

**PHYSIOLOGICAL RESPONSES AT VARIOUS LACTATE MARKERS FOR
RUNNING AT 4 AND 8 MINUTE TREADMILL INCREMENTS**

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ABSTRACT

We compared physiological responses corresponding to speeds at plasma lactate markers between incremental treadmill running of 4 and 8 min stages in fifteen healthy men (23 ± 4 yrs, 1.78 ± 0.49 m, 72.7 ± 10.8 kg). Treadmill speed, oxygen uptake ($\dot{V}O_2$), heart rate (HR), rating of perceived exertion (RPE) and plasma lactate were measured for each stage, and calculated at: fixed blood lactate accumulation (FBLA) 4.0 mmol/L, an initial 1 mmol/L rise, deviation maximum (D_{\max}), lactate threshold (LT) and log-log LT. There was no effect ($p > 0.05$) of stage duration on speed, $\dot{V}O_2$, HR and RPE at fixed markers. For 8 min stages, speed was lower at modelled markers: D_{\max} (-1.1 km/h; $p = 0.001$), LT (-0.9 km/h; $p = 0.008$) and log-log LT (-0.8 km/h; $p = 0.006$), yet RPE was higher and $\dot{V}O_2$ lower for LT (1.1, $p = 0.02$; -0.27 L/min, $p = 0.01$) and log-log LT (1.4, $p = 0.03$; -0.29 L/min, $p = 0.002$). Lactate and $\dot{V}O_2$ were greater at 8 km/h for 4 min ($p = 0.0001$), then similar until 11 km/h, with a trend towards elevated plasma lactate for 4 min thereafter. When applying lactate threshold markers to assess physiological responses to incremental running, protocols using prolonged stage durations may underestimate marker running speed.

Key words: Incremental exercise, Lactate markers, Oxygen uptake, Stage duration, Threshold.

INTRODUCTION

Incremental exercise with measurement of physiological responses, coupled with blood lactate sampling, is used to predict endurance performance (Coyle, 1999; Roecker, Schotte, Niess, Horstmann & Dickhuth, 1998) prescribe exercise intensity and monitor adaptation (Bentley, Newell & Bishop, 2007). Aerobic capacity and the anaerobic threshold (AT) can be assessed in this manner during sub-maximal tests (Davis, 1985; Wasserman, Whipp, Koysl & Beaver, 1973) in both athletic (Coen, Urhausen & Kindermann, 2001) and clinical populations (Katz, Berkowitz & Lejemtel, 1992). Running speed eliciting AT has greater association with endurance performance in comparison to other predictive indices such as running economy, maximal oxygen uptake ($\dot{V}O_{2\max}$) and muscle fibre composition (Farrell, Wilmore, Coyle, Billing & Costill, 1979; Sjodin & Jacobs, 1981). To better describe the workload -response relationship, fixed and modelled lactate markers can be calculated from blood samples collected at each increment. Lundberg et al (1986) established the LT via a computational method, whereby the lactate-workload response was modelled with two phase linear regression. Advancing upon this, Beaver and colleagues (1985) suggested applying a log transform to both lactate and workload values. Alternatively, fixed blood lactate accumulations (FBLA), such as 2.0 and 4.0 mmol/L (Heck et al., 1985; Kindermann, Simon & Keul, 1979) and the initial 1 mmol/L rise from baseline, rely upon blood lactate concentration responding in a monotonous, curvilinear fashion to workload increments. Fixed markers allow ease of use, yet ignore individual lactate variability at rest and during exercise. Considering individual variation, the deviation maximum (D_{\max}) detects workload yielding the maximum perpendicular from a line connecting the first and the final lactate-workload points to the estimated polynomial (Cheng et al., 1992). The calculation of the workload corresponding to the D_{\max} is therefore dependent on the individual's resting blood lactate concentration.

Lactate kinetics are sensitive to exercise incrementation rate and duration, both of which may be manipulated according to sporting performance demands and the individual (Stockhausen, Grathwohl, Burklin, Spranz & Keul, 1997). Typically stage increments between 3 and 6 min duration are used, based upon the attainment of lactate and oxygen uptake ($\dot{V}O_2$) steady-states (Bentley, Newell & Bishop, 2007). Previous research have found shorter stage durations to result in overestimation of the AT (Coen, Urhausen & Kindermann, 2001; Whipp, Davis, Torres & Wasserman, 1981), in addition to increasing workload at the LT (Bentley, McNaughton & Batterham, 2001). The latter reported 3 min stage increments had the effect of elevating power output at LT, in comparison to 8 min stages for cycling exercise. Yet, no effect was seen for workload, $\dot{V}O_2$ and heart rate (HR) at the FBLA 4.0 mmol/L. Step durations less than 3 min are unlikely to satisfy steady-state, and thus physiological response corresponding to particular lactate markers. Prolonged stages, in contrast, may allow lactate equilibrium, yet compromise peak $\dot{V}O_2$ attainment and promote premature fatigue. Differing muscular recruitment patterns (Beneke & von Duvillard, 1996) and maximal lactate steady-state responses (Beneke, Leithauser & Hutler, 2001) make direct comparisons between running and cycling difficult. For running, stages beyond 6 min have been recommended with the FBLA 4.0 mmol/L to avoid delay in blood lactate response (Foxdal, Sjodin & Sjodin, 1996). To expand upon findings of Bentley's (2001) and Foxdal's (1996) groups, our aim was to examine physiological responses corresponding to speeds at lactate markers using incremental treadmill running of traditional, 4 min, and prolonged, 8 min stage durations.

