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Interval Training for Performance: A Scientific and Empirical Practice

Special Recommendations for Middle- and Long-Distance Running. Part II: Anaerobic Interval Training

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Abstract

Studies of anaerobic interval training can be divided into 2 categories. The first category (the older studies) examined interval training at a fixed work-rate. They measured the time limit or the number of repetitions the individual was able to sustain for different pause durations. The intensities used in these studies were not maximal but were at about 130 to 160% of maximal oxygen uptake ($\dot{V}O_{2max}$). Moreover, they used work periods of 10 to 15 seconds interrupted by short rest intervals (15 to 40 seconds). The second category (the more recent studies) asked the participants to repeat maximal bouts with different pause durations (30 seconds to 4 to 5 minutes). These studies examined the changes in maximal dynamic power during successive exercise periods and characterised the associated metabolic changes in muscle.

Using short-interval training, it seems to be very difficult to elicit exclusively anaerobic metabolism. However, these studies have clearly demonstrated that the contribution of glycogenolysis to the total energy demand was considerably less than that if work of a similar intensity was performed continuously. However, the latter studies used exercise intensities that cannot be described as maximal. This is the main characteristic of the second category of interval training performed above the minimal velocity associated with $\dot{V}O_{2max}$ determined in an incremental test ($v\dot{V}O_{2max}$).

Many studies on the long term physiological effect of supramaximal intermittent exercise have demonstrated an improvement in $\dot{V}O_{2max}$ or running economy.

1. Anaerobic Interval Training

1.1 Acute Physiological Responses

Studies of anaerobic interval training can be divided into 2 categories. The first category (the older studies) examined interval training at a fixed work-rate.[1] They measured the time limit or the number of repetitions the athlete was able to sustain for different pause duration. The intensities used in these studies were not maximal but were at about 130 to 160% of maximal oxygen uptake ($\dot{V}O_{2max}$). Moreover, they used work periods of 10 to 15 seconds interrupted by short rest intervals (15 to 40 seconds).

The second category (the more recent studies) asked the participants to repeat maximal bouts with different pause durations (30 seconds to 4 to 5 minutes). These studies examined the changes in maximal dynamic power during successive exercise periods and characterised the associated metabolic changes in muscle.[2]

However, the purpose of both of these approaches was to understand the involvement of the different types of metabolism (alactic, lactic anaerobic and aerobic) by comparing the influence of pause duration [time for creatine phosphate (CP) resynthesis] on the decrease in the time limit and total work performed and the decrease in the velocity or power output. These decreases in total work performed or of the work-rate were correlated with the substrates (glycogen, CP) and the reactants of the different metabolic pathways by biopsy techniques, blood analysis and gas exchange measurements.

1.1.1 Fixed Work-Rate Studies

In the first category, Margaria et al.[1] demonstrated that for supramaximal intermittent exercise [about 160% of the minimal velocity associated with $\dot{V}O_{2max}$ determined in an incremental test ($v\dot{V}O_{2max}$)], the total time leading to exhaustion depended on the pause duration. Moreover, when the pause was 0 (uninterrupted running), 10, 20 and 30 seconds, the total times run at the supramaximal velocity were 32, 100 and 200 seconds and indefinite, respectively. When the pause was 10 seconds, the total running time could be increased about 3 times and when the rest period was 20 seconds, 6 times. Doubling the period of rest allowed the athlete to work twice as long. Indeed, blood lactate accumulation was equal to 11 and 7.5 mmol/L and stayed at the steady state of 2 mmol/L, respectively, for the 10-, 20- and 30-second pauses. This study demonstrates that the pause duration was the determinant for the utilisation of anaerobic and aerobic pathways. By plotting the blood lactate accumulation as a function of the length of the rest period, Margaria et al.[1] showed that the minimal time of rest at which no lactic acid accumulation took place was about 25 seconds. This duration corresponds to the half-reaction time for the alactic oxygen deficit payment (i.e. CP resynthesis). These authors measured oxygen uptake ($\dot{V}O_2$) breath by breath in a runner performing 10 bouts of exercise with a 20-second pause and they showed that there was a very fast increase in $\dot{V}O_2$ at the beginning of the exercise and there was no appreciable difference during the first pause compared with the first running period. At steady state, from the fifth repetition, $\dot{V}O_2$ was slightly lower during the pause than during running (e.g. 3.5 *vs* 4 L/min for 1 participant). The first run took place almost exclusively (90%) at the expense of phosphagen. During the following 25 seconds of rest, the alactic pool was restored to about 45% by the oxygen debt payment (fast post oxygen component).

During the second run, energy was provided more from the oxidative mechanism: 25 versus 10% of the total energy requirement in the first and second bouts, respectively. This is too high to be sustained by the alactic anaerobic metabolism, since the phosphagen pool is only equal to 45% of the initial resting storage value. Consequently, anaerobic glycolysis was involved and blood lactate accumulated. This is why the 30 seconds of pause, which is sufficiently long to allow the phosphagen pool to be resynthesised, also allows the athlete to run for a very long time with a steady-state blood lactate below 2.5 mmol/L. With pauses of only 10 seconds, $\dot{V}O_{2max}$ is reached earlier and blood lactate accumulates. This kind of interval training performed at supramaximal intensity can elicit a completely different metabolism, depending upon the length of the pause.

If the oxygen deficit during the period of supramaximal work is higher than the alactic anaerobic oxygen equivalent (0.5 L), the interval training will involve lactic anaerobic processes. For instance, a female runner who has a $v\dot{V}O_{2max}$ equal to 16 km/h (4.44 m/sec) will run $10 \times 4.44 \times 1.6 = 71.1$m during a 10-second supramaximal run at 160% of $v\dot{V}O_{2max}$. If she weighs 50kg and has a gross oxygen cost of running equal to 0.210 ml/kg/m, she will need to consume a volume of oxygen equal to $71.1\text{m} \times 0.210 = 15$ml of oxygen/kg, i.e. 750ml. This is equal to 150% of the oxygen equivalent of anaerobic metabolism. We can therefore calculate

that for the 10-second exercise bouts, the velocity should be proportionately less, i.e. close to $v\dot{V}O_{2max}$. However, the pause should be at least 25 seconds to be sure that the CP consumed during the work interval is restored. As demonstrated by Margaria et al.,[1] in these conditions interval training can be performed for a very long time with no blood lactate accumulation. Hence, the work : pause ratio is equal to 1 : 3 and $\dot{V}O_2$ is submaximal (82% of $\dot{V}O_{2max}$).[3]

Using short–short interval training, it seems to be very difficult to elicit exclusively anaerobic metabolism. However, these studies have clearly demonstrated that the contribution of glycogenolysis to the total energy demand was considerably less than that if work of a similar intensity was performed continuously. However, the latter studies used exercise intensities that cannot be described as maximal. This is the main characteristic of the second category of interval training performed above $v\dot{V}O_{2max}$.

More recently, Tabata et al.[4] have compared the metabolic profiles of 2 different types of high intensity interval training at a fixed percentage of $\dot{V}O_{2max}$ equivalent. Participants cycled 6 bouts of 20 seconds at 170% $\dot{V}O_{2max}$ with a 10-second rest between each bout. The second interval training involved 4 bouts of 30 seconds at 200% of $\dot{V}O_{2max}$ with a 2-minute rest between each bout. The first interval training protocol elicited both the maximal accumulated oxygen deficit and the maximal oxygen consumption. Therefore, the authors concluded that this supramaximal interval training might tax both the anaerobic and aerobic energy releasing systems at close to their maximal capacity.

1.1.2 Fixed Intensity Studies

It should be noted that we cannot really call these exercises 'interval training', but rather 'repeated maximal sprints'. Maximal dynamic exercise has been recently (for 10 years) used by physiologists to study the regulation of metabolic pathways and to shed light on the aetiology of fatigue (inability to sustain a given power output) during high intensity exercise. In this type of study, Bogdanis et al.[5] demonstrated that in a second 30 seconds of all-out cycling exercise performed 4 minutes after the first bout, CP, which was at 17% of the resting value after the first all-out cycling exercise, was resynthesised to 79% of the resting value even though the pH remained at a low level (pH 6.8). Despite the 41% reduction in anaerobic energy, the total work carried out during the second 30-second sprint was reduced by only 18%. Aerobic metabolism provided a significant part (49%) of the energy and the rate of CP synthesis determined the work-rate performed in the first 10 seconds of the second 30-second all-out exercise.

