

Research article

Age-associated changes in $\dot{V}O_2$ and power output – A cross-sectional study of endurance trained New Zealand cyclists

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Abstract

Age-associated changes in power and maximal oxygen consumption ($\dot{V}O_{2max}$) were studied in a cross section of endurance trained cyclists. Subjects ($n = 56$) performed incremental cycling exercise, during which capillary blood lactate $[La^-]$ was measured. Power output increased by 30 Watts during each 5 minutes stage, with initial power output based on individual ability. When $[La^-]$ was >4.5 mmol·L⁻¹, subjects were given a 10 min recovery at a power output approximately 50% below estimated power at $[La^-]_{4mmol}$. Subjects then performed an incremental test (1 minute stages) to $\dot{V}O_{2max}$. Decline in $\dot{V}O_{2max}$ was 0.65 ml·kg⁻¹·min⁻¹·year⁻¹ ($r = -0.72$, $p < 0.01$) for males, and 0.39 ml·kg⁻¹·min⁻¹·year⁻¹ ($r = -0.54$, $p < 0.05$) for females. Power at $\dot{V}O_{2max}$ decreased by 0.048 W kg⁻¹·year⁻¹ ($r = -0.72$, $p < 0.01$) in males. Power at $[La^-]_{4mmol}$ decreased by 0.044 W kg⁻¹·year⁻¹ ($r = -0.76$, $p < 0.01$) in males, and by 0.019 W kg⁻¹·year⁻¹ ($r = -0.53$, $p < 0.05$) in females. Heart rate at $\dot{V}O_{2max}$ (HR_{max}) showed a weaker correlation with age in males ($r = -0.36$, $p < 0.05$). The age-associated changes in maximum aerobic power and sub-maximal power were gender-specific, thus suggesting different age-related effects on the systems which support exercise in males and females.

Key words: Maximal oxygen consumption, aging, exercise, performance.

Introduction

For both males and females, a decline in the functional reserve of the systems which support exercise occurs with age (Goldspink, 2005), although regular exercise is known to slow the rate of decline. Trained adult athletes across the age range experience a decline in physiological functional capacity (Tanaka and Seals, 2003), generally with an accelerated decline during (and after) the 6th decade (Tanaka and Seals, 2003). The rate of decline in $\dot{V}O_{2max}$ with age is often reported to be higher in endurance trained compared to sedentary adults, although endurance trained individuals have consistently higher absolute $\dot{V}O_{2max}$ values than their sedentary counterparts (Tanaka and Seals, 2003).

An age-associated decline in $\dot{V}O_{2max}$ of 0.47 ml·kg⁻¹·min⁻¹·year⁻¹ for trained distance runners aged between 35 and 70 years has been reported (Wells et al., 1992), whereas others (Pimentel et al., 2003) reported that $\dot{V}O_{2max}$ declined by 0.54 ml·kg⁻¹·min⁻¹·year⁻¹ in endurance trained subjects ($n = 89$, age range 21-74 years) and by

0.39 ml·kg⁻¹·min⁻¹·year⁻¹ in sedentary subjects ($n = 64$, age range 20-75 years). However, when stratifying for age, endurance trained subjects showed declines of 0.2 ml·kg⁻¹·min⁻¹·year⁻¹ when aged 20 – 50 years, increasing to 0.89 ml·kg⁻¹·min⁻¹·year⁻¹ when aged 50 – 74 years. A cross-sectional study (Katzel et al., 2001) reported declines of 0.42 ml·kg⁻¹·min⁻¹·year⁻¹ and 0.43 ml·kg⁻¹·min⁻¹·year⁻¹ in trained athletes and sedentary subjects respectively, whereas in the longitudinal (8 year) follow up, the rate of decline increased to 1.46 ml·kg⁻¹·min⁻¹·year⁻¹ in the athletes and 0.48 ml·kg⁻¹·min⁻¹·year⁻¹ in the sedentary subjects. When a variety of mathematical techniques were used to describe the age-associated declines in $\dot{V}O_{2max}$ with age (Rosen et al., 1998), all models indicated that the rates of decline were not different in athletes and sedentary males, and an 8 year longitudinal study (Stathokostas et al., 2004) reported a lower rate of decline in $\dot{V}O_{2max}$ with age in endurance trained master athletes compared to sedentary controls.

