CHAPTER 6

STUDY 3

$\dot{V}O_2$ and mOxy slow components determined during heavy- and severe-intensity exercise in well-trained young and middle-aged cyclists

OVERVIEW

The purpose of Study Three was to examine the effect of age on the development of the $\dot{V}O_2$ and mOxy slow components during heavy- and severe-intensity SWT in well-trained cyclists. The results suggest no significant effect of age in the $\dot{V}O_2$ or mOxy slow components in either the heavy- or severe-intensity SWT in well-trained cyclists. However, both the $\dot{V}O_2$ and mOxy slow components demonstrated significant main effects of intensity in the TD_s but not in the A_s or τ_s. The $\dot{V}O_2$ wMRT was significantly longer in the heavy-intensity SWT, whereas the mOxy wMRT was not significantly affected by SWT intensity in either age group.

No significant relationships were observed between the $\dot{V}O_2$ or mOxy slow components with any of the hematological responses or histochemical and enzymatic characteristics of either the young or middle-aged cyclists. Additionally, the sEMG responses of the VL and VM demonstrated no significant effects of age in the well-trained cyclists during the heavy or severe-intensity SWT. However, several significant effects of time were observed for the sEMG measures in the VL and VM across both SWT intensities in both age groups. The changes observed in the

neuromuscular activity of the VL and VM were not significantly related to the development of the $\dot{V}O_2$ or mOxy slow components in either age group.

In summary, the major finding of Study Three was the absence of any age-related differences in the $\dot{V}O_2$ or mOxy slow components in well-trained cyclists or the proposed causal mechanisms across the high- and severe-intensity SWT.

RESULTS

$\dot{V}O_2$ Slow Component

The time and amplitude parameters of the $\dot{V}O_2$ slow component measured during the heavy and severe-intensity SWT are shown in Table 6.1.

No significant main effects of age, intensity or age x intensity interactions were observed for the $\dot{V}O_2$ A_s. A strong and positive relationship was observed between the $\dot{V}O_2$ A_p and A_s during the heavy-intensity SWT ($r= 0.86$, $p = 0.01$) in the young cyclists but not the middle-aged cohort ($r= 0.27$, $p = 0.55$).

No significant main effects of age, intensity or age x intensity interactions were observed for the $\dot{V}O_2$ TD_s and τ_s. However, *post-hoc* analysis revealed a significantly faster $\dot{V}O_2$ τ_s in the young cyclists in the severe-intensity ($F(1,5)= 7.497$, $p=0.041$, $\eta^2=0.600$) compared to that of the heavy-intensity SWT.

Table 6.1: Mean (± SD) time and amplitude values of the $\dot{V}O_2$ slow component of the young and middle-aged cyclists measured during the heavy- and severe-intensity square wave transition.

	Young		Middle-aged	
	Heavy	**Severe**	**Heavy**	**Severe**
A_s ($mL \cdot min^{-1}$)	550 ± 264	708 ± 236	643 ± 253	350
TD_s (s)	127.7 ± 23.8	85.7 ± 29.6	89.6 ± 23.9	96
τ_s (s)	105.8 ± 35.0	62.4 ± 21.8 ¥	111.3 ± 42.1	62.3
G_s ($mL \cdot min^{-1} \cdot W^{-1}$)	1.9 ± 0.8	2.2 ± 0.7	2.2 ± 0.7	1.2
G_o ($mL \cdot min^{-1} \cdot W^{-1}$)	8.8 ± 1.2	7.7 ± 1.3	8.3 ± 1.3	6.3 ± 1.5
EE$\dot{V}O_2$ ($mL \cdot min^{-1}$)	3487 ± 510	3405 ± 418	3648 ± 345	3636 ± 363
wMRT (s)	54.4 ± 11.8	38.7 ± 11.7 ¥	51.2 ± 14.5	29.2 ± 7.5 ¥

A_s = Slow Component Amplitude
TD_s = Slow Component Time Delay
τ_s = Slow Component Time Constant
G_s = Slow Component Gain
G_o = Overall Gain
EE$\dot{V}O_2$ = End-Exercise $\dot{V}O_2$
wMRT= Weighted Mean Response Time

¥ significant difference between heavy and severe-intensities ($p<0.05$); € no subject completed the six minute severe-intensity SWT.

mOxy Slow Component

The time and amplitude parameters of the mOxy slow component across the heavy and severe-intensity SWT are shown in Table 6.2.

No significant main effect of age or age x intensity interaction was observed in the mOxy $A_{s.}$ However, the mOxy A_s demonstrated a significant main effect of intensity ($F(2,22)=15.422$, $p=0.002$, $\eta^2=0.794$). This effect of intensity was not observed in the separated young or middle-aged cohorts.

No significant main effect of age, intensity or age x intensity interaction was observed for the mOxy TD_s. However, the mOxy TD_s was found to be significantly (t = -5.434, $p<0.001$) longer than the $\dot{V}O_2$ TD_s during the heavy-intensity SWT in the middle-aged cyclists, but not the young cyclists,.

