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Abstract: This study compared V*O2, heart rate (HR) and electromyographic (iEMG) responses to speeds above the velocity associated with V*O2 max (v-V*O2 max). Eight male, middle-distance runners performed a graded exercise test to determine V*O2 max and v-V*O2 max and runs to fatigue at 100% and 110% v-V*O2 max. Breath-by-breath V*O2 and HR were continuously recorded; lactate [La-] measured pre and post-run and iEMG measures of Rectus Femoris (RF) and Vastus Lateralis were recorded during the first and last 20 s of each run. Analysis indicated longer time to fatigue in the 100% v-V*O2 max run with no differences between conditions for V*O2 or HR amplitudes or post-run [La-] (p>0.05). There were significantly faster tau values (p<0.05) in the 110% condition in V*O2 and HR. No significant correlations were observed between V*O2 or HR tau values and time to fatigue. RF iEMG was significantly larger in 110% compared to 100% run in the first 20 s(p<0.05). While no association between treadmill performance and V*O2 response was evident, faster running speeds resulted in faster V*O2 and HR responses, with no difference in amplitude or % V*O2max attained. This may potentially be as a result of an increased muscle fibre recruitment stimulus during the faster running velocity resulting in faster cardio-dynamic responses.

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Title: **$\dot{V}O_2$ responses to running speeds above $\dot{V}O_{2\ max}$.**

Abstract:

This study compared $\dot{V}O_2$, heart rate (HR) and electromyographic (iEMG) responses to speeds above the velocity associated with $\dot{V}O_{2\ max}$ (v-$\dot{V}O_{2\ max}$). Eight male, middle-distance runners performed a graded exercise test to determine $\dot{V}O_2$ max and v-$\dot{V}O_2$ max and runs to fatigue at 100% and 110% v-$\dot{V}O_{2\ max}$. Breath-by-breath $\dot{V}O_2$ and HR were continuously recorded; lactate [La^-] measured pre and post-run and iEMG measures of Rectus Femoris (RF) and Vastus Lateralis were recorded during the first and last 20 s of each run. Analysis indicated longer time to fatigue in the 100% v-$\dot{V}O_{2\ max}$ run with no differences between conditions for $\dot{V}O_2$ or HR amplitudes or post-run [La^-] ($p>0.05$). There were significantly faster tau values ($p<0.05$) in the 110% condition in $\dot{V}O_2$ and HR. No significant correlations were observed between $\dot{V}O_2$ or HR tau values and time to fatigue. RF iEMG was significantly larger in 110% compared to 100% run in the first 20 s($p<0.05$). While no association between treadmill performance and $\dot{V}O_2$ response was evident, faster running speeds resulted in faster $\dot{V}O_2$ and HR responses, with no difference in amplitude or % $\dot{V}O_{2max}$ attained. This may potentially be as a result of an increased muscle fibre recruitment stimulus during the faster running velocity resulting in faster cardio-dynamic responses.

Key words: VO_2 kinetics, Severe intensity, Athletics, Running Performance

Introduction:

Previous research has highlighted the role of aerobic metabolism (as measured by $\dot{V}O_2$) during middle-distance running events [15, 29]. A dominant proportion of energy is supplied by aerobic metabolism for events of 800-m and longer, with a dominance within 50 s of event commencement [12]. Along with this dominance of the percentage aerobic energy contribution, previous research has outlined the rise in $\dot{V}O_2$ to above 90% of $\dot{V}O_{2\,max}$ during events as short as 2 min (800-m) [9,30] and even higher for longer middle-distance events [10,29]. Further, recent research has also reported an association between race performance and both in-race $\%\dot{V}O_{2\,peak}$ and the speed of the $\dot{V}O_2$ response during on-track, middle-distance trials [10]. However, given the short duration of some events (2 – 4 min) and high running speeds (at or above $\dot{V}O_{2\,max}$), a limiting factor for race performance may potentially relate to the speed at which $\dot{V}O_{2\,peak}$ can be attained. Whether a faster $\dot{V}O_2$ response to these exercise intensities results in an improved race performance is unclear and further, whether faster speeds alter the speed of the $\dot{V}O_2$ response is also the topic of some debate [6].

