

Isometric strength training lowers the O₂ cost of cycling during moderate-intensity exercise

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Abstract The effect of maximal voluntary isometric strength training of knee extensor muscles on pulmonary V'O₂ on-kinetics, the O₂ cost of cycling and peak oxygen uptake (V'O_{2peak}) in humans was studied. Seven healthy males (mean ± SD, age 22.3 ± 2.0 years, body weight 75.0 ± 9.2 kg, V'O_{2peak} 49.5 ± 3.8 ml kg⁻¹ min⁻¹) performed maximal isometric strength training lasting 7 weeks (4 sessions per week). Force during maximal voluntary contraction (MVC) increased by 15 % ($P < 0.001$) after 1 week of training, and by 19 % ($P < 0.001$) after 7 weeks of training. This increase in MVC was accompanied by no significant changes in the time constant of the V'O₂ on-kinetics during 6 min of moderate and heavy cycling intensities. Strength training resulted in a significant decrease (by ~7 %; $P < 0.02$) in the amplitude of the fundamental component of the V'O₂ on-kinetics, and therefore in a lower O₂ cost of cycling during moderate

cycling intensity. The amplitude of the slow component of V'O₂ on-kinetics during heavy cycling intensity did not change with training. Training had no effect on the V'O_{2peak}, whereas the maximal power output reached at V'O_{2peak} was slightly but significantly increased ($P < 0.05$). Isometric strength training rapidly (i.e., after 1 week) decreases the O₂ cost of cycling during moderate-intensity exercise, whereas it does not affect the amplitude of the slow component of the V'O₂ on-kinetics during heavy-intensity exercise. Isometric strength training can have beneficial effects on performance during endurance events.

Keywords Exercise · Oxygen uptake kinetics · Muscle force · Strength training

Introduction

Muscle force is an important factor in determining human locomotory performance and physical activity (e.g., Sargeant and Jones 1995; Zoladz et al. 2000; Sargeant and de Haan 2006). It is well known that the force determined during a maximal voluntary contraction (MVC) can be significantly increased following a few weeks of strength training (Jones and Rutherford 1987). In athletes performing short-term supramaximal events, such as power lifting, throwing, sprinting, etc., strength training usually focuses on improving maximal muscle strength (Zatsiorsky 2003). In recent years, however, strength training in various forms has been widely used by several groups of subjects, including patients (Ada et al. 2006; Brooks et al. 2006; Hoff et al. 2007), the elderly (Macaluso and De Vito 2004; Narici et al. 2004), as well as the endurance athletes (Paavolainen et al. 1999; Millet et al. 2002; Støren et al. 2008).

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It is well known that both maximal oxygen uptake ($\dot{V}O_{2\max}$) and the endurance capacity of strength trained athletes are substantially lower than in endurance athletes (for an overview see, e.g., Astrand and Rodahl 1986). However, one should distinguish the effects on muscle endurance capacity and oxidative metabolism deriving from several years of “pure” strength training from those deriving from a relatively short period (several weeks) of strength training. It has indeed been reported that strength training might also have some beneficial effects on endurance performance. A series of recent studies (Johnston et al. 1997; Paavolainen et al. 1999; Saunders et al. 2006; Støren et al. 2008; Taipale et al. 2010) reported an improvement of running economy of distance runners and an enhancement of endurance performance after strength training. Similarly, it was recently reported that, in competitive cyclists, strength training improves the economy of cycling and increases time to exhaustion at maximal aerobic power (Sunde et al. 2010). Millet et al. (2002) evaluated the effects of concurrent endurance training and strength training on running economy and oxygen uptake $\dot{V}O_2$ kinetics. According to these authors, in elite triathletes resistance training in combination with endurance training resulted in an improvement of maximal strength and running economy, with no significant effects on the $\dot{V}O_2$ kinetics pattern in heavy exercise.

It seems to be relevant to ask whether strength training increases muscle efficiency to the same degree during moderate- and heavy-intensity exercise. In other words, does strength training increase mechanical efficiency to the same degree in type I muscle fibres, recruited predominantly during moderate-intensity exercise, and in type II muscle fibres, recruited to a greater extent during heavy-intensity exercise? (for an overview of the concept of different muscle recruitment, see, e.g., Sargeant and Jones 1995). Moreover, what are the specific effects of “pure” (i.e., not associated with endurance training) isometric strength training on parameters of pulmonary $\dot{V}O_2$ kinetics? To our best knowledge, no data have been published so far concerning this aspect. However, the effects of various kinds of strength training on $\dot{V}O_{2\max}$ have been investigated before, although with controversial results (Hickson et al. 1980, 1988; Frontera et al. 1990; Goreham et al. 1999; Paavolainen et al. 1999; Støren et al. 2008), surprisingly little is known concerning the effect of isometric strength training on the $\dot{V}O_{2\max}$ as well as on the rate of oxygen uptake at the onset of exercise in humans ($\dot{V}O_2$ on-kinetics) (for overview see Rossiter 2011) or on the oxygen cost of cycling.

We have hypothesized that “pure” isometric strength training would decrease oxygen cost of cycling. Assuming that the strength training-induced improvement of endurance

exercise performance is caused by enhanced mechanical efficiency of type I muscle fibres, we have hypothesized that the strength training-induced decrease of the amplitude of the fundamental $\dot{V}O_2$ on-kinetics will be more pronounced during moderate than during heavy-intensity cycling. Moreover, we have also aimed to determine the effect of 7-week “pure” isometric strength training on the magnitude of the slow component of the $\dot{V}O_2$ on-kinetics in young healthy men.

