Abstract The purpose of the present study was to examine comprehensively the kinetics of oxygen uptake ($\dot{V}O_2$) during treadmill running across the moderate, heavy and severe exercise intensity domains. Nine subjects [mean (SD age, 27 (7) years; mass, 69.8 (9.0) kg; maximum $\dot{V}O_2$, $\dot{V}O_{2\text{max}}$, 4,137 (697) ml·min$^{-1}$] performed a series of "square-wave" rest-to-exercise transitions of 6 min duration at running speeds equivalent to 80% and 100% of the $\dot{V}O_2$ at lactate threshold (LT; moderate exercise); and at 20%, 40%, 60%, 80% and 100% of the difference between the $\dot{V}O_2$ at LT and $\dot{V}O_{2\text{max}}$ ($\Delta$, heavy and severe exercise). Critical velocity (CV) was also determined using four maximal treadmill runs designed to result in exhaustion in 2–15 min. The $\dot{V}O_2$ response was modelled using non-linear regression techniques. As expected, the amplitude of the $\dot{V}O_2$ primary component increased with exercise intensity [from 1,868 (136) ml·min$^{-1}$ at 80% LT to 3,296 (218) ml·min$^{-1}$ at 100% $\Delta$, $P < 0.05$]. However, there was a non-significant trend for the "gain" of the primary component to decrease as exercise intensity increased [181 (7) ml·kg$^{-1}$·km$^{-1}$ at 80% LT to 160 (6) ml·kg$^{-1}$·km$^{-1}$ at 100% $\Delta$]. The time constant of the primary component was not different between supra-LT running speeds (mean value range = 17.9–19.1 s), but was significantly shorter during the 80% LT trial [12.7 (1.4) s, $P < 0.05$]. The $\dot{V}O_2$ slow component increased with exercise intensity from 139 (39) ml·min$^{-1}$ at 20% $\Delta$ to 487 (57) ml·min$^{-1}$ at 80% $\Delta$ ($P < 0.05$), but decreased to 317 (84) ml·min$^{-1}$ during the 100% $\Delta$ trial ($P < 0.05$). During both the 80% $\Delta$ and 100% $\Delta$ trials, the $\dot{V}O_2$ at the end of exercise reached $\dot{V}O_{2\text{max}}$ [4,152 (242) ml·min$^{-1}$ and 4,154 (114) ml·min$^{-1}$, respectively]. Our results suggest that the "gain" of the primary component is not constant as exercise intensity increases across the moderate, heavy and severe domains of treadmill running. These intensity-dependent changes in the amplitudes and kinetics of the $\dot{V}O_2$ response profiles may be associated with the changing patterns of muscle fibre recruitment that occur as exercise intensity increases.

Keywords Running exercise · Oxygen consumption slow component · Oxygen consumption primary component

Introduction

The pulmonary oxygen uptake (\(\dot{V}O_2\)) response in the transition from rest to constant-load exercise has been well described for cycling exercise. During moderate-intensity exercise (i.e. below the lactate threshold, LT), following the cardiodynamic phase (phase 1), rises in an approximately monoexponential fashion (phase 2) to attain a new steady state (phase 3) within 2–3 min. Previous research in cycling exercise has shown the amplitude of the primary component of \([A_{c+p}]\); i.e. the combined amplitudes of phase 1 (cardiodynamic phase, \(A_c\)) and phase 2 (primary phase, \(A_p\)) to be linearly related to exercise intensity. The primary “gain” (\(A_{c+p}\)/power output) therefore remains essentially constant over a broad range of submaximal power outputs, with values of \(\leq 10\) ml·min$^{-1}$·W$^{-1}$ commonly reported (Barstow and Mole 1991; Paterson and Whipp 1991). During heavy exercise above the LT, the kinetics are more complex and have been suggested to be dependent upon the proximity of the exercise intensity to the critical power (CP). CP is the asymptote of the hyperbolic power/time relationship, and therefore represents the
upper limit of sustained, tolerable work (Poole et al. 1988a). Between the LT and the CP, \( A_{e+p} \)' is supplemented by an additional slow component \( (A_{s}^p) \) that causes to rise for several minutes until a delayed steady state is achieved. This slow component causes to rise above the value expected from extrapolation of the sub-LT/power output relationship. In the severe exercise domain, that is above CP, a steady state in may not be attained and maximum (\( \dot{V} \)) may be reached if exercise is continued (Poole et al. 1988a).

Controversy exists over whether the time constant for the primary component (\( \tau_p \)) is slower (Paterson and Whipp 1991) or is unchanged (Barstow and Mole 1991) for exercise above, as compared to below the LT. The accurate estimation of \( \tau_p \) is of great importance in understanding the control of the on-kinetic response. A slower \( \tau_p \) in heavy exercise has been interpreted to indicate a reduced oxygen availability (MacDonald et al. 1997; Paterson and Whipp 1991). In contrast, an invariant \( \tau_p \) supports the notion that oxygen delivery is not limiting during heavy exercise and that muscle kinetics are controlled by intracellular processes (Grassi 2000; Grassi et al. 1998).

