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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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Contents

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.

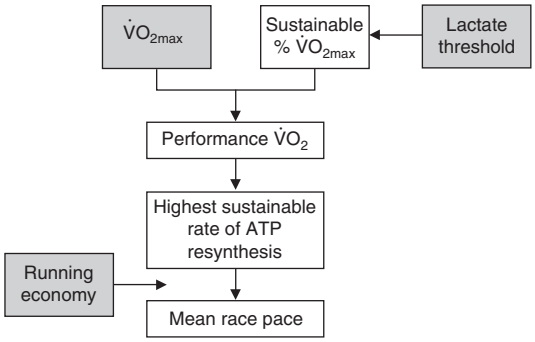

Fig. 1. Flow diagram showing that long-distance running performance is predominantly determined by maximal oxygen uptake ($\dot{V}O_{2max}$), the lactate threshold (determines the fraction of $\dot{V}O_{2max}$ that can be sustained) and running economy. Performance oxygen uptake ($\dot{V}O_2$) represents 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 intensities 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 (18–25[b])	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 4.0[e]		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% HR_{max} 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	19–23[c]	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]			Comments
						$\dot{V}O_{2max}$	LT/OBLA	RE	
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 $\dot{V}O_{2max}$ (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.[39]	9 M LDR	48 ± 3	55.1 ± 4.2	6	2 × vMLSS (30–60 min); 3 × LSD (vMLSS ≈85% $\dot{V}O_{2max}$)	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% v$\dot{V}O_{2max}$ G2: 3 × intervals at 132% v$\dot{V}O_{2max}$ G3: 3 × continuous at 94% v$\dot{V}O_{2max}$ 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% HR_{max}. 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% v$\dot{V}O_{2max}$)	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% $v\dot{V}O_{2max}$ + 3.2 km 'all-out' run G2: 2 × intervals at 100% $v\dot{V}O_{2max}$ + 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 (speciality distance not specified); **F** = female; **G** = group; $\mathbf{HR_{max}}$ = maximal heart rate; **LDR** = long-distance runners; **LSD** = long slow distance; **LT** = lactate threshold; **M** = male; **MDR** = middle-distance runners; $\mathbf{p\dot{V}O_{2max}}$ = 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; $\mathbf{v\dot{V}O_{2max}}$ = 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]

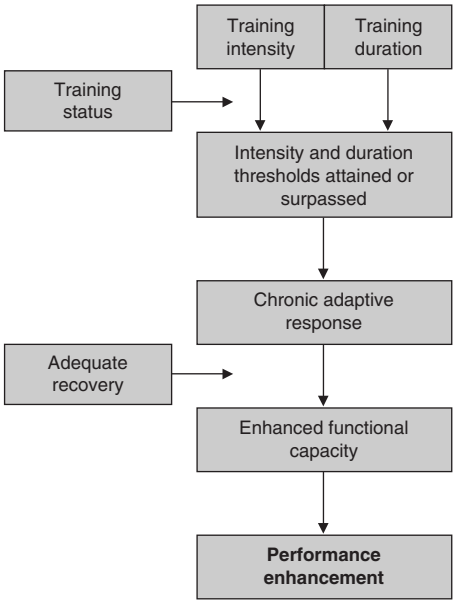

Fig. 2. Model of the threshold concept relating to training-induced stimuli (physiological stress) leading to physiological adaptation and enhanced running performance. Note that the adaptation threshold is specific to an individual runner and the runner's training status at any given time.

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 (≈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 (≈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 (≈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

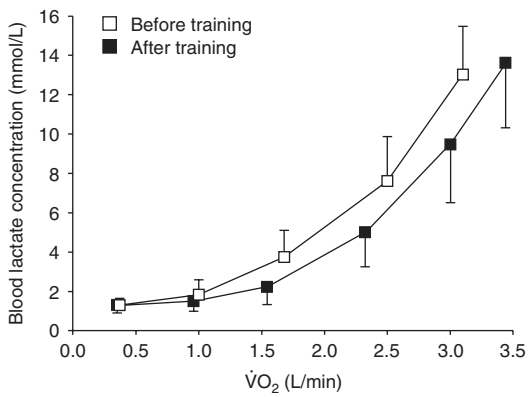

Fig. 3. A downward and rightward shift of the mean blood lactate curve of ten male students in response to 16 weeks of physical training. The error bars represent the standard deviation of the mean.[128] $\dot{V}O_2$ = oxygen uptake.

