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The Scientific Basis for High-Intensity Interval Training

Optimising Training Programmes and Maximising Performance in Highly Trained Endurance Athletes

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Abstract

While the physiological adaptations that occur following endurance training in previously sedentary and recreationally active individuals are relatively well understood, the adaptations to training in already highly trained endurance athletes remain unclear. While significant improvements in endurance performance and corresponding physiological markers are evident following submaximal endurance training in sedentary and recreationally active groups, an additional increase in submaximal training (i.e. volume) in highly trained individuals does not appear to further enhance either endurance performance or associated physiological variables [e.g. peak oxygen uptake ($\dot{V}O_{2peak}$), oxidative enzyme activity]. It seems that, for athletes who are already trained, improvements in endurance

performance can be achieved only through high-intensity interval training (HIT). The limited research which has examined changes in muscle enzyme activity in highly trained athletes, following HIT, has revealed no change in oxidative or glycolytic enzyme activity, despite significant improvements in endurance performance ($p < 0.05$). Instead, an increase in skeletal muscle buffering capacity may be one mechanism responsible for an improvement in endurance performance. Changes in plasma volume, stroke volume, as well as muscle cation pumps, myoglobin, capillary density and fibre type characteristics have yet to be investigated in response to HIT with the highly trained athlete. Information relating to HIT programme optimisation in endurance athletes is also very sparse. Preliminary work using the velocity at which $\dot{V}O_{2max}$ is achieved (V_{max}) as the interval intensity, and fractions (5075%**((Author: 50 to 75%?)**)) of the time to exhaustion at V_{max} (T_{max}) as the interval duration has been successful in eliciting improvements in performance in long-distance runners. However, V_{max} and T_{max} have not been used with cyclists. Instead, HIT programme optimisation research in cyclists has revealed that repeated supramaximal sprinting may be equally effective as more traditional HIT programmes for eliciting improvements in endurance performance. Further examination of the biochemical and physiological adaptations which accompany different HIT programmes, as well as investigation into the optimal HIT programme for eliciting performance enhancements in highly trained athletes is required.

Compared with the volume of research that describes the physiological adaptations to endurance-exercise training in sedentary and recreationally trained individuals, relatively little work has examined the physiological and performance responses of already highly trained athletes to a modified training programme. In part, this may be because of the difficulty of persuading highly trained athletes to alter their training programmes to accommodate the interests of exercise scientists.[1] Consequently, recommendations made by exercise scientists to coaches and athletes are largely based on training studies completed with sedentary and recreationally trained individuals coupled with anecdotal hearsay of some successful coaches.[1,2] Highly trained athletes already have a high aerobic capacity, lactate threshold (T_{lac}), and economy of motion.[3,4] Therefore, the physiological adaptations that generally account for improved performance in sedentary or recreationally trained individuals[5,6] may not necessarily apply to the highly trained athlete.[7] Indeed, in the highly trained athlete, an additional increase in submaximal exercise training (i.e. volume) does not appear to further enhance either endurance performance or associated variables such as maximal oxygen uptake ($\dot{V}O_{2max}$), anaerobic threshold, economy of motion and oxidative muscle enzymes.[8-10]

In these individuals, it appears that further improvements in performance can only be achieved through high-intensity interval training (HIT).[8] The only research to date that has examined the physiological responses of already highly trained athletes to HIT[7] indicates no up-regulation of oxidative or glycolytic enzyme activity, despite significant improvements in 40km cycling time-trial performance and peak power output (P_{peak}) obtained during a progressive exercise test ($p < 0.05$). Skeletal muscle buffering capacity significantly increased ($p < 0.05$), but the increase was not related to improvements in P_{peak} and 40km time-trial performance. Thus, the mechanisms responsible for improvements in performance following HIT in the already highly trained athlete remain unclear. This article will: (i) briefly review the adaptations to endurance training known to occur in sedentary and recreationally active individuals; (ii) outline some physiological mechanisms that may further

enhance performance following HIT in the already highly-trained athlete; and (iii) consider the issue of HIT programme optimisation in the highly-trained athlete.

1. Endurance Training in the Untrained and Recreationally Active

1.1 Submaximal Endurance Training

It is generally accepted that many of the biochemical and physiological adaptations that accompany endurance training occur in response to an increase in muscle cell energy demands.[11,12] Indeed, manipulation of the intensity and duration of work and rest intervals changes the relative demands on particular metabolic pathways within muscle cells, as well as oxygen delivery to muscle. The subsequent adaptations that occur, both at the cellular and systemic level, are specific to the particular characteristics of the training programme employed.

Several short- and long-term training studies performed with sedentary individuals have challenged the aerobic energy system through daily submaximal training (i.e. 2 h/d, 65 to 75% $\dot{V}O_{2max}$).[13-15] In these studies, the improvements in physical work capacity (i.e. P_{peak} [13]), were attributed to an increased delivery of oxygen to the exercising muscles (central adaptations),[6,16] coupled with increased utilisation of oxygen by the working muscles (peripheral adaptations).[17-20] Central adaptations to endurance training result in a lower heart rate at pre-training workrates[6] coupled with an increase in blood and plasma volume (hypervolaemia).[6,13] These changes are accompanied by a greater cardiac output (stroke volume),[6,21] and increases in muscle and cutaneous blood flow during exercise at the same pre-training workrate.[22,23] Although these central adaptations may allow relatively rapid (i.e. 3 days) improvements in physical work capacity ($p < 0.05$),[13] a longer period of training (three to five times per week, 12 to 38 days) may be needed for increases in $\dot{V}O_{2max}$ to occur ($p < 0.05$).[16,24,25] Indeed, several weeks of training may be needed before changes in muscle capillary density and mitochondrial volume are observed ($p < 0.05$).[26-29] Other adaptations to endurance training include a reduction in glucose[30-32] and muscle glycogen utilisation,[33] as well as lower blood lactate levels at the same absolute workload ($p < 0.05$).[6,17,20,34-37]

Thus, with previously untrained individuals, exercise-induced cellular hypoxia increases blood flow, oxygen delivery, oxygen extraction and fat metabolism in working muscles during submaximal exercise after training. As a result, muscle contraction becomes more efficient and physical work capacity increases. However, when submaximal endurance training becomes habitual, such as for the endurance athlete, further improvements in exercise performance with an increase in training volume do not normally occur.[8,9,25,38] Indeed, the muscle of trained athletes has three to four times higher oxidative enzyme activity, up to three times more capillaries per muscle fibre, and a greater percentage of slow twitch fibres when compared with untrained muscle.[39] In these individuals, additional improvements in endurance performance and associated physiological markers appear to require a different training stimulus than simply an increase in volume.[8,25,40,41]

1.2 High-Intensity Interval Training (HIT)

It is generally believed that in sedentary ($\dot{V}O_{2max} < 45$ ml/kg/min) and recreationally active individuals ($\dot{V}O_{2max} \approx 45$ to 55 ml/kg/min), several years are required to increase $\dot{V}O_{2max}$ to that of the highly trained athlete ($\dot{V}O_{2max} > 60$ ml/kg/min).[21,42] However, Hickson et al.[43] showed, in eight sedentary and recreationally active individuals, that $\dot{V}O_{2max}$ could be markedly increased (+44%; $p < 0.05$) after 10 weeks of high-intensity exercise training (alternating 40 minutes cycling intervals at $\dot{V}O_{2max}$ 1 day, with 40 minutes high-intensity running the next, 6 d/wk). Interestingly, in four of these individuals, $\dot{V}O_{2max}$ approached or exceeded 60 ml/kg/min. This clearly shows how an increase in high-intensity exercise training can elicit a rapid improvement in 'aerobic fitness'.[43]