Research design and methods

Subjects

Seven healthy, physically active males (mean \pm SD: age 22.3 ± 2.0 years, body weight 75.0 ± 9.2 kg, height 180.2 ± 7.4 cm, body fat 13.5 ± 2.4 % of BW) participated in this study. The $\dot{V}O_{2\max}$ determined during the incremental exercise test (see the following section) of the subjects was 49.5 ± 3.8 ml $\text{kg}^{-1} \text{min}^{-1}$. Basic blood variables, taken at rest before training, such as haematocrit (Ht), haemoglobin (Hb), erythrocyte (Er), leucocyte (Leu), sodium (Na^+), potassium (K^+) and creatinine (Cr) concentrations were in the normal range (Table 1). Abnormalities in basic blood variables were considered as exclusion criteria. All subjects were experienced in laboratory exercise tests. The study protocol was approved by the Local Ethical Committee and was performed in accordance with the Declaration of Helsinki.

Study design

In the pre-training phase, all laboratory measurements and exercise tests were completed as follows (for details, see Fig. 1). After routine medical screening and familiarization with the exercise testing procedure, the participants performed the incremental exercise test to exhaustion on the cycle ergometer, followed by at least a 2-day rest. Then, the MVC was measured and after 1 day of rest, the constant power output tests were conducted. During the 7-week long isometric strength training program, subjects underwent 28 training sessions (4 times a week). The isometric strength exercise sessions normally took place on Monday, Tuesday, Thursday and Friday except for the 2nd week of training when the strength exercise sessions took place on Thursday, Friday, Saturday and Sunday, since during this week the constant power output tests were performed after the 1st week of training (see Fig. 1). At the end of 1st, 3rd, 5th and 7th week, the MVC testing was performed at the beginning of Friday’s exercise session. The post-training phase schedule was similar to the pre-training phase schedule (see Fig. 1).

Table 1 Basic blood variables

	Ht (vol%)	Hb (g 100 ml ⁻¹)	E (×10 ⁶ mm ⁻³)	Leu (×10 ³ mm ⁻³)	[Na ⁺] (mmol l ⁻¹)	[K ⁺] (mmol l ⁻¹)	[Cr] (μmol l ⁻¹)
Min.	41.4	13.8	4.74	4.69	141.5	4.17	92.0
Max.	45.9	15.6	5.05	8.21	145.0	4.92	109.7
Mean ± SD	43.06 ± 1.62	14.60 ± 0.67	4.88 ± 0.11	5.53 ± 1.23	142.8 ± 1.2	4.47 ± 0.31	98.27 ± 6.31

Data are given as minimal (Min.), maximal (Max.), mean ± SD values of haematocrit (Ht), haemoglobin (Hb), erythrocyte (Er), leucocyte (Leu), sodium [Na⁺], potassium [K⁺] and creatinine [Cr] concentrations in venous blood samples

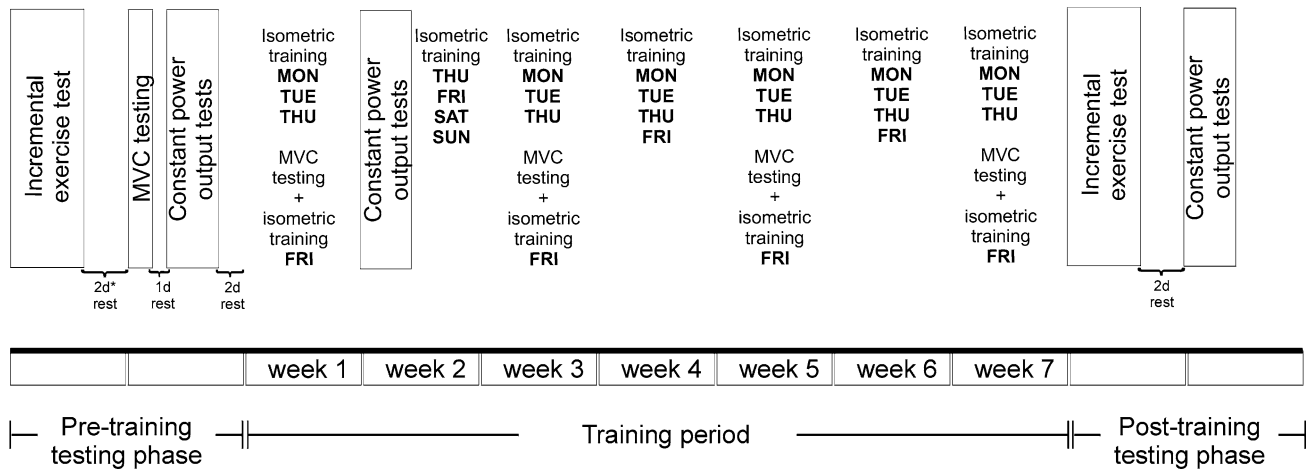


Fig. 1 Scheme of the study design

Exercise protocols

Force during maximal voluntary contraction

The measurement of force during MVC of the knee extensor muscle was performed on a prototype device equipped with a strain gauge, according to Edwards et al. (1977). The subject sat on the chair with their hips and knees fixed at 90° of flexion. The pelvis and the thigh were firmly stabilized by restraining straps to minimize unwanted movement. Another inextensible strap was placed around the ankle above the medial malleolus and was attached to the strain gauge located at the back of the chair. The signal from the strain gauge was amplified and sent to the computer. Purpose-made software allowed continuous monitoring and storing of the signal. The testing session began with a 10-min warm-up consisting of low-intensity cycling at 60 W and individually chosen lower extremity stretching exercises.