MATERIALS AND METHODS

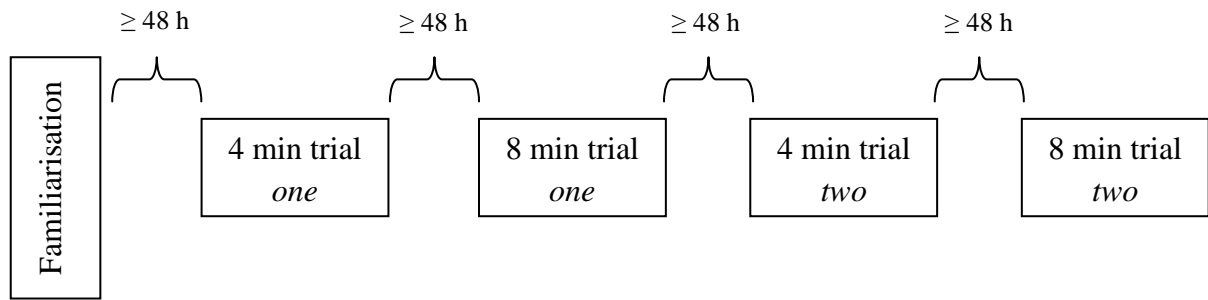
Participants

Fifteen healthy men (mean \pm SD; age 23 ± 4 yrs, height 1.78 ± 0.49 m, body mass 72.7 ± 10.8 kg) volunteered and provided written informed consent for participation in the study.

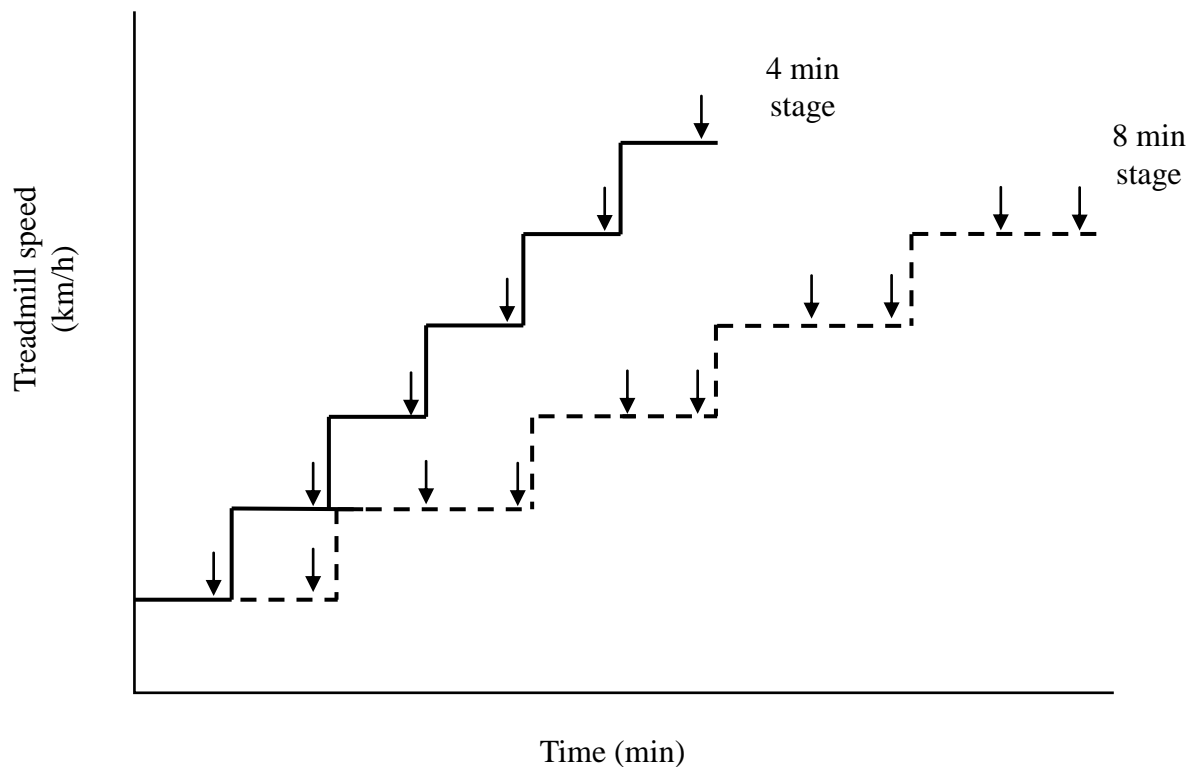
Volunteers completed a medical and an International Physical Activity Questionnaire (IPAQ), conforming to high levels of habitual physical activity (2877.5 ± 1304.4 MET·min/wk) (Bauman et al., 2009). Ethical approval for the research protocol was approved of by the University of Chichester Ethics Committee and conducted in accordance with the Helsinki Declaration.

