

What is Best Practice for Training Intensity and Duration Distribution in Endurance Athletes?

Stephen Seiler

Successful endurance training involves the manipulation of training intensity, duration, and frequency, with the implicit goals of maximizing performance, minimizing risk of negative training outcomes, and timing peak fitness and performances to be achieved when they matter most. Numerous descriptive studies of the training characteristics of nationally or internationally competitive endurance athletes training 10 to 13 times per week seem to converge on a typical intensity distribution in which about 80% of training sessions are performed at low intensity (2 mM blood lactate), with about 20% dominated by periods of high-intensity work, such as interval training at approx. 90% $\dot{V}O_{2\max}$. Endurance athletes appear to self-organize toward a high-volume training approach with careful application of high-intensity training incorporated throughout the training cycle. Training intensification studies performed on already well-trained athletes do not provide any convincing evidence that a greater emphasis on high-intensity interval training in this highly trained athlete population gives long-term performance gains. The predominance of low-intensity, long-duration training, in combination with fewer, highly intensive bouts may be complementary in terms of optimizing adaptive signaling and technical mastery at an acceptable level of stress.

Keywords: elite athletes, training organization, $\dot{V}O_{2\max}$, lactate threshold, interval training

Endurance training involves manipulation of intensity, duration, and frequency of training sessions over days, weeks, and months. *Long slow distance*, *lactate threshold training*, and *high-intensity interval training* (HIT) are all familiar terms for exercising within different regions on the intensity scale. The relative impact of different combinations of intensity and duration of endurance training has been studied and debated for decades among athletes, coaches, and scientists. Currently, HIT has come into focus again based in part on recent findings suggesting superior central adaptations to short-term interval programs compared with continuous exercise at lower intensity.^{1,2} However, the application of these findings to the long-term training of endurance athletes is unclear. The purpose of this brief review

Stephen Seiler is with the Faculty of Health and Sport Sciences, University of Agder, Kristiansand, Norway.

is to discuss the roles of training duration and training intensity in the long-term physiological and performance development of endurance athletes.

Measuring Training Intensity

A review of training intensity and duration issues in endurance training should begin with some discussion of how these variables are quantified. Measuring exercise duration is straightforward. Training volume can be measured in terms of distance (eg, yearly cycling or running kilometers) or time (annual training hours). The most readily comparable unit across endurance sports is effective training hours. Quantifying training intensity is more complicated. Describing and comparing training intensity distribution requires a common intensity scale. Most national sport governing bodies employ a guiding intensity scale based on ranges of heart rate relative to maximum and blood lactate concentration. Often, *aerobic* endurance training in the intensity range of approximately 50% to 100% of VO_2max is divided into five somewhat arbitrary intensity zones. Table 1 gives as an example a scale used by the Norwegian Olympic Committee. Standardizing an intensity scale can be criticized because the approach fails to account for individual variation in the relationship between heart rate and blood lactate concentration, or activity-specific variation, such as the tendency for maximal steady-state concentrations of blood lactate to be higher in activities activating less muscle mass.^{3,4} In the practical performance setting, these potential sources of error seem to be outweighed by the improved communication that a common scale facilitates between coach and athlete and across sports disciplines. A standardized training intensity “language” may be particularly important in improving the match between the intensity prescription from a coach and an athlete’s interpretation of that prescription. For example, Foster and colleagues quantified the tendency for midlevel athletes to train harder than planned on easy days and at lower intensity than planned on hard days, relative to coach prescriptions.⁵ It is important to point out that integrated approaches that multiply training session time by a physiological or perceptual measure of intensity (yielding TRIMPS⁶ or LOAD^{7,8}) have also been developed and used to quantify training

Table 1 Example of a five-zone intensity scale to prescribe and monitor training of endurance athletes

| Intensity zone | VO_2 (% max) | Heart rate (% max) | Lactate (mmol·L ⁻¹) | Typical accumulated duration within zone |
|----------------|-----------------------|--------------------|---------------------------------|--|
| 1 | 50–65 | 60–72 | 0.8–1.5 | 1–6 h |
| 2 | 66–80 | 72–82 | 1.5–2.5 | 1–3 h |
| 3 | 81–87 | 82–87 | 2.5–4 | 50–90 min |
| 4 | 88–93 | 88–92 | 4.0–6.0 | 30–60 min |
| 5 | 94–100 | 93–100 | 6.0–10.0 | 15–30 min |

Note. This scale is typical of intensity zone scales used for endurance training prescription and monitoring. The scale above was developed by the Norwegian Olympic Federation as a general guideline based on years of testing of cross-country skiers, rowers, and biathletes.

exposure. However, in this review, I will focus on training intensity distribution, and these integrated approaches will not be presented in detail.

Several recent studies examining training intensity distribution^{9–11} or performance intensity distribution in multiday events^{12,13} have employed individually determined first and second ventilatory turn points to demarcate three intensity zones (Zone 1, Zone 2, and Zone 3; Figure 1). Intensity distribution studies based on ventilatory threshold–derived zones are not directly comparable with the five-zone model, but what is typically identified as “lactate threshold intensity,” or the approximately 2 to 4 mM blood lactate concentration range, corresponds well in practice with the intensity zone demarcated by the first and second ventilatory turn points. Thus, for practical purposes, the three-zone model and five-zone model have common intensity anchor points around the lactate threshold. For well-trained athletes, I will use the term *low-intensity training* (LIT) to refer to work eliciting a stable lactate concentration of less than approximately 2 mM. *High-intensity training* (HIT) will refer to training above maximum lactate steady-state intensity (≥ 4 mM blood lactate). Training in the region bounded by about 2 and 4 mM blood lactate will be referred to as *threshold training* (ThT). For untrained / recreationally trained subjects, we find that a 2 mM lactate turn point is difficult to identify because blood lactate often approaches this concentration already at very low workloads (unpublished observations).

