Effects of prior heavy-intensity exercise on pulmonary O₂ uptake and muscle deoxygenation kinetics in young and older adult humans

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DeLorey, Darren S., John M. Kowalchuk, and Donald H. Paterson. Effects of prior heavy-intensity exercise on pulmonary O₂ uptake and muscle deoxygenation kinetics in young and older adult humans. J Appl Physiol 97: 998–1005, 2004. First published May 7, 2004; 10.1152/japplphysiol.01280.2003.—Pulmonary O₂ uptake (V̇O₂p) and muscle deoxygenation kinetics were examined during moderate-intensity cycling (80% lactate threshold) without warm-up and after heavy-intensity warm-up exercise in young (n = 6; 25 ± 3 yr) and older (n = 5; 68 ± 3 yr) adults. We hypothesized that heavy warm-up would speed V̇O₂p kinetics in older adults consequent to an improved intramuscular oxygenation. Subjects performed step transitions (n = 4; 6 min) from 20 W to moderate-intensity exercise preceded by either no warm-up or heavy-intensity warm-up (6 min). V̇O₂p was measured by breath by breath. Oxy-, deoxy-(HHb), and total hemoglobin and myoglobin (Hbtot) of the vastus lateralis muscle were measured continuously by near-infrared spectroscopy (NIRS). V̇O₂p (phase 2; 7) and HHb data were fit with a monoeponential model. After heavy-intensity warm-up, oxyhemoglobin (younger subjects: 9 ± 9 μM; young subjects: 9 ± 8 μM) and Hbtot (older subjects: 12 ± 8 μM; young subjects: 14 ± 10 μM) were elevated (P < 0.05) relative to the no warm-up pretransition baseline. In older adults, V̇O₂p adapted at a faster rate (P < 0.05) after heavy warm-up (30 ± 7 s) than no warm-up (38 ± 5 s), whereas in young subjects, V̇O₂p was similar in no warm-up (26 ± 7 s) and heavy warm-up (25 ± 5 s). HHb adapted at a similar rate in older and young adults after no warm-up; however, in older adults after heavy warm-up, the adaptation of HHb was slower (P < 0.01) compared with young and no warm-up. These data suggest that, in older adults, V̇O₂p kinetics may be limited by a slow adaptation of muscle blood flow and O₂ delivery.

The technique of near-infrared spectroscopy (NIRS) provides noninvasive and continuous monitoring of relative concentration changes in deoxy- (HHb), oxy- (HbO₂), and total hemoglobin (Hb) and myoglobin (Mb) (Hbtot) in the microvasculature (small arterioles, capillaries, and venules) (9) of the muscle. The NIRS-derived HHb signal reflects the balance between O₂ delivery and O₂ utilization in the region of NIRS interrogation and, when used in combination with measurements of pulmonary V̇O₂p, provides information on the time course of local muscle O₂ utilization (20, 21, 29).

Therefore, the purpose of the present study was to examine the effect of prior heavy-intensity exercise on the adaptation of V̇O₂p and muscle deoxygenation at the onset of a subsequent moderate-intensity exercise bout in young and older adults. We hypothesized that 1) after prior heavy-intensity exercise, V̇O₂p kinetics would be accelerated in older and unchanged in young adults during subsequent moderate-intensity exercise; 2) prior heavy-intensity exercise would result in an elevation of HR in older and younger adults, reflecting improved muscle perfusion and O₂ delivery before the onset and throughout subsequent moderate-intensity exercise in both groups; and 3) after heavy-intensity warm-up exercise, muscle deoxygenation would adapt at a slower rate in older adults (despite faster V̇O₂p kinetics) and be unchanged in young adults, reflecting an improvement in local muscle perfusion in older adults. Collectively, these results would suggest that the adaptation of muscle blood flow may limit the adaptation of V̇O₂p and muscle O₂ uptake (V̇O₂p) in older adults during the on-transient of moderate-intensity exercise without prior warm-up exercise.
METHODS

Subjects. Six young (25 ± 3 yr) and five older (68 ± 3 yr) healthy and physically active adults volunteered and gave written, informed consent to participate in the study. All procedures were approved by The University of Western Ontario Ethics Committee for Research on Human Subjects.

