REVIEW ARTICLE

The prediction of endurance performance from work rates at fixed blood lactate concentrations is a mathematical not a physiological phenomenon – a novel hypothesis

I Cook (BA (Phys Ed) Hons, BSc (Med) Hons)

Department of Kinesiology and Physical Education, University of Limpopo, Polokwane

Abstract

Traditionally it is accepted that physiological mechanisms underpin the oft-demonstrated highly significant relationship between work rates at fixed blood lactate concentrations of 4 mmol.l^{-1} (WR_{fblc}) and endurance performance (EP). The objective of this paper was to suggest an alternative non-physiological mechanism for the ability of WR_{fblc} to predict EP. Important observations are that WR_{fblc} occur at a constant percentage of maximum work rate (WR_{max}) in trained athletes and there are high correlations between WR_{fblc}, WR_{max} and EP. These observations suggest that WR_{fblc} is a marker of WR_{max}. Using data from published reports, the association between WR_{fblc} and EP is dramatically reduced after removing the effect of WR_{max}. Furthermore, the between-subject variation in slopes (first derivative of exponential plus constant function) at fixed blood lactate concentrations is significantly associated with WR_{fblc}/WR_{max} ratios with exponential but not linear relationships using the same blood lactate concentration - work rate data. This paper argues that WR_{fblc} is related to EP through its relationship with WR_{max}, and contends that the relationship between WR_{fblc} and EP is not causal because of physiological mechanisms, specifically skeletal muscle metabolism. The WR_{fblc}-EP relationship is rather the result of the peculiar properties of the exponential relationship between blood lactate concentrations and incremental work rate. Re-analyses of existing data sets with valid measures of WR_{max} and homogeneous and heterogeneous samples in terms of endurance performance, to confirm or reject this hypothesis, are warranted.

CORRESPONDENCE:

I Cook
Department of Kinesiology and Physical Education
University of Limpopo (Turfloop Campus)
PO Box 459
Fauna Park
0787
Polokwane
Tel + Fax: 015-268 2390
E-mail: ianc@ul.ac.za

Introduction

Certain paradigms propagated by exercise scientists are based on research that was conducted during the early part of the 20th century.[3] Consequently, the conclusions made by these early scientists were based on data obtained using rather indirect and limited methods. The techniques available to exercise scientists today have allowed much more detailed, accurate and direct measurements to be made.[3]

Arguably, 2 of the most pervasive paradigms in exercise science, in need of revision, are that human endurance performance is limited by whole-body maximal oxygen uptake (VO_{2max}) and that 'lactic acid' is the result of skeletal muscle anaerobiosis and causes fatigue.[3,13,31,39] In other words, VO_{2max} is limited by the availability and supply of oxygen to the working skeletal muscles, and lack of oxygen to the muscles leads to the production of 'lactic acid' which inhibits skeletal muscle contractile performance (this will be referred to as the Meyerhof-Hill-Wasserman postulate, so-named after the major contributors to the development of the model). Predictably, from this paradigm developed the use of blood lactate concentrations to monitor and predict performance in endurance athletes.[44] This practice continues,[37] specifically the use of the fixed blood lactate thresholds, and in particular the work rate at a fixed blood lactate concentration of 4 mmol.l^{-1} (WR_{fblc}), to predict endurance performance (EP).[15,22]

The objective of this paper is to suggest an alternative non-physiological mechanism for the ability of WR_{fblc} (4 mmol.l^{-1}) to predict EP. The paper is divided in 2 parts. The first section briefly reviews the evidence cited in support of WR_{fblc}, and then shortly elaborates on some important aspects related to the exponential increase in blood lactate concentration with increasing work rate. The second part of this paper is devoted to developing an alternative non-physiological hypothesis for the ability of WR_{fblc} to accurately predict EP. Readers are referred to more comprehensive and detailed reviews regarding blood lactate metabolism.[3,9,13]

What evidence has been presented by proponents of WR_{fblc} to justify the use of fixed blood lactate concentrations to predict endurance performance?

The use of WR_{fblc} probably originated during the early 1970s, the main proponent being Alois Mader, a German sports scientist and medical doctor, now retired. In 1976 Mader and his

colleagues from the German Sports University, Cologne published 2 papers, in German, suggesting that the 4 mmol.l^{-1} blood lactate threshold represented the 'aerobic-anaerobic threshold'.[18] During the 1970s the use of blood lactate concentration for prescribing training intensities and monitoring training adaptation in endurance athletes, in particular swimmers, was widespread in pre-unification Germany.[5] It could be argued that the phenomenal success of the East German swimmers[5] served as empirical evidence in support of the validity of WR_{fblc} for prescribing training intensities and monitoring training adaptation.

Support from the scientific community came from a number of sources, but most importantly the Meyerhof-Hill-Wasserman postulates[44] formed the bulwark of the defence.[18,22] Indeed, Karlsson and Jacobs have suggested a similarity between the 4 mmol.l^{-1} blood lactate threshold, and a stable V_E/VO_2 ratio.[22] However, Davis *et al.*[7] showed that the metabolic rate (ml O_2.min^{-1}) at the anaerobic threshold, determined by gas exchange,[44] differed from the metabolic rate at the blood lactate turn point and fixed blood lactate concentrations (2 mmol.l^{-1} and 4 mmol.l^{-1}) by 44 ml O_2.min^{-1}, 280 ml O_2.min^{-1}, and 1 028 ml O_2.min^{-1}, respectively. The metabolic rates at the fixed blood lactate concentrations were significantly different from the gas exchange anaerobic threshold ($p < 0.05$).

In 1978, Jorfeldt *et al.*[20] presented *in vivo* human data suggesting a plateau in the lactate released from working skeletal muscle (~4 mmol.min^{-1}) when lactate concentration in working skeletal muscle reaches about 4 - 5 mmol.l^{-1}. In citing the findings of Jorfeldt *et al.* as evidence for the 4 mmol.l^{-1} threshold, some have suggested that 'since the density of muscle and blood are almost identical, a blood lactate concentration of 4 - 5 mmol.l^{-1} corresponds to the saturation part for the speed of lactate release'.[22] However, it has been noted that if the lactate efflux data of Jorfeldt *et al.* are plotted against the lactate gradients, no saturation point is seen[21] and to their credit, Mader and Heck have conceded this point.[27] Furthermore, recent studies have found lactate efflux rates as high as 7.6 mmol.min^{-1}, without evidence of a saturation point in lactate efflux.[21] Therefore, although the data of Jorfledt *et al.* is suggestive of lactate transporter mechanisms between muscle and blood,[21] the absolute values of lactate efflux and working skeletal muscle lactate concentration are fortuitous and cannot be used to justify the 4 mmol.l^{-1} threshold, as has been done by proponents of the threshold.[15,22]

During the early 1980s a number of papers were published that endeavoured to provide further physiological support for the 4 mmol.l^{-1} threshold.[22] The support in these papers for the 4 mmol.l^{-1} threshold was based largely on studies examining the relationships (correlations) between the 4 mmol.l^{-1} threshold and skeletal muscle fibre typing and enzyme concentrations.[22] However, the underlying basis of these studies was the belief that in order to prevent an 'oxygen deficit', the ability of skeletal muscle respiratory capacity and cardiorespiratory capacity must be improved.[22] Essentially these papers found significant associations between WR_{fblc} and percentage slow twitch fibre area, ratio of oxidative to glycolytic enzyme activities, skeletal muscle respiratory capacity and capillary density, total heart volume and maximal oxygen uptake.[22]

