

Pulmonary O₂ uptake on-kinetics in sprint- and endurance-trained athletes

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Abstract: Pulmonary O₂ uptake kinetics during “step” exercise have not been characterized in young, sprint-trained (SPT), athletes. Therefore, the objective of this study was to test the hypotheses that SPT athletes would have (i) slower phase II kinetics and (ii) a greater oxygen uptake “slow component” when compared with endurance-trained (ENT) athletes. Eight sub-elite SPT athletes (mean (± SD) age = 25 (±7) y; mass = 80.3 (±7.3) kg) and 8 sub-elite ENT athletes (age = 28 (±4) y; mass = 73.2 (±5.1) kg) completed a ramp incremental cycle ergometer test, a Wingate 30 s anaerobic sprint test, and repeat “step” transitions in work rate from 20 W to moderate- and severe-intensity cycle exercise, during which pulmonary oxygen uptake was measured breath by breath. The phase II oxygen uptake kinetics were significantly slower in the SPT athletes both for moderate (time constant, τ ; SPT 32 (±4) s vs. ENT 17 (±3) s; $p < 0.01$) and severe (SPT 32 (±12) s vs. ENT 20 (±6) s; $p < 0.05$) exercise. The amplitude of the slow component (derived by exponential modelling) was not significantly different between the groups (SPT 0.55 (±0.12) L·min⁻¹ vs. ENT 0.50 (±0.22) L·min⁻¹), but the increase in oxygen uptake between 3 and 6 min of severe exercise was greater in the SPT athletes (SPT 0.37 (±0.08) L·min⁻¹ vs. ENT 0.20 (±0.09) L·min⁻¹; $p < 0.01$). The phase II τ was significantly correlated with indices of aerobic exercise performance (e.g., peak oxygen uptake (moderate-intensity $r = -0.88$, $p < 0.01$; severe intensity $r = -0.62$; $p < 0.05$), whereas the relative amplitude of the oxygen uptake slow component was significantly correlated with indices of anaerobic exercise performance (e.g., Wingate peak power output; $r = 0.77$; $p < 0.01$). Thus, it could be concluded that sub-elite SPT athletes have slower phase II oxygen uptake kinetics and a larger oxygen uptake slow component compared with sub-elite ENT athletes. It appears that indices of aerobic and anaerobic exercise performance differentially influence the fundamental and slow components of the oxygen uptake kinetics.

Key words: fitness, aerobic, anaerobic, oxygen uptake kinetics, slow component, exercise.

Résumé : La cinétique de la consommation d'oxygène n'a pas encore fait l'objet d'étude chez de jeunes athlètes entraînés au sprint (SPT). La présente étude se propose donc de vérifier l'hypothèse selon laquelle les athlètes SPT passent par une phase II plus lente en ce qui concerne la cinétique de la consommation d'oxygène et que son ampleur dépasse celle des individus entraînés à l'endurance (ENT). Huit sujets SPT presque rendus au niveau élite (moyenne (± é-t) âge 25 (± 7) ans; masse 80,3 (±7,3) kg) et 8 sujets ENT (âge 28 (± 4) ans; masse 73,2 (± 5,1) kg) également presque rendus au niveau élite participent à un test d'effort progressif sur ergocycle, soit l'épreuve de Wingate d'une durée de 30 s et à une séance répétée de transition brusque de palier depuis une intensité de travail égale à 20 W jusqu'à une intensité de travail modéré et très intense ; durant ces exercices de transition, on évalue la consommation d'oxygène à chaque respiration. Tant à l'effort modéré qu'à l'effort très intense, on observe chez les athlètes SPT au cours de la Phase II une cinétique de la consommation d'oxygène significativement plus lente que chez les athlètes ENT (effort modéré : constante de temps, τ ; SPT: 32 (± 4) s comparativement à ENT: 17 (± 3) s; $p < 0,01$) ; effort très intense : (SPT: 32 (± 12) s comparativement à ENT: 20 (± 6) s; $p < 0,05$). On n'observe pas de différence d'ampleur de la composante lente de la consommation d'oxygène (estimée par modélisation exponentielle) : SPT: 0,55 (± 0,12) L·min⁻¹ comparativement à ENT: 0,50 (± 0,22) L·min⁻¹, mais on observe chez les athlètes SPT au cours de l'effort très intense une plus grande augmentation de la consommation d'oxygène entre la troisième et la sixième minute (SPT: 0,37 (± 0,08) L·min⁻¹ comparativement à ENT: 0,20 (± 0,09) L·min⁻¹; $p < 0,01$). On observe en plus une corrélation significative entre la constante τ de la phase II et des indices de la performance aérobie (la consommation d'oxygène de crête : effort modéré, $r = -0,88$, $p < 0,01$ et effort très intense, $r = -0,62$; $p < 0,05$) et une corrélation significative entre l'amplitude relative de la composante lente et des indices de la performance anaérobie (puissance développée au cours du test de Wingate; $r = 0,77$; $p < 0,01$). Comparativement aux athlètes ENT presque rendus au niveau élite, on observe chez les athlètes SPT une cinétique plus lente de la consommation d'oxygène et une plus grande ampleur de ce dernier au cours de la phase II. Les indices de performance au cours d'un effort aérobie et anaérobie influencent de façon différente les composantes fondamentales et lentes de la cinétique de la consommation d'oxygène.

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Introduction

The speed with which oxygen uptake ($\dot{V}O_2$) rises to or toward the requisite “steady-state” following the onset of exercise dictates the proportional contribution of oxidative and non-oxidative metabolism to energy transfer. The slower the $\dot{V}O_2$ kinetics, the larger the incurred O_2 deficit, and the greater the contribution from substrate phosphorylation, involving the depletion of intramuscular high-energy phosphates (chiefly phosphocreatine (PCr)), and the anaerobic breakdown of glycogen to lactate. It has been shown that high levels of aerobic fitness and endurance training are associated with faster $\dot{V}O_2$ kinetics, whereas senescence, deconditioning, and a variety of disease states result in slower $\dot{V}O_2$ kinetics (Hagberg et al. 1980; Powers et al. 1985; Sietsema 1992; Babcock et al. 1994; Sietsema et al. 1994; Chilibeck et al. 1996; Regensteiner et al. 1998; Bauer et al. 1999; Koppo et al. 2004a; Kilding et al. 2006). The extent to which $\dot{V}O_2$ kinetics are determined by central (i.e., cardiovascular delivery of O_2 to skeletal muscle) or peripheral (i.e., related to the oxidative metabolic processes within the myocytes) factors is unclear, but it appears to depend upon the exercise modality and intensity, as well as on the physiological characteristics of the subjects (Poole and Jones 2005).