Procedures

Participants visited the same temperature controlled laboratory ($19 - 22^{\circ}\text{C}$) on five occasions to perform: a familiarisation, and repeated incremental running trials of 4 and 8 min stages in a randomised order within a four wk period (Schematic 1). All were instructed to refrain from strenuous exercise 24 h prior to each visit and a minimum of 48 h was allocated between sessions. Volunteers reported to the laboratory euhydrated, having not eaten, or consumed caffeine 3 h prior. Participants ran at a 1% gradient with an incrementation rate of 1 km/h every 4 or 8 min on a Woodway Ergo ELG 70 powered treadmill (Weil am Rhein, Germany). Subsequently, lactate-speed data was plotted using lactate analysis software (Newell et al., 2007) with treadmill speed, $\dot{V}\text{O}_2$, HR and rating of perceived exertion (RPE) calculated corresponding to the markers: FBLA 4.0 mmol/L, an initial 1 mmol/L rise, D_{max} , LT and log-log LT.



↓ Blood lactate, $\dot{V}O_2$, HR and RPE measured.



SCHEMATIC 1. Schematic diagram of methodology and incremental exercise protocol for 4 and 8 min stage trials (trial order was randomised; above is an example of trial sequence).

Trials began with explanation of experimental protocol, then height was measured with a free-standing stadiometer (Holtain Ltd., Crymych, UK) and body mass using digital health scales (Seca Ltd., Birmingham, UK). All were familiarised with Borg's scale (Borg, Hassmen & Lagerstrom, 1987) for RPE and positioned with a heart rate monitor (Polar Electro Oy,

Kempele, Finland). At rest, heart rate and plasma lactate were recorded in the final 30 s of a 5 min, seated period. Next participants warmed up at 8 km/h for 5 min on the treadmill. Following a brief interval, exercise commenced with participants lowering themselves onto the treadmill belt when the start speed of 8 km/h was attained. Volunteers performed between four and ten stages with an incrementation rate of 1 km/h every 4 or 8 min. Participants were asked for RPE in the final 30 s of each stage, before straddling the treadmill upon stage completion during which a finger-tip capillary blood sample was collected.

Measurement Procedures

Respiratory gases were monitored breath-by-breath, concurrent with HR, using a portable metabolic cart (Cosmed K4b², Rome, Italy). Prior to each trial, the system was calibrated according to manufacturer's recommendations (Cosmed K4b² User Manual). For analysis, $\dot{V}O_2$ and HR were recorded as 30 s averages, and then reported during the final 30 s of each stage with treadmill speed, RPE and blood lactate concentration.

Plasma lactate was analysed via a ~25 μ l finger prick blood sample collected from the right index finger from an incision made using a Haemolance plus high flow lancet (HaeMedic AB, Munka Ljungby, Sweden). At rest and on stage completion, fingertip capillary blood samples were drawn into non-lysed, EDTA-treated microvettes (Sarstedt Aktiengesellschaft & Co., Nümbrecht, Germany) and analysed in duplicate for plasma lactate (2300 STAT PlusTM, YSI Life Sciences, Yellow Springs, USA). Where lactate concentrations varied less than 0.4 mmol/L between samples, the mean was reported. Where lactate concentration varied greater than 0.4 mmol/L, a third analysis was conducted with the mean of the two closest samples recorded (Grant et al., 2002). Lactate analysis software (Newell et al., 2007)

was used to establish treadmill speed, $\dot{V}O_2$, HR and RPE at the lactate markers: FBLA 4.0 mmol/L, the initial 1 mmol/L rise, D_{max} , LT and log-log LT.

Statistical Analysis

Physiological responses between 4 and 8 min stages were analysed by a Student's t-test for paired samples. One-way repeated measures analyses of variance (ANOVA) with pairwise comparisons were used to assess mean lactate and $\dot{V}O_2$ for: 4 min, 4 min at 8 min, and 8 min stages. Statistical analyses were calculated using PASW Statistics 18.0 for Windows (California, USA) with statistical significance set to a Type I error level of $p < 0.05$.

