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Is there an Optimal Training Intensity for Enhancing the Maximal Oxygen Uptake of Distance Runners?

Empirical Research Findings, Current Opinions, Physiological Rationale and Practical Recommendations

Adrian W. Midgley, Lars R. McNaughton and *Michael Wilkinson*

Department of Sport, Health and Exercise Science, University of Hull, Hull, England

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Abstract

The maximal oxygen uptake ($\dot{V}O_{2max}$) is considered an important physiological determinant of middle- and long-distance running performance. Little information exists in the scientific literature relating to the most effective training intensity for the enhancement of $\dot{V}O_{2max}$ in well trained distance runners. Training intensities of 40–50% $\dot{V}O_{2max}$ can increase $\dot{V}O_{2max}$ substantially in untrained individuals. The minimum training intensity that elicits the enhancement of $\dot{V}O_{2max}$ is highly dependent on the initial $\dot{V}O_{2max}$, however, and well trained distance runners probably need to train at relative high percentages of $\dot{V}O_{2max}$ to elicit further increments. Some authors have suggested that training at 70–80% $\dot{V}O_{2max}$ is optimal. Many studies have investigated the maximum amount of time runners can maintain 95–100% $\dot{V}O_{2max}$ with the assertion that this intensity is optimal in enhancing $\dot{V}O_{2max}$. Presently, there have been no well controlled training studies to support this premise.

Myocardial morphological changes that increase maximal stroke volume, increased capillarisation of skeletal muscle, increased myoglobin concentration, and increased oxidative capacity of type II skeletal muscle fibres are adaptations associated with the enhancement of $\dot{V}O_{2max}$. The strength of stimuli that elicit adaptation is exercise intensity dependent up to $\dot{V}O_{2max}$, indicating that training at or near $\dot{V}O_{2max}$ may be the most effective intensity to enhance $\dot{V}O_{2max}$ in well trained distance runners. Lower training intensities may induce similar adaptation because the physiological stress can be imposed for longer periods. This is probably only true for moderately trained runners, however, because all cardiorespiratory adaptations elicited by submaximal training have probably already been elicited in distance runners competing at a relatively high level.

Well trained distance runners have been reported to reach a plateau in $\dot{V}O_{2max}$ enhancement; however, many studies have demonstrated that the $\dot{V}O_{2max}$ of well trained runners can be enhanced when training protocols known to elicit 95–100% $\dot{V}O_{2max}$ are included in their training programmes. This supports the premise that high-intensity training may be effective or even necessary for well trained distance runners to enhance $\dot{V}O_{2max}$. However, the efficacy of optimised protocols for enhancing $\dot{V}O_{2max}$ needs to be established with well controlled studies in which they are compared with protocols involving other training intensities typically used by distance runners to enhance $\dot{V}O_{2max}$.

Runners of all levels of ability, from the competitive club runner to the elite performer, seek effective training methods to enhance performance in an attempt to better personal best performances, break records, or improve medal prospects. Long-term performance enhancement requires training loads of sufficient intensity and duration to progressively overload and stress the physiological structures and processes that determine performance. The effect of manipulating training intensity and duration to elicit adaptations that enhance particular physiological determinants of performance has been considered for over 30 years,[1] although probably more questions relating to this topic remain unanswered than have been presently resolved.

Many training methods are largely a product of the trial-and-error approach used by coaches of successful athletes and teams, or based on training schedules of current world-class athletes.[2,3] Although the trial-and-error approach has probably advanced the overall effectiveness of the training programmes given to athletes, many of the common methods employed have received little scientific support.[4] During the trial-and-error process many training and non-training variables may be responsible for improving an athlete's performance, and isolating the effect of a particular change in training strategy is difficult. Basing training programmes on those used by world-class athletes is also problematic, because these individuals undoubtedly possess genotypes and training responses uncharacteristic of most competitive athletes.[5,6] Confidence in the effectiveness of a training method to enhance a particular physiological determinant of performance is therefore limited, and scientific scrutiny of well established and contemporary training methods is required.

The maximal oxygen uptake, or $\dot{V}O_{2max}$, is the maximum rate that oxygen can be taken up from the ambient air and transported to and used by cells for cellular respiration during physical activity.[7] The $\dot{V}O_{2max}$ has been considered by some to be an important physiological determinant of middle- and long-distance running performance.[8-10] Many distance runners strive to enhance $\dot{V}O_{2max}$[11] and therefore training routines that most effectively enhance $\dot{V}O_{2max}$ would seem valuable. Several authors have suggested that intensity is the most im-

portant training variable that can be manipulated for eliciting the training-induced enhancement of $\dot{V}O_{2max}$,[12-14] although the total work performed has also been considered most important.[1] There appears to be little information in the scientific literature relating to the most effective training intensity for the enhancement of $\dot{V}O_{2max}$ in well trained distance runners. Furthermore, there has been no comprehensive attempt to describe the physiological rationale for choosing a particular intensity as the most effective for enhancing $\dot{V}O_{2max}$.

The main aim of this article is to review findings of research and highlight current opinions on which is the most effective training intensity to enhance the $\dot{V}O_{2max}$ of well trained distance runners. A second aim is to review physiological adaptations associated with the training-induced enhancement of $\dot{V}O_{2max}$ and discuss the effect of training intensity in eliciting these adaptations. Finally, practical recommendations for distance runners are made primarily based on the preceding review.

The $\dot{V}O_{2max}$ was chosen as the focus of the present review because historically it was probably the first physiological determinant of distance running performance to be identified. However, effective training methods for distance runners that target its enhancement remain poorly defined. Moreover, as highlighted earlier, $\dot{V}O_{2max}$ is an important physiological determinant of both middle- and long-distance running performance.[8-10] Distance runners in the context of this article refer to runners that compete in running events of between 1500m and 42.2km, and distance running is defined accordingly.