Protocol. Subjects reported to the laboratory on nine separate occasions. An incremental ramp (25 W/min) exercise test to the limit of tolerance was performed by cycle ergometry on the first day of testing for the determination of the estimated lactate threshold ($\theta_L$) and peak VO$_2$, $\theta_P$. Each moderate-intensity WR transition was 6 min in duration and was preceded by either no warm-up exercise or 6 min of heavy-intensity warm-up exercise at a WR selected to elicit a VO$_2$peak equivalent to ~50% of the difference ($\Delta$VO$_2$peak) between the VO$_2$peak at $\theta_L$ and peak VO$_2$. In the heavy-intensity warm-up condition, heavy-intensity exercise was preceded and followed by 6 min of 20-W cycling. Four transitions were performed in each of the warm-up conditions in random order.

Measurements. Gas-exchange measurements were similar to those described previously (2). Briefly, inspired and expired flow rates were measured with a low dead-space (90 ml) bidirectional turbine (Alpha Technologies VMM 110), which was calibrated before each test with a syringe of known volume. Inspired and expired gases were sampled continuously at the mouth and analyzed for concentrations of O$_2$, CO$_2$, and N$_2$ by mass spectrometry (Morgan Medical) after calibration in minute ventilation/VO$_2$ and peak VO$_2$ at which CO$_2$ output began to increase out of proportion in relation to VO$_2$, with a systematic rise in minute ventilation/VO$_2$ and end-tidal PO$_2$ while minute ventilation/CO$_2$ output and end-tidal PCO$_2$ were stable. The incremental ramp test also served as a medical screening of older subjects, was monitored by a physician, and included a 12-lead ECG.

After this test, subjects returned to the laboratory on eight separate occasions to perform step transitions in work rate (WR) from 20 W to a moderate-intensity WR selected to elicit a VO$_2$peak, corresponding to 80% $\theta_P$. Each moderate-intensity WR transition was 6 min in duration and was preceded by either no warm-up exercise or 6 min of heavy-intensity warm-up exercise at a WR selected to elicit a VO$_2$peak equivalent to ~50% of the difference ($\Delta$VO$_2$peak) between the VO$_2$peak at $\theta_P$ and peak VO$_2$. In the heavy-intensity warm-up condition, the heavy-intensity exercise was preceded and followed by 6 min of 20-W cycling. Four transitions were performed in each of the warm-up conditions in random order.

Analysis. Breath-by-breath gas-exchange data were filtered for aberrant data points, interpolated to 1-s intervals, ensemble averaged, and then averaged into 5-s time bins to yield a single response for each subject. Phase 2 VO$_2$peak kinetics were determined by the use of a monoexponential model of the form

$$Y(t) = Y_b + A \cdot \left[1 - e^{-(t - TD)/\tau}\right]$$

where $Y$ represents VO$_2$peak at any time (t), $A$ is the baseline value of $Y$ at the point in time from which the data were fitted, $A$ is the amplitude of the increase in $Y$ above the baseline value, $\tau$ is the time constant defined as the duration of time through which $Y$ increases to a value equivalent to 63% of $A$, and TD is the time delay. Data were fit from the phase 1-phase 2 interface to minute 4 of exercise.

The NIRS-derived O$_2$Hb, HHb, and Hb$_{tot}$ data were time aligned and ensemble averaged to 5-s time bins to yield a single response for each subject. The time delay before an increase in HHb after exercise onset was determined as the first point greater than one standard deviation above the mean of the pretransit (20 W) baseline. This analysis was performed on the second-by-second data for each of the individual trials; the time delay was calculated as the average of the four trials for each subject. HHb data were fit from the time of initial increase in HHb to 180 s with a monoexponential model of the form in Eq. 1 to determine the time course of muscle deoxygenation. Although we are not certain that the underlying processes determining muscle deoxygenation are exponential in nature, visual inspection of the NIRS-derived HHb signal and analysis of least squares residuals suggested that fitting with a monoexponential model would provide a reasonable estimate of the time course of muscle deoxygenation (i.e., effective $\tau$). The time course for the overall change in HHb was also determined as the sum of the time delay before an increase in HHb and effective $\tau$-HHb [mean response time (MRT)]. In addition, the time course of increase of HHb during the on-transient was also determined as the time to reach 63% (63%) of the steady-state response observed at 3–4 min. The O$_2$Hb and Hb$_{tot}$ signals did not approximate an exponential response as changes in local blood volume will influence these signals, whereas the HHb signal is essentially
blood volume insensitive (16). The O₂-Hb and Hb_rest signals were not modeled; however, the response of these signals was compared qualitatively to the HHb data at corresponding time intervals.