No significant effect of age or age x intensity interaction was observed for the mOxy τ_s. However, a significant main effect of intensity ($F(28,)=8.296$, $p=0.011$, $\eta^2=0.675$) was observed for the mOxy τ_s in the well-trained cyclists. This significant effect of intensity was only observed in the middle-aged cyclists ($F(2,6)=11.854$, $p=0.008$, $\eta^2=0.798$) between the heavy- and severe-intensity SWT.

Table 6.2: Mean (± SD) time and amplitude values of the mOxy slow component of the young and middle-aged cyclists measured during the heavy- and severe-intensity square wave transition.

	Young		Middle-Aged	
	Heavy	**Severe** [€]	**Heavy**	**Severe** [€]
A_s (%)	18.5 ± 5.9	20.0 ± 10.2	17.4 ± 4.3	17.0 ± 7.8
TD_s (s)	184.5 ± 67.3	92.5 ± 11.2	182.4 ± 61.59	75.1 ± 25.1
τ_s (s)	105.6 ± 31.5	70.2 ± 32.8	179.1 ± 56.9	55.9 ± 15.3 [¥]
G_s (%•W^{-1})	0.065 ± 0.024	0.052 ± 0.022	0.04 ± 0.04	0.04 ± 0.02
G_o (%•W^{-1})	0.18 ± 0.10	0.17 ± 0.07	0.19 ± 0.05	0.16 ± 0.05 [¥]
EEmOxy (%)	31.0 ± 19.9	7.8 ± 8.4 [¥]	35.6± 12.2	32.3 ± 15.0 [¥]
wMRT (s)	47.6 ± 20.2	37.6 ± 18.2	64.4 ± 37.2	34.5 ± 12.3

A_s = Slow Component Amplitude
TD_s = Slow Component Time Delay
τ_s = Slow Component Time Constant
G_s = Slow Component Gain
G_o = Overall Gain
EEmOxy = End-Exercise mOxy
wMRT= Weighted Mean Response Time

[¥] significant difference between heavy and severe-intensities ($p<0.05$); [€] no subject completed the six minute severe-intensity SWT.

Electromyographic Responses

The mean (± SEM) changes in the iEMG responses on the VL and VM are shown in Figure 6.1. The mean (± SEM) changes in the MPF of the sEMG signal from the VL and VM across the two high-intensity SWT are shown in Figure 6.2.

The majority of sEMG parameters demonstrated no significant effect of age, with only the iEMG response of the VM during the heavy-intensity SWT being significantly different between the two age-groups ($F(1,12)=5.464$, $p=0.039$, $\eta^2=0.332$). No significant age x time interactions were observed in the iEMG or MPF response from the VL and VM. However, a significant main effect of time was observed for the changes in the increasing iEMG response of the VL during the heavy-intensity ($F(12,144)=4.674$, $p<0.001$, $\eta^2=0.280$) SWT. Significant main effects of time were also observed in decreases in the MPF of the VL during the heavy-intensity SWT ($F(12,144)=3.467$, $p<0.001$, $\eta^2=0.224$).

No significant changes were observed in the iEMG of the VM across either SWT intensity. The middle-aged cyclists demonstrated a significant increase across of time ($F(12,72)=3.602$, $p<0.001$, $\eta^2=0.375$) in the iEMG of the VL during the heavy-intensity SWT. The young cyclists demonstrated a significant decrease across time in the MPF of VL during the heavy-intensity SWT ($F(12,72)=3.832$, $p<0.001$, $\eta^2=0.390$).