To date, research in this area has focussed almost exclusively on cycle ergometry. Recent research from our laboratory has shown a tendency for \( \tau_p \) to be slower in heavy- than in moderate-intensity treadmill running, although the difference did not reach statistical significance (Carter et al. 2000a, b). Previous studies reporting differences in \( \tau_p \) between moderate and heavy exercise have selected relatively few exercise intensities, typically examining the response at 80–90% LT and halfway between the LT and (Barstow et al. 1996; Engelen et al. 1996; Paterson and Whipp 1991). Furthermore, none of these studies measured CP, the parameter that demarcates the transition between the heavy and severe exercise intensity domains (Poole et al. 1988a). This may have implications for the interpretation of \( \tau_p \) since kinetics differ markedly in these two domains (Poole et al. 1988a).

The purpose of this study was to describe comprehensively kinetics throughout the moderate, heavy and severe exercise intensity domains during treadmill running. Based upon previous studies, we hypothesised that \( \tau_p \) would be shorter during exercise at a sub-LT intensity than at a supra-LT intensity, that the gain \( (G_{c+p}) \) of the primary component (calculated in this study as per kg body mass per unit of distance run; i.e. ml kg
\(-1\) km
\(-1\)) would be constant across the range of exercise intensities, and that the amplitude of the slow component would increase as exercise intensity increased above the LT.

Methods

Subjects

Nine recreationally active subjects [six men, mean (SD): age, 27 (7) years; mass, 69.8 (9.0) kg; , 4,137 (697) ml min
\(-1\)] volunteered to take part in this study, which was approved by the Institution Ethics Committee. The subjects gave written informed consent to participate, after the experimental procedures, the associated risks and the benefits of participation were explained. The subjects were all fully familiar with laboratory exercise testing procedures.

The subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h post-prandial, and to avoid strenuous exercise in the 48 h preceding a test session. For each subject, tests took place at the same time of day (± 2 h) to minimise the effects of diurnal biological variation on the results.

Experimental design

The subjects were required to visit the laboratory for three stages of experimentation. The first stage involved the determination of LT and . The second stage involved seven laboratory sessions, the subjects performing 2–3 repetitions of “square-wave” transitions from rest to one of seven exercise intensities: 80% LT, 100% LT, 20% Δ (20% of the difference in between that at LT and ), 40% Δ, 60% Δ, 80% Δ, and 100% Δ. No more than two transitions were completed in 1 day, with at least 1 h of recovery in between. The third stage involved determination of the subjects' critical velocity (CV, being analogous to CP in cycling exercise) from four treadmill runs to exhaustion (on separate days). The square-wave transitions and the exercise bouts to determine CV were performed in random order. The study was completed within 3–4 weeks for all subjects.

Procedures

All tests were performed on a motorised treadmill (Woodway, CardioKinetics, Salford, UK) with the grade set at 1% (Jones and Doust 1996b). During the exercise tests, pulmonary gas exchange was determined breath-by-breath. Subjects breathed through a low-dead-space (90 ml), low-resistance (0.65 mmH2O l–1) at 8 l s–1 mouthpiece and turbine assembly. Gases were drawn continuously from the mouthpiece through a 2-m capillary line of small bore (0.5 mm) at a rate of 60 ml min–1, and analysed for oxygen, carbon dioxide and nitrogen concentrations by a quadrupole mass spectrometer (CaSE QP9000, Gillingham, Kent, UK), which was calibrated before each test using gases of known concentration. Expiratory volumes were determined using a turbine volume transducer (Interface Associates, California, USA). The sampling rate for both gas flow and gas concentration was 50 Hz. The volume and concentration signals were integrated by computer, following analog-to-digital conversion, with account taken of the gas transit delay through the capillary. Respiratory gas exchange variables (carbon dioxide output and pulmonary ventilation) were calculated and displayed for every breath. Heart rate was recorded telemetrically throughout the exercise tests (Polar Electro Oy, Kempele, Finland).

Subjects performed incremental exercise to volitional exhaustion in order to determine LT. The initial running speed was 6.0–7.0 km h–1 for the female subjects and 8.0–9.0 km h–1 for the male subjects. Subjects completed 6–8 submaximal stages of 4 min duration with running speed increased by 1.0 km h–1 between stages (Jones and Doust 1997). At the end of each stage, subjects supported their weight with their hands and moved their feet to the sides of the treadmill belt. Finger tip capillary blood samples (≈25 μl) were collected in capillary tubes and subsequently analysed for lactate concentration ([La]) using an automated analyser (YSI 2300, Yellow Springs, Ohio, USA). Subjects recommenced running within 10–15 s. When blood [La] exceeded 4 mM, or heart rate exceeded 90% of the known or age-predicted maximum heart rate, the treadmill gradient was increased by 1% every minute until the subject reached volitional exhaustion. In a previous study (Jones and Doust 1996a), we demonstrated that the measured following a 25-min LT determination was not significantly different from that measured with a conventional 10-min fast-ramp protocol.

Plots of blood [La] against running speed and were provided to two independent reviewers who determined the LT as the first