The study of the speed of the $\dot{V}O_2$ response ($\dot{V}O_2$ kinetics) to square-wave transition exercise has primarily focussed on exercise intensities below $\dot{V}O_{2\,max}$ [5]; however, a renewed focus on exercise intensities around $\dot{V}O_{2\,max}$ has recently occurred [14,16]. Research has reported mixed findings regarding the characteristics of the $\dot{V}O_2$ response to severe exercise intensities at or above intensities associated with $\dot{V}O_{2\,max}$. To date, there are conflicting research findings on the modelling of the speed of the $\dot{V}$

O_2 response, or tau (τ), to severe exercise intensities with literature indicating that increases in exercise intensity near $\dot{V}O_{2\,max}$ result in no difference [28], a speeding [6] or a slowing [20] in the speed (τ) of the $\dot{V}O_2$ response. Hughson et al. [18] also reported faster τ values for higher exercise intensities but concluded that this did not indicate a faster $\dot{V}O_2$ response, as slower responses for higher intensities were reported when the data was modelled with a semi-logarithmic method. This method was used due to concerns regarding the curve fitting properties of exponential modelling for $\dot{V}O_2$ responses at severe exercise intensities. Whether the $\dot{V}O_2$ response is speeded as exercise intensities increase further above $\dot{V}O_{2\,max}$ is unclear. Further, as limited research has investigated the possible practical applications of these data to maximal exercise performance, this area is yet to be explored.

Despite the interest in the characteristics of the $\dot{V}O_2$ response to high-intensity exercise, few studies have investigated the relationship between the $\dot{V}O_2$ response and exercise performance in maximal aerobic events. Previously, we have reported an association between faster $\dot{V}O_2$ kinetics (τ) and both faster starting speeds and faster subsequent race performance in trained middle-distance runners during simulated on-track 1500-m trials, and near significance for 800-m trials [11]. However, only a single trial for each subject at each distance was analysed and the races were not constant exercise intensities and as such did not conform to the first order assumptions when modelling $\dot{V}O_2$ kinetics.

Therefore, the aim of this study was to determine whether faster running speeds were associated with faster $\dot{V}O_2$ kinetics in middle-distance runners at running speeds

encountered in middle-distance events. It was hypothesised that faster running speeds would promote a faster physiological response, as evidenced by faster τ values at these higher running speeds.

Methods:

Participants:

Eight trained middle-distance runners (mean ± SD: age 20 ± 1 y, mass 70.1 ± 7.8 kg, $\dot{V}O_{2\ max}$ 60.6 ± 2.6 $mL \cdot kg^{-1} \cdot min^{-1}$) volunteered to participate in this study. Participants were informed of the study requirements, benefits and risks before giving written informed consent. Approval for the study's procedures was granted by the Institutional Research Ethics Committee.

Experimental overview:

All participants were required to perform five testing sessions at the same time of day, each separated by at least 48 h and by no more than 72 h. Participants were in a rested state, and were required to record and maintain a similar dietary pattern, to not consume any food or drink apart from water in the 3 h prior to testing, and to avoid alcohol in the 24 h prior to testing. During the first testing session, participants performed a graded exercise test (GXT) consisting of continual increments in treadmill velocity every minute until volitional exhaustion was reached to determine $\dot{V}O_{2\ max}$ and the velocity associated with $\dot{V}O_{2\ max}$ (v-$\dot{V}O_{2\ max}$). The remaining testing sessions involved repeated sessions of a constant-intensity run to fatigue (RTF) at 100% and 110% v-$\dot{V}O_{2\ max}$ respectively (two trials at each speed). These sessions were randomised in a counter-balanced order to avoid any learning or practice effects.

During all sessions, pulmonary gas exchange was measured breath-by-breath by determining O_2 and CO_2 concentrations and ventilation to calculate $\dot{V}O_2$ using a metabolic gas analysis system (ParvoMedics True2400, Utah, USA). The gas analysers were calibrated immediately before and verified after each test using a certified gravimetrically determined gas mixture, while the ventilometer was calibrated pre-exercise and verified post-exercise using a three litre syringe in accordance with the manufacturer's instructions. Heart rate (Polar, Kempe, Finland) was continuously monitored throughout all RTF sessions with a heart rate value recorded for each breath-by-breath data value recorded and electromyographic (EMG) data was recorded from the Rectus femoris (RF) and Vastus lateralis (VL) muscles of the lower limb during the first and last 20 s of each RTF.