1. Maximal Oxygen Uptake ($\dot{V}O_{2max}$) as an Important Determinant of Distance Running Performance

Important physiological determinants of distance running performance may include the $\dot{V}O_{2max}$,[9] the lactate threshold,[15] running economy[16] and anaerobic capacity.[17] Performance $\dot{V}O_2$ is the maximum rate of oxygen utilisation sustainable for the duration of a race.[18] Its importance in dictating running velocity becomes greater as race distances increase and contributions to total energy production from anaerobic metabolism decrease. Although performance $\dot{V}O_2$ in long-distance running events is largely dictated by the lactate threshold velocity, $\dot{V}O_{2max}$ is arguably the most important factor in dictating performance $\dot{V}O_2$, as the $\dot{V}O_{2max}$ sets the upper limit for the $\dot{V}O_2$ at the lactate threshold.[19] Moreover, well trained long-distance runners who have exclusively trained at submaximal velocities for many years will probably have a very high fractional utilisation of $\dot{V}O_{2max}$,[20] and the enhancement of $\dot{V}O_{2max}$ may be necessary to increase performance $\dot{V}O_2$. In middle-distance events, runners sustain velocities with a $\dot{V}O_2$ demand greater than $\dot{V}O_{2max}$.[8] In events of sufficient duration to allow $\dot{V}O_2$ to reach its maximum, such as 1500m and 3000m races, a higher $\dot{V}O_{2max}$ will invariably increase sustainable metabolic power, regardless of lactate threshold velocity.

Several studies have used correlation coefficients to demonstrate that $\dot{V}O_{2max}$ is a poor predictor of performance in well trained distance runners.[16,21,22] However, the runners used in these studies were homogeneous in terms of $\dot{V}O_{2max}$ values and performance times, and the use of correlation coefficients is inappropriate because the size of a correlation coefficient is strongly dependent on the range of the values that are being correlated.[23] Regardless, the question relating to the strength of the relationship between $\dot{V}O_{2max}$ and distance running performance is probably of significance only when considering predicting a runner's performance from laboratory test results. A more important question in relation to the context of the present review is whether an increase in $\dot{V}O_{2max}$ will enhance distance running performance. The previous discussion pertaining to the influence of $\dot{V}O_{2max}$ on performance $\dot{V}O_2$ suggests that an appreciable increase in $\dot{V}O_{2max}$ will improve performance in middle- and long-distance running.

2. Changes in the $\dot{V}O_{2max}$ of Distance Runners

Ekblom[24] suggested the training-induced enhancement of $\dot{V}O_{2max}$ plateaus after several years of

Table I. Summary of training studies that reported changes in the maximal oxygen uptake ($\dot{V}O_{2max}$) of elite or well trained runners in response to high-intensity training

Subjects	Age (y)[a]	Initial $\dot{V}O_{2max}$ (mL/kg/min)[a]	Study duration (wk)	Characteristics of high-intensity training included in the weekly training	Increase in $\dot{V}O_{2max}$ (%)	Statistically significant?	Reference
8 male MDR and LDR	24 (3.2)	71.2 (5.0)	4	One interval session ran at v$\dot{V}O_{2max}$	2.1	No	37
5 male and 4 female elite marathon runners	34 (6.0)	66.3 (9.2)	8	Two interval sessions at 3km and 10km race pace	5.4	Yes	35
7 male LDR	22 (3.4)	65.3 (2.4)	8	One interval session ran at 90–95% HR_{max} and two Fartlek sessions at just below and above 10km pace	0.7	No	43
7 male LDR	24 (9.3)	61.0 (3.7)	6	Maximal effort runs eliciting heart rates at or above 190 beats/min	4.1	No	38
7 male MDR and LDR	25 (11.9)	61.0 (11.6)	8	Two interval sessions ran at vΔ50 (≈93% v$\dot{V}O_{2max}$)	4.0	No	39
5 male MDR	23 (10.1)	61.0 (6.6)	4	Two interval sessions ran at v$\dot{V}O_{2max}$	4.9	No	40
27 male MDR, LDR and triathletes[b]	25 (6.8)	60.5 (9.8)	4	Two interval sessions ran at v$\dot{V}O_{2max}$	5.0	No	41

a Mean (SD). Where standard error of the mean was reported, the standard deviation was calculated using the formula: SEM $\sqrt{n}$.

b Two experimental groups and one control group each with nine subjects. Increase in $\dot{V}O_{2max}$ is reported as the mean of the two experimental groups. These groups differed only by a small difference in work internal length.

HR_{max} = maximum heart rate; **LDR** = long-distance runners; **v$\dot{V}O_{2max}$** = minimal running velocity that elicits $\dot{V}O_{2max}$ during incremental running to volitional exhaustion; **vΔ50** = velocity midway between the lactate threshold velocity and v$\dot{V}O_{2max}$.

training. If this were true, training to enhance $\dot{V}O_{2max}$ would be of value only to novice and moderately trained runners, and well trained runners should target other physiological determinants of performance. Longitudinal changes in the $\dot{V}O_{2max}$ of well trained runners reported in the literature have mostly been small, or otherwise, no change has occurred.[24-28] In fact, Ekblom, a 1964 Swedish orienteering champion, reported that his own $\dot{V}O_{2max}$ had not changed between 1960 and 1968.[24] Martin et al.[28] reported no significant change in the $\dot{V}O_{2max}$ of nine elite distance runners during a 2.5-year build up to the 1984 Olympic Games. No significant changes in $\dot{V}O_{2max}$ occurred in three well trained runners during 5 years of training,[24,26,27] or seven university track and cross-country runners during 1 year of training.[25]