lactate threshold. However, only four of the studies reviewed involved well trained individuals, and only one of these studies involved training above OBLA (≈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. MacDougall[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 significantly 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

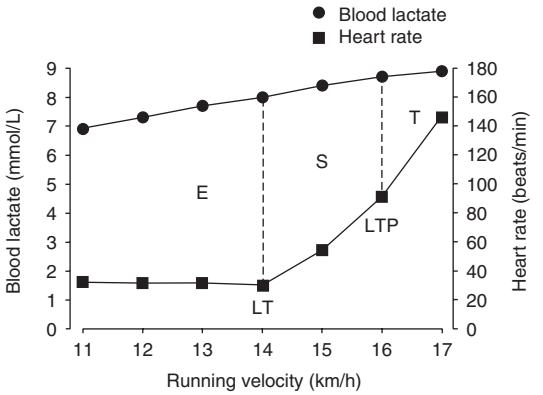

Fig. 4. Blood lactate and heart rate responses of a competitive club runner during an incremental exercise test. Training zones are demarcated by the running velocities and heart rates associated with the lactate threshold (LT) and the lactate turnpoint (LTP). The easy (E), steady (S) and tempo (T) running 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.

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References

1. Billat V, Demarle A, Slawinski J, et al. Physical and training characteristics of top-class marathon runners. Med Sci Sports Exerc 2001; 33: 2089-97
2. Foster C. $\dot{V}O_{2max}$ and training indices as determinants of competitive running performance. J Sports Sci 1983; 1: 13-22
3. Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the $\dot{V}O_{2max}$ test predicts running performance. J Sports Sci 1990; 8: 35-45
4. Farrell PA, Wilmore JH, Coyle EF, et al. Plasma lactate accumulation and distance running performance. Med Sci Sports Exerc 1979; 11: 338-44
5. Tanaka K, Matsuura Y. Marathon performance, anaerobic threshold, and onset of blood lactate accumulation. J Appl Physiol 1984; 57: 640-3
6. Conley DL, Krahenbuhl GS. Running economy and distance running performance of highly trained athletes. Med Sci Sports Exerc 1980; 12: 357-60
7. Morgan DW, Baldini FD, Martin PE, et al. Ten kilometre performance and predicted velocity at $\dot{V}O_{2max}$ among well-trained male runners. Med Sci Sports Exerc 1989; 21: 79-83
8. di Prampero PE, Atchou G, Brückner JC, et al. The energetics of endurance running. Eur J Appl Physiol 1986; 55: 259-66
9. Rupert JL. The search for genotypes that underlie human performance phenotypes. Comp Biochem Physiol A 2003; 136: 191-203
10. Wolfarth B, Bray MS, Hagberg JM, et al. The human gene map for performance and health-related fitness phenotypes: the 2004 update. Med Sci Sports Exerc 2005; 37: 881-903
11. Hawley JA. State of the art training guidelines for endurance performance. S Afr J Sports Med 1995; 2: 7-12
12. Unal M, Unal DO. Gene doping in sports. Sports Med 2004; 34: 357-62
13. Hoffman RL. Effects of training at the ventilatory threshold on the ventilatory threshold and performance in trained distance runners. J Strength Cond Res 1999; 13: 118-23
14. Priest JW, Hagan RD. The effects of maximum steady state pace training on running performance. Br J Sports Med 1987; 21: 18-21
15. Yoshida T, Udo M, Chida M, et al. Specificity of physiological adaptation to endurance training in distance runners and competitive walkers. Eur J Appl Physiol 1990; 61: 197-201
16. Midgley AW, McNaughton LR, Wilkinson M. Is there an optimal training intensity for enhancing the $\dot{V}O_{2max}$ of distance runners? Empirical research findings, current opinions, physiological rationale and practical recommendations. Sports Med 2006; 36: 117-32
17. Londeree B. Effect of training on lactate/ventilatory thresholds: a meta-analysis. Med Sci Sports Exerc 1997; 29: 837-43
18. Pate RR, Branch JD. Training for endurance sport. Med Sci Sports Exerc 1992; 24 Suppl.: S340-3
19. Berg K. Endurance training and performance in runners: research limitations and unanswered questions. Sports Med 2003; 33: 59-73