Published studies reporting the training characteristics of endurance athletes have employed several methods of quantifying intensity distribution. Self-report of training pace based on questionnaire and anchoring with different running paces (eg, below-marathon pace, 10 K pace, 3 K pace) has been used alone¹⁴ and in conjunction with physiological testing.¹⁵ Intensity distribution based on standardized blood lactate ranges and representative sampling during workouts has been reported for elite swimmers¹⁶. “Time-in-zone” heart rate analysis has been employed based on quantification of the training time spent within different heart rate ranges identified from preliminary threshold testing.^{9,10,17} The latter method gives total duration and percentage of time with heart rate within each intensity zone. This method is

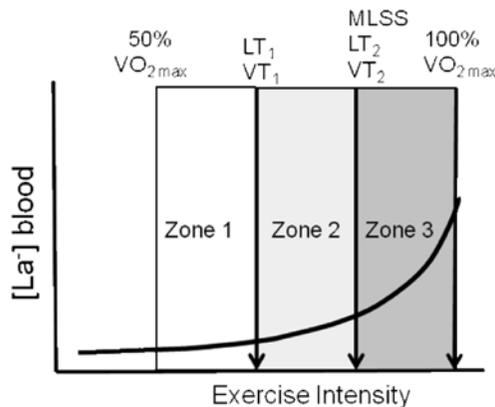


Figure 1 — A three-intensity-zone model based on identification of ventilatory thresholds.

appealing since it is noninvasive, individualized, and straightforward analytically. However, heart rate time-in-zone tends to underestimate the time spent working at high intensity (due to heart rate lag time during intervals). More importantly, it does not seem to correspond well with perceived effort for a given workout.¹⁰ For example, applying heart rate time-in-zone analysis to an interval session such as 4 × 4 min at a workload eliciting 95% VO_2max preceded by a 20 min warm-up and followed by a 20 min cool-down will result in both average session heart rate and time-in-zone distribution (dominated by time spent at low intensity) that misrepresent the perceived effort and blood lactate profile of the session and probably also underrepresent the autonomic stress load.¹⁸ Nominally allocating each training session to an intensity zone based on the intensity of the primary part of the workout, the “session goal approach,” yields better matching between heart rate analysis and athlete perception of session effort, or “session RPE,” in both cross-country skiers¹⁰ and 1st-division Norwegian soccer players (unpublished data). Typical software-based heart rate analysis methods overestimate the amount of time spent training at low intensity and underestimate the time spent at very high workloads, compared with athlete perception of effort for a training bout. In training organization, the unit of stress perceived and responded to by the athlete is the stress of entire training sessions or perhaps training days, not minutes in any given heart rate zone.

How Do Elite Endurance Athletes Train?

Good empirical descriptions of the distribution of training intensity in well-trained athletes constitute a fairly recent addition to the sport science literature. In 1991, Robinson et al¹⁹ published “the first attempt to quantify training intensity by use of objective, longitudinal training data.” They studied training characteristics of 13 national-class male New Zealand runners with favorite distances ranging from 1500 m to the marathon. They used heart rate data collected during training and related it to results from standardized treadmill determinations of heart rate and running velocity at 4 mM blood lactate concentration. Over a data collection period of 6 to 8 wk corresponding to the preparation phase, athletes reported that only 4% of all training sessions were interval workouts or races. For the remaining training sessions, average heart rate was 77% of their heart rate at 4 mM blood lactate (which translates to approx. 60% of VO_2max).

Billat et al performed physiological testing and training diary data collection of elite French and Portuguese marathoners.¹⁵ They classified training intensity in terms of several specific velocities: less than *v*-marathon, *v*-10,000m, and *v*-3,000m. During the 12 wk preceding an Olympic trials marathon, the athletes ran 78% of their training kilometers at below-marathon velocity, only 4% at marathon-race velocity (likely to be between VT_1 and VT_2), and 18% at *v*-10K or *v*-3K (likely to be $>\text{VT}_2$). This distribution of training intensity was identical in both high-level (< 2 h 16 min or < 2 h 38 min for males or females) and elite performers (< 2 h 11 min or < 2 h 32 min for males and females). But the elite athletes ran more total kilometers and proportionally more kilometers at or above *v*-10K. Examination of data from another descriptive study by Billat et al on elite male and female Kenyan 5 and 10 K runners demonstrated that approximately 85% of their weekly training kilometers were run at below-lactate threshold velocity.²⁰

Esteve-Lanao et al⁹ analyzed over 1000 heart rate records using the time-in-zone approach to quantify the training of eight regional- and national-class Spanish distance runners over a 6 mo period. Intensity zones were established with treadmill testing. On average these athletes ran 70 km·wk⁻¹ during the 6 mo period. Seventy-one percent of running time was <VT₁, 21% between VT₁ and VT₂, and 8% >VT₂. Mean training intensity was 64% VO₂max. They also reported that performance times in both long and short races were inversely correlated with total training time in zone 1. They found no correlation between the volume of HIT performed and race performance.

Rowers compete over a 2000 m distance requiring 6 to 7 min. Steinacker et al²¹ reported that extensive endurance training (60 to 120 min sessions at <2 mM blood lactate) dominated the training volume of German, Danish, Dutch, and Norwegian elite rowers. Rowing at higher intensities was performed about 4% to 10% of the total rowed time. The data also suggested that German rowers preparing for the world championships performed essentially no rowing at ThT intensity, but instead trained either LIT or HIT in the 6 to 12 mM range.