In 1985, Mader and associates published a paper attempting to justify the 4 mmol.l^{-1} threshold,[15] probably because of concerns raised by others about the generalisability of a single absolute blood lactate threshold as representative of steady state conditions.[40] They found that the mean maximal lactate steady state concentration (MLSS) obtained during five 25-minute constant load tests on a treadmill for 16 trained males was on average 4.02 + 0.73 mmol.l^{-1}.[15] The blood lactate concentrations, of the corresponding MLSS speed, for incremental treadmill tests of 3 and 5 minutes stage lengths were 3.50 + 0.60 mmol.l^{-1} and 4.05 + 0.86 mmol.l^{-1}, respectively. Different stage lengths and treadmill types had significant effects on work rates at 4 mmol.l^{-1} ($p < 0.04$) and various running surfaces and treadmill inclinations produced varying blood lactate responses. The length of blood sampling periods between work increments for an incremental test had no effect on the work rate at 4 mmol.l^{-1} ($p = 0.362$).[15] Mader and associates also reported a mean MLSS of 4.01 + 0.75 mmol.l^{-1} in 59 elite swimmers after a continuous 30-minute swim.[34] Furthermore, the mean swimming speed during the 30-minute swim test was not significantly different from the speed at a blood lactate concentration of 4 mmol.l^{-1} determined from a two-speed swim test (2 x 400 m) (1.361 m.s^{-1} v. 1.353 m.s^{-1}). Moreover, a significant association was found between these two speeds ($r = 0.97$, $p < 0.001$).[34]

The findings of Mader and associates showed, as expected, that testing conditions must remain constant between tests to ensure reliable blood lactate concentration - work rate relationships.[15] However, this hardly serves as justification for the use of the 4 mmol.l^{-1} threshold. The primary evidence for justifying the 4 mmol.l^{-1} threshold was firstly, that the swimming and running speeds at MLSS produced blood lactate concentrations very close to 4 mmol.l^{-1}.[15,34] Secondly linear regression for the swimming and running speeds at 4 mmol.l^{-1} obtained during incremental tests versus the swimming and running speed at MLSS, were highly correlated ($r > 0.95$) and very close to the line of identity.[15,34] However the primary validation seems to be the appeal to MLSS, which Mader and associates found to be ~4 mmol.l^{-1}.[15,34] Brooks *et al.*[3] note that although a MLSS at ~4 mmol.l^{-1} blood lactate concentration is often found, the association is not causal. Furthermore, recent work suggests the appeal to MLSS does not serve to justify the 4 mmol.l^{-1} threshold because the amount of active skeletal muscle mass has a significant effect on MLSS.[2] Moreover, highly variable MLSS between subjects of 2 mmol.l^{-1} - 6 mmol.l^{-1} have been demonstrated.[40]

Finally, in 1986, further support for the 4 mmol.l^{-1} threshold came from a computer simulation of a 2-compartment lactate kinetics model which predicted that MLSS would occur in the range of 3 - 4 mmol.l^{-1}.[27] Significantly, Mader and Heck did not explain, on the basis of their model,[27] why some studies have found highly variable MLSS ranging from 2 mmol.l^{-1} to 6 mmol.l^{-1} which are at odds with their 4 mmol.l^{-1} threshold.[40]

From the preceding discussion it can be concluded that the

evidence forwarded to justify the 4 mmol.l^{-1} is flawed for at least 4 reasons: (*i*) the appeal to questionable paradigms[44] to explain the rise in blood lactate concentrations with increasing work rate;[18,22] (*ii*) the reliance on 1 study suggesting a saturation point in muscle lactate efflux at a blood lactate concentration of ~4 mmol.l^{-1};[20] (*iii*) the fortuitous findings of MLSS values of ~4 mmol.l^{-1};[15,27,34] and (*iv*) the use of associations between skeletal muscle fibre types, skeletal muscle enzyme profiles and WR_{fblc} to establish causality between WR_{fblc} and EP.[22]

Lactate is an important, dynamic metabolite not a 'dead-end' substrate

Studies have shown repeatedly and convincingly that blood lactate concentrations do not reflect the rate of production or removal of lactate, nor do blood lactate concentrations reflect an 'anaerobic' state of the skeletal muscle.[3] Moreover, lactate does not cause fatigue but is an important substrate.[3] Gladden summarises appropriately that 'lactate can no longer be considered the usual suspect for metabolic "crimes", but is instead a central player in cellular, regional and whole body metabolism'.[13]

Rising blood lactate concentrations are more likely the result of greater ATP resynthesis and myosin ATPase activity using carbohydrate as a fuel source, and greater recruitment of type II skeletal muscle fibres.[9] The outcome of this is a greater production of skeletal muscle lactate with a resultant increased co-transport of lactate and hydrogen ions out of the skeletal muscle cell. Consequently blood lactate concentrations start to rise exponentially. Importantly, skeletal muscle lactate is 'dumped' into the circulation to reduce rising hydrogen ion concentration in skeletal muscle cytosol during high work rates.[9] Minute ventilation therefore increases exponentially with increasing work rate because of increasing carbohydrate-supported myosin ATPase activity.[9]

Low blood lactate concentrations are not synonymous with enhanced endurance performance

The traditional paradigm[44] that 'lactic acid' results from an oxygen deficit predicts that low blood lactate concentrations are synonymous with improved endurance performance. However, this prediction is at odds with the findings of higher blood lactate concentrations and enhanced endurance performance with sodium citrate ingestion,[36] high carbohydrate diets,[25] and fast versus slow marathon runners.[33]

Potteiger *et al.*[36] found significantly faster performance times (1 min 43 sec, $p < 0.05$) and higher blood lactate concentrations (> 1.5 mmol.l^{-1}, $p = 0.021$) during a 30 km laboratory cycling time trial, in trained cyclists after the ingestion of sodium citrate compared with a placebo trial. Compared with a low-carbohydrate diet, a high-carbohydrate diet resulted in an improved endurance time (23 min v. 49 min, $p < 0.002$) and higher peak blood lactate concentrations (3.2 mmol.l^{-1} v. 5.6 mmol.l^{-1}, $p < 0.002$) for a cycling test conducted at 67% of maximal work rate in trained cyclists.[25] Furthermore, improved performance was associated with an upward shift in the blood lactate concentration - work rate curve for a high-carbohydrate diet compared with a low-carbohydrate diet.[25] During a treadmill marathon run, blood lactate concentrations were significantly higher in fast marathon runners compared with slow marathon runners (2.1 mmol.l^{-1} v. 1.2 mmol.l^{-1}, $p < 0.05$).[33] Paradoxically, peak blood lactate values correlated directly and significantly ($r = 0.68$ to $r = 0.71$, $p < 0.01$) with 10 - 42.2 km running performances in 43 marathon and ultra-marathon runners.[32] Against prediction, MacRae *et al.*[26] demonstrated similar lactate appearance rates before and after 9 weeks of endurance training (215 v. 244 μmol.min^{-1}.kg^{-1}, $p > 0.05$), despite a significant 14.5% ($p < 0.05$) improvement in VO_{2max}.

Indeed, Connett *et al.*[6] posit that skeletal muscle lactate is essential in maintaining a favourable reduction-oxidation potential in the cytosol (high NAD^+/NADH ratio) and consequently enhanced mitochondrial function. Low cytosolic NAD^+/NADH ratios will inhibit pyruvate dehydrogenase, slow down glycolysis and reduce the delivery of NADH to the mitochondria to produce ATP.[3] Lactate thus serves as an important buffer to maintain a high cytosolic NAD^+/NADH ratio and therefore to preserve lactate and NADH concentrations proportional to VO_2.[6] In other words, a high skeletal muscle lactate concentration and similarly a high blood lactate concentration are necessary to obtain a high VO_{2max} and WR_{max}. Interestingly, as early as 1929 Herbst stated that 'the greater the amount of lactic acid that can accumulate in the blood during exercise before exhaustion, the higher the endurance level'.[17] More than 60 years later, MacRae *et al.*[26] concluded that 'during endurance events in which athletes perform sustained exercise at ~80 - 90% VO_{2max}, lactate production is probably equal to or, more likely, greater than that in less well-conditioned individuals'.