20. Kurz MJ, Berg K, Latin R, et al. The relationship of training methods in NCAA division I cross-country runners and 10,000-meter performance. J Strength Cond Res 2000; 14: 196-201
21. Hawley JA, Myburgh KH, Noakes TD, et al. Training techniques to improve fatigue resistance and endurance performance. J Sports Sci 1997; 15: 325-33
22. Wells CL, Pate RR. Training for performance of prolonged exercise. In: Lamb DR, Murray R, editors. Perspectives in exercise science and sports medicine: volume 1, prolonged exercise. Indianapolis (IN): Benchmark Press, 1988, 357-91
23. Billat V, Flechet B, Petit B, et al. Interval training at $\dot{V}O_{2max}$: effects on aerobic performance and overtraining markers. Med Sci Sports Exerc 1999; 31: 156-63
24. Sjödin 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
25. Lehmann M, Dickhuth HH, Gendrisch G, et al. Training-overtraining: a prospective, experimental study with experienced middle- and long-distance runners. Br J Sports Med 1991; 12: 444-52
26. Londeree BR. The use of laboratory test results with long distance runners. Sports Med 1986; 3: 201-13
27. Hamilton RJ, Paton CD, Hopkins WG. Effect of high-intensity resistance training on performance of competitive distance runners. Int J Sports Physiol Perf 2006; 1: 40-9
28. Acevedo EO, Goldfarb AH. Increased training intensity effects on plasma lactate, ventilatory threshold, and endurance. Med Sci Sports Exerc 1989; 21: 563-8
29. Tanaka K, Watanabe H, Konishi Y, et al. Longitudinal associations between anaerobic threshold and distance running performance. Eur J Appl Physiol 1986; 55: 248-52
30. Daniels JT, Yarbrough RA, Foster C. Changes in $\dot{V}O_{2max}$ and running performance with training. Eur J Appl Physiol 1978; 39: 249-54
31. Paavolainen L, Häkkinen K, Hämäläinen I, et al. Explosive-strength training improves 5-km running time by improving running economy and muscle power. J Appl Physiol 1999; 86: 1527-33
32. Smith TP, McNaughton LR, Marshall KJ. Effects of 4-wk training using V_{max}/T_{max} on $\dot{V}O_{2max}$ and performance in athletes. Med Sci Sports Exerc 1999; 31: 892-6
33. Smith TP, Coombes JS, Geraghty DP. Optimising high-intensity treadmill training using the running speed at maximal O_2 uptake and the time for which this can be maintained. Eur J Appl Physiol 2003; 89: 337-43
34. Slawinski J, Demarle A, Koralsztein JP, et al. Effect of supralactate training on the relationship between mechanical stride descriptors and aerobic energy cost in trained runners. Arch Physiol Biochem 2001; 109: 110-6
35. Mikesell KA, Dudley GA. Influence of intense endurance training on aerobic power of competitive distance runners. Med Sci Sports Exerc 1984; 16: 371-5
36. Laffite LP, Mille-Hamard L, Koralstein JP, et al. The effect of interval training on oxygen pulse and performance in supra-threshold runs. Arch Physiol Biochem 2003; 111: 202-10
37. Bickham DC, Bentley DJ, Le Rossignol PF, et al. The effects of short-term sprint training on MCT expression in moderately endurance-trained runners. Eur J Appl Physiol 2006; 96: 636-43
38. Spurrs RW, Murphy AJ, Watsford ML. The effect of plyometric training on distance running performance. Eur J Appl Physiol 2003; 89: 1-7
39. Billat V, Sirvent P, Lepretre PM, et al. Training effect on performance, substrate balance and blood lactate concentration at maximal lactate steady state in master endurance runners. Pflugers Arch 2004; 447: 875-83
40. Franch J, Madsen K, Djurhuus MS, et al. Improved running economy following intensified training correlates with reduced ventilatory demands. Med Sci Sports Exerc 1998; 30: 1250-6