Fiskerstrand and Seiler²² examined historical developments in training organization among elite rowers. Using questionnaire data, athlete training diaries, and physiological testing records, they quantified training intensity distribution in 27 Norwegian athletes who had won world or Olympic medals in the 1970s, 1980s, or 1990s. They documented that over the three decades (1) training volume had increased about 20% and LIT volume increased relatively more, (2) the monthly hours of HIT had actually been reduced by one-third, (3) very high intensity over-speed sprint training had declined dramatically in favor of longer interval training at 85% to 95% of VO₂max, and (4) the number of altitude camps attended by the athletes increased dramatically. Over this 30 y timeline, athletes had about 12% higher VO₂max and a 10% improvement in rowing ergometer performance with no change in average height or body mass. However, most of this increase was seen between the 1970s and 1980s when major adjustments in training intensity distribution were made.

Guellich et al²³ described the training of world-class junior rowers from Germany during a 37-wk period culminating in national championships and qualification races for the world championships. Twenty-seven of the 36 athletes studied won medals in the junior world championships that followed the training period analyzed. Using the time-in-zone heart rate analysis method described above, fully 95% of all endurance training time was performed as LIT. This heavy dominance of extensive endurance training persisted throughout the 9 mo period. However, the relatively small volume of ThT and HIT shifted toward higher intensities from the basic preparation phase to the competition phase. That is, the overall intensity distribution became more *polarized* as athletes approached competition.

Professional road cyclists are known for performing very high training volumes, up to 30 to 35,000 km·yr⁻¹. Zapico and colleagues used the three-intensity zone model to track training characteristics from November to June in a group of elite Spanish U23 riders.¹¹ In addition, physiological testing was performed at season start and at the end of the winter and spring mesocycles to compare training changes and physiological test results. Figure 2 compares the training intensity distribution in the winter and spring mesocycles. Figure 3 shows physiological test results at baseline, and at the end of each training mesocycle. Comparison of the training

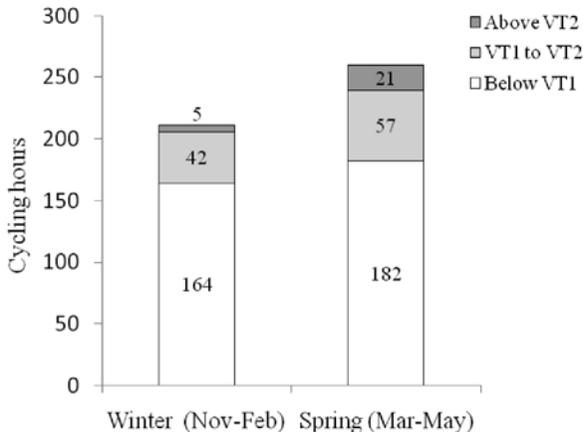


Figure 2 — Cycling intensity and volume of elite Spanish U23 cyclists training in the period November to June. Data redrawn from Zapico et al.¹¹

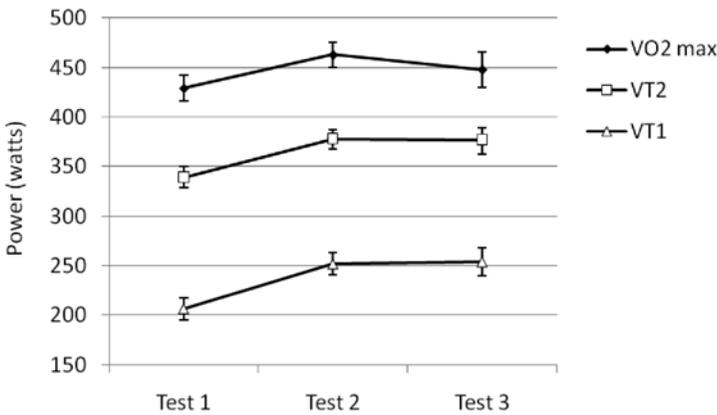


Figure 3 — Response to periodization of training intensity and volume in elite Spanish U23 cyclists (see Figure 2). Results from tests performed before starting the winter mesocycle (test 1), at the end of the winter mesocycle (test 2), and at the end of the spring mesocycle (test 3). Data redrawn from Zapico et al.¹¹

intensity distributions in the two periods shows that there was both an increase in total training volume and a 4× increase in HIT training during the spring mesocycle. However, physiological testing revealed no further improvement in power at VT₁, VT₂, or at VO₂max between the end of the winter and spring mesocycles, despite a clear training intensification. Anecdotally, this is not an unusual finding. Time at VO₂max or time at VT₂ power may be more sensitive variables to evaluate the impact of intensified training in highly trained athletes with stable threshold and VO₂max results.

Cross-country skiing has adopted spectator-friendly 1000 to 1500 m sprint races in the last decade (contested as a knockout tournament). Recently, Sandbakk et al compared the training and physiology of eight international-class and eight national-class (Norway) sprint cross-country skiers.²⁴ The internationally elite skiers distinguished themselves with higher $\text{VO}_{2\text{peak}}$, $v\text{VO}_{2\text{peak}}$, and exercise time at $\text{VO}_{2\text{peak}}$. Over a 6 mo registration period, the world-class skiers trained about one-third greater volume (445 h vs 341), with almost all of this difference in training time due to greater volumes of low-intensity training (86 more hours) and speed training (9 more hours). The two groups performed identical volumes of HIT over 6 mo (19 h in both groups, or about 45 min·wk⁻¹).