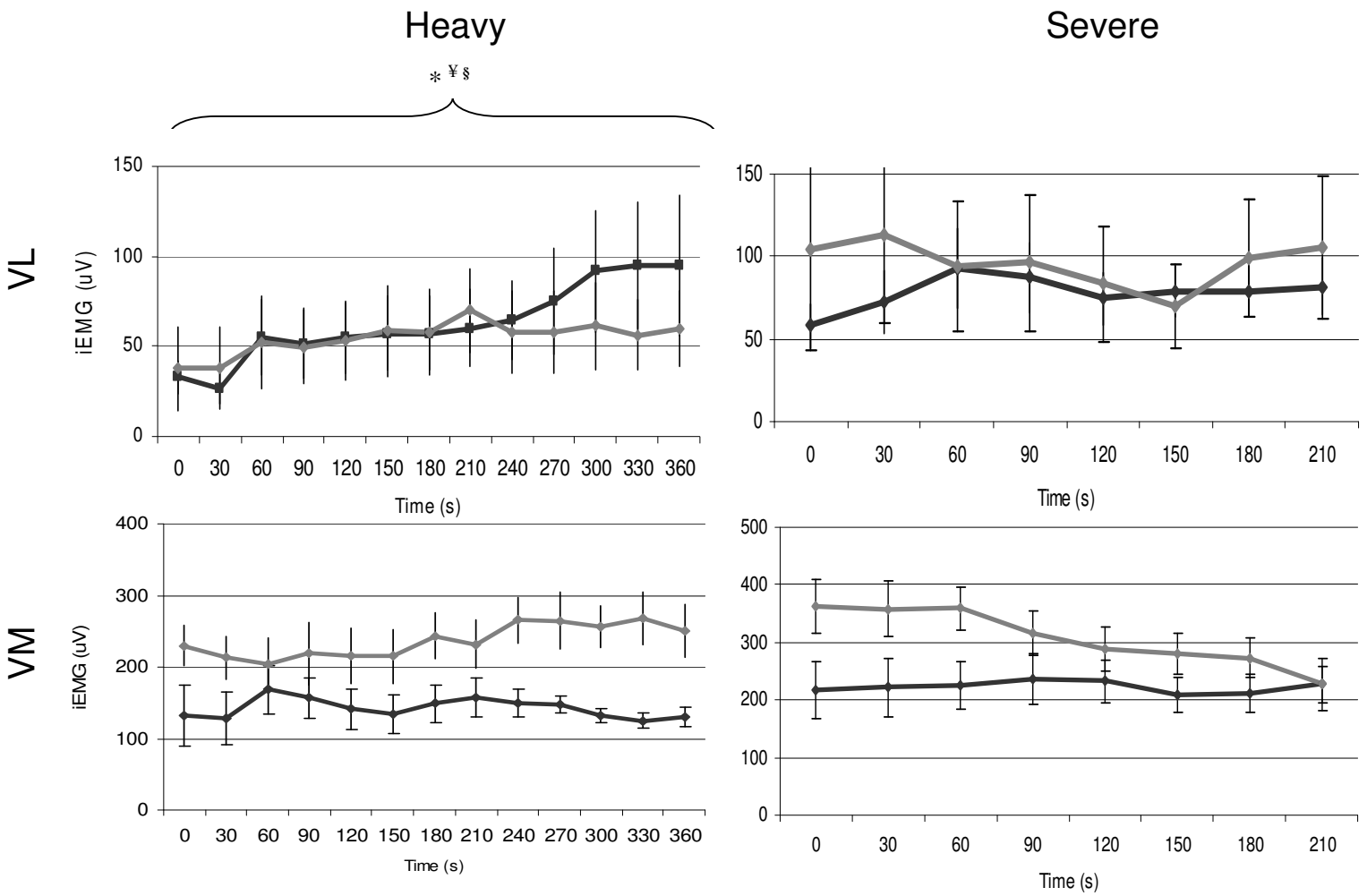

Figure 6.1: Mean (± SEM) iEMG responses from the vastus lateralis (VL) (top) and vastus medialis (VM) (bottom) during the heavy- (left) and severe-intensity(right) SWT in the young ( ) and middle-aged ( ) cyclists (* significant effect of age ($p<0.05$); ¥ significant main effect of time in vastus lateralis ($p<0.05$); § significant increase across time in vastus lateralis of middle-aged cyclists ($p<0.05$)).