During constant work-rate exercise performed above the so-called lactate threshold (LT), $\dot{V}O_2$ does not attain an early steady-state; rather, it continues to rise until either a delayed steady-state is attained (for heavy exercise below the critical power, CP) or the peak $\dot{V}O_2$ is reached (for severe exercise above the CP; Poole et al. 1988). This “slow component” of $\dot{V}O_2$ reflects a reduction in muscle efficiency during exercise above LT (Poole et al. 1991; Rossiter et al. 2002a), with consequent implications for exercise tolerance (Poole et al. 1994). Although the underpinning mechanisms remain obscure, the $\dot{V}O_2$ slow-component phenomenon has been most frequently attributed to an increased recruitment of type II muscle fibres during exercise above LT (Poole et al. 1994; Barstow et al. 1996; Pringle et al. 2003; Krstrup et al. 2004).

The kinetics of $\dot{V}O_2$ during exercise have been characterized in a wide range of populations including children, the senescent, endurance athletes, and those with cardiovascular or metabolic diseases, and the results have provided useful insight into the factors that might limit $\dot{V}O_2$ kinetics under different circumstances (Hagberg et al. 1980; Powers et al. 1985; Sietsema 1992; Babcock et al. 1994; Sietsema et al. 1994; Chilibeck et al. 1996; Regensteiner et al. 1998; Bauer et al. 1999; Williams et al. 2001; Fawcner and Armstrong 2003; Koppo et al. 2004a; Kilding et al. 2006). One population that has not been well studied to date is that of sprint or power-trained athletes. To our knowledge, $\dot{V}O_2$ kinetics in sprint and power-trained athletes has only been reported in a limited number of studies using sinusoidal (Fukuoka et al.

1995, 1997) or pseudorandom binary sequence (PRBS; Edwards et al. 1999, 2003) work-rate forcing functions. This lack of information on the $\dot{V}O_2$ kinetics of sprint and power-trained athletes, particularly in response to “step” exercise, is perhaps surprising given that these athletes would be expected to have distinctive muscle metabolic properties (relative both to the general population and to endurance-trained athletes), including a higher proportion of type II fibres in the relevant muscles, a greater muscle phosphocreatine concentration ([PCr]), greater anaerobic enzyme activities, and perhaps a reduced muscle capillary density (Costill et al. 1976; Bergh et al. 1978; Abernethy et al. 1990; Bouchard et al. 1992; Ross and Leveritt 2001). As a consequence, these athletes might be expected to exhibit distinctive $\dot{V}O_2$ kinetic responses to exercise (Jones et al. 2005).

Therefore, the principal purpose of this study was to characterize $\dot{V}O_2$ kinetics following the onset of step exercise in athletes who were training exclusively for competition in sprint sports, and to contrast their responses to those elicited by endurance-trained athletes. We hypothesized that (i) the pulmonary $\dot{V}O_2$ kinetics would be slower (i.e., the phase II time constant (τ), which reflects muscle $\dot{V}O_2$ kinetics (Grassi et al. 1996; Koga et al. 2005), would be longer) in the sprint-trained athletes (SPT) than in the endurance-trained athletes (ENT) during both moderate- (below LT) and severe-intensity exercise, and (ii) the magnitude of the $\dot{V}O_2$ slow component would be greater during severe exercise in the SPT athletes than in the ENT athletes. Previous studies have suggested that the speed of the $\dot{V}O_2$ adaptation to exercise is negatively related to the $\dot{V}O_{2\text{peak}}$ (Weltman and Katch 1976; Powers et al. 1985). However, it is not known whether other variables, including those that are considered to represent non-oxidative metabolic function, correlate more strongly with $\dot{V}O_2$ kinetics. A secondary purpose of the study was therefore to examine the relationship between the key parameters of the $\dot{V}O_2$ kinetic response to exercise (i.e., phase II τ and $\dot{V}O_2$ slow component amplitude) and indices of physical fitness as derived from specific tests of aerobic and anaerobic exercise performance.

Materials and methods

Subjects

Eight SPT athletes and 8 ENT athletes (all male) volunteered and gave their written informed consent to participate in this study, which had previously received approval from the local Research Ethics Committee. The SPT athletes comprised 5 track sprinters, 1 sprint cyclist, a long jumper, and a decathlete; the ENT athletes included 3 distance runners, 3 endurance cyclists, and 2 tri-athletes. Their physical characteristics are shown in Table 1. The SPT and ENT athletes were of a similar relative performance standard and could be described as sub-elite (i.e., they competed at re-

Table 1. Mean (\pm SD) physical and physiological characteristics of the sprint- and endurance-trained subjects.

	Sprint trained	Endurance trained
Age (y)	25 (\pm 7)	28 (\pm 4)
Height (m)	1.80 (\pm 0.08)	1.81 (\pm 0.07)
Body mass (kg)	80.3 (\pm 7.3)	73.2 (\pm 5.1)*
$\dot{V}O_{2\text{ peak}}$ (L \cdot min $^{-1}$)	3.78 (\pm 0.30)	4.39 (\pm 0.39)**
$\dot{V}O_{2\text{ peak}}$ (mL \cdot kg $^{-1}$ \cdot min $^{-1}$)	47.1 (\pm 4.0)	60.2 (\pm 4.6)**
Peak work rate (W)	343 (\pm 34)	434 (\pm 49)**
Estimated LT $\dot{V}O_2$ (L \cdot min $^{-1}$)	1.83 (\pm 0.13)	2.39 (\pm 0.27)**
Estimated LT work rate (W)	139 (\pm 24)	204 (\pm 37)**
Wingate peak power (W)	1187 (\pm 165)	937 (\pm 181)*
Wingate peak power (W \cdot kg $^{-1}$)	14.7 (\pm 1.0)	12.8 (\pm 2.0)*
Wingate mean power (W)	767 (\pm 92)	680 (\pm 79)*
Wingate fatigue index (%)	61 (\pm 7)	41 (\pm 13)**

Note: *, significantly different at $p < 0.05$; **, significantly different at $p < 0.01$.

gional and national championships in the UK, but were not of international standard). The SPT athletes had been engaged in specific sprint and power training for 9 ± 5 y, for an average of 9 ± 3 h/week; the ENT athletes had been training for endurance sports for 9 ± 7 y, for an average of 11 ± 6 h/week. Each of the subjects visited the laboratory on 4 occasions within a period of 21 d (see below). On test days, the subjects were required to report to the laboratory well hydrated and at least 2 h after the consumption of a light meal, and having completed no strenuous exercise in the preceding 24 h.