Schumacher and Mueller²⁵ demonstrated the validity of power balance modeling in predicting “gold medal standards” for physiological testing and power output in the 4,000 m pursuit cycling race. However, less obvious from the title was the detailed description of the training program followed by the gold medal-winning team monitored in the study. These athletes trained to maintain an average competition intensity of over 100% of power at $\text{VO}_{2\text{max}}$ with a program dominated by LIT (29,000–35,000 km·y⁻¹). In the 200 d preceding the Olympics, the pursuit team performed “low-intensity, high-mileage” training at 50 to 60% of $\text{VO}_{2\text{max}}$ on approximately 140 d. Stage races comprised approximately 40 d. Specific track cycling at near competition intensities was performed on fewer than 20 d between March and September. In the approximately 110 d preceding the Olympic final, high-intensity interval track training was performed on only 6 d.

The descriptive studies above highlight the paradoxical finding that even though all Olympic endurance events are performed at or above the lactate threshold (or $\geq 85\%$ $\text{VO}_{2\text{max}}$), the large majority of the training performed is completed below lactate threshold intensity. The duration of monitoring from published studies varies from weeks to an entire season but seems to converge on a common intensity distribution: about 80% of training sessions are LIT intensity and the remaining 20% are performed as ThT or HIT. For an athlete training 10 to 14 times per week, this means that two to three of these sessions would be ThT or HIT training bouts. This distribution fits well with findings that adding two interval sessions per week for 4 to 8 wk improves performance by 2% to 4% among well-trained endurance athletes doing only basic endurance training.^{26–29} Additional increases in HIT frequency do not induce further improvements and tend to induce symptoms of overreaching/overtraining.^{30,31}

Training Intensification Studies

Despite the consistency with which this general distribution is observed, one can question whether the “80-20” training intensity distribution is a really a self-organized optimum for high-performance athletes, or a product of tradition and/or superstition. Several studies have examined the impact of training intensification (with or without corresponding volume reduction) on physiology and/or performance in well-trained athletes.

In 1997, Evertsen et al published the first of three papers from a study involving training intensification in 20 well-trained junior cross-country skiers competing at the national or international level.^{32–34} In the 2 mo before study initiation, 84% of training was carried out at 60% to 70% $\text{VO}_{2\text{max}}$, with the remainder at 80% to

90% of VO_2max . They were then randomized to a moderate-intensity (MOD) or a high-intensity training group (HIGH). The MOD group maintained essentially the same training intensity distribution, but training volume was increased from 10 to 16 $\text{h}\cdot\text{wk}^{-1}$. The HIGH group reversed their baseline intensity distribution so that 83% of training time was performed at 80% to 90% of VO_2max , with only 17% performed as low-intensity endurance training. The HIGH group trained 12 $\text{h}\cdot\text{wk}^{-1}$. The training intervention period lasted 5 mo. Intensity control was achieved using heart rate monitoring and blood lactate sampling throughout the training period. Despite 60% more training volume in MOD and approximately four times more training at an intensity greater than or equal to lactate threshold in HIGH, physiological and performance changes were quite modest in both groups of already well-trained athletes (Table 2).

Gaskill et al reported the results of a 2 y project involving 14 cross-country skiers.³⁵ During the first year, athletes trained similarly, averaging 660 training hours with 16% HIT (nominal distribution of sessions). Physiological test results and race performances during the first year were used to identify seven athletes who responded well to the training and seven who showed poor VO_2max and lactate threshold progression, and race results. In the second year, the positive responders continued using their established training program whereas the nonresponders performed a markedly intensified training program with a slight reduction in training hours. They observed that the nonresponders from year 1 showed a positive response to the intensified program in year 2 (VO_2max , lactate threshold, race result points). The positive responders from year 1 showed a similar development in year 2 as year 1.

Esteve-Lanao et al randomized 12 subelite distance runners to one of two training groups (Z1 and Z2) that were carefully monitored for 5 mo.³⁶ They based

Table 2 Summary of responses to training intensification in well-trained cross-country skiers^{32–34}

| | Intensity Increase (n = 10) | Volume Increase (n = 10) |
|-------------------------|--------------------------------|-----------------------------|
| VO_2max | ↔ | ↔ |
| Lactate-threshold speed | ↑ 3% | ↔ |
| 20-min run at 9% grade | ↑ 3.8% | ↑ 1.9% |
| Fiber type | ↔ | ↔ |
| Enzyme activities | | |
| MCT 1 transporter | ↔ | ↓ 12% |
| MCT 4 transporter | ↔ | ↔ |
| Citrate synthase | ↔ | ↔ |
| Succinate dehydrogenase | ↑ 6% | ↔ |
| Na/K pump | ↑ ?% | ↑ ?% |

Note. A summary of results from refs. 32–34.

their training intensity distribution on the three-zone model described earlier. Based on time-in-zone heart rate monitoring, Z1 performed 81, 12, and 8% of training in zones LIT, ThT, and HIT respectively. The Z2 group performed more ThT, with 67, 25, and 8% of training performed in the three respective zones. Anecdotally, the authors reported that in pilot efforts, they were unable to increase the total time spent in intensity zone 3, as it was too hard for the athletes. Total training load was matched between the groups using a modification of TRIMPS. Improvement in a time trial performed before and after the 5 mo period revealed that the group that had trained more zone 1 training showed significantly greater race time improvement (-157 ± 13 s vs -121.5 ± 7.1 s, $P = .03$).