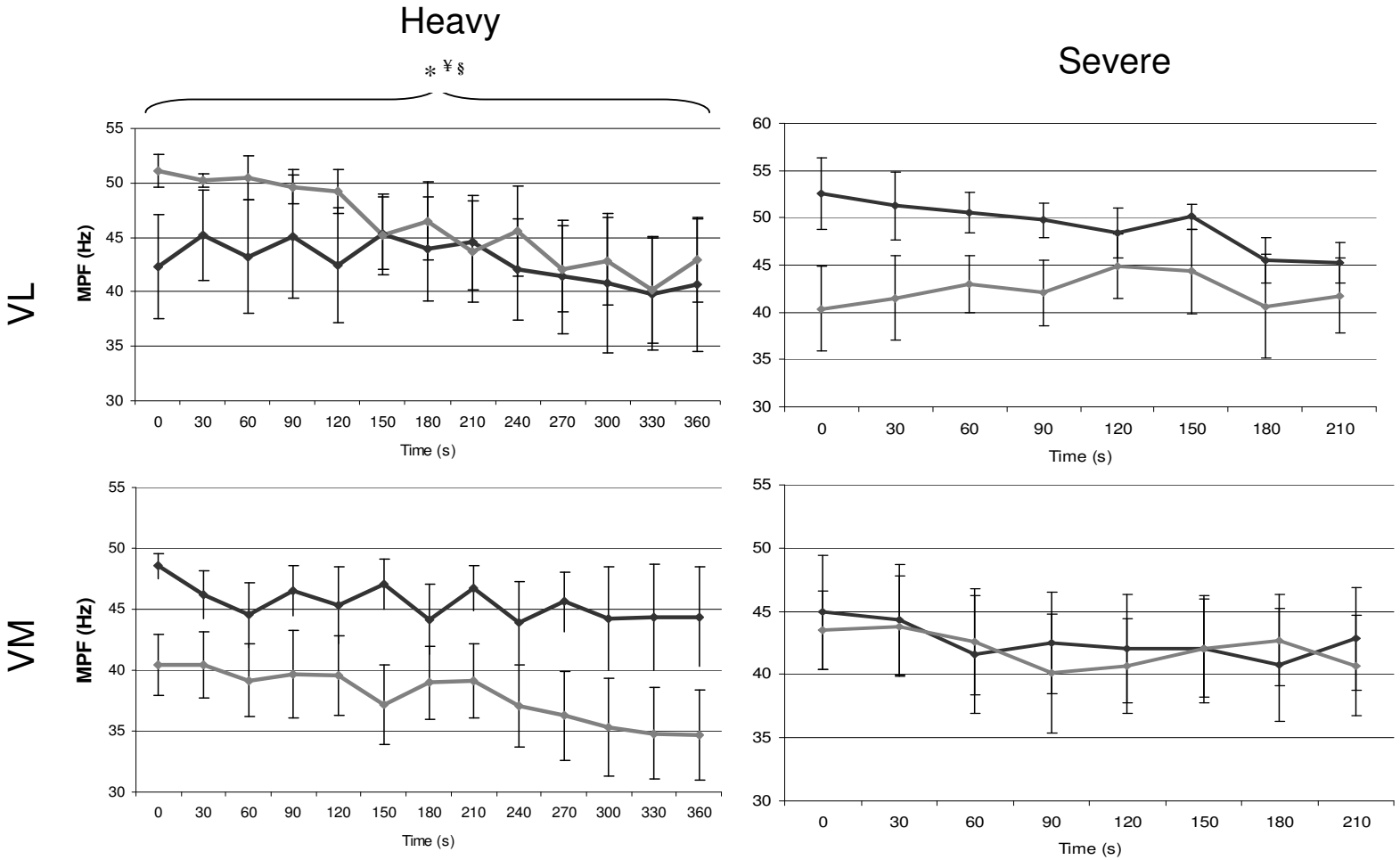

Figure 6.2: Mean (± SEM) MPF responses from the vastus lateralis (VL) (top) and vastus medialis (VM) (bottom) during the heavy- (left) and severe-intensity (right) square wave transition in the young ( ) and middle-aged ( ) cyclists (* significant effect of age ($p<0.05$); ¥ significant main effect of time in vastus lateralis ($p<0.05$); § significant decrease with time in vastus lateralis of young cyclists ($p<0.05$)).

DISCUSSION

The purpose of Study Three was to examine the effect of age on the development of the $\dot{V}O_2$ and mOxy slow components observed across heavy- and severe-intensity SWT in well-trained cyclists. The present study is the first to investigate the effect of age on the development of these slow components during high-intensity exercise in well-trained cyclists.

The results of the present study demonstrated no significant effect of age or age x intensity interactions in either the $\dot{V}O_2$ or mOxy slow components. Furthermore, the present results demonstrated no significant relationships between the $\dot{V}O_2$ or mOxy slow components and changes in the hematological (blood pH, pO_2, $[HCO_3^-]$, $[BLa^-]$) or neuromuscular (iEMG, MPF) parameters across the two high-intensity SWT. Finally, no significant relationships were observed between the development of the slow components and the muscle histochemical or enzymatic characteristics of the VL in either age group. Therefore, the present study suggests that the nature of the $\dot{V}O_2$ and mOxy slow component responses and possible causal mechanisms are similar in matched young and middle-aged cyclists.

At present, the physiological mechanisms responsible for the development of the $\dot{V}O_2$ slow component are not fully understood. A number of possible causal mechanisms have been hypothesised to be responsible for the observed decrease in mechanical or metabolic efficiency during prolonged high-intensity exercise. These factors may include increases in physiological factors such as muscle temperature, adrenaline, ventilation or $[BLa^-]$, or

changes in muscle fibre-specific recruitment patterns (Poole 1994; Poole et al. 1994; Whipp 1994; Gaesser and Poole 1996; Saunders et al. 2000).

Although the exact underlying mechanisms of the slow component remain to be elucidated, it is widely accepted that the majority of this decrease in metabolic efficiency and slow component development occurs within the working muscle (Poole 1994; Demarie et al. 2001). In support of this suggestion, previous evidence has reported that the nature of the $\dot{V}O_2$ slow component is influenced by physiological characteristics such as $\dot{V}O_2$max (Gaesser and Poole 1996; Carter et al. 2000a) and both muscle fibre composition and CSA (Barstow et al. 1996; Pringle et al. 2003b). To date, little research has examined these characteristics and their relationship to the development of the mOxy slow component. Further, no research has examined this relationship in older well-trained athletes. Therefore, it is plausible to suggest that the $\dot{V}O_2$ and mOxy slow components are maintained in well-trained middle-aged cyclists to similar levels of a matched younger cohort. This is most likely a result of the similar physiological and performance characteristics between the two age groups.