Graded Exercise Test

The GXT was performed on a motorised treadmill (h/p/cosmos quasar, Nusseldorf, Germany) commencing at a starting speed of 8 $km \cdot h^{-1}$ followed by continuous increments of 1 $km \cdot h^{-1}$ every min until volitional exhaustion. The test was stopped when the participant could no longer maintain the required treadmill velocity. Strong verbal encouragement was provided to each participant as they came to the end of the test. Expired gases were analysed continuously throughout the duration of the GXT. $\dot{V}O_{2\,max}$ was determined as the highest 30-s rolling average during the GXT and the treadmill speed at which $\dot{V}O_{2\,max}$ occurred (v-$\dot{V}O_{2\,max}$) were accordingly used in the RTF.

Run to Fatigue Test

Participants initially performed a standardised warm up consisting of a 5-min run at 10 $km \cdot h^{-1}$, followed by 5 min of stretching. Following the warm up, a 5-min resting baseline $\dot{V}O_2$ measure was obtained while subjects were stationary on the treadmill, before commencing the square-wave transition run (100% or 110% v-$\dot{V}O_{2\,max}$). Subjects performed the RTF until they could no longer maintain the required treadmill velocity. Strong verbal encouragement was provided to each participant as they came to the end of the test. Through out the baseline period and RTF, $\dot{V}O_2$ was measured on a breath-by-breath basis, while capillary blood lactate concentration [La^-] (Lactate Pro, Arkray, Kyoto, Japan) was measured prior to and following each RTF from a hyperaemic earlobe (Finalgon, Boehringer Ingelheim, Germany). During each RTF, heart rate was continuously recorded via a receiver linked to the metabolic system. On each RTF, the run duration was timed for each participant from the moment they released their grip on the treadmill railing (after stepping onto the moving belt at the required speed) until volitional exhaustion (T_{fat}).

To compare the initial muscle recruitment and change over the duration of the respective RTF, electomyographic (EMG) data from the RF and VL muscles was measured during the first and last 20 s of each RTF (Bagnoli 4, DelSys Inc, Boston, Mass., USA). Prior to the warm up on each RTF trial, double differential EMG electrodes with a bar configuration (1 × 10 mm) and bandwidth of 20 – 450 Hz (DelSys Inc, Boston, Mass., USA) were attached to the RF and VL of the right leg and a Ag/AgCl reference electrode was attached to the spine of the right scapula. These muscles were chosen due their importance and high level of recruitment in high-speed running [22]. The location of each electrode was determined by locating

the belly of the muscle while avoiding micro-innervation zones of the lower quadriceps. Each location was marked on the skin and subjects were required to ensure the mark was maintained for each ensuing test to ensure identical placement of the electrode for each muscle between trials. EMG electrodes and the reference electrode were connected to a signal acquisition apparatus (Bagnoli 4, DelSyss Inc, Boston, Mass, USA) and host computer with raw data sampled at 2048 Hz for all tests.

Data Calculation

Transition times from stationary to v-$\dot{V}O_{2\,max}$ intensity running took 2 – 4 s. Following removal of outliers (± 3 SD) to exclude errant breaths, breath-by-breath $\dot{V}O_2$ data were interpolated to give 1-s values and smoothed using rolling 5-s averages to enhance the underlying $\dot{V}O_2$ response characteristics. For both exercise intensities (100% and 110% respectively) the smoothed $\dot{V}O_2$ data was time aligned for the two respective trials and collapsed into a single $\dot{V}O_2$ response (for each participant). As HR measures are recorded in intervals and averaged against the previous interval measurement and displayed as beats per minute, this essentially acts as a smoothing process and as such, no further smoothing function was performed. A mono-exponential model [24] was fit to the $\dot{V}O_2$ and HR response data respectively using the following iterative, non-linear regression (customised, Microsoft Excel). As comparisons were made between the responses to the respective speeds, the same model was required to be fit to both sets of data. As a minimal slow-component is evident for the 110% RTF and Özyener [24] report that at supra-maximal speeds the

VO_2 response reverts to a mono-exponential curve, a single exponential function was chosen to model the data.

$$VO_2(t) = VO_2(b) + A_1 \cdot (1- e^{-(t - td1)/\tau 1})$$

Note: $VO_2(t)$ represents the VO_2 at any given time; $VO_2(b)$ is the VO_2 baseline value; A_1 is the asymptotic amplitude; τ_1 is the time constant and td_1 is the time delay.