Experimental procedures

All exercise testing took place in an air-conditioned laboratory (maintained at 18 °C) at the same time of day (± 2 h) for each subject. On their first visit to the laboratory, the subjects completed a ramp incremental exercise test to volitional exhaustion on an electrically braked cycle ergometer (Excalibur Sport, Lode, Netherlands) for the estimation of the LT using gas-exchange indices and for the determination of $\dot{V}O_{2\text{ peak}}$. Following 3 min of “unloaded” baseline cycling, the work rate was increased in a ramp fashion (30 W \cdot min $^{-1}$) until the subject was unable to continue. Subjects typically became exhausted within 10–14 min. The saddle and handlebar heights were recorded and reproduced in subsequent tests. Subjects cycled at a pedal rate of 80 r \cdot min $^{-1}$ in the incremental test and in all subsequent step tests. The $\dot{V}O_{2\text{ peak}}$ was defined as the highest 30 s mean value recorded before the subject’s volitional termination of the test. The LT was estimated from a cluster of gas-exchange indices including (i) the first disproportionate increase in carbon dioxide output ($\dot{V}CO_2$) from visual inspection of individual plots of $\dot{V}CO_2$ vs. $\dot{V}O_2$; and (ii) an increase in expiratory ventilation ($\dot{V}E$) divided by $\dot{V}O_2$ with no increase in $\dot{V}E/\dot{V}CO_2$. The work rates estimated to require ~90% of the LT (moderate exercise) and ~70% of the difference (Δ) between the LT and $\dot{V}O_{2\text{ peak}}$ (severe exercise) were calculated, with account taken of the mean response time of the $\dot{V}O_2$ adaptation to ramp exercise (Whipp et al. 1981).

On their second visit to the laboratory, the subjects com-

pleted a standard Wingate anaerobic sprint test (Bar-Or 1987). Briefly, the subjects mounted a calibrated, mechanically braked, friction-loaded, cycle ergometer (Monark 874E, Varberg, Sweden) that was fitted with toe-clips. The subjects were instructed not to pace themselves, to remain seated, and to provide a maximum effort throughout the test. They were asked to bring the pedal rate up to 60 r \cdot min $^{-1}$ with no resistive load and were given a 5 s countdown before the individualized resistive load (set at 7.5% of body mass) was applied. They were then required to complete a 30 s bout of supra-maximal exercise. They received strong verbal encouragement from the experimenters throughout the test. An optical sensor was used to measure rotation of the ergometer flywheel and commercial software (Cranlea, Birmingham, UK) was used to calculate the inertia-corrected power output. Performance indices that were computed included the peak power output and mean power output (expressed in both absolute (W) and relative (W \cdot kg $^{-1}$) terms) and the “fatigue index”, which is defined as the degree of power drop-off during the test ($[\text{peak power} - \text{minimum power} / \text{peak power}] \times 100$).

On their third and fourth visits to the laboratory, the subjects completed two moderate exercise bouts, followed by one severe exercise bout, with all exercise bouts separated by 10 min of rest. Each step exercise test began with 3 min of baseline pedalling (at 20 W) before an abrupt transition to the target work rate, which was maintained for 6 min.

Measurements

Pulmonary gas exchange was measured breath-by-breath throughout all exercise tests, except for the Wingate anaerobic test. Subjects wore a nose-clip and breathed through a low dead space, low-resistance mouthpiece and volume sensor assembly (Jaeger Triple V, Hoechberg, Germany). Gas was continuously drawn down a capillary line into rapid-response gas analyzers (Jaeger Oxycon Alpha). Gas-exchange variables (at the mouth) were calculated and displayed breath-by-breath once the delay between the volume and concentration signals had been accounted for. The volume transducer was calibrated before each test with a 3 L calibration syringe, and the analyzers were calibrated with gases of known concentration. Heart rate was recorded every 5 s using short-range telemetry (Polar PE 4000, Kempele, Finland). A fingertip blood sample was collected in a capillary tube immediately before and after one of the moderate exercise bouts and one of the severe exercise bouts and subsequently analyzed for blood [lactate] using an automated analyzer (YSI 1500 Sport lactate analyzer, Yellow Springs Instruments, Ohio). The difference between the pre- and post-exercise blood [lactate] values (i.e., Δ blood [lactate]) was calculated.

The breath-by-breath $\dot{V}O_2$ data from each test were initially examined to exclude errant breaths caused by coughing, swallowing, sighing, etc., and those values lying more than 4 standard deviations from the local mean (based on 5 breaths) were deleted. The breath-by-breath data were subsequently linearly interpolated to provide 1 s values. For each subject and each exercise condition, the identical repetitions of each work rate were then time aligned to the start of exercise and ensemble averaged to reduce the breath-to-breath noise and enhance the underlying physiological response

characteristics (Lamarra et al. 1987). The baseline $\dot{V}O_2$ was defined as the average $\dot{V}O_2$ measured during baseline cycling (20 W) between 150 and 30 s before the start of exercise. The first 20 s of data after the onset of exercise (i.e., the phase I response) were deleted. Subsequently, a single exponential model was used to analyze the $\dot{V}O_2$ responses to moderate exercise and a bi-exponential model was used for severe exercise as described in the following equations:

$$[1] \quad \dot{V}O_{2(t)} = \dot{V}O_{2 \text{ baseline}} + A_p(1 - e^{-(t-T_{dp})/\tau_p})$$

(moderate exercise)

$$[2] \quad \dot{V}O_{2(t)} = \dot{V}O_{2 \text{ baseline}} + A_p(1 - e^{-(t-T_{dp})/\tau_p}) + A_s(1 - e^{-(t-T_{ds})/\tau_s})$$

(severe exercise).