Ingham et al³⁷ randomized 18 experienced U.K. national standard male rowers into two training groups that were initially equivalent based on performance and physiological testing. All the rowers had completed a 25-d postseason “training-free” period just before baseline testing, followed by a 12 wk period of rowing ergometer training. One group performed 98% of all training between 60 and 75% of peak oxygen consumption (LIT). The other group performed 70% training at 60% to 75% VO_2max , as well as 30% of training at an intensity 50% of the way between power at LT and power at VO_2peak (MIX). In practice, the MIX group performed HIT on 3 d·wk⁻¹. The two groups performed virtually identical volumes of training (approx. 1140 km on the ergometer), with $\pm 10\%$ individual variation. Results of the study are summarized in Table 3. Sixteen of 18 subjects set new personal bests for the 2000 m ergometer test at the end of the study. The authors concluded that LIT and MIX training had similar positive effects on performance and VO_2max . The LIT regimen appeared to induce a greater right-shift in the blood lactate profile during submaximal exercise, but this did not translate to a significantly greater gain in ergometer performance.

Table 3 Physiological and performance changes after two rowing programs³⁷

| | LOW (n = 9) | MIXED (n = 9) |
|-------------------------|----------------|------------------|
| 2000-m ergometer time | ↓ 2% | ↓ 1.4% |
| VO_2max | ↑ 11% | ↑ 10% |
| Power at 2 mM lactate | ↑ 10%* | ↑ 2% |
| Power at 4 mM lactate | ↑ 14%* | ↑ 5% |
| VO_2 kinetics | ↔ | ↔ |

* $P < .05$ vs. LOW vs. MIXED.

Periodization of Training Variables

Elite endurance athletes train systematically >11 mo out of the year and may perform over 600 individual training sessions, all with the goal of achieving maximal performance at a specific time in the season. Further, peak athlete development may take 10 y of specific training,³⁸ with highly successful athletes often using

a 2- or 4-year cycle of preparation for world championships or Olympic events. Training is planned in different periods or training cycles. Periodization language often incorporates phase-duration terms such as *micro-*, *meso-*, and *macrocycle*, but this taxonomy has evolved from coaching practice, not research. For the purposes of this review I use the term *short-term periodization* to describe manipulation of daily training variables over a few days up to a few weeks. *Long-term periodization* of training refers to manipulation of training into cycles lasting weeks to several months. Short-term manipulation of intensity and duration loads seems to be very important for maintaining the athlete's health and tolerance for training. Long-term periodization is designed to facilitate the development of capacity over time, and ensure that peak performance is timed appropriately.

Since Matveyev introduced his now-classic model of periodization of volume and intensity in training four decades ago,³⁹ there has been considerable debate regarding how best to organize long-term exposure to training stimuli (ie, volume, intensity, mode) for modern endurance athletes. A number of long-term periodization structures have been conceptualized and described.³⁹⁻⁴³ However, controlled studies comparing the impact of these different organizational structures on endurance performance are lacking. One underlying assumption that influences long-term training organization principles in endurance training seems to be that adaptation of peripheral and central components of the respiratory chain are differentially impacted by training intensity and duration, with differing time courses and adaptive scope. Myocardial function may be somewhat more responsive to the greater ventricular filling and preload associated with near-maximal exercise intensity.^{1,2} The physiological and performance impact of adding HIT to endurance-trained athletes who have not been performing HIT is rapid.²⁶⁻²⁸ However, other rapidly derived benefits of HIT, such as increased buffer capacity,²⁸ and relevant pacing experience are likely to be integrated into this performance impact as well. The cardiovascular impact of further intensity amplification in already well-trained (LIT+HIT) subjects appears limited at best.^{11,30} In contrast, peripheral adaptations such as capillary densification and mitochondrial volume expansion (measured directly or indirectly as improvements in fractional utilization capacity) appear to (1) continue to respond to training over many months⁴⁴ and (2) appear responsive to large volumes of LIT.^{11,37,45} At the same time, there is some evidence suggesting that the blood lactate–power relationship may actually be neutral to, or even negatively impacted by, large volumes of HIT in well-trained athletes.^{37,45} However, mechanistic explanations for these observations are lacking.

Few studies have actually documented the intensity and volume distribution of endurance athletes over multiple phases of their annual training cycle.^{11,23,25,35} These studies—unpublished case histories of elite performers, and feedback from coaches—all suggest that although there is a clear increase in HIT moving from the preparation to competition period, the emphasis on substantial volumes of low-intensity training remains quite strong. Very little is documented regarding the correlation between responses to training in the preparation period and capacity or performance months later in the competition period.⁴⁶ For example, we have recently observed that whereas lactate profile responses to standardized testing before and after a 12 wk period of basic preparation in national-class German track cyclists varied from strongly positive to negative, these results were not correlated with end-of-season success in championship events.⁴⁵ Progress in understanding

long-term periodization will likely require systematic athlete monitoring by governing bodies or Olympic centers in cooperation with sport scientists.

Short-term periodization of training, involving day-to-day manipulation of intensity and duration over a few weeks, has been investigated more extensively. Endurance athletes train, rest, and repeat. Training (intensity, duration) and recovery (rest interval, nutrition) variables interact to induce both *fitness* (ie, physiological adaptations) and *fatigue* (ie, stress responses and associated negative health outcomes). This practical dichotomization was introduced by Banister and colleagues in their modeling studies of the training process.^{6,47,48} The predictive value and stability of their mathematical approach to the relationship between training input and fitness outcome has been challenged.⁴⁹ Conceptually, the model remains useful in that it predicts that day-to-day organization of training, recovery, and nutritional strategies should tend to maximize the gain in fitness for a given long-term cost (fatigue, stress, and risk of negative health outcomes).