A meta analysis of previously published studies (Wilson and Tanaka, 2000) reported age-associated declines in $\dot{V}O_{2max}$ of 0.40 , 0.39 , and 0.46 ml·kg⁻¹·min⁻¹·year⁻¹ in sedentary ($n = 6,231$), active ($n = 5,621$), and endurance trained ($n = 1,967$) male subjects. A similar analysis on published female data (Fitzgerald et al., 1997) reported age-associated declines in $\dot{V}O_{2max}$ of 0.35 , 0.44 , and 0.62 ml·kg⁻¹·min⁻¹·year⁻¹ in sedentary ($n = 2,256$), active ($n = 1,717$), and endurance trained ($n = 911$) female subjects. Cross-sectional studies (Wiswell et al., 2000; 2001) have reported age-associated declines in $\dot{V}O_{2max}$ of 0.36 ml·kg⁻¹·min⁻¹·year⁻¹ in trained female athletes (age range 40 – 70+ years), and 0.67 ml·kg⁻¹·min⁻¹·year⁻¹ in trained male athletes (age range 40 – 70+ years).

Despite the numerous studies reporting the rate of decline in $\dot{V}O_{2max}$ with age, there is a paucity of data describing the age-associated changes in sport-specific power output. For example, Martin et al. (2000) reported that anaerobic power output during cycling decreased by approximately 7.5% per decade following the teenage years, and Seiler et al. (1998) reported approximate declines in power of 3% per decade between ages 24 and 50 years, and 7% between ages 50 and 74 years, in elite rowers.

Given the limited availability of information on changes in power with age, the aims of the current study were: (1) to describe the age-associated decline in $\dot{V}O_{2max}$

in endurance trained cyclists, and (2) model age-associated changes in performance specific power output at both $\dot{V}O_{2\max}$ and at a sub-maximal level. It was hypothesised that physiological function would decline with age and this would be accompanied by similar rates of decline in cycling specific power output.

Methods

Subjects

Fifty six trained subjects, 36 male (age range 17 - 64 yr; body mass 81.4 ± 11.3 kg), and 20 female (16 - 54 yr; body mass 63.0 ± 5.9 kg) gave written informed consent, and completed a medical screening questionnaire. Procedures used in this study have local ethics committee approval.

Selection criteria: All subjects had participated in regular training for endurance (events longer than 1 hour) cycling events for more than 3 years, and at the time of the study, participated in at least two high-intensity training sessions per week for cycling exercise. Throughout the 12 months prior to the study, subjects participated in competitive cycle racing (road racing and / or time trial events) at a local and/or national level. The cohort included current male and female under 21 years NZ road race medallists, former male and female senior NZ road race champions, former male and female professional road cyclists, and current male and female NZ Master and Veteran National road race medallists.

No subjects presented with a medical history which excluded them from a high intensity exercise test, and potential subjects were excluded if they were on any prescribed medication, had experienced illness (e.g. stomach upset, cold or flu like symptoms) in the preceding week, or had a musculo-skeletal injury which affected their normal routine training. Subjects reported to the laboratory in a 4 hour post-prandial state, and were asked to refrain from strenuous exercise for at least 24 hours before the test. Subjects were asked to refrain from drinks containing caffeine for at least 4 hours before testing. Water was provided ad-libitum throughout the test.

Exercise protocol

Subjects performed a 10 minute warm up on an electronically braked cycle ergometer (Lode Excalibur, Lode BV, Groningen, Netherlands) at 60 Watts. Subjects were asked to ride at their normal cadence, and the ergometer was set up to replicate their normal riding position. Subjects performed a single bout of exercise which consisted of a step-wise incremental test to determine their lactate threshold, followed by incremental exercise to volitional exhaustion to determine their $\dot{V}O_{2\max}$.