Effect of endurance training on blood lactate accumulation

The right-shift in the blood lactate concentration-work rate relationship after endurance training has been well documented and has been ascribed to decreases in catecholamine release and β-adrenergic stimulation, and increases in plasma volume and endurance-trained muscle oxidative capacity.[9,26] Rising blood lactate concentrations are a complex, dynamic process described by the rate of blood lactate appearance (R_a) and disappearance (R_d). Consequently, blood lactate concentrations alone provide no information regarding the underlying lactate R_a and lactate R_d. Using radio isotopes, MacRae *et al.*[26] demonstrated that only for exercise intensities < 60% VO_{2max} was lactate R_a significantly lower after training, but remained the same at higher intensities. In contrast, lactate R_d was significantly higher only at exercise intensities > 60% VO_{2max}, and remained constant after training at low work rates (< 60% VO_{2max}). Considering the contrasting intensity-dependent responses of blood lactate R_a and R_d to endurance training, it is difficult to understand the continued support for blood lactate testing to monitor adaptation to training and for prescription of training intensities.

Unlike the absolute values of the blood lactate concentration-work rate relationship, the percentage of maximal work rate at which WR_{fblc} occurs remains constant after training.[8,38,48] In other words, even though endurance training results in an increase in WR_{max} and a right shift in the blood lactate concentration - work rate curve, WR_{fblc} still closely mirrors WR_{max}. This suggests that the constant WR_{fblc}/WR_{max} ratio is not determined by the physiological mechanisms responsible for changes in WR_{fblc}, WR_{max} or EP. Importantly, this would suggest that WR_{fblc}/WR_{max} ratios are independent of either the Meyerhof-Hill-Wasserman postulates[44] or more recent integrated systems approaches.[39]

Effect of body mass and active skeletal muscle size on blood lactate accumulation

Because blood lactate concentrations and lactate R_a are volume-dependent measures, any changes or differences in plasma volume or the lactate distribution volume can have significant effects on blood lactate accumulation.[2,12,16,26] There is evidence of dilution of blood lactate concentrations through an increased plasma volume after 1 week of endurance training.[12] Furthermore, a blunted increase in blood lactate concentrations for larger subjects,[16] and increased active skeletal muscle mass has also been found.[2]

Unpublished results (Dennis SC, Albertyn R, Bosch AN, Noakes TD, University of Cape Town, 1991), obtained from competitive and elite cyclists and runners, suggest that the major determinants of rises in blood lactate concentration (measured from the slopes of the curves), were starting work rate and body mass. These investigators recruited endurance athletes of different body masses and athletic ability, and used different testing protocols (treadmill v. bicycle, slow work rate increments v. fast work rate increments, higher starting work rate v. lower starting work rate) to investigate the influence of the type of work, the work rate increments, the starting work rate, body mass and athletic ability on blood lactate curves. To simplify the lactate slope units, the blood lactate - work rate curves were expressed as mmol of lactate and VO_2. To correct for plasma volume the blood lactate concentrations were multiplied by 0.05 $l.kg^{-1}$. The blood lactate - VO_2 data were fitted with an exponential function ($y = a + b.e^{c.x}$) and the work rate ($mmol.min^{-1}$ VO_2) at a slope of 0.10 mmol per $mmol.min^{-1}$ was used to compare blood lactate -VO_2 curves. The rates of blood lactate accumulation (blood lactate slopes) were not affected ($p > 0.05$) by the type of work (running: 83 mmol $O_2.min^{-1}$, cycling 77 mmol $O_2.min^{-1}$) or the work rate increment (15 watts per min: 77 mmol $O_2.min^{-1}$, 40 watts per 6 min: 71 mmol $O_2.min^{-1}$). Irrespective of the athletic ability and work type, the higher starting work rates resulted in higher blood lactate slope values ($r = 0.96$, $p < 0.0001$). Blood lactate slopes were significantly related to body mass with and without plasma volume corrections ($r = 0.85$, $p < 0.01$ and $r = 0.91$, $p < 0.001$). There were no significant relationships between blood lactate slopes (absolute or expressed as a percentage of peak VO_2) and 10 km running time ($r < -0.43$, $p > 0.05$).

Similarly, using unpublished results (Cook I and van Wyk GJ, University of Pretoria, 1991) for 6 elite and competitive male triathletes of varying body masses (73 kg - 85 kg), the correlation between work rates at a tangent of 45° to the curvilinear regression fit and maximal work rate was 0.96 ($p = 0.0018$). When the work rate at a tangent of 45° to the curve (an arbitrary slope index suggested by Hughson *et al.*[19] which is the increase in blood lactate concentration of 1 $mmol.l^{-1}$ for every 1 unit increase in work rate) was expressed as a percentage of maximal work rate, to correct for differences in body mass, the correlation between blood lactate accumulation and maximal work rate was insignificant ($r = 0.21$, $p = 0.6853$). These findings are indicative of a significant effect of body mass on blood lactate accumulation.

Some have suggested that the determination of workloads at MLSS might carry more validity than inferring 'thresholds' from incremental exercise testing.[3] However, by using 3 different modes of testing (rowing, cycling, skating) Beneke and von Duvillard[2] have shown that MLSS is very dependent on exercising skeletal muscle mass.

Why do work rates at fixed blood lactate concentrations predict endurance performance?

While some have correctly questioned endurance training based on WR_{fblc},[3,40] what is not clear is why WR_{fblc} correlate extremely well with endurance performance in the face of little physiological support.[3,9,14] In spite of the lack of physiological support, WR_{fblc} determinations are still advocated for elite athlete evaluation.[37] Insightfully, 20 years ago, Hagberg sounded a warning regarding the use of blood lactate concentration thresholds to predict endurance performance:[14] 'The close correlation between LT [lactate threshold] and endurance performance capacity does not mean that the relationship between them is necessarily cause-and-effect. Therefore, while it is tempting to theorise that the relationship between LT and endurance performance is cause-and-effect for these events, little data is available to indicate that this is the case. It is more likely that the blood lactate level may provide an index of some other physiological signal which is actually the mechanism limiting performance in prolonged steady state competitive events.'

A number of fundamental lines of evidence, taken together, suggest that the close relationship between WR_{fblc} and EP might not be a causal relationship, explained by physiological mechanisms specifically skeletal muscle metabolism:

1. A crucial observation is that WR_{fblc} occur at constant percentages of maximal work rate in trained athletes with low between-subject variation (coefficient of variation: 3.0% - 4.1%).[1,23,24,35,42,43] In other words, WR_{fblc} mirror maximal work rates.

2. In heterogeneous populations of trained and untrained individuals or trained and elite endurance athletes, WR_{fblc} occur at high and low percentages of maximal work rate respectively.[28,45,47] The effect of this is to inflate the correlation coefficient

between WR_{fblc} and endurance performance for pooled data.

3. The association between WR_{fblc} and endurance performance is stronger when work rate is expressed in speed or power units than VO_2 units.[1,41] This is probably because athletes with the same VO_{2max}, can have very different maximal work rates in speed or power units.[30]

4. WR_{fblc}, maximal work rate, and endurance performance are strongly correlated ($r \geq 0.85$),[46] and using linear regression endurance performance accurately predicts WR_{fblc} ($r \geq 0.85$).[34,45,47]

5. The percentage of maximal work rate at which WR_{fblc} occurs remains constant after training.[8,38,48]

6. Physiological signals related[29] and unrelated[4,50] to lactate metabolism both increase exponentially with increasing work rate.

7. Metabolites unrelated to lactate metabolism which increase exponentially with increasing work rate correlate with measures of endurance performance ($r = 0.92$).[50]

These findings suggest that consideration should be given to other non-physiological mechanisms as an explanation for the tight relationship between WR_{fblc} and EP. Specifically, consideration should be given to the idea that WR_{fblc} is, in fact, a covariate, a reflection of a major determinant of endurance performance, namely maximal work rate achieved during an incremental or performance test.[31] This is in agreement with the observation of Lacour *et al.*[23] that, because of the close interrelationships between EP, WR_{max} and WR_{fblc}, the constant percentage of maximal work rate of WR_{fblc} must be 'interpreted as being an expression of the correlation between Va_{max} (maximal work rate) and v (best middle distance track speed)'. The hypothesis that WR_{fblc} (4 $mmol.l^{-1}$) is a covariate between endurance performance and maximal work rate is illustrated in Fig. 1.

An alternative mechanism explaining why fixed blood lactate concentrations predict endurance performance

Thus far, it has been argued in this paper that there is sufficient indirect evidence, from a number of sources, to suggest that the causality proposed between WR_{fblc} and EP has not been convincingly proven by traditional models and that other mechanisms might better explain the tight relationship between WR_{fblc} and EP.