The parameters of the model were determined using a non linear least-square algorithm. In the equations above, $\dot{V}O_{2(t)}$ represents the absolute $\dot{V}O_2$ at a given time t and $\dot{V}O_{2 \text{ baseline}}$ represents the average $\dot{V}O_2$ throughout the baseline cycling period. For moderate exercise, the $\dot{V}O_2$ response was appropriately fit with a single exponential curve that described the amplitude (A_p), the time delay (T_{dp}), and the time constant (τ_p) of the fundamental increase in $\dot{V}O_2$ above baseline. For severe exercise, the $\dot{V}O_2$ response was modelled with two independent exponential curves, one describing the fundamental $\dot{V}O_2$ response and one describing the $\dot{V}O_2$ slow-component response. Therefore, this equation also includes terms describing the amplitude of the $\dot{V}O_2$ slow component (A_s), the time delay before the appearance of the slow component (T_{ds}), and the time constant of the slow-component development (τ_s). Because the asymptotic value (A_s) of the exponential term describing the $\dot{V}O_2$ slow component may represent a higher value than is actually reached at the end of the exercise, the actual amplitude of the $\dot{V}O_2$ slow component at the end of exercise was defined as A'_s . The magnitude of the $\dot{V}O_2$ slow component was also expressed as the difference in $\dot{V}O_2$ between 3 and 6 min of exercise ($\dot{V}O_{2 [6-3]}$) and the difference in $\dot{V}O_2$ between 2 and 6 min of exercise ($\dot{V}O_{2 [6-2]}$); the latter was calculated because the $\dot{V}O_2$ slow component typically emerges before 3 min of exercise has elapsed.

Statistics

The responses of the SPT and ENT athletes to the ramp incremental test, the Wingate anaerobic test, and the step tests to moderate- and severe-intensity constant work-rate exercise were compared using independent-sample t tests, following confirmation of homogeneity of variance, using the Social Science Statistical Package (version 11.0). The relationships between indices of aerobic and anaerobic exercise performance and $\dot{V}O_2$ kinetics during the step exercise tests were examined using Pearson's product-moment correlation coefficients. Statistical significance was accepted when $p < 0.05$. Data are presented as mean \pm SD.

Results

The physical and physiological characteristics of the SPT and ENT athletes are shown in Table 1. The SPT and ENT

athletes were of similar age and stature, but the SPT athletes were significantly heavier than the ENT athletes (Table 1). The SPT athletes achieved a significantly higher peak power output and mean power output on the Wingate anaerobic test and also had a significantly higher fatigue index compared with the ENT athletes (Table 1). The ENT athletes achieved a significantly higher $\dot{V}O_{2 \text{ peak}}$ during the ramp incremental cycle test compared with the SPT athletes and their superior aerobic fitness was also evident in the work rates achieved at the estimated LT and at exhaustion (Table 1). In the SPT group, the mean peak power output achieved during the Wingate test was 3.4 \times higher than that attained during the ramp incremental test, whereas in the ENT group the value was 2.2. The work rates applied during the square-wave tests were 184 (± 33) W and 125 (± 21) W for moderate exercise, and 355 (± 41) W and 268 (± 29) W for severe exercise, for ENT and SPT athletes, respectively. These work rates were equivalent to 57% and 52% $\dot{V}O_{2 \text{ peak}}$ for moderate exercise and 87% and 84% $\dot{V}O_{2 \text{ peak}}$ for severe exercise (at the end of the primary phase, rising to 99% $\dot{V}O_{2 \text{ peak}}$ after 6 min) for ENT and SPT athletes, respectively.

The parameters of the $\dot{V}O_2$ kinetics and the blood [lactate] values for SPT and ENT athletes are displayed in Tables 2 (moderate exercise) and 3 (severe exercise). For moderate exercise, the baseline $\dot{V}O_2$, initial time delay, and gain were not significantly different between the two groups. However, the phase II τ was significantly longer in the sprinters (SPT: 32 (± 4) vs. ENT: 17 (± 3) s; $p < 0.001$). The 95% confidence intervals for the estimation of the phase II τ were 3 (± 1) and 1 (± 1) for SPT and ENT, respectively. For severe exercise, the baseline $\dot{V}O_2$, initial time delay, and fundamental component gain were also not significantly different between the two groups but, again, the phase II τ was significantly longer in the sprinters (SPT 32 (± 9) s vs. ENT 20 (± 6) s; $p < 0.01$). The 95% confidence intervals for the estimation of the phase II τ were 4 (± 2) and 3 (± 1) for SPT and ENT, respectively. The modelled parameters for the $\dot{V}O_2$ slow component were not significantly different between the two groups, although the relative contribution made by the $\dot{V}O_2$ slow component to the end-exercise $\dot{V}O_2$ tended to be greater in the SPT athletes ($p < 0.10$). However, when the $\dot{V}O_2$ slow component was expressed either as $\dot{V}O_{2 [6-2]}$ or $\dot{V}O_{2 [6-3]}$, it was significantly greater in the SPT athletes (Table 3). The differences in $\dot{V}O_2$ kinetics between the SPT and ENT athletes are illustrated, using the responses of representative subjects, in Figs. 1A (moderate exercise) and 1B (severe exercise).

Table 4 presents the correlation coefficients between the principal parameters of the $\dot{V}O_2$ kinetics for moderate and severe exercise (phase II τ and $\dot{V}O_2$ slow component amplitude) and several measures of aerobic and anaerobic exercise performance (as derived from the ramp incremental test and the Wingate anaerobic test, respectively). The variables that correlated most strongly with the phase II τ for moderate exercise were the indices of aerobic exercise performance such as the $\dot{V}O_{2 \text{ peak}}$ ($r = -0.88$), the peak work rate ($r = -0.85$), and the work rate at the estimated LT ($r = -0.80$). These same variables were also significantly correlated with the phase II τ for severe exercise, although the corre-

Table 2. Mean (\pm SD) parameters of the $\dot{V}O_2$ kinetic response to moderate-intensity exercise in sprint- and endurance-trained subjects.