41. Johnston RE, Quinn TJ, Kertzer R, et al. Strength training in female distance runners: impact on running economy. J Strength Cond Res 1997; 11: 224-9
42. Turner AM, Owings M, Schwane JA. Improvement in running economy after 6 weeks of plyometric training. J Strength Cond Res 2003; 17: 60-7
43. Olsen R, Berg K, Latin R, et al. Comparison of two intense interval training programs on maximal oxygen uptake and running performance. J Sports Med Phys Fitness 1988; 28: 158-64
44. Viru A. The mechanism of training effects: an hypothesis. Int J Sports Med 1984; 5: 219-27
45. Billat V, Demarle A, Paiva M, et al. Effect of training on the physiological factors of performance in elite marathon runners (males and runners). Int J Sports Med 2002; 23: 335-41
46. Pollock ML. The quantification of endurance training programs. Exerc Sport Sci Rev 1973; 1: 155-88
47. Casaburi R, Storer TW, Sullivan CS, et al. Evaluation of blood lactate elevation as an intensity criterion for exercise testing. Med Sci Sports Exerc 1995; 27: 852-62
48. Saltin B, Essén B, Pederson PK. Intermittent exercise: its physiology and some practical applications. In: Jokl E, editor. Advances in exercise physiology. Basel: Karger, 1976: 23-51
49. Smirnov M. Do we need a methodological reform? Mod Athl Coach 1998; 36 (2): 33-6
50. Smith DJ. A framework for understanding the training process leading to elite performance. Sports Med 2003; 33: 1103-26
51. Hagan RD, Smith MG, Gettman LR. Marathon performance in relation to maximal aerobic power and training indices. Med Sci Sports Exerc 1981; 13: 185-9
52. Billat V, Lepretre PM, Heugas AM, et al. Training and bioenergetic characteristics in elite male and female Kenyan runners. Med Sci Sports Exerc 2003; 35: 297-304
53. Foster C, Florhaug JA, Franklin J, et al. A new approach to monitoring exercise training. J Strength Cond Res 2001; 15: 109-15
54. Seiler KS, Kjerland GØ. Quantifying training intensity distribution in elite endurance athletes: is there evidence for an 'optimal' distribution. Scand J Med Sci Sports 2006; 16: 49-56
55. Neumann G, Gohlitz D. Training control in running with event-specific endurance tests [in German]. Leistungssport 1996 (1); 26: 63-7
56. Karboviak RJ. Using GPS technology to monitor intensity, speed, and training volume in outdoor athletes. Strength Cond J 2005; 27: 24-5
57. Schutz Y, Herren R. Assessment of speed of human locomotion using a differential satellite global positioning system. Med Sci Sports Exerc 2000; 32: 642-6
58. Weltman A, Snead D, Seip R, et al. Percentages of maximal heart rate, heart rate reserve and $\dot{V}O_{2max}$ for determining endurance training intensity in male runners. Int J Sport Med 1990; 11: 218-22
59. Calvert TW, Banister EW, Savage MV, et al. A systems model of the effects of training on physical performance. IEEE Trans Syst Man Cybernet 1976; 6: 94-102
60. Morton RH. Fitz-Clarke, JR, Banister EW. Modeling human performance in running. J Appl Physiol 1990; 69: 1171-7
61. Garcin M, Fleury A, Ansart L, et al. Training content and potential impact on performance: a comparison of young male and female endurance-trained runners. Res Q 2006; 77: 351-61
62. Jones AM, Doust JH. Limitations to submaximal exercise performance. In: Eston R, Reilly T, editors. Kinanthropometry and exercise physiology laboratory manual, vol. 2. London: Routledge, 2001: 235-62
63. Wells JG, Balke B, van Fossan DD. Lactic acid accumulation during work: a suggested standardization of work classification. J Appl Physiol 1957; 10: 51-5
64. Jones AM. The physiology of the world record holder for the women's marathon. Int J Sports Sci Coach 2006; 1: 101-16
65. Esteve-Lanao J, San Juan AF, Earnest CP, et al. How do endurance runners actually train? Relationships with competition performance. Med Sci Sports Exerc 2005; 37: 496-504
66. Faria EW, Parker DL, Faria IE. The science of cycling: physiology and training – part 1. Sports Med 2005; 35: 285-312
67. Fry RW, Morton AR, Keast D. Periodisation of training stress: a review. Can J Sport Sci 1992; 17: 234-40
68. Viru A, Viru M. Biochemical monitoring of sport training. Champaign (IL): Human Kinetics, 2001
69. Verhoshansky Y. Main features of a modern scientific sports training theory. New Stud Athlet 1998; 13: 9-20
70. Hewson DJ, Hopkins WG. Prescribed and self-reported seasonal training of distance runners. J Sports Sci 1995; 13: 463-70
71. Verhoshansky Y. The end of 'periodization' in the training of high-performance sport. Mod Athl Coach 1999; 37 (2): 14-8
72. Karikosk O. Training volume in distance running. Mod Athl Coach 1984; 22 (2): 18-20
73. Basset FA, Chouinard R, Boulay MR. Training profile counts for time-to-exhaustion performance. Can J Appl Physiol 2003; 28: 654-66
74. Föhrenbach R, Mader A, Hollman W. Determination of endurance capacity and prediction of exercise intensities for training and competition in marathon runners. Int J Sports Med 1987; 8: 11-8
75. Robinson DM, Robinson SM, Hume PA, et al. Training intensity of elite male distance runners. Med Sci Sports Exerc 1991; 23: 1078-82
76. Laursen PB, Jenkins DJ. The scientific basis of high-intensity interval training: optimising training programmes and maximising performance in highly trained athletes. Sports Med 2002; 32: 53-73
77. Arrese AL, Ostáriz ES, Mallén JAC, et al. The changes in running performance and maximal oxygen uptake after long-term training in elite athletes. J Sports Med Phys Fitness 2005; 45: 435-40
78. Berg K, Latin RW, Hendricks T. Physiological and physical performance changes in female runners during one year of training. Sports Med Training Rehab 1995; 5: 311-9
79. Daniels J. Running with Jim Ryan: a five year case study. Phys Sports Med 1974; 2: 63-7
80. Jones AM. A five year physiological case study of an Olympic runner. Br J Sports Med 1998; 32: 39-43
81. Martin EE, Vroon DH, May DF, et al. Physiological changes in elite male distance runners training. Phys Sports Med 1986; 14: 152-71
82. Sjödin B, Svedenhag J. Applied physiology of marathon running. Sports Med 1985; 2: 83-99
83. Fox EL, Bartels RL, Billings CE, et al. Intensity and distance of interval training and changes in aerobic power. Med Sci Sports 1973; 5: 18-22
84. Shephard RJ. Intensity, duration and frequency of exercise as determinants of the response to a training regime. Int Z Angew Physiol 1968; 26: 272-8
85. Wenger HA, Bell GJ. The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. Sports Med 1986; 3: 346-56