After the subjects were seated and positioned in the testing chair, they performed (with each leg) 3–4 short (1–3 s duration) submaximal isometric contractions, with a 30-s interval between each contraction and followed by 3 min of recovery. MVC of the knee extensors (for which

the subjects were asked to push as forcefully as possible for about 3 s against the strap) was determined for both the right and the left leg with a 2-min break in between. This procedure was repeated three times and the MVC was calculated as the average force for the consecutive measurements. The length of the lever arm during the MVC measurement was kept constant before and after the training program.

Isometric strength training

The subjects performed, 4 times per week, maximal isometric strength training sessions over a total period of 7 weeks (for overview, see Fig. 1). During each session the subjects performed 10 maximal voluntary contractions lasting 5 s each, separated by 30 s of rest, involving both the right and the left leg. Training started with 5 maximal contractions performed by the right leg, followed by 5 maximal contractions performed by the left leg. After a short pause, lasting about 3 min, the subjects performed once again 5 maximal contractions involving each leg. Generated forces were displayed on a monitor situated in front of the subjects. During training, individual values of MVC determined before training were displayed on the

monitor in order to motivate the subjects to achieve the maximal forces during each contraction.

The incremental exercise tests

All subjects performed the incremental exercise test until exhaustion on a cycloergometer (Ergoline 800 S, Germany) to determine $\dot{V}O_{2\max}$ and LT. The test started with 6 min of rest (subject seated on the ergometer) followed by a gradual increase of power output, by 30 W every 3 min. The pedalling rate was maintained at 60 rev min^{-1} . The tests were stopped when the subjects could no longer maintain the required pedalling rate or power output despite vigorous encouragement by the operators (see Zoladz et al. 1998). The incremental tests were performed again 3 days after finishing the training program, about 1 week before the constant power output tests (for overview, see Fig. 1).

The constant power output tests

The subjects performed two constant power output exercise tests (each for 6 min duration). The tests started with 6 min of rest (subject seated on the cycloergometer), followed by 6 min of cycling at 60 rev min^{-1} and by 6 min of passive recovery. The power output during the first constant power output test (moderate intensity) was the same for all of the subjects, and it amounted to 90 W. In all subjects this power output was below LT. The second constant power output test (heavy intensity) was performed at the power output corresponding to the 50 % difference between the power outputs at LT and at $\dot{V}O_{2\text{peak}}$ (50 % Δ) (for a review of this concept, see, e.g., Roston et al. 1987; Barstow et al. 1996).

The tests were performed in the morning hours after a light breakfast. During one visit to the laboratory the subjects performed two tests, starting with the moderate exercise intensity test followed about 1 h later by the heavy-intensity test. In order to improve the signal-to-noise ratio of $\dot{V}O_2$ breath-by-breath measurements and to facilitate curve fitting, each of these tests was performed three times, on separate days. Measurements were repeated after 1 week of training and after finishing the strength training program (i.e., after 7 weeks) (for overview, see Fig. 1).

Measurements

Gas exchange variables

Both during the incremental exercise test and during the constant power output tests gas exchange variables were measured continuously breath-by-breath using the Oxycon

Champion Jaeger (Germany), starting from the sixth minute prior to the exercise. Before and after each test was finished, the gas analysers were calibrated with certificated calibration gases, as previously described by Zoladz et al. (1995). During the incremental exercise test a medium-sized Rudolph mask was used (death space equal to 90 ml), whereas during the constant power output tests the subjects were breathing via a small-sized mouthpiece (death space equal to 40 ml).

Heart rate

The heart rate was monitored by the Hellige SMS 181 unit (Germany) and recorded beat-by-beat using S810 Polar Electro (Finland).

Blood sampling and analyses

Venous blood samples were taken using an Abbot Int-Catheter, from Ireland (18G/1.2 \times 45 mm), inserted into the antecubital vein about 30 min prior to the onset of the exercise. The catheter was connected with the extension set (a tube 10 cm in length), using a “T” Adapter SL Abbot (Ireland). Immediately before taking the blood sample for appropriate analysis, 1 ml of blood was taken and discarded, in order to eliminate the blood originally residing in the catheter and the T-set. Blood samples (1 ml each) were taken 1 min before starting the first bout of the incremental exercise test and at the end of each minute of work (during the last 15 s).

Blood PO_2 and PCO_2 , as well as $[H^+]_b$ were determined immediately using a Ciba-Corning 238 analyser (England). Blood bicarbonate concentration ($[HCO_3^-]_b$) and base excess (BE) were calculated by the same unit. Plasma lactate concentration ($[La]_{pl}$) was measured using the automatic analyser Ektachem XR 700, Kodak (USA). Serum sodium (Na^+) and potassium (K^+) concentrations were determined using a flame photometer, Ciba-Corning Model 480 (England). Blood creatinine level was determined by the kinetics method, based on its reaction with picric acid, using the automatic analyser Express 550 CBI (England). Blood haemoglobin concentration (Hb), haematocrit (Ht), erythrocyte count (Er) and leucocyte count (Leu) were determined using an automatic haematological analyser (Baker 9000, USA).

Data analysis

The lactate threshold (LT), as previously described (see Zoladz et al. 1995), was identified as the highest power output above which $[La]_{pl}$ showed a sustained increase of $>0.5 \text{ mmol l}^{-1} \text{ step}^{-1}$.

Modelling and analysis of $\dot{V}O_2$ on-kinetics

Raw breath-by-breath data were pre-processed to remove occasional, artifactual values resulting from, e.g., swallowing or coughing, by means of a procedure similar to that described by Lamarra et al. (1987). $\dot{V}O_2$ responses from the repeated bouts of exercises were time-aligned to the onset of the exercise and interpolated on a second-by-second basis. The resulting data were then, for each subject, superimposed, averaged over the transitions and averaged over consecutive 10 s time intervals (Rossiter et al. 2001), in order to reduce the noise. $t = 0$ was set at the onset of exercise. The cardio-dynamic phase of the response (Whipp 1996) was not modelled. Correspondingly, data from the first 20 s of the on-transients were ignored.