Slow Component Amplitude Responses

In the present study, no significant effects of age, intensity or age x intensity interaction were demonstrated in the $\dot{V}O_2$ or mOxy A_s across the heavy and severe-intensity SWT. This finding suggests that physical training into middle-age can maintain the $\dot{V}O_2$ and mOxy A_s, and prevent the decline observed with sedentary aging (Chick et al. 1991; Scheuermann et al. 2002; Sabapathy et al. 2004). The $\dot{V}O_2$ A_s measured in the present study

(Y: ~550 mL•min^{-1}; MA: ~650 mL•min^{-1}) was similar to that observed in previous investigations reporting on both young (Scheuermann et al. 2001; Russell et al. 2002) and older (Sabapathy et al. 2004) populations. The mOxy A_s was difficult to compare to existing literature, given the different methods used to measure this parameter between investigations (e.g. difference between 6th and 3rd minutes vs. separate exponential function) (Demarie et al. 2001).

The absence of a significant effect of age in $\dot{V}O_2$ and mOxy A_s is most likely due to the similar physiological and muscle histochemical characteristics of the well-trained cyclists reported in Study One of the present series of investigations. Despite the majority of previous literature suggesting that the muscle fibre composition of the working muscle has a primary influence on the development of the slow component (Barstow et al. 1996; Pringle et al. 2003b; Garland et al. 2004; Krustrup et al. 2004b), no such effect was observed in the present study.

The reported influence of muscle fibre composition on the slow component is thought to be related to the changes within the metabolic environment in the working muscle across high-intensity constant-load exercise (Poole 1994). The similar changes in blood pH measures across the high-intensity exercise in the young and middle-aged cyclists of the present study may suggest that the magnitude of anaerobic metabolism was comparable between age groups. These changes in blood pH may have resulted from an exhausted aerobic metabolism and an increase in the magnitude of anaerobic metabolic contribution required to match the energetic demands of the high-intensity exercise (Barstow et al. 1993).

Surprisingly, no significant correlations were observed between the $\dot{V}O_2$ and mOxy slow components and the hematological responses in the present study. This finding contrasts those of previous investigations which have observed such relationships across similar exercise intensities as those used in the present study (Demarie et al. 2001). This difference between studies may be the result of the well-trained nature of the two cohorts in the current study. Therefore, the present data suggests that there is no significant relationship between the magnitude of anaerobic metabolism and the amplitude of the $\dot{V}O_2$ and mOxy slow components. Such an absence of significant relationship was also observed with the speed of development of the two slow components.

Slow Component Speed Responses

Within the present study, the $\dot{V}O_2$ and mOxy TD_s demonstrated no significant effect of age or age x intensity interaction. Previous research has reported a significant effect of age on the $\dot{V}O_2$ TD_s during heavy-intensity exercise in sedentary populations (Sabapathy et al. 2004). However, to date no such data are available on the effect of age on the mOxy slow component, or in older well-trained subjects. Both the young (Y) and middle-aged (MA) cyclists examined in the present study demonstrated similar $\dot{V}O_2$ TD_s (Y: 127.7 ± 23.8 s; MA: 85.7 ± 29.6 s) to that reported by Sabapathy and colleagues (2004) for young (21.7 ± 0.9 y) sedentary subjects (118 ± 10 s). However, the elderly (71.6 ± 0.8 y) subjects in this previous study demonstrated a considerably longer $\dot{V}O_2$ TD_s (178 ± 14 s) than the middle-aged cyclists in the present study (Sabapathy et al. 2004). This difference may be due to previously reported and age-related physiological changes of the elderly cohort (Sabapathy et al. 2004). This finding may suggest that if age-related changes in muscle fibre

composition are offset through physical training, then the development of the slow components may not be subject to a significant effect of aging.

In the present study, the $\dot{V}O_2$ and mOxy τ_s demonstrated no significant effect of age or age x intensity interaction. This finding further supports the origin of the slow component being within the working muscle during high-intensity constant-load exercise. Limited previous research has examined the $\dot{V}O_2$ and mOxy τ_s across high-intensity constant-load exercise in any population (Carter et al. 2002). Carter and colleagues (2002) reported that the average $\dot{V}O_2$ τ_s ranged between 220-290 s for supra-threshold exercise intensities between 20-100%Δ in a young (27 ± 7 y) male population. In contrast, the present study is the first to examine the effect of exercise intensity on the mOxy τ_s in any population. In the present study, the young and middle-aged cyclists demonstrated similar $\dot{V}O_2$ and mOxy τ_s across the heavy- (Y: 105.8 ± 35.0 s; MA: 111.3 ± 42.1 s) and severe-intensity (Y: 62.4 ± 21.8 s; MA: 62.3 s) SWT. This may again reflect the similar physiological, muscle histochemical and enzymatic characteristics reported in the two age groups in Study One. These physiological and muscle characteristics have consistently been shown to be related to the observed changes in metabolic efficiency and the slow component across bouts of high-intensity constant-load exercise (Poole 1994; Poole et al. 1994; Whipp 1994; Gaesser and Poole 1996; Saunders et al. 2000).