Beat-by-beat HR data were filtered for aberrant beats, time-aligned, and averaged to 5-s time bins. These data were then fitted from exercise onset with a monoexponential model of the form in Eq. 1.

Statistical analysis. Analysis of the effects of age and warm-up on V̇O₂p, HR, and HHb kinetics was by two-way repeated-measures ANOVA. Significant differences were further tested by Student-Newman-Keuls post hoc analysis. Comparisons of V̇O₂p, HR, and HHb kinetics within a group (young vs. older) and within condition (no warm-up vs. heavy-intensity warm-up) was by independent t-test. Relationships among key variables were determined by Pearson product correlation. All data are presented as means ± SD. A P value of <0.05 was considered statistically significant.

RESULTS

The older adults (68 ± 3 yr) were similar (i.e., not significantly different) in height (1.78 ± 0.03 m) and body mass (84 ± 11 kg) to the young adults (25 ± 3 yr; height 1.78 ± 0.03 m; mass 77 ± 6 kg). The V̇O₂p peak (2.3 ± 0.3 l/min; 27 ± 4 ml·kg⁻¹·min⁻¹) and peak WR (220 ± 29 W) of older adults was lower (P < 0.05) than that of young adults (3.6 ± 0.6 l/min; 48 ± 7 ml·kg⁻¹·min⁻¹ at 343 ± 63 W).

V̇O₂ kinetics. The adaptation of V̇O₂p at the onset of moderate-intensity exercise after no warm-up and heavy-intensity warm-up for a representative older and young subject is illustrated in Fig. 1. To confirm that a moderate-intensity steady-state V̇O₂p had been achieved and that a slow component of V̇O₂p characteristic of heavy-intensity exercise performed above 0.1v_1 did not exist, the slopes of the V̇O₂p responses between 3 and 6 min of no warm-up and heavy-intensity warm-up in older and young adults were calculated. The V̇O₂p slope was not different from zero for the older or young adults during either no warm-up (older: 4 ± 4 ml/min; young: 8 ± 4 ml/min) or heavy-intensity warm-up (older: 2 ± 5 ml/min; young: 8 ± 13 ml/min) conditions. Additionally, end-exercise V̇O₂p (calculated as the average V̇O₂p during the last 30 s of the exercise transient) was similar to the sum of the V̇O₂p baseline and amplitude from modeling in older and young adults after no warm-up and heavy-intensity warm-up exercise.

The 20-W pretransition baseline V̇O₂p was similar in older and young adults in the no warm-up condition. Heavy-intensity warm-up resulted in a higher (P < 0.01) pretransition baseline V̇O₂p in both older and young adults. As expected in relation to the lower absolute WR during heavy-intensity warm-up in older adults, the increase in baseline V̇O₂p after heavy-intensity warm-up exercise was smaller in older (0.11 l/min) than in young adults (0.17 l/min), such that baseline V̇O₂p was lower (P = 0.046) in older compared with young adults after heavy-intensity warm-up (Table 1). The amplitude of the increase in V̇O₂p was lower (P < 0.01) in older compared with young adults after both warm-up conditions, reflecting the lower absolute moderate-intensity WR in older adults. The amplitude of the increase in V̇O₂p was lower (P < 0.01) after heavy-intensity warm-up compared with no warm-up in young subjects, whereas the increase in V̇O₂p was not different between warm-up conditions in older adults (Table 1). The resulting end-exercise V̇O₂p was lower (P < 0.05) in no warm-up

Table 1. Parameters of V̇O₂p kinetics for moderate-intensity cycle exercise with NWU and after HWU in young and older adults

<table>
<thead>
<tr>
<th>Young</th>
<th>Older</th>
</tr>
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<tbody>
<tr>
<td>NWU</td>
<td>HWU</td>
</tr>
<tr>
<td>Baseline V̇O₂p l/min</td>
<td>0.91±0.12</td>
</tr>
<tr>
<td>V̇O₂p amplitude, l/min</td>
<td>0.79±0.21</td>
</tr>
<tr>
<td>τV̇O₂p, s</td>
<td>29±7</td>
</tr>
</tbody>
</table>