Over a period of days, athletes normally perform LIT and ThT/HIT sessions. Horses are trained similarly, with alternating “easy days” of continuous running and “hard days” of interval training. Bruin and colleagues⁵⁰ performed a long-term training study of horses in which they manipulated the hard-easy rhythm of the horses’ training in two ways. After 187 d of daily training in hard-easy fashion, hard training days were intensified by performing more total high-intensity running, with easy days left unchanged. The horses exhibited improved running performance over the next 75 d. After 261 d, the easy days were intensified by having the horses run faster for the same duration. Within 5 d, the horses were no longer able to complete the HIT and showed clear signs of decompensation and overtraining symptoms. Foster extended this finding to human athletes and conceptualized training *monotony* as increasing the risk of negative adaptations to training.⁵¹ High *training stress* was quantified as a product of large training volumes, high perceived intensity, and low day-to-day variation in training load. Elite athletes often train twice or even three times per day, making the rest interval between training sessions typically between 4 and 12 h. Achieving this training frequency without excessive stress appears to require careful management of training intensity.

Connecting Training Characteristics to Cellular Signaling and Stress Responses

The studies outlined above combine to suggest that over the long term, (1) successful endurance athletes achieve excellent results when accumulating a high training volume by emphasizing frequent exposure to 60 to 180 min bouts performed at approximately 60 to 75% of VO_2max (ie, LIT) in combination with a modest proportion of training performed at intensities between 85 and 100% of VO_2max (about 20% of training sessions), and (2) when HIT is heavily emphasized by adding interval workouts and decreasing the volume of LIT, the effects are equivocal at best. While these conclusions are based on a growing body of published studies, they are unrevealing and unsatisfying from a mechanistic viewpoint.

Ultimately, endurance training is a stimulus for cell signaling, gene expression, and resulting increased rates of protein synthesis. Changes in physiological capacity over time are hypothesized to be the net result of transient increases in gene expression during recovery from repeated bouts of exercise.⁵² It is therefore

appealing to try to link training behavior to cellular events associated with training adaptation. Unfortunately, details regarding how intensity and duration of exercise combine to modulate cell signaling are only beginning to emerge in the literature. What is known is that multiple signaling pathways exist;⁵³ redundancies among mechanical, metabolic, neuronal, and hormonal signaling factors are likely;⁵² intensity and duration effects on signaling may interact in fiber type-specific ways;⁵⁴ and the potency of the gene expression response to a given exercise signal (intensity \times duration) changes rapidly with repeated exercise.^{55,56} At present, any attempt to reconcile training behavior in elite performers with the molecular biology of cellular signaling is doomed to some measure of both incompleteness and overinterpretation. Accepting that, one simple reconciliation of signaling studies with athlete practice might be that (1) exercise duration and exercise intensity can drive gene expression for mitochondrial protein proliferation through different pathways and (2) ceiling effects for signal amplitude are seen rapidly with repeated high-intensity interval exercise, whereas increased exercise frequency at reduced intensity may provide greater scope for expansion of the total signal (amplitude \times frequency) for gene expression.

Training induces stress responses as well. Increased training intensity is associated with a nonlinear increase in sympathetic stress that appears to track well with relative intensity increases and the lactate profile.⁵⁷ In highly trained athletes, training more frequently and/or for longer durations at relatively low exercise intensities may induce a lower overall stress load and facilitate more rapid recovery compared with highly intensive training sessions above the lactate threshold.¹⁸ An intensity distribution strategy that allows frequent training (twice daily) may give an important long-term adaptive advantage via what can be conceptually described as optimization of the ratio between adaptive signal and stress response. Recent studies comparing twice daily training with training the same total volume every other day suggest that training twice daily induced greater peripheral adaptations.^{58,59} One mechanism for this benefit may be the signal-amplifying effect of reduced muscle glycogen (in the second daily workout). We have also found that autonomic nervous system recovery (measured via heart rate variability) is very rapid after training bouts at 60% VO_2max for up to 120 min, but becomes markedly delayed in highly trained subjects when exercise intensity increases to an intensity eliciting >3 mM blood lactate. We also observed that highly trained subjects (often training twice daily) recovered parasympathetic control after a standardized HIT session dramatically faster than a group of subjects training about once a day.¹⁸ Similarly, elite female rowers can train for 2 h at 60% VO_2max with only minor hormonal or immune system disturbance.⁶⁰ Unfortunately, longitudinal data are needed to reveal whether progression in training volume and frequency gradually induces, or is naturally facilitated by, more rapid recovery of the autonomic nervous system and hormonal balance after training. Thus, the question could be posed as, is rate of recovery from training a trainable characteristic of the endurance athlete?

Conclusions

There is reasonably strong evidence for concluding that an approximate 80-to-20 ratio of LIT to ThT/HIT intensity training gives excellent long-term results among endurance athletes. Frequent, low-intensity (≤ 2 mM blood lactate), longer

duration training is effective in stimulating physiological adaptations. The idea of a dichotomous physiological impact of HIT and LIT is probably exaggerated, as both methods seem to generate overlapping physiological adaptation profiles and are likely complementary. Over a broad range, increases in total training volume correlate well with improvements in physiological variables and performance. HIT is a critical component in the training of all successful endurance athletes. However, about two HIT training sessions per week seems to be sufficient for inducing physiological adaptations and performance gains without inducing excessive stress over the long term. When *already well-trained athletes* markedly intensify training over weeks to months, the impact is equivocal, with reported effects varying widely. In athletes with an established endurance base and tolerance for relatively high training loads, intensification of training may yield small performance gains at acceptable risk of negative outcomes. An established endurance base built from high volumes of training may be an important precondition for tolerating and responding well to a substantial increase in training intensity over the short term. Periodization of training by elite athletes is achieved with modest reductions in total volume and a careful increase in the volume of training performed above the lactate threshold as athletes transition from preparation to competition training phases. Greater *polarization* of training intensity characterizes this transition, both in terms of the net training distribution as well as within micro- and macrocycles of training. However, compared with classic training periodization models, with large swings in volume and intensity, the basic intensity distribution remains quite similar throughout the year. Almost no research is available investigating the impact of different models of long-term training periodization for endurance athletes.