In contrast to submaximal exercise training, which is characterised by prolonged, continuous activity, HIT is normally achieved through the use of intervals. HIT can be broadly defined as repeated bouts of short to moderate duration exercise (i.e. 10 seconds to 5 minutes) completed at an intensity that is greater than the anaerobic threshold. Exercise bouts are separated by brief periods of low-intensity work or inactivity that allow a partial but often not a full recovery. The purpose of HIT is to repeatedly stress the physiological systems that will be used during a specific endurance-type exercise[44] to a greater extent than that which is actually required during the activity. Despite the fact that coaches have long used HIT to improve the performance of endurance athletes,[1] studies describing the influence of HIT on muscle respiration have largely been limited to previously sedentary or recreationally active individuals (**table I**).[45]

Several studies have indicated that intermittent HIT may increase fat oxidation when compared with continuous training. Essen and associates[61] compared 1 hour of continuous exercise at 50% $\dot{V}O_{2max}$ with 1 hour of intermittent exercise (15 seconds work at P_{peak}, 15 seconds rest) of the same mean workload (157W). In these previously untrained individuals, more lipids and less glycogen were used when exercise was performed intermittently, as opposed to continuously. In another study with untrained individuals,[45] HIT (5 × 4 minutes at 100% $\dot{V}O_{2max}$, 2 minutes rest; n = 13) was found to enhance the oxidative capacity (succinate dehydrogenase and cytochrome oxidase) of type II fibres ($p < 0.05$), when compared with a continuous exercise training group (n = 8) which performed exercise of a similar duration at the same average intensity (79% $\dot{V}O_{2max}$). A recent study in rats has provided support for this earlier finding; mitochondrial fatty acid oxidation rates have been shown to increase to a greater extent following HIT than following continuous submaximal endurance training ($p < 0.05$).[62]

Somewhat in contrast, however, Gorostiaga and colleagues[63] reported an increase in citrate synthase (CS) activity in response to continuous but not HIT. The opposite trend was seen in the activity of adenylate kinase, which increased by 25% following HIT, but not continuous training. The authors compared the physiological effects of continuous training (50% $\dot{V}O_{2max}$; n = 6) versus HIT (repeated 30 seconds at 100% $\dot{V}O_{2max}$, 30 seconds rest; n = 6). Their participants cycled 30 min/d, 3 d/wk, for 8 weeks, with both groups exercising at the same mean intensity. Following training, $\dot{V}O_{2max}$, exercising work rates and P_{peak} obtained during the incremental test were all higher ($p < 0.05$) in the HIT group (+9 to 16%) compared with the continuous training group (+5 to 7%). However, the exercise intensity in the continuous training group was adjusted so that individuals trained at the same heart rate throughout the 8 weeks of training. It is generally accepted that continuous submaximal training will reduce the heart rate corresponding to a pre-training workrate.[6] Thus, the continuous training group likely received an increase in their continuous training intensity throughout their 8 weeks of training, which may help explain why CS activity was improved more than in the HIT group in this study.

In a more recent study by Franch et al.,[64] the effects of continuous and HIT were compared in recreational runners (n = 36; $\dot{V}O_{2max}$ = 54.8 ± 3.0 ml/kg/min). Individuals were equally matched into three groups; either short HIT (30 to 40 × 15 seconds at 20.4 km/h, 15 seconds inactive rest), long HIT (4 to 6 × 4 minutes at 16.6 km/h, 2 minutes inactive rest), or continuous running (15 km/h, ~26 minutes). All groups trained three times per week (2.2 h/wk) at a mean exercise intensity of ~65% maximum heart rate for 6 weeks. Both the continuous running and the long HIT groups improved their $\dot{V}O_{2max}$ significantly more than the short HIT group (6*vs* 3%; $p < 0.05$). Furthermore, time to exhaustion at 85% $\dot{V}O_{2max}$ increased significantly more in the continuous running group (+93%; $p < 0.05$) compared with the long (+67%) and short (+65%) HIT groups. It is important to note, however, that the individuals in this study had low levels of initial fitness, so the finding of a greater im-

Table I. Findings from high-intensity interval-training studies in sedentary and recreationally active individuals [a]

Reference	n	Mode	Frequency (d/wk)	Weeks	Reps	Intensity	Work duration	Rest duration	Results
Hickson et al.[43]	8 M	R and C	6	10	6	100% $\dot{V}O_{2max}$	5 min	2 min	↑$\dot{V}O_{2max}$, ↑T_{lim}
Green et al.[46]	10 M	C	1	1	16	90% $\dot{V}O_{2max}$	6 min	54 min	↑PCr, ↑Gly, ↓Lac^-
Green and Fraser[47]	6 M	C	3	1	12-24	120% $\dot{V}O_{2max}$	1 min	4 min	↑UA
Keith et al.[48]	7 M	C	2-4	8	2	T_{lac} + 30%	7.5 min	30 min	↑$\dot{V}O_{2max}$, ↑P_{peak}, ↑CS, ↑3-HcoA, ↑T_{lac}
Keith et al.[48]	8 M	C	2-4	8	1	T_{lac}	30 min	0	↑$\dot{V}O_{2max}$, ↑P_{peak}, ↑CS, ↑3-HCoA, ↑T_{lac}
Burke et al.[49]	21 F	C	4	7	NR	85-98% $\dot{V}O_{2max}$	30-120 sec	30-120 sec	↑$\dot{V}O_{2max}$, ↑T_{lac}, ↑T_{vent}
Simoneau et al.[50]	24M, F**((Author: n =?))**	C	4-5	15	4-15	60-90% P_{peak}	15-90 sec	HR = 120-130 bpm	↑type I, ↓type IIb, ↔ type IIa
Rodas et al.[51]	5 M	C	7	2	4-7	All-out	15-30 sec	45s-12 min	↑PCr, ↑Gly, ↑CK, ↑PFK, ↑LDH, ↑3-HcoA, ↑CS, ↑$\dot{V}O_{2max}$, ↑P_{peak}, ↔ WIN
Parra et al.[52]	5 M	C	2	6	4-7	All-out	15-30 sec	45s-12 min	↑PFK, ↑ALD, ↑CS, ↑3-HCoA, ↑P_{peak}, ↑WIN
MacDougall et al.[53]	12 M	C	3	7	4-10	All-out	30 sec	2.5-4 min	↑HK, ↑PFK, ↑CS, ↑SD, ↑MD, ↑P_{peak}, ↑WIN, ↑$\dot{V}O_{2max}$
Linossier et al.[54]	10 M, F**((Author: n =?))**	C	4	7	8-13	All-out	5 sec	55 sec	↑WIN, ↑Lac^-, ↑PFK, ↑LDH, ↑Type I, ↓Type IIb, ↔ Type IIa
Simoneau et al.[55]	19M, F**((Author: n =?))**	C	2-3	15	10-15	60-90% P_{peak}	15-30 sec	HR = 120-130 bpm	↑HK**((Author: hexokinase activity?))**, ↑PFK, ↑LDH, ↑MD, ↑3-HcoA, ↑OGDH
Henritze et al.[56]	23 F	C	5	12	1	T_{lac} –T_{lac} + 69W	NR	NA	↑T_{lac}, ↔ $\dot{V}O_{2max}$
Nevill et al.[57]	8 M, F**((Author: n =?))**	R	3-4	8	2-10	All-out	6-30 sec	1-10 min	↑WIN, ↑Lac^-, ↑NE, ↔ β_m, ↑H^+
Tabata et al.[58]	7 M	C	5	6	7-8	170% $\dot{V}O_{2max}$	20 sec	10 sec	↑$\dot{V}O_{2max}$, ↑AN_{cap}
Ray[59]	6 M	C	4	6	5	90-100% $\dot{V}O_{2max}$	5 min	3 min	↑$\dot{V}O_{2max}$, ↓HR_{rest}, ↓MAP, ↓MSNA
Harmer et al.[60]	7 M	C	3	7	4-10	All-out	30 sec	3-4 min	Before maximum work-rate: ↑T_{lim}, ↓$Lac^-_{m, pl}$, ↓H^+, ↓$_{an}ATP_{prod}$, ↓IMP, ↓Gly, ↓ATP_{deg}, ↓K^+, ↓NE. After maximum work-rate: ↔Lac^-_m, ↑Lac^-_{pl}, ↓H^+_m, ↑H^+_{pl}, ↑NE, ↓ATP_{deg}, ↓$_{an}ATP_{prod}$, ↓IMP

a Changes indicated based on statistical significance at the p< 0.05 level.