Experimentally, whether a runner has reached his or her trainable limit for $\dot{V}O_{2max}$ enhancement is difficult to ascertain. Laursen and Jenkins[29] suggested all cardiorespiratory adaptations that could be elicited by submaximal training have probably already occurred in distance runners competing at a relatively high level. It is possible that many well trained runners do not include sufficient volumes of high intensity training in their training programmes to reach their trainable limit for $\dot{V}O_{2max}$ enhancement. Basset et al.[30] reported that well trained long-distance runners used in their study invariably trained at running velocities below v$\dot{V}O_{2max}$ (the minimal running velocity that elicits $\dot{V}O_{2max}$ during incremental running to volitional exhaustion[31]). Robinson et al.[32] reported that 17 nationally ranked distance runners performed <4% of their training sessions as high-intensity interval training, with one-third performing no interval training. Average training intensity was 64% $\dot{V}O_{2max}$. A retrospective study by Hewson and Hopkins[33] found that most of the 123 distance-running coaches surveyed favoured long slow distance training, with limited time allocated to either 'hard' continuous training or high-intensity interval training. Favouring training duration over intensity is also reflected in the high weekly training distances reported for well trained distance runners.[34-36]

Results of studies that reported changes in the $\dot{V}O_{2max}$ of elite and well trained runners in response to high-intensity training (table I) suggest that the $\dot{V}O_{2max}$ values of these runners have not reached a plateau and are responsive to high-intensity training, even during relatively short training periods. However, valid inferences cannot be made from these studies due to several methodological limitations. Only one of these studies reported statistically significant increases in $\dot{V}O_{2max}$.[35] Other studies demonstrated meaningful but statistically insignificant increases in $\dot{V}O_{2max}$ of 2–5%.[37-41] The small sample sizes used in these studies and the associated statistical power of <30% (Power and Precision, Biostat, NJ, USA) in all but one of these studies was probably a major cause of the statistical insignificance.[42] Several studies[37,39-41] reported changes in relative $\dot{V}O_{2max}$ but did not report whether any changes in body mass occurred. It is therefore not possible to quantify how much of the increase in relative $\dot{V}O_{2max}$ was due to changes in cardiorespiratory fitness and how much was due to any changes in body mass. The relatively large standard deviations in $\dot{V}O_{2max}$ in some of these studies[39-41] also suggest that some of the runners were not well trained. To more accurately estimate the effect of high-intensity training on the $\dot{V}O_{2max}$ of well trained and elite distance runners, further studies are clearly required involving larger sample sizes that possess adequate statistical power to detect small but meaningful changes in $\dot{V}O_{2max}$ and result in relatively narrow confidence intervals for the mean difference.

Although the $\dot{V}O_{2max}$ of well trained distance runners may be very similar when retested several years later,[24,26-28] substantial transient changes may occur.[26,44,45] Athletes frequently undertake periods of low training loads or stop training because of illness, injury or post-season breaks.[46] Reduced training loads are typically also incorporated into a periodised training plan.[33] Conley et al.[47] reported that the $\dot{V}O_{2max}$ of the mile record holder Steve Scott increased by 6% (0.34 L/min) in response to a progressive increase in high-intensity interval training and a reduction in long slow distance training over 9 months of periodised training. Effective training protocols to enhance $\dot{V}O_{2max}$ to competition level subsequent to transient reductions may therefore prove valuable. Circumstances in which effective training methods to enhance $\dot{V}O_{2max}$ may prove valuable are summarised in table II.

3. Current Opinions on Effective Training Intensities to Enhance the $\dot{V}O_{2max}$ of Distance Runners

During the initial stages of an endurance training programme, rapid increases in $\dot{V}O_{2max}$ may occur[48,49] and can be elicited with training intensities as low as 40–50% $\dot{V}O_{2max}$.[50,51] However, a review by Swain and Franklin,[52] highlighted that the minimal training intensity that elicits the enhancement of $\dot{V}O_{2max}$ is highly dependent on the initial $\dot{V}O_{2max}$. Well trained distance runners, who invariably possess $\dot{V}O_{2max}$ values almost double the population average,[10,34,53] therefore probably need to train at high percentages of $\dot{V}O_{2max}$ to enhance $\dot{V}O_{2max}$. Several authors have suggested that runners approaching their trainable limit for $\dot{V}O_{2max}$ enhancement may even need to attain and maintain $\dot{V}O_{2max}$ during training to elicit further increments.[20,29,54]

In addition to identifying a training intensity threshold above which $\dot{V}O_{2max}$ will increase, the rate of increase in $\dot{V}O_{2max}$ and the efficiency of training to enhance $\dot{V}O_{2max}$ in relation to the investment of time may be important considerations. MacDougall and Sale[55] suggested that training at approximately 75% $\dot{V}O_{2max}$ is optimal in enhancing $\dot{V}O_{2max}$ because myocardial stress and therefore the stimulus for myocardial adaptation are greatest at this intensity. Mader[56] suggested that the optimal training intensity is 60–80% $\dot{V}O_{2max}$, primarily based on the premise that higher training intensities are detrimental because of mitochondrial degeneration at high oxidation rates. Presently, there is insufficient evidence to support the efficacy of these training intensities for enhancing the $\dot{V}O_{2max}$ of well trained distance runners.