References

1. Daussin FN, Ponsot E, Dufour SP, et al. Improvement of VO₂max by cardiac output and oxygen extraction adaptation during intermittent versus continuous endurance training. *Eur J Appl Physiol*. 2007;101:377–383.
2. Helgerud J, Hoydal K, Wang E, et al. Aerobic high-intensity intervals improve VO₂max more than moderate training. *Med Sci Sports Exerc*. 2007;39:665–671.
3. Beneke R, Leithauser RM, Hutler M. Dependence of the maximal lactate steady state on the motor pattern of exercise. *Br J Sports Med*. 2001;35:192–196.
4. Beneke R, von Duvillard SP. Determination of maximal lactate steady state response in elected sports events. *Med Sci Sports Exerc*. 1996;28:241–246.
5. Foster C, Heiman KM, Esten PL, et al. Differences in perceptions of training by coaches and athletes. *South African Journal of Sports Medicine*. 2001;8:3–7.
6. Banister EW, Good P, Holman G, et al. Modeling the training response in athletes. In: Landers DM, ed. *Sport and elite performers*. Champaign: Human Kinetics; 1986:7–23.
7. Foster C, Daines E, Hector L, et al. Athletic performance in relation to training load. *Wis Med J*. 1996;95:370–374.
8. Foster C, Hector LL, Welsh R, et al. Effects of specific versus cross-training on running performance. *Eur J Appl Physiol Occup Physiol*. 1995;70:367–372.
9. Esteve-Lanao J, San Juan AF, Earnest CP, et al. How do endurance runners actually train? Relationship with competition performance. *Med Sci Sports Exerc*. 2005;37:496–504.
10. Seiler KS, Kjerland GO. Quantifying training intensity distribution in elite endurance athletes: is there evidence for an “optimal” distribution? *Scand J Med Sci Sports*. 2006;16:49–56.

11. Zapico AG, Calderon FJ, Benito PJ, et al. Evolution of physiological and haematological parameters with training load in elite male road cyclists: a longitudinal study. *J Sports Med Phys Fitness*. 2007;47:191–196.
12. Lucia A, Hoyos J, Carvajal A, et al. Heart rate response to professional road cycling: the Tour de France. *Int J Sports Med*. 1999;20:167–172.
13. Lucia A, Hoyos J, Santalla A, et al. Tour de France versus Vuelta a Espana: which is harder? *Med Sci Sports Exerc*. 2003;35:872–878.
14. Karp JR. Training characteristics of qualifiers for the U.S. Olympic Marathon Trials. *Int J Sports Physiol Perform*. 2007;2:72–92.
15. Billat VL, Demarle A, Slawinski J, et al. Physical and training characteristics of top-class marathon runners. *Med Sci Sports Exerc*. 2001;33:2089–2097.
16. Mujika I, Chatard JC, Busso T, et al. Effects of training on performance in competitive swimming. *Can J Appl Physiol*. 1995;20:395–406.
17. Esteve-Lanao J, Lucia A, deKoning JJ, et al. How do humans control physiological strain during strenuous endurance exercise? *PLoS ONE*. 2008;3:e2943.
18. Seiler S, Haugen O, Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. *Med Sci Sports Exerc*. 2007;39:1366–1373.
19. Robinson DM, Robinson SM, Hume PA, et al. Training intensity of elite male distance runners. *Med Sci Sports Exerc*. 1991;23:1078–1082.
20. Billat V, Lepretre PM, Heugas AM, et al. Training and bioenergetic characteristics in elite male and female Kenyan runners. *Med Sci Sports Exerc*. 2003;35:297–304; discussion 305–296.
21. Steinacker JM, Lormes W, Lehmann M, et al. Training of rowers before world championships. *Med Sci Sports Exerc*. 1998;30:1158–1163.
22. Fiskerstrand A, Seiler KS. Training and performance characteristics among Norwegian international rowers 1970–2001. *Scand J Med Sci Sports*. 2004;14:303–310.
23. Guellich A, Seiler S, Emrich E. Training Methods and Intensity Distribution of Young World-Class Rowers. *Int J Sports Physiol Perform*. 2009;4:448–460.
24. Sandbakk Ø, Holmberg HC, Leirdal S, et al. The Physiology of World Class Sprint Skiers. *Scand J Med Sci Sports*. 2010. doi: 10.1111/j.1600-0838.2010.01117.x.
25. Schumacher YO, Mueller P. The 4000-m team pursuit cycling world record: theoretical and practical aspects. *Med Sci Sports Exerc*. 2002;34:1029–1036.
26. Lindsay FH, Hawley JA, Myburgh KH, et al. Improved athletic performance in highly trained cyclists after interval training. *Med Sci Sports Exerc*. 1996;28:1427–1434.
27. Stepto NK, Hawley JA, Dennis SC, et al. Effects of different interval-training programs on cycling time-trial performance. *Med Sci Sports Exerc*. 1999;31:736–741.
28. Weston AR, Myburgh KH, Lindsay FH, et al. Skeletal muscle buffering capacity and endurance performance after high-intensity interval training by well-trained cyclists. *Eur J Appl Physiol Occup Physiol*. 1997;75:7–13.
29. Driller MW, Fell JW, Gregory JR, et al. The effects of high-intensity interval training in well-trained rowers. *Int J Sports Physiol Perform*. 2009;4:110–121.
30. Billat VL, Flechet B, Petit B, et al. Interval training at VO₂max: effects on aerobic performance and overtraining markers. *Med Sci Sports Exerc*. 1999;31:156–163.
31. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. *Sports Med*. 2004;34:967–981.
32. Evertsen F, Medbo JI, Bonen A. Effect of training intensity on muscle lactate transporters and lactate threshold of cross-country skiers. *Acta Physiol Scand*. 2001;173:195–205.
33. Evertsen F, Medbo JI, Jebens E, et al. Effect of training on the activity of five muscle enzymes studied on elite cross-country skiers. *Acta Physiol Scand*. 1999;167:247–257.
34. Evertsen F, Medbo JI, Jebens E, et al. Hard training for 5 mo increases Na⁽⁺⁾-K⁽⁺⁾ pump concentration in skeletal muscle of cross-country skiers. *Am J Physiol*. 1997;272:R1417–R1424.