ALD = aldosterone; **$_{an}$ ATP $_{prod}$** = anaerobic ATP production; **AN $_{cap}$** = anaerobic capacity; **ATP $_{deg}$** = ATP degradation; **b** = blood; **((Author: "b" does not seem to be used in this table. Delete from abbrev list?))** **C** = cycle training; **CK** = creatine kinase activity; **CS** = citrate synthase activity; **F** = female; **Gly** = glycogen content; **H $^+$** = hydrogen ions; **HR** = heart rate; **HR $_{rest}$** = resting HR; **IMP** = inosine monophosphate; **K $^+$** = potassium ions; **Lac $^-$** = lactate; **LDH** = lactate dehydrogenase activity; **M** = male; **m** = muscle; **MAP** = mean arterial pressure; **MD** = malate dehydrogenase activity; **MSNA** = muscle sympathetic nerve activity; **n** = number of participants; **NA** = not applicable; **NE** = plasma norepinephrine (noradrenaline); **NR** = not reported; **OGDH** = oxoglutarate dehydrogenase activity; **PCr** = phosphocreatine; **PFK** = phosphofructokinase activity; **pl** = plasma; **P $_{peak}$** = peak power output, **R** = run training; **Reps** = repetitions; **SD** = succinate dehydrogenase activity; **T $_{lac}$** = lactate threshold; **T $_{lim}$** = time to exhaustion; **T $_{vent}$** = ventilatory threshold; **type I, IIa, IIb** = type I, IIa, and IIb muscle fibres; **UA** = **((Author: please define))**; $\dot{V}$**O $_{2max}$** = maximal oxygen uptake; **WIN** = Wingate anaerobic test performance; **3-HCoA** = 3-hydroxyacyl coenzyme A dehydrogenase activity; β = buffering capacity; ↓ = **((Author: decrease?))**; ↑ = **((Author: increase?))**; ↔ = **((Author: no change?))**.

provement in $\dot{V}O_{2max}$ and time to exhaustion following the continuous training is not surprising. Had highly trained athletes been exposed to the same training stimulus, it is unlikely that the same magnitude of changes would have been observed, as highly trained athletes train regularly at these continuous submaximal intensities[65,66] at least once per week (generally called tempo training).[1]

The effects of repeated supramaximal HIT in previously untrained individuals have also been examined by Harmer et al.,[60] MacDougall et al.,[53] Parra et al.[52] and Rodas et al.[51] MacDougall and co-workers[53] examined the influence of supramaximal HIT on muscle enzyme activity and exercise performance in 12 previously active students ($\dot{V}O_{2max}$ = 3.73 ± 0.13 L/min). For 7 weeks, individuals performed 4 weekly HIT sessions that became progressively more challenging, in terms of a progressive increase in the number of interval bouts (4 to 10 × 30 seconds all-out cycle sprints) and a progressive reduction in the duration of recovery between interval bouts (4 to 2.5 minutes). Individuals significantly enhanced their peak anaerobic power output and total work done over 30 seconds, as well as their $\dot{V}O_{2max}$. The maximal enzyme activities of CS, hexokinase (HK), phosphofructokinase (PFK), succinate dehydrogenase and malate dehydrogenase also significantly increased following training ($p < 0.05$). Thus, in contrast to submaximal endurance training, that has little or no effect on glycolytic enzyme activity,[67,68] relatively brief but intense supramaximal HIT training can elicit concurrent up-regulation of both glycolytic and oxidative enzyme activity, maximum short-term power output, and $\dot{V}O_{2max}$ in untrained individuals. The findings of the concurrent up-regulation of aerobic and anaerobic metabolism may have been due to the progressive reduction in recovery periods between HIT bouts, which likely would have created for a greater reliance on aerobic metabolism.[69] A more recent study has produced similar findings.[51] The authors had their five moderately active individuals sprint train on a cycle ergometer (8 to12 × 15 seconds all out, 45 seconds rest) each day for 2 weeks. Significant increases were found in the muscle activities of creatine kinase (CK) [+44%], PFK (+106%), lactate dehydrogenase (LDH) [+45%], 3-hydroxyacyl coenzyme A (CoA) dehydrogenase (+60%) and CS (+38%) [all $p < 0.05$]. While participants did not show improvements in the 30-second sprint after only 1 day of rest (compared with their pre-training cycle sprint performance), a reassessment 5 days later revealed significant increases in $\dot{V}O_{2max}$ (+11.3%) and P_{peak} (+10.0%) obtained during the progressive exercise test ($p < 0.05$). Finally, Harmer et al.[60] have reported that sprint training (4 to 10 all-out cycle sprints, 3 to 4 minutes rest, 3 d/wk, 7 weeks) improves time to fatigue (+21%; $p < 0.001$) at 130% of the pre-training $\dot{V}O_{2max}$ workload. This increase in exercise capacity was attributed to reduced anaerobic ATP generation, and an increased contribution of aerobic metabolism to the energy yield.

There is some evidence to suggest that as the recovery time between repeated sprints declines, so does the contribution of glycolysis to the energy yield during subsequent sprints.[69] Consequently, aerobic metabolism increases to meet the energy deficit.[53,60] Linossier et al.[54] have suggested that aerobic metabolism during recovery from high-intensity exercise is important for the resynthesis of phosphocreatine and for the oxidation (i.e. removal) of lactic acid. It would appear, therefore, that intermittent high-intensity sprint training, that involves a significant contribution of energy derived from aerobic sources, improves the capacity for aerobic metabolism.[53,60]

From these studies in previously untrained individuals,[51,53,60] one significant advantage of HIT is the simultaneous up-regulation of both oxidative and glycolytic energy systems, producing an improved energy state in the working muscle through the preservation of high-energy phosphates.[11] As a final example to illustrate this point, Tabata et al.[58]compared the effects of HIT (8 × 20 seconds at 170% P_{peak}, 10 seconds rest, 5 d/wk for 6 weeks), and submaximal training (70% $\dot{V}O_{2max}$, 60 min/d, 5 d/wk) with two groups of active, but relatively untrained individuals (n = 7 per group; $\dot{V}O_{2max}$,

~50 ml/kg/min). While the submaximal exercise training group significantly increased their $\dot{V}O_{2max}$ (+9.4%; $p < 0.05$), there was no effect on their anaerobic capacity measured through maximal accumulated oxygen deficit.[70] However, those in the HIT group significantly improved both their $\dot{V}O_{2max}$ (+15%) and their anaerobic capacity (+28%) [$p < 0.05$].

In summary, HIT in sedentary and recreationally active individuals improves endurance performance to a greater extent than does continuous submaximal training alone. This improvement appears due, in part, to an up-regulated contribution of both aerobic and anaerobic metabolism to the energy demand,[51,53,55] which enhances the availability of ATP and improves the energy status in working muscle. An improved capacity for aerobic metabolism, as evidenced by an increased expression of type I fibres,[54] capillarisation and oxidative enzyme activity[53,61,62,71] is the most common response to HIT in untrained or moderately active individuals.

