

Adaptation Signal Determinants in Aerobic Exercise Training.

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Introduction

Historically the method for optimizing the performance of endurance athletes has been to design training plans based around large amounts of very low intensity training. The basic premise of this philosophy is that low intensity training more effectively signals the up-regulation of structures and pathways related to the uptake of oxygen. However, contemporary research suggests that high intensity training may be more effective at increasing maximal rates of oxygen consumption, and absolutely necessary to achieve any training response in elite endurance athletes.

Literature Review

Endurance exercise performance is a crucial field, the study of which affects not only athletes, but also those with respiratory capacity reduced by illness or injury. One of the primary factors limiting endurance exercise performance is the quantity of oxygen that an athlete can remove from the atmosphere and provide to the working muscle. Training to improve these elements of endurance exercise performance has historically been governed by the idea that accumulated volume of training is the most important factor relating to positive exercise induced changes (Karlsen & Patterson, 1998). However, more recent scientific literature suggests that the intensity of exercise is the most important training variable for endurance exercise performance (Cooper, 1997; Hudlicka, Brown, & Egginton, 1992; Midgley, McNaughton, & Wilkinson, 2006).

Since the pioneering work of Dill and others nearly 80 years ago (Dill, Edwards, & Talbott, 1932; Hill, 1925), a sophisticated understanding of the structural and functional determinants limiting oxygen flux has emerged (Levine, 2008; Weibal, 1984). The quantitative relationship produced by our understanding of the aerobic system

illustrates that the sustainable rate of performance of an athlete depends upon the interaction of their maximum aerobic capacity (VO_2 max), the intensity at which lactate begins to accumulate in their blood stream, and the effectiveness with which they convert metabolic energy to mechanical performance (efficiency) (Costill, 1972). While the training improvement of efficiency is thought negligible (McArdle, Katch, & Katch, 2007) and power output at lactate threshold is observed to increase by only 36% (Coyle, 2005; Joyner & Coyle, 2008), the largest potential for performance improvement is seen in VO_2 max, with up to 300% difference observed between elite (highly trained endurance athletes) and sedentary individuals (Joyner & Coyle, 2008).

In order for endurance athletes to augment their VO_2 max, they must stimulate additional growth in the structures and pathways that enable oxygen delivery to the working muscle. Traditionally, this training goal has been achieved by dedicating many hours per day, every month of the year to exercise training. For example, yearly totals for elite cross-country skiers reach 1000+ hours of training (3-4 hours/day), with elite cyclists exceeding this total. Current popular coaching literature based on this historical philosophy of training suggests spending approximately 80% of those hours at a low intensity (60-70% maximum heart rate) (Karlsen & Patterson, 1998). These existing training protocols are governed by the idea that it is the accumulated volume of training that is the most important factor relating to positive exercise induced changes. However, after analyzing the current scientific literature, it appears that intensity is a more important training variable.

In order to review the effectiveness of focusing on volume or intensity, I will present the existing understanding on the degree of response possible within the steps of

the oxygen cascade. The volume of oxygen taken in and used by the body ($\dot{V}O_2$) is a function of many different structures and functions, each of which is a potential limiting step in the flux of oxygen. These determinants of the possible quantity of oxygen from the lungs to the working muscle cell are characterized by the cardiovascular Fick equation (Weibel, 1984):

$$\dot{V}O_2 = \dot{Q} \times [CaO_2 - C\bar{v}O_2]$$

where Q is cardiac output, CaO_2 is the arterial oxygen concentration, and $C\bar{v}O_2$ is the venous oxygen concentration.

Cardiac Output

Cardiac Output (Q) is the total amount of blood pumped by the heart in liters per minute, and is the product of heart rate (HR) and stroke volume (SV). In both non-human mammals, and human subjects, Q increases at the same rate as $\dot{V}O_2$, suggesting that it accounts for most of the variation between maximal rates of oxygen consumption (Ekblom & Hermansen, 1968; Taylor, Karas, Weibel, & Hoppeler, 1987). HR responds to exercise with a slightly greater than 2-fold increase from resting to maximum $\dot{V}O_2$ in sedentary subjects (more than 3-fold in elite endurance athletes), while SV can increase by nearly 2-fold to a maximum of about 150ml. These changes result in a cardiac output at maximum $\dot{V}O_2$ that is four times higher than resting (Levine, 2008; McArdle et al., 2007).

Due to the inability of an athlete's maximum heart rate to increase with training, the primary limitation to increases in cardiac output over time is stroke volume, or the amount of blood ejected by the heart per contraction (Saltin & Rowell, 1980). For instance, more aerobic mammals have been found to have stroke volumes that are approximately 1.5 times larger in athletic versus nonathletic species, while elite human athletes have 1.6 times larger SV than sedentary individuals (Blomqvist & Saltin, 1983; Karas, Taylor, et al., 1987; McArdle et al., 2007). Not only do athletes have larger stroke volumes, they are also able to increase their SV throughout graded exercise more than sedentary subjects (Gledhill, Cox, & Jamnik, 1994). More detailed findings suggest that in elite athletes, stroke volume continues to rise until VO_2 max is reached, as opposed to sedentary subjects whose SV plateaus at a low to moderate exercise intensity (Warburton et al., 2002; Zhou et al., 2001)