Initially, subjects performed a continuous incremental test with a minimum of 5 stages, each lasting 5 min. During each stage, a capillary blood sample was collected from a finger tip after 4 min and used for measurement of blood lactate concentration $[La^-]$ (Lactate Pro, Arkray, Kyoto, Japan). The lactate analyser was calibrated with supplied standards prior to each test, and test-retest reliability of this device was within 5% (unpub-

lished observations). Heart rate was continuously monitored throughout the test (S610, Polar Electro, Finland). For all subjects, power output was increased by 30 Watts at the onset of each stage, and starting power output was equivalent to that delivered when the heart rate was approximately 100 beats per minute. When a lactate value >4.5 mmol was achieved, exercise intensity was reduced and subjects were given a 10 min active recovery at a power output approximately 50% below their estimated power at 4mmol $[La^-]$. If by the end of the 5 stages a lactate value of >4.5 mmol was not obtained, subjects performed additional stages (to a maximum of 7), by which all subjects had achieved a lactate >4.5 mmol.

Subjects then performed a continuous incremental test to volitional exhaustion, with 1 minute stages, starting at a power output equivalent to that achieved at a $[La^-]$ of approximately 4.5mmol, during which power output was increased by 20 Watts \cdot min $^{-1}$. Throughout each minute, exhaled air was collected and analysed for oxygen and carbon dioxide (Servomex CO₂ + O₂, 1440, East Sussex, UK) content, and total volume (Dry Gas Meter, Harvard Apparatus, Kent, UK). The gas analysers were calibrated with known concentrations of O₂ and CO₂ prior to each test, and test-retest for this method to determine $\dot{V}O_{2\max}$ was between 5 and 10% (unpublished observations). Maximum oxygen pulse was calculated by dividing $\dot{V}O_{2\max}$ (in ml \cdot kg $^{-1}\cdot$ min $^{-1}$) by HR $_{\max}$ (in beats \cdot min $^{-1}$), thus giving the unit for oxygen pulse as ml \cdot kg $^{-1}\cdot$ beat $^{-1}$.

Statistics

The males and females were compared using a Student *t*-test for unpaired samples of equal variance, and significance level was set at $p < 0.05$. Linear regression was used to describe the relation between age and: (1) $\dot{V}O_{2\max}$, (2) power at $\dot{V}O_{2\max}$, and (3) power at $[La^-]_{4\text{mmol}}$, when normalised for body mass. Linear regression was also used to describe changes in HR $_{\max}$, and maximum oxygen pulse with age. In each case, Pearson's correlation coefficients were used to quantify the degree to which the points clustered about the regression line. Significance level was set at $p < 0.05$.

Results

All subjects completed the lactate threshold and $\dot{V}O_{2\max}$ protocols. All subjects terminated the maximal oxygen uptake test at volitional exhaustion. At test termination, analysis of the expired breath indicated a respiratory exchange ratio exceeding 1.1. The HR $_{\max}$ values are shown in Figure 1, which also shows regression lines used to predict the maximum heart rate based on age (Tanaka et al., 2001). Deviation of measured HR $_{\max}$ values from the predictions were 3.1 (± 6.9)% and 2.8 (± 5.7)% for females, and 1.9 (± 6.4)% and 2.3 (± 5.4)% for males using the 220-age and 208-0.7age equations respectively. Table 1 shows the mean (\pm sd) characteristics of the group as a single cohort, and shows the descriptive statistics for the males and females when separated into sub-groups. Males were significantly heavier than females (males: 81.4 ± 11.3 kg vs. females: 63.1 ± 5.9 kg, $p < 0.01$), and could

Table 1. Descriptive characteristics of all subjects, and for male and female sub-groups. Values are means (\pm SD).