2. Endurance Training in Highly Trained Athletes

Many exercise scientists base their advice to athletes on training principles developed from studies completed with previously untrained or recreationally active individuals. As shown in the previous section, this is problematic; research has consistently shown that endurance training in previously untrained individuals will increase $\dot{V}O_{2max}$, capillary density, oxidative enzyme activity and plasma volume. However, changes in these variables do not occur when already highly trained athletes increase the volume of their submaximal training.[9,39] Indeed, endurance trained athletes and untrained individuals do not show the same response to submaximal (continuous) training.[8]

2.1 Submaximal Continuous Training

It appears that once an individual has reached a $\dot{V}O_{2max} > 60$ ml/kg/min, endurance performance is not improved by a further increase in submaximal training volume.[8] Indeed, Costill and associates[9] showed that when swim-training distance was more than doubled from 4 266 to 8 979 m/d over a 10-day period (while average training intensity was maintained), there was no change in swimming performance, aerobic capacity or CS activity in the deltoid muscle. While it might be argued that well trained athletes need more time for adaptations to occur than the time frame used in the former study, it should be noted that adaptations to a submaximal training stimulus are generally observed within this time period in untrained individuals.[6,16,20,72,73] In all likelihood, athletes in the trained state will have reached a plateau in the metabolic adaptations that result from submaximal endurance training. Evidence for this comes from a meta-analysis completed by Londeree.[8] This group compared training status (trained *vs* untrained) with the influence of continuous training at an exercise intensity corresponding to either the ventilatory threshold (T_{vent}) or T_{lac}. Although studies using previously untrained individuals have consistently shown a marked influence of training in terms of performance and associated physiological variables, analysis by Londeree et al. [8] showed that continuous training failed to elicit further improvements in already highly trained athletes. The authors did note, however, that trained individuals tended to respond better to higher intensity training.

2.2 HIT

Generally, training programmes undertaken by highly trained endurance athletes consist of an early 'aerobic base' component, complemented by HIT sessions nearer to the competitive season. Despite the fact that coaches have long used HIT to improve the performance of their elite endurance athletes,[1] exercise scientists have only recently sought to understand the physiological mechanisms behind the practice.[7,74-84] Despite this increased attention, the mechanisms responsible for these improvements in endurance performance following HIT remain unclear.[7,80]

2.2.1 Quantifying the Demands of HIT in Highly Trained Athletes

Recent work has examined the short-term[85] and long-term[82]influences of HIT in highly trained cyclists. Stepto and colleagues[85] investigated the metabolic demands of a single session of HIT [8 × 5 minutes at 86% peak oxygen uptake ($\dot{V}O_{2peak}$), 60 seconds recovery] in seven highly trained cyclists ($\dot{V}O_{2peak}$ = 5.14 L/min). These cyclists showed high rates of carbohydrate oxidation (340 μmol/kg/min), coupled with a progressive increase in fat oxidation (16 to 25 μmol/kg/min) measured throughout the HIT session. Laursen and colleagues[82] recently reported changes in cardiorespiratory and performance variables following four HIT sessions (20 × 60 seconds at P_{peak}, 2 minutes recovery) over 2 weeks in seven highly trained cyclists ($\dot{V}O_{2peak}$ = 68.7 ± 1.3 ml/kg/min). These individuals were able to perform a greater number of HIT bouts and complete more total work following training. The improved HIT performance was accompanied by reductions in both the respiratory exchange ratio and 1-minute recovery heart rates from the first to the fourth HIT session ($p < 0.05$); T_{vent} and P_{peak} obtained during the progressive exercise test also improved as a result of the four HIT sessions ($p < 0.05$).

2.2.2 The Influence of HIT on Performance and Related Variables

HIT has been shown to improve 3km (+3%),[86,87] and 10km (+3%)[74] running performance in middle- and long-distance runners, as well as 40km time-trial performance in endurance-trained cyclists (+2.1 to 4.5%) [$p < 0.05$].[7,80,81,83] Well established laboratory-based markers of endurance performance have also changed following HIT. These include T_{vent},[74,82] and the P_{peak} obtained during a progressive exercise test ($p < 0.05$).[7,80-83] However, improvements in economy of motion[10,75,88] and $\dot{V}O_{2max}$ have not been observed.[74,75,82] It should be mentioned however that most of these studies did not involve control individuals.[7,74,75,80,81,83,88] Indeed, improvements in performance-related variables may, at least in part, be attributed to 'psychological factors' that are naturally inherent with training studies (i.e. 'the last test'). Some studies have shown improvements in P_{peak}, time to exhaustion and time-trial performance, merely as a result of it being the last of a series of tests ($p < 0.05$).[89,90]

The following section will examine studies that have investigated potential mechanisms responsible for an improved endurance performance following HIT in the already highly trained athlete.

Potential Mechanisms to Improved Performance

Insight into the potential ways in which HIT may benefit endurance performance is possible through examining the particular physiological parameters that have been identified as being important in endurance events. $\dot{V}O_{2max}$, the sustainable fractional utilisation of $\dot{V}O_{2max}$, and economy of motion all contribute to endurance performance.[4] Included in the following section is a review of the processes that regulate these variables and determine the delivery of oxygen to working muscles and facilitate the utilisation of oxygen by working muscles.[11]

Central Adaptations

Central adaptations to endurance training facilitate improved delivery of oxygen to working muscles. Given that maximal heart rate remains unchanged in response to endurance training,[91] improvements in oxygen delivery to exercising muscles during high-intensity exercise can be attributed to an increase in stroke volume.[21] Stroke volume can increase through a higher left-ventricular contractile force and/or through an increase in cardiac filling pressure, which raises end-diastolic volume and resultant stroke volume.[21] Surprisingly, potential changes in stroke volume and plasma volume in response to HIT in the already highly trained athlete have, to our knowledge, not been examined. However, if stroke volume does increase following HIT in the highly trained athlete, it may be difficult to detect, as $\dot{V}O_{2max}$, which is strongly related to maximal cardiac output,[16] has rarely been shown to change following HIT in the highly trained athlete.[86]

The increase in plasma volume, caused by either training or heat acclimation, has been regarded as

the single most important event in promoting cardiovascular stability and improving thermoregulation during prolonged exercise.[92] Hypervolaemia serves to minimise cardiovascular stress by preventing significant reductions in mean arterial pressure, central venous pressure, and cardiac filling,[93] thereby maintaining or improving stroke volume.[94] Plasma volume expansion through either training or heat acclimation has been attributed to elevated plasma renin levels, vasopressin and plasma albumin content, which facilitates water and sodium retention in the blood.[95] Artificial plasma volume expansion (i.e. 200 to 300ml above normal) has been reported to increase both $\dot{V}O_{2max}$ and exercise time to fatigue by 4 and 11% ($p < 0.05$), respectively, in untrained individuals despite a 4% reduction in haemoglobin concentration.[94] However, artificial plasma volume expansion does not appear to significantly enhance stroke volume in highly trained individuals, who already have a relatively high plasma volume.[96]

One other potential mechanism that might be partially responsible for enhanced endurance performance following HIT in the highly trained athlete is an improvement in heat tolerance via an augmented cutaneous blood flow and/or sweating rate.[97] Although experimental HIT sessions are normally completed under controlled thermoneutral environments, high-intensity exercise produces high core temperatures (~40°C),[98] and endurance training itself has been shown to independently expand plasma volume, creating partial heat acclimatisation.[99]Because a strong association has been established between volitional fatigue and elevated core temperatures,[100,101] it is possible that highly trained athletes may adapt somewhat to successive HIT sessions by means of improved temperature regulation.[102] Indeed, HIT can elicit improved work-heat tolerance in physically active individuals,[103] but this has yet to be investigated in highly trained athletes. The fact that endurance trained athletes have an enhanced capacity for sweating and cutaneous blood flow supports this as a possible adaptive response to HIT.[22]

Peripheral Adaptations

Peripheral adaptations to exercise training refer to an improved ability of working muscle to produce and utilise ATP. The integration of the metabolic pathways, which serve to resynthesise ATP and the excitation-contraction processes utilising ATP, determine this efficiency.[11] Due to the absence of data relating to peripheral changes following training in already highly trained athletes, understanding of this area has been limited to studies which have used untrained and recreationally active individuals.