In another major study on intermittent maximal exercise training, 10 × 6-second maximal sprints with 30 seconds of recovery between each sprint were performed.[6] Needle biopsy samples were taken from the vastus lateralis muscle before and after the first sprint and 10 seconds before and immediately after the tenth sprint. In this study, the high mean power output was more than 3 times the minimal power output which elicits $\dot{V}O_{2max}$ determined in an incremental test ($p\dot{V}O_{2max}$). The peak power (the maximum power output in 1 second) was 5 times greater than $p\dot{V}O_{2max}$. Mean power outputs significantly decreased from the fourth repetition and the peak power decreased from the fifth. The last repetition was reduced to only 73% of power generated during the first sprint. The energy required to sustain the high mean power output that was generated over the first 6-second sprint was provided by an equal contribution from CP degradation (–57% of rest concentration) and anaerobic glycolysis (muscle lactate had increased to 28.6 mmol/kg dry weight). In the tenth sprint, muscle lactate concentration did not increase. The authors suggested that during the last sprint, the power output (which was 73% of the first repetition) was maintained by energy that was derived mainly from CP degradation and an increase in aerobic metabolism.

For training purposes, because of the drop in the power output and because of the high muscle lactate accumulation, this configuration of intermittent maximal sprint is not used. The purpose of sprint training is to increase the maximal velocity

for 6 to 10 seconds (60 to 100m in running); in order to achieve this, the intermittent maximal run is separated by 4-minute pauses to allow the CP to be restored so that each repetition can be run fast. To increase the glycolysis pathways, which account for 40 to 50% of the energy necessary to cover 100m, the intermittent training consists of a series of 100, 120 and 150m runs at 88 to 90% of the best performance with a passive rest of 5 to 6 minutes between each bout. However, in this protocol, it has been demonstrated that during passive rest almost all the CP was resynthesised after 4 minutes (from 19.8 to 36.9 mmol/kg dry muscle, instead of 39 ± 3.2 mmol/kg).[7] The half time of CP resynthesis is 170 seconds.[8]

However, these studies have a direct application for sprint training. Indeed, Balsöm et al.[2] have compared the physiological responses to maximal intensity intermittent running for 15m (in 2.6 seconds), 30m (in 4.5 seconds) and 45m (in 5.6 seconds). These sprints were performed every 30 seconds but covered the same distance (work) of 600m (40 × 15m, 20 × 30m, 15 × 40m). The time of the last sprint of 15m was not significantly different from that of the first (2.63 ± 0.04 *vs* 2.62 ± 0.02 seconds). This was not the case for the 30m and 40m sprint times, which increased significantly. Velocity at 40m decreased after the third sprint, associated with a net loss of the adenine nucleotide pool. Balsöm et al.[9] also compared the effect of recovery duration (120, 60 and 30 seconds) on the 40m maximal intensity intermittent exercise. The performance (time) during the first 15m (acceleration phase) was only affected by the shortest recovery (30 seconds). Blood lactate reached 17 mmol/L for this short recovery compared with the value registered using 60 and 120 seconds recovery (12.1 ± 1.3 and 13.9 ± 1.2 mmol/L, respectively). Although these work bouts could be classified primarily as anaerobic exercise, oxygen uptake measured during rest periods (Douglas bag method) increased to 52, 57 and 66% of $\dot{V}O_{2max}$. However, to improve performance in competitions lasting 1 minute (1km in track cycling or 100m in swimming and 400m in running), generally performed at 150% of $\dot{V}O_{2max}$, it is important to practise aerobic interval training, since the aerobic metabolism contribution to the total energy is about 30%.[2]

The increase in recovery time allowed the runners to maintain their performance during more trials; however, the increment of recovery duration had no effect on the total adenine nucleotide pool estimated by plasma hypoxanthine and uric acid concentrations. When the rate of ATP hydrolysis exceeds the rate at which it can be resynthesised, AMP is eliminated via deamination of AMP to IMP and subsequent oxidation to hypoxanthine and uric acid.

It is well known that both anaerobic pathways – lactic (glycolysis) and alactic (CP degradation) – are activated instantaneously at the onset of maximal activity.[10] However, the ability to repeat maximal sprints depends on the duration of recovery, which does not have the same effect on the 2 anaerobic pathways. The resynthesis of CP depends on the endurance level of the participant.[5,11] The percentage of CP resynthesis and the percentage restoration of the mean power output and pedalling speed measured on the first 10 seconds in the second 30-second all-out exercise were highly correlated (r = 0.84 and 0.91, respectively). Indeed, the time course of CP resynthesis after a 30-second sprint was found to be parallel with the time course of the peak power output (PPO) restoration.[12]

As described above, glycogenolysis is largely elicited in these supramaximal intermittent exercises. Intensity of work interval training is well above the crossover point: i.e. the power output at which energy from carbohydrate-derived fuels predominates over energy from lipids.[13] Jenkins et al.[14] have demonstrated that a high (83% of the total energy intake) and moderate (58% of the total energy intake) carbohydrate diet for 3 days preceding supramaximal intermittent exercises would allow performance to be maintained during 5 all-out cycling bouts. Each of these 5 exercise periods, at about 125% of $p\dot{V}O_{2max}$, were separated by 5 minutes of passive recovery.

1.2 Long Term Physiological Effects

Many studies on the long term physiological effect of supramaximal intermittent exercises have demonstrated an improvement in $\dot{V}O_{2max}$. In the 1970s, Fox and his team reported that this kind of interval training was efficient in inducing an improvement in $\dot{V}O_{2max}$ (+15%) in men[15] and in women.[16] Indeed, Lesmes et al.[16] compared the effects of 2 types of supramaximal interval training over an 8-week period on $\dot{V}O_{2max}$ (2 or 4 days/week) in women with a $v\dot{V}O_{2max}$ equal to about 12.9 km/h, using high intensity, short distance (50, 101, 201m) at 170% of $v\dot{V}O_{2max}$ (22 km/h) or high intensity, long distance (604, 805, 1208m) at 130% of $v\dot{V}O_{2max}$ (15 km/h).

For both types of interval training, the relief intervals consisted of walking 2 to 3 times the duration of the work intervals. The improvement was the same for both short and long supramaximal interval training and for both frequencies (2 or 4 training days/week). The authors concluded that the change in aerobic power and submaximal heart rate for females was independent of frequency, distance and intensity. In contrast, for men, it has been shown that training intensity, rather than frequency or distance, was the most important factor to improve $\dot{V}O_{2max}$.[17] The decrease in submaximal heart rate during a run of 5 minutes at 50% of $v\dot{V}O_{2max}$ using high intensity interval training was due to a decrease in sympathetic drive and probably due to the increase of stroke volume. The fact that the response was independent of intensity and frequencies of interval training could mean that the maximal responsiveness to this programme was reached for the women, who started with a $\dot{V}O_{2max}$ of only 40 ml/min/kg.

As demonstrated in rats,[18,19] any kind of training programme would have the same effect on $\dot{V}O_{2max}$ (+15%) and on $p\dot{V}O_{2max}$. However, the problem with such longitudinal studies is the low degree of fitness of the participants and the choice of so-called 'supramaximal intermittent exercises', which are often less intensive than imagined by the authors. Indeed, before drawing conclusions on the inefficiency of high intensity intermittent training in improving anaerobic capacity, it is important to check that the input is well above the power output associated with $\dot{V}O_{2max}$ ($p\dot{V}O_{2max}$). For instance, no increase was found in either the level of anaerobic metabolic enzymes or the percentage of type IIb fibres due to the effect of training (only an increase in the absolute number).[20-22] In fact, the intensity of intermittent exercise (10- or 15-fold × 15 to 30 seconds) was set at 60% of the absolute maximal velocity, which corresponds to 110 to 120% of $p\dot{V}O_{2max}$. The intensity of the longer high intensity intermittent training (4- or 5-fold × 60 to 90 seconds) was set at 70% of the maximal velocity sustained for 90 seconds. All these intensities correspond, effectively, to 90 to 95% of $v\dot{V}O_{2max}$, knowing that an exercise sustained for 1.5 minutes is at about 130% of $p\dot{V}O_{2max}$.[23] It should be noted that the intensities of these exercises were probably too low to enhance anaerobic metabolism or modify the percentage of type II fibres. The response was the same as in interval training for improving aerobic metabolism, using velocities between 90 and 120% of $v\dot{V}O_{2max}$ to give participants the time to reach and therefore to elicit $\dot{V}O_{2max}$.[23]

In contrast, 1 study[24] has used true supramaximal exercise: 2 series of 4 repetitions of 200m at 90% of the participants' maximum velocity over 200m (which gave 29 seconds) with a recovery of 2 minutes (3 to 4 times per week) for 5 weeks. At the end of the first series the blood lactate level was 14.5 mmol/L; after a rest of 10 minutes between the 2 series, blood lactate was still 13 mmol/L, and at the end of the second series of 200m blood lactate was 17.6 mmol/L. This type of interval training clearly elicited anaerobic glycolysis. Indeed, this high intensity interval training with a 1 : 4 work : rest ratio resulted in an increase in the activity of key enzymes involved in glycogenolysis and anaerobic glycolysis in skeletal muscle. The increase in succinate dehydrogenase (SDH) [+17.5%] was not statistically significant. Participants had improved their time limit in a run at 16 km/h with a 15% slope inclination (about 25 km/h equivalent on level treadmill according to Margaria et al.[25]), i.e. at about 140 to 150% for these men.