RESULTS

Mean maximal running speed was faster during 4 min (14.4 ± 1.4 km/h; $p=0.001$), in comparison to 8 min trials (12.6 ± 1.3 km/h) (Table 1). There was no effect of duration on speed, $\dot{V}O_2$, HR and RPE at fixed markers: FBLA 4.0 mmol/L and the initial 1 mmol/L rise. Speed, $\dot{V}O_2$ and HR were similar; yet RPE appeared elevated for 4 min at the FBLA 4.0 mmol/L (0.7; $p=0.46$) and the initial 1 mmol/L rise (0.9; $p=0.38$), in comparison to 8 min. For 8 min, speed was lower at modelled markers: D_{max} (-1.1 km/h; $p=0.001$), LT (-0.9 km/h; $p=0.008$) and log-log LT (-0.8 km/h; $p=0.006$), with HR unchanged between stage durations at respective markers. RPE was higher and $\dot{V}O_2$ lower at the LT (1.1, $p=0.02$; -0.27 L/min, $p=0.01$) and log-log LT (1.4, $p=0.03$; -0.29 L/min, $p=0.002$) for 8 min trials. This was not observed for D_{max} ($p=0.11$).

TABLE 1. Physiological responses corresponding to speed at fixed and modelled plasma lactate markers during incremental treadmill running trials of 4 and 8 min stage durations (mean \pm SD).

Plasma lactate marker	4 min trial				8 min trial			
	Speed (km/h)	HR (beats/min)	$\dot{V}O_2$ (L/min)	RPE	Speed (km/h)	HR (beats/min)	$\dot{V}O_2$ (L/min)	RPE
FBLA (4.0 mmol/L)	12.9 \pm 1.5	183.6 \pm 8.6	3.78 \pm 0.53	15.3 \pm 1.5	13.0 \pm 1.9	186.6 \pm 12.4	3.65 \pm 0.45	14.6 \pm 1.9
Initial 1 mmol/L rise	10.7 \pm 1.8	169.0 \pm 13.6	3.32 \pm 0.65	16.2 \pm 1.8	11.0 \pm 1.5	167.5 \pm 14.7	3.33 \pm 0.52	15.3 \pm 1.8
D _{max}	13.3 \pm 1.4	184.2 \pm 8.5	4.00 \pm 0.82	16.2 \pm 1.1	12.2 \pm 1.8 *	182.1 \pm 14.5	3.80 \pm 0.72	16.3 \pm 0.9
LT	11.4 \pm 1.2	175.8 \pm 7.8	3.51 \pm 0.48	14.7 \pm 1.3	10.5 \pm 0.9 *	170.3 \pm 15.9	3.26 \pm 0.48*	15.8 \pm 1.6 *
Log-log LT	10.4 \pm 0.9	171.6 \pm 11.0	3.39 \pm 0.54	13.3 \pm 1.9	9.6 \pm 0.6 *	160.5 \pm 15.0	3.08 \pm 0.53*	14.7 \pm 2.1 *

* Significant difference from 4 min values, p<0.05.

TABLE 2. Plasma lactate concentration at the completion of each running speed (km/h) during incremental treadmill running trials of 4 and 8 min stage durations, as well as for 4 min at 8 min (mean \pm SD).

Speed (km/h)	4 min trial			8 min trial		
	4 min lactate (mmol/L)	4 min lactate (mmol/L)	8 min lactate (mmol/L)	4 min lactate (mmol/L)	4 min lactate (mmol/L)	8 min lactate (mmol/L)
8 (n = 15)	1.3 \pm 0.5	1.3 \pm 0.6	1.1 \pm 0.6 *†	4.5 \pm 2.2	3.3 \pm 1.5	3.0 \pm 1.4
9 (n = 15)	1.5 \pm 0.7	1.4 \pm 0.7	1.4 \pm 0.8	4.7 \pm 1.9	3.4 \pm 1.4	3.3 \pm 2.0
10 (n = 15)	1.9 \pm 0.9	1.8 \pm 1.0	2.0 \pm 1.2	4.7 \pm 1.8	2.9 \pm 0.8	4.1 \pm 1.5
11 (n = 15)	2.5 \pm 1.4	2.5 \pm 1.5	2.8 \pm 1.9 *†	6.3 \pm 1.9	4.5 \pm 1.1	
12 (n = 12)	3.5 \pm 2.1	3.2 \pm 2.2	2.8 \pm 1.7	7.3 \pm 1.1		

* Significant difference from 4 min values, p<0.05. † Significant difference from 4 min at 8 min values, p<0.01. n= participant number.

Following stage one (8 km/h), plasma lactate concentration was 0.2 mmol/L lower for 8 min than 4 min ($p=0.008$), and 4 min at 8 min ($p=0.03$) respectively. Thereafter, plasma lactate was similar until the end of stage four (11 km/h) ($p=0.05$). For subsequent speeds (12 to 15 km/h), there was a trend towards higher lactate concentrations during 4 min stages, than 8 min (Table 2). For example, at 12 km/h lactate was 3.5 mmol/L for 4 min and 2.8 mmol/L for 8 min. At 13 km/h lactate was 4.5 mmol/L for 4 min and 3.0 mmol/L for 8 min, respectively.