Because a significant association by itself does not infer causality, it is important to establish *a priori* possible causal relationships through evidence that has already been shown to provide a strong case for a causal relationship. Therefore, to propose an alternative hypothesis, the following questions need to be answered:

1. Are there causal relationships between EP and WR_{max}, EP and WR_{fblc} and WR_{max} and WR_{fblc}?

2. Is the exponential nature of the rise in blood lactate concentration with increasing work rate a phenomenon peculiar to curvilinear relationships such that WR_{fblc} / WR_{max} ratios are very constant, or can these constant ratios be duplicated by linear relationships?

That there is a causal relationship between EP and WR_{max} has been argued in detail elsewhere.[31,39] The argument for causality between EP and WR_{max} suggests that WR_{max} is evaluating an important aspect of EP, namely skeletal muscle power output. In other words, both EP and WR_{max} share the same physiological underpinning.[31,39]

Several independent sources separated by more than 10 years, suggest that there is little evidence to support the causality usually ascribed to the EP-WR_{fblc} relationship.[3,9,14] As has been highlighted earlier in this paper causality has been proposed based largely on the fortuitous results from MLSS findings, skeletal muscle lactate efflux studies and computer simulation results, with additional support from correlational studies and arguably the sporting success of principally 1 nation. Furthermore, because the traditional physiological explanation[44] for the relationship between EP and WR_{fblc} is at odds with the more recently proposed paradigms which provide an alternative physiological explanation for the relationship between EP and WR_{max},[31,39] it would be difficult to ascribe causality to the relationship between EP and WR_{fblc} on the basis of the Meyerhof-Hill-Wasserman hypothesis.[44]

It follows then that WR_{max} and WR_{fblc} cannot be causally related based on the traditional Meyerhof-Hill-Wasserman model. WR_{max} and WR_{fblc} can only be causally related by a different physiological mechanism. This appears unlikely and there do not seem to be data to support an alternative physiological mechanism. The only causal relationship that could be argued for is the requirement of a high skeletal muscle lactate and thus blood lactate concentration to achieve high work rates.[6]

Based on the above, the argument could be made that WR_{max} is a covariate between WR_{fblc} and EP, which is what explains the tight relationship between WR_{fblc} and EP. Consequently, by partialling out the effect of WR_{max}, the relationship between WR_{fblc} and EP will be removed or substantially reduced, particularly in homogeneous samples. The relationship between WR_{fblc} and EP in heterogeneous samples will be less affected because the percentage of maximal work rate at which WR_{fblc} occurs is highly variable.

Correlational analysis (zero-order and first-order partial) of data obtained from published reports supports the foregoing arguments (Table I).[23,24,28,30,35,42,43] The association between WR_{fblc} and endurance performance was highly significant across all events with 5 of the 6 correlation coefficients > 0.8. Of note was the highly significant correlations between WR_{fblc}, endurance performance and maximal work rate; the majority of coefficients (16/18) were > 0.8. Once the effect of WR_{max} was partialled out, the association between WR_{fblc} and endurance performance was dramatically altered. Three of the 6 partial correlation coefficients were no longer significant and 4 of the 6 partial correlation coefficients were significantly lower after controlling for WR_{max}. All the partial correlation coefficients were < 0.6, except for the swimming and 16 km road-running sample. Because the 16 km road-running sample was heterogeneous the variation in running performance, maximal work rate

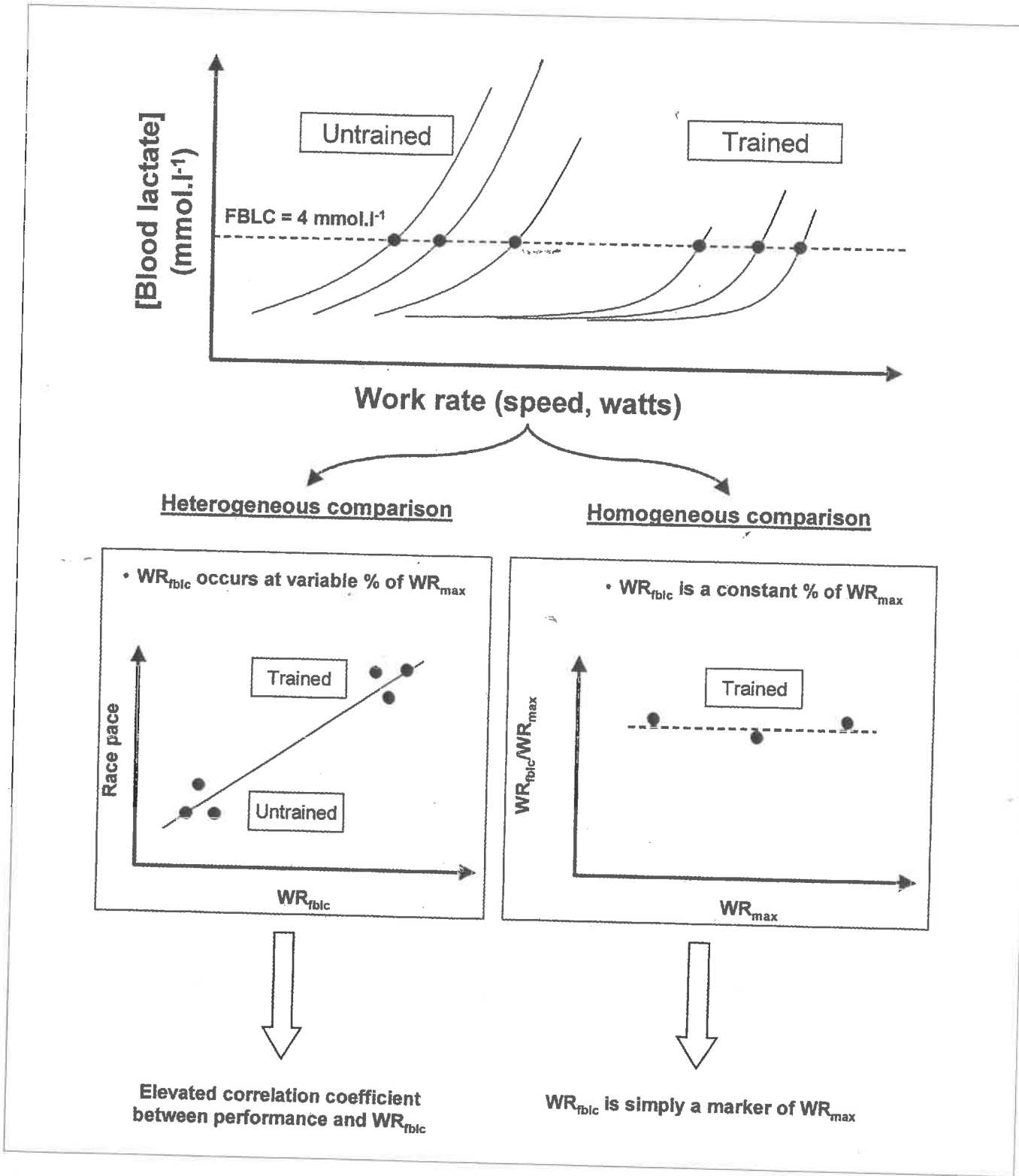

Fig. 1. Endurance performance prediction from work rates at fixed blood lactate concentrations: a mathematical not physiological phenomenon. This diagram illustrates an alternative, non-physiological mechanism why WR_{fblc} predicts endurance performance. Analysis of groups with heterogeneous athletic ability or training status, results in high correlation coefficients between WR_{fblc} and endurance performance (race pace). The reason for this is that WR_{fblc} occurs at a low percentage of WR_{max} in individuals with low athletic ability. However, for trained individuals, WR_{fblc} occurs at a high percentage of WR_{max}. The consequence of this is a large variation in the dependent (race pace) and independent (WR_{fblc}) variables, resulting in a high correlation coefficient. Homogeneous groups on the other hand, produce very similar WR_{fblc}/WR_{max} ratios. In other words, WR_{fblc} occurs at a high and constant percentage of WR_{max}. Accordingly, WR_{fblc} is a relatively accurate surrogate measure of WR_{max}, and since WR_{max} is a significant predictor of endurance performance, WR_{fblc} correlates strongly with endurance performance. (FBLC = fixed blood lactate concentration, WR_{fblc} = work rate at fixed blood lactate concentration, WR_{max} = maximal work rate).

