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REVIEW ARTICLE

Training to Enhance the Physiological Determinants of Long-Distance Running Performance
Can Valid Recommendations be Given to Runners and Coaches Based on Current Scientific Knowledge?

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Abstract

This article investigates whether there is currently sufficient scientific knowledge for scientists to be able to give valid training recommendations to long-distance runners and their coaches on how to most effectively enhance the maximal oxygen uptake, lactate threshold and running economy. Relatively few training studies involving trained distance runners have been conducted, and these studies have often included methodological factors that make interpretation of the findings difficult. For example, the basis of most of the studies was to include one or more specific bouts of training in addition to the runners' 'normal training', which was typically not described or only briefly described. The training status of the runners (e.g. off-season) during the study period was also typically not described. This inability to compare the runners' training before and during the training intervention period is probably the main factor that hinders the interpretation of previous training studies. Arguably, the second greatest limitation is that only a few of the studies included more than one experimental group. Consequently, there is no comparison to allow the evaluation of the relative efficacy of the particular training intervention. Other factors include not controlling the runners' training load during the study period, and employing small sample sizes that result in low statistical power. Much of the current knowledge relating to chronic adaptive responses to physical training has come from studies using sedentary

individuals; however, directly applying this knowledge to formulate training recommendations for runners is unlikely to be valid. Therefore, it would be difficult to argue against the view that there is insufficient direct scientific evidence to formulate training recommendations based on the limited research. Although direct scientific evidence is limited, we believe that scientists can still formulate worthwhile training recommendations by integrating the information derived from training studies with other scientific knowledge. This knowledge includes the acute physiological responses in the various exercise domains, the structures and processes that limit the physiological determinants of long-distance running performance, and the adaptations associated with their enhancement. In the future, molecular biology may make an increasing contribution in identifying effective training methods, by identifying the genes that contribute to the variation in maximal oxygen uptake, the lactate threshold and running economy, as well as the biochemical and mechanical signals that induce these genes. Scientists should be cautious when giving training recommendations to runners and coaches based on the limited available scientific knowledge. This limited knowledge highlights that characterising the most effective training methods for long-distance runners is still a fruitful area for future research.

The physiological determinants of long-distance running performance have been well documented, and include maximal oxygen uptake ($\dot{V}O_{2max}$),[1-3] lactate threshold[3-5] and running economy (figure 1).[3,6,7] These three determinants explain >70% of the between-subject variance in long-distance running performance.[8] Although athletic performance is known to be related to genetic-[9,10] and training-related[11] factors, the former is normally a fixed factor (gene doping[12] being the exception). In contrast, physical training may exert profound effects on physiological adaptation and athletic performance.[13-15] Distance runners often seek the most effective training methods to enhance performance,[16] and is probably most evident in elite runners where the rate of training adaptation and performance enhancement may have reached a plateau.[17] Since $\dot{V}O_{2max}$, lactate threshold and running economy have been regarded as the most important physiological determinants of long-distance running performance, effective training programmes for long-distance runners should focus on their enhancement.[18] However, it is unclear which training methods are most effective.[16,17,19,20]

Current training methods have largely developed from the trial-and-error approach of runners and several prominent running coaches, whereas contributions from scientists have been relatively small.[11,21,22] This may be due to the reluctance of runners and coaches to acknowledge the potential merit of scientific research for improving training methods, or because scientific knowledge is too limited to allow worthwhile contributions. Sports scientists sometimes give training advice to runners during sports science support work, or after runners have participated in experimental research. Awareness of the current state of scientific knowledge in this area should therefore be valuable.

The main purpose of this article is to evaluate whether there is currently sufficient scientific knowledge to allow scientists to give valid training recommendations to long-distance runners and their coaches on how to most effectively enhance $\dot{V}O_{2max}$, lactate threshold and running economy. This article focuses mainly on training intervention studies (summarised in table I) that involved runners and reported changes in these physiological determinants of long-distance running performance. Training intervention studies involving other athletes and sedentary individuals have been highlighted where deemed appropriate.
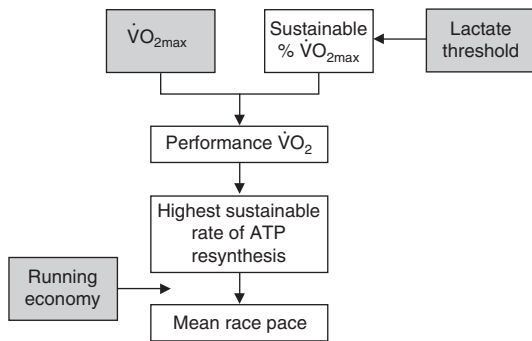
Fig. 1. Flow diagram showing that long-distance running performance is predominantly determined by maximal oxygen uptake ($\dot{V}O_{2max}$), the lactate threshold (determines the fraction of $\dot{V}O_{2max}$ that can be sustained) and running economy. Performance oxygen uptake ($\dot{V}O_2$) represents the highest mean $\dot{V}O_2$ that can be sustained during the race. Running economy refers to how efficient the runner is at converting available energy into running speed. **ATP** = adenosine triphosphate.

1. Training and Adaptation

Training-induced stress imposed on a physiological process or structure is the stimulus for adaptation resulting in an enhanced functional capacity.[44] The training frequency of distance runners is typically relatively constant,[29] with elite and sub-elite runners performing approximately 10–14 sessions per week.[45] Training stress is therefore predominantly imposed by the manipulation of training intensity and duration.[46] Chronic adaptation will occur only if the training intensity is sufficient to elicit an adaptive response.[47] This minimum intensity has been termed the training intensity threshold.[17] Similarly, for a given training intensity there must be a training duration threshold, below which no chronic adaptive response occurs. The intensity and duration thresholds combined form the adaptation threshold, which must be surpassed to enhance any of the physiological determinants of long-distance running performance highlighted in figure 1. A model for performance enhancement based on this threshold concept is shown in figure 2. Although the thresholds define the minimum training intensity and duration required to elicit a chronic adaptive response, these thresholds may not be optimal for enhancing a particular physiological performance determinant. Distance runners should seek optimal training inten-sities and durations rather than threshold values, since training protocols that are not optimal would reduce the overall efficiency of the training process.[48] Optimising training methods may also negate the need to continually increase the training volumes of runners to extreme levels, which has been implicated in premature stagnation of performance.[49]

2. Training Quantification

Training load is the product of intensity, duration and frequency.[50] Although training loads for runners may be prescribed based on training volume (duration or hours multiplied by frequency), such as kilometres or hours run per week,[51] this approach does not incorporate exercise intensity, which has been suggested to be the most important and heavily debated of the variables relating to exercise prescription.[52-54] Training intensity has been prescribed based on percentages of maximum velocity or race pace,[55] made more practical by the relatively recent development of global position system (GPS) monitors.[56,57] Training intensity has also been based on physiological measures such as percentages of the maximal heart rate or $\dot{V}O_{2max}$.[58] The common factor to physiological measures is that they are all indicators of relative physiological strain. However, the relative percentage method has been criticised, since it does not take into account between-subject differences in lactate accumulation at particular percentages of maximal heart rate and $\dot{V}O_{2max}$, and may therefore be a poor indicator of physiological strain.[58] A more complex model of training prescription and quantification based on heart rate and training duration has been developed and termed the training impulse (TRIMP).[59] This method has been further refined by the inclusion of a weighting factor based on blood lactate concentration.[60]

Several authors have suggested that some physiological responses can be used to demarcate training zones, with the cumulative time spent in each of these training zones being indicative of the total physiological strain over a given training period.[54,61] For example, the blood lactate response to

Table I. Summary of 23 training intervention studies that have reported changes in the maximal oxygen uptake ($\dot{V}O_{2max}$), lactate threshold (or onset of blood lactate accumulation [OBLA]) or running economy of trained distance runners. Studies are ordered in relation to the mean relative $\dot{V}O_{2max}$ of the runners participating in the studies