	Males (n=36)	Females (n=20)	All (n=56)
Age (years)	42.1 (10.7)	37.7 (11.9)	40.5 (11.2)
Mass (kg)	81.4 (11.3)	63.1 (5.9) **	74.8 (13.1)
HR _{max} (beat·min ⁻¹)	175 (11)	177 (10)	176 (11)
VO _{2max} (L·min ⁻¹)	4.5 (.6)	3.2 (.5) **	4.1 (.41)
VO _{2max} (ml·kg ⁻¹ ·min ⁻¹)	56.2 (9.6)	51.3 (8.6)	54.5 (9.5)
O _{2max} (ml·kg ⁻¹ ·beat ⁻¹)	.321 (.056)	.289 (.041) *	.310 (.053)
Power at VO _{2max} (Watts·kg ⁻¹)	4.4 (.7)	4.2 (.6)	4.4 (.7)
Power at [La] _{4mmol}	3.1 (.6)	2.9 (.5)	3.0 (.6)

* and ** denote $p < 0.05$ and $p < 0.01$ using a Student *t*-test for unpaired samples of equal variance.

use more oxygen at VO_{2max} (males: 4.5 ± 0.6 L·min⁻¹ vs. females: 3.2 ± 0.5 L·min⁻¹, $p < 0.01$). Male and female sub-groups were not different in terms of age (males: 42.1 ± 10.7 years vs. females: 37.7 ± 11.9 years, $p > 0.05$) or HR_{max} (males: 175 ± 11.2 beats·min⁻¹ vs. females: 177 ± 9.7 beats·min⁻¹, $p > 0.05$). When normalised for body mass, males were not different compared to females for VO_{2max} when measured in ml·kg⁻¹·min⁻¹, power at VO_{2max}, or sub-maximal power at [La]_{4mmol}.

Pearson's correlation coefficients for male and female subjects, and where these were significant, the accompanying linear regression equations, are shown in Table 2. There was no significant correlation between age and HR_{max} for the females, and no significant correlation between age and maximum oxygen pulse for the females. The rate of decline in VO_{2max} with age was 0.65 ml·kg⁻¹·min⁻¹·year⁻¹ and 0.39 ml·kg⁻¹·min⁻¹·year⁻¹ for males and females respectively.

Power at VO_{2max} showed a negative correlation with age for males, where power decreased by 0.048 Watts·kg⁻¹·year⁻¹ (see Figure 1). There was no apparent age-associated decline in power at VO_{2max} for the females. Power at [La]_{4mmol} decreased at a rate of 0.044 Watts·kg⁻¹·year⁻¹ in males, and at $.019$ Watts·kg⁻¹·year⁻¹ in females (see Figure 2).

Discussion

The power output at VO_{2max}, when normalised for body mass, is an index which considers both the power output of recruited skeletal muscle and the maximal aerobic capacity. Therefore, for cyclists, this is a measure of the

functional capacity of skeletal muscle at VO_{2max}. This study reported an age-associated decrease in relative power at VO_{2max} in males but not in females, thus suggesting that the female cohort maintained peak aerobic power throughout the age range. Although speculative, this may suggest that peak aerobic power may be maintained if there are no age-associated changes in maximal oxygen pulse and HR_{max}, both findings consistent with current data.

A sub-maximal sustainable power (for example the power output at the onset of the accumulation of blood lactate) may represent a measure of muscle performance potentially unaffected by the age-associated declines in maximal cardiac output, reduced peak muscle blood flow, and decreases in oxygen extraction. The current study reported that power output at [La]_{4mmol}, when normalised for body mass, declined with age in both males and females. The near parallel age-associated declines in peak aerobic power and power at [La]_{4mmol} in males may suggest a common mechanism, for example, a decrease in peripheral oxygen uptake (indirectly supported by the age-associated decline in maximum oxygen pulse), or a decrease in cardiac output (indirectly supported by the age-associated decline in HR_{max}). The apparent divergence of the regression lines for the female data should be treated with caution, however, the age-associated decline in power at [La]_{4mmol} in the females may suggest age-associated changes in the kinetics of lactate production and/or clearance. Further work on the mechanisms responsible for gender specific changes in sub-maximal and peak aerobic power is required.