Even very short, repeated, 5-second all-out sprints followed by a recovery of 55 seconds induced an increase in the proportion of slow twitch fibres, in accordance with Simoneau's studies.[20-22] Moreover, 7 weeks of this kind of sprint training not only increased lactate production after a training session [associated with a 20% higher activity of both phosphofructokinase and lactate dehydrogenase (LDH)], but also increased the proportion of slow twitch fibres which was closely related to a concomitant decrease in fast IIb fibres.[26] However, $\dot{V}O_{2max}$ did not increase and the decrease in power output during each sprint was not reported. Notably, 55 seconds is too short to reconstitute the CP reserve. Thus, intense and repetitive exercise could induce the conversion of intermediate fibres to the oxidative type, aerobic metabolism being involved in the recovery and probably increasingly during the repetition of the sprints.[26] Maximal power output measured with the force-velocity test increased significantly after sprint training (+13%). This was similar for the average power output in an all–out test of 30 seconds (+27%) and for the maximal work-rate reached in the Wingate test. These improvements in performance were not related to a significant increase in the mean resting muscle CP concentration, but to an increase in the energy production from anaerobic glycolysis, as demonstrated by Nevill et al.[27]

Because the training exercises were performed intermittently, and because of their greater oxidative capacity, type I fibres could have been more extensively involved in the replenishment of the depleted CP stores during rest periods than fast twitch fibres. Moreover, type I fibres are more involved in the removal of the lactate accumulated during exercise periods (52.8 mmol/kg of dry mass) than type II fibres. Indeed, in order to increase alactic anaerobic metabolism, interval training acts: (i) by increasing the ability to decrease CP as rapidly as possible by sprint exercises separated by sufficient rest (at least 4 minutes) to restore the CP reserve, avoiding the involvement of anaerobic glycolysis; and (ii) by increasing the ability to replenish as quickly as possible the CP reserve. To accomplish this, it is necessary to have muscle fibres with a high oxidative capacity. However, with short pauses, as in the study by Linossier et al.[26] (55 seconds), anaerobic lactic metabolism is increasingly involved. Acidosis could impair CP production via mitochondrial creatine kinase during recovery.

Hargreaves et al.[28] studied 6 men during 4 × 30-second all-out exercise bouts. The first 3 exercise bouts were separated by 4 minutes of passive recovery; after the third bout there was a 4-minute rest period, followed by a 30-minute exercise period at 30 to 35% peak $\dot{V}O_2$. This was followed by a further 60-minute rest period before completing the fourth exercise bout. The performance was maintained in the fourth bout because CP resynthesis is increased. Indeed, peak power and total work were not significantly decreased in the fourth bout compared with the first bout and were higher than in the second and third bouts. Before the fourth bout, muscle CP concentration was above the resting level (probably because of increased mitochondrial creatine kinase activity during recovery); IMP, lactate, pH and sarcoplasmic reticulum calcium uptake were the same as before the first bout. However, ATP and glycogen were still depleted. Since, in these metabolic conditions, the performance was the same as in the first bout, the authors concluded that the maintenance in performance observed during the fourth bout does not appear to be related to a reduction in muscle glycogen.[28]

Heugas et al.[29] reported on elite 400m runners (including an Olympic champion), in the precompetitive phase, who performed training sessions with maximal all-out 30-second runs with 4 minutes of rest in between, followed by a long rest (10 minutes) before a last 30-second maximal run. The capacity to sustain exercise at about 150% of $v\dot{V}O_{2max}$ with high acidosis and the ability to run a relay race (4 × 400m) a couple of hours later is improved by using this type of intermittent high intensity training.[29]

In a recent study[30] where the purpose was to examine the effects of sprint interval training on muscle glycolytic and oxidative enzyme activity and exercise performance, it was reported that 4 high interval training sessions per week (consisting

of 4 × 30 seconds of all-out exercise spaced by a recovery of 4 minutes, or 2 minutes 30 seconds in weeks 1 and 2) enhanced PPO, total work over 30 seconds and $\dot{V}O_{2max}$. Maximal enzyme activity of hexokinase, phosphofructokinase (glycolysis enzymes), citrate synthase, SDH and malate dehydrogenase (oxidative enzymes) was enhanced after this type of training. The participants were students in kinesiology, who were physically active but were not athletes. From the seventh week of such training, they were able to perform 10 intervals with 2 minutes 30 seconds of recovery per session. This type of high interval training induced an increase in blood lactate of up to 32 mmol/L after the tenth exercise interval. The power output ranged from 210% of $\dot{V}O_{2max}$ on the first interval to 140% $\dot{V}O_{2max}$ by the ninth and tenth intervals. At such intensities, the rate of production of pyruvate may be considered to be almost maximal and one would expect major increases in the velocity of catalytic activity of the competing enzymes pyruvate dehydrogenase (PDH) and LDH. However, the increase of 7% in LDH after such training was not significantly different; PDH was not measured, but the authors speculated that the increase in PDH activity (which provokes an increased rate of pyruvate entry into the mitochondria) could be the stimulus for the upregulation of mitochondrial enzymes. This could explain the fact that 3 to 4 repetitions of 5 minutes of effective high interval training per week (hence 20 minutes per week only) can result in an increase in both glycolytic and oxidative muscle enzyme activity, maximum short term power output and $\dot{V}O_{2max}$ (+7%). The authors concluded that the increase in power output (+24% on average after 4 × 30-second bouts) may have been the result of an increase in the maximal activity of the glycolytic enzymes (+49% for phosphofructokinase) and in Na^+/K^+ pump capacity, whereas the increased mitochondrial enzyme activity (+65% for SDH) may have been a result of increased pyruvate flux rate during this intense sprint interval training.[30]

Thus, interval training using short all-out bouts of exercise elicits the glycolytic pathway and can be used to prepare long sprint (200 to 400m) and middle distance (800 to 1500m) runners whose anaerobic metabolism is a determinant for performance. However, victory in longer races such as 5000 and 10 000m depends on the ability to cover the last lap at a velocity well above $v\dot{V}O_{2max}$,[31] although there are also 5000 and 10 000m races with 1 runner 50 to 100m ahead when starting the last lap, and this runner can win at a modest last lap speed. The last 400m of a 10 000m race can be run in 52 seconds, i.e. 27.7 km/h (110% of $v\dot{V}O_{2max}$ for the best performers, who have a $v\dot{V}O_{2max}$ of 25 km/h). Also, athletes must enhance their anaerobic capacity to be able to accelerate in championship races. For this, they can use supramaximal interval training of longer duration: 1 minute at about 130% of $v\dot{V}O_{2max}$ with a rest of 5 minutes between. Eight weeks of this type of training (3 times a week) has been reported to efficiently enhance anaerobic capacity as measured as the y-intercept of the relationship between work rate and time limit.[32] The critical power, i.e. the slope of this relationship, was not modified. This critical power depends on aerobic capacity and has been shown to be related to $\dot{V}O_{2max}$ and lactate threshold.[33] This type of high intermittent training has also been used to enhance performance in high level cyclists (see section 3), in whom it is more difficult to obtain an improvement in performance or physiological modifications.