Investigations focused on the limitations of stroke volume suggest that the end diastolic volume is the primary constraint (Baggish, Wang, et al., 2008; Baggish, Yared, et al., 2008; Levine, 2008; Levine, Lane, Buckey, Friedman, & Blomqvist, 1991). End diastolic volume is determined by the left ventricle's ability to relax and fill. The coronary structures that prevent full relaxation of the myocardium include the extracellular matrix (ECM) of the muscle itself and the serous membrane that separates the heart from the pleural cavity (the pericardium). Research in aging mice has shown that chronic exercise decreases the collagen cross-linking, increasing pliability of the ECM (Thomas, McCormick, Zimmerman, Vadlamudi, & Gosselin, 1992; Thomas, Zimmerman, Hansen, Martin, & McCormick, 2000). In addition to a potential change in cardiac elasticity, habitual exercise also increases blood volume by 8-10%, which aids in the diastolic

filling of the left ventricle (Gledhill, Warburton, & Jamnik, 1999; Sawka, Convertino, Eichner, Schnieder, & Toung, 2000).

Within the heart, the hypothesized signal for collagen biosynthesis is volume overload, or the stretching of heart wall with the influx of venous blood (Clausen 1977; Kainulainen, Takala, Myllyla, Hassinen, & Vihko, 1983). Since volume overload is dependent upon SV, the bigger the stretch and resulting stroke volume, the greater the signal for change (Clausen, 1977; Cooper, 1997; Midgley et al., 2006). More specifically, one of the primary markers of collagen cross-linking is prolyl 4-hydroxylase (P-4-H), which directly correlates with the biosynthesis of collagen (Choi et al., 2009; Takala et al., 1991; Thomas et al., 2000). Training in mice, dogs, and rats, has been shown to increase P-4-H levels without increased collagen mass (Choi et al., 2009; Kainulainen et al., 1983; Takala et al., 1991; Thomas et al., 2000).

While diastolic elasticity is usually thought to be the primary limiter in cardiac distension, the pericardium may also limit passive filling of the left ventricle (Atherton et al., 1997; Hammond, White, Bhargavam, & Shabetai, 1992; LeWinter & Pavelec, 1982; Stray-Gundersen et al., 1986). Thus, removal of imposed pericardial restrictions increases $\dot{V}O_{2\max}$ 8% and 30%, in dogs and pigs, respectively (Hammond et al., 1992; Stray-Gundersen et al., 1986). Although nonhuman models suggest that exercise training leads to increased pericardial elasticity, it is currently unknown whether similar pericardial remodeling occurs following endurance training in humans (Esch, Bredin, Haykowsky; LeWinter & Pavelec, 1992).

Arterial Oxygen Content

Arterial Oxygen Content (CaO_2) is determined by the flow of gas across the alveolar-capillary membrane. The flux from the atmosphere to the arterial blood depends upon the pressure difference between the alveoli and the vasculature, and the diffusive capacity of the lung membrane (Weibal, 1984). Work in humans has shown that the diffusive capacity does not change with the onset of exercise or with training (Gale, Torre-Bueno, Moon, Saltzmand, & Wagner, 1985). The alveolar oxygen pressure is determined by the atmospheric oxygen pressure and by ventilation (i.e., breath, volume, and frequency). Once in the arterial system, the delivery of oxygen is determined by the two factors: (a) the amount of oxygen bound to hemoglobin, and (b) the amount of oxygen dissolved in solution, which is minimal (McArdle et al., 2007). The importance of hemoglobin is evidenced by short-term altitude exposure increasing oxygen carrying capacity by increasing hemoglobin mass and red cell volume in elite athletes (Heinicke, Heinicke, Schmidt, & Wolfarth, 2005). Also, in nonhuman animals, athletic species (dogs, ponies) have 1.6 times higher hemoglobin concentrations than sedentary species (goats, calves) (Karas, Taylor, Rosler, et al., 1987). In human subjects, artificially elevating hemoglobin mass by blood doping increases VO_2 max significantly (Gledhill et al., 1999; Russell, Gore, Ashendon, Parisotto, & Hahn, 2002). However, since increases in erythrocyte mass in man plateaus after only 30 days of exposure to either altitude induced hypoxia or endurance training, the long-term significance of these changes is probably small (Midgley et al., 2006; Sawka et al., 2000).

Some elite male athletes have developed such high demand for oxygen through maximal exercise that the pulmonary system limits oxygen uptake. In contrast, the

physical size of the pulmonary structures have been thought to limit oxygen supply in small women, causing exercise induced arterial desaturation (Dempsey, 1986; Karas, Taylor, Rosler, et al., 1987; McArdle et al., 2007).

Venous Oxygen Content

Venous Oxygen Content ($C\bar{v}O_2$) is limited by the oxygen pressure gradient between the capillaries and the muscle cells, and the diffusive capacity of the cellular membranes. The cellular oxygen pressure head is largely determined by volume of mitochondria, which sets the demand (Ingjer, 1979). Some research suggests that the oxidative capacity of the mitochondria also affects demand, but this is a contentious subject (Larson-Meyer, Newcomer, Hunter, Hetherington, & Weinsier, 2000; Richardson, 2000; Richardson et al., 1999; Roy et al., 2006). Myoglobin is also thought to contribute to this local oxygen cellular concentration gradient by binding with oxygen