86. Brisswalter J, Legros P. Variability in energy cost of running during one training season in high level runners. J Sports Med Phys Fitness 1994; 34: 135-40
87. Conley DL, Krahenbuhl GS, Burkett LN, et al. Following Steve Scott: physiological changes accompanying training. Phys Sports Med 1984; 12: 103-6
88. Svedenhag J, Sjödin B. Physiological characteristics of elite male runners in and off-season. Can J Appl Sport Sci 1985; 10: 127-33
89. Wilcox AR, Bulbulian R. Changes in running economy to $\dot{V}O_{2max}$ during a cross-country season. J Sports Med Phys Fitness 1984; 24: 321-6
90. Daniels J, Scardina N. Interval training and performance. Sports Med 1984; 1: 327-34
91. Hill DW, Rowell AL. Responses to exercise at the velocity associated with $\dot{V}O_{2max}$. Med Sci Sports Exerc 1997; 29: 113-6
92. Laursen PB, Shing CM, Peake JM, et al. Interval training program optimisation in highly trained endurance cyclists. Med Sci Sports Exerc 2002; 34: 1801-7
93. Tabata I, Irisawa K, Kouzaki M, et al. Metabolic profile of high intensity intermittent exercises. Med Sci Sports Exerc 1997; 29: 390-5
94. Vuorimaa T, Karvonen J. Recovery time in interval training for increasing aerobic capacity. Ann Sports Med 1988; 3: 215-9
95. Wenger HA, McNab RBJ. Endurance training: the effects of intensity, total work, duration and initial fitness. J Sports Med Phys Fitness 1975; 15: 199-211
96. Gledhill N, Cox D, Jamnik R. Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. Med Sci Sports Exerc 1994; 26: 1116-21
97. Zhou B, Conlee RK, Jensen R, et al. Stroke volume does not plateau during graded exercise in elite male distance runners. Med Sci Sports Exerc 2001; 33: 1849-54
98. Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev 1977; 57: 779-815
99. Cooper G. Basic determinants of myocardial hypertrophy: a review of molecular mechanisms. Annu Rev Med 1997; 48: 13-23
100. Ouellet Y, Poh SC, Becklake MR. Circulatory factors limiting maximal aerobic exercise capacity. J Appl Physiol 1969; 27: 874-80
101. Saltin B, Rowell LB. Functional adaptations to physical activity and inactivity. Fed Proc 1980; 39: 1506-13
102. Billat VL. Interval training for performance: a scientific and empirical practice – special recommendations for middle- and long-distance running. Part I: aerobic interval training. Sports Med 2001; 3: 13-31
103. Midgley AW, McNaughton LR. Time at or near $\dot{V}O_{2max}$ during continuous and intermittent running: a review with special reference to considerations for the optimisation of training protocols to elicit the longest time at or near $\dot{V}O_{2max}$. J Sports Med Phys Fitness 2006; 46: 1-14
104. Åstrand I, Åstrand PO, Christensen EH, et al. Intermittent muscular work. Acta Physiol Scand 1960; 48: 448-53
105. MacDougall D, Sale D. Continuous vs interval training: a review for the athlete and the coach. Can J Appl Sport Sci 1981; 6: 93-7
106. Moffatt RJ, Stamford BA, Weltman A, et al. Effects of high-intensity aerobic training on maximal oxygen uptake capacity and field test performance. J Sports Med Phys Fitness 1977; 17: 351-9
107. Demarie S, Koralsztein JP, Billat V. Time limit and time at $\dot{V}O_{2max}$, during a continuous and an intermittent run. J Sports Med Phys Fitness 2000; 40: 96-102