The $\dot{V}O_2$ and mOxy τ_s responses both demonstrated effects of intensity in the well-trained young and middle-aged cyclists in the present study. The young cyclists demonstrated a significantly shorter $\dot{V}O_2$ τ_s in the severe-

intensity compared to the heavy-intensity in the present study. However, this was not observed in the middle-aged cyclists. This observation should be taken with caution given that only one middle-aged cyclist demonstrated a $\dot{V}O_2$ slow component during the severe-intensity SWT, which made statistical comparisons difficult. In contrast, the mOxy τ_s demonstrated a significant effect of intensity between the heavy (Y: 105.6 ± 31.5 s; MA: 179.1 ± 56.9 s) and severe-intensity (Y: 70.2 ± 32.8 s; MA: 62.4 ± 21.8 s) SWT in the middle-aged cyclists. Therefore, it appears that the controlling mechanisms associated with the development of the slow components may be influenced by exercise intensity.

The different $\dot{V}O_2$ and mOxy responses across the severe-intensity SWT are most likely due to the increased anaerobic energetic demands in the well-trained cyclists of the current study. This near-maximal SWT intensity may have facilitated greater disturbance in the metabolic environment and faster fibre-specific fatigue of the working muscle prior to fatigue. In comparison to pasta literature, the slower $\dot{V}O_2$ and mOxy slow components observed in the present study may be a result of the difference in the SWT relative intensity, and the ability of the cyclists to complete the heavy-intensity SWT. During the heavy-intensity SWT, the proposed changes within the metabolic environment and recruitment patterns of the working muscle may not have occurred as quickly as that in the severe-intensity SWT. As such, it is likely that the magnitude of anaerobic metabolism and shifts in muscle fibre recruitment patterns may be augmented with increasing exercise intensity. This was supported by the changes in several hematological parameters in the present study. This difference in the energetic responses in response to the two work intensities

may be reflected as a significant effect of intensity in the development of the $\dot{V}O_2$ and mOxy slow components. However, the present data contrast previous literature suggesting that the $\dot{V}O_2$ τ_s remains stable across increasing high-intensity work rates (Carter et al. 2002). The present data are the first to report upon a significant effect of intensity on the mOxy slow component, with the severe-intensity mOxy slow component developing significantly faster than that observed during heavy-intensity slow component. However, this may reflect the elevated [BLa^-] prior to commencing the severe-intensity SWT in the present study, which may have helped O_2 unloading within the muscle and a faster decrease in efficiency. Alternateivly, it may suggest differences in the energy metabolism requirements of the working muscle between the heavy and severe intensities.

The results of Study Three suggested no significant effect of age in the $\dot{V}O_2$ or mOxy slow components observed in the well-trained cyclists across the high-intensity exercise bouts (Sabapathy et al. 2004). Past investigations have suggested that the development of the $\dot{V}O_2$ slow component during high-intensity constant-load exercise is slowed with sedentary aging, whereas the present investigation did not report such an effect in the well-trained middle-aged cyclists. The effect of intensity observed in the speeding of development of the $\dot{V}O_2$ and mOxy slow components suggests that the causal mechanisms lie within gradual changes in the energetic processes within the actual working muscle. In summary, no significant relationships were observed between the cyclist's physical characteristics and the development of the slow components in the present study. This similar absence of relationships between the two age

groups most likely reflects their similar $\dot{V}O_2$max and muscle histochemical and enzymatic characteristics

Slow Component Physiological Mechanisms

Previous investigations have suggested the slow components are developed due to increases in muscle temperature, ventilation, adrenaline or [BLa^-] (Poole et al. 1994; Gaesser and Poole 1996; Zoladz and Korzeniewski 2001). More recent studies have suggested that a decrease in efficiency of the working muscle may due in part to shifts in fibre-type recruitment during high-intensity constant-load exercise (Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004b; Sabapathy et al. 2005). However, in contrast to this suggestion, the present data observed no significant correlations between the $\dot{V}O_2$ or mOxy slow components and any changes in the hematological parameters or muscle fibre composition and recruitment patterns across the high-intensity work bouts.

At present, the two most plausible physiological explanations for the $\dot{V}O_2$ and mOxy slow components appear to be muscle fibre composition (Barstow et al. 1996; Pringle et al. 2003b; Krustrup et al. 2004b) and changes in fibre-specific recruitment patterns (Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004b; Sabapathy et al. 2005). As such, the VL muscle has been identified to be of practical importance for researchers examining the $\dot{V}O_2$ slow component in cycling, given it is a prime mover of the cycle stroke and its activity is linearly related to cycling intensity (Jorge and Hull 1986; Akima, Kinugasa and Kuno 2005; Raymond et al. 2005). The development of the $\dot{V}O_2$ slow component has been previously related to the muscle fibre composition

and recruitment patterns of the VL in numerous investigations (Barstow et al. 1996; Saunders et al. 2000; Borrani et al. 2001; Pringle et al. 2003b; Krustrup et al. 2004b; Sabapathy et al. 2005). The muscle fibre composition appears to influence the metabolic processes within the working muscle during the high-intensity exercise bouts.