Some authors[45,104] have assumed that further adaptations in the already highly trained athlete in response to continued training originate from the same well-established adaptations known to occur with untrained and recreationally active individuals. In particular, endurance training promotes an improved energy state in the working muscle as indicated by better protection of the high-energy phosphate potential.[11] Increases in both oxidative and glycolytic enzyme activity coupled with an improved exercise capacity have been shown to occur in untrained and recreationally active individuals following HIT.[51-54] To our knowledge, however, only one published study to date[7] has examined the underlying metabolic adaptations responsible for improved endurance performance following HIT in already highly trained athletes. Weston and associates[7] found that the activities of HK, PFK, CS and 3-hydroxyacyl-CoA dehydrogenase did not change with HIT. Nevertheless, there was a significant improvement in 40km time-trial performance, P_{peak}, and time to fatigue at 150% P_{peak} in these six highly trained cyclists following six HIT sessions over 3 weeks ($p < 0.05$; **table II**). Therefore, factors other than the activities of the enzymes measured by the authors must have contributed to the observed improvements in performance. Despite the findings of Weston and associates,[7] Billat[104] contends that HIT may promote a greater use of fatty acids, even in the highly trained athlete. In support of this possibility, Shepley and co-workers[105] while examining the effects of tapering (i.e. reduction in volume before an endurance contest) on endurance performance

Table II. Summary of findings in high-intensity interval-training (HIT) studies in highly trained cyclists [a]

Reference	n	HIT sessions	Reps	Intensity (% P_{peak})	Work duration	Rest duration	HIT duration (wk)	Results
Lindsay et al.[83]	8	6	6-8	80	5 min	60 sec	4	$\uparrow P_{peak}$, $\uparrow TF_{150}$, $\uparrow TT_{40}$
Weston et al.[7]	6	6	6-8	80	5 min	60 sec	4	$\uparrow P_{peak}$, $\uparrow TF_{150}$, $\uparrow TT_{40}$, $\uparrow \beta$, $\leftrightarrow$HK, $\leftrightarrow$PFK, $\leftrightarrow$CS, $\leftrightarrow$3-HCoA
Westgarth-Taylor et al.[80]	8	12	6-9	80	5 min	60 sec	6	$\uparrow P_{peak}$, $\uparrow TT_{40}$, $\downarrow CHO_{OX}$ **((Author: please define))**
Stepto et al.[81]	4	6	4	80	8 min	1 min	3	No change **((Author: $\leftrightarrow$?))**
Stepto et al.[81]	4	6	8	85	4 min	1.5 min	3	$\uparrow P_{peak}$, $\uparrow TT_{40}$
Stepto et al.[81]	4	6	12	90	2 min	3 min	3	No change **((Author: $\leftrightarrow$?))**
Laursen et al.[82]	7	4	20	100	1 min	2 min	2	$\uparrow P_{peak}$, $\uparrow T_{vent}$ **((Author: ventilatory threshold?))**, $\uparrow TF_{100}$,,
Stepto et al.[81]	3	6	12	100	1 min	4 min	3	No change **((Author: $\leftrightarrow$?))**
Stepto et al.[81]	4	6	12	175	30 sec	4.5 min	3	$\uparrow P_{peak}$, $\uparrow TT_{40}$

a Changes indicated based on statistical significance at the $p < 0.05$ level.

CS = citrate synthase activity; **HK** = hexokinase activity; **n** = number of participants; **PFK** = phosphofructokinase activity; **P_{peak}** = peak aerobic power output; **Reps** = repetitions; **TF_{100}** = time to fatigue at 100%; **TF_{150}** = time to fatigue at 150% of P_{peak}; **TT_{40}** = 40km time-trial performance; **3-HCoA** = 3-hydroxyacyl coenzyme A dehydrogenase activity; β = buffering capacity; $\downarrow$ = **((Author: decrease??))**; $\uparrow$ = **((Author: increase?))**; $\leftrightarrow$ = **((Author: no change?))**.

and CS activity in highly trained middle distance runners, showed that a high-intensity taper (3 to 5 × 500m at 120% $\dot{V}O_{2peak}$, 800m jog recovery, five times per week) improved run time to exhaustion at ~115% $\dot{V}O_{2peak}$ (+22%) and CS activity (+18%), compared with low-intensity taper and no taper scenarios ($p < 0.05$). Interestingly, the high-intensity taper programme consisted of training intensities that were greater than those typically completed by the athletes during their normal training programme .[105] This suggests that an increase in training intensity may enhance oxidative enzyme activity, even in highly trained athletes. Moreover, some authors have reported lower respiratory exchange ratio values at submaximal workloads following HIT.[80,82] Thus, further examination of potential changes in oxidative enzyme activity as a result of HIT in the already highly trained athlete is needed.

Increased glycogenolytic capacity is another avenue through which endurance performance could be improved. However, while the simultaneous increase in both aerobic and anaerobic capacity has been documented in untrained individuals following HIT,[58,106] Weston et al.[7] showed that with already highly trained athletes, the activities of HK and PFK remained unchanged following HIT. Considering that highly-trained athletes already have high muscle glycolytic enzyme activities,[107] it is possible that the intervals used by Weston et al.[7] were performed at too low an intensity (85% of P_{peak}) for adaptations to the glycolytic pathway to occur. In addition to potential changes to key glycolytic enzyme activity, there are other peripheral mechanisms that could contribute to an improved performance in the highly trained athlete following HIT. These include the capacity of skeletal muscle to buffer H^+ ions, and an up- or down-regulation of muscle cation pumps.

The capacity of working muscle to buffer H^+ ions is related to sprint performance in untrained[108] and highly trained individuals.[7] Moreover, sprint training has been shown to improve skeletal muscle buffering capacity in untrained individuals[109] and in already highly trained athletes.[7] Weston et al.[7] reported a significant increase in skeletal muscle buffering capacity following only 3 weeks of HIT ($p < 0.05$). They

also found a significant relationship between 40km time-trial performance and skeletal muscle buffering capacity in their six highly trained cyclists ($r = 0.82$; $p < 0.05$). The findings of these workers[7] suggest that improvements in endurance performance following HIT may be related to an increased ability to buffer H^+ ions. The findings of enhanced 30km time-trial performance in moderately trained cyclists ($\dot{V}O_{2max}$ = 54.7 ± 1.7 ml/kg/min) following sodium citrate consumption also supports this premise.[110]

More evidence for skeletal muscle buffering capacity as a key regulatory mechanism in the highly trained athlete arises from the findings of Stepto and colleagues.[81] These workers used trend analysis to examine the effects of different HIT programmes on the rate of 40km time-trial performance improvements in already highly trained cyclists. They found that improvements in P_{peak} and 40km time-trial performance arose from two distinctly different HIT programmes. One of these programmes (8 × 4 minutes at 85% P_{peak}, 1 minute recovery) has been shown to consistently produce performance improvements in highly trained cyclists (table II).[7,80,81,83] However, similar improvements in performance have also resulted from repeated 'supramaximal' HIT (12 × 30 seconds at 175% P_{peak}, 4.5 minutes recovery). Although skeletal muscle buffering capacity was not measured, the improved performance following supramaximal HIT may well have been accompanied by an increase in muscle buffering capacity, as has been shown following repeated supramaximal sprint training in untrained individuals.[109]

A high concentration of H^+ ions has a known inhibitory effect on enzyme activity, including PFK.[111] Thus, improved skeletal muscle buffering capacity could indirectly contribute to an improved glycolytic ATP yield and higher exercise intensity by improving the activity of PFK. Although, further examination of this mechanism is required, endogenous skeletal muscle buffering capacity remains a prospective mechanism to the improvement in performance found in highly trained athletes following HIT.