For moderate-intensity exercises, a mono-exponential model

$$\dot{V}O_2(t) = \dot{V}O_2(0) + A_1 \times (1 - e^{-(t-TD_1)/\tau_1})$$

was used.

The on-transient $\dot{V}O_2$ responses for heavy-intensity exercises were modelled as

$$\begin{aligned} \dot{V}O_2(t) = & \dot{V}O_2(0) + A_1 \times (1 - e^{-(t-TD_1)/\tau_1}) \\ & + A_2 \times (1 - e^{-(t-TD_2)/\tau_2}) \end{aligned}$$

(cf., e.g., Borrani et al. 2001; Zoladz et al. 2005). In these equations, $\dot{V}O_2(0)$ is the baseline value, calculated as an average over the 3 min before the onset of the exercise. The first exponential term starts at $t = TD_1$ and the second one at $t = TD_2 > TD_1$.

In all cases the relevant models were fitted to the corresponding data by the least-squares method, using the *Non-linear Estimation* module of STATISTICA, version 6. Several starting points for the iterative minimization procedure were tried in order to determine the best fit. (cf., e.g., Borrani et al. 2001; Zoladz et al. 2005). The initial data smoothing reveals the shape of the estimated curve and makes it easier to properly set starting points for the iterative fitting procedures, which is crucial for the quality of the estimates. It should be noted, however, that, according to our experience, provided the starting points are set properly, fitting to original non-smoothed data gives practically the same results, at least for the time constant τ_1 and the amplitudes, which were the main parameters of interest in this study.

The essential amplitude of the slow component of the $\dot{V}O_2$ on-kinetics was assessed by two approaches: (1) as the difference between the $\dot{V}O_2$ values during the last 30 s of exercise and the asymptotic value of the fundamental component, (2) as the difference between the $\dot{V}O_2$ measured in the sixth minute minus the $\dot{V}O_2$ measured in the third minute of cycling.

$\dot{V}O_{2peak}$

In the present study, we have observed a $\dot{V}O_2$ plateau at the maximal power output (considered as the only criterion of $\dot{V}O_{2max}$, see Poole et al. 2008) in most but not in all subjects. Therefore, accordingly with the notion of the study by Poole et al. (2008), the highest $\dot{V}O_2$ reached during the incremental exercise test was considered as $\dot{V}O_{2peak}$.

Statistical analyses

Data are presented as mean \pm SD. Although we have performed the Friedman test at a preliminary stage of our analysis, we mainly report the results of the final comparisons between weeks 0, 1 and 7. We do so because pairwise comparisons between weeks not only reveal the pattern of the change but also detect consistent changes, as the Friedman test does. The significant difference between τ_1 (and other variables) measured before and after the training was tested by the matched-pairs Wilcoxon signed-ranks test; for each subject the change $\Delta\tau_1$ of τ_1 was computed and the Wilcoxon signed-ranks test was used to check whether the differences were significantly different from zero. We report the P values obtained in multiple pairwise comparisons in the Fisherian spirit as a measure of evidence rather than in the strict decision-theoretic sense. Exact (i.e., non-asymptotic), two-sided P values were computed. A non-parametric test with the exact P values was used because of the small sample size (7 subjects) (for details, see Zoladz et al. 2005). With a $n = 7$ it is indeed impossible to reliably verify the assumption of normality needed for the application of standard parametric tests. For the non-parametric Wilcoxon signed-ranks test, one only needs to assume the symmetry of the distribution with respect to its median, which is much weaker than the assumption of normality and, in our opinion is acceptable for the present study.

Results

Force during maximal voluntary contraction (MVC)

Values of force during MVC are presented in Table 2. After the first week of training force values during MVC increased significantly by about 15 % in relation to the pre-training level (Table 2). After 3 weeks of training force values during MVC further increased, and thereafter they remained relatively stable up to the 7th week of training, when they were about 19 % higher than before training.

Table 2 Values of force during maximal voluntary contraction (MVC) obtained before and during the weeks of training

	Pre-training level (week)	Weeks of training			
	0	1	3	5	7
MVC (N)	675 ± 54	776 ± 79*	811 ± 60* [#]	813 ± 85* [#]	806 ± 83*

N Newton

* Significantly different from pre-training level ($P < 0.02$)

[#] Significantly different from the value at week 1 ($P < 0.03$) as determined by the matched-pairs Wilcoxon signed rank test. Consistent pattern of the changes in MVC was also detected by the preliminary Friedman test ($P < 0.001$)

The incremental exercise tests

Values of maximal power output (PO_{\max}), selected cardio-respiratory variables and basic variables of plasma acid–base status obtained at exhaustion during the incremental exercise test performed before and after strength training are presented in Table 3.