Training at or near $\dot{V}O_{2max}$ has also been suggested to be the optimal intensity to elicit further increments,[4,54,57-59] particularly in well trained ath-

Table II. Circumstances in which optimal training methods to enhance or maintain maximal oxygen uptake ($\dot{V}O_{2max}$) may prove valuable

Progressively increase $\dot{V}O_{2max}$ to its maximum trainable limit over the many years of a runner's competitive career
After a transient decrease in $\dot{V}O_{2max}$ due to scheduled periods of low-intensity training and relatively low total training loads as part of a periodised training programme
After an absence from training due to a scheduled lay-off (e.g. a long vacation)
After an absence from training due to an unscheduled lay-off (e.g. due to illness or injury)
When resuming a competitive career subsequent to retiring
Peaking prior to competition when all physiological capacities are maximised to their trainable limit
Decrease the amount of training time spent on enhancing $\dot{V}O_{2max}$ in an attempt to prevent excessive training loads, under recovery, and overtraining
Decrease the amount of training time spent on increasing $\dot{V}O_{2max}$ to allow more time to improve other performance determinants such as running economy and the lactate threshold
Decrease the total amount of training time while still stimulating or maintaining a high level of cardiorespiratory fitness. This may be valuable for competitive runners who have many other time-consuming commitments, and therefore have constraints on time that can be allocated for training

letes.[60] The interest in training at or near $\dot{V}O_{2max}$ as the optimal stimulus for its enhancement is reflected by numerous studies that have investigated the maximum amount of time that ≥95% $\dot{V}O_{2max}$ can be maintained during intermittent[61-63] and constant velocity running protocols.[54,62,64] Support for the premise that training at or near $\dot{V}O_{2max}$ is the optimal intensity for its enhancement comes from a review of 59 training studies in which it was concluded that the degree of enhancement in $\dot{V}O_{2max}$ was positively related to training intensity in the range of 50–100% $\dot{V}O_{2max}$.[14] This relationship existed almost irrespective of training frequency and duration, programme length, and initial $\dot{V}O_{2max}$. Table III summarises studies that compared changes in $\dot{V}O_{2max}$ in response to training at intensities associated with the attainment of $\dot{V}O_{2max}$, with other training intensities. These results suggest that training at $\dot{V}O_{2max}$ is no more effective at enhancing $\dot{V}O_{2max}$ than many other training intensities. However, because these studies mostly employed untrained or moderately trained individuals, used cycle ergometry, and did not report whether any of the subjects actually elicited $\dot{V}O_{2max}$ during training, valid inferences cannot be made relating to the efficacy of training at or near $\dot{V}O_{2max}$ in well trained distance runners. A training study that did include well trained distance runners did not equate the total work completed by the experimental and control groups, and so the results could not be interpreted.[41]

A limitation to research that bases the efficacy of training protocols to enhance $\dot{V}O_{2max}$, solely on time at or near $\dot{V}O_{2max}$, is that $\dot{V}O_{2max}$ can be elicited with different physiological responses. An extreme example is the attainment of $\dot{V}O_{2max}$ due to the superimposition of the $\dot{V}O_2$ slow component on the underlying $\dot{V}O_2$ response during severe continuous velocity exercise,[70] compared with interval training with 10-second work intervals.[71] Future studies investigating the efficacy of training protocols that elicit $\dot{V}O_{2max}$ should therefore evaluate the amount and type of stress these protocols impose on the physiological determinants of $\dot{V}O_{2max}$.

The question of whether or not training at or near $\dot{V}O_{2max}$ is the optimal method of enhancing $\dot{V}O_{2max}$ dates back at least to Daniels and Scardina.[57] Currently, this question as it pertains to competitive distance runners remains unresolved, and appropriate empirical research is required before the relative efficacy of this type of training in enhancing $\dot{V}O_{2max}$ can be ascertained. Future training studies that address this question need to: (i) recruit only well trained competitive distance runners; (ii) compare training at or near $\dot{V}O_{2max}$ with other training intensities typically performed by distance runners to enhance $\dot{V}O_{2max}$; (iii) employ stratified random sampling to help ensure the mean $\dot{V}O_{2max}$ of runners in each group is similar; (iv) equate the total work performed in each training group; (v) check that the training protocols designed to elicit $\dot{V}O_{2max}$ did actually elicit $\dot{V}O_{2max}$ and report the time that ≥95% $\dot{V}O_{2max}$ was maintained; and (vi) report changes in $\dot{V}O_{2max}$ in absolute values.

Table III. Summary of studies that have compared training at workloads associated with 100% maximal oxygen uptake ($\dot{V}O_{2max}$) with other training intensities

Subjects	Initial $\dot{V}O_{2max}$ (mL/kg/min)[a]	Exercise mode	Study duration (wk)	Work equated?[b]	Findings (training intensity and % improvement in absolute $\dot{V}O_{2max}$)	Reference
38 male cyclists/ triathletes	64.5 (5.1)	CE	4	No	100% $\dot{V}O_{2max}$ = 9%; 175% $\dot{V}O_{2max}$ = 3%	60
12 male runners	45.9 (–)	TR	8	Yes	100% $\dot{V}O_{2max}$ = 5%; 92% $\dot{V}O_{2max}$ = 6%	65
29 active males	44.1 (–)	CE	10	Yes	100% $\dot{V}O_{2max}$ = 9%; 120% $\dot{V}O_{2max}$ = 16%; 80% $\dot{V}O_{2max}$ = 8%	66
17 sedentary males	43.4 (–)	CE	8	Yes	105% $\dot{V}O_{2max}$ = 15%; 70% $\dot{V}O_{2max}$ = 8%; 50% $\dot{V}O_{2max}$ = 15%	51
14 sedentary males/ females	41.7 (–)	CE	7	Yes	100% $\dot{V}O_{2max}$ = 14%; 70% $\dot{V}O_{2max}$ = 15%	67
40 males (mixed fitness)	40.0 (–)	CE	8	Yes	100% $\dot{V}O_{2max}$ = 14%; 72% $\dot{V}O_{2max}$ = 18%; 55% $\dot{V}O_{2max}$ = 14%	68
36 males (mixed fitness)	38.0 (8.0)	CE	7	Yes	100% $\dot{V}O_{2max}$ = 33%; 60% $\dot{V}O_{2max}$ = 22%	59
12 sedentary males	36.5 (1.4)	CE	8	No	100% $\dot{V}O_{2max}$ = 11%; 70% $\dot{V}O_{2max}$ = 7%	69

a Mean (SD). Missing standard deviations due to the relative $\dot{V}O_{2max}$ being calculated from the absolute $\dot{V}O_{2max}$ and body mass that were reported in the original paper.

b Total work performed by each group similar.