Another possible mechanism that may contribute to improved endurance performance following HIT in already highly trained individuals is altered expression of Na^+-K^+-ATPase and sarcoplasmic reticulum Ca^{2+}-ATPase. These enzymes are responsible for regulating the activity of pumps involved in cation transport, which in turn maintain muscle membrane potential.[112] Resistance training,[113] endurance training[114] and altitude acclimatisation[115,116] have all been shown to alter the levels of these enzymes. Improved submaximal cycling efficiency was recently shown to be related to a down-regulation in Na^+-K^+-ATPase pump density in well trained mountain climbers following prolonged exercise at high altitude.[115,117,118] A similar response could be associated with HIT in the already highly trained athlete. Given that highly trained athletes can become hypoxaemic during exercise at high intensities,[85,119] and since hypoxaemia appears to be a stimulus for alteration in Na^+-K^+-ATPase pump density,[114,115] further research needs to examine the possibility that HIT training evokes an altered expression of cation pumps in already highly trained athletes.

Other factors that may contribute to the enhanced endurance performance of the highly trained athlete following HIT include biomechanical changes, adaptation of the central nervous and endocrine systems, as well as other peripheral changes such as increases in myoglobin, capillary density and fibre type characteristics. Biomechanical changes could improve exercise efficiency following HIT. However, Lake and Cavanagh[10] investigated the effects of 6 weeks HIT on various biomechanical variables in a group of moderately trained runners ($\dot{V}O_{2max}$ = 57.7 ± 6.2 ml/kg/min), and found no relationship between changes in performance, $\dot{V}O_{2max}$, running economy and biomechanical variables. The authors concluded that improvements in performance following HIT were more likely to be caused by physiological rather than biomechanical factors.[10]

The effect of HIT on the central nervous and endocrine systems has not yet been examined in highly trained athletes. In untrained individuals,

muscle sympathetic nerve activity following exercise training appears attenuated during exercise,[59] suggesting a reduced sympathetic outflow at a given submaximal workload. However, the capacity for noradrenaline (norepinephrine) release during a progressive exercise test appears superior following HIT.[57]

Myoglobin stores, which represent ~10% of the accumulated oxygen deficit,[120] have been reported to increase,[121,122] decrease[123] and remain unchanged[124] following endurance training in untrained individuals. Myoglobin stores, which are yet to be examined following HIT in already highly trained athletes, may be related to the enhanced oxygen uptake ($\dot{V}O_2$) witnessed during HIT;[104,125] the reloading of myoglobin stores during recovery phases could increase oxygen availability during subsequent interval bouts.[126] This mechanism could, in part, explain our observation that highly trained athletes can complete a greater number of high-intensity intervals following successive HIT sessions.[82] Myoglobin levels have been shown to increase in response to a hypoxic stress.[11,127] Considering that athletes become hypoxaemic during high-intensity exercise,[85,119] an examination of muscle myoglobin levels before and after HIT in the highly trained athlete warrants consideration.

An increased expression of type I fibres has been reported following multiple sprint training in untrained individuals.[54] Type I fibres may play an important role during the recovery phase of HIT for the resynthesis of phosphocreatine and for the removal (oxidation) of lactic acid. It is questionable, however, whether the expression of type I fibres would be altered following HIT in the highly trained athlete, as highly trained athletes already have a high proportion of type I fibres.[107]

A large number of capillaries and a high capillary to fibre area ratio are characteristic of highly trained skeletal muscle.[107,128,129] It is therefore unlikely that further enhancement of capillary density could occur following HIT in already highly trained athletes. Interestingly, in a group of highly trained female cyclists, Bishop and co-workers[130]recently reported a negative correlation ($r = -0.77$, $p < 0.01$) between the diameter of the type II fibres and 1-hour cycling performance. The authors suggested that a reduction in type II fibre size might allow for an increase in capillary density and improve lactate removal.

2.2.3 Enhanced Physiological Efficiency: An Issue of Practical Versus Statistical Significance?

The highly trained athlete already has a high aerobic capacity,[4] and a high degree of adaptation in a number of physiological variables associated with oxygen delivery and utilisation.[9,107,128] Moreover, improvements in endurance performance following HIT, although statistically significant, have been relatively small (2 to 4%).[7,74,80,81,83] One issue is that while these improvements in performance are extremely important to an elite athlete, they may be too small to statistically detect and explain.

In summary, HIT, but not continuous submaximal training, elicits significant enhancements in endurance performance. These performance improvements have been shown to parallel the enhancements in T_{vent} and P_{peak}, but generally not $\dot{V}O_{2max}$ or economy of motion. Very little research has examined the adaptation of central and peripheral factors following HIT in highly trained athletes. However, in the only study to analyse muscle tissue following HIT, there was no evidence of an up-regulation in the glycolytic and oxidative enzyme activity. Instead, this study revealed that an improved skeletal muscle buffering capacity may play an important role in the enhancement of endurance performance following HIT. Other mechanisms warranting future examination following HIT in the highly trained athlete include the expression of muscle cation pumps, neuromuscular and endocrinological adaptations, as well as the adjustment of myoglobin levels, capillary density, and fibre type expression.

3. HIT Programme Optimisation

Very little information is available concerning HIT programme optimisation in highly trained endurance athletes. Optimisation in the current con-

text refers to the optimal exercise intensity, exercise duration and number of interval bouts, in addition to the type (active*vs* passive) and duration of the recovery between exercise bouts. These variables require manipulation according to the periodisation phase of annual training programmes,[1] training status and the individual response that an athlete has to a training stimulus. The last major section of this review will focus on how best to use HIT in the preparation of trained athletes for competition.

Research has generated a wide range of variables for use in prescribing exercise intensities to individuals undertaking endurance training. Some of these include $\dot{V}O_{2max}$,[78,131,132] anaerobic threshold,[133,134] T_{lac},[56,135,136] T_{vent},[4,137,138] onset of blood lactate accumulation (OBLA)[139-141] and critical power.[142-149] However, the physiological significance, feasibility and rationale for using such measures to establish suitable and effective exercise intensities have been questioned.[150] A variable that has been used with reasonable success in runners is the velocity at which $\dot{V}O_{2max}$ is achieved (V_{max}),[66,78,125,132,150-157] defined as the running speed during an incremental test at which $\dot{V}O_{2max}$ is attained.[155]

3.1 Significance of the Time to Exhaustion at the Velocity at Which $\dot{V}O_{2max}$ is achieved (V_{max}) (T_{max}) in Highly Trained Runners

V_{max} has been shown to predict performance in middle- and long-distance running events,[79,131,158,159] and appears useful for prescribing HIT programmes.[78,86,125,160] The rationale for using V_{max} in HIT programme prescription is based on the assumption that further improvements $\dot{V}O_{2max}$ in the highly trained athlete will only result from exercise training at or above $\dot{V}O_{2max}$. Moreover, V_{max} may be the lowest velocity at which $\dot{V}O_{2max}$ is elicited.[104,106,125,155] The basis for this premise is that the onset of muscular fatigue during high-intensity exercise performed near $\dot{V}O_{2max}$ is dependent on oxygen delivery to the sarcolemma.[161] Although most studies employing HIT have not sampled $\dot{V}O_2$ during the training intervention, Billat et al.[125] recently showed that repeated bouts of intermittent running (30 seconds at 100% V_{max}, 30 seconds 50% V_{max}) enabled runners to maintain $\dot{V}O_{2max}$ from the 5th to the 18th repetition (~10 minutes). This is nearly three times longer than $\dot{V}O_{2max}$ can be sustained during a single timed-to-exhaustion bout at V_{max} ($p < 0.05$).[66,125]

If one accepts that V_{max} is an appropriate exercise intensity to use in HIT programming, then what remains is to decide on the optimal exercise duration for each bout of exercise. Because the time that an athlete can run at his or her V_{max} [the time to exhaustion at V_{max} (T_{max})] is highly subjective, even amongst runners with the same V_{max},[66,152,153] the fractional utilisation of T_{max} emerges as an appropriate marker for establishing interval duration.