For the assessment of EMG activity, raw EMG signals were full wave rectified and movement artefact removed using a high-pass second order Butterworth filter with a cut-off frequency of 15 Hz; then smoothed using a low-pass second order Butterworth filter with a cut-off frequency of 5 Hz. Subsequently, EMG data was quantified as an integrated EMG signal (iEMG) by calculating the average between each data point and multiplying the total sum of these averages by the time frame of collection (EMGworks, version 2.0 DelSys Inc, Boston, Mass., USA). For data analysis, iEMG data collected will be reported as both the smoothed data from the first 20 s, while the change over time will be reported as a percentage change of the final value based on the initial and final 10 s.

Statistical Analysis

A one-way, repeated-measures ANOVA with Tukey's post-hoc tests was used to determine any significant differences in the measured variables between each RTF speed. Relationships between VO_2 response parameters (particularly τ) and run to fatigue performance were analysed by way of Pearson product-moment correlations. The alpha level for statistical significance was set at 0.05. All results are reported as mean ± SD unless otherwise stated. Data analysis was performed with the statistical software package SPSS v14.0 (Illinois, USA)

Results:

Results for measures of $\dot{V}O_2$, HR, [La^-], iEMG and T_{fat} for the 100% and 110% v-$\dot{V}O_{2\,max}$ RTF are presented in Table 1. The average velocities for the 100% and 110% v-$\dot{V}O_{2\,max}$ RTF were 17.7 ± 0.8 and 19.4 ± 0.8 $km \cdot h^{-1}$ respectively. Time to fatigue was significantly longer in the 100% v-$\dot{V}O_{2\,max}$ run ($p<0.01$). Significant differences were present ($p<0.05$) between 100% and 110% v-$\dot{V}O_{2\,max}$ for τ values in both $\dot{V}O_2$ and HR and for the gain of the $\dot{V}O_2$ primary amplitude, all of which were lower in the 110% v-$\dot{V}O_{2\,max}$ condition. However, no significant differences were present for the baseline, primary amplitude, total amplitude or %max reached for either $\dot{V}O_2$ or HR data ($p>0.05$) at T_{fat}. Further, no significant differences were present between exercise intensities for post-run [La^-] ($p>0.05$). Smoothed iEMG data showed a significant difference ($p<0.05$) in the first 20 s between 110% and 100% v-$\dot{V}O_{2max}$ for RF (0.183 ± 0.061 v 0.090 ± 0.051 mV), however not for VL (0.100 ± 0.043 v 0.071 ± 0.058 mV; $P>0.05$). Further, the increase in RF iEMG as the run progressed when comparing the initial to final 10 s occurred at both speeds. There was a trend ($p=0.09$) for this pre to post-run iEMG increase to be greater in the 110% compared to 100% run. No significant correlations ($p>0.05$) were observed between T_{fat} and $\dot{V}O_2$ or HR τ values ($r = -0.50$ and -0.10 for 100% and $r = -0.41$ and -0.09 for 110%). The $\dot{V}O_2$, HR and iEMG responses to 100% and 110% v-$\dot{V}O_{2\,max}$ for representative subjects are presented in Figures 1, 2 and 3 respectively.

TABLE 1 AND FIGURE 1, 2 AND 3 TO GO NEAR HERE.

Discussion:

The aim of the current study was to determine whether faster running speeds above v-$\dot{V}O_{2max}$ were associated with faster $\dot{V}O_2$ responses. In accordance with the hypothesis, faster running speeds during constant-speed treadmill runs above v-$\dot{V}O_{2max}$ were associated with a speeding of the $\dot{V}O_2$ response (smaller τ values). Further, faster heart rate responses, and no differences in the amplitude or %max of either $\dot{V}O_2$ or HR, were evident between the respective speeds. In contrast to the hypothesis, the correlations between the speeds of the respective $\dot{V}O_2$ and HR responses (τ) and T_{fat} for either run were not-significant, indicating no association with treadmill run to fatigue performance. Finally, greater iEMG activity was recorded in the RF but not the VL during the faster run velocity.