CE = cycle ergometry; **TR** = treadmill running.

4. Training Intensity and Adaptations Associated with $\dot{V}O_{2max}$ Enhancement

Characterising the physiological response to increasing exercise intensity and how this response may elicit physiological adaptations that are associated with the enhancement of $\dot{V}O_{2max}$ may provide a physiological rationale for recommending a particular training intensity to enhance the $\dot{V}O_{2max}$ of distance runners. The following section therefore discusses the potential effect of training intensity in eliciting adaptations associated with $\dot{V}O_{2max}$ enhancement.

In accordance with a derivation of the Fick equation,[72] $\dot{V}O_{2max}$ is a product of the maximal cardiac output ($\dot{Q}_{max}$) and the maximal arterial-mixed venous oxygen difference (maximal $a-\bar{v}O_2$ difference). Any physiological structures or processes that determine these two variables could therefore potentially limit $\dot{V}O_{2max}$. It would also appear reasonable to theorise that any training-induced increase in the functional capacity of any one of these structures or processes should enhance $\dot{V}O_{2max}$.

4.1 Maximal Cardiac Output

Although alternative hypotheses have been presented,[73,74] most authors agree that in young healthy individuals performing maximal whole body exercise at sea level, $\dot{V}O_{2max}$ is predominantly limited by $\dot{Q}_{max}$[7,75,76] (readers are directed to a series of papers[77-80] debating this issue). Training-induced increases in $\dot{Q}_{max}$ are due to increased maximal stroke volume (SV_{max}), because the maximal heart rate either decreases or remains the same.[81] The main stimulus for myocardium morphological adaptation associated with SV_{max} enhancement is mechanical overload imposed by a volume overload-induced increase in ventricular diastolic stretch and increased resistance to ventricular emptying due to increased afterload.[82,83]

MacDougall and Sale[55] suggested that training at approximately 75% $\dot{V}O_{2max}$ is optimal in stimulating favourable myocardial adaptations, because stroke volume plateaus at approximately 40–50% $\dot{V}O_{2max}$ and mean arterial pressure at 70–80%

$\dot{V}O_{2max}$, so therefore the contractile force of the myocardium is probably maximum at approximately 75% $\dot{V}O_{2max}$. Early studies demonstrated that stroke volume plateaus at approximately 40–75% $\dot{V}O_{2max}$[84,85] and may even decrease as $\dot{V}O_{2max}$ is approached.[86] More recent studies found that in well trained individuals, stroke volume[87-89] and systolic and mean arterial blood pressures[87,90,91] progressively rise in response to increasing exercise intensity up to $\dot{V}O_{2max}$, and therefore myocardial stress should be maximal at $\dot{V}O_{2max}$.

Neuroendocrine factors such as thyroxine, testosterone, angiotensin II, and the catecholamines stimulate myocardial growth.[83,92] Threshold intensities exist for the release of these hormones, and once surpassed, their rate of release increases curvilinearly with increasing exercise intensity.[93,94] Although training intensities above the threshold intensities appear obligatory to benefit from their potentiating effects, exercise intensity and duration interactions relating to hormone release and effects on physiological myocardial hypertrophy require further empirical research.

Plasma volume, erythrocyte mass and blood volume increase in response to endurance training.[81,95] A review of 18 studies by Sawka et al.[96] highlighted that plasma volume expansion plateaus after approximately 15 days of training and total erythrocyte mass after approximately 30 days. Consequently, under normal physiological conditions, significant changes in blood volume occur only in poorly conditioned individuals, with little change in the already well trained.[97,98] Even if a particular training strategy could increase blood volume in well trained runners, it is unlikely to enhance SV_{max} to any significant extent. The SV_{max} of nine elite cyclists increased very little in response to an experimental 547mL increase in blood volume,[88] probably because well trained endurance athletes are at or near their diastolic reserve capacity.[88,99]

4.2 Maximal Arterial-Mixed Venous Oxygen Difference

Skeletal muscle capillarisation increases in response to endurance training[100,101] and has been considered a major physiological adaptation in the enhancement of $\dot{V}O_{2max}$.[76] The main stimulus for inducing capillarisation is increased shear stress and capillary pressure resulting from a critical increase in blood flow velocity.[102] Since cardiac output and blood flow increase with increasing exercise intensity up to $\dot{V}O_{2max}$, there should be an intensity-dependent increase in capillary shear stress and stimulus for capillarisation up to $\dot{V}O_{2max}$.

Skeletal muscle myoglobin enhances the movement of oxygen from the sarcolemma to the mitochondrial surface[103] and in rats has been shown to increase with training.[104-108] However, more recent studies involving human subjects have found no significant changes in myoglobin or myoglobin messenger ribonucleic acid concentration in response to endurance training performed in normoxic conditions.[109-113] The training intensities used in the human studies were 60–75% $\dot{V}O_{2max}$. In contrast, except for the study conducted by Lawrie,[107] the studies employing rats included running speeds of up to 42–44 m/min, speeds that have been found to elicit $\dot{V}O_{2max}$ in rats.[114,115] Training studies conducted in hypoxic conditions suggest that intracellular hypoxia is a stimulus for increased myoglobin gene expression.[112,113] Since oxymyoglobin saturation has been found to decrease in relation to increasing exercise intensity,[116] high percentages of $\dot{V}O_{2max}$ may be required to increase myoglobin concentration. However, the non-significant differences in myoglobin concentrations in the vastus lateralis muscles of endurance-trained and untrained males reported by Jansson et al.[117] suggests that increases in myoglobin may not be elicited by training in normoxic conditions in humans. Studies involving exposing well trained runners to high-intensity training are therefore required.