Study	Subjects[a]	Age (y) [mean ± SD][c]	Initial $\dot{V}O_{2max}$ (mL/kg/min) [mean ± SD][c]	Study duration (wk)	Weekly training	Mean % improvement[b]			Comments
						$\dot{V}O_{2max}$	LT/OBLA	RE	
Billat et al.[23]	8 M MDR/LDR	24 ± 3	71.2 ± 5.0	8	0-4wk: 1 × vOBLA, 1 × v$\dot{V}O_{2max}$, 4 × 60-70 v$\dot{V}O_{2max}$, 5-8wk: 1 × vOBLA, 3 × v$\dot{V}O_{2max}$, 2 × 60-70 v$\dot{V}O_{2max}$ (vOBLA ≈85% v$\dot{V}O_{2max}$)	2.1 -0.4	1.1 3.4	6.1* 7.7*	85 km/wk throughout study. Runners did only 85-90 km/wk easy running 2y before study
Sjödin et al.[24]	8 M MDR/LDR	20 (18-25b)	68.7 ± 2.6	14	1 × vOBLA (20 min) + normal training (vOBLA ≈85% v$\dot{V}O_{2max}$)	2.2	4.3*	2.8*	Runners normal training included supra-OBLA velocities. Study conducted in the off-season
Lehmann et al.[25]	8 MDR/LDR	33 ± 7	66.8 ± 5.6	4	6 d/wk; 90-98% training volume at 50-70% long-distance race pace; the rest high-intensity intervals		0.6		Training volume increased by ≈33% each week
Priest and Hagan[14]	12 M CCR	21 ± 3	66.0 ± 5.9 51.7 ± 4.8	7	4 × 104% 10km[d] pace 4 × 109% 10km[d] pace	6.2* 12.3*			Training was at the estimated maximum steady-state (running velocity at 2.2 mmol/L blood lactate). Normal training not specified
Hamilton et al.[27]	10 M DR	28 ± 8	66.0 ± 7.0	5-7	G1: normal endurance training (controls) G2: 1-3 × tethered maximal effort treadmill runs at a 5% grade and maximal effort single-leg jumps replaced part of normal training		0.5 4.0e		Number of resistance training sessions dependent on subject availability
Acevedo and Goldfarb[28]	7 M LDR	22 ± 3	65.3 ± 6.2	8	1 × 90-95% HRmax intervals (85-95% $\dot{V}O_{2max}$); 2 × fartlek around 10km pace; 3-4 × LSD	0.7	5.7*		Normal training 80-105 km/wk at 8-19km per session. Normal training maintained except 3d LSD training was substituted for higher intensity training
Tanaka et al.[29]	20 M MDR	19-23c	64.4 ± 3.8	17	2 or more × vLT or slightly above vLT (60-90 min) + normal training (vLT = 70 ± 5% $\dot{V}O_{2max}$)	4.8*	3.8*		Normal training increased from 90 km/wk to 120 km/wk for study period. Normal training intensity not specified

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Table I. Contd

Study	Subjects[a]	Age (y) [mean ± SD][c]	Initial $\dot{V}O_{2max}$ (mL/kg/min) [mean ± SD][c]	Study duration (wk)	Weekly training	Mean % improvement[b] $\dot{V}O_{2max}$	LT/OBLA	RE	Comments
Daniels et al.[30]	15 DR	22 ± 3	63.9 ± 4.1	8	Increase in training volume from 20 to 30km easy running (2mo control period) to 50–70km	0.0		–0.7	5 subjects = continuous fast runs; 5 subjects = 100–600m sprints; 5 subjects = 600–1200m runs (groups pooled for analyses). Normal training not specified
Paavolainen et al.[31]	10 M CCR	23 ± 3	63.7 ± 2.7	9	G1: normal training (controls) G2: 32% of normal training replaced with sport-specific explosive strength training	4.9* −1.3	0.8 1.7	−1.0 7.7*	Endurance training = 84% <vLT (62% $\dot{V}O_{2max}$) and 16% >vLT (86% $\dot{V}O_{2max}$). Study conducted in the off-season
Smith et al.[32]	5 M MDR	23 ± 4	61.5 ± 6.6	4	2 × intervals at $v\dot{V}O_{2max}$; 1 × continuous at 60% $v\dot{V}O_{2max}$	4.9			Normal training included LSD, tempo, speed and over-speed work, and weight training
Smith et al.[33]	18 M MDR/LDR/Tr	25 ± 7	61.4 ± 5.2	4	2 × intervals at $v\dot{V}O_{2max}$; 1 × continuous at 60% $v\dot{V}O_{2max}$[f]	5.0		2.0	Normal training not specified
Slawinsky et al.[34]	6 LDR	27 ± 4	61.2 ± 6.0	8	2 × vΔ50 intervals (≈93% $v\dot{V}O_{2max}$); 3 × continuous at 60–70% $v\dot{V}O_{2max}$	0.7	2.5	3.6*	Normal training not specified. Study conducted during return to training after recovery from competition period
Mikesell and Dudley[35]	7 M LDR	24 ± 9	61.1 ± 3.7	6	3 × 'all-out' runs (40 min) eliciting heart rates ≥190 bpm; 3 × cycling intervals at ≈95% $p\dot{V}O_{2max}$	4.1			Training was alternate days of running and cycling. Instructed to run as far as possible during runs. Normal running mileage decreased by 60%. Normal training not specified
Laffite et al.[36]	7 M MDR/LDR	25 ± 4	60.6 ± 4.4	8	2 × vΔ50 intervals; 3 × LSD (vΔ50 = 93 ± 1%)	4.0	0.6	5.4*	Normal training not specified
Bickham et al.[37]	7 DR	28 ± 8	58.1 ± 5.3	6	3 × sprint intervals (90–100% perceived maximum effort) + normal training	−2.2			Subjects had no recent history of sprint training. Normal training ≈50 km/wk sub-vLT
Spurrs et al.[38]	8 M DR	25 ± 4	57.6 ± 7.7	6	G1: regular endurance training (controls) G2: as G1 + 2–3 × plyometric training	6.4 3.3		−0.2 5.7*	Normal training = 60–80 km/wk (intensity unspecified). No history of structured plyometric training

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Table I. Contd

| Study	Subjects[a]	Age (y) [mean ± SD][c]	Initial V̇O2max (mL/kg/min) [mean ± SD][c]	Study duration (wk)	Weekly training	Mean % improvement[b]			Comments
						V̇O2max	LT/OBLA	RE	
Billat et al.[39]	9 M LDR	48 ± 3	55.1 ± 4.2	6	2 × vMLSS (30–60 min); 3 × LSD (vMLSS ≈85% V̇O2max)	3.6*	3.3*		Previously only performed LSD training (65 ± 17 min/wk). Two LSD sessions replaced by vMLSS
Franch et al.[40]	36 M DR	30 ± 5	54.8 ± 3.0	6	G1: 3 × intervals at 106% vV̇O2max G2: 3 × intervals at 132% vV̇O2max G3: 3 × continuous at 94% vV̇O2max Subjects also performed 1–3 LSD sessions	6.0* 3.6* 5.9*		3.0* 0.9 3.1*	Training 4wk before study averaged 2.2 h/wk at ≈65% HRmax. The volume of LSD training was adjusted so that runners maintained the same volume as before the study
Hoffman[13]	8 M/F DR	25 ± 7	52.0 ± 7.1	6	G1: 1 × vVT (20 min) + normal training G1: 3 × vVT (20 min) + normal training	8.1* 14.1*			Normal training averaged ≈42 km/wk (intensity not specified). vVT training replaced the same volume of normal training so that subjects maintained same training volume as before the study
Yoshida et al.[15]	6 F MDR/LDR	19 ± 1	51.8 ± 3.2	8	6 × vOBLA (20 min) + normal training (vOBLA ≈91% vV̇O2max)	2.5	10.3*	2.8	Normal training ≈120 min at vLT
Johnston et al.[41]	6 F DR	30 ± 2	50.5 ± 5.4	10	G1: 4–5 × steady-state running (controls) G2: as G1 + 3 × strength training	-1.9 -2.1		0.0 4.0*	G1 and G2 groups both did 32–48 km/wk for 12wk prior to and during the study. Subjects instructed to maintain same endurance training as before the study period. The strength training was additional in G1. Subjects had not weight trained for ≥3mo
Turner et al.[42]	10 M/F DR	31 ± 9	50.4 ± 8.0	6	G1: normal endurance training (controls) G2: as G1 + 3 × plyometric training	0.4 0.0		0.0 2.3*	Subjects instructed to continue normal training (not specified)