	Sprint trained	Endurance trained
Δ Work rate (W)	105 (\pm 21)	164 (\pm 33)**
Δ Blood [lactate] (mmol/L)	0.5 (\pm 0.5)	0.8 (\pm 0.4)
Baseline $\dot{V}O_2$ (L \cdot min $^{-1}$)	0.87 (\pm 0.12)	0.80 (\pm 0.08)
Time delay (s)	14 (\pm 5)	15 (\pm 2)
Time constant (s)	32 (\pm 4)	17 (\pm 3)**
Amplitude (L \cdot min $^{-1}$)	1.10 (\pm 0.22)	1.69 (\pm 0.32)**
Gain (mL \cdot min $^{-1}$ \cdot W $^{-1}$)	10.5 (\pm 0.8)	10.3 (\pm 0.3)
O $_2$ deficit (L)	0.83 (\pm 0.11)	0.90 (\pm 0.18)

Note: **, significantly different at $p < 0.01$.

lations were generally not as strong (Table 4). In contrast, indices of anaerobic exercise performance, such as the peak power output achieved during the Wingate anaerobic test ($r = 0.77$), were more strongly correlated with the relative amplitude of the $\dot{V}O_2$ slow component. The $\dot{V}O_{2 [6-3]}$ and $\dot{V}O_{2 [6-2]}$ estimates of the $\dot{V}O_2$ slow component were also correlated with Wingate test performance ($r = 0.65$ ($p < 0.05$) and 0.72 ($p < 0.01$), respectively, for peak power output, and $r = 0.64$ ($p < 0.05$) and 0.71 ($p < 0.01$), respectively, for fatigue index). The relationships between the $\dot{V}O_2$ kinetics (phase II τ ; $\dot{V}O_2$ slow component amplitude) and selected indices of aerobic and anaerobic exercise performance are illustrated in Figs. 2 and 3, respectively.

Discussion

The results of the study were consistent with our hypothesis that the phase II $\dot{V}O_2$ kinetics would be slower in the SPT than in the ENT athletes. Our results extend earlier reports that the speed of the fundamental component $\dot{V}O_2$ kinetics is inversely related to aerobic fitness (Cerretelli et al. 1979; Powers et al. 1985) and that sprint and power-trained athletes have slower $\dot{V}O_2$ kinetics than endurance-trained athletes (sinusoidal exercise, Fukuoka et al. 1995, 1997; PRBS, Edwards et al. 1999, 2003). Our second hypothesis that the magnitude of the $\dot{V}O_2$ slow component would be greater in the SPT than in the ENT athletes was only partly supported: the amplitude of the $\dot{V}O_2$ slow component, when derived from bi-exponential modelling of the $\dot{V}O_2$ response to severe exercise, was not significantly different between SPT and ENT athletes, whereas the $\dot{V}O_{2 [6-3]}$ and $\dot{V}O_{2 [6-2]}$ estimates of the slow component were significantly greater in the SPT athletes. This difference might be related to the earlier appearance of the $\dot{V}O_2$ slow component in the ENT athletes (mean $T_{ds} = 103$ vs. 133 s). Several indices of anaerobic exercise performance were positively correlated both with the relative amplitude of the $\dot{V}O_2$ slow component and with the $\dot{V}O_{2 [6-3]}$ and $\dot{V}O_{2 [6-2]}$ estimates. These results therefore suggest that a subject's propensity for aerobic or anaerobic exercise has the potential to differentially influence the fundamental and slow components of the $\dot{V}O_2$ kinetics.

We were successful in recruiting two cohorts of athletes

of similar age and training status, but with considerable differences in aerobic and anaerobic exercise performance. The athletes were among the best in the locale and were highly committed to their sports, but could best be described as sub-elite rather than elite (i.e., they competed at regional and national levels rather than at the international level). The SPT athletes had significantly lower values for $\dot{V}O_{2 \text{ peak}}$ and estimated LT (as determined from the ramp incremental cycle ergometer test) and significantly higher values for peak power output and fatigue index (as measured during the Wingate anaerobic test) compared with the ENT athletes. The ENT athletes (comprising runners, cyclists, and tri-athletes) in our study had $\dot{V}O_{2 \text{ peak}}$ values measured during cycle ergometer exercise of approximately 60 mL \cdot kg $^{-1}$ \cdot min $^{-1}$. This is similar to that reported by Kilding et al. (2005) for endurance runners tested on a motorized treadmill (\sim 59 mL \cdot kg $^{-1}$ \cdot min $^{-1}$), but somewhat lower than that reported by Koppo et al. (2004a) for elite cyclists performing cycle ergometer exercise (\sim 67 mL \cdot kg $^{-1}$ \cdot min $^{-1}$). The SPT athletes had relative $\dot{V}O_{2 \text{ peak}}$ values of approximately 47 mL \cdot kg $^{-1}$ \cdot min $^{-1}$, slightly higher than those previously reported for age-matched subjects who were not specifically trained (i.e., 41–45 mL \cdot kg $^{-1}$ \cdot min $^{-1}$; Jones and Carter 2004; Koppo et al. 2004a), but somewhat lower than those reported previously by Fukuoka et al. (1995) for American football players (53 mL \cdot kg $^{-1}$ \cdot min $^{-1}$) and by Granier et al. (1995) for track sprint runners (52 mL \cdot kg $^{-1}$ \cdot min $^{-1}$). The % $\dot{V}O_{2 \text{ peak}}$ at the estimated LT was lower in both our SPT and ENT athletes than has been reported in earlier studies (Schneider et al. 1990; Fukuoka et al. 1995). The cause of these differences is not clear, but they might be related to differences in the protocols and procedures used for the measurement of $\dot{V}O_{2 \text{ peak}}$ or estimation of LT, or to differences in the relative training status of the subjects. Our SPT subject cohort predominantly comprised track and field athletes who trained exclusively for these events and performed minimal aerobic exercise training. In this respect, it is especially noteworthy that the SPT athletes recorded high peak power output values on the Wingate test that were similar to previously reported values for other athletes who compete in sprint and power sports (Heller et al. 1998; Jones et al. 1999). The SPT athletes achieved Wingate peak power outputs that were 3.4 \times greater than the peak power output attained during the incremental test (mean values of 1187 and 343 W), considerably greater than the value of 2.2 in the ENT athletes (mean values of 937 and 434 W).

Phase II time constant

To our knowledge, this is the first study to examine $\dot{V}O_2$ kinetics in young highly trained sprint athletes during step cycle exercise. Breath-by-breath measurements of O $_2$ uptake at the mouth possess inherent variability, but the signal-to-noise ratio can be greatly increased by averaging together the responses to a suitable number of like transitions (Lamarra et al. 1987). In the present study, the subjects completed 4 repeat transitions to moderate exercise and 2 repeat transitions to severe exercise, resulting in acceptable 95% confidence intervals for the estimation of the phase II τ (1–3 s for ENT and 3–4 s for SPT).