Enhanced skeletal muscle fibre oxidative capacity associated with training[118] does not appear to play a major role in $\dot{V}O_{2max}$ enhancement in well trained individuals,[81] because the oxidative capacity of skeletal muscle is greater than the cardiovascular system's oxygen delivery capacity during whole body exercise.[119] However, during maximal exercise, blood perfusing areas of muscle that contain

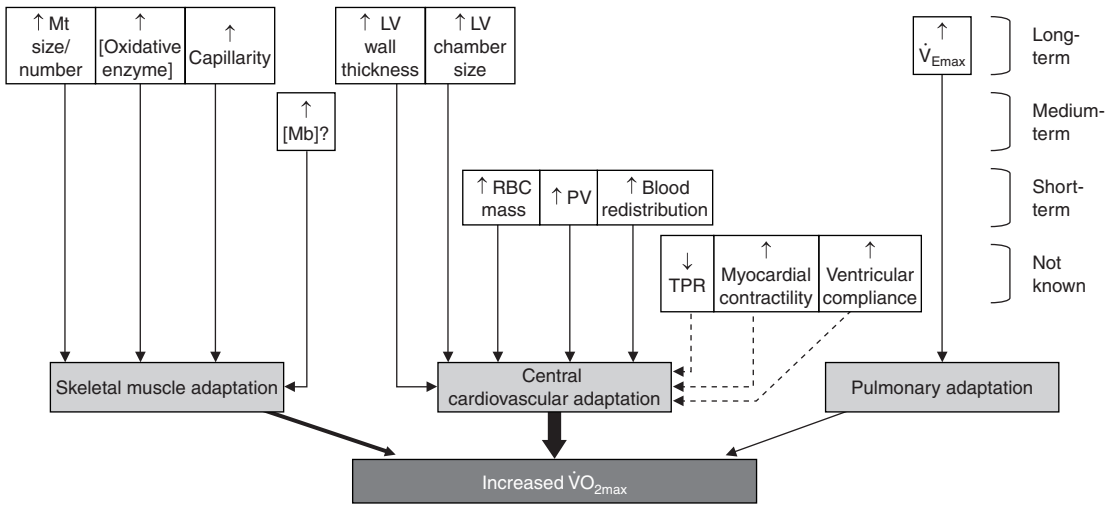

Fig. 1. Training-induced physiological adaptations associated with the enhancement of maximal oxygen uptake ($\dot{V}O_{2max}$). Short-, medium- and long-term adaptations typically have a maximum period of adaptability of days, months and years, respectively. The arrows with broken lines indicate the time course of those adaptations has presently not been elucidated. The width of the three shaded arrows at the bottom of the figure broadly represent the total contribution of those adaptations in the long-term enhancement of $\dot{V}O_{2max}$. Maximum period of adaptability for myoglobin concentration based on rat studies. **LV** = left ventricular; [**Mb**] = myoglobin concentration; **Mt** = mitochondrial; [**Oxidative enzyme**] = oxidative enzyme concentration; **PV** = plasma volume; **RBC** = red blood cell; **TPR** = total peripheral resistance; $\dot{V}_{Emax}$ = maximal minute ventilation; ↑ indicates increase; ↓ indicates decrease; **?** indicates presently unknown if training-induced increases occur in humans under normoxic conditions.

type II fibres is likely to possess a relatively high oxygen content as it leaves the venule end of the capillary, due to the relatively low oxidative capacity of these fibres.[120] An increase in the oxidative capacity of type II fibres should therefore increase their oxygen uptake for the same blood perfusion and partial pressure of oxygen (pO_2), and widen the maximal $a-\bar{v}O_2$ difference. However, because of the high threshold motor neurons associated with these fibres,[121] exercise intensities of at least 90–100% $\dot{V}O_{2max}$ are required to substantially recruit them and elicit adaptive responses.[122]

Another method of widening the maximal $a-\bar{v}O_2$ difference is by increasing the oxygen content of the blood via increased erythrocyte and haemoglobin mass.[123] Experimentally induced increases in erythrocyte and haemoglobin mass have been shown to enhance $\dot{V}O_{2max}$ significantly in the absence of any change in $\dot{Q}_{max}$.[124,125] As highlighted earlier, however, training-induced increases in total erythrocyte mass plateau after about 30 days,[96] and is therefore probably not involved in the longitudinal enhancement of $\dot{V}O_{2max}$ in well trained distance runners.

Physiological adaptations associated with the enhancement of $\dot{V}O_{2max}$ and their time course for adaptation is shown in figure 1. The physiological significance of these adaptations and the influence of training intensity are summarised in table IV.

5. Recommendations for Enhancing the $\dot{V}O_{2max}$ of Distance Runners

Presently, there appears to be little evidence as to which training intensity is most effective in enhancing the $\dot{V}O_{2max}$ of well trained distance runners. The following recommendations should therefore be considered 'best practice' based on the limited knowledge that currently exists.

5.1 Physiological Rationale

Physiological structures or processes that demonstrate substantial long-term plasticity (figure 1) should be the target of training-induced adaptations for the longitudinal enhancement of a distance run-

Table IV. Summary of physiological variables associated with the endurance training-induced enhancement of $\dot{V}O_{2max}$, their physiological significance and the potential influence of training intensity on their adaptive responses

