (O pre, 1.20 and 1.16 mmol/l) 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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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>
</tr>
</thead>
<tbody>
<tr>
<td>POpeak, W</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</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>
<td></td>
<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)a,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)a,b,c,d</td>
</tr>
<tr>
<td>VO2max, ml·kg⁻¹·min⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<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)p</td>
<td>36.6 (6.5)a,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)a</td>
<td>53.1 (6.5)p</td>
<td>55.4 (5.5)a,b,c,d</td>
</tr>
<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)b</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>
<td></td>
<td></td>
<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>SVmax, 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,d</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,d</td>
<td>152.3 (12.6)b</td>
</tr>
</tbody>
</table>

Maximal a-VO2diff, ml O2/100 ml blood

| O         | 13.5 (2.2)  | 14.0 (2.2)b  | 14.2 (1.7)b  | 14.0 (1.9)  | 14.7 (2.1)a   |
| Y         | 14.7 (0.9)  | 15.8 (1.2)b  | 15.4 (1.3)b  | 14.8 (1.4)c | 15.7 (0.9)a   |

Values are means (SD). POpeak, peak power output; VO2max, maximal oxygen 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 (Qsub) remained unchanged in both groups throughout the training. The Qsub/VO2sub was higher (P < 0.05) by week 3 compared with pretraining, with no further changes during the training program. Submaximal a-vO2diff (a-vO2diffsub) in O and Y was not affected by training.

The absolute VO2 corresponding to θ1 (l/min) significantly increased after 3 wk of training in both O and Y. A further increase in θ1 (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 θ1 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 VO2max was similar in O and Y; 2) the percent increase in VO2max was significantly larger in O (31 ±...
V̇O_2 max in O reflected a relatively lower initial level of fitness (see RESULTS) as well as HRmax (ally, secondary criteria for determination of V̇O_2 max such as constant-load protocol as previously described (42). Additionally, the ramp test with those observed during the 2-to-3-min central and peripheral changes were tracked at 3-wk intervals. Interestingly, the relative contribution from Q̇ max and maximal a-vO_2_diff in explaining the larger V̇O_2 max in O remained the same from pretraining to week 3 and from week 3 to postraining (the testing times at which V̇O_2 max was significantly increased), suggesting that central adaptations are important in establishing increases in V̇O_2 max in O and also that these central adaptations occur rapidly (within the first 3 wk of starting training). Considering that the overall HRmax response was unchanged pre- to postraining, the greater Q̇ max observed in O postraining was a consequence of a larger SV̇ max (pre- to postraining 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 HRsub and an increase in SV̇ sub. Training-induced increases in SV̇ sub in O have been reported previously (17, 32, 48). The larger SV̇ 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 Q̇ 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, TRḢ sub, submaximal arterial-venous O_2 difference; SV̇ sub, submaximal stroke volume; a-vO_2_diffsub, submaximal arterial-venous O_2 difference; hL, estimated lactate threshold. aSignificantly different from pretraining values (P < 0.05). bSignificantly different from week 3 (P < 0.05). cSignificantly different from week 6. dSignificantly different from week 9. eSignificantly different from Y (P < 0.05).

10%) than in Y (18 ± 10%); 3) the mechanisms explaining the time course of increase in V̇O_2 max were different in O compared with Y.

Measurements of V̇O_2 max in this study were rigorous. We confirmed no further increments in V̇O_2 (suggesting that a true V̇O_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 V̇O_2 max such as pre- and postraining end-exercise lactate concentration and RER (see RESULTS) as well as HRmax (~95% of the estimated HṘ max for each age group) (Table 2) suggest a maximal effort was achieved.

Our finding that V̇O_2 max increased by 31% from pre- to postraining 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 V̇O_2 max in the present study may be explained by the relatively high training intensity used (~70% of V̇O_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 V̇O_2 max in older adults (15, 44). Similarly, Makrides et al. (30) reported a 38% increase in V̇O_2 max in older subjects in response to a 12-wk interval training regimen where the training intensity was adjusted to ~85% of the initial V̇O_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 V̇O_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 V̇O_2 max. Although in the present study the absolute increase in V̇O_2 max was similar in both O and Y, the percent increase was larger in O (31%; Y, 18%), reflecting the lower absolute pretraining V̇O_2 max in O. It is unlikely that the higher percent increase in V̇O_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 V̇O_2 max predicted for age-matched populations (1, 35).

Approximately two-thirds of the pre- to postraining increase in V̇O_2 max in O was explained by an increase in Q̇ max with a larger postraining maximal a-vO_2_diff accounting for the remaining approximately one-third of the change. A similar pattern of adaptation, with Q̇ max being the primary mechanism for the increase in V̇O_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 Q̇ max and maximal a-vO_2_diff in explaining the larger V̇O_2 max in O remained the same from pretraining to week 3 and from week 3 to postraining (the testing times at which V̇O_2 max was significantly increased), suggesting that central adaptations are important in establishing increases in V̇O_2 max in O and also that these central adaptations occur rapidly (within the first 3 wk of starting training). Considering that the overall HRmax response was unchanged pre- to postraining, the greater Q̇ max observed in O postraining was a consequence of a larger SV̇ max (pre- to postraining 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 HRsub and an increase in SV̇ sub. Training-induced increases in SV̇ sub in O have been reported previously (17, 32, 48).

The larger SV̇ 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 Q̇ 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
A training-induced increase in a-VO_{2\text{diff}} also provided a significant contribution to the increase in VO_{2\text{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 VO_{2\text{max}} from pre- to post-training was attributed to a greater Q_{\text{max}}, similar to that observed in O (i.e., ~66%). However, in Y the early increase in VO_{2\text{max}} (i.e., during the first 3 wk of training) was a consequence of a greater a-VO_{2\text{diff}}. Unlike O who showed a more steady response during the 12 wk of training, in Y, VO_{2\text{max}} remained unchanged between weeks 3 and 9, followed by an increase between weeks 9 and 12, a consequence of a greater Q_{\text{max}}. It is unclear why increases in VO_{2\text{max}} in Y men relied more on a-VO_{2\text{diff}} 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 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_{\text{max}} did not change from pre- to posttraining, the improvements in Q_{\text{max}} that explained the further increase in VO_{2\text{max}} with training were solely explained by a higher SV_{\text{max}}. Similarly, an improved SV was also observed at submaximal intensities as previously reported (30, 43, 49).

A training-induced increase in VO_{2} corresponding to the \theta_{\text{1}} was observed after weeks 3, 6, and 12 in both O and Y. Similar increases in \theta_{\text{1}} 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 \theta_{\text{1}} 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 VO_{2\text{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 VO_{2\text{max}} remains sufficient to produce increments in VO_{2\text{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 VO_{2\text{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 VO_{2\text{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 VO_{2\text{max}} in both older and young men. Increments in VO_{2\text{max}} from pre- to posttraining in O were achieved through changes in Q_{\text{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_{2\text{diff}} (first 3 wk) with further increases in aerobic power being explained exclusively by a larger Q_{\text{max}} whereas O showed consistent improvements in Q_{\text{max}} (~2/3 increase) throughout the training program.

ACKNOWLEDGMENTS

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DISCLOSURES

No conflicts of interest are declared by the authors.

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