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Table I. Contd

Study	Subjects[a]	Age (y) [mean ± SD][c]	Initial $\dot{V}O_{2max}$ (mL/kg/min) [mean ± SD][c]	Study duration (wk)	Weekly training	Mean % improvement[b]			Comments
						$\dot{V}O_{2max}$	LT/OBLA	RE	
Olsen et al.[43]	12 M DR	25 ± 4	45.9 ± 3.6	8	G1: 2 × intervals at 92% $\dot{V}O_{2max}$ + 3.2 km 'all-out' run G2: 2 × intervals at 100% $\dot{V}O_{2max}$ + 3.2 km 'all-out' run	6.2	5.4*		Recreational runners. Normal training = 24–40 km/wk (intensity not specified)

a   Excluding any control subjects.

b   Where percentage improvement was not reported, it was calculated using the following equation: change score/initial score * 100.

c   The range is included where either the mean or SD was not reported.

d   3km race pace ≈100% $\dot{V}O_{2max}$, 10km race pace ≈89% $\dot{V}O_{2max}$.[26]

e   True treatment effect likely, based on 90% confidence limits.

f   Two experimental groups were used but only a small difference in the interval length separated the two groups.

bpm = beats per minute; **CCR** = cross-country runners; **DR** = distance runners; **F** = female; **G** = group; **HR$_{max}$** = maximal heart rate; **LDR** = long-distance runners; **LSD** = long slow distance; **LT** = lactate threshold; **M** = male; **MDR** = middle-distance runners; **p$\dot{V}O_{2max}$** = power output at $\dot{V}O_{2max}$; **RE** = running economy; **Tr** = triathletes; **vΔ50** = velocity midway between vLT and v$\dot{V}O_{2max}$; **vLT** = velocity at the lactate threshold; **vMLSS** = velocity at the maximal lactate steady-state; **vOBLA** = velocity at the onset of blood lactate accumulation; **v$\dot{V}O_{2max}$** = velocity at $\dot{V}O_{2max}$; **vVT** = velocity at the ventilatory threshold; * indicates statistically significant.

an incremental exercise test can be used to approximate the range of heart rates and running velocities in the light (or moderate), heavy and severe exercise intensity 'domains' associated with continuous exercise.[62,63] These domains are characterised by markedly different blood acid-base and pulmonary gas exchange responses.[62-64] This approach towards training prescription has also been used with exercise intensity domains demarcated by ventilatory measures such as the ventilatory threshold and respiratory compensation point.[54,65,66] Coaches and governing bodies have used additional zones, but the physiological rationale underlying this approach has been criticised, since the zones are not based on definable physiological events.[49,54] Particular chronic physiological adaptations may be expected to be largely exercise domain specific, and future research should explore this possibility.

The distribution of training loads over the weeks, months and years of an athlete's training plan is an important but complex aspect of prescribing long-term training known as periodisation.[67] Periodisation allows runners to tolerate periods of relatively high training loads necessary to optimise performance, by following a systematic progression in training load and physiological strain, interspersed with periods of low training loads that are necessary for regeneration to avoid overtraining.[68] Training periodisation originated in the former Soviet Union in the 1950s,[69] and some degree of periodisation appears to have been adopted by modern distance runners and coaches across the world.[20,65,70] However, this approach to training has been criticised,[71] and there is currently little scientific evidence to support the effectiveness of periodised training. It is particularly noteworthy that world-class African runners typically have not followed periodised training plans.[71] Finally, it is important to highlight that the total training load that can be tolerated and that results in peak performance is specific to each individual.[72]
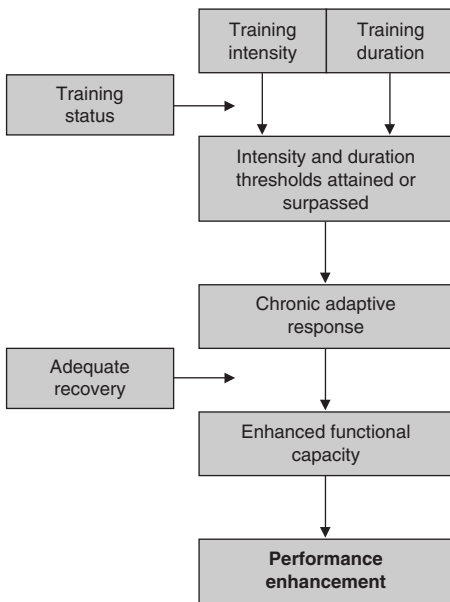
Fig. 2. Model of the threshold concept relating to training-induced stimuli (physiological stress) leading to physiological adaptation and enhanced running performance. Note that the adaptation threshold is specific to an individual runner and the runner's training status at any given time.

3. Enhancing the Maximal Oxygen Uptake

Distance runners and their coaches have typically favoured long slow distance (LSD) training and comparatively little time has been allocated to high-intensity training.[70,73-75] LSD training involves relatively high mileage of moderately paced running.[22] Increasing the volume of LSD training has been suggested to be ineffective for enhancing the $\dot{V}O_{2max}$ of already well trained athletes,[76] and may explain the observation that the enhancement of the $\dot{V}O_{2max}$ of some well trained distance runners has seemingly reached a plateau.[77-81] However, Tanaka et al.[29] reported that increasing the training volume of well trained distance runners from 90 to 120 km/week resulted in a statistically significant 4.8% increase in $\dot{V}O_{2max}$. This study also incorporated 2 days per week of training slightly above the lactate threshold velocity (vLT), and since the runners' normal training was not specified, it is not known whether this represented a training intensity that was

higher than that previously performed. The $\dot{V}O_{2max}$ enhancement may therefore have been related to an increased training intensity rather than an increase in training volume. Well trained runners in another study demonstrated no increase in $\dot{V}O_{2max}$ in response to a >2-fold increase in training volume, even though training intensity was increased simultaneously.[30] However, the percentage increase in training volume was in relation to a 2-month pre-experimental period during which subjects were restricted to 20–30 km/week of easy running. The findings are therefore difficult to interpret. There is little experimental evidence that increasing the volume of LSD training is either effective or ineffective for enhancing the $\dot{V}O_{2max}$ of well trained distance runners, and more research is required. There is also an interest in identifying whether there is an LSD training volume threshold, beyond which no $\dot{V}O_{2max}$ enhancement will occur.[82]

Training intensity has been regarded as the most important variable that can be manipulated for $\dot{V}O_{2max}$ enhancement,[83-85] and may explain the increase in $\dot{V}O_{2max}$ of distance runners during the transition between the off-season and pre-competitive period, during which training intensity is typically increased.[86-89] Accordingly, some authors have suggested that to enhance $\dot{V}O_{2max}$, runners should train at 90–100% $\dot{V}O_{2max}$,[85] or that training at or very close to $\dot{V}O_{2max}$ is most effective for enhancing $\dot{V}O_{2max}$.[90-95] Billat et al.[45] reported that the $\dot{V}O_{2max}$ of well trained distance runners increased by 5.4% (p < 0.05) in response to the inclusion of training between 90–100% $\dot{V}O_{2max}$, despite a 10% decrease in training volume. Several other studies that incorporated training intensities of 90–100% $\dot{V}O_{2max}$ reported similar but statistically insignificant increases in $\dot{V}O_{2max}$.[32,33,35,36] The statistically insignificant results may have been type II errors due to the small sample sizes and associated low statistical power, making interpretation of the findings difficult. Acevedo and Goldfarb[28] reported that the inclusion of training close to $\dot{V}O_{2max}$ did not enhance the $\dot{V}O_{2max}$ of well trained distance runners. The contrasting findings of Billat et al.[45] and Acevedo and Goldfarb[28] cannot be explained at

present and do not appear to be related to the initial $\dot{V}O_{2max}$ of the runners or the volume of high-intensity training performed during the study period. Two studies have reported statistically significant increases in the $\dot{V}O_{2max}$ of runners in response to the addition of training at approximately 70% and 85% $\dot{V}O_{2max}$.[29,39] These studies highlight that the optimal training intensity for enhancing the $\dot{V}O_{2max}$ of trained distance runners may not lie between 90% and 100% $\dot{V}O_{2max}$. Since no studies involving trained runners have compared training at 90–100% $\dot{V}O_{2max}$ with training intensities below this range, the relative efficacy of the two approaches is presently unknown.