108. Billat VL, Slawinski J, Bocquet V, et al. Very short (15s–15s) interval-training around the critical velocity allows middle-aged runners to maintain $\dot{V}O_{2max}$ for 14 minutes. Int J Sports Med 2001; 22: 201-8
109. Billat V, Slawinski J, Bocquet V, et al. Intermittent runs at the velocity associated with maximal oxygen uptake enables subjects to remain at maximal oxygen uptake for a longer time than intense but submaximal runs. Eur J Appl Physiol 2000; 81: 188-96
110. Midgley AW, McNaughton LR, Carroll S. Physiological determinants of time to exhaustion during intermittent treadmill running at $v\dot{V}O_{2max}$. Int J Sports Med 2007; 28: 273-80
111. Coyle EF. Very intense exercise-training is extremely potent and time efficient: a reminder. J Appl Physiol 2005; 98: 1983-4
112. Paavolainen L, Nummela A, Rusko H. Muscle power factors and $\dot{V}O_{2max}$ as determinants of horizontal and uphill running performance. Scand J Med Sci Sports 2000; 10: 286-91
113. Burleson Jr MA, O'Bryant HS, Stone MH, et al. Effect of weight training exercise and treadmill exercise on post-exercise oxygen consumption. Med Sci Sports Exerc 1998; 30: 518-22
114. MacDougall JD, Sale DG, Moroz JR, et al. Mitochondrial volume density in human skeletal muscle following heavy resistance training. Med Sci Sports Exerc 1979; 11: 164-6
115. Moritani T, deVries HA. Neural factors versus hypertrophy in the time course of muscle strength gain. Am J Phys Med 1979; 58: 115-30
116. Bangsbo J, Juel C. Lactic acid accumulation is a disadvantage during muscle activity. J Appl Physiol 2006; 100: 1412-3
117. Cairns SP. Lactic acid and exercise performance: culprit or friend? Sports Med 2006; 36: 279-91
118. Lamb GD, Stephenson DG. Lactic acid accumulation is an advantage during muscle activity. J Appl Physiol 2006; 100: 1410-2
119. Billat VL. Use of blood lactate measurements for prediction of exercise performance and for control of training recommendations for long-distance running. Sports Med 1996; 22: 157-75
120. MacDougall JD. The anaerobic threshold: its significance for the endurance athlete. Can J Appl Sport Sci 1977; 2: 137-40
121. Tanaka K. Lactate-related factors as a critical determinant of endurance. Ann Physiol Anthrop 1990; 9: 191-202
122. Anderson O. Lactate lift-off. Lansing (MI): SSS Publishing Inc., 1998
123. Ivy JL, Withers RT, Van Handel PJ, et al. Muscle respiratory capacity and fibre type as determinants of the lactate threshold. J Appl Physiol 1980; 48: 523-7
124. Coen B, Schwarz L, Urhausen A, et al. Control of training in middle- and long-distance running by means of the individual anaerobic threshold. Int J Sports Med 1991; 12: 519-24
125. Henritze J, Weltman A, Schurrer RL, et al. Effects of training at and above the lactate threshold on the lactate threshold and maximal oxygen uptake. Eur J Appl Physiol 1985; 54: 84-8
126. Weltman A, Seip RL, Snead D, et al. Exercise training at and above the lactate threshold in previously untrained women. Int J Sports Med 1992; 13: 257-63
127. Belman MJ, Gaesser GA. Exercise training below and above the lactate threshold in the elderly. Med Sci Sports Exerc 1991; 23: 562-8