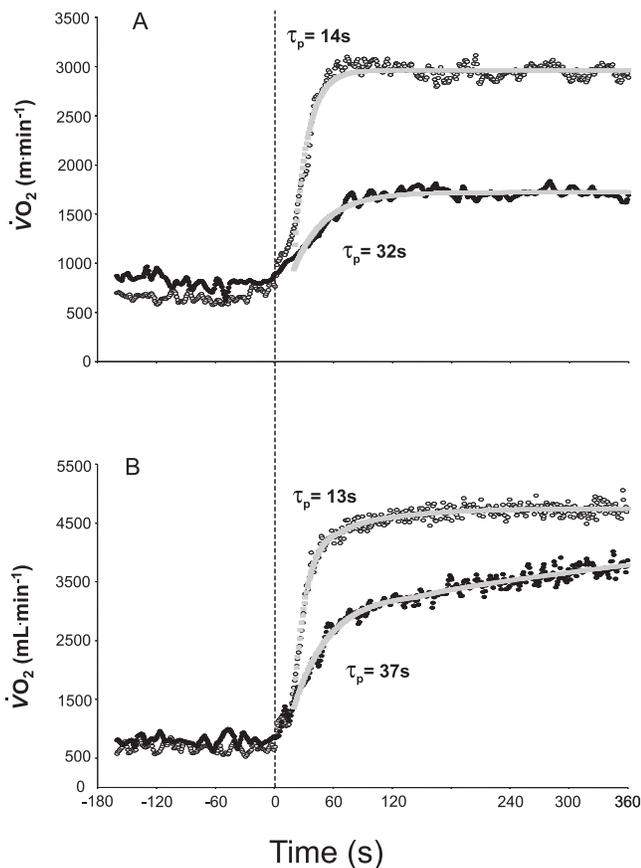
In the present study, the ENT subjects had phase II τ val-

Table 3. Mean (\pm SD) parameters of the $\dot{V}O_2$ kinetic response to severe-intensity exercise in sprint- and endurance-trained subjects.

	Sprint trained	Endurance trained
Δ Work rate (W)	248 (\pm 29)	335 (\pm 41)**
Δ Blood [lactate] (mmol/L)	5.7 (\pm 1.6)	5.8 (\pm 1.1)**
Baseline $\dot{V}O_2$ (L \cdot min $^{-1}$)	0.86 (\pm 0.15)	0.81 (\pm 0.07)
Time delay (s)	11 (\pm 4)	11 (\pm 5)
Time constant (s)	32 (\pm 10)	20 (\pm 6)*
Primary amplitude (L \cdot min $^{-1}$)	2.28 (\pm 0.30)	3.04 (\pm 0.39)**
Gain (mL \cdot min $^{-1}$ \cdot W $^{-1}$)	9.2 (\pm 1.0)	9.1 (\pm 0.9)
$\dot{V}O_2$ SC time delay (s)	133 (\pm 28)	103 (\pm 43)
$\dot{V}O_2$ SC amplitude (L \cdot min $^{-1}$)	0.55 (\pm 0.12)	0.50 (\pm 0.22)
Relative $\dot{V}O_2$ SC (%)	20 (\pm 5)	14 (\pm 7)
$\dot{V}O_2$ [6-3] (L \cdot min $^{-1}$)	0.37 (\pm 0.08)	0.20 (\pm 0.09)**
$\dot{V}O_2$ [6-2] (L \cdot min $^{-1}$)	0.58 (\pm 0.05)	0.37 (\pm 0.11)**

Note: *, significantly different at $p < 0.05$; **, significantly different at $p < 0.01$; $\dot{V}O_2$ SC = $\dot{V}O_2$ slow component.

Fig. 1. $\dot{V}O_2$ responses following the onset of moderate-intensity (A) and severe-intensity (B) exercise in a representative sprint-trained athlete (closed symbols) and a representative endurance-trained athlete (open symbols). Solid lines represent the model fits to the data. Notice the significantly slower $\dot{V}O_2$ response in phase II (as given by τ_p values) for both exercise intensities and the relatively larger $\dot{V}O_2$ slow component during severe exercise in the sprint-trained athlete.



ues of ~ 17 – 20 s. Although these values are considerably shorter (i.e., the kinetics are faster) than those reported for sedentary or recreationally active subjects (i.e., phase II τ

of 25–32 s for moderate-intensity exercise; e.g., Brittain et al. 2001; DeLorey et al. 2003; Koppo et al. 2004b; Roberts et al. 2005; Wilkerson et al. 2005; Berger et al. 2006a), they are somewhat longer than those that have been reported previously for highly trained endurance cyclists and runners (phase II τ of ~ 12 s; Koppo et al. 2004a; Kilding et al. 2005). This difference is likely a function of the higher fitness of the cyclists studied by Koppo et al. (2004a) and the use of treadmill as opposed to cycle ergometry by Kilding et al. (2005). The phase II τ in the SPT athletes was 32 s, on average, for both moderate- and severe-intensity exercise. It is of interest that despite having higher $\dot{V}O_{2\text{ peak}}$ values, the SPT athletes had phase II τ values that were somewhat larger than those reported for untrained or recreationally active subjects (Brittain et al. 2001; DeLorey et al. 2003; Pringle et al. 2003; Koppo et al. 2004b; Roberts et al. 2005; Wilkerson et al. 2005; Berger et al. 2006a).

The phase II τ was significantly correlated with indices of aerobic fitness and performance (i.e., $\dot{V}O_{2\text{ peak}}$, estimated LT, and the highest power output attained during the ramp incremental test; Fig. 2). This is consistent with several early studies reporting that higher aerobic fitness (as evidenced by $\dot{V}O_{2\text{ peak}}$) was associated with faster overall $\dot{V}O_2$ kinetics (Weltman and Katch 1976; Cerretelli et al. 1979; Powers et al. 1985) and that endurance training resulted in faster overall $\dot{V}O_2$ kinetics (Cerretelli et al. 1979; Hagberg et al. 1980). Higher levels of aerobic fitness are linked with improved cardiovascular function, greater muscle capillarity, and higher muscle oxidative capacity (Jones and Carter 2000), which likely improves the capacity for both muscle O_2 delivery and utilization across a metabolic transient.