The data from the present study did not support a relationship between muscle fibre composition and the development of the $\dot{V}O_2$ and mOxy slow component. Previously, the $\dot{V}O_2$ slow component has been associated with gradual increases in the phosphate cost of high-intensity exercise, which may occur as a result of decreases in muscle oxidative capacity and Type I fibres, or changes in the fibre-specific recruitment patterns during high-intensity exercise (Rossiter et al. 2001). No such relationships were observed between the development of the $\dot{V}O_2$ and mOxy slow components and any muscle histochemical characteristics in the present study. This contrasts with previous investigations that have reported significant inverse correlations between the $\dot{V}O_2$ A_s and Type I fibre composition of the VL during heavy- and severe-intensity cycling (Barstow et al. 1996; Pringle et al. 2003b). The Type I fibre composition of the working muscle may be representative of oxidative capacity of the muscle, given their preferable functional and metabolic characteristics for aerobic metabolism (He et al. 2000). Since Type I muscle fibres possess such higher oxidative capacities and enzyme activities, myoglobin concentrations and capillarisation, it is plausible that these previously reported effects of muscle fibre composition may be related to the development of both the $\dot{V}O_2$ and mOxy slow components. Given that the current data did not lend itself to

this hypothesis, further research is required to support the relationship between muscle fibre composition and the development of the slow component.

The significant relationship previously reported between the $\dot{V}O_2$ slow component development and muscle fibre composition (Barstow et al. 1996; Pringle et al. 2003b; Krustrup et al. 2004b) may suggest that individuals that possess lower oxidative potential (i.e. higher Type II fibre composition) within working muscles are likely to demonstrate an augmented $\dot{V}O_2$ slow component. The significance of muscle fibre composition to the development of the $\dot{V}O_2$ slow component may explain the previously observed effect of sedentary aging on the $\dot{V}O_2$ A_s and τ_s (Sabapathy et al. 2004). While no definitive mechanism has been identified for this effect of age in the $\dot{V}O_2$ slow component, it is possible that it is related to the muscle fibre-specific atrophy reported to occur in sedentary aged populations typically older than that recruited for the present investigation (Deschenes 2004).

Apart from the influence of muscle fibre composition, the consensus of previous research suggests that the $\dot{V}O_2$ slow component is also developed as a result of Type I fibre fatigue and the subsequent gradual recruitment of the less aerobic and less efficient Type II fibres during high-intensity constant-load exercise (Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004a; 2004b). This change in the muscle fibre recruitment patterns is commonly reported through changes in both muscle activity (iEMG) and recruitment frequencies (MPF) of the working muscle (Miura et al. 1999; Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004a; 2004b). The iEMG and MPF responses of the VL have previously been shown to be significantly related to

both the $\dot{V}O_2$ (Saunders et al. 2000; Borrani et al. 2001; Krustrup, et al. 2004a; Krustrup et al. 2004b) and mOxy slow components (Miura et al. 1999; Demarie et al. 2001). However, many investigations have not observed this relationship between changes in muscle fibre recruitment patterns as either iEMG or MPF measures and the development of the $\dot{V}O_2$ slow component (Scheuermann et al. 2001; Cleuziou et al. 2004).

The present study did not show any significant effect of age in either the iEMG or MPF responses during heavy or severe-intensity SWT. Thus, the results of the present study contrast previous investigations that have reported significant relationships between the $\dot{V}O_2$, mOxy and EMG responses across high-intensity constant-load exercise in young healthy males (Miura et al. 1999; Saunders et al. 2000; Demarie et al. 2001). However, the present study observed significant effects of time in both the iEMG and MPF of the VL during the heavy-intensity SWT in both the young and middle-aged cyclists, respectively.

The observed changes in neuromuscular activity of the VL and VM muscles during the heavy-intensity SWT of the present study are suggestive of Type II fibre fatigue and a resultant increase in Type I fibre recruitment. The reported changes in fibre recruitment patterns contrasts that of previous investigations which have proposed that the $\dot{V}O_2$ slow component is due to Type I fibre fatigue and increases in Type II fibre recruitment (Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004b). However, given the high relative power output of the heavy-intensity SWT (50%Δ), it is possible that at the commencement of the high-intensity SWT, a large number of Type II fibres

are recruited to overcome this change in work rate and to maintain the constant power output required. As Type II fibres possess relatively lower oxidative capacities than Type I fibres, they may have fatigued more quickly, as demonstrated by increases in anaerobic metabolism and decreases in metabolic efficiency. The Type II fibre fatigue may have facilitated an increased dependence on Type I fibres to sustain the SWT power output (Bottinelli and Reggiani 2000). Due to their smaller force production capacities, a greater number of Type I fibres may have then been recruited in order to sustain the power output across the high-intensity SWT. These proposed changes in the fibre-specific recruitment patterns are represented as increases in iEMG and decreases in the MPF of the working muscles across the high-intensity SWT, both of which were observed in the present study across the two high-intensity SWT.