128. Ekblom B, Åstrand PO, Saltin B, et al. Effect of training on circulatory response to exercise. J Appl Physiol 1968; 24: 518-28
129. Boileau RA, Mayhew JL, Riner WF, et al. Physiological characteristics of elite middle and long distance runners. Can J Appl Sport Sci 1982; 7: 167-72
130. Sjödin B, Jacobs I. Onset of blood lactate accumulation and marathon running performance. Int J Sports Med 1981; 2: 23-6
131. Gollnick PD, Piehl K, Saltin B. Selective glycogen depletion pattern in human muscle fibres after exercise of varying intensity and at varying pedalling rates. J Physiol 1974; 241: 45-57
132. Anderson GS, Rhodes EC. A review of blood lactate and ventilatory methods of detecting transition thresholds. Sports Med 1989; 8: 43-55
133. Mader A. Evaluation of the endurance performance of marathon runners and theoretical analysis of test results. J Sports Med Phys Fitness 1991; 31: 1-19
134. Rerych SK, Scholz PM, Sabiston DC, et al. Effects of exercise training on left ventricular function in normal subjects: a longitudinal study by radionuclide angiography. Am J Cardiol 1980; 45: 244-52
135. Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training. Annu Rev Physiol 1983; 45: 169-89
136. Saltin B, Blomqvist G, Mitchell JH, et al. Response to exercise after bed rest and after training: a longitudinal study of adaptive changes in oxygen transport and body composition. Circulation 1968; 38 Suppl. 7: 1-78
137. Hagberg JM, Goldberg AP, Lakatta L, et al. Expanded blood volumes contribute to the increased cardiovascular performance of endurance-trained older men. J Appl Physiol 1998; 85: 484-9
138. Warburton DER, Haykowsky MJ, Quinney HA, et al. Blood volume expansion and cardiorespiratory function: effects of training modality. Med Sci Sports Exerc 2004; 36: 991-1000
139. Ingjer F. Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. J Physiol 1979; 294: 419-32
140. Brodal P, Ingjer F, Hermansen L. Capillary supply of skeletal muscle fibres in untrained and endurance-trained men. Am J Physiol 1977; 232: H705-12
141. Hoppeler H, Howald H, Conley K, et al. Endurance training in humans: aerobic capacity and structure of skeletal muscle. J Appl Physiol 1985; 59: 320-7
142. Hoppeler H, Flück M. Plasticity of skeletal muscle mitochondria: structure and function. Med Sci Sports Exerc 2003; 35: 95-104
143. Harms SJ, Hickson RC. Skeletal muscle mitochondria and myoglobin, endurance, and intensity of training. J Appl Physiol 1983; 54: 798-802
144. Wittenberg JB, Wittenberg BA. Myoglobin function reassessed. J Exp Biol 2003; 206: 2011-20
145. Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol 1984; 56: 831-8
146. Dubouchaud H, Butterfield GE, Wolfel EE, et al. Endurance training, expression, and physiology of LDH, MCT1, and MCT4 in human skeletal muscle. Am J Physiol 2000; 278: E571-9
147. Marcinik EJ, Potts J, Schlabach G, et al. Effects of strength training on lactate threshold and endurance performance. Med Sci Sports Exerc 1991; 23: 739-43
148. Crow MT, Kushmerick MJ. Chemical energetics of slow-and fast-twitch muscles of the mouse. J Gen Physiol 1982; 79: 147-66
149. Nelson RC, Gregor RJ. Biomechanics of distance running: a longitudinal study. Res Q 1976; 47: 417-28
150. Williams KR, Cavanagh PR. Relationship between distance running mechanics, running economy, and performance. J Appl Physiol 1987; 63: 1236-45
151. Craib MW, Mitchell VA, Fields KB, et al. The association between flexibility and running economy in sub-elite distance runners. Med Sci Sports Exerc 1996; 28: 737-43
152. Bosquet L, Léger L, Legros P. Methods to determine aerobic endurance. Sports Med 2002; 32: 675-700