Another type of anaerobic high interval training is circuit training consisting of explosive jumping exercises. Recently, with this type of circuit training, Paavolainen et al.[34] reported an improvement of 5km time in well trained endurance athletes with no changes in their $\dot{V}O_{2max}$. This was due to improvement of the running economy (RE), i.e. the oxygen consumption at a given sublactate threshold velocity was decreased by 3% after 6 weeks of 3 × 20 to 30 minutes of circuit training per week with long interval training or exhaustive distance training, but not with short interval training.[35] This improvement of RE was related to improved neuromuscular characteristics, allowing an increase of velocity over 20m and over a distance covered in 5 jumps that were transferred into improved RE. Improved

RE following intensive training was correlated with reduced ventilatory demands (VE). This improvement in RE was related to a decrease in VE, whereas muscle fibre composition and respiratory exchange ratio, stride length and frequency during running were unaltered by training.

If long interval training and long distance running induce a decrease in RE, circuit training composed of jump exercises can yield the same result, but probably by different mechanisms through the stretch-shortening cycle.[36] In fact, it is probable that the fitness level of participants plays a prominent role in the results of the long term effect of different types of training (continuous or intermittent).

Finally, for not very well trained participants, the intensity of exercise is more important than the type of exercise (cycling and running). Flynn et al.[37] have reported that in well trained runners, 6 weeks of high intensity interval training (between 95 and 105% $p\dot{V}O_{2max}$) performed either with cross-training (cycling and running) or specific running training yielded the same performance improvement (–30 seconds over 18 minutes 15 seconds on a 5km race). However, in all these studies, conclusions are based on results obtained in low-level athletes. This underlies the fact that sports physiologists have a limited impact on the training practices of successful competitors. Training programmes are more often based on the experience of track and field coaches and athletes.[38,39]

1.3 Anaerobic Interval Training and Creatine Ingestion

In a study of the effects of creatine supplementation on cycling performance (10 × 6-second bouts at 880W and 140 rev/min, separated by passive 30-second rest periods), performance could be enhanced by creatine towards the end of each exercise bout, as demonstrated by a smaller decline in work output from a baseline throughout the 10 trials in the last 2 seconds of the 6-second exercise periods.[40] Moreover, although more work was performed in this intermittent maximal test after versus before the administration period, blood lactate accumulation decreased from 10.8 to 9.1 mmol/L. This improvement was due to a higher availability of phosphate or to an increased rate of CP resynthesis during the recovery periods. This could explain why glycolysis is less involved in interval training at maximal velocity.

High intensity intermittent training has been shown to be a very effective way of increasing the maximal oxygen uptake. Tabata et al.[41] reported that a protocol of 6 × 20 seconds at 170% of $\dot{V}O_{2max}$ for 6 weeks increased $\dot{V}O_{2max}$ by 13% and anaerobic capacity by 28%. In fact, it seems that protocols that elicit $\dot{V}O_{2max}$, even if they are performed at supra-$v\dot{V}O_{2max}$ velocities, can increase $\dot{V}O_{2max}$ and anaerobic capacity. To do this, the recovery has to be short, as in Tabata's interval training (10 seconds).[41]

2. Interval Training for the Elderly and the Young

2.1 The Elderly

Of the various methods used to train elderly people, interval training has been reported to be effective in improving aerobic capacity ($\dot{V}O_{2max}$ and lactate threshold).

In 1 study, interval training was more easily accepted (more than 70% of participants completed both test and training sessions) than continuous training (40%).[42] The interval training was performed on the track, taking as a reference the heart rate at the ventilatory threshold (HR_{th}) of 125 ± 5 beats/min, corresponding to 60% $\dot{V}O_{2max}$. The interval training consisted of 1 minute exercise at HR_{th} and 1 minute active recovery (20 beats/min below HR_{th}); 2 minutes exercise at HR_{th}, 1 minute active recovery; 4 minutes exercise at HR_{th}, 1 minute active recovery; and 10 minutes exercise at HR_{th}, 3 minutes active recovery.

From session to session, the order and duration of the exercise/recovery sequences were varied in such a way that the participants progressively increased their total exercise time from 30 to 60 minutes by the eighth week of training. This procedure can be performed on the track and needs only a

cardiofrequency meter. As for younger runners, the elderly benefit from this interval training by increasing oxidative enzyme activity,[43] $\dot{V}O_2$ at the ventilatory threshold and cardiac output, since maximal oxygen pulse increased by 19% (as $\dot{V}O_{2max}$). Moreover, the attendance at these training sessions during periods as long as 3 months (24 sessions and 12 interval training sessions) has been reported to be more than 97.3% for the 73% of participants who had been considered to have completed training and test protocols. Interval training significantly increased $\dot{V}O_{2max}$ by 20% (from 25 ± 1.2 to 31 ± 2.3 ml/min/kg) and the velocity associated with the ventilatory threshold (vVT) by 26%.

However, as for younger participants, the training effect depends on the basal level of the participants. In training camp, we often have senior runners (>60 years) who are able to run for 6 minutes at 18 km/h. They are generally very long distance runners (marathon and 100km races). For those who already have a high $\dot{V}O_{2max}$ (60 ml/min/kg at age 60 years) and maximal lactate steady-state velocity (vMLSS = 85% of $v\dot{V}O_{2max}$) and are not familiar with interval training, very short repetitions are well supported.[44] Figure 1 shows the $\dot{V}O_2$ kinetics for a 60-year-old runner (eighth in senior 100km world championship 1999) during a very short interval of 15 seconds at 90% and 15 seconds at 80% of $v\dot{V}O_{2max}$. The average velocity was at MLSS velocity (85% $v\dot{V}O_{2max}$). It should be pointed out that this very narrow amplitude training interval allows him to remain for more than 10 minutes at $\dot{V}O_{2max}$. Blood lactate accumulation was only 8 mmol/L. More research is needed to allow these seniors who are already in good shape to improve their performance, but by running fewer kilometres.

Nowadays, many runners participating in popular races over 5 to 100km are middle-aged (45 to 60 years). After several years of slow long distance training, they no longer improve their performance. Following this type of training, these long distance runners have a high endurance index, which is de-

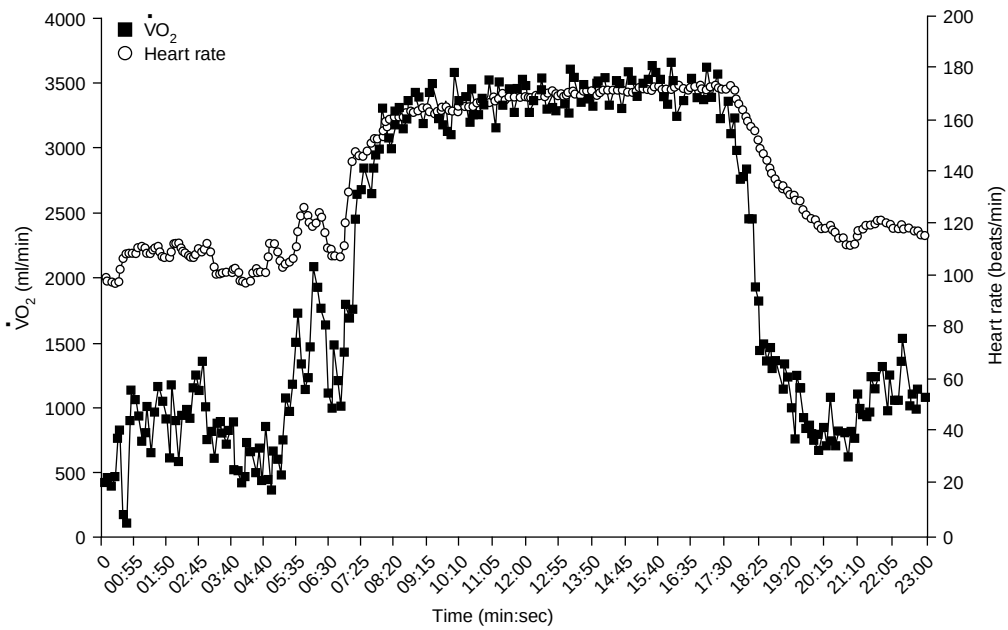

Fig. 1. Time course of oxygen uptake ($\dot{V}O_2$) and heart rate during short interval training [15 seconds at 90% of $v\dot{V}O_{2max}$ (minimal velocity associated with maximal oxygen uptake, $\dot{V}O_{2max}$) alternated with 15 seconds at 80% $v\dot{V}O_{2max}$] in a long distance runner aged 60 years. His $\dot{V}O_{2max}$ was 3350 ml/min.