TABLE I. Zero-order and first order partial correlation coefficients between performance measures and endurance performance

Correlation coefficient type and relationship		Endurance event: Swim† 400 m (*N* = 16)	Track running‡ 1 500 m (*N* = 55)	Track running‡ 3 000 m (*N* = 28)	Track running‡ 5 000 m (*N* = 13)	Road running 10 km§ (*N* = 10)	Road running 16 km¶ (*N* = 20)
Zero-order	WR_{fblc} v. EP	0.92**	0.83**	0.86**	0.63**	0.82**	0.91**
	WR_{max} v. EP	0.93**	0.87**	0.84**	0.85**	0.87**	0.88**
	WR_{fblc} v. WR_{max}	0.87**	0.89**	0.82**	0.65**	0.98**	0.87**
1st-order partial*	WR_{fblc} v. EP	0.64‖,**	0.26‖,††	0.56‖,**	0.19††	0.30††	0.65‖,**

See text for references, data reported as correlation coefficients, EP = endurance performance ($m.s^{-1}$); WR_{fblc} = work rate at blood lactate concentration of 4 $mmol.l^{-1}$ ($m.s^{-1}$); WR_{max} = maximal work rate ($m.s^{-1}$); WR_x obtained through linear interpolation of the two closest measured values; *partialled out WR_{max}, †Homogeneous sample: coefficient of variation (CV) for WR_x/WR_{max} = 2.7%, WR_{max} = critical velocity determined in swimming pool or swimming flume; ‡Homogeneous sample: CV for WR_x/WR_{max} = 3.4%, WR_{max} = treadmill speed at VO_{2max} determined from an intermittent incremental test; §Homogeneous sample: CV for WR_x/WR_{max} = 2.9%, WR_{max} = treadmill speed at VO_{2max} calculated from running speed and VO_2; ¶Heterogeneous sample: CV for WR_x/WR_{max} = 16.2%, WR_{max} = treadmill speed at exhaustion during incremental test; ‖ significant difference between zero-order and first-order partial correlation coefficients ($p < 0.04$); **$p < 0.05$; ††$p > 0.05$.

and WR_{fblc} as a percentage of maximal work rate was inflated.[28] Consequently, it would be expected that removing the effect of maximal work rate would not reduce the association between WR_{fblc} and endurance performance to the level of insignificance. The much lower variation in WR_{fblc} as a percentage of maximal work could explain why the association between WR_{fblc} and endurance performance was not affected similarly in the homogeneous track running group.[23,24,35] The maximal work rate for the swimming sample was determined by means of a critical velocity test,[42,43] which is not a true maximal test. Critical velocity measurements in swimmers is 4 - 6% lower than 400 m freestyle swimming speeds.[42,43] The use of the critical velocity test might have resulted in greater variation in WR_{fblc}/WR_{max} ratios. Lacour *et al.*[23] emphasise that in order to achieve constant WR_{fblc}/WR_{max} ratios, a valid and reliable maximal testing procedure is required.

The correlational analysis reported in Table I is noteworthy in that as predicted the relationship between WR_{fblc} and EP was significantly modified by WR_{max}. Moreover, the validity of the results of the correlational analysis are increased because of the *a priori* examination of the causality between WR_{fblc}, WR_{max} and EP.

Is the constant WR_{fblc}/WR_{max} ratio a peculiar phenomenon of curvilinear relationships?

A number of studies, extending across gender and athletic ability, have shown that exponential relationships (blood lactate concentration v. speed) are more closely related to EP than linear relationships (oxygen consumption v. speed).[10,11,23,30,32,49] Correlation coefficients between running economy and running speed over distances of 1.5 km - 42.2 km have ranged from 0.08 to 0.64 (median: $r = 0.57$).[10,11,23,30,32,49] In contrast, correlation coefficients between WR_{fblc} and running speed over the same distances have ranged from 0.63 to 0.98 (median: $r = 0.91$).[10,11,23,30,32,49]

To examine this postulate further, unpublished data (Cook I and van Wyk GI, University of Pretoria, 1991) from elite and competitive male triathletes was used. Using the same data, the relationship between blood lactate concentration and power output was described in a curvilinear and linear manner (Figs 2a and b). The latter was achieved by linearising the abscissa (transformed power output = $e^{c \cdot \text{power output}}$).[19] Work rates and gradients at fixed blood lactate concentrations of 1.5, 2.0, 4.0, 6.0 and 8.0 $mmol.l^{-1}$ were determined for both curvilinear and linear relationships. The coefficient of variation for the WR_{fblc}/WR_{max} ratios and gradients at the fixed blood lactate concentrations were calculated for both curvilinear and linear relationships. The results are presented in Figs 2c-e.

For both the curvilinear and linear relationships the mean WR_{fblc}/WR_{max} ratios increased with increasing blood lactate concentrations (Fig. 2c). However, as expected the variability, expressed as the confidence intervals for the mean, decreased for the curvilinear relationship, but increased for the linear relationship (Fig. 2c). In other words, the variability in the WR_{fblc}/WR_{max} ratios decreased with increasing blood lactate concentration for the curvilinear relationship but increased for the linear relationship. The variability, expressed as a coefficient of variation, is shown in Fig. 2d, and, as expected, a clear pattern emerges of decreasing variability of gradients and WR_{fblc}/WR_{max} ratios with increasing blood lactate concentrations for the curvilinear relationship, but not the linear relationship. The much higher variability for the linear gradients compared with the curvilinear gradients is also evident (Fig. 2d). As expected, a tight relationship exists between the variability of the gradients with increasing blood lactate concentrations and the WR_{fblc}/WR_{max} ratios for the curvilinear relationship but not the linear relationship (Fig. 2e). In other words, with the exponential accumulation of blood lactate as work rate increases, the slopes or gradients at fixed blood lactate concentrations become steeper, but the variability of the gradients between subjects decreases. The resultant effect of the decreasing variability in gradients is a concomitant decrease in the variability of the WR_{fblc}/WR_{max} ratios with increasing blood lactate accumulation. Therefore, in a homogeneous sample of trained athletes, the WR_{fblc}/WR_{max} ratios will be constant such that the ratios are a surprisingly accurate reflection of WR_{max}.

The significance of this analysis was that, using the same data, a clear distinction could be shown between the level of variation in the gradients and WR_{fblc}/WR_{max} ratios for the curvi-