153. Caird SJ, McKenzie AD, Sleivert GG. Biofeedback and relaxation techniques improve running economy in sub-elite long distance runners. Med Sci Sports Exerc 1999; 31: 717-22
154. Morgan DW, Bransford DR, Costill DL, et al. Variation in the aerobic demand of running among trained and untrained subjects. Med Sci Sports Exerc 1995; 27: 404-9
155. Pate RR, Macera CA, Bailey SP, et al. Physiological, anthropometric, and training correlates of running economy. Med Sci Sports Exerc 1992; 24: 1128-33
156. Mayhew JL. Oxygen cost and energy expenditure of running in trained runners. Br J Sports Med 1977; 11: 116-21
157. Jones AM, Carter H. The effect of endurance training on parameters of aerobic fitness. Sports Med 2000; 29: 373-86
158. Morgan DW, Martin PE, Krahenbuhl GS. Factors affecting running economy. Sports Med 1989; 7: 310-30
159. Abe T, Kojima K, Kearns CF, et al. Whole body muscle hypertrophy from resistance training: distribution and total mass. Br J Sports Med 2003; 37: 543-5
160. Costill DL. The relationship between selected physiological variables and distance running performance. J Sports Med Phys Fitness 1967; 7: 61-6
161. Krause V. Marathon preparations. Mod Athl Coach 1989; 27 (2): 17-20
162. Billat VL. Current perspectives on performance improvement in the marathon: from universalisation to training optimisation. New Stud Athl 2005; 3: 21-39
163. Croker N. The body balance of female distance runners. In: Jarver J, editor. Long distances: contemporary theory, technique and training. Mountain View (CA): Tafnews Press, 1995: 119-21
164. Jones AM. Running economy is negatively related to sit-and-reach test performance in international-standard runners. Int J Sports Med 2002; 23: 40-3
165. Andersen JC. Stretching before and after exercise: effect on muscle soreness and injury risk. J Athl Train 2005; 40: 218-20
166. Beaudoin CM, Blum JW. Flexibility and running economy in female collegiate track athletes. J Sports Med Phys Fitness 2005; 45: 295-300
167. Nelson AG, Kokkonen J, Eldredge C, et al. Chronic stretching and running economy. Scand J Med Sci Sports 2001; 11: 260-5
168. Bailey SP, Pate RR. Feasibility of improving running economy. Sports Med 1991; 12: 228-36
169. Podolin DA, Munger PA, Mazzeo RS. Plasma catecholamine and lactate response during graded exercise with varied glycogen conditions. J Appl Physiol 1991; 71: 1427-33
170. Suslov F. How much strength is needed in endurance events? Mod Athl Coach 1997; 35 (4): 9-12
171. Carpinelli RN, Otto RM, Winett RA. A critical analysis of the ACSM position stand on resistance training: insufficient

evidence to support recommended training protocols. J Exerc Physiol 2004; 7: 1-60
172. Bushman BA, Flynn MG, Andres FF, et al. Effect of deep water run training on running performance. Med Sci Sports Exerc 1997; 29: 694-9
173. Wilber RL, Moffatt RJ, Scott BE, et al. Influence of water-run training on the maintenance of aerobic performance. Med Sci Sports Exerc 1996; 28: 1056-62
174. Lydiard A. Training for running races. N Z Vet J 1979; 23: 260-1
175. Smith GC, Jones AM. The relationship between critical velocity, maximal lactate steady-state velocity and lactate turnpoint velocity in runners. Eur J Appl Physiol 2001; 85: 19-26
176. Coffey VG, Hawley JA. Training for performance: insights from molecular biology. Int J Sports Physiol Perf 2006; 1: 284-92
177. MacArthur DG, North KN. Genes and human elite performance. Hum Genet 2005; 116: 331-9
178. Allen DL, Harrison BC, Leinwand LA. Molecular and genetic approaches to studying exercise performance and adaptation. Exerc Sport Sci Rev 2002; 30: 99-105

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