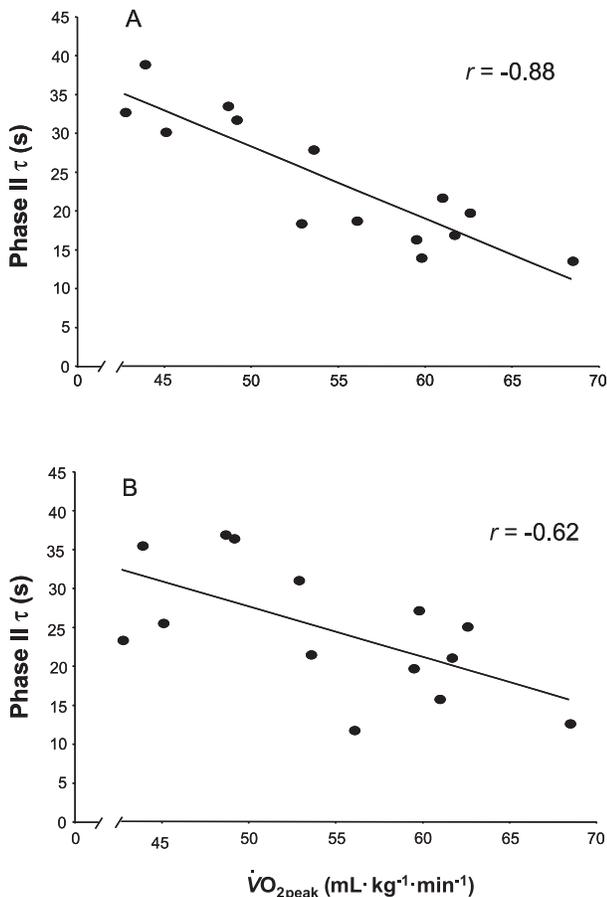
The significant difference in the phase II τ between the SPT and ENT subjects in the present study is in agreement with the earlier results of Edwards et al. (1999), who reported that endurance runners had significantly shorter phase shift components in the frequency domain when $\dot{V}O_2$ kinetics were measured using a PRBS exercise protocol. Similar results have been reported by other groups (Eßfeld et al. 1987; Fukuoka et al. 1995, 1997; Edwards et al. 2003). That $\dot{V}O_2$ kinetics might discriminate between athletes with different specialist events is also suggested by the results of

Table 4. Matrix of correlation coefficients between key parameters of the $\dot{V}O_2$ kinetics and selected indices of aerobic and anaerobic exercise performance.

	Phase II τ : moderate	Phase II τ : severe	Relative $\dot{V}O_2$ SC (%)
$\dot{V}O_{2\text{ peak}}$ (mL·kg ⁻¹ ·min ⁻¹)	-0.88**	-0.62*	-0.53*
Peak work rate (W·kg ⁻¹)	-0.85**	-0.58*	-0.55*
Estimated LT work rate (W·kg ⁻¹)	-0.80**	-0.68**	-0.46
Wingate peak power (W·kg ⁻¹)	0.56*	-0.05	0.77**
Wingate mean power (W·kg ⁻¹)	0.23	-0.26	0.60*
Wingate fatigue index (%)	0.75**	0.33	0.58*

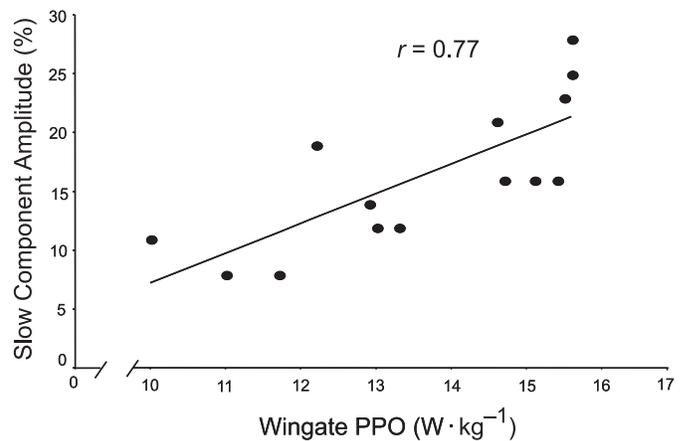
Note: *, significantly different at $p < 0.05$; **, significantly different at $p < 0.01$.

Fig. 2. Relationships between $\dot{V}O_{2\text{ peak}}$ (mL·kg⁻¹·min⁻¹) and the phase II time constant (τ) for moderate-intensity (A) and severe-intensity (B) exercise across all subjects. The correlation coefficients were significant at the 1% and 5% levels, respectively, for moderate- and severe-intensity exercise.



Kilding et al. (2006). These authors reported that the mean phase II τ during moderate-intensity treadmill running was ~12 s in long-distance runners and ~16 s in middle-distance runners. One possible explanation for these results is that ENT athletes have better cardiovascular responsiveness (i.e., faster cardiac output kinetics and more rapid vasodilatation) leading to improved delivery of O_2 to the contracting muscles compared with SPT athletes. There is evidence that muscle blood flow kinetics become faster as a consequence of endurance training (Shoemaker et al. 1996). Furthermore,

Fig. 3. Relationship between the peak power output attained during the Wingate anaerobic sprint test (W·kg⁻¹) and the relative contribution made by the $\dot{V}O_2$ slow component to the total increase in $\dot{V}O_2$ above baseline during severe-intensity exercise. The correlation coefficient was significant at the 1% level.



muscle capillary density is likely to be greater in the ENT than in the SPT athletes owing, in part, to the greater relative muscle hypertrophy in the SPT athletes (Tesch et al. 1984), which might facilitate a more homogeneous O_2 distribution within the muscles. However, although the putative determinant(s) of $\dot{V}O_2$ kinetics is (are) debated, it is generally agreed that peripheral (muscle metabolic) factors and not O_2 supply limit $\dot{V}O_2$ kinetics, at least during moderate-intensity exercise in most circumstances (Poole and Jones 2005; cf. Hughson 2005). In this regard, significant differences both in muscle fibre type distribution and in oxidative and non-oxidative metabolic enzyme activity would be expected between the two groups of athletes (Costill et al. 1976; Holloszy and Coyle 1984; Abernethy et al. 1990; Andersen et al. 1994; MacDougall et al. 1998; Ross and Leveritt 2001). In rodents, at least, muscles with a preponderance of type II fibres appear to have much slower $\dot{V}O_2$ kinetics than muscles with a high proportion of type I fibres (Crow and Kushmerick 1982; Jones et al. 2005), an effect that might be a function of blunted microvascular PO_2 profiles (Behnke et al. 2003) and (or) impaired ability to extract and utilize available O_2 (Kindig et al. 2003). It has been demonstrated that the kinetics of $\dot{V}O_2$ closely reflect the kinetics with which muscle [PCr] declines following the onset of exercise (Rossiter et al. 2002a, 2002b), suggesting that [PCr] or the products of PCr hydrolysis are important in the