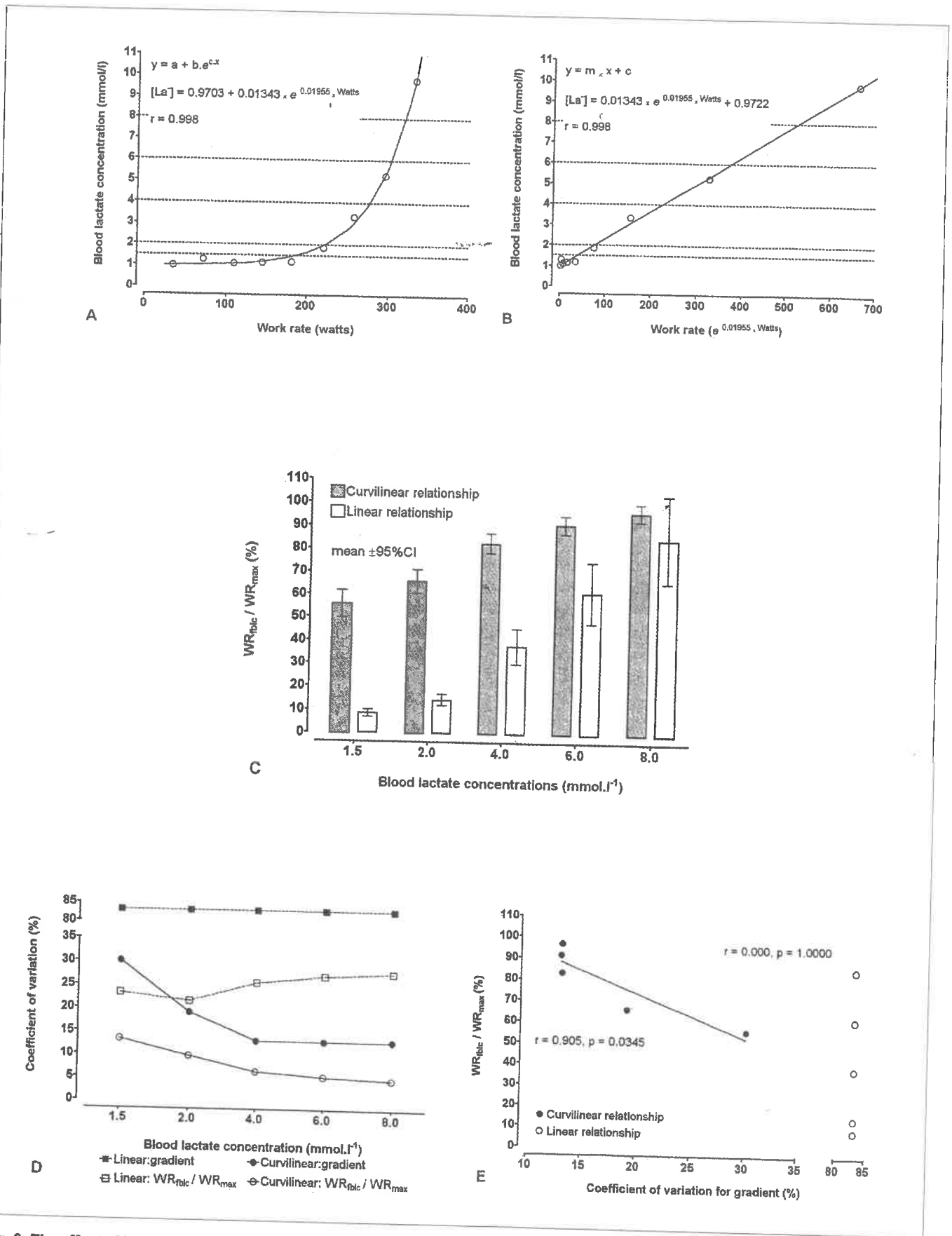

Fig. 2. The effect of linear and curvilinear relationships on the variability of gradients and WR_{fblc}/WR_{max} ratios obtained from work rates at fixed blood lactate concentrations. See text for explanation.

linear and linear relationships. Importantly, a tight relationship is seen between the decreasing variation in gradients and increasing WR_{fblc}/WR_{max} ratios. The argument could be made that the mechanism for the constant WR_{fblc}/WR_{max} ratios in a homogeneous sample is the result of the peculiar properties of the exponential rise in blood lactate accumulation with increasing work rate.

Slope indices and prediction of endurance performance (Figs 3a - e)

Tokmakidis and Leger found that the work rate at a slope determined from the log-log transformation of the blood lactate concentration-work rate relationship (Fig. 3e) produced stronger and more consistent correlations with running performance (300 m - 42.2 km) than a slope index at a 45° tangent (Fig. 3d).[41] This finding would be expected because firstly, only the data points at the steep portion of the curve are used for the log-log method (Fig. 3e). Secondly, these selected points are used to obtain a work rate at an individualised fixed blood lactate concentration (Fig. 3e) which will always fall on the steep portion of the curve. In other words, the method of obtaining a work rate using the log-log transformation method (Fig. 3e) is simply producing an individualised WR_{fblc}. Therefore, even though the blood lactate concentrations at these individualised work rates would differ between individuals, the inter-correlations between WR_{max}, individualised WR_{fblc}, and standard WR_{fblc} ranging from 2.0 mmol.l^{-1} to 6.0 mmol.l^{-1} would be high.[45-47] In fact, there is no reason why work rates at blood lactate concentrations > 6 mmol.l^{-1} would not correlate significantly and strongly with WR_{max} and EP.

In this regard, Tokmakidis and Leger found that of all the blood lactate concentration models that they investigated, the work rate determined from the gradient of the upper segment of the log-log transformation (Fig. 3e) was the highest of all the methods employed (17.36 km.hr^{-1} v. 15.09 km.hr^{-1}, $p < 0.01$) and correlated the best with WR_{max} ($r = 0.90$, $p < 0.01$).[41] Using data obtained from treadmill testing, Weltman *et al.* have reported high inter-correlations ($r = 0.85$ to $r = 0.99$) between WR_{fblc} (2.0, 2.5, and 4 mmol.l^{-1}) and WR_{max} in trained male runners and sedentary female subjects.[45-47] Furthermore, WR_{fblc} (2.0, 2.5, and 4 mmol.l^{-1}) and WR_{max} were all significant predictors of average running speed for 3 200 m ($r = 0.84$ to $r = 0.94$, $p < 0.01$).[45,47] Lastly, using unpublished data (Cook I and van Wyk GJ, University of Pretoria, 1991), inter-correlations between WR_{max}, WR_{fblc} at blood lactate concentrations of 4.0, 6.0 and 8.0 mmol.l^{-1}, and the work rate determined from the gradient of the upper segment of the log-log transformation (Fig. 3e), ranged from $r = 0.837$ to $r = 0.997$ ($p < 0.04$). The work rate determined from the gradient of the upper segment of the log-log transformation was significantly related to WR_{max} ($r = 0.980$, $p = 0.001$) and WR_{fblc} at blood lactate concentrations of 4, 6 and 8 mmol.l^{-1} ($r = 0.837$, $r = 0.887$ and $r = 0.914$ respectively, $p < 0.04$).

However, unlike the work rate determined from the gradient of the upper segment of the log-log transformation (Fig. 3e), the 45° tangent method (Fig. 3d) removes any reference to the maximal work rate by simply providing a gradient at a particular point on the blood lactate concentration - work rate curve. Similarly, unpublished results (Dennis SC, Albertyn R, Bosch AN, Noakes TD, University of Cape Town, 1991) found that the work rate (mmol.min^{-1} VO_2) at a slope of 0.1 mmol per mmol.min^{-1} did not predict endurance performance ($r = -0.03$ to $r = -0.43$, $p > 0.05$) for elite and competitive endurance athletes. In other words, the blood lactate concentration - work rate curves independent of WR_{max} were not related to endurance performance.

Finally, using unpublished data (Cook I and van Wyk GI, University of Pretoria, 1991), WR_{fblc} calculated by linear interpolation (Fig. 3b) or numerical methods (Fig. 3c) correlated significantly with maximal work rate ($r = 0.86$ to $r = 0.84$, $p < 0.04$). Moreover, both methods of determining WR_{fblc} (linear interpolation and numerical methods) were significantly related to each other ($r = 0.996$, $p < 0.0001$; mean difference = 0.98 W). Similarly, the log-log method (Fig. 3e) was significantly related to maximal work rate ($r = 0.98$, $p = 0.0005$) and significantly related to WR_{fblc} using linear interpolation or numerical methods ($r = 0.83$ to $r = 0.85$, $p < 0.05$). However, the slopes at WR_{fblc}, from either linear interpolation or numerical methods, were not significantly related to maximal work rate ($r = -0.29$ to $r = -0.30$, $p > 0.5$).

The results presented in the preceding discussion, taken together, suggest firstly that a work rate at any fixed blood lactate concentration will correlate significantly with WR_{max} and EP, particularly if the blood lactate concentration falls on the steep portion of the blood lactate - work rate curve. Secondly, once any reference to WR_{max} is removed, associations between blood lactate concentration indices and maximal work rate are dramatically reduced.