Values are means ± SD. NWU, no warm-up; HWU, high-intensity warm-up; V̇O₂p, pulmonary O₂ uptake; τ, time constant of the response. Significant difference between HWU and NWU conditions within an age group at *P < 0.05 and †P < 0.01. Significant difference between young and older adults at *P < 0.05 and †P < 0.01.
compared with heavy-intensity warm-up in older (no warm-up: 1.16 ± 0.15 l/min; heavy-intensity warm-up: 1.24 ± 0.17 l/min) and young adults (no warm-up: 1.70 ± 0.28 l/min; heavy-intensity warm-up: 1.78 ± 0.29 l/min); end-exercise \( \dot{V}_{O_2}\) was lower (\( P < 0.01 \)) in older compared with young adults in both warm-up conditions. The end-exercise change in \( \dot{V}_{O_2}\)-to-change in WR ratio (\( \Delta \dot{V}_{O_2}/\Delta \dot{W}_R \)) was similar in older and young adults for both no warm-up (older: 10 ± 1 ml/W; young: 11 ± 4 ml/W) and heavy-intensity warm-up (older: 9 ± 1 ml/W; young: 9 ± 3 ml/W).

\( \tau \dot{V}_{O_2} \) was greater (\( P < 0.01 \)) in older compared with young adults in the no warm-up condition (Fig. 1, Table 1). After heavy-intensity warm-up, \( \tau \dot{V}_{O_2} \) was reduced (\( P < 0.01 \)) in older adults but was unchanged in the young adults, such that after heavy-intensity warm-up \( \tau \dot{V}_{O_2} \) was similar in older and young adults (Table 1).

HR. The adaptation of HR at the onset of moderate-intensity exercise after no warm-up and heavy-intensity warm-up for a representative older and young subject is illustrated in Fig. 2. Baseline HR during no warm-up was similar in older and young adults (Table 2) and was elevated (\( P < 0.001 \)) after heavy-intensity warm-up in older (17 beats/min) and young (18 beats/min) adults. The amplitude of the increase in HR was lower (\( P < 0.01 \)) in older compared with young adults during both warm-up conditions (Table 2). The rate of adaptation of HR (\( \tau \text{HR} \)) was not statistically different in older (47 ± 24 s) and young adults (23 ± 12 s) in the no warm-up condition (Table 2). When the \( \tau \text{HR} \) data in the no warm-up condition were analyzed separately by independent \( t \)-test, the apparent difference in \( \tau \text{HR} \) between older and young adults was significant (\( P < 0.05 \)). After heavy-intensity warm-up, \( \tau \text{HR} \) was similar to the no warm-up condition in older adults, but in young adults \( \tau \text{HR} \) was 22 s greater (\( P < 0.05 \)), resulting in a similar \( \tau \text{HR} \) in older and young adults after heavy-intensity warm-up (older: 51 ± 31 s; young: 45 ± 36 s).

\textit{NIRS.} The adaptation of change in HHb (\( \Delta \text{HHb} \)) at the onset of moderate-intensity exercise after no warm-up and heavy-intensity warm-up for a representative older and young subject is illustrated in Fig. 3. The time delay before an increase NIRS-derived \( \Delta \text{HHb} \) was similar in older and young adults in both warm-up conditions (Table 3). The time delay before an increase in HHb was shorter (\( P < 0.05 \)) after heavy-intensity warm-up compared with no warm-up in older and young adults (Table 3). After the time delay, \( \Delta \text{HHb} \) increased, with the effective \( \tau \text{HHb} \) and MRT-\( \text{HHb} \) being similar in older and young adults after no-warm-up; in older adults with heavy-intensity warm-up, \( \tau \text{HHb} \) was 31 s; young: 45 s).

Table 2. Parameters of HR kinetics for moderate-intensity cycle exercise with NWU and after HWU in young and older adults

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Older</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NWU</td>
<td>HWU</td>
</tr>
<tr>
<td>Baseline HR, beats/min</td>
<td>86±7</td>
<td>104±10†</td>
</tr>
<tr>
<td>HR amplitude, beats/min</td>
<td>26±7</td>
<td>15±4†</td>
</tr>
<tr>
<td>( \tau \text{HR}, ) s</td>
<td>23±12</td>
<td>45±36</td>
</tr>
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</table>