Strength training significantly increased PO_{\max} (265 ± 23 vs. 255 ± 25 W after and before training program, respectively, $P < 0.05$), RER (1.15 ± 0.05 vs. 1.12 ± 0.04 , $P < 0.05$), and V'_E (120 ± 22 vs. 107 ± 17 min^{-1} , $P = 0.06$). Conversely, PO_{LT} , $V'O_{2\text{peak}}$, $V'CO_2$, HR, $[La^-]_{pl}$, $[H^+]_b$, and $[HCO_3^-]_b$ were not significantly different after the training period (see Table 3). HR, RER, and $[La^-]_{pl}$ values

Table 3 Values of maximal power output, selected cardio-respiratory variables and basic variables of plasma acid–base status obtained at exhaustion during the incremental exercise test performed before and after strength training

Variables	Before training	After training
$V'O_{2\text{peak}}$ (ml min^{-1})	3,700 ± 423	3,690 ± 499
V'_E (l min^{-1})	107 ± 17	120 ± 22 [#]
$V'CO_2$ (ml min^{-1})	4,119 ± 430	4,222 ± 529
RER	1.12 ± 0.04	1.15 ± 0.05*
HR (bt min^{-1})	191 ± 4	189 ± 10
$[La^-]_{pl}$ (mmol l^{-1})	9.80 ± 1.25	10.40 ± 1.48
$[H^+]_b$ (nmol l^{-1})	53.79 ± 5.11	53.56 ± 3.54
$[HCO_3^-]_b$ (mmol l^{-1})	19.96 ± 1.15	20.11 ± 1.02
PO_{LT} (W)	116 ± 32	129 ± 38
PO_{\max} (W)	255 ± 25	265 ± 23*

Data are given as mean ± SD

Peak oxygen uptake ($V'O_{2\text{peak}}$), minute ventilation (V'_E), carbon dioxide production ($V'CO_2$), respiratory exchange ratio (RER), heart rate (HR), plasma lactate concentration ($[La^-]_{pl}$), blood hydrogen ion concentration ($[H^+]_b$) and blood bicarbonate concentration ($[HCO_3^-]_b$), power output reached at the lactate threshold (PO_{LT}), power output reached at $V'O_{2\text{peak}}$ obtained during the incremental exercise test (PO_{\max}) performed before and after strength training

* Significantly different ($P < 0.05$) from pre-training value

[#] Non-significant tendency to higher post-training value ($P = 0.06$) as determined by the matched-pairs Wilcoxon signed rank test

confirm that both before and after training program, the incremental exercises were maximal.

The constant power output exercise tests

Values of the main gas exchange and cardiovascular variables obtained at steady-state during constant power output exercise <LT and in the final stage of exercise >LT, carried out before and after 1 and 7 weeks of strength training, are presented in Table 4.

During moderate-intensity exercise (<LT), $V'O_2$ was lower after 1 and 7 weeks of training compared to before training; no significant difference was observed between 1 and 7 weeks. $V'CO_2$ was slightly lower after 7 weeks of training compared to pre-training level (Table 4).

During heavy-intensity exercise (>LT) $V'O_2$ was lower, with respect to before training, after 1 week of training, but not after 7 weeks. $V'CO_2$ was lower after 1 and 7 weeks of training versus before training. HR was lower after 7 weeks of training versus before training (Table 4).

$V'O_2$ on-kinetics

$V'O_2$ on-kinetics of a typical subject, determined during moderate-intensity exercise before and after strength training are presented in Fig. 2a. The lower amplitudes of the response after 1 and 7 weeks of training are evident.

Values of $V'O_2$ on-kinetics parameters for moderate-intensity exercise are presented in Table 5. A_1 and EE $V'O_2$ were lower after 1 and 7 weeks of training versus before training; no significant differences were observed between the values obtained after 1 and 7 weeks of training. Changes in A_1 (ΔA_1) following training are shown in Fig. 3a. No significant effects of strength training were described for BL $V'O_2$, TD_1 , τ_1 and MRT. As expected, no slow component of the $V'O_2$ on-kinetics occurred during this moderate intensity bout of cycling exercise.

$V'O_2$ on-kinetics of a typical subject, determined during heavy-intensity exercise before and after strength training are presented in Fig. 2b. No significant effects of training on the time-courses of the variable can be seen. Values of $V'O_2$ on-kinetics parameters for heavy-intensity exercise are presented in Table 6. No significant effects of training were

Table 4 Values of the main gas exchange and cardiovascular variables obtained at steady-state during constant power output exercise of moderate intensity [below the lactate threshold (LT)] and

in the final stage of heavy intensity cycling (above LT), carried out before and after 1 and 7 weeks of strength training

Power output	Below LT (90 W)			Above LT (189 ± 24 W)		
	Week of training			Week of training		
	0	1	7	0	1	7
$\dot{V}O_2$ (ml min ⁻¹)	1,573 ± 62	1,516 ± 58 [#]	1,515 ± 44 [#]	2,812 ± 369	2,702 ± 355 [#]	2,776 ± 346
$\dot{V}CO_2$ (ml min ⁻¹)	1,448 ± 80	1,415 ± 52	1,379 ± 56 [#]	2,909 ± 382	2,807 ± 430 [#]	2,829 ± 390 [#]
\dot{V}_E (l min ⁻¹)	35 ± 4	34 ± 4	34 ± 3	70 ± 14	66 ± 13	67 ± 15
RER	0.92 ± 0.05	0.93 ± 0.02	0.91 ± 0.04	1.04 ± 0.02	1.04 ± 0.04	1.02 ± 0.04
HR (bt min ⁻¹)	111 ± 10	110 ± 10	110 ± 10	159 ± 8	155 ± 8	154 ± 8 [#]

Data are given as mean ± SD, and were calculated between the 4th and the 6th minute of cycling for moderate cycling intensity (<LT), and during the 6th minute of high-intensity cycling (>LT)

$\dot{V}O_2$ oxygen uptake, $\dot{V}CO_2$ carbon dioxide production, \dot{V}_E pulmonary ventilation, RER respiratory exchange ratio, HR heart rate

[#] Significantly lower ($P < 0.05$) than pre-training ("0"), as determined by the matched-pairs Wilcoxon signed rank test