fined as the ability to use a high fraction of maximal oxygen consumption ($\dot{V}O_{2max}$) for a given running duration.[45] Therefore, to improve their performance, they have to increase both $\dot{V}O_{2max}$ and the velocity associated with $\dot{V}O_{2max}$ ($v\dot{V}O_{2max}$).[46-48] For this purpose, interval training involves repeated bouts of work, each lasting from about 30 seconds at $v\dot{V}O_{2max}$ to 5 minutes at 95% of $v\dot{V}O_{2max}$.[49] Gorostiaga et al.[50] showed that interval training with repetitions of 30 seconds of work at 100% of $v\dot{V}O_{2max}$, separated by 30 seconds of rest, produced larger increases in $\dot{V}O_{2max}$ than continuous training at 50% $v\dot{V}O_{2max}$. Recently, as seen above, it has been shown that the time spent specifically at $\dot{V}O_{2max}$ was much higher in 30/30-second light-heavy exercise intervals than in a continuous severe run performed at an intermediate velocity between the lactate threshold and $v\dot{V}O_{2max}$.[51]

However, the types of interval training described above seem to be too hard for middle-aged runners who want to begin interval training. It may be possible to use shorter durations of less than 20 seconds. Christensen et al.[3] showed previously that very short intermittent runs alternating with heavy intensity repetitions of 15 seconds at 100% $v\dot{V}O_{2max}$ with complete rests of 15 seconds also allowed the runners to reach 90% $\dot{V}O_{2max}$. In this case, the intermittent exercise was continued for 30 minutes with a blood lactate level of only 4 mmol/L until the 20th minute (reaching 7 mmol/L at the end of the exercise). This low blood lactate level was obtained by the unloading of myoglobin oxygen and CP stores and by their rapid recharging during the passive recovery period. To elicit $\dot{V}O_2$ at its maximum with this 'short–short' interval training, active pause may be preferable to passive recovery[51] and a small range of velocities between the high and low velocity bouts has to be used. This difference in velocity is called the 'amplitude' of the Interval training.[52,53] The amplitude describes the degree to which the work intensity in the different periods of the exercise varies from the average velocity.[51] For instance, for an interval training using high and low velocity bouts of 100 and 50% of $v\dot{V}O_{2max}$, the average velocity is 75% $v\dot{V}O_{2max}$, and the amplitude of the interval training is equal to $[(100 - 75)/75] \times 100 = 33\%$.

For elderly runners, the average velocity of interval training has been chosen to correspond to the critical velocity, i.e. the vertical asymptote of the velocity-time relationship. The critical velocity is known to be sustained for 30 minutes[54] and to be the velocity above which runners reach $\dot{V}O_{2max}$ with exercising time.[55] Moreover, in a group of low level marathon runners (3 hours 51 minutes ± 27 minutes), the critical velocity was more closely correlated with the marathon performance than $v\dot{V}O_{2max}$.[56]

Therefore, the purpose of this study[56] was to compare run time at $\dot{V}O_{2max}$ using a short–short interval training protocol with the same average velocity (the critical velocity) but with different amplitudes. We hypothesised that for middle-aged runners, a very short interval training (15 seconds of hard alternated with 15 seconds of easier run) with a small amplitude would allow them to run for a longer distance at a higher velocity and for a longer time at $\dot{V}O_{2max}$. In our group of middle-aged runners, having an average critical velocity of 85.6 ± 1.2% $v\dot{V}O_{2max}$, this study compared the following types of interval training:

- 90 to 80% $v\dot{V}O_{2max}$ (for hard bouts and active recovery periods, respectively), amplitude equal to 6%
- 100 to 70% $v\dot{V}O_{2max}$ (for hard bouts and active recovery periods, respectively), amplitude equal to 18%
- 110 to 60% $v\dot{V}O_{2max}$ (for hard bouts and active recovery periods, respectively), amplitude equal to 30%.

The results showed that short intermittent exercise of 15 seconds at $v\dot{V}O_{2max}$ alternated with 15 seconds of active recovery, run at an average velocity equal to the critical velocity (85% of $v\dot{V}O_{2max}$ in this group), allowed middle-aged runners to reach and sustain $\dot{V}O_{2max}$. In fact, the result shows distinctly that the low and medium amplitudes induced the same metabolic (blood lactate and time spent at $\dot{V}O_{2max}$) and performance (distance, time limit) responses.

However, as underlined by Astrand and Rodahl,[57] 'it is an important but unsolved question which type of training is most effective: to maintain a level representing 90% of the maximal oxygen uptake for 40 minutes, or to tax 100% of the oxygen uptake capacity for about 16 minutes'. This remains an open question. But before beginning longitudinal studies to try to resolve this, it is first necessary to identify the metabolic response during different types of interval training (very short to long; see Daniels & Scardina[49]) used by trainers, especially for special (new) populations of runners such as middle-aged runners.

Even if optimum improvement in cardiorespiratory fitness is thought to occur following training at an intensity corresponding to 90 to 100% of $\dot{V}O_{2max}$,[58] this central factor is not the only one to induce an improvement in performance.

If we consider the distance run at a high velocity, the longer distance (2390 ± 815m) has to be performed with the lower amplitude 90 to 80% $v\dot{V}O_{2max}$ and the shorter with the higher amplitude 110 to 60% $v\dot{V}O_{2max}$ (1048 ± 365m). However, the distance run was not significantly different between the 90 to 80% $v\dot{V}O_{2max}$ and 100 to 70% $v\dot{V}O_{2max}$ interval training runs (2390 ± 815m *vs* 1980 ± 611m, $p = 0.23$). When we consider the distance run at the high velocity only, the difference was smaller because of the higher peak velocity in the 100 to 70% $v\dot{V}O_{2max}$ procedure compared with the 90 to 80% $v\dot{V}O_{2max}$ procedure (1265 ± 431 *vs* 1165m, $p = 0.6$).

However, the time spent at $\dot{V}O_{2max}$ is not the only parameter to be taken into account to judge the possible efficiency of a given type of interval training on the improvement of $\dot{V}O_{2max}$. Even if the 90 to 80% of $v\dot{V}O_{2max}$ interval training elicited $\dot{V}O_{2max}$ for the same duration as the 100 to 70% of $v\dot{V}O_{2max}$, the difference in peak velocity is 10% of $v\dot{V}O_{2max}$, i.e. 1.6 km/h. This difference could reduce the involvement of the fast IIa fibres, and prevent them from enhancing their oxidative capacity. However, even if running at 90% of $v\dot{V}O_{2max}$ instead of 100% of $v\dot{V}O_{2max}$ elicits the type IIa fibres less, we must emphasise that 90% of $v\dot{V}O_{2max}$ was just 1 km/h above the lactate threshold velocity. Therefore we can hypothesise that even at 90% of $v\dot{V}O_{2max}$, type IIa fibres are recruited.

In fact, central factors related to oxygen uptake are not the only limiting factors, even in long distance running. In addition to aerobic processes, neuromuscular and anaerobic characteristics are also involved.[47,59] Indeed, as suggested by Noakes,[47] the benefits of training also depend on the distance covered at a high velocity, which determines muscular adaptation maximising the number of powerful muscle contractions, interval training at 100 to 70% of $v\dot{V}O_{2max}$ is preferable to that at 90 to 80% of $v\dot{V}O_{2max}$.

For elderly runners who are used to train at long slow distances, a very short (15/15 seconds) interval training at 100% of $v\dot{V}O_{2max}$ with a recovery at 70% of $v\dot{V}O_{2max}$ seems be a good procedure to conciliate central and peripheral adaptations.[44] However, longitudinal studies are now necessary to confirm this hypothesis.

2.2 The Young

As for elderly runners, few studies have focused on interval training in children and adolescents. The influence of pretraining condition on effectiveness of the interval training has been tested in 15- to 19-year-olds.[60] It was found that the increase in work rate at a heart rate of 170 beats/min was significant for untrained participants versus trained athletes (+15 *vs* +5%). Interval training consisted of 4 to 8 repetitions of 200m (3 times per week for 5 weeks). Velocity and recovery were not specified. Given that the number of repetitions was similar for trained and untrained participants, the stimulus was perhaps insufficient for trained participants even if they ran at the same relative workrate.