Conclusion

Because of the exponential rise in lactate R_a and lactate R_d with incremental work rates,[19] rising blood lactate concentrations are expressed as a continuous, single rate or monotonic, exponential function, $y = a + b.e^{c.x}$.[19] The growth constant c results in an ever-increasing slope, such that small changes in the abscissa (work rate) result in large changes in the ordinate (blood lactate concentration). The higher the work rate the steeper the slope such that the magnitude of the slopes tend to infinity as work rates increase. Consequently, at high work rates, blood lactate concentration-work rate curves between subjects can visually appear very similar. It is this property of exponential functions which results in very constant WR_{fblc}/WR_{max} ratios. Although the exponential blood lactate concentration-work rate curve does have a plausible physiological explanation, attempting to infer dynamic physiological processes or oxygen status from this curve is not supported by data. This being the case, a causal link cannot be established between WR_{fblc} and skeletal muscle metabolism on the basis of the relationship between WR_{fblc} and endurance performance.

This paper suggests that WR_{fblc} is a marker of maximal work rate, as alluded to by Hagberg[14] and Lacour *et al.*[23] This would explain the tight relationship between endurance performance and WR_{fblc}. Re-analyses of existing data sets, containing blood lactate concentration - work rate results from incremental tests,

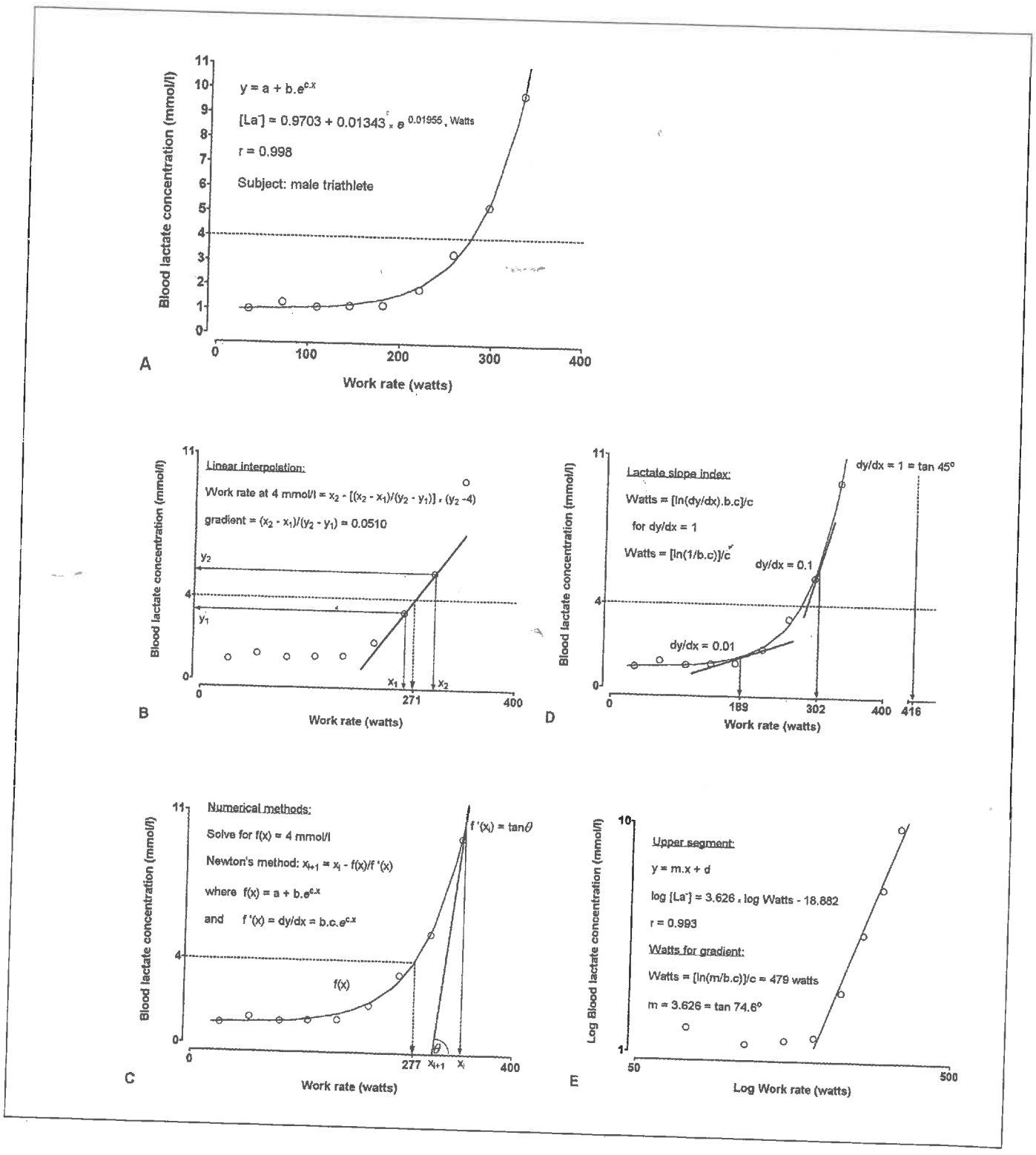

Fig. 3. Some commonly used methodologies to determine WR_{max}-dependent and -independent blood lactate accumulation indices as markers of endurance performance. Fig. 3A shows typical raw data and the line of best fit for an exponential plus constant model. Fig. 3B illustrates linear interpolation routinely used to determine WR_{fblc} when the desired work rate falls between two blood lactate - work rate data points. Alternatively, when the required work rate falls beyond two blood lactate - work rate data points, an equation for linear extrapolation is easily derived. Fig. 3C shows a common iterative, numerical method (Newton's Method) that is used to determine a work rate (x-value) for a given blood lactate concentration (y-value). Fig. 3D demonstrates the calculation of work rates at different slopes or slope indices for an exponential plus constant model. Fig. 3E requires that the original blood lactate concentration - work rate data is transformed by taking the base-10 logarithms of the abscissa and ordinate. The linear regression equation for the transformed axes is determined and the gradient of the linear equation is used to determine a work rate, using coefficients of the exponential plus constant model. For more detail regarding the exponential plus constant model (Fig. 3A) and the methods in Figs 3D and E, readers are referred to Hughson et al.[19] and Tokmakidis and Leger.[41] For more detail on numerical methods such as Newton's Method (Fig. 3C), readers are referred to texts dealing with numerical methods.

are warranted. Importantly, only data sets containing valid measures of maximal work rate and endurance performance should be re-analysed. Moreover, an obvious prediction of the hypothesis proposed in this paper could be tested using either existing data sets or by conducting new studies. The prediction is that work rates at fixed minute ventilation volumes and fixed blood concentrations of catecholamines, ammonia and human growth hormone would similarly predict endurance performance because of the exponential increase of these parameters with increasing work rate.[4,9,29,50] In fact, any parameter which increases exponentially with increasing work rate would predict endurance performance. If the mechanism argued in this paper is confirmed by further analyses and investigation, the efficacy of obtaining blood lactate measurements from athletes as a marker of endurance performance must be questioned