Values are means ± SD, HR, heart rate. †Significant difference between HWU and NWU conditions within an age group, \( P < 0.001 \). *Significant difference between young and older adults, \( P < 0.01 \).
(ΔVO_{2p}) was similar in older and young adults after no warm-up [older: 13 ± 4 μM·l^{-1}·min^{-1}; young: 7 ± 2 μM (l/min)], whereas after heavy-intensity warm-up the ΔHHb-to-ΔVO_{2p} ratio was greater (P = 0.044) in older compared with young adults [older: 16 ± 9 μM/l/min); young: 10 ± 4 μM/l/min]; the ΔHHb-to-ΔVO_{2p} ratio was similar after both warm-up conditions in older [no warm-up: 13 ± 4 μM/l/min); heavy-intensity warm-up: 16 ± 9 μM/l/min)] and young adults [no warm-up: 7 ± 2 μM/l/min); heavy-intensity warm-up: 10 ± 4 μM/l/min]. After heavy-intensity warm-up, baseline O_{2}Hb and Hb_{tot} were elevated before the onset of subsequent moderate-intensity exercise compared with the no warm-up baseline in both older (O_{2}Hb: 13 ± 9 μM increase; P < 0.05; Hb_{tot}: 12 ± 8 μM increase; P < 0.05) and young (O_{2}Hb: 9 ± 8 μM increase; P < 0.05; Hb_{tot}: 14 ± 10 μM increase; P < 0.05) adults. The increase in O_{2}Hb and Hb_{tot} was similar in older and young adults. The pretransition baseline HHb concentration was not different in young and older adults with no warm-up (older: -4 ± 2 μM; young: -7 ± 3 μM) or after heavy-intensity warm-up (older: -5 ± 2 μM; young: -4 ± 3 μM).

Relationships among VO_{2p}, HR, and HHb kinetics. The effective τHHb during the on-transient of exercise was less than (P < 0.001) the τVO_{2p} in older and young subjects after no warm-up and heavy-intensity warm-up exercise (Tables 1 and 3). The MRT-HHb was less (P < 0.001) than the τVO_{2p} in older adults after no warm-up, whereas after heavy-intensity warm-up the MRT-HHb and τVO_{2p} were similar. In young adults, the MRT-HHb and τVO_{2p} were similar in both warm-up conditions. In older adults, τVO_{2p} was not correlated with the MRT-HHb after no warm-up; however, the VO_{2p} was negatively correlated with the MRT-HHb (r = -0.889; P = 0.043) after heavy-intensity warm-up. In contrast, the τVO_{2p} was not correlated with the MRT-HHb after either warm-up in young adults.

**DISCUSSION**

This study examined the effect of prior heavy-intensity exercise on the adaptation of VO_{2p} and muscle HHb at the onset of a subsequent bout of moderate-intensity leg-cycling exercise in older and young adults. The main findings were 1) without prior warm-up exercise, VO_{2p} kinetics were slower (P < 0.01) in older compared with young adults; 2) after heavy-intensity warm-up, VO_{2p} kinetics were similar in older and young adults as a consequence of a faster (P < 0.05) adaptation in older adults; 3) the adaptation of HHb at exercise onset (i.e., τHHb...
and MRT-HHb) was similar in older and young adults without warm-up; and 4) with heavy-intensity warm-up, the adaptation of HHb was slowed ($P < 0.01$) in older but not in young adults. Collectively, these results suggest that faster moderate-intensity VO$_{2p}$ kinetics in older adults after heavy-intensity warm-up were related to an improvement in local muscle perfusion and O$_2$ delivery at the onset of exercise. Furthermore, the slower VO$_{2p}$ kinetics in older adults without prior warm-up exercise may be due to an O$_2$ delivery limitation.

VO$_{2p}$ kinetics were slower during moderate-intensity exercise (without prior warm-up) in older adults consistent with previous reports from our laboratory (2, 6, 13, 14, 20, 41). In agreement with Scheuermann et al. (41), after heavy-intensity warm-up, VO$_{2p}$ kinetics were speeded in older ($P < 0.05$), but not in young adults, and resulted in similar VO$_{2p}$ kinetics in older and young adults. Thus this influence of heavy-intensity warm-up exercise on VO$_{2p}$ kinetics in older but not in young adults suggests that the limitation to the adaptation of VO$_{2p}$ (and presumably muscle VO$_2$) is different in older and young adults in the moderate-intensity exercise domain.