Mean $\dot{V}O_2$ was elevated during 4 min (2.91 L/min), in comparison to 8 min trials (2.57 L/min; $p=0.0001$), following the first stage, 8 km/h until 11 km/h (Figure 1). At 4 min during 8 min, mean $\dot{V}O_2$ was also reduced when compared to that measured at the stage end, at 8 min (2.48 L/min; $p=0.0002$). At 9 km/h during 4 min stages, $\dot{V}O_2$ (3.17 L/min) remained above both, 4 min at 8 min (2.86 L/min; $p=0.0001$) and 8 min (2.89 L/min; $p=0.001$). At 10 and 11 km/h, $\dot{V}O_2$ was similar between stage lengths, and no difference was observed above 11 km/h. This may be attributable to fewer participants completing stage increments above this speed, resulting in insufficient paired data sets for statistical analysis. In accordance to our plasma lactate data, for 12 km/h $\dot{V}O_2$ sampled was not different between 4 (3.61 L/min) and 8 min stages (3.4 L/min) ($p=0.13$). Dissimilar to our plasma lactate response, at 13, 14 and 15 km/h, $\dot{V}O_2$ for 8 min appeared to rise steeply, $\dot{V}O_2$ showing a trend of surpassing that of 4 min trials at 14 and 15 km/h. Therefore, the effect of stage duration on $\dot{V}O_2$ response was not reflected by plasma lactate response at running speeds above 12 km/h.

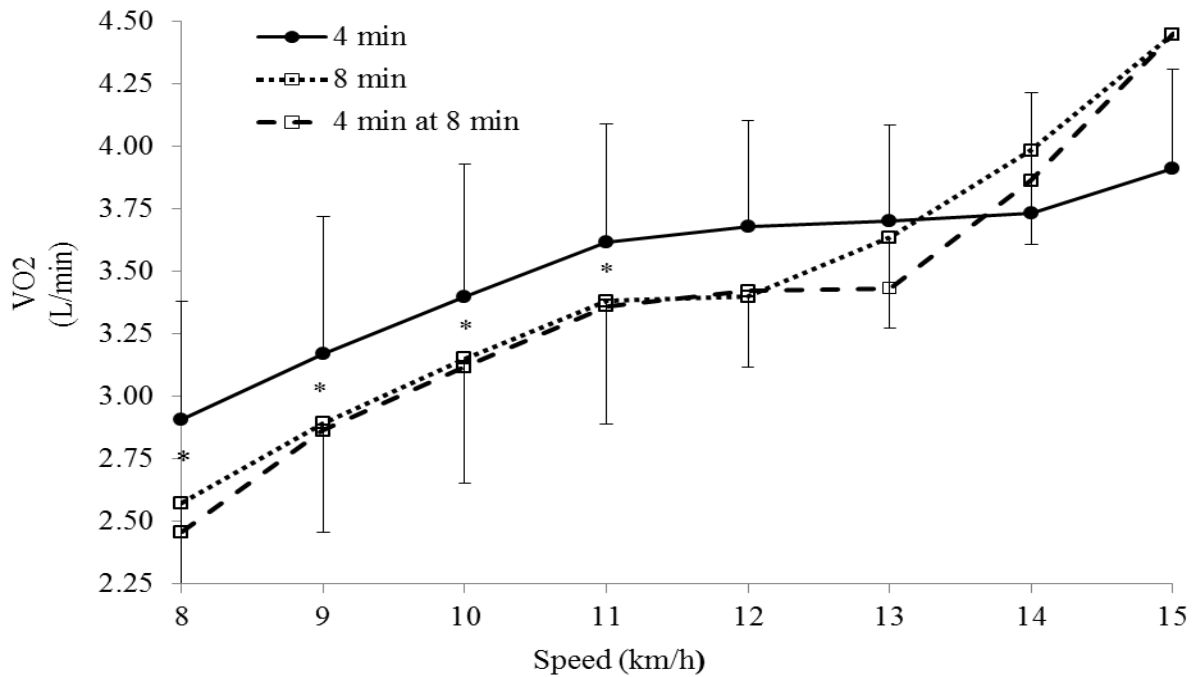


FIGURE 1. Oxygen uptake ($\dot{V}O_2$) (L/min) measured at the completion of each running speed (km/h) during incremental running trials of 4 and 8 min stages, as well as for 4 min at 8 min (mean \pm SD). * Significant difference from 4 min values, $p < 0.05$.

DISCUSSION

The incrementation rate and stage duration of incremental exercise protocols are adapted according to activity-specific demands and the characteristics of the population under assessment. The purpose of the present study was to compare physiological responses ($\dot{V}O_2$, HR and RPE) corresponding to speeds at plasma lactate markers during repeat, incremental treadmill running trials of 4 and 8 min stage durations in young men. These findings suggest that trials using stage durations of 8 min have the effect of reducing the running speed, but increasing the perception of exercise intensity at the LT and log-log LT. Furthermore, there was no effect of stage duration on selected physiological responses when using the fixed plasma lactate markers: FBLA 4.0 mmol/L and the initial 1 mmol/L rise, in our cohort.