Midgley et al.[16] suggested that training at or near $\dot{V}O_{2max}$ should place maximal stress on the physiological processes and structures that limit $\dot{V}O_{2max}$, providing the optimal stimulus for adaptation. In trained individuals, myocardial pressure and volume overload reach their maximal values at the exercise intensity associated with the attainment of $\dot{V}O_{2max}$.[96,97] This mechanical overload is the main stimulus for myocardial adaptation associated with the enhancement of the maximal stroke volume.[98,99] Since the maximal stroke volume is thought to predominantly limit $\dot{V}O_{2max}$ in relatively well trained individuals,[100,101] training at $\dot{V}O_{2max}$ would appear to be the optimal stimulus for $\dot{V}O_{2max}$ enhancement. However, this physiological rationale was based on studies that investigated the physiological responses to incremental exercise. Since training protocols used by distance runners that include velocities that elicit $\dot{V}O_{2max}$ are typically intermittent,[102] the validity of applying this physiological rationale to intermittent training protocols is questionable. Furthermore, intermittent training protocols that elicit $\dot{V}O_{2max}$ can vary considerably.[103] Åstrand et al.[104] reported that subjects elicited $\dot{V}O_{2max}$ during intermittent running with 10-second intervals. MacDougall and Sale[105] suggested that the oxidative stress during such short intervals would be less than for 2- to 3-minute intervals, since much of the energy requirement of the work interval is derived from hydrolysis of phosphocreatine and oxygen bound to myoglobin. Even if eliciting

95–100% $\dot{V}O_{2max}$ has been found most effective in enhancing the $\dot{V}O_{2max}$ of distance runners, extrapolating those findings to all protocols that allow similar amounts of time at 95–100% $\dot{V}O_{2max}$ would be invalid.

Moffatt et al.[106] speculated that as $\dot{V}O_{2max}$ is approached, the differentiation between stimuli decreases. Olsen et al.[43] reported no difference in the increase in $\dot{V}O_{2max}$ of runners who trained at 92% or 100% of the velocity associated with $\dot{V}O_{2max}$ (v$\dot{V}O_{2max}$), therefore supporting this view. However, training at 92% v$\dot{V}O_{2max}$ has been shown to elicit $\dot{V}O_{2max}$.[107] The study by Olsen et al.[43] therefore does not oppose the view held by some[76,91] that training at $\dot{V}O_{2max}$ is optimal, or even obligatory, for enhancing the $\dot{V}O_{2max}$ of well trained runners.

Two studies that investigated intermittent training at 100% $\dot{V}O_{2max}$ reported that the mean time ran at v$\dot{V}O_{2max}$ was 9[108] and 9.5 minutes.[109] Distance runners with large differences between their vLT and work interval velocity have been reported to have particularly low times to exhaustion during intermittent training protocols designed to elicit $\dot{V}O_{2max}$.[110] If training at $\dot{V}O_{2max}$ is found to be the optimal stimulus for $\dot{V}O_{2max}$ enhancement, it would therefore be questionable whether the relatively low volume of high-intensity training is sufficient to provide the optimal training effect. If the duration threshold could not be attained during training at $\dot{V}O_{2max}$, no $\dot{V}O_{2max}$ enhancement would occur. Conversely, only short periods may be needed at $\dot{V}O_{2max}$ in order to enhance $\dot{V}O_{2max}$, since high-intensity training may be particularly potent in eliciting physiological adaptation.[111] Further training studies and research investigating acute and chronic adaptive responses to constant and intermittent running protocols ≤100% $\dot{V}O_{2max}$ are required.

If high-intensity training is a potent signal in eliciting chronic adaptive responses,[111] supra-v$\dot{V}O_{2max}$ training velocities may be considered effective for enhancing $\dot{V}O_{2max}$. There is no theoretical basis to support such a premise, since there would be no additional stress on the oxygen transport system compared with maximal exercise,[16] and the greater proportional contribution of anaer-

obic metabolism would considerably reduce time to exhaustion.[103] Bickham et al.[37] found that $\dot{V}O_{2max}$ did not change when 5- to 15-second sprint intervals (work : relief ratios between 1 : 5 and 1 : 3) were added to the training programmes of distance runners who previously performed only LSD training. Similar results have been reported for well trained cyclists, triathletes and duathletes.[92] However, Franch et al.[40] reported a significant increase in the $\dot{V}O_{2max}$ of runners with a similar initial $\dot{V}O_{2max}$ and training history to those used in the study by Bickham et al.,[37] in response to the inclusion of intermittent running incorporating 15-second work intervals at 132% $v\dot{V}O_{2max}$ (work : relief interval ratio 1 : 1). Very high-intensity training may therefore enhance the $\dot{V}O_{2max}$ of distance runners only if relief intervals between work bouts are short. Franch et al.[40] reported that interval training at 106% $\dot{V}O_{2max}$ resulted in significantly greater increases in $\dot{V}O_{2max}$ than interval training at 136% $\dot{V}O_{2max}$. Although this suggests that very high-intensity training is not optimal for enhancing $\dot{V}O_{2max}$, this type of training may have other important benefits, such as eliciting improvement in the so-called muscle power factors.[112]

There is no physiological rationale to suggest that resistance training would be effective for enhancing the $\dot{V}O_{2max}$ of distance runners. Burleson Jr et al.[113] reported that the average exercise intensity during a traditional strength training session was only 44% $\dot{V}O_{2max}$. Accordingly, studies have consistently demonstrated that adding traditional strength training or plyometric training to the training programmes of distance runners does not enhance $\dot{V}O_{2max}$.[31,38,41,42] The increased muscle mass associated with traditional strength training[114] could potentially reduce the relative $\dot{V}O_{2max}$ of distance runners. Body mass did not significantly change in any training studies involving the addition of resistance training to runners' normal training programmes.[27,31,38,41,42] However, these studies were only between 6 and 10 weeks in duration. Since neural adaptation and not skeletal muscle hypertrophy is the predominant source of the increased strength in the early stages of a resistance training

programme,[115] the long-term effects of resistance training on the body mass and $\dot{V}O_{2max}$ of distance runners requires investigation. Furthermore, the female distance runners in the study by Johnston et al.[41] demonstrated a mean 1.3kg increase in body mass. Although this was statistically insignificant, the experimental group included only six subjects and therefore may have been a type II error.

In summary, some authors have suggested that training at or near $\dot{V}O_{2max}$ is optimal for enhancing the $\dot{V}O_{2max}$ of athletes,[90-95] and that increasing the volume of submaximal training is ineffective.[76] However, submaximal,[29,39] maximal[45] and supramaximal[40] training intensities have all been shown to enhance the $\dot{V}O_{2max}$ of distance runners. Since only two studies[40,43] have compared different training intensities, the relative efficacy of a particular training intensity is unknown. Different forms of resistance training have consistently been reported to be ineffective in enhancing the $\dot{V}O_{2max}$ of distance runners.