Previous studies have attributed slower VO$_{2p}$ kinetics in older adults to 1) a slower adaptation of muscle blood flow/O$_2$ delivery (20, 41) and/or 2) a slower rate of activation and a slowing of the biochemical processes regulating oxidative phosphorylation (7). Heavy-intensity warm-up has been presumed to increase muscle blood flow (28) with the implication that the faster VO$_{2p}$ kinetics after heavy-intensity warm-up in older adults in the present study may be the result of an improvement in muscle perfusion and O$_2$ delivery before exercise onset. Previous studies have demonstrated the potential for an O$_2$ delivery limitation in older adults at the onset of moderate-intensity exercise. Age-related changes, which suggest a potential O$_2$ delivery limitation in older adults, include 1) a lower resting cardiac output (Q) (39) and lower exercise Q relative to VO$_{2p}$ (33, 44), 2) a lower resting limb blood flow and limb vascular conductance (22–24), 3) lower leg blood flow during submaximal exercise (38, 40, 48), 4) an impaired ability to redistribute blood flow from the splanchnic and renal circulations to muscle during exercise (30), and 5) decrements in endothelium-dependent vasodilation (35, 43) and myogenic responsiveness (34), which have implications for blood flow distribution.

In the present study, the adaptation of HR was used as an indicator of the rate of adaptation of Q and presumably muscle O$_2$ delivery. HR kinetics during moderate-intensity exercise were slower in older ($\tau_{HR}$, 47 s) compared with young adults ($\tau_{HR}$, 23 s) without warm-up, in agreement with others (41). Nevertheless, after heavy-intensity warm-up, baseline HR was significantly elevated compared with the no warm-up condition, suggesting that Q, muscle blood flow, and O$_2$ delivery were elevated before the onset of the subsequent exercise bout in both older and young adults. An inherent limitation to the use of HR kinetics is that it provides an indirect estimate of the adaptation of local muscle blood flow. However, the adaptation of HR is commonly used to examine the adaptation of Q because stroke volume probably changes little after the initial adaptation from rest to the initial WR (20 W in the present study), and further increases in stroke volume during the exercise transient likely occur over the first heart beats of the transient secondary to the muscle pump effect on end-diastolic volume. Therefore, HR kinetics provide a reasonable approximation of Q kinetics, as suggested by the data of De Cort et al. (19) and Yoshida and Whipp (49).

To date, technical limitations have precluded study of muscle blood flow and O$_2$ delivery at the level of the microvasculature within the active muscle in the exercising human. NIRS, however, allows the relative concentration changes of HHb, O$_2$Hb, and Hb$_{tot}$ to be monitored noninvasively and continuously throughout the transition to and into the steady state of dynamic exercise (21). NIRS measurements primarily reflect changes in the small arterioles, capillaries, and venules (9), and, assuming that the largest proportion of vascular volume resides in the capillaries (37), any change in HHb reflects the balance between O$_2$ delivery and O$_2$ utilization in the microvasculature of the region of muscle being interrogated. In contrast to measurement of an arteriovenous O$_2$ difference calculated across the limb, NIRS provides an indirect estimate of muscle O$_2$ extraction. However, Bangsbo et al. (3) have reported that the measurement of venous PO$_2$ may not accurately reflect the dynamics of VO$_2$ in active muscle due to the need to correct for tissue-to-sampling site delays and the influence of blood draining vascular beds from inactive tissues. In agreement, Van Beekveldt et al. (46) have reported that the Fick method of determining metabolic rate provides a global assessment of muscle VO$_2$, and, on the basis of the presence of metabolism/perfusion mismatching in an exercising limb, agreement between NIRS estimates of O$_2$ utilization in an active area of muscle tissue and the more global Fick estimate is unlikely. NIRS data have also been shown to closely reflect the muscle metabolic rate as determined by magnetic resonance spectroscopy-derived PCr changes (a proxy for muscle VO$_2$) (11). Thus the ability to noninvasively monitor the balance between O$_2$ delivery and utilization in the microvasculature of the exercising human with NIRS is a significant advance in the study of muscle VO$_2$. In the present study, NIRS data provided further evidence in support of an increase in muscle blood flow and O$_2$ delivery. Relative to no warm-up, after heavy-intensity warm-up, O$_2$Hb and Hb$_{tot}$ concentrations were significantly elevated in both older (O$_2$Hb: 13 ± 9 µM; Hb$_{tot}$: 12 ± 8 µM) and young (O$_2$Hb: 9 ± 8 µM; Hb$_{tot}$: 14 ± 10 µM) adults, suggesting improved local muscle oxygenation in the region of NIRS interrogation before exercise onset.