Physiological variable	Physiological significance of enhancement	Potential influence of training intensity
Mitochondria size/volume + aerobic enzymes[118]	Increased oxidative capacity of fast twitch fibres may widen the maximal $a-\bar{v}O_2$ difference	Fast twitch fibres are substantially recruited only at exercise intensities at or above 90–100% $\dot{V}O_{2max}$[122] and therefore 90–100% $\dot{V}O_{2max}$ should set the lower training limit to enhance their oxidative capacity
Skeletal muscle capillarity[100,101]	Increased oxygen diffusion and uptake for any given arterial pO_2 and blood flow. Increased maximal $a-\bar{v}O_2$ difference	Capillarisation is stimulated by increased shear stress and capillary blood pressure from increased blood flow velocities.[102] During whole-body endurance exercise such as running, blood flow velocities through the active musculature increase with increasing exercise intensity
Myoglobin[104,105]	Facilitation of oxygen diffusion from the sarcolemma to the mitochondria. Increased oxygen uptake for any given pO_2 and blood flow. Increased maximal $a-\bar{v}O_2$ difference	Under normoxic conditions probably only enhanced in response to high relative exercise intensities in humans
LV wall thicknesses[126-128]	Increased force of LV contraction, increased ejection fraction and SV_{max}. Maintains normal wall stress during hypertrophy	Systolic and mean arterial blood pressures increase with increasing exercise intensity up to $\dot{V}O_{2max}$[87,90,91] causing an exercise intensity-dependent myocardial pressure overload and stimulus for myocardial adaptation
LV chamber size[126-129]	Increased end-diastolic volume and SV_{max}	Stroke volume increases with increasing exercise intensity up to $\dot{V}O_{2max}$[87-89] causing an exercise intensity-dependent myocardial volume overload and stimulus for myocardial adaptation
Erythrocyte mass[81,95]	Increased blood volume, venous return, end-diastolic volume and SV_{max}.[123,130-132] Increased arterial oxygen content and maximal $a-\bar{v}O_2$ difference	Reduced blood flow to the kidney is positively related to exercise intensity.[133] Reduced blood flow decreases oxygen delivery to the kidney, providing the stimulus for erythropoietin production and erythropoiesis[134]
Plasma volume[95,98]	Increased blood volume, venous return, end-diastolic volume and SV_{max}[123,130-132]	The release of hormones responsible for the enhancement of plasma volume is exercise intensity dependent[94,135]
More efficient blood redistribution[24,136]	Decreased vascular conductance in tissues operating at low rates of respiration, such as the kidneys and splanchnic regions, and increased vascular conductance in heavily respiring active skeletal muscle.[137] Increased maximal $a-\bar{v}O_2$. Early adaptations in blood redistribution are somewhat reversed in the well trained state,[133] however, and do not appear trainable in well trained runners	Presently unknown
Total peripheral resistance[82]	Reduced afterload and increased ejection fraction and SV_{max}	Presently unknown
Myocardial contractility[24,138]	Increased force of LV contraction. Increased ejection fraction and SV_{max}	Presently unknown
Ventricular compliance[87]	Attenuation of myocardial stiffness as ventricular wall thicknesses increase. Increased end-diastolic reserve, end-diastolic volume and SV_{max}	Presently unknown
Maximal minute ventilation[136,138-140]	Increased alveolar pO_2 and oxygen diffusion pressure gradients across the alveoli-pulmonary capillary interface during maximal exercise	Presently unknown

$a-\bar{v}O_2$ = arterial-mixed venous oxygen difference; **LV** = left ventricular; **pO_2** = partial pressure of oxygen; **SV_{max}** = maximal stroke volume.

ner's $\dot{V}O_{2max}$. Myocardial morphological adaptations that increase SV_{max} would appear most important. Other important adaptations include increased capillarisation of skeletal muscle and increased oxidative capacity of type II skeletal muscle fibres. The strength of stimuli that elicit adaptation is exercise intensity dependent up to $\dot{V}O_{2max}$, indicating that training at or near $\dot{V}O_{2max}$ may be the most effective intensity to enhance $\dot{V}O_{2max}$ in well trained distance runners. However, Moffatt et al.[141] suggested that as $\dot{V}O_{2max}$ is approached, the differentiation between stimuli decreases. Research is therefore needed to understand the chronic adaptive effects elicited by different training intensities in the range of 90–100% $\dot{V}O_{2max}$.

5.2 Moderately Trained Distance Runners

It could be argued that lower training intensities than those associated with the attainment of $\dot{V}O_{2max}$ would induce greater adaptation because the physiological stress could be imposed for longer periods. This may be true for moderately trained runners, and the prolonged moderate stress elicited by traditional continuous intensity exercise used to enhance $\dot{V}O_{2max}$, such as 65–80% $\dot{V}O_{2max}$, will probably be effective in enhancing the $\dot{V}O_{2max}$ of these runners. Runners who train <60–80 km/week may enhance $\dot{V}O_{2max}$ with increased submaximal training loads, although further research is required to confirm typical limits beyond which further enhancement does not occur, such as the 120km suggested by Sjödin and Svedenhag.[22] Training at intensities that elicit $\dot{V}O_{2max}$ may be beneficial to moderately trained runners; however, the total volume of training at these intensities should be low and slowly increased over many years.

5.3 Well Trained Distance Runners

Until further information is available to suggest otherwise, in order to target the enhancement of $\dot{V}O_{2max}$ we recommend well trained distance runners should progressively increase training intensity to those that elicit $\dot{V}O_{2max}$. The volume of this type of training should then be subsequently increased over many years of a periodised training plan. The reader is directed to references[40,41,61-64] for examples of appropriate interval training protocol design.

5.4 Elite Distance Runners

High training volumes are typical of most contemporary elite distance runners,[142] and increased submaximal training loads are unlikely to appreciably increase training stress.[22,29] Elite runners who have employed submaximal training intensities exclusively in their training programmes may demonstrate considerable increases in $\dot{V}O_{2max}$ if exposed to progressively higher volumes of high-intensity training during each additional year of a periodised annual training programme. This premise has been supported by studies involving elite cross-country skiers.[143] Elite runners who habitually employ running velocities that elicit $\dot{V}O_{2max}$ in their training programmes are probably close to their trainable limit for $\dot{V}O_{2max}$ enhancement, and any improvement is likely to exert only small effects on performance. However, small improvements in the performance of elite runners can be highly significant in terms of competitive success,[32,144] and progressive increases in the volume of training at or near $\dot{V}O_{2max}$ may prove beneficial. Runners and their coaches should use a cost-benefit approach to decide whether any enhancement in $\dot{V}O_{2max}$ is beneficial after taking into account possible negative stress associated with increased volumes of high-intensity training. Physiological laboratory testing would greatly facilitate this process.