68 Nigerian schoolgirls aged 15.5 years performed aerobic interval training [4 minutes at 90% of maximum heart rate (HR_{max}) and 4 minutes of jogging] 3 times a week for 8 weeks.[61] Another group of young girls (of the same level, i.e. $\dot{V}O_{2max}$ 39 ± 5 ml/min/kg) performed continuous work (4.8km at 80 to 85% HR_{max}). Improvement in $\dot{V}O_{2max}$ was similar for both groups (11%) and was significantly

higher than in the control group. It should be pointed out that the intensity was not very different (5% of HR_{max}) and it is probable that at the end of the severe run of 4.8km the girls were at $\dot{V}O_{2max}$ because of the slow component of $\dot{V}O_2$. The end blood lactate level for both types of training was not reported in this study.

Bar-Or[62] considered that children achieved steady state during intense exercise and recovered more quickly than adults following intense exercise. A practical implication is that, during high intensity interval training, children may need shorter resting periods than adults. It can be added that the aerobic contribution is higher. Fleck and Kraemer[63] underlined the fact that children should not copy elite athletic programmes.

11-year-old children carried out interval training at 25 and 50% above their anaerobic threshold (measured with a ventilatory method) [50 minutes per session, 5 times per week for 6 weeks].[64] They improved their anaerobic threshold (expressed in $\%\dot{V}O_{2max}$) by 22%.

As for adults, variation of training velocity from lactate (or ventilatory) velocity to $v\dot{V}O_{2max}$ might be the best stimulus to improve aerobic capacity. Indeed, a combination of continuous steady-state running (2 days per week) and interval training (2 days per week) was used for 8 weeks in boys aged from 10 to 14 years.[65] However, the biological age, which is a critical factor in this age group, was not reported in these studies. Continuous steady state was run at the ventilatory threshold velocity that the boys were able to sustain for 15 minutes at the beginning of the training period to twice this duration (30 minutes) in the eighth and final week of training. Interval training was run at 90 to 100% $\dot{V}O_{2max}$ and was equivalent to 135% of the ventilatory threshold $\dot{V}O_2$. Interval training consisted of repeated bouts of set distances in the range of 100 to 800m. The total distance run in this type of training ranged from 1.5km at the beginning of the 8 weeks of training to 2.5km at the end of the training period. $\dot{V}O_{2max}$ was improved by 8% (45.9 to 49.4 ml/min/kg). During the same time, the ventilatory threshold was improved by 19%. Therefore, before training the $\dot{V}O_2$ at ventilatory threshold appeared at 66.6% of $\dot{V}O_{2max}$ and after training at 73.8% of $\dot{V}O_{2max}$. We suggest that the higher efficiency of training for improving ventilatory threshold rather than $\dot{V}O_{2max}$ may come from the fact that the interval training increased both ventilatory threshold and $\dot{V}O_{2max}$. Therefore, the children had 4 training sessions per week for ventilatory threshold improvement and 2 training sessions per week (interval training only) for $\dot{V}O_{2max}$ improvement. This means that young untrained boys need to have high intensity training to improve $\dot{V}O_{2max}$. During the 8 weeks, height and bodyweight had not changed. Some authors have reported that children have higher lactate levels or ventilatory thresholds relative to their $\dot{V}O_{2max}$ than adults.[65] However, it is probably only a question of oxygen kinetics, and when a steady-state protocol is used to determine MLSS, children have the same MLSS values as adults when expressed as a percentage of $\dot{V}O_{2max}$.[66]

However, children have, or at least used to have, a style of life that is more comparable to interval training at or above $v\dot{V}O_{2max}$. Therefore, to improve $\dot{V}O_{2max}$, this would imply a high intensity of work with very short intermittent training (10 to 15 seconds) above $\dot{V}O_{2max}$ or long intervals, long enough (3 minutes of work at $v\dot{V}O_{2max}$, 3 minutes at 50% $v\dot{V}O_{2max}$) to elicit $\dot{V}O_{2max}$ for a long time. Children have the same time limit at $v\dot{V}O_{2max}$ as adults[67] and the improvement in $v\dot{V}O_{2max}$ is not systematically, as for adults,[67,68] accompanied by an improvement in the time limit at $v\dot{V}O_{2max}$.[66]

In 10- to 11-year-old boys, 9 weeks of interval training was found to improve both aerobic ($\dot{V}O_{2max}$ per kilogram of bodyweight: +8%) and anaerobic (mean power and peak power on Wingate test: +10 and 14%, respectively) capacity. These improvements were correlated with those of a 1200m run.[69] Docherty et al.[70] showed that changes in aerobic and anaerobic functions were independent of physiological maturity as determined by serum testosterone levels in young boys.

3. Studies Performed on Top Athletes

Acevedo and Goldfarb's study[71] remains one of the most cited papers, since they demonstrated that increased training intensity improved athletic performance in previously trained runners ($\dot{V}O_{2max}$ 65.8 ± 2.4 ml/min/kg). The training consisted of 8 weeks of increased intensity running 3 days per week at 48-hour intervals while continuing to run 8 to 19km (5 to 12 miles) per day on the other days of the week. One day was dedicated to interval training at 90 to 95% of HR_{max} with limited recovery times between runs (distance was not specified). As soon as heart rate returned to 120 beats/min they started again. The other 2 runs were fartlek workouts covering 10 to 16km at the specific pace of a 10km run, i.e. about 92% $v\dot{V}O_{2max}$. The total distance of the week was maintained; therefore the modifications could be attributed to an increase in training intensity. The performance over 10km was improved in relation to the decrease in blood lactate level at 85 and 90% $v\dot{V}O_{2max}$ and with no increase in $\dot{V}O_{2max}$. The authors hypothesised that lactate production and clearance (as already reported in rats by Denis et al.[72]) could be improved. This assumption has been recently confirmed by Fukuba et al.[73] in triathletes. An improvement in performance with no increase in $\dot{V}O_{2max}$ has already been reported in well trained participants by Daniels et al.[74]

A series of longitudinal studies performed by a South African group on well trained endurance cyclists examined metabolic and performance adaptation to interval training.[75] They replaced a portion (15%) of their 300km per week base endurance training with high intensity interval training (HIT). HIT consisted of 6 to 9 × 5-minute rides at 80% of PPO reached and sustained for 1 minute in an incremental test as defined by Noakes et al.[59] (This can be a little higher than $v\dot{V}O_{2max}$, since $\dot{V}O_{2max}$ can be reached before the highest work-rate; see Billat and Koralsztein.[48]) Recovery time was 1 minute. The programme lasted 6 weeks and the cyclists carried out 2 HIT sessions per week. In all these studies, an increase in performance during the 40km trial (reported as being a reproducible test) was obtained with an increase in the peak work-rate values. Cyclists were generally able to sustain a higher absolute and relative work-rate during the time trials, which lasted about 1 hour.[76,77] This HIT programme allowed time to fatigue to be increased by 150% at peak (1 minute) power output without any change in oxidative and glycolytic enzyme metabolism. The skeletal muscle buffering capacity was improved after this HIT programme.[76] This programme improved performance but it was not specified whether $\dot{V}O_{2max}$ was increased. There was, as in many longitudinal studies, no control group.

To determine whether a short-fast or a longer-slower interval training programme produced better improvements in performance, Septo et al.[78] compared 5 types of interval training sessions: (i) 12 × 30 seconds (rest interval 4.5 minutes) at 175% of PPO according to the definition by Noakes et al.;[59] (ii) 12 × 60 seconds at 100% PPO (rest 4.0 minutes); (iii) 12 × 2 minutes at 90% PPO (rest 3.0 minutes); (iv) 8 × 4 minutes at 85% PPO (rest 1.5 minutes); or (v) 4 × 8 minutes at 80% PPO (rest 1.0 minutes). Cyclists completed 6 sessions over a 3-week period in addition to their usual aerobic base training. The percentage improvement in the 40km time trial was modelled as a polynomial function of the rank order of the intensity of the training intervals. The cubic trend was strong and statistically significant and predicted the greatest enhancement for the intervals performed at 85% PPO and at 175% PPO. PPO was correlated with performance, i.e. the 40km time trial. Intervals performed at 100% PPO and 80% PPO did not produce any statistically significant enhancements in performance. The authors concluded that interval training with work bouts of 3 to 6 minutes at an intensity of 85% of PPO gave the maximal enhancement of performance for this exercise over 40km, lasting about 1 hour at this power output. However, according to the principle of specificity, the 30-second work bouts, which would have been achieved by a substantial contribution from oxygen-independent glycolysis, would not enhance performance over a 40km time trial, which depends almost entirely on power provided

by the aerobic system. However, 12 × 30 seconds also involves aerobic metabolism, and it is probable that buffering capacity plays a part in an all-out exercise of 1 hour where glycolysis is at a high rate.