An age-associated decrease in muscle mass and/or changes in the expression of the myosin heavy chain

Table 2. Pearson's correlation coefficients for AGE in years, versus selected variables for males and females.

	Males (n = 36) Age range: 17-64 years	Females (n = 20) Age range: 16-54 years
HR _{max} (beat·min ⁻¹)	-.36 * y = -0.35x + 190	-.30
VO _{2max} (L·min ⁻¹)	-.63 ** y = -0.035x + 5.97	-.55 *
VO _{2max} (ml·kg ⁻¹ ·min ⁻¹)	-.72 ** y = -0.65x + 83.4	-.54 *
O _{2max} (ml·kg ⁻¹ ·beat ⁻¹)	-.57 ** y = -0.003x + .45	-.42
Power at VO _{2max} (Watts·kg ⁻¹)	-.72 ** y = -0.048x + 6.45	-.10
Power at [La] _{4mmol}	-.76 ** y = -0.044x + 4.98	-.53 * y = -0.019x + 3.47

* and ** denote $p < 0.05$ and $p < 0.01$. For significant associations, the regression equation is shown, where y is the variable and x is age.

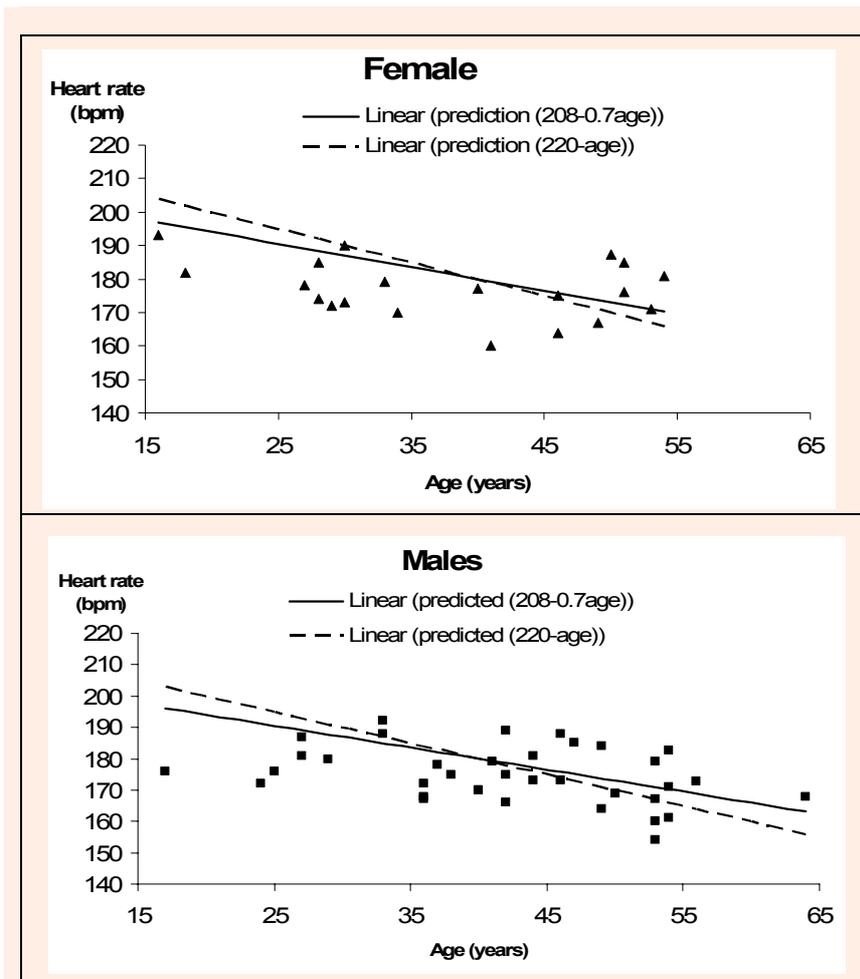


Figure 1. Maximum heart rate for female (upper) and male (lower) endurance trained cyclists. Reference linear regression lines for 220-age (dashed line) and 208-0.7age (solid line) are shown.