3.1.1 Use of T_{max} to Prescribe HIT Sessions

Despite wide variation in times between individuals with similar $\dot{V}O_{2max}$ values (coefficient of variation = 25%), Billat et al.[153] have demonstrated the reproducibility of T_{max} in sub-elite runners (404 ± 101 seconds *vs* 402 ± 113 seconds; $r = 0.86$; $p < 0.05$). T_{max} has been shown to correlate negatively with $\dot{V}O_{2max}$ [154,162] and V_{max},[66,154] and positively with the anaerobic threshold.[132,152-154]

Hill and Rowell[150] found that the minimal time it took for $\dot{V}O_{2max}$ to be reached was 60% of T_{max} in a group of highly trained female middle-distance runners. Consequently, 60% of T_{max} has been accepted as an optimal interval duration for use in studies examining the effects of HIT at V_{max} (**table III**). In a recent study, Smith and colleagues[86] trained five middle-distance runners twice a week for 4 weeks (8 × 60 to 75% of T_{max}, 2 : 1 work to rest ratio) and found that mean V_{max}, T_{max}, and 3000m-run performance increased ($p < 0.05$). In further work, the same group[87] used a larger cohort of well-trained runners (n = 26) that were randomly assigned into either a control group, or one of two HIT groups (V_{max} intervals at 60 or 70% of T_{max}; 1 : 2 work to rest ratio). Both T_{vent} and

Table III. Summary of findings of high-intensity interval-training (HIT) studies in highly-trained runners ($\dot{V}O_{2max}$ > 60 ml/kg/min) [a]

Reference	n	HIT sessions	Reps	Intensity	Work duration	Rest duration	HIT Duration (wk)	Results
Acevedo and Goldfarb[74]	7	24	NR	90-95% HR_{max}	NR	NR	8	↓10km time, ↑T_{vent}, ↔$\dot{V}O_{2max}$
Zavorsky et al.[88]	12	3	10	96% V_{max}	NR	60-180 sec	2	↑RE, ↓RER
Smith et al.[86]	5	8	5	V_{max}	60-75% T_{max}	1 : 2	4	↑$\dot{V}O_{2max}$, ↑V_{max}, ↑T_{max}, ↓3000m time
Billat et al.[78]	8	4	5	V_{max}	50% T_{max}	1 : 1	4	↑$_v\dot{V}O_{2max}$, ↑RE, ↔$\dot{V}O_{2max}$, ↔T_{max}
Smith et al.[87]	9	8	8	V_{max}	60% T_{max}	1 : 2	4	↑T_{max}, ↓ 3000m time, ↔$\dot{V}O_{2max}$, ↔$_v\dot{V}O_{2max}$
Smith et al.[87]	9	8	8	V_{max}	70% T_{max}	1 : 2	4	↔T_{max}, ↔ 3000m time, ↔$\dot{V}O_{2max}$, ↔V_{max}

a Changes indicated based on statistical significance at the $p < 0.05$ level.

HR_{max} = maximum heart rate; **n** = number of participants; **NR** = not reported; **RE** = running economy; **Reps** = repetitions; **RER** = respiratory exchange ratio; **T_{max}** = time to exhaustion while running at V_{max}; **T_{vent}** = ventilatory threshold; **V_{max}** = running speed at $\dot{V}O_{2max}$; **$\dot{V}O_{2max}$** = maximal oxygen consumption; **v $\dot{V}O_{2max}$ = ((Author: please define. Do you mean V_{max} ?))**; ↓ = **((Author: decrease?))**; ↑ = **((Author: increase?))**; ↔ = **((Author: no change?))**.

3000m-running performance improved significantly more in the 60% T_{max} group than in either the 70% T_{max} or control group ($p < 0.05$). However, despite randomisation, runners performing 60% T_{max} intervals had slower overall 3000m-run times to begin with, and this disparity may have biased the results. Thus, it cannot be unequivocally stated that intervals performed at 60% T_{max} are the optimal HIT programme duration for improving endurance performance. Indeed, Billat et al.[78] examined the effects of 4 weeks of HIT in highly trained runners ($\dot{V}O_{2max}$ = 71.6 ± 4.8 ml/kg/min), using only one session per week at 50% of T_{max}, and found that V_{max} (+5%) and running economy (–6.1%) significantly improved, despite there being no change in T_{max} or $\dot{V}O_{2max}$. Although further work is required in terms of HIT optimisation, preliminary data suggest that HIT performed somewhere between 50 to 60% of T_{max} may be optimal for improving endurance performance.

Another variable that some researchers have suggested to be an important component for enhancing endurance performance is the distance run at V_{max} during a given HIT session.[104,163] Accordingly, Billat et al.[160] reported that 16 highly trained male runners ($\dot{V}O_{2max}$ = 69.1 ± 4.3 ml/kg/min) were able to run 2.5 times their T_{max} distance during a HIT workout using a 1 : 1 work : rest ratio at 50% T_{max}, with recovery between bouts approximating 60% V_{max}.[104]

Longer HIT performed at an intensity between T_{lac} and V_{max} (also known as critical velocity[164]) has the potential to increase $\dot{V}O_2$ to the level of $\dot{V}O_{2max}$ as a result of the $\dot{V}O_2$ slow component phenomenon.[165,166] Poole and Gaesser[136] have stated that the critical velocity may be the threshold intensity for eliciting $\dot{V}O_{2max}$. However, Billat et al.[167] reported that 14 highly trained runners ($\dot{V}O_{2max}$ = 74.9 ± 3.0 ml/kg/min) reached steady-state $\dot{V}O_2$ at 93% of $\dot{V}O_{2max}$ during a time to exhaustion test (~17 minutes) at 90% of V_{max}. Indeed, endurance training has also been shown to reduce the magnitude of the $\dot{V}O_2$ slow component.[168,169] Thus, although critical velocity/critical power may be an appropriate exercise intensity for use with moderately trained individuals,[170-172] a more demanding exercise intensity is needed for use with elite athletes.[104]

3.2 Cycling Studies

Research with cyclists has taken a more conventional approach to HIT programme optimisation, but as is the case with runners, scientific data are sparse (table II). In a heterogeneous group of previously trained, but not highly trained cyclists ($\dot{V}O_{2max}$ = 56.8 ± 6.6 ml/kg/min), Norris and

Petersen[173] reported increases in $\dot{V}O_{2max}$ (+7%), T_{vent} (+16%), and 40km time-trial performance (+8%) after 8 weeks of HIT at T_{vent} heart rate ($p < 0.05$). Thus, improvements in aerobic capacity and T_{vent} can occur following HIT in previously or moderately trained athletes. Only one study to date, however, has attempted to examine HIT programme optimisation in highly trained cyclists. In this study, Stepto and colleagues[81] investigated the effects of five different HIT programmes, performed twice per week for 3 consecutive weeks, on the rate of performance improvements in twenty endurance-trained cyclists. The authors found that performance (40km time trial and P_{peak}) improvements resulted from two markedly different HIT programmes. One of these was a commonly used HIT programme of 'aerobic' type intervals (8×4 minutes at 85% P_{peak}, 90 seconds recovery) that has previously been shown to improve endurance performance.[7,80,81,83] However, a comparable improvement in performance resulted from intermittent supramaximal training (12×30 seconds at 175% P_{peak}, 4.5 minutes recovery). The other HIT programmes (table II) failed to significantly improve endurance performance. That intermittent supramaximal training improved 40km time-trial performance is intriguing, as training of this nature has not previously been associated with improvements in endurance performance. Thus, because the mechanisms remain unknown, repeated sprint training may be more important to the endurance athlete than was previously thought. Further research into HIT programme optimisation is required with cyclists, including the use of those concepts that have shown success in highly trained runners; namely the prescription of a HIT programme using T_{max}.