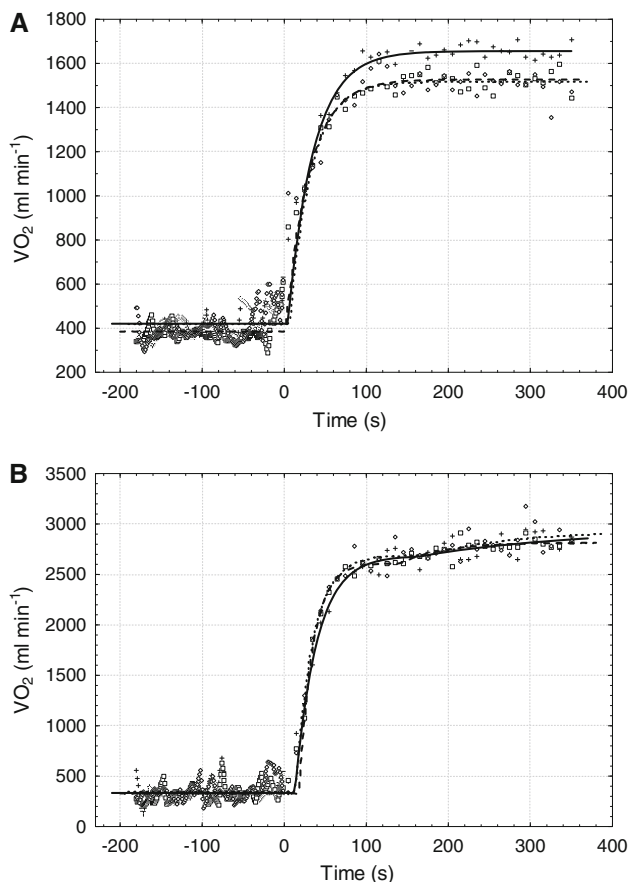


Fig. 2 Typical individual examples of the $\dot{V}O_2$ on-kinetics during moderate intensity (a) and heavy intensity (b) during cycling before training (0) and after the 1st and 7th week of training. Legend: weeks of training: 0-solid and crosses, 1-dashed and squares, 7-dotted and diamonds. See text for further details

observed for any variable, with the exception of τ_1 and EE $\dot{V}O_2$, which was lower after 1 week of training. Changes in A_1 (ΔA_1) following training are shown in Fig. 3b.

In absolute terms, for the heavy-intensity exercise, the A_1 decrease observed after 1 week of training was very similar to the statistically significant decrease observed for this variable during the moderate-intensity exercise. For the heavy-intensity exercise, however, the decrease was not statistically significant, presumably as a consequence of higher dispersion of data (see Fig. 3b). As expected, a slow component of the $\dot{V}O_2$ on-kinetics was observed during heavy-intensity exercise. The amplitude of the slow component (which corresponded to about 8 % of the total amplitude of the $\dot{V}O_2$ response) was unaffected by training.

Discussion

Maximal isometric strength training, performed 4 times per week for 7 weeks, resulted as expected in a significant increase (by about 19 %) in the developed force during maximal voluntary contraction (MVC) of the knee extensor muscles. The main and original finding of this study was that the adopted strength training protocol resulted in a significant decrease (by about 90 ml min⁻¹) in the amplitude of the primary component of the $\dot{V}O_2$ on-kinetics during moderate-intensity cycling indicating a reduced O₂ cost of cycling by about 7 %. This effect was significant after only 1 week of training, in association with a 15 % increase of muscle force. During heavy-intensity cycling a slightly less pronounced decrease in the amplitude of the primary component of the $\dot{V}O_2$ on-kinetics was observed; this decrease, however, did not reach statistical significance, due to higher data dispersion. Our results suggest that isometric strength training decreases the O₂ cost of cycling, and that this effect is more pronounced during moderate-intensity exercise.

Table 5 Values of $\dot{V}O_2$ on-kinetics parameters for moderate-intensity exercise

Variables	Pre-training level (week)	Week of training	
	0	1	7
BL $\dot{V}O_2$ (ml min ⁻¹)	354 ± 42	331 ± 56	353 ± 57
TD ₁ (s)	7.7 ± 8.1	10.1 ± 5.9	9.6 ± 4.7
τ ₁ (s)	26.7 ± 7.0	22.3 ± 5.5	22.2 ± 4.6
MRT (s)	34.4 ± 7.6	32.4 ± 2.5	31.3 ± 3.8
A ₁ (ml min ⁻¹)	1,260 ± 40	1,199 ± 74* versus 0	1,173 ± 50** versus 0
EE $\dot{V}O_2$ (ml min ⁻¹)	1,611 ± 50	1,528 ± 64** versus 0	1,524 ± 48** versus 0
Slow component $\dot{V}O_2$ (ml min ⁻¹)			
$\dot{V}O_2$ (6) – $\dot{V}O_2$ (3)	1.1 ± 3.0	0.4 ± 1.9	0.0 ± 1.6

Data are given as mean ± SD

BL $\dot{V}O_2$ baseline $\dot{V}O_2$ value, TD₁ time delay, τ₁ time constant, MRT mean response time, A₁ asymptotic amplitude for the exponential term, EE $\dot{V}O_2$ end exercise oxygen uptake

No slow component was present during moderate-intensity cycling. Values significantly different from pre-training level are marked with asterisks (* $P < 0.05$, ** $P < 0.02$, as determined by the matched-pairs Wilcoxon signed rank test). No significant differences between weeks 1 and 7 were detected by the Wilcoxon test