REFERENCES

1. Barlow K, Weltman A, Schurrer R, Henritze J. Prediction of maximal effort bicycle ergometer endurance performance. *Int J Sports Med* 1985; **6:** 190-6.
2. Beneke R, von Duvillard SP. Determination of maximal lactate steady state response in selected sports events. *Med Sci Sports Exerc* 1996; **28:** 241-6.
3. Brooks GA, Fahey TD, White TP. *Exercise Physiology: Human Bioenergetics and its Applications.* Mountain View, CA: Mayfield Publishing, 1996.
4. Chwalbinska M, Krysztofiak F, Ziemba A, Nazar K, Kaciuba-Uscilko H. Threshold increases in plasma growth hormone in relation to plasma catecholamine and blood lactate concentrations during progressive exercise in endurance-trained athletes. *Eur J Appl Physiol* 1996; **73:** 117-20.
5. Colwin CM. *Swimming into the 21st Century.* Champaign, IL: Leisure Press, 1992.
6. Connett RJ, Gayeski TE, Honig CR. Energy sources in fully aerobic rest-work transitions: a new role for glycolysis. *Am J Physiol* 1985; **248:** H922-9.
7. Davis JA, Caiozzo VJ, Lamarra N, *et al.* Does the gas exchange anaerobic threshold occur ar a fixed blood lactate concentration of 2 or 4mM? *Int J Sports Med* 1983; **4:** 89-93.
8. Denis C, Dormois D, Lacour JR. Endurance training, VO_{2max}, and OBLA: a longitudinal study of two different age groups. *Int J Sports Med* 1984; **5:** 167-73.
9. Dennis SC, Noakes TD. Physiological and metabolic responses to increasing work rates: relevance for exercise prescription. *J Sports Sci* 1998; **16:** S77-84.
10. Farrell PA, Wilmore JH, Coyle EF, Billing JE, Costill DL. Plasma lactate accumulation and distance running performance. *Med Sci Sports* 1979; **11:** 338-44.
11. Fay L, Londeree BR, Lafontaine TP, Volek MR. Physiological parameters related to distance running performance in female runners. *Med Sci Sports Exerc* 1989; **21:** 319-24.
12. Gaesser GA, Poole DC. Blood lactate during exercise: time course of training adaptation in humans. *Int J Sports Med* 1988; **9:** 284-8.
13. Gladden LB. Lactate metabolism: a new paradigm for the third millennium. *J Physiol* 2004; **558:** 5-30.
14. Hagberg JM. Physiological implications of the lactate threshold. *Int J Sports Med* 1984; **5:** 106-9.
15. Heck H, Mader A, Hess G, *et al.* Justification of the 4-mmol/l lactate threshold. *Int J Sports Med* 1985; **6:** 117-30.
16. Helgerud J. Maximal oxygen uptake, anaerobic threshold and running economy in women and men with similar performances level in marathons. *Eur J Appl Physiol* 1994; **68:** 155-61.
17. Hollmann W. Historical remarks on the development of the aerobic-anaerobic threshold up to 1966. *Int J Sports Med* 1985; **6:** 109-16.
18. Hollmann W. 42 Years ago - Development of the concepts of ventilatory and lactate threshold. *Sports Med* 2001; **31:** 315-20.
19. Hughson RL, Weisiger KH, Swanson GD. Blood lactate concentration increases as a continuous function in progressive exercise. *J Appl Physiol* 1987; **62:** 1975-81.
20. Jorfeldt L, Juhlin-Dannfelt A, Karlsson J. Lactate release in relation to tissue lactate in human skeletal muscle during exercise. *J Appl Physiol* 1978; **44:** 350-2.
21. Juel C. Lactate-proton co-transport in skeletal muscle. *Physiol Rev* 1997; **77:** 321-58.
22. Karlsson J, Jacobs I. Onset of blood lactate accumulation during muscular exercise as a threshold concept. I. Theoretical considerations. *Int J Sports Med* 1982; **3:** 190-201.
23. Lacour JR, Padilla M, Barthelemy JC, Dormois D. The energetics of middle-distance running. *Eur J Appl Physiol* 1990; **60:** 38-43.
24. Lacour JR, Padilla M, Chatard JC, Arsac L, Barthelemy JC. Assessment of running velocity at maximal oxygen uptake. *Eur J Appl Physiol* 1991; **62:** 77-82.
25. Maassen N, Busse MW. The relationship between lactic acid and work load: a measure for endurance capacity or an indicator of carbohydrate deficiency? *Eur J Appl Physiol* 1989; **58:** 728-37.
26. MacRae HS, Dennis SC, Bosch AN, Noakes TD. Effects of training on lactate production and removal during progressive exercise in humans. *J Appl Physiol* 1992; **72:** 1649-56.
27. Mader A, Heck H. A theory of the metabolic origin of 'anaerobic threshold'. *Int J Sports Med* 1986; **7:** 45-65.
28. Marti B, Abelin T, Howald H. A modified, fixed blood lactate threshold for estimating running speed for joggers in 16-km races. *Scandinavian Journal of Sports Science* 1987; **9:** 41-5.
29. Mazzeo RS, Marshall P. Influence of plasma catecholamines on the lactate threshold during graded exercise. *J Appl Physiol* 1989; **67:** 1319-22.
30. Morgan DW, Baldini FD, Martin PE, Kohrt WM. Ten kilometer performance and predicted velocity at VO_{2max} among well-trained male runners. *Med Sci Sports Exerc* 1989; **21:** 78-83.
31. Noakes TD. JB Wolffe Memorial Lecture. Challenging beliefs: ex Africa semper aliquid novi. *Med Sci Sports Exerc* 1997; **29:** 571-90.
32. Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the VO_{2max} test predicts running performance. *J Sports Sci* 1990; **8:** 35-45.
33. O'Brien MJ, Viguie CA, Mazzeo RS, Brooks GA. Carbohydrate dependence during marathon running. *Med Sci Sports Exerc* 1993; **25:** 1009-17.
34. Olbrecht J, Madsen O, Mader A, Liesen H, Hollmann W. Relationship between swimming velocity and lactic concentration during continuous and intermittent training exercises. *Int J Sports Med* 1985; **6:** 74-7.
35. Padilla S, Bourdin M, Barthelemy JC, Lacour JR. Physiological correlates of middle-distance running performance. A comparative study between men and women. *Eur J Appl Physiol* 1992; **65:** 561-6.
36. Potteiger JA, Nickel GL, Webster MJ, Haub MD, Palmer RJ. Sodium citrate ingestion enhances 30 km cycling performance. *Int J Sports Med* 1996; **17:** 7-11.
37. Sports Information and Science Agency High Performance Programme. *Cycling: Physical Protocol.* Pretoria: South African Sports Information and Science Agency, 2000.
38. Sjodin B, Jacobs I, Svedenhag J. Changes in onset of blood lactate accumulation (OBLA) and muscle enzymes after training at OBLA. *Eur J Appl Physiol* 1982; **49:** 45-57.
39. St Clair Gibson A, Noakes TD. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br J Sports Med* 2004; **38:** 797-806.
40. Stegmann H, Kindermann W. Comparison of prolonged exercise tests at the individual anaerobic threshold and the fixed anaerobic threshold of 4 mmol.l(-1) lactate. *Int J Sports Med* 1982; **3:** 105-10.
41. Tokmakidis SP, Leger LA. Comparison of mathematically determined blood lactate and heart rate 'threshold' points and relationship with performance. *Eur J Appl Physiol* 1992; **64:** 309-17.
42. Wakayoshi K, Yoshida T, Udo M, *et al.* Does critical swimming velocity represent exercise intensity at maximal lactate steady state? *Eur J Appl Physiol* 1993; **66:** 90-5.
43. Wakayoshi K, Yoshida T, Udo M, *et al.* A simple method for determining critical speed as swimming fatigue threshold in competitive swimming. *Int J Sports Med* 1992; **13:** 367-71.
44. Weltman A. *Current Issues in Exercise Science: The Blood Lactate Response to Exercise.* Champaign, IL: Human Kinetics, 1995.
45. Weltman A, Snead D, Seip R, *et al.* Prediction of lactate threshold and fixed blood lactate concentrations from 3200-m running performance in male runners. *Int J Sports Med* 1987; **8:** 401-6.
46. Weltman A, Snead D, Stein P, *et al.* Reliability and validity of a continuous incremental treadmill protocol for the determination of lactate threshold, fixed blood lactate concentrations, and VO_{2max}. *Int J Sports Med* 1990; **11:** 26-32.
47. Weltman J, Seip R, Levine S, *et al.* Prediction of lactate threshold and fixed blood lactate concentrations from 3200-m time trial running performance in untrained females. *Int J Sports Med* 1989; **10:** 207-11.
48. Westgarth-Taylor C, Hawley JA, Rickard S, *et al.* Metabolic and performance adaptations to interval training in endurance-trained cyclists. *Eur J Appl Physiol* 1997; **75:** 298-304.
49. Yoshida T, Udo M, Iwai K, *et al.* Significance of the contribution of aerobic and anaerobic components to several distance running performances in female athletes. *Eur J Appl Physiol* 1990; **60:** 249-53.
50. Yuan Y, Chan KM. A longitudinal study on the ammonia threshold in junior cyclists. *Br J Sports Med* 2004; **38:** 115-9.