Production and elimination of intramuscular lactate determines circulating blood lactate concentration (MacRae, Dennis, Bosch & Noakes, 1992), and as such, blood lactate responds

in a steady-state manner at low- and moderate-running speeds sub LT. At speeds above the LT, blood lactate accumulation will occur, unabating even when the intensity is reduced to constant speed. Blood lactate response will only decrease when running speed is reduced to those intensities below the LT. The duration an individual is exposed to a workload during incremental exercise remains controversial. Previously, Yoshida and co-workers (1984) observed no difference in $\dot{V}O_2$ at the initial rise above baseline and FBLA 4.0 mmol/L during incremental cycling protocols of 1 and 4 min stages. However, workloads at the respective markers were greater for the shorter, 1 min stages. It has been proposed that stage increments of at least 3 min are adequate to reach lactate steady-state; stages of longer duration allow lactate diffusion to reach an equilibrium-state between muscle and blood, thus permitting greater sensitivity for blood lactate sampling (Billat, Sirvent, Py, Koralsztein & Mercier, 2003). The reliability of 3 min increments to identify fixed blood lactate markers and the LT was tested by Weltman and colleagues (1990) in a cohort of male runners. Assessed against a discontinuous, criterion protocol of 10 min stages; the 3 min protocol elicited similar running speeds at the FBLAs: 2.0, 2.5 and 4.0 mmol/L and the LT, as well as $\dot{V}O_2$, suggesting high reliability. For repeat trials of 3 min increments, mean running speed and $\dot{V}O_2$ were 16.2 km/h and 4.13 L/min at FBLA 4.0 mmol/L; and 13.0 km/h and 3.28 L/min at the LT, respectively. Dissimilarities in our data (12.9 km/h and 3.78 L/min at FBLA 4.0 mmol/L; 11.4 km/h and 3.51 L/min at the LT) may be due to differences in the training status of populations sampled. Weltman et al (1990) recruited male runners accumulating a minimum of 40 km per wk; whereas we used recreationally active, male students who did not all undertake regular running. Following endurance training, higher running speeds can be attained at a specific lactate marker leading to a rightward shift in the lactate-speed relationship. If this does not sufficiently explain the disparities in our findings, marker suitability for stage duration may. Similarly to Weltman (1990) stage duration had no effect

on running speed at FBLA 4.0 mmol/L; yet for 4 min stages we observed faster speed concomitant with elevated $\dot{V}O_2$ at LT. Being regular runners, it is plausible that their cohort had a superior lactate buffering capacity, allowing them to tolerate the 10 min criterion protocol to a greater extent than ours during the 8 min protocol. Thus, reaching metabolic breakpoint at similar running speeds to shorter stage durations, which may explain the attainment of LT at slower running speeds during 8 min stages in our investigation. Interestingly, workload at D_{\max} was elevated for 4 min, in comparison to 8 min trials, without influence upon $\dot{V}O_2$, HR and RPE. Previously we found poor reproducibility for the D_{\max} during repeat, 4 and 8 min protocols. D_{\max} identifies threshold speed corresponding to the maximal perpendicular of a line connecting the first and the final lactate concentrations from a third-degree polynomial lactate-speed curve; thus dependent upon the curve expressing a smooth, parabolic function (Figure 2). As can be seen in Figure 3, this marker is unsuitable with the occurrence of a breakpoint, and relies upon the individual exerting maximal effort throughout the trial. As little consensus exists to an established fitting procedure, it appears apt to consider the incremental protocol design when selecting lactate markers to determine exercise intensity and monitor adaptation. Furthermore, the practice of applying a single marker for analysis of lactate-workload relationships should be discouraged.