4. Enhancing the Lactate Threshold

Although the role of lactate as a cause of fatigue during physical activity is still a controversial issue,[116-118] scientists widely agree that an increase in the lactate threshold typically results in improved endurance performance.[21,64,119-121] Anecdotal evidence supports the view traditionally held by distance runners and running coaches that prolonged, moderate-intensity running is optimal for enhancing the lactate threshold.[105,122] However, a 103% increase in the volume of sub-lactate threshold training over 4 weeks did not enhance the lactate threshold of well trained distance runners.[25] The short study duration may explain the absence of any detectable change in the lactate threshold. The observation that long-distance runners typically possess higher lactate thresholds than middle-distance runners has been suggested to be indirect evidence that high volumes of sub-lactate threshold training are effective in enhancing the lactate threshold.[120] The basis for this premise was that long-distance runners predominantly perform LSD training, whereas middle-distance runners tend to rely more on interval

training at or close to $\dot{V}O_{2max}$.[120] Natural selection may be an alternative explanation for differences in the lactate thresholds of middle- and long-distance runners and challenges the hypothesis of MacDougall.[120] A runner with a genetically determined high preponderance of slow-twitch skeletal muscle fibres would probably possess a relatively high lactate threshold,[123] would more likely perform better in long-distance running events,[3-5] and would therefore more likely compete in these events. We could find no experimental evidence to support the premise that prolonged moderate-intensity exercise is effective for enhancing the lactate threshold of distance runners.

Several authors have suggested that training can be effectively prescribed based on the blood lactate response to an incremental exercise test.[124-126] Many training intervention studies have used this approach to prescribe training loads (table I). However, Hawley[11] stated that there was no scientific evidence to support the use of blood lactate responses to exercise tests for training prescription. Untrained women demonstrated a significant increase in the lactate threshold during 4 months of training at lactate threshold intensity; however, there was no further enhancement during the following 8 months of training.[126] This study suggests that training at the vLT would probably be ineffective for enhancing the lactate threshold of distance runners. The basis for this statement is that distance runners typically have a history of relatively high volumes of this type of training[15] and so the rate of adaptive responses has probably already reached a plateau.[76] However, the addition of very long runs at or below the vLT (over-distance training) to the training programmes of distance runners, who habitually undertake high training mileages, may enhance the lactate threshold; future research could investigate this possibility.

Wells and Pate[22] suggested that training slightly above the vLT is effective for enhancing the lactate threshold of endurance athletes. However, this assertion appears to be based on the training responses of untrained individuals. Henritze et al.[125] reported that training slightly above vLT ($\approx$59% $\dot{V}O_{2max}$)

increased the oxygen uptake ($\dot{V}O_2$) at the lactate threshold by 48% in untrained college women, compared with an insignificant 18% increase in the group that trained at the vLT ($\approx$44% $\dot{V}O_{2max}$). In a study by Weltman et al.,[126] subjects training at the lactate threshold intensity demonstrated a plateau in lactate threshold enhancement after 4 months, whereas subjects training at supra-lactate threshold intensities demonstrated continued improvement over the next 8 months of the study period. Another study reported that supra-lactate threshold training was no more effective for enhancing the lactate threshold than sub-lactate threshold training.[127] To our knowledge, only one study that involved trained runners has reported the enhancement of the lactate threshold in response to the inclusion of training at or slightly above vLT.[29] Although this study highlights that training at or slightly above the lactate threshold may enhance the lactate threshold of well trained distance runners, only one experimental group was used. The relative efficacy of training at or slightly above the lactate threshold, for enhancing the lactate threshold of trained distance runners, is therefore unknown.

Several studies involving runners incorporated higher training velocities, with contrasting results.[15,34,36,39] Two studies that incorporated intermittent running at vΔ50 (the velocity midway between vLT and v$\dot{V}O_{2max}$) reported no significant increase in the lactate threshold.[34,36] Two other studies incorporating intermittent runs at the velocities at the maximal lactate steady-state (vMLSS) and onset of blood lactate accumulation (vOBLA) reported significant increases in lactate threshold.[15,39] Since the duration of the studies and the relative training intensity expressed as a percentage of $\dot{V}O_{2max}$ were similar between studies, these factors did not appear to explain the contrasting findings. The study that reported the largest mean increase in the lactate threshold of 10.6% involved 20 minutes of running at vOBLA for 6 days per week, in addition to the runners' normal training.[15] This volume of additional high-intensity training ($\approx$91% $\dot{V}O_{2max}$) may explain the large increase in the lactate threshold in these runners. The subjects in the second

study that reported a significant increase in the lactate threshold had a previous history of performing only LSD training.[39] Runners who favour high volumes of moderate-intensity training and do not engage in relatively high-intensity training may demonstrate a plateau in lactate threshold enhancement.[76] Since the LSD training was performed at an average of 12.4 km/h, the addition of 2 days per week of training at the vMLSS (average 13.8 km/h) may have provided an unaccustomed training stimulus resulting in lactate threshold enhancement.

Londeree[17] suggested that the lactate threshold and fixed blood lactate concentrations responded similarly to training and should not confound the interpretation of training studies. This is highlighted in figure 3 by a rightward shift of the whole blood lactate curve, and is characteristic of the training response of trained distance runners.[15,28,64] By inference from reported changes in OBLA, two further studies involving well trained distance runners demonstrated statistically significant increases in the lactate threshold,[24,28] and one study reported a statistically insignificant change.[23] Since these studies all involved similarly intense training and similarly well trained runners, the reason for the contrasting findings cannot be ascertained.

A meta-analysis by Londeree[17] concluded that highly trained individuals may need to train at much higher than lactate threshold intensity to enhance the
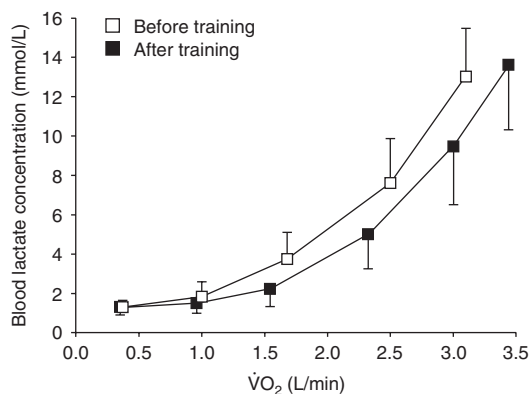
Fig. 3. A downward and rightward shift of the mean blood lactate curve of ten male students in response to 16 weeks of physical training. The error bars represent the standard deviation of the mean.[128] $\dot{V}O_2$ = oxygen uptake.

lactate threshold. However, only four of the studies reviewed involved well trained individuals, and only one of these studies involved training above OBLA ($\approx 85\%$ $\dot{V}O_{2max}$),[24,39] making this supposition questionable. If well trained runners do require high relative training intensities to enhance the lactate threshold, the physiological basis for this is unclear. One explanation is that distance runners typically perform high weekly training loads,[1,45,129] with little time allocated to high-intensity training.[73,75,130] Since type II skeletal muscle fibres are not recruited to any great extent until around 90% $\dot{V}O_{2max}$,[131] these fibres may be relatively untrained compared with the frequently recruited type I fibres. Training responses associated with lactate threshold enhancement are thought to be related to skeletal muscle adaptations (table II) that reduce lactate production and increase its disposal at higher running velocities.[132] The high-intensity training required for lactate threshold enhancement suggested by Londeree[17] may therefore be related to these training adaptations in type II skeletal muscle fibres. Mac-Dougall[120] presented this hypothesis almost 30 years ago and recommended that work intervals of 2–3 minutes duration at 90–100% $\dot{V}O_{2max}$ would be most effective for lactate threshold enhancement. Since some well trained runners possess lactate thresholds at or close to 90% $\dot{V}O_{2max}$,[75,82] training slightly above the lactate threshold would represent high-intensity training for these runners. An alternative explanation is that these physiological adaptations are elicited by an elevated lactate concentration associated with high-intensity exercise. Mader[133] stated that lactate does not elicit adaptive responses that enhance endurance performance, but did not provide empirical or theoretical support for this view. Intuitively, lactate would be a prime candidate molecule for gene induction that resulted in adaptations associated with lactate threshold enhancement. However, if elevated blood lactate concentrations are necessary to enhance the lactate threshold, some distance runners may need to train at high percentages of $\dot{V}O_{2max}$. Weltman et al.[58] reported that 20 of 31 runners had blood lactate concentrations <2.5 mmol/L at a running velocity associated with 90% $\dot{V}O_{2max}$, and 10 of the 31