Thus, after heavy-intensity warm-up, there was a similar increase in muscle O$_2$ delivery (evidenced by elevation of baseline HR and local oxygenation) in young and older adults, but a significant speeding of VO$_{2p}$ kinetics was observed only in older adults. These results are consistent with the hypothesis that muscle O$_2$ delivery imposed a limitation to VO$_{2p}$ kinetics in older adults during moderate-intensity upright cycle exercise, whereas in young adults VO$_{2p}$ kinetics may be limited by factors other than blood flow and O$_2$ delivery.

Further support for an increase in local muscle O$_2$ delivery being responsible for the speeding of VO$_{2p}$ in older adults is evidenced by the adaptation of the local muscle HHb. Despite slower VO$_{2p}$ kinetics (and presumably muscle VO$_2$) in older compared with young adults without warm-up, the adaptation of HHb was similar in older and young adults. After heavy-intensity warm-up, $\tau$VO$_{2p}$ was not changed in young, but the heavy-intensity warm-up was associated with a faster adaptation of VO$_{2p}$ in older adults, whereas HHb adapted at a slower rate in older compared with young adults and compared with the no warm-up condition. Reflecting the balance between

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local O2 delivery and utilization, the slower adaptation of HHb during the exercise-transient in older adults after heavy-intensity warm-up suggests that O2 delivery was increasing at a faster rate than O2 utilization.

As discussed by Scheuermann et al. (41), factors such as substrate provision and changes in the activation and/or rate of metabolic processes regulating oxidative phosphorylation cannot be overlooked when the mechanisms responsible for the speeding of $\tau_{O2p}$ are considered in older adults after heavy-intensity warm-up. However, as argued in that study, there is little evidence supporting a greater metabolic limitation to mitochondrial respiration in older compared with young adults during moderate-intensity exercise. Presumably, an increase in substrate provision and/or metabolic activation would impact primarily on the ability of the muscle to utilize O2 and, as such, would be reflected in the NIRS-HHb signal as a faster rate of muscle deoxygenation at the onset of exercise. However, in the present study, a slower, not faster, adaptation of HHb was observed in older adults after heavy-intensity warm-up.

Thus an increase in local muscle O2 delivery before the onset of exercise may be particularly important to the older adult because it may serve to alleviate decrements in the ability to deliver and distribute blood flow and O2 delivery to active skeletal muscles during the exercise on-transient.

Also, after no warm-up, a similar time delay before an increase in the NIRS-derived HHb signal above preexercise baseline levels was observed in older (11 ± 2 s) and young (12 ± 2 s) adults. After heavy-intensity warm-up, this time delay was shorter in both older (8 ± 2 s) and young (10 ± 2 s) adults. We (21) and others (29) have previously documented a delay before an increase in HHb in young subjects at the onset of moderate-intensity cycling exercise, and the potential explanations for this delay have been discussed in detail (21). We believe that the HHb delay reflects a complex balance between Hb/Mb deoxygenation, O2 delivery, and the effect of muscle contraction on microvascular volume, such that muscle $V_O2$ is actually increasing during the delay, and an increase in HHb is “masked” by other factors, which impact on the volume of Hb in the field of NIRS interrogation.

In conclusion, this study demonstrated slower $V_O2p$ kinetics but a similar adaptation of muscle HHb in older compared with young adults during a moderate-intensity WR transition without prior warm-up exercise. After heavy-intensity warm-up exercise, $V_O2p$ kinetics were accelerated in older adults, and the adaptation of HHb was slowed during subsequent moderate-intensity exercise. Prior heavy-intensity warm-up did not alter the adaptation of $V_O2p$ kinetics and HHb in young adults. These results suggest that heavy-intensity warm-up exercise resulted in an improvement in local muscle perfusion and O2 delivery at the onset of a subsequent moderate-intensity exercise bout and that the speeding of $V_O2p$ kinetics in older adults is due to an improvement in muscle O2 delivery. Furthermore, these results suggest that, without prior warm-up exercise, muscle blood flow in older compared with young subjects may be lower and adapt at a slower rate, and thus the slower $V_O2p$ kinetics observed in older adults may be the result of a lower O2 delivery in older relative to young adults. These results also demonstrate that, in young adults, heavy-intensity warm-up exercise does not alter the adaptation of $V_O2p$ kinetics and muscle deoxygenation at the onset of a subsequent moderate-intensity exercise bout, and thus blood flow and O2 delivery are unlikely to limit $V_O2p$ kinetics in young adults during moderate-intensity exercise.

GRANTS

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