(MHC) isoforms in the recruited motor unit pool (Doherty, 2003; Goldspink, 2005) may contribute to the decline in power with age. Farina et al. (2007) reported a correlation between % MHC type 1 isoform with power at the lactate threshold and at $\dot{V}O_{2\max}$ in trained subjects (aged 25 ± 4 years, $\dot{V}O_{2\max}$ $52.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), whereas Mattern et al. (2003) found no differences between young and old subjects in the MHC isoforms expressed in skeletal muscle. However, Mattern et al. (2003) did show that age and MHC type 1 combined to account for 58% of the variance in power output at the maximum sustainable stable blood lactate concentration when expressed as a % of maximal aerobic capacity. A lower power output at the maximum sustainable stable blood lactate concentration in older, trained endurance athletes, has been reported (Mattern et al., 2003) where subjects ($n = 9$, $\dot{V}O_{2\max}$ $67.7 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) aged 25 years produced $3.5 \text{ W}\cdot\text{kg}^{-1}$, and subjects ($n = 9$, $\dot{V}O_{2\max}$ $47.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) aged 65 years produced $2.2 \text{ W}\cdot\text{kg}^{-1}$. The present study predicted power at $[\text{La}]_{4\text{mmol}}$ when aged 25 to be $3.41 \text{ W}\cdot\text{kg}^{-1}$, and when aged 65 to be $2.41 \text{ W}\cdot\text{kg}^{-1}$.

There is certainly some variability in the age-associated decline in $\dot{V}O_{2\max}$ in trained subjects, whether approximate rates are derived from longitudinal or cross-sectional studies, and both experimental designs have

limitations. We have included all ages into gender-specific regression models - consistent with previous reports (Fitzgerald et al, 1997; Wilson and Tanaka, 2000), and acknowledge that a selection bias, whereby the oldest athletes are selected from a decreasing pool of available subjects (Katzel et al., 2001) exists in the current data.

The cross-sectional age-associated decrease in $\dot{V}O_{2\max}$ reported in the present study was lower than that reported by Tanaka et al. (1997) for endurance athletes, but higher than that reported by Katzel et al. (2001) in their cross-sectional study. Katzel et al. (2001) reported higher values for the rate of decline in their longitudinal study, and Marcell et al. (2003) reported a rate of $1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\cdot\text{year}^{-1}$ in trained men and women aged 40 – 60 years over an approximate 6 year period. It has been reported that longitudinal studies generally report higher rates of decline in $\dot{V}O_{2\max}$ with age (Eskurza et al., 2002; Katzel et al., 2001), and Stathokostas et al. (2004) reported that the longitudinal rate of decline in men was higher than that reported in a cross-section of their original sample population. Pimental et al. (2003), when stratifying for age, reported that endurance trained subjects showed declines of $0.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\cdot\text{year}^{-1}$ when aged 20-50 years, and this increased to $0.89 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\cdot\text{year}^{-1}$ when aged 50-75 years. We acknowledge that the age range used in the current study does not include 'old' athletes, and the