Table II. Training-induced adaptations that have been associated with the enhancement of the maximal oxygen uptake ($\dot{V}O_{2max}$), lactate threshold and running economy

Training adaptation	Physiological significance
$\dot{V}O_{2max}$	
Increased left ventricular chamber size and wall thickness[134]	Increased maximal stroke volume[135]
Increased erythrocyte mass[136]	Increased blood volume, maximal stroke volume and arterial oxygen content[137]
Increased plasma volume[138]	Increased blood volume and maximal stroke volume[137]
Increased skeletal muscle capillarity[139]	Increased oxygen diffusion and uptake for any given arterial pO_2 and blood flow[140]
Increased skeletal muscle mitochondrial density and oxidative enzyme concentration[24,141]	Increased $\dot{V}O_2$ and widening of the maximal arterial-mixed venous oxygen difference[142]
Increased myoglobin concentration[143]	Facilitation of oxygen diffusion from the sarcolemma to the mitochondria. Increased $\dot{V}O_2$ for any given pO_2 and blood flow. Increased maximal arterial-mixed venous oxygen difference[144]
Lactate threshold	
Decreased PFK-1 concentration and PFK-1 : CS ratio[24]	Decreased lactate production[24]
Increased skeletal muscle mitochondrial density and oxidative enzyme concentration[24,141]	Increased percentage of pyruvate that enters the Krebs cycle, as opposed to lactate formation through the LDH reaction[24]
Increased β-oxidation enzymes[24]	Increased lipid oxidation, decreased demand for carbohydrate metabolism and decreased lactate production[145]
Change in LDH expression that favours the heart isoform[24]	Decreased pyruvate-to-lactate conversion rate[24]
Increased MCT expression[146]	Increased lactate disposal[146]
Increased muscle strength[147]	Reduced recruitment of type II skeletal muscle fibres and reduced blood flow occlusion[147]
Running economy	
Change in the expression of fast-twitch skeletal muscle fibres towards a more slow-twitch phenotype[146]	Reduced energy cost for developing a particular level of force[148]a
Decreased minute ventilation for a particular running velocity[40]	Reduced respiratory energy demand[40]
Improved mechanical efficiency[149]	Reduced whole body energy demand[150]
Increased musculotendonous stiffness[38]	Increased storage and return of elastic energy and muscle stabilising activity[151]

a   Based on research involving mouse skeletal muscle.

CS = citrate synthase; **LDH** = lactate dehydrogenase; **MCT** = monocarboxylate transporter; **PFK-1** = phosphofructokinase-1; **pO_2** = partial pressure of oxygen; **$\dot{V}O_2$** = oxygen uptake.

runners at 95% $\dot{V}O_{2max}$. Consequently, the same training intensity (i.e. 90–100% $\dot{V}O_{2max}$) could be optimal for the enhancement of the lactate threshold and the $\dot{V}O_{2max}$ of well trained runners. Empirical research is required to investigate this hypothesis.

Marcinik et al.[147] reported a significant 12% increase in the lactate threshold of sedentary males in response to a 12-week traditional resistance training programme. The authors suggested that the improvement in lactate threshold may have been related to increased muscle strength, an associated attenuation of blood flow occlusion, and a decrease in type II skeletal muscle fibre recruitment. Hamilton et al.[27] reported a 4% increase in the vOBLA of

distance runners in response to replacing part of their normal training with resistance training, consisting of single-leg jumps and maximal effort tethered treadmill running. The control group, who performed only their normal training, demonstrated a 0.5% increase in vOBLA. The 90% confidence limits highlighted that the difference between groups was likely to be a true treatment effect. Conversely, Paavolainen et al.[31] reported that the lactate threshold did not change when 32% of the runners' normal run training was replaced with sport-specific explosive resistance training. The improvement in the study by Hamilton et al.[27] may have been due to the combination of high resistance

and movement specificity afforded by the tethered running, compared with the plyometric training and sprints performed in the study by Paavolainen et al.[31] Alternatively, the apparent lack of improvement in the $\dot{V}O_2$ at the lactate threshold in the study by Paavolainen et al.[31] may have been an artefact of the 7.7% improvement in running economy. The lactate threshold, expressed as $\dot{V}O_2$ or running velocity, is dependent on running economy, with improved running economy tending to increase the velocity at lactate threshold[152] and decrease the $\dot{V}O_2$ at the lactate threshold.[153] Further research is clearly required to investigate the efficacy of different types and volumes of resistance training for enhancing the lactate threshold of distance runners.

In summary, we found only one study that investigated the effects of an increase in the volume of sub-vLT or vLT training on the lactate threshold of distance runners.[25] This study reported no significant increase in the lactate threshold. Several training studies have reported a significant increase in the lactate threshold of distance runners in response to the inclusion of supra-vLT training velocities,[15,29,39] although these findings have not been consistent.[34,36] Similar contrasting findings have also been reported with studies that added resistance training to the training programmes of distance runners.[27,31] This limited and contrasting research highlights the need for further studies.

5. Enhancing Running Economy

There is a belief that, over time, runners adopt their most economical running style.[149] Accordingly, high training volumes and the number of years of running experience have been suggested to be important for improved running economy.[154] Pate et al.[155] reported that training volume was not associated with better running economy, even after controlling for potential confounding variables. However, no training intervention studies that measured running economy have increased the training volume of runners while maintaining the same mean training intensity, so the effect of increasing training volume on running economy is not known. Mayhew[156] found that the years of training was signifi-

cantly correlated (r = 0.62) with running economy in relatively well trained distance runners. The critically important factor in the enhancement of running economy may therefore be the cumulative distance the runner has covered over the years of training and not training volume *per se*. This may be due to continued long-term adaptations in skeletal muscle (table II), or a slow but progressive long-term improvement in mechanical efficiency.[149] If the cumulative distance the runner has covered over the years of training is a determinant of running economy, a tendency for older runners to have better running economy than younger runners may be expected. However, running economy has been found to be negatively associated with age in runners aged 20–60 years.[155] This has been suggested to be due to a reduced ability to store and use elastic energy.[155] Further research is required to investigate the relationship between running economy and cumulative training distance.

Studies that have incorporated relatively high-intensity training into the training programmes of distance runners have reported equivocal results in relation to improving running economy. Interval training at 93–106% $\dot{V}O_{2max}$[23,34,36,40] and continuous running at vOBLA[24] have been shown to improve running economy significantly. Other studies using similar training intensities have reported no significant improvement.[15,33] However, some of the studies that found contrasting findings actually reported similar improvements in running economy,[15,24,33,40] and the contrasting findings were actually an artefact of statistical power. These studies therefore indicate that high-intensity training improves running economy, but they do not allow evaluation of its relative efficacy. Additionally, whereas $\dot{V}O_{2max}$ has been shown to increase significantly during the transition between the off-season and pre-competitive period, during which training intensity is increased,[86-89] the same studies reported either a significant improvement[87,88] or no change[86,89] in running economy. Further research is therefore required to establish the relative efficacy of high-intensity training for improving the running economy of long-distance runners. Franch et al.[40]

compared interval training at 94%, 106% and 132% $\dot{V}O_{2max}$ and found that running economy significantly improved in the 94% and 106% groups, but not in the group that trained at 132% $\dot{V}O_{2max}$. This suggests that very high-intensity running is not effective in improving running economy, possibly due to a loss of running form at very high running velocities, or an inability to complete a sufficient training volume to elicit a training effect.