Research comparing the $\dot{V}O_2$ kinetics of exercise intensities associated with $\dot{V}O_{2\,max}$ has reported inconsistent results. However, the present results are consistent with several studies that have reported faster τ values as exercise intensities increase above $\dot{V}O_{2\,max}$ [2,6,8]. While Scheuermann and Barstow [28] reported no significant difference between τ values at 100 and 120% $\dot{V}O_{2\,peak}$, their data showed similar trends to both Carter et al. [6] and the current study; however, significance may not have been achieved due to heterogeneity of the subject population (high SD values for τ values), combined with a significantly greater amplitude at the faster speed. In addition, the $\dot{V}O_{2\,max}$ values for subjects in the Scheuermann and Barstow [28] study were much lower than for the subjects in those studies reporting a speeding of $\dot{V}O_2$ kinetics with an increase in intensity above $\dot{V}O_{2\,max}$ [6,8]. Within the current study, the participants were trained endurance runners, with homogenous τ values, allowing

detection of significantly faster τ values for the higher exercise intensity above $\dot{V}O_{2max}$.

iEMG activity recorded from RF and VL muscles during the first 20 s indicated a significantly greater activity in RF at 110% compared to 100% v-$\dot{V}O_{2max}$, with no difference for VL. Further, a trend for a greater increase in RF activity during the faster running speeds was also evident. Previous iEMG data for high-speed running has reported an increased EMG activity with faster running velocities in the two-joint muscles, specifically muscles such as RF, possibly resulting from an increased firing rate or increased muscle fibre recruitment [22]. While data from the current study only incorporates measures recorded at two specific muscle sites, the assumption is that a larger whole body muscle recruitment is occurring during the faster running velocity. If a larger active muscle mass is recruited at higher exercise intensities, as indicated by RF values in the current study and previously by Kryolainen et al. [22], it is likely there will be a resultant increase in the O_2 requirement. While faster $\dot{V}O_2$ kinetics can not be directly attributed to a greater muscle mass involvement in the present study, it is possible that the extent of the active musculature provides feedback to regulate and adjust the speed and magnitude of the cardio-dynamic response to exercise [6]. Accordingly, a larger muscle recruitment stimulus may invoke the faster cardio-dynamic responses noted in the present study. Further, the less than maximal iEMG activity at $\dot{V}O_{2max}$, combined with larger iEMG activity in faster speeds with no difference in [La^-] accumulation, may indicate that skeletal muscle recruitment is not maximal at $\dot{V}O_{2max}$. Consequently, this may indicate a role for centrally mediated, rather than peripheral regulation of exercise performance in this exercise intensity domain.

Potentially linked to this muscle recruitment explanation is that the rate of increase in $\dot{V}O_2$ to a given (maximal) amplitude was faster and earlier in the 110% run due to the previously demonstrated link between faster PCr degradation and faster $\dot{V}O_2$ kinetics [26]. Rossiter et al. [26] have reported a temporal alignment between the respective speeds of PCr degradation and muscle $\dot{V}O_2$ kinetics. As such, if a greater or faster recruitment promotes a faster PCr degradation, it is feasible that the metabolic feedback provided by the 110% condition results in faster kinetics of oxidative metabolism and allows for the provision of a greater oxidative involvement during the early stages of maximal aerobic exercise. This result however, must be tempered by the observation that the gain ($\Delta\dot{V}O_2/\Delta$velocity) was greater for the lower exercise intensity, consistent with the results of Carter et al. [6], indicating that a larger rise in $\dot{V}O_2$ occurred relative to the increase in velocity at the lower speed, or that the relationship between muscle recruitment and $\dot{V}O_2$ is not linear.

Heart rate responses indicated a significantly faster attainment of the peak amplitude for heart rate in the higher-intensity run, similar to the $\dot{V}O_2$ response previously detailed. Hughson and Morrissey [19] have previously reported slower τ values for HR responses with increases in sub-maximal exercise intensity (40 to 80% of anaerobic threshold), similar to their previously reported $\dot{V}O_2$ kinetics data [18]. They suggested that this association between changes in HR and $\dot{V}O_2$ kinetics reflected the importance of a delivery mechanism for the regulation of the primary $\dot{V}O_2$ response. This premise is further supported by later work of Hughson et al. [17] who demonstrated that manipulation of muscle blood flow, and therefore adjustment of O_2