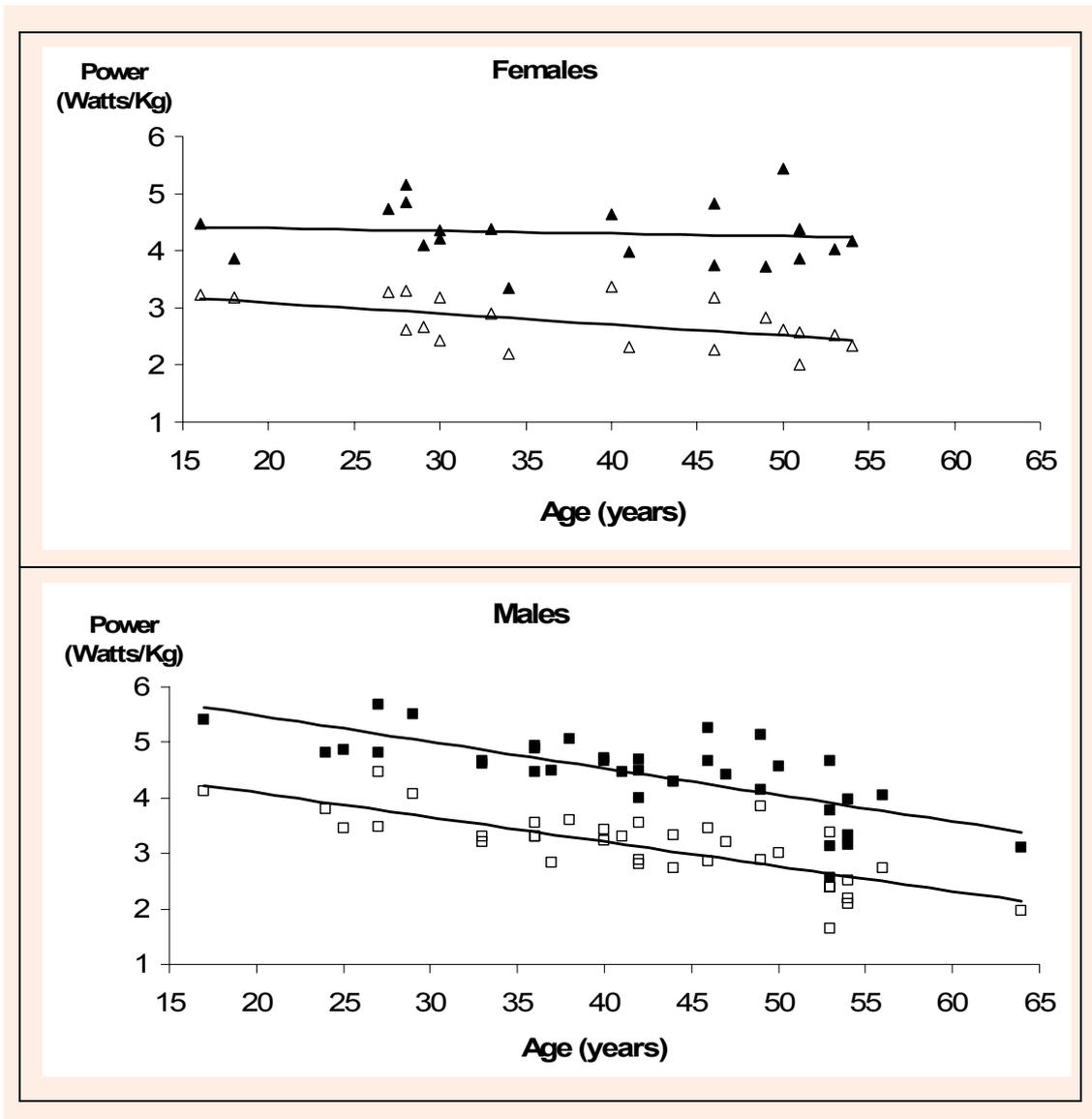


Figure 2. Decreases in power at $\dot{V}O_{2max}$ (closed symbols) and at $[La^-]_{4mmol}$ (open symbols) for female (upper) and male (lower) endurance trained cyclists.

limited sample size prevents stratification by age.