Previous studies described, after various programs of strength training, a significant improvement of running economy, accompanied by an enhancement of running performance (Johnston et al. 1997; Paavolainen et al. 1999; Millet et al. 2002; Saunders et al. 2006; Støren et al. 2008). An improvement in cycling efficiency, accompanied by an improvement of performance at maximal aerobic power was also described after strength training in competitive cyclists (Sunde et al. 2010). These data suggest that the introduction of strength training into endurance training programs of athletes may result in an enhancement of muscle efficiency. Beneficial effects of strength training on endurance performance have also been reported in previously untrained subjects (see, e.g., Hickson et al. 1988; Marcinik et al. 1991; McCarthy et al. 1995). The physiological mechanism responsible for the strength training-induced improvement of muscle efficiency remains unclear, although some research has pointed improvement in neuromuscular characteristics (see, e.g., Paavolainen et al. 1999; Støren et al. 2008). Our results, showing a very rapid effect (i.e., occurring only after 1 week) of isometric strength training on the oxygen cost of cycling during moderate-intensity cycling support this observation. However, one should not exclude in this case some muscle adaptive responses leading to an improvement of muscle metabolic stability (see Zoladz et al. 2006; Majerczak et al. 2012). An improvement of muscle metabolic stability during exercise could enhance muscle efficiency (Woledge 1988).

It is well established that during moderate-intensity cycling exercise type I muscle fibres are predominantly recruited, whereas during heavy exercise intensity type II muscle fibres are recruited to a greater extent (for an overview, see, e.g., Sargeant and Jones 1995). In the

present study, we have found a greater decrease of the amplitude of the fundamental component of $\dot{V}O_2$ kinetics (indicating a lower O₂ cost of cycling) during moderate versus heavy-intensity exercise. Therefore, our results suggest that strength training resulted in an enhancement of mechanical efficiency predominantly in type I muscle fibres.

In the present study, we have also evaluated the effect of strength training on the amplitude of the slow component of the $\dot{V}O_2$ on-kinetics, which was not significantly affected by the training program. Thus, the substantial increase in force during MVC of the knee extensor muscles had no significant effect on the amplitude of the slow component of the $\dot{V}O_2$ on-kinetics. This was rather surprising, since strength training could decrease the need of recruitment of type II muscle fibres, considered to be less efficient than type I muscle fibres. An increased recruitment of type II fibres is usually considered responsible for the occurrence of the slow component (Jones et al. 2011). On the other hand, there is a growing body of evidence that not the type II fibres recruitment per se, but rather its consequence, i.e., the accumulation of metabolites in the muscles, such as ADP_{free}, P_i, H⁺, AMP, etc., would be responsible for the slow component of $\dot{V}O_2$ on-kinetics and the associated decreases in “metabolic stability” and muscle efficiency (Zoladz et al. 2008; Vanhatalo et al. 2011; Cannon et al. 2011; Jones et al. 2011). According to this scenario, the training-induced increase in force during MVC of the knee extensor muscles observed in our study did not improve muscle metabolic stability during the 6 min bout of heavy exercise intensity cycling. This was associated with an unchanged amplitude of the slow component of the $\dot{V}O_2$ on-kinetics.

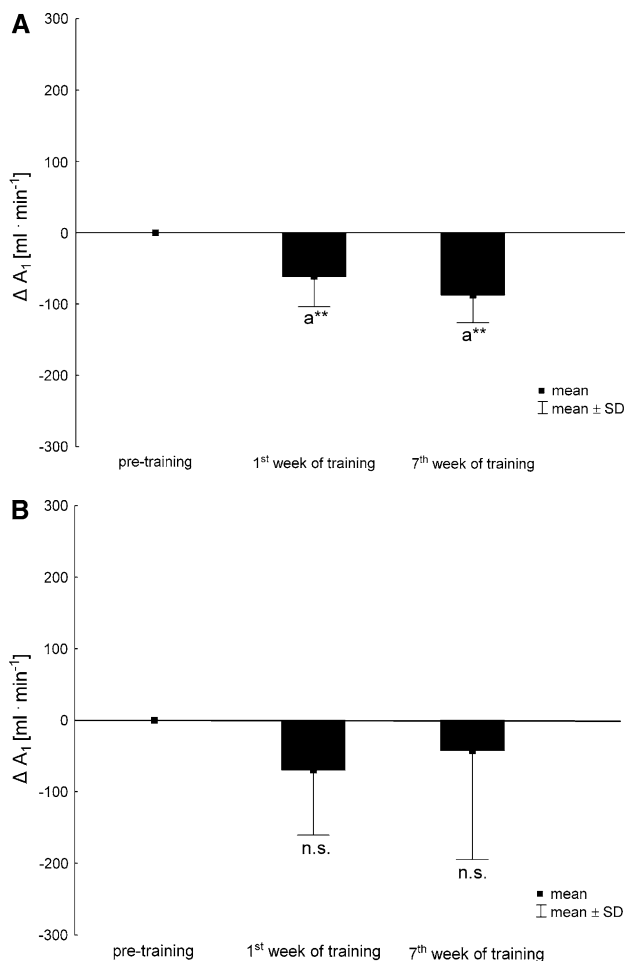


Fig. 3 Mean \pm SD changes in the amplitude of the fundamental component of the $\dot{V}O_2$ on-kinetics (ΔA_1), following 1 and 7 weeks of training, with respect to the baseline values obtained before training. **a** Moderate-intensity exercise (** $P < 0.02$ as determined by the matched-pairs Wilcoxon signed rank test. Preliminary Friedman test resulted in $P = 0.006$). **b** Heavy-intensity exercise [no significant differences were detected by the Wilcoxon test ($P > 0.05$) and no consistent changes in A_1 were detected with the preliminary Friedman test ($P > 0.05$)]