Interval training should be practised by cyclists, since Palmer et al.[79] showed that elite cyclists racing in a pack randomly vary their work rates from around 50% to almost 100% of the peak sustained power output, independently of the track.

Elite runners train at a higher level of intensity (relative to their $v\dot{V}O_{2max}$) than lower level athletes.[59,80] The authors of these studies suggested that $\dot{V}O_{2max}$ and endurance performance may be limited not only by central cardiovascular factors related to $\dot{V}O_2$ but also by the so-called 'muscle power' factors affected by the interaction of both neuromuscular and anaerobic characteristics. Therefore, supramaximal interval training can also be included in a programme for middle distance runners.

Even if more than 75% of the energy is provided by aerobic metabolism,[81,82] anaerobic capacity is very important for 1500m runners. They must be able to run the first 800m in 1 minute 50 seconds, as seen in the last world athletic championship in 1999 (26.2 km/h, which corresponds to a pace of 3 minutes 25 seconds, faster than the world record). Even for the 10 000m event, the last 1000m and 300m in the last world championship were run in 2 minutes 25.2 seconds (24.8 km/h) and 39.8 seconds (27.1 km/h), respectively. This means that if the world champion Gebreselassie has a $v\dot{V}O_{2max}$ of 25.5 km/h estimated from his best performance at 3000m, he is able to run at 106% of $v\dot{V}O_{2max}$ during the last (25th) lap of the 10 000m race.

4. Conclusion

Much more study is needed to understand the physiological basis of interval training, especially in elite athletes. However, sports records are performed with variable velocity, even in long distance running. Considering the last 3 world records in middle and long distance running (1500, 3000, 5000 and 10 000m), it can be observed that the range of the coefficient of variation of velocity is 1 to 5% (in the 10 000m of 1997 by Gebreselassie and in the 1500m of 1995 by Morcelli, respectively). This could mean that interval training could be individualised taking into account this individual stochastic pace, allowing athletes to reach their best performance.

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References

1. Margaria R, Oliva RD, di Prampero PE, et al. Energy utilization in intermittent exercise of supramaximal intensity. J Appl Physiol 1969; 26: 752-6
2. Balsöm PD, Seger JY, Sjodin B, et al. Physiological responses to maximal intensity intermittent exercise. Eur J Appl Physiol 1992; 65: 144-9
3. Christensen EH, Hedman R, Saltin B. Intermittent and continuous running. Acta Physiol Scand 1960; 50: 269-6
4. Tabata I, Irisawa K, Kouzaki M, et al. Metabolic profile of high intensity intermittent exercises. Med Sci Sports Exerc 1997; 29: 390-5
5. Bogdanis GC, Nevill ME, Boobis LH, et al. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. J Appl Physiol 1996; 80: 876-84
6. Gaitanos GC, Williams C, Boobis LH, et al. Human muscle metabolism during intermittent maximal exercise. J Appl Physiol 1993; 75: 712-9
7. Spriet LL, Lindinger MI, McKelvie RS, et al. Muscle glycogenolysis and H+ concentration during maximal intermittent cycling. J Appl Physiol 1989; 66: 8-13
8. Hirvonen J, Rehunen S, Rusko H, et al. Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. Eur J Appl Physiol 1987; 56: 253-9
9. Balsom PD, Seger JY, Sjodin B, et al. Maximal-intensity intermittent exercise: effect of recovery duration. Int J Sports Med 1992, 13: 528-33
10. Spriet LL. Anaerobic metabolism in human skeletal muscle during short-term, intense activity. Can J Physiol Pharmacol 1992; 70: 157-65
11. Yoshida T, Watari H. Metabolic consequences of repeated exercise in long distance runners. Eur J Appl Physiol 1993; 67: 261-5
12. Bogdanis GC, Nevill ME, Boobis LH, et al. Recovery of power output and muscle metabolites following 30s of maximal sprint cycling in man. J Physiol (Lond) 1995; 15: 467-80
13. Brooks GA, Mercier J. Balance of carbohydrate and lipid utilization during exercise: the 'crossover' concept. J Appl Physiol 1994; 76: 2253-61
14. Jenkins DG, Palmer J, Spillman D. The influence of dietary carbohydrate on performance of supramaximal intermittent exercise. Eur J Appl Physiol 1993; 67: 309-14
15. Fox EL, Bartels RL, Billing CE, et al. Frequency and duration of interval training programs and changes in aerobic power. J Appl Physiol 1975; 38: 481-4

16. Lesmes GR, Fox EL, Stevens C, et al. Metabolic responses of females to high intensity interval training of different frequencies. Med Sci Sport Exerc 1978; 10: 229-32
17. Fox EL, Bartels RL, Billings CE, et al. Intensity and distance of interval training programs and changes in aerobic power. Med Sci Sports Exerc 1973; 5: 18-22
18. Davies KJA, Packer L, Brooks GA. Biochemical adaptation of mitochondria, muscle, and whole-animal respiration to endurance training. Arch Biochem Biophys 1981; 209: 539-54
19. Davies KJA, Packer L, Brooks GA. Exercise bioenergetics following sprint training. Arch Biochem Biophys 1982; 215: 260-5
20. Simoneau JA, Lortie G, Boulay MR, et al. Inheritance of human skeletal muscle and anaerobic capacity adaptation to high-intensity intermittent training. Human skeletal muscle fiber type alteration with high-intensity intermittent training. Int J Sports Med 1986; 7: 167-71
21. Simoneau JA, Lortie G, Boulay MR, et al. Human skeletal muscle fiber type alteration with high-intensity intermittent training. Eur J Appl Physiol 1985; 54: 250-3
22. Simoneau JA, Lortie G, Boulay MR, et al. Effects of two high-intensity intermittent training programs interspersed by detraining on human skeletal muscle and performance. Eur J Appl Physiol 1987; 56: 516-21
23. Billat V, Blondel N, Berthoin N. Determination of the velocity associated with the longest time to exhaustion at maximal oxygen uptake. Eur J Appl Physiol 1999; 80: 159-61
24. Roberts AD, Billeter R, Howald H. Anaerobic muscle enzyme changes after interval training. Int J Sports Med 1982; 3: 18-21
25. Margaria R, Cerretelli P, Aghemo P, et al. Energy cost of running. J Appl Physiol 1963; 18: 367-70
26. Linossier MT, Denis C, Dormois D, et al. Ergometric and metabolic adaptation to a 5-s sprint training programme. Eur J Appl Physiol 1993; 67: 408-14
27. Nevill ME, Boobis LH, Brooks S, et al. Effect of training on muscle metabolism during treadmill sprinting. J Appl Physiol 1989; 67: 2376-82
28. Hargreaves M, McKenna MJ, Jenkins DG, et al. Muscles metabolites and performance during high-intensity, intermittent exercise. J Appl Physiol 1998; 84: 1687-91
29. Heugas AM, Brisswalter J, Vallier JM. Effet d'une période d'entraînement de trois mois sur le déficit maximal en oxygène chez des sprinters de haut niveau de performance. Can J Appl Physiol 1997; 22: 171-81
30. MacDougall DJ, Hicks AL, MacDonald JR, et al. Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol 1998; 84: 2138-42
31. Houmard JA, Costill DL, Mitchell JB, et al. The role of anaerobic ability in middle distance running performance. Eur J Appl Physiol 1991; 62: 40-3
32. Jenkins DG, Quigley BM. The influence of high-intensity exercise training on the Wlim-Tlim relationship. Med Sci Sports Exerc 1993; 25: 275-82
33. Lechevalier JM, Vandewalle H, Chatard JC, et al. Relationship between the 4 mMol running velocity, the time-distance relationship and the Leger-Boucher test. Arch Int Physiol Biochem 1989; 97: 355-60
34. Paavolainen L, Hakkinen K, Hamalainen 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
35. Franch J, Madsen K, Djurhuus MS, et al. Improved running economy following intensified training correlates with reduces ventilatory demands. Med Sports Sci Exerc 1998; 30: 1250-6
36. Komi PV, Kyröläiinen H. Mechanical efficiency of stretch-shortening cycle exercise. In: Marconnet P, Saltin B, Komi P, et al., editors. Human muscular function during dynamic exercise. Basel: Karger, 1996: 44-56
37. Flynn MG, Fahlman M, Braun WA, et al. Effects of resistance training on selected indices of immune function in elderly women. J Appl Physiol 1999; 86: 1905-13
38. Wells CL, Pate RR. Training for performance of prolonged exercise. In: Lamb DR, Murray R, editors. Perspectives in exercise science and sports medicine. Vol. 1. Indianapolis (IN): Benchmark Press, 1988: 357-91
39. Tanaka H, Swensen T. Impact of resistance training on endurance performance. Sports Med 1985; 6: 266-70
40. Balsom PD, Ekblom B, Söderlund K, et al. Creatine supplementation and dynamic high-intensity intermittent exercise. Scand J Med Sci Sports 1993; 3: 143-9
41. Tabata I, Nishimura K, Kouzaki M, et al. Effects of moderate-intensity endurance and high-intensity intermittent training on anaerobic capacity and $\dot{V}O_2$max. Med Sci Sports Exerc 1996; 28: 1327-30
42. Ahmaidi S, Masse-Biron J, Adam B, et al. Effects of interval training at the ventilatory threshold on clinical and cardiorespiratory responses in elderly humans. Eur J Appl Physiol 1998; 78: 170-6
43. Suominen H, Heikkinen E, Liesen H, et al. Effect of 8-weeks endurance training on skeletal muscle metabolism in 56-70 year old sedentary men. Eur J Appl Physiol 1977; 37: 173-80
44. Billat V, Slawinski J, Bocquet V, et al. Very short interval training (15s-15s) around the critical velocity allows middle-aged runners to maintain $\dot{V}O_{2max}$ for 14 min. Int J Sports Med. In press
45. Péronnet F, Thibault G. Mathematical analysis of running performance and world running records. J Appl Physiol 1989; 67: 453-65
46. Daniels J. Jacks Daniels' formula. Champaign (IL): Human Kinetics Publishers, 1998
47. Noakes T. Lore of running. Champaign (IL): Human Kinetics Publishers, 1991
48. Billat V, Koralsztein JP. Significance of the velocity at $\dot{V}O_2$max and its time to exhaustion at this velocity. Sports Med 1996; 22: 90-108
49. Daniels J, Scardina N. Interval training and performance. Sports Med 1984; 1: 327-34
50. Gorostiaga EM, Walter CB, Foster C, et al. Uniqueness of interval and continuous training at the same maintained exercise intensity. Eur J Appl Physiol 1991; 63: 101-7
51. Billat V, Slawinski J, Bocquet V, et al. Intermittent runs at v$\dot{V}O_2$max enables subjects to remain at $\dot{V}O_2$max for a longer time than submaximal runs. Eur J Appl Physiol 2000; 81: 188-96
52. Saltin B, Essen B. Muscle glycogen, lactate, ATP, and CP in intermittent exercise. In: Pernow B, Saltin B, editors. Muscle metabolism during exercise. Advances in experimental medicine and biology. Vol. 11. New York: Plenum Press, 1971: 419-24
53. Saltin B, Essen B, Pedersen PK. Intermittent exercise: its physiology and some practical applications. In: Joekle E, Anand RL, Stoboy H, editors. Advances in exercise physiology. Basel: Karger, 1976: 9: 23-51 (Medicine Sport; 5)