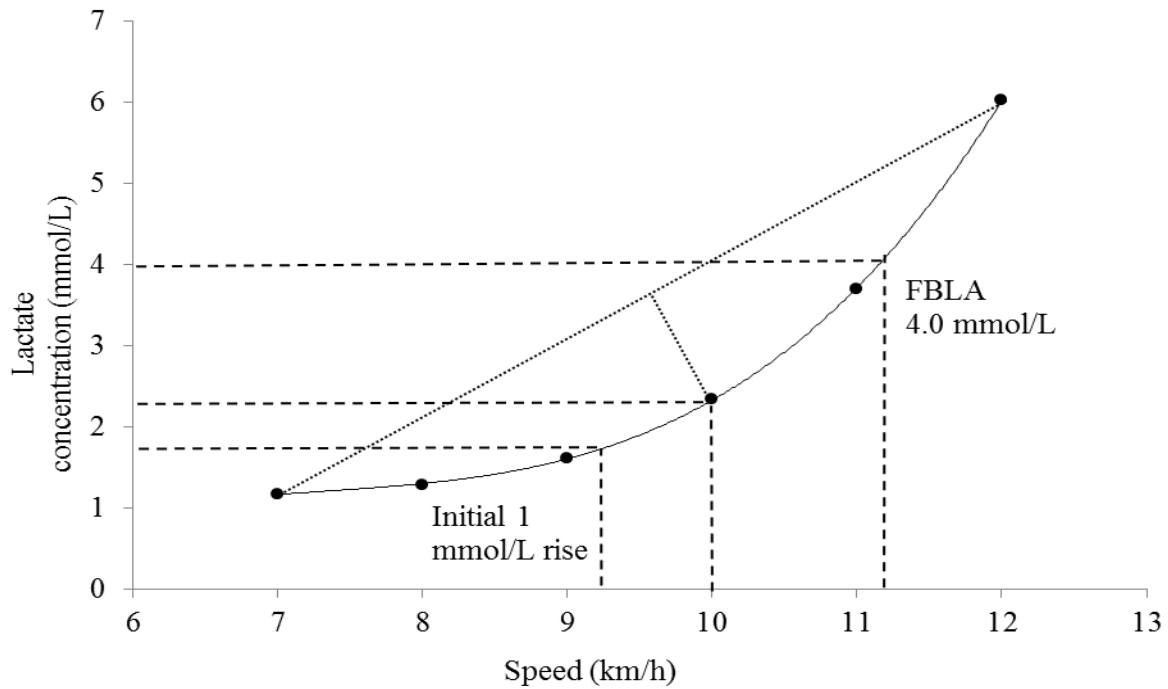


FIGURE 2. Plasma lactate-speed relationship for a single participant analysed from a 4 min stage incremental treadmill running trial with selected lactate markers (FBLA 4.0 mmol/L = 11.1 km/h; initial 1 mmol/L rise = 9.2 km/h; D_{\max} = 9.9 km/h).

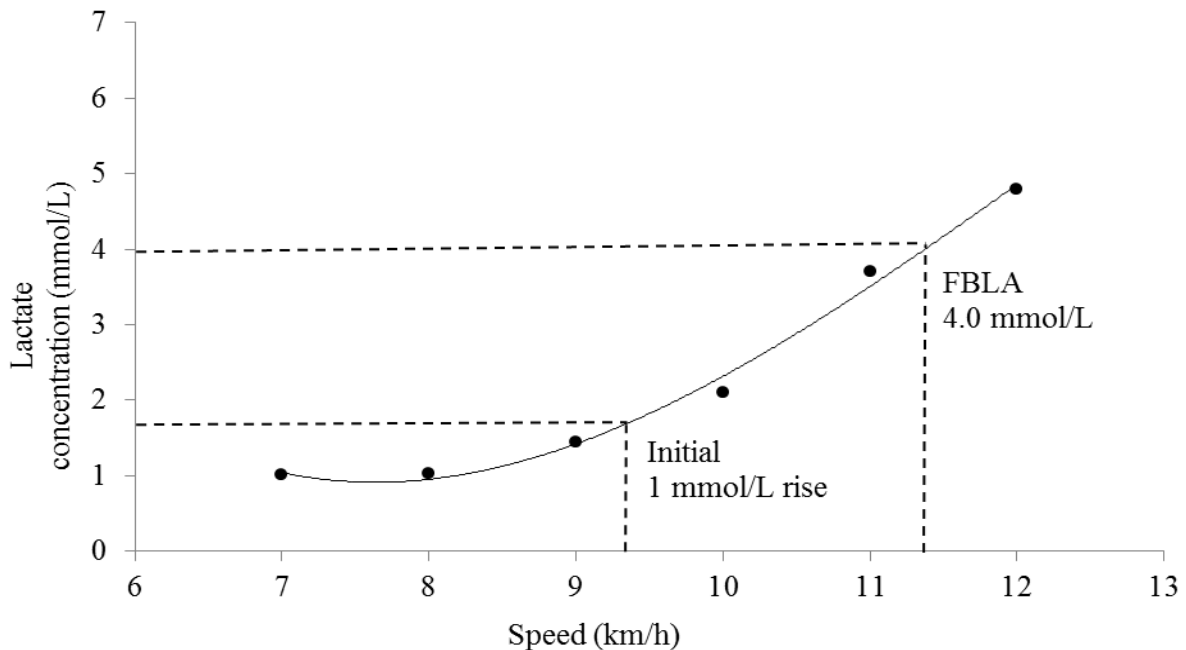


FIGURE 3. Plasma lactate-speed relationship for a single participant analysed from a 4 min stage incremental treadmill running trial with selected lactate markers repeated less than a week after that of Figure 2 (FBLA 4.0 mmol/L = 11.4 km/h; initial 1 mmol/L rise = 9.5 km/h; D_{\max} = 15.3 km/h).