Jones and Carter[157] suggested that runners are typically most economical at the running velocities at which they habitually train. Since running economy is only of practical importance at intended race pace, this would suggest that training at intended race pace is optimal for enhancing running economy. Although intuitively this would seem correct, to our knowledge no studies have investigated the specificity of training velocity on running economy. In fact, Morgan et al.[158] suggested that the type of run training exerts a negligible effect on improving running economy, based on the observation that several studies reported no differences in the running economy of distance runners despite the runners engaging in different training.

Although the addition of traditional strength training and plyometric training to the training programmes of distance runners has been shown not to enhance $\dot{V}O_{2max}$,[31,38,41,42] a significant improvement in running economy has consistently been reported.[31,38,41,42] The authors suggested that greater mechanical efficiency resulting from increased muscle strength, improved motor unit recruitment patterns and increased tendon stiffness were possible explanations for the improved running economy.[31,38,41] Although resistance training may appear to be an effective training method to enhance the performance of distance runners, the long-term effects of different forms of resistance training have not been investigated. Traditional strength training may eventually lead to an increased body mass,[159] which may impair performance.[160] Plyometric training may avoid this potential problem and has the benefit of being able to be incorporated into the warm-up or cool-down of a running workout.[42] This latter point may be of particular importance to runners who are reluctant to engage in traditional resistance training. Increased resistance to movement may also be attained by hill training, bungee running and running in sand. Uphill running and bounding have been used to enhance the strength of distance runners,[161] whereas Billat[162] suggested that high-velocity running exerts similar training effects as resistance training in distance runners. These training methods may prove to be more effective alternative forms of resistance training due to higher levels of movement specificity. However, no training studies have investigated these alternative forms of training for improving the running economy of runners. Further research is clearly required to investigate the relative efficacy of different forms of resistance training for improving running economy.

Stretching is recommended to runners to prevent injury and improve performance.[163] Two studies have reported that flexibility was inversely related to running economy in trained distance runners,[151,164] indicating that runners should perform little or no stretching, to prevent decrements in running performance due to decreased running economy. Furthermore, there is currently no scientific evidence to support the premise that stretching reduces injury risk in runners.[165] Craib et al.[151] hypothesised that less flexibility minimised muscle stabilising activity and increased the storage and return of elastic energy. However, two studies found no relationship between flexibility and running economy in runners.[155,166] A 10-week chronic stretching programme significantly increased flexibility, but running economy remained unchanged.[167] The relationship between flexibility and running economy is therefore currently unclear and further studies are required.

In summary, high training volumes have been suggested to be effective for improving running economy,[154] although no studies that increased training volume while maintaining a constant training intensity have been conducted to support this view. The inclusion of high-intensity interval training,[23,34,36,40] continuous running at vOBLA,[24] and resistance training[31,38,41,42] in the training program-

mes of distance runners have been shown to improve running economy. However, no studies have compared the relative efficacy of the different forms of resistance training, and there has been limited research[40] investigating the relative efficacy of different training intensities on the running economy of distance runners.

6. Can Valid Training Recommendations be Given to Runners and Coaches Based on Current Scientific Knowledge?

There have been relatively few long-term training studies involving trained distance runners.[78-81] This lack of long-term training studies has been perceived as a major limitation in characterising effective training methods for enhancing the physiological determinants of long-distance running performance.[19] Although we agree with this view, training studies of 4–8 weeks duration, which account for 19 of the 23 studies in table I, may still be insightful. Runners often target specific physiological performance determinants during training cycles of similar duration.[20,65,70] However, when considering both short- and long-term training studies involving runners, there have still been relatively few. This may be due to the reluctance of runners to have scientists dictate their training schedules over the study period.[21] Consequently, it would be difficult to argue against the view that there is insufficient direct scientific evidence to formulate training recommendations based on this limited research. This is particularly evident when considering the many methodological factors associated with these training studies that make interpretation of the findings difficult (summarised in table III). Previous studies, for example, mostly did not control the runners' training load, and sometimes the basis of the training study was to include one or more specific bouts of training in addition to the runner's 'normal training',[13,15,24,29,37] which was typically not described or only briefly described (table IV). The training status of the runners (e.g. off-season, pre-competitive training phase) during the study period was seldom reported (table IV). The nature of the training in the weeks, months and years before the study

period could profoundly affect training responses during the study period. For example, if high-intensity training bouts were added to runners' 'normal training', runners who have previously performed only LSD training[74,75] would probably demonstrate greater training responses than those who have already habitually performed high-intensity training. The same would probably be true for runners studied in the off-season compared with the pre-competitive phase of training. There is also the possibility that the training intervention used in some of the studies in table I did not involve an appreciable change in training intensity or volume for some of the runners. This inability to compare the runners' training before and during the training intervention period is probably the main factor that hinders the interpretation of previous training intervention studies. Researchers are therefore encouraged to report as much detail as practically possible relating to the training history of subjects involved in training studies. A washout period has been used to minimise the influence of the runners' previous training on the responses to a training intervention.[30] This involved 2 months of 20–30km per week of easy running. A limitation to this approach is that most runners will probably be unwilling to undertake a long period of low-intensity, low-volume training. Furthermore, a washout period reduces ecological validity since runners would not normally undertake such low training loads.

Arguably the second greatest limitation in the interpretation of the studies summarised in table I is that only 5 of the 23 studies[13,14,33,40,43] included more than one experimental group. Consequently, there is no comparison to allow the evaluation of the relative efficacy of a particular training intervention. If a training intervention study was conducted in the runners' off-season, for example, any increase in training volume or intensity may enhance the physiological determinants of performance, regardless of the specific characteristics of that intervention. Researchers are therefore encouraged to include at least two experimental groups with clearly defined differences in the training programmes of the groups. Presenting at least one clear hypothesis,

Table III. Potential limitations to some of the training intervention studies included in table I that make interpretation of the findings difficult

Factor	Impact on interpretation of results
Subjects' prior training not described	Does not allow evaluation of how the runners' training programmes have changed due to the training intervention
No control group	Does not allow identification of any increase in a dependent variable[a] not due to the experimental treatment (e.g. learning effect during repeated testing)
Only one experimental group	Does not allow evaluation of the relative efficacy of an experimental treatment in enhancing a particular dependent variable[a]
Small sample size	Low statistical power that increases the type II error rate (i.e. an increased chance that the authors of a training study declare that no change has occurred in the dependent variable,[a] when in reality a change has occurred). This is particularly relevant to studies that have investigated interaction effects (time × group) between control and experimental groups
Degree of compliance with training programme not monitored/reported	Lack of compliance to the training intervention could result in more or less training being completed than expected, or could result in the inclusion of a type or intensity of training that the runner should not have been doing. For example, Smith et al.[33] stated that in their previous training study[32] some runners performed additional training because they thought the prescribed training volume was insufficient
Inappropriate test methodology	Decreases the chance of detecting a change in a dependent variable[a] if it has occurred. For example, using an inclined treadmill test for runners who have trained on relatively level surfaces[24,28,30]
Inappropriate units of measurement	Relative $\dot{V}O_{2max}$ can change due to training-induced changes in body mass and does not reflect a change in cardiorespiratory fitness. Changes in absolute $\dot{V}O_{2max}$ should therefore be reported. A training-induced increase in fat oxidation would tend to increase submaximal $\dot{V}O_2$[168] and would tend to mask any improvement in running economy. Applying a correction factor to the $\dot{V}O_2$ value based on the change in the respiratory exchange ratio would increase the validity of running economy measurements
Interaction between dependent variables[a]	The lactate threshold expressed in relation to $\dot{V}O_2$ or running speed is dependent on running economy.[152] Improved running economy tends to decrease the lactate threshold when expressed as $\dot{V}O_2$, and increase it when expressed as a velocity
Subject pre-test preparation not controlled/reported	Increases the noise in detecting changes in a dependent variable.[a] For example, differences in muscle glycogen concentration from variations in dietary carbohydrate intake may alter the measurement of the lactate threshold pre- and post-experimental treatment[169]
Subject habituation to test procedures and equipment not conducted/reported	A change in a particular dependent variable[a] could be due to a learning effect rather than a true training effect in runners who are not adequately habituated

a Dependent variables refer to $\dot{V}O_{2max}$, lactate threshold and running economy.