regulation of oxidative phosphorylation (Whipp and Mahler 1980; Grassi 2005). The faster $\dot{V}O_2$ kinetics in ENT than in SPT athletes might therefore be explained by differences in [PCr] kinetics, the sensitivity of mitochondria to creatine or adenosine diphosphate, or the activity of one or more of the (potentially rate-limiting) oxidative enzymes (Whipp and Mahler 1980; Grassi 2005).

$\dot{V}O_2$ slow component

The magnitude of the $\dot{V}O_2$ slow component (as assessed using the $\dot{V}O_{2 [6-2]}$ or $\dot{V}O_{2 [6-3]}$ indices) was significantly greater in the SPT athletes than in the ENT athletes. This finding was in agreement with our second hypothesis. SPT athletes would be expected to have a high relative distribution of type II muscle fibres in the primary locomotory muscles compared with ENT athletes and the general population (Abernethy et al. 1990; Bouchard et al. 1992; Andersen et al. 1994; Ross and Leveritt 2001). Among other factors, a high proportion of type II muscle fibres should facilitate a greater, and more rapid, generation of muscle power, which would clearly be an advantage during short-term, supramaximal intensity exercise. However, type II fibres are generally considered less efficient (i.e., to have both a higher energy cost of force production and a higher O_2 cost of high-energy phosphate production) than type I fibres (Crow and Kushmerick 1982; Willis and Jackman 1994). For this reason, the recruitment of these higher-order fibres to meet the requirement for increased muscle power generation during exercise above LT has been proposed as a candidate mechanism for the $\dot{V}O_2$ slow component (Poole et al. 1994; Barstow et al. 1996; Jones et al. 2005). There is evidence that type II muscle fibres are active following the onset of high-intensity exercise, which elicits the $\dot{V}O_2$ slow component, and that these fibres continue to be recruited as exercise proceeds (Krustrup et al. 2004). The relative amplitude of the $\dot{V}O_2$ slow component during exercise above LT has been reported as significantly correlated with the proportion of type II muscle fibres in the quadriceps muscles (Barstow et al. 1996; Pringle et al. 2003). Moreover, a variety of other interventions (e.g., previous exercise, glycogen depletion, and cadence alterations) and measurement techniques (e.g., electromyogram, magnetic resonance imaging, and spectroscopy) have provided further indirect evidence for an important role for muscle fibre recruitment in the development of the $\dot{V}O_2$ slow component (Jones et al. 2005).

Several previous studies have shown that, for the same relative exercise intensity, the $\dot{V}O_2$ slow component is negatively related to aerobic fitness and can be attenuated by endurance training (Casaburi et al. 1987; Jones and Koppo 2005; Koppo et al. 2004a; Berger et al. 2006b). However, an interesting finding in the present study was that the relative amplitude of the $\dot{V}O_2$ slow component was much more strongly correlated with indices of anaerobic exercise performance than with indices of aerobic exercise performance. Specifically, the relative amplitude of the $\dot{V}O_2$ slow component was significantly correlated with peak power output (Fig. 3), mean power output, and fatigue index as measured during the Wingate anaerobic test. To the extent that various aspects of Wingate test performance have been related to the proportional distribution of type II fibres in the exercising

muscles (Esbjornsson et al. 1993; Mannion et al. 1995), these data lend support to the prevailing theory that the $\dot{V}O_2$ slow component is linked to the recruitment of type II muscle fibres. The present data are also consistent with the recent study of Garland et al. (2004), in which it was reported that the relative amplitude of the $\dot{V}O_2$ slow component during knee extension exercise was significantly greater in 6 power-trained athletes than in 6 endurance-trained athletes (8% vs. 3%, on average). In this study, the functional contractile profile and fatigability of the knee extensors assessed using an electrical stimulation protocol was consistent with the presence of a higher proportion of type II muscle fibres in the muscles of the power-trained athletes.

Potential limitations to the present study should be acknowledged. Firstly, although our subjects were serious athletes who had been committed to their sports for many years, they might be described as sub-elite rather than elite (i.e., they competed at national rather than international levels). The relative training status of the two cohorts might have been more effectively normalized if all the subjects had been elite. This might have led to differences in $\dot{V}O_2$ kinetics that were even more pronounced than those reported herein. Secondly, 5 of the 8 ENT athletes, but only 1 of the 8 SPT athletes, routinely performed cycle exercise training and it cannot be discounted that this influenced the results. A future study might therefore compare $\dot{V}O_2$ kinetics in elite ENT and SPT athletes who are either all cycling specialists or who are all non-cyclists.

Conclusions

In conclusion, sub-elite SPT athletes evidenced slower phase II $\dot{V}O_2$ kinetics and a greater $\dot{V}O_2$ slow component compared with sub-elite ENT athletes. The mean phase II τ of ~ 32 s in the SPT athletes was surprisingly long for young, healthy subjects involved in regular exercise training; indeed, it was slightly longer than the values normally reported in untrained or recreationally active young subjects with similar or lower $\dot{V}O_{2 \text{ peak}}$ values. The much slower phase II $\dot{V}O_2$ kinetics in the SPT compared with the ENT athletes is presumably a function of inherent (genetic) and (or) training-induced differences in muscle O_2 transport and utilization. The phase II τ was significantly correlated with indices of aerobic exercise performance, confirming the importance of aerobic fitness as an important determinant of the speed of the $\dot{V}O_2$ adjustment in the fundamental phase of the response. The relative amplitude of the $\dot{V}O_2$ slow component, on the other hand, was significantly correlated with indices of anaerobic exercise performance, namely the peak power output, the mean power output, and the fatigue index measured during the Wingate test. It appears, therefore, that indices of aerobic and anaerobic fitness can differentially influence the fundamental and slow components of the $\dot{V}O_2$ response following the onset of constant work-rate exercise.

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