54. Pepper ML, Housh TJ, Johnson GO. The accuracy of the critical velocity test for predicting time to exhaustion during treadmill running. Int J Sports Med 1992; 13: 121-4
55. Gaesser GA, Poole DC. The slow component of oxygen uptake kinetics in humans. Exerc Sports Sci Rev, 1996; 24: 35-70
56. Florence SL, Weir JP. Relationship of critical velocity to marathon running performance. Eur J Appl Physiol 1997; 75: 274-8
57. Astrand PO, Rodahl K. Textbook of work physiology. 3nd ed. New York: McGraw Hill, 1986
58. Robinson DM, Robinson SM, Hume PA, et al. Training intensity of elite male distance runners. Med Sci Sports Exerc 1991; 23: 1078-82
59. Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the $\dot{V}O_2$max test predicts running performance. J Sports Sci 1990; 8: 35-45
60. Watson AWS, O'Donovan DJ. The effects of five weeks of controlled interval training on youths of diverse pre-training condition. J Sports Med Phys Fitness 1977; 17: 139-46
61. Adeniran SA, Toriola AL. Effects of continuous and interval running programmes on aerobic and anaerobic capacities in schoolgirls aged 13 to 17 years. J Sports Med Phys Fitness 1988; 28: 260-6
62. Bar-Or O. The young athlete: some physiological considerations. J Sports Sci 1995; 13: 531-3
63. Fleck SJ, Kraemer WJ. Designing resistance training programs. Champaign (IL): Human Kinetics Publishers, 1997
64. Haffor AA, Harrison AC, Catledge-Kirk PA. Anaerobic threshold alterations caused by interval training in 11-years olds. J Sports Med Phys Fitness 1990; 30: 53-6
65. Mahon AD, Vaccaro P. Ventilatory threshold and $\dot{V}O_2$max changes in children following endurance training. Med Sci Sports Exerc 1989; 4: 425-31
66. Billat V, Gratas-Delamarche A, Monnier M, et al. Test to approach maximal lactate steady-state in 12-year old boys and girls. Arch Biochem Physiol 1995; 103: 65-72
67. Berthoin S, Manteca F, Gerbeaux M, et al. Effect of a 12-week training programme on maximal aerobic speed (MAS) and running time to exhaustion at 100% of MAS for students aged 14 to 17 years. J Sports Med Phys Fitness 1995; 35: 251-6
68. Billat V, Flechet B, Petit B, et al. Interval training at $\dot{V}O_2$max: effects on aerobic performance and overtraining markers. Med Sci Sport Exerc 1999; 31: 156-63
69. Rostein A, Dotan R, Bar-Or O, et al. Effect of training on anaerobic threshold, maximal aerobic power and anaerobic performance of preadolescent boys. Int J Sports Med Phys 1986; 7: 281-6
70. Docherty D, Wenger HA, Collis ML. The effects of resistance training on aerobic power of young boys. A test to approach maximal lactate steady-state in 12-year old boys and girls. Med Sci Sport Exerc 1987; 19: 389-92
71. Acevedo EO, Goldfarb AH. Increased training intensity effects on plasma lactate, ventilatory threshold, and endurance. Med Sci Sports Exerc 1989; 21: 563-8
72. Denis C, Fouquet R, Poty P, et al. Effect of 40 weeks of endurance training on the anaerobic threshold. Int J Sports Med 1982; 3: 208-13
73. Fukuba Y, Walsh ML, Morton RH, et al. Effect of endurance training on blood lactate clearance after maximal exercise. J Sports Sci 1999; 17: 239-48
74. Daniels JT, Yarbough RA, Foster C. Changes in $\dot{V}O_2$max and running performance with training. Eur J Appl Physiol 1978; 39: 249-54
75. Westgarth-Taylor C, Hawley JA, Richard S, et al. Metabolic and performance adaptations to interval training in endurance-trained cyclists. Eur J Appl Physiol 1997; 75: 298-304
76. Weston AH, Myburgh KH, Lindsay FH, et al. Skeletal muscle buffering capacity and endurance performance after high-intensity interval-training well-trained cyclists. Eur J Appl Physiol 1997; 75: 7-13
77. Lindsay FH, Hawley JA, Myburgh KH, et al. Improved athletic performance in highly trained cyclists after interval training. Med Sci Sports Exerc 1996; 28: 1427-37
78. Septo NK, Hawley JA, Dennis SC, et al. Effects of different interval training programs on cycling time-trial performance. Med Sci Sports Exerc 1999; 31: 736-41
79. Palmer GS, Hawley JA, Dennis SC, et al. Heart rate responses during a 4-d cycle stage race. Med Sci Sports Exerc 1994; 26: 1278-83
80. Green H, Patla M. Maximal aerobic power: neuro-muscular and metabolic considerations. Med Sci Sports Exerc 1992; 24: 38-46
81. Spencer MR, Gastin PB, Payne WR. Energy system contribution during 400 to 1500 metres. New Studies Athletics 1996; 4: 59-65
82. Weyand PG, Cureton KJ, Conley DS, et al. Peak oxygen deficit predicts sprint and middle-distance track performance. Med Sci Sports Exerc 1994; 9: 1174-80

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