$\dot{V}O_2$ = oxygen uptake; $\dot{V}O_{2max}$ = maximal oxygen uptake.

based on previous experimental research or some theoretical physiological rationale, should also aid in the interpretation of the findings.

Several studies incorporated resistance training into the training programmes of runners and included a control group that maintained only their normal run training.[27,31,38,41,42] These studies provide an insight into the effectiveness of resistance training for enhancing the physiological determinants of long-distance running performance. Resistance training has consistently been shown to improve the running economy of trained runners,[31,38,41,42] and this would indicate that scientists should recommend that runners routinely undertake some sort of

resistance training. However, it is not known which type of resistance training is most effective, or how resistance training loads and types should be integrated into the different phases of a training plan.[170] Furthermore, the studies highlighted above were of only 6–10 weeks duration, and any negative long-terms effects are unknown.

Much of the current knowledge relating to chronic adaptive responses to training has been derived from studies using sedentary individuals. For example, the belief that training slightly above the lactate threshold is optimal for enhancing the lactate threshold[22] appears to have been based on studies involving sedentary individuals.[125,126] To our knowledge,

Table IV. Number of training intervention studies (out of 23) involving distance runners that reported particular details of the runners' training prior to participation in the study

Aspect of previous training	No. of studies
Weekly training distance	10
Training intensities	3
Weekly training distance and training intensities	0
Weekly training time	2
Type of training (e.g. interval training, hill work, resistance training)	1
Stage of training (e.g. off-season, pre-competition phase)[a]	5

a   At the time of commencing participation in the study.

no studies involving runners have corroborated these findings. However, although we cannot assume that trained individuals respond in the same way as sedentary individuals, little research has so far been conducted that identifies any differences in chronic adaptive responses to training between trained and sedentary individuals. The rate of progression in physiological adaptation may be the only appreciable difference,[171] particularly after previously sedentary individuals have undertaken several weeks of training. Until appropriate research has been conducted, scientists should be very cautious, when extrapolating findings from training studies that used untrained individuals, in their advice to long-distance runners and their coaches on effective training methods. In this context, it may be possible that training intervention studies using trained animals may have greater validity than those using untrained humans. The validity of making inferences from training studies using different athletes, such as cyclists and swimmers, or distance runners of different ability (e.g. from recreational club runner to elite), is also questionable.

Many training methods have not been investigated in relation to enhancing the physiological determinants of long-distance running performance. For example, the physiological effects of replacing on-land running with deep-water running have been investigated in distance runners,[172,173] but the use of deep-water running as supplemental training to on-land running has not been investigated. Anecdotal evidence suggests that hill running is effective for improving running economy;[174] however, no training studies have investigated this premise. Other training methods that have not been investigated in training studies using runners are over-speed training using declined running and bungee ropes, over-distance training, and running in sand. Valid recommendations cannot be given to runners or coaches regarding the efficacy of some of these 'novel' training methods until appropriate research has been conducted.

Although scientists are unlikely to be able to formulate valid training recommendations based solely on 'direct' scientific evidence (i.e. training studies), other sources of scientific knowledge are available. The physiological structures and processes that limit $\dot{V}O_{2max}$, the lactate threshold and running economy, and the adaptations associated with their enhancement, have been identified (table II). Scientists can use this information, along with knowledge of the acute physiological responses to different exercise intensities, to guide training recommendations. This approach has been used recently by Midgley et al.[16] to formulate recommendations for effective training intensities to enhance the $\dot{V}O_{2max}$ of distance runners. We believe that the demarcation of training zones based on the blood lactate response to incremental exercise, highlighted in figure 4, is also useful for prescribing training for long-distance runners. Some scientists have had vast experience working with runners and running coaches, and this will probably also prove valuable in interpreting the body of scientific literature relating to enhancing the physiological determinants of long-distance running performance.

In the future, molecular biology may make an increasing contribution to identifying optimal training methods. At present, little is known about the biochemical, mechanical and electrochemical signals that modify gene expression resulting in adaptations associated with the enhancement of the physiological determinants of long-distance running performance. Relatively recent advances in genetic technology have allowed the identification of genetic factors that contribute to particular aspects of athletic performance.[176,177] Future research may
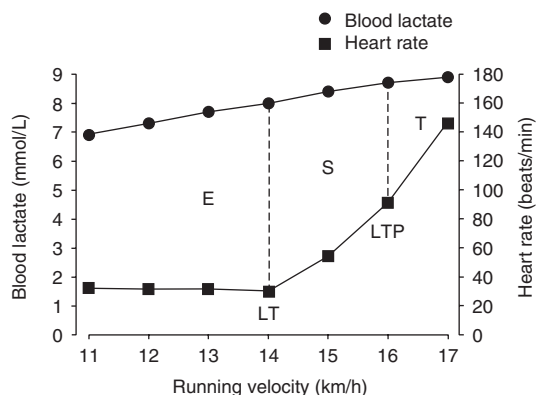
Fig. 4. Blood lactate and heart rate responses of a competitive club runner during an incremental exercise test. Training zones are demarcated by the running velocities and heart rates associated with the lactate threshold (LT) and the lactate turnpoint (LTP). The easy (E), steady (S) and tempo (T) running training zones are associated with the moderate, heavy and severe exercise domains, respectively. This is based on the assumption that the running velocity and heart rate at the LTP, measured during an incremental exercise test, is a reasonable approximation of the maximal lactate steady-state.[175]

identify the genes that contribute to the variation in $\dot{V}O_{2max}$, the lactate threshold and running economy, as well as the biochemical, mechanical and electrochemical signals that induce these genes.[178] If the effects of different exercise intensities and modalities on signal strength can be characterised for each of these genes, this information may prove very effective in identifying optimal training methods for long-distance runners.

7. Conclusions

Current training methods have developed predominantly from the trial-and-error approach of runners and several prominent running coaches, while the contributions made by scientists have been comparatively small. The present article suggests that this can be largely explained by the lack of available scientific knowledge. Upon consideration of the limitations of current research, we believe that there is little direct scientific evidence to allow identification of the most effective training methods to enhance the $\dot{V}O_{2max}$, lactate threshold and running economy of long-distance runners. Previous

research suggests that high-intensity running and resistance training may be effective training methods; however, this research is still too limited to guide the formulation of specific recommendations with a sufficient level of confidence. Although direct scientific evidence is limited, we believe that scientists can still formulate worthwhile training recommendations by integrating the information derived from training intervention studies with other scientific knowledge. This includes the acute physiological responses in the various exercise intensity domains, the structures and processes that limit the physiological determinants of long-distance running performance, and the adaptations associated with their enhancement. The scientists' own experience in providing sports science support and knowledge of appropriate anecdotal evidence should also help formulate worthwhile recommendations that are predominantly based on the available scientific evidence.

Readers should be aware that although this review has focused on identifying optimal training methods for enhancing the three major physiological determinants of long-distance running performance, enhancing competition performance is the ultimate goal. Optimal training methods for enhancing particular determinants of long-distance running performance may not be optimal for enhancing performance, since 'optimal' training methods may impact negatively on other determinants of long-distance running performance. Furthermore, an increased risk of overtraining should be an important consideration.

Scientists should be cautious when giving training recommendations to runners and coaches based on the current limited scientific knowledge. This limited knowledge highlights that characterising the most effective training methods for long-distance runners is still a fruitful area for future research.

Acknowledgements

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