In the present study, we have also assessed the effect of 7-week strength training on the rate of $\dot{V}O_2$ adjustment at the onset of moderate- and heavy-intensity cycling exercise, i.e., the so-called “fundamental” component of the $\dot{V}O_2$ on-kinetics (Whipp 1996; Rossiter 2011). Before training the time constants of the fundamental component were about 27–28 s for both moderate and heavy exercise intensity; these values are typical for young and healthy subjects (see, e.g., Whipp et al. 2002). The time constants were essentially not affected by strength training, both during moderate- and heavy-intensity exercise, although it should be noticed that during high-intensity cycling a transient acceleration of $\dot{V}O_2$ on-kinetics after 1 week of the isometric strength training occurred (τ_1 amounting to 28.3 ± 4.2 vs. 24.2 ± 6.3 s ($P < 0.05$), before training

and after 1 week of training, respectively) (see Table 6). This suggests that isometric strength training in its early stage can induce some temporary muscle adaptive responses leading to acceleration of the $\dot{V}O_2$ on-kinetics (for overview, see, e.g., Wilkinson et al. 2008; Wang et al. 2011).

Using a computer model of oxidative phosphorylation (Korzeniewski and Zoladz 2001) we have demonstrated that the rate of $\dot{V}O_2$ on-kinetics in the human skeletal muscle reflects the changes in muscle metabolic stability at a given ATP usage (power output) (Korzeniewski and Zoladz 2003). Faster $\dot{V}O_2$ on-kinetics is accompanied by a lesser decrease in muscle PCr concentrations and a smaller accumulation of metabolites associated with muscle fatigue, such as P_i , ADP_{free} , IMP, etc. (Korzeniewski and Zoladz 2003, 2004; Zoladz et al. 2006; Grassi et al. 2011). Therefore, our results suggest that the isometric strength training-induced adaptive response(s) resulting in an increase of muscle force is/are not harmful to the mechanism determining muscle metabolic stability during moderate and heavy exercise intensity (at least within 7 weeks).

The results of the present study, concerning the effect of the strength training on the $\dot{V}O_2$ on-kinetics, are in accordance with the study by Millet et al. (2002), showing that in well-trained triathletes heavy weight training in combination with endurance training resulted in an improvement of maximal strength and running economy with no significant effects on the $\dot{V}O_2$ kinetics during heavy-intensity exercise.

In the present study, we have also evaluated the effect of strength training on $\dot{V}O_{2\text{peak}}$ and the power output at $\dot{V}O_{2\text{peak}}$. We showed that “pure” isometric strength training (i.e., not associated with endurance exercise), resulting in a significant increase of muscle force, did not affect the $\dot{V}O_{2\text{peak}}$ in young and healthy men. Interestingly, however, power output reached at $\dot{V}O_{2\text{peak}}$ during the incremental test was slightly but significantly higher after the 7th week of the strength training, when compared to that determined before training. This slight increase in maximal power output, occurring in the presence of an unchanged $\dot{V}O_{2\text{peak}}$, is likely attributable to the lower O_2 cost of cycling after training. A similar effect, i.e., an improvement of performance at maximal aerobic power in competitive cyclists after maximal strength training, was recently reported by Sunde et al. (2010).

Conclusion

In conclusion, in young, healthy and previously untrained men 7 weeks of isometric strength training, resulting in a significant (about 19 %) increase of force during MVC of the knee extensor muscles, was not “harmful” for $\dot{V}O_{2\text{peak}}$

Table 6 Values of $\dot{V}O_2$ on-kinetics parameters for heavy-intensity exercise

Variables	Pre-training level (week)	Week of training	
	0	1	7
BL $\dot{V}O_2$ (ml min ⁻¹)	354 ± 56	339 ± 49	369 ± 49** versus 1
TD ₁ (s)	10.29 ± 3.13	12.77 ± 3.28	11.83 ± 4.03
τ ₁ (s)	28.3 ± 4.2	24.2 ± 6.3* versus 0	26.0 ± 8.7
MTR (s)	38.6 ± 4.24	36.9 ± 3.9	37.8 ± 5.6
A ₁ (ml min ⁻¹)	2,272 ± 284	2,202 ± 259	2,229 ± 254
EE $\dot{V}O_2$ (ml min ⁻¹)	2,827 ± 350	2,724 ± 356* versus 0	2,796 ± 359* versus 1
Slow component $\dot{V}O_2$ (ml min ⁻¹)			
$\dot{V}O_2$ (6) – (A ₁ + BL $\dot{V}O_2$)	203 ± 104	178 ± 124	190 ± 165
$\dot{V}O_2$ (6) – $\dot{V}O_2$ (3)	175 ± 79	134 ± 80	158 ± 84

Data are given as mean ± SD

BL $\dot{V}O_2$ $\dot{V}O_2$ baseline value, TD₁ time delay, τ₁ time constant, MTR mean time response, A₁ asymptotic amplitude for the exponential term, EE $\dot{V}O_2$ end exercise oxygen uptake

Significant differences are marked with asterisks (* $P < 0.05$, ** $P < 0.02$, as determined by the matched-pairs Wilcoxon signed rank test)

and muscle metabolic stability, as judged by the fundamental component of pulmonary $\dot{V}O_2$ on-kinetics. Rather surprisingly, the training-induced increase in MVC had no effect on the amplitude of the slow component of $\dot{V}O_2$ on-kinetics. Interestingly, strength training significantly decreased the O₂ cost of cycling during moderate-intensity exercise. This observation appears compatible with the increased running economy and cycling efficiency observed after strength training in endurance athletes, and confirms that strength training can have beneficial effects on performance during endurance events.

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Conflict of interest The authors declare that they have no conflict of interest.

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