35. Gaskill SE, Serfass RC, Bacharach DW, et al. Responses to training in cross-country skiers. *Med Sci Sports Exerc.* 1999;31:1211–1217.
36. Esteve-Lanao J, Foster C, Seiler S, et al. Impact of training intensity distribution on performance in endurance athletes. *J Strength Cond Res.* 2007;21:943–949.
37. Ingham SA, Carter H, Whyte GP, et al. Physiological and performance effects of low-versus mixed-intensity rowing training. *Med Sci Sports Exerc.* 2008;40:579–584.
38. Balyi I. Long-term athletic development: the B.C. approach. *Sports Aider.* 2002;18:1–4.
39. Matwejew LP. *Periodisierung des sportlichen Trainings.* Berlin: Bartels & Wernitz; 1972.
40. Issurin V. Block periodization versus traditional training theory: a review. *J Sports Med Phys Fitness.* 2008;48:65–75.
41. Issurin V. A modern approach to high performance training: the block composition. In: Blumenstein B, Lidor R, Tenenbaum G, eds. *Psychology of Sport Training.* Oxford: Meyer & Meyer Sport; 2007:216–234.
42. Tschiene P. Einige neue Aspekte zur Periodisierung des Hochleistungstrainings. *Leistungssport.* 1977;7:379–382.
43. Tschiene P. Veränderungen in der Struktur des Jahrestrainingszyklus. *Leichtathletik.* 1985;29:1519–1522.
44. Tyler CM, Golland LC, Evans DL, et al. Skeletal muscle adaptations to prolonged training, overtraining and detraining in horses. *Pflügers Arch.* 1998;436:391–397.
45. Guellich A, Seiler S. Lactate profile changes in relation to training characteristics in junior elite cyclists. *Int J Sports Physiol Perform.* 2010;5:316–327.
46. Ingjer F. Maximal oxygen uptake as a predictor of performance ability in women and men elite cross country skiers. *Scand J Med Sci Sports.* 1991;1:25–30.
47. Banister EW, Calvert TW. Planning for future performance: implications for long term training. *Can J Appl Physiol.* 1980;5:170–176.
48. Morton RH, Fitz-Clarke JR, Banister EW. Modeling human performance in running. *J Appl Physiol.* 1990;69:1171–1177.
49. Hellard P, Avalos M, Lacoste L, et al. Assessing the limitations of the Banister model in monitoring training. *J Sports Sci.* 2006;24:509–520.
50. Bruin G, Kuipers H, Keizer HA, et al. Adaptation and overtraining in horses subjected to increasing training loads. *J Appl Physiol.* 1994;76:1908–1913.
51. Foster C. Monitoring training in athletes with reference to overtraining syndrome. *Med Sci Sports Exerc.* 1998;30:1164–1168.
52. Fluck M, Hoppeler H. Molecular basis of skeletal muscle plasticity—from gene to form and function. *Rev Physiol Biochem Pharmacol.* 2003;146:159–216.
53. Coffey VG, Hawley JA. The molecular bases of training adaptation. *Sports Med.* 2007;37:737–763.
54. Hildebrandt AL, Pilegaard H, Neufer PD. Differential transcriptional activation of select metabolic genes in response to variations in exercise intensity and duration. *Am J Physiol Endocrinol Metab.* 2003;285:E1021–E1027.
55. McConell GK, Lee-Young RS, Chen ZP, et al. Short-term exercise training in humans reduces AMPK signalling during prolonged exercise independent of muscle glycogen. *J Physiol.* 2005;568:665–676.
56. Yu M, Stepto NK, Chibalin AV, et al. Metabolic and mitogenic signal transduction in human skeletal muscle after intense cycling exercise. *J Physiol.* 2003;546:327–335.
57. Chwalbinska-Moneta J, Kaciuba-Uscilko H, Krysztofiak H, et al. Relationship between EMG blood lactate, and plasma catecholamine thresholds during graded exercise in men. *J Physiol Pharmacol.* 1998;49:433–441.
58. Hansen AK, Fischer CP, Plomgaard P, et al. Skeletal muscle adaptation: training twice every second day vs. training once daily. *J Appl Physiol.* 2005;98:93–99.

59. Yeo WK, Paton CD, Garnham AP, et al. Skeletal muscle adaptation and performance responses to once a day versus twice every second day endurance training regimens. *J Appl Physiol*. 2008;105:1462–1470.
60. Nieman DC, Nehlsen-Cannarella SL, Fagoaga OR, et al. Immune response to two hours of rowing in elite female rowers. *Int J Sports Med*. 1999;20:476–481.