3.2.1 T_{max} During Cycle Ergometry

As reviewed earlier(**(Author: please specify in which section)**), although T_{max} has been developed as a practical method for determining the appropriate length of the interval bout at V_{max} in highly trained runners,[86] HIT programme prescription using this method has yet to be applied to cycling. Use of the fractional utilisation of T_{max} for HIT prescription in cyclists is possible; Billat et al.[174] determined T_{max} in nine elite cyclists to be 222 ± 91 seconds, which was not significantly different to that of elite runners (321 ± 84 seconds). Future research could utilise multisport athletes (triathletes and duathletes) to determine whether or not true statistical differences in T_{max} exist between exercise modes.

In brief, HIT prescription with highly trained runners has been reasonably successful when V_{max} is used to establish the intensity, and 50 to 60% of T_{max} is used for the exercise duration. Despite the feasibility of using T_{max} for prescribing HIT programmes in cyclists, longitudinal studies in cyclists have used more conventional programmes, revealing that supramaximal sprinting may be a more effective means of endurance performance enhancement than previously thought.

3.3 Rate of Performance Enhancement Following HIT

Very little information is available concerning the rate at which endurance performance improves following a given HIT stimulus. This is probably due, at least in part, to the challenging nature of carrying out research of a longitudinal repeated-measure design. Lindsay et al.[83] showed, with eight endurance-trained cyclists, that HIT elicited no change in P_{peak} or 40km time-trial performance after 2 weeks, but there was an increase in both P_{peak} (+4.3%; $p = 0.01$) and 40km time-trial performance (+3.5%; $p < 0.001$) after 4 weeks. Interestingly, a similar time course of change in these variables has been shown in another study using the same HIT programme repeated over 6 weeks,[80] suggesting that regular assessments of training status and subsequent adjustments to HIT programmes are required to maximise improvements in endurance performance. Indeed, Laursen and co-workers[82]have recently shown that the increases in $T\text{-}_{vent}$ (+22%) and P_{peak} (+4.3%) in highly trained cyclists are possible following just four HIT sessions (20×60 seconds at P_{peak}, 120 seconds recovery) over 2 weeks during the off-season ($p < 0.05$).

In well trained runners, a single HIT session has failed to elicit changes in $\dot{V}O_{2max}$ and $T\text{-}_{max}$,[162] or running economy in elite long-distance runners.[159] In contrast, a recent study in untrained individuals has shown that skeletal muscle adaptations begin to occur after just one 16-hour training session involving cycling for 6 minutes each hour at ~90% $\dot{V}O_{2max}$.[46] In this study, muscle was biopsied from the vastus lateralis during a two-stage (2 × 20 minutes) standardised submaximal cycle protocol before and 36 to 48 hours after the HIT session. Analysis revealed an attenuated decline in phosphocreatine and glycogen use, as well as a smaller rise in muscle lactate after the training. There was no effect, however, on the maximal activities of CS and malate dehydrogenase, suggesting that adjustments in high-energy phosphates are an early adaptive event that occur before increases in oxidative potential following endurance exercise training, at least in untrained individuals. Future research is required to describe the time course of adaptations resulting from HIT with highly trained athletes.

3.4 Recovery Considerations

The importance of recovery following a HIT session has been demonstrated in untrained individuals. Balsom et al.[175] showed that the distance of repeated sprints (15, 30, 40m; 30 seconds recovery) was positively related to a reduction in sprint performance and an associated decline in the adenine nucleotide pool. Furthermore, Rodas et al.[51] found significant increases in both oxidative and glycolytic enzyme activities after 2 weeks of supramaximal cycle sprint training (table I), but after only one rest day there was no change in 30 seconds all-out performance. Nevertheless, marked increases were found in $\dot{V}O_{2max}$ (+11.3%) and P_{peak} (+10%) measured 5 days later ($p < 0.05$), suggesting that fatigue or overtraining may have played a role in preventing a significant improvement in the 30 seconds all-out test. In another study by the same authors,[52] the aforementioned training group was compared with a matched group of individuals completing the same training programme, except that a 2-day rest period separated each HIT session in the latter group. This lengthened the entire training programme to 6 weeks. While muscle enzyme activities were not significantly increased in this study, performance in the 30 seconds all-out test was significantly improved ($p < 0.05$). A comparison of these studies suggests that muscle fibres experience fatigue or injury following HIT, indicating that sufficient recovery following the final training session is necessary for the benefits of training to be detected.

Unfortunately, very little information is available concerning the optimal recovery duration between HIT bouts. Generally, coaches and researchers have used fixed work-recovery ratios (i.e. 2 : 1, 1 : 1, 1 : 2),[78,85,86] or recovery durations based on heart rate returning to a fixed percentage of its maximum.[55,74] A recent study[106] compared the contribution of aerobic and anaerobic metabolism of different HIT programmes in active ($\dot{V}O_{2max}$ = 57 ± 6 ml/kg/min) but not highly trained individuals, using maximal accumulated oxygen deficit as a measure of anaerobic capacity.[70] A short recovery HIT protocol (6 to 7 × 20 seconds at 170% $\dot{V}O_{2max}$, 10 seconds recovery) resulted in a higher accumulated oxygen deficit and $\dot{V}O_2$ than a long recovery HIT protocol (4 to 5 × 30 seconds at ~200% $\dot{V}O_{2max}$, 2 minutes recovery), suggesting that supramaximal HIT with short recovery periods may maximally tax aerobic and anaerobic capacities. One apparent limitation to a shortened recovery in untrained individuals, is that a reduced number of intervals might be completed and thus less work achieved.[175] However, evidence for this was not found in a recent study by Zavorsky et al.[88] In this study, 12 highly trained runners (VO_{2max} = 72.5 ± 4.3 ml/kg/min) were assigned to different HIT recovery groups (duration = 1, 2 or 3 minutes) following three HIT sessions (10 × 400m running) run at 4% below V_{max} over 2 weeks. While there was an overall increase in running economy when the group was considered as a whole, no differences were found between the groups. Thus, the optimal recovery duration between HIT bouts is yet to be determined.

The importance of active versus passive recovery bouts following HIT work bouts has recently been addressed.[104] Because high lactate levels develop during interval training performed at an intensity greater than T_{lac}, active recovery facilitates lactate removal[176,177] and allows athletes to tolerate heavy work rates for longer periods.[104,125] The use of active recovery in the prescription of HIT programmes therefore appears justified. Interestingly, training status seems to be unrelated to the decline in plasma lactate during passive recovery from exercise at equivalent relative maximal work intensities.[178]

In athletes, the importance of a taper following a phase of increased training volume and intensity appears essential.[105,179] Most recently, Mujika et al.[180] examined the physiological and performance responses to different 6-day tapers in eight well-trained male middle-distance runners. In this study, runners completed 15 weeks of their regular training programme, and were then assigned to either a moderate (50% reduction in training volume and intensity) or a low volume taper (75% reduction in training volume and intensity). The type of taper showed no effect on either 800m-run performance or changes in haematological status, suggesting that middle-distance runners can progressively reduce their usual training volume by at least 75% during a 6-day taper.

4. Conclusion

Considerable information is available relating to the physiological responses that result from submaximal training and HIT in untrained individuals. In contrast, very little is known of how already highly trained athletes respond to a modified training programme. However, it does not appear that additional submaximal endurance training volume improves endurance performance or related physiological variables in this particular population.[9] In contrast, HIT, in many forms, can elicit significant improvements in endurance performance in already highly trained athletes.[81,82,86] To date, however, researchers have been unsuccessful in explaining the reasons for this improvement.[7,74,80] Further investigation into the response of central and peripheral factors to HIT in the highly trained athlete is therefore warranted. Finally, coaches and athletes are in need of more knowledge concerning HIT programme optimisation; the optimal HIT programme intensity, duration and recovery that elicit the greatest rate of improvement in endurance performance are yet to be reported.

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