This alternative hypothesis of fibre-specific recruitment patterns is also supported by the current study's finding of a non-significant decrease in the MPF of the VL and VM across the high-intensity SWT. Type I fibres are innervated by smaller motor neurons and possess lower threshold frequencies than Type II fibres, as demonstrated through reductions in the MPF of the recruited muscles (De Luca 1985). While Type I muscle fibres possess greater metabolic efficiency and oxidative capacities, their continued recruitment may facilitate decreases in metabolic efficiency across sustained high-intensity exercise (He et al. 2000). He and others (2000) have reported that Type I and II muscle fibres possess similar thermodynamic efficiencies during repeated contractions, but their optimal efficiency occurs at significantly different power outputs and contraction speeds. Therefore, taking into account the high relative

intensities and cadences of the high-intensity SWT in the present study, it might be suggested that the Type I fibres were recruited outside their range of optimal efficiency. This may have been observed as a gradual increase in $\dot{V}O_2$ and decrease in mOxy across the sustained high-intensity exercise bouts.

This physiological hypothesis may contribute to a greater understanding of the mechanisms underlying the $\dot{V}O_2$ and mOxy slow components across such high-intensity work bouts. Therefore, while no observed effect of age was demonstrated in the iEMG or MPF responses in the present study, these results offer a novel alternative to explain the development of the $\dot{V}O_2$ and mOxy slow components.

While the present study's sEMG responses differ to that reported within previous investigations (Saunders et al. 2000; Borrani et al. 2001; Krustrup et al. 2004b; Sabapathy et al. 2005), this may be the result of the novel method of sEMG analysis used within the present investigation. In the present study, the iEMG and MPF analysis of the raw sEMG signal was only performed through the angular range of the crank cycle where the VL (315-110°) and VM (305-135°) are proposed to be recruited (Jorge and Hull 1986). It may be suggested that frequency spectral analysis across such a dynamic task as cycling may be greatly influenced through the inclusion of larger periods of inactivity of the isolated muscles than previous EMG studies have used (Cram et al. 1998). Further, the reporting of the specific sEMG responses across the proposed recruitment range may also decrease the effect of muscle crosstalk and noise (Cram et al. 1998). The present iEMG and MPF responses from the VL and VM are therefore representative of the muscle activity through the narrow muscle-

specific angular recruitment range of the VL and VM muscles from the well-trained young and middle-aged cyclists. This angular-specific analysis technique provides novel and mechanistic information and thus adds to the body of literature attempting to identify the causal mechanisms of the $\dot{V}O_2$ and mOxy slow components.

SUMMARY

In conclusion, the results of Study Three demonstrated that the $\dot{V}O_2$ and mOxy slow components were not significantly influenced by age in well-trained cyclists matched for $\dot{V}O_2$max and muscle histochemical and enzymatic characteristics. To the researcher's knowledge, the present study is the first to demonstrate that concurrent aging and physical training maintains the $\dot{V}O_2$ and mOxy slow components into middle-age compared to a similarly trained younger cohort.

The current results contrast with previous data that have reported a significant effect of sedentary aging on the $\dot{V}O_2$ slow component (Sabapathy et al. 2004), and thus suggest that physical training into older age may maintain energy metabolism and potential slow component causal mechanisms within the peripheral muscle. The absence of a significant effect of age on the $\dot{V}O_2$ and mOxy slow components may be the result of the similar physiological and muscle characteristics reported for the young and middle-aged cyclists. The differences in the speed of the $\dot{V}O_2$ and mOxy slow components suggest that the causal mechanism of the slow component is related to exercise intensity, and changes with subsequent energetic metabolism within the working muscle.

Despite the proposed developmental role of the working muscle on the slow component, the present study did not observe any significant relationships between the slow component development and muscle histochemical characteristics. Previous investigations have suggested that the increased recruitment of Type II fibres may be responsible for the development of the slow component (Miura et al. 1999; Saunders et al. 2000; Demarie et al. 2001). The observed trends in the sEMG responses in the present study contrast previous findings, and suggest an increased recruitment of Type I fibres during prolonged high-intensity constant-load exercise in both age groups. The present study failed to identify any definitive mechanism responsible for the $\dot{V}O_2$ and mOxy slow components. In summary, the development of the $\dot{V}O_2$ and mOxy slow components appears to be maintained in well-trained young and middle-aged cyclists, matched for $\dot{V}O_2$max and muscle histochemical and enzymatic characteristics.