5.5 Periodisation and Recovery

If training stress is too severe, the capacity to adapt will be surpassed, resulting in maladaptation and decreased functional capacity.[145] High-intensity training that is performed too often, or without appropriate preparatory training, may predispose runners to under-recovery[37] and immunosuppression.[146] Billat et al.[37] found that well trained runners who performed three high-intensity training sessions per week for 4 weeks developed signs indicative of overtraining. One or two sessions per week of high-intensity training with at least 48 hours between sessions is recommended. If higher fre-

quencies are employed, such as during shock microcycles,[147] a period of low training loads should follow. A shock microcycle before an important race, for example, would necessitate a long tapering period to reduce accumulated fatigue associated with such training.[148] Short bouts of training at or near $\dot{V}O_{2max}$ may also be effective in maintaining $\dot{V}O_{2max}$ during low training loads such as during tapering.[149]

Preparatory training should include several months of base training at intensities of 65–70% $\dot{V}O_{2max}$ followed by transition training at around 85% $\dot{V}O_{2max}$.[2] During the subsequent high-intensity training phase, when $\dot{V}O_{2max}$ is targeted, it may be effective to increase training intensity progressively from vΔ50 (the velocity midway between the lactate threshold velocity and $v\dot{V}O_{2max}$) to supra-$v\dot{V}O_{2max}$ velocities. This would maintain maximal stress on the oxygen transport system while simultaneously eliciting other physiological adaptations associated with supra-$\dot{V}O_{2max}$ training intensities.[20]

5.6 Event-Specific and Other Considerations

Adaptive responses to training protocols designed to enhance a particular physiological performance determinant are not exclusive to that determinant. For example, supra-$\dot{V}O_{2max}$ interval protocols maximally stress aerobic and anaerobic metabolism[4] and have been shown to significantly enhance $\dot{V}O_{2max}$ and anaerobic capacity simultaneously.[150] The training protocol chosen to target $\dot{V}O_{2max}$ enhancement should therefore be influenced by the relative importance of other physiological performance determinants of the event. Supra-$v\dot{V}O_{2max}$ interval protocols have been recommended for middle-distance runners,[58] and work intervals ran at 105–140% $v\dot{V}O_{2max}$ are probably appropriate. Runners who compete in longer races where anaerobic capacity is not an important determinant of performance may derive more benefit from intervals ran at vΔ50–105% $v\dot{V}O_{2max}$, particularly as this intensity range may be necessary to enhance the lactate threshold velocity in well trained runners.[151] Physiological performance profiling of a runner that indicates a particular weakness may also dictate which training protocol would be most beneficial. The principle of individuality as it pertains to the training response,[5] and the runner's age and training history, should also be considered.

Impact forces between the runner and the running surface increase in relation to running speed,[152] and training at relatively high running speeds could therefore expose the runner to a greater risk of musculoskeletal injury. However, it could be theorised that the addition of high-intensity training to a training programme may result in no increased risk, or even reduce the risk, of injury by allowing a reduction in training volume. A review by Hreljac[152] highlighted that the methodological limitations of existing studies do not allow the identification of any relationship between training intensity and injury risk in runners. Regardless, we recommend that the volume of high-intensity interval running is increased gradually to allow the musculoskeletal system time to adapt, while at the same time monitoring for signs of adverse training responses (e.g. musculoskeletal pain after a training session). Runners with a relatively short training history may have an increased susceptibility to injury,[153] and these runners should be especially cautious if increasing the volume of high-intensity training. Injured runners should probably temporarily avoid high-intensity training.[154]

6. Conclusion

Effective training methods to enhance the $\dot{V}O_{2max}$ of well trained distance runners have not been clearly defined since its conception in 1923.[7] Physiological responses to increasing exercise intensity indicate that training at or near $\dot{V}O_{2max}$ may be the optimal stimulus to enhance the $\dot{V}O_{2max}$ of well trained distance runners. The physiological rationale for this premise, however, was based on studies that mostly investigated the physiological responses to incremental exercise. Further research is therefore required to identify the stress that is imposed on the physiological determinants of $\dot{V}O_{2max}$ during interval training protocols designed to elicit very high percentages of $\dot{V}O_{2max}$.

Well trained distance runners have been reported to reach a plateau in $\dot{V}O_{2max}$ enhancement;[24-28] however, several studies[35,38,40,41] have demonstrated that the $\dot{V}O_{2max}$ of well trained runners can be enhanced when training protocols known to elicit 95–100% $\dot{V}O_{2max}$ are included in their training programmes. This suggests that training at or near $\dot{V}O_{2max}$ may be effective or even necessary for well trained distance runners to enhance $\dot{V}O_{2max}$. Thus far, however, there have been no well controlled training studies that support this premise. The efficacy of optimised protocols for enhancing $\dot{V}O_{2max}$ need to be established with well controlled studies in which they are compared with protocols involving other training intensities typically used by distance runners to enhance $\dot{V}O_{2max}$. Clearly, large gaps exist in our current knowledge of the most effective training intensities to enhance $\dot{V}O_{2max}$, and recommendations made in this article are based on this limited knowledge and therefore should not be regarded as definitive.

The $\dot{V}O_{2max}$ is one of many physiological determinants of distance running performance, and the most effective training protocols for the enhancement of all other determinants also need to be characterised. Synergistic and interference effects between optimised training protocols designed to target specific physiological performance determinants and the influence of individuality then need to be established before sports scientists can make recommendations to runners and running coaches, with a high level of confidence, on components of an effective training programme.

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Correspondence and offprints: Prof. *Lars R. McNaughton*, Department of Sport, Health and Exercise Science, University of Hull, Cottingham Road, Hull, HU6 7RX, England. E-mail: l.mcnaughton@hull.ac.uk