delivery, altered the $\dot{V}O_2$ response. In contrast, Grassi et al. [13] argue that a delivery mechanism is not supported by Near-infrared spectroscopy measures and that the "metabolic inertia" at the commencement of exercise was due to the intrinsic slowness of peripheral aerobic metabolism. This in turn is supported by Knight et al. [21] who showed no slowing of the $\dot{V}O_2$ response of occluded limbs, indicating that reduced skeletal blood flow was not a direct cause of changes in $\dot{V}O_2$ kinetics. However, the influence of exercise intensity needs to be considered when inferring from these contrasting theories for the current data, as this previous research has been conducted at sub-maximal intensities. For higher intensities, there has been little research on the association between O_2 delivery and the $\dot{V}O_2$ response. It has previously been established however, that increasing O_2 delivery can increase $\dot{V}O_{2\,max}$ [25]. Hence, while the mechanisms limiting maximal oxidative metabolism are unlikely to be solely related to delivery or utilisation respectively, the faster HR responses in the 110% v-$\dot{V}O_{2\,max}$ condition suggests a possible role of increased delivery or availability of O_2 to promote faster $\dot{V}O_2$ kinetics during exercise above v-$\dot{V}O_{2\,max}$. Alternatively, increases in muscle fibre recruitment may stimulate increases in both $\dot{V}O_2$ and HR kinetics.

The speeding of the $\dot{V}O_2$ response with higher exercise intensities may be of benefit for performance in maximal aerobic exercise such as middle-distance running. While previous studies have not shown altering pacing strategy to have any effect on $\dot{V}O_2$, these studies either did not measure $\dot{V}O_2$ kinetics or focussed on end-race $\dot{V}O_{2\,peak}$ [1,3,23]. Bishop et al. [4] have reported higher $\dot{V}O_2$ values at 30 and 45 s (without measuring kinetics) for higher-speed all-out starts when compared to even-pacing

strategies on a kayak ergometer. Similar results were reported by Sandals et al. [27], where a faster acceleration phase at the start of a simulated treadmill 800-m trial promoted a larger $\dot{V}O_2$ earlier in the effort (no kinetics measured). They also hypothesised that a larger total muscle or type II recruitment imposed a larger O_2 demand to promote the greater $\dot{V}O_2$ attained. As such, there is some support for the notion of faster running velocities at the commencement of an exercise bout promoting faster $\dot{V}O_2$ adaptations as suggested by the current study.

To date, little research has attempted to apply the measurement of $\dot{V}O_2$ kinetics in square-wave laboratory conditions to athletic performance. While the mechanisms regulating adjustments in τ with changes in exercise intensity require further investigation, particularly above $\dot{V}O_{2max}$, an association between faster $\dot{V}O_2$ responses (resulting from faster running speeds) and middle-distance track running performance has previously been reported [11]. While no relationships between $\dot{V}O_2$ response and treadmill run performance were noted in the present-square-wave conditions, race conditions allow athletes to adjust their pace to ensure completion of the race distance. From an applied perspective, if an initial, faster running speed promotes a faster $\dot{V}O_2$ response it may be of benefit in high-speed, variable paced, maximally-aerobic events such as middle-distance running where $\dot{V}O_2$ reaches a plateau. Data from Draper et al. [8], Carter et al. [6] and the current study showed no significant difference in the final $\dot{V}O_2$ amplitude for exercise intensities above $\dot{V}O_{2\,max}$. Bishop et al. [4] have previously shown that changes in pacing strategy do not affect total anaerobic contribution during maximal intensity exercise. Therefore, from a metabolic model standpoint, a greater aerobic response, combined with an unchanged

anaerobic contribution, will increase total metabolic ATP supply and may provide performance benefits. Possible race strategies may involve starting at (individually relative) faster velocities before settling into a more sustainable race pace so that peak $\dot{V}O_2$ is reached and maintained within an earlier time frame.

In conclusion, at running speeds above v-$\dot{V}O_{2\,max}$, a faster $\dot{V}O_2$ response was noted which was evident alongside faster heart rate kinetics. This occurred without any difference in the amplitude, percent of maximum $\dot{V}O_2$ reached or [La^-] accumulation in the higher running velocity. When placed in the context of previous track-based $\dot{V}O_2$ kinetics data, it may be beneficial for middle-distance athletes to commence track races at faster (individually relative) running speeds to promote a faster and earlier aerobic response, however this may be the case only if exercise performance is linked to oxygen supply.

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