Previous studies have observed elevated lactate response during shorter stage protocols, when compared against longer stage increments (Bentley, McNaughton & Batterham, 2001; Kuipers, Rietjens, Verstappen, Schoenmakers & Hofman, 2003). To our knowledge, only one investigation has compared difference in blood lactate response to incremental running exercise of 4 and 8 min stages (Foxdal, Sjodin & Sjodin, 1996). These authors found 4 and 6 min stages insufficient to achieve lactate steady-state in a group of firemen and marathon runners. Furthermore, the greater relationship between blood lactate sampled from incremental and constant-speed exercise observed in the running cohort is indicative of a training-induced regulatory effect likely to influence the use of FBLA 4.0 mmol/L for training prescription. Here, faster running speed at the FBLA 4.0 mmol/L was noted for 4 min, when compared to 8 min stages. In contrast, we found no effect of stage duration on speed at FBLA 4.0 mmol/L; we used non-lysed, plasma capillary samples, whereas Foxdal's group (1996) used haemolysed capillary blood for analysis. Although capillary samples are known to reflect arterial lactate values, the disparity may be attributable to the non-homogenous distribution of lactate in blood. Blood sampling and analysis media vary throughout the literature, making direct comparison of data difficult. Whole venous samples have been reported to produce lower concentrations than fingertip samples during 4 min stage incremental treadmill running (El-Sayed, George & Dyson, 1993). Analytically, samples from plasma tend to be higher than those of whole- and haemolysed blood (Williams, Armstrong & Kirby, 1992); this is due to the erythrocyte membrane acting as a barrier to lactate transit between plasma and red blood cells with changing lactate concentration. Various have cited difference in workload at fixed lactate markers (Bishop, Smith, Kime, Mayo & Tin, 1992; Foxdal, Sjodin, Ostman & Sjodin, 1991; Williams, Armstrong & Kirby, 1992), but not the LT (Robergs et al., 1990; Yoshida, 1984) when collecting from venous or capillary, and analysing via plasma or haemolysed blood.

A secondary, but important finding of this study was the possible dissociation of plasma lactate concentration and $\dot{V}O_2$ for faster running speeds during 4 min stage trials. There appeared an increased blood lactate response from stages five to seven (12 to 15 km/h) during 4 min stages, with the difference increasing with advancing stages (stage eight, mean plasma lactate concentrations: 6.3 mmol/L (4 min); 4.5 mmol/L (8 min)). This was reflected by an observation of higher $\dot{V}O_2$ from stages one to six (8 to 13 km/h) during 4 min stages, yet by stages six and seven (14 and 15 km/h) $\dot{V}O_2$ during 8 min had exceeded that of 4 min. Mean maximal treadmill running speed achieved during 4 min trials was greater than that of 8 min trials, with the length of all 8 min trials in excess of 4 min trials. The contrasting natures of the two protocols impose specific physiological demands upon the individual, therefore influencing the particular selection of lactate markers and the subsequent prescription of training intensities. When comparing power output, HR and $\dot{V}O_2$ between 3 and 8 min stage protocols for incremental cycling, Bentley et al (2001) reported 3 min increments may be adequate when using the FBLA 4.0 mmol/L, but not the LT. Prolonged stages had the effect of increasing power output at the LT, which may explain the possible $\dot{V}O_2$ slow component observed for our 8 min trials. Lactate has been implicated as a causative factor leading to the $\dot{V}O_2$ slow component and muscle fatigue (Zoladz, Gladden, Hogan, Nieckarz & Grassi, 2008). Steady-state $\dot{V}O_2$ occurs during low- to moderate-intensity exercise, however with transition to high-intensity exercise beyond the LT, $\dot{V}O_2$ rises continuously until the cessation of exercise (Whipp & Wasserman, 1972). Once the LT has been surpassed during incremental running exercise, the $\dot{V}O_2$ -speed relationship exhibits a non-proportional, curvilinear response indicative of muscular fatigue. In this manner, fixed plasma lactate markers may overestimate HR and $\dot{V}O_2$ when using stages of prolonged duration as such markers are

unable to discern individual metabolic differences, and may lack sensitivity to distinguish influence of muscular fatigue.

This investigation compared the treadmill speed, $\dot{V}O_2$, HR, and RPE at fixed and modelled plasma lactate markers from data recorded during repeat incremental running trials of 4 and 8 min stages in healthy men. The findings suggest the 8 min trials have the effect of reducing the speed, but increasing the perception of exercise intensity at the LT and log-log LT. When assessing physiological responses for incremental treadmill running, the use of these markers with protocols of longer stage duration may underestimate marker running speed. Secondly, the observed dissociation of plasma lactate concentration and $\dot{V}O_2$ between 4 and 8 min trials suggests consideration of lactate marker usage in accordance to: participant training status and conditioning, exercise mode and step duration.

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