An age-associated decline in HR_{max} has been previously reported (Fitzgerald et al., 1997; Tanaka et al., 1997; Pimentel et al., 2003; Wilson and Tanaka, 2000), although in the present study there was no obvious trend in HR_{max} for females and only a weak age-associated decrease in male HR_{max} . The lack of an age-associated decline in HR_{max} in the present study may be a result of fatigue, given that the $\dot{V}O_{2max}$ protocol was at the end of the exercise period. However, we were satisfied that all subjects reached $\dot{V}O_{2max}$ using this protocol. Decreases (Rogers et al., 1990; Fleg et al., 2005) or no changes (Ogawa et al., 1992; Stathokostas et al., 2004) in maximal oxygen pulse with age have been previously reported without consistent directional changes in HR_{max} .

Differences in the rate at which $\dot{V}O_{2max}$ declined with advancing age has been attributed to gender-specific changes in components of the cardiovascular system (Weiss et al., 2006). The current study reported no corre-

lation between age and maximum oxygen pulse in females, but a significant negative correlation in males, thus further supporting gender-specific differences in the rate of decline in cardiovascular performance. However, it should be noted that the small sample size and lack of old (60 years +) subjects (particularly females) used in the current study, make such statements speculative.

Linear equations have been used to express the maximal aerobic capacity of skeletal muscle as a function of muscle mass where Proctor and Joyner (1997) reported the same gradient for young compared to old muscle. This indicated that for a given change in muscle mass, the proportional change in aerobic capacity of young and old muscle was the same. The authors concluded that it was unlikely that skeletal muscle oxidative capacity or capillarisation was responsible for the age-associated reduction in aerobic capacity per Kilogramme muscle in trained older subjects (Proctor and Joyner, 1997). Mattern et al. (2003) also reported no differences in the citrate synthase activity of skeletal muscle samples taken from young,

middle aged, and older trained endurance athletes, thus supporting the conclusions of Proctor and Joyner (1997). However, the ordinary least-squares model used by Rosen et al. (1998) suggested that 35% of the age-associated decline in $\dot{V}O_{2\max}$ was due to a loss of fat free mass, accounting for approximately $8 \text{ ml min}^{-1} \text{ year}^{-1}$.

In the current study, we chose to use the same testing protocol for all ages and both genders - a decision based on the selection of the subjects (all well trained and in regular competition), and following medical screening. Variations in training regimes are likely to occur with aging (Spirduso et al., 2005), although subjects in the current study all participated in at least 2 high intensity training sessions each week – in most cases this was in addition to a weekly competitive event. Our experiences in recruiting trained athletes across a wide age-range is that older subjects do not have a lower training volume in comparison to their younger counterparts, however, we acknowledge that medical conditions which impact on the ability to perform high-intensity exercise may define the appropriate testing protocols suitable for older subjects (Huggett et al., 2005).

Conclusion

This cross-sectional study reports an age-associated decline in $\dot{V}O_{2\max}$ in both males and females, consistent with previous reports. The concomitant decline in maximum oxygen pulse with minimal trend in HR_{\max} in males has been rarely reported in previous literature. The present study uniquely reports an age-associated decline in power at $\dot{V}O_{2\max}$ in males, and at a $[La]_{4\text{mmol}}$ sub-maximal level for both males and females.

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Key points

- $\dot{V}O_{2\max}$ decreased with age by $0.65 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\cdot\text{year}^{-1}$ in male, and by $0.39 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\cdot\text{year}^{-1}$ in female endurance trained cyclists.
- Power at $\dot{V}O_{2\max}$ decreased with age by $0.048 \text{ Watts}\cdot\text{kg}^{-1}\cdot\text{year}^{-1}$ in male endurance trained cyclists.
- Sub-maximal power at a blood lactate concentration of $4 \text{ mmol}\cdot\text{L}^{-1}$ decreased by $0.044 \text{ Watts}\cdot\text{kg}^{-1}\cdot\text{year}^{-1}$ in male, and by $0.019 \text{ Watts}\cdot\text{kg}^{-1}\cdot\text{year}^{-1}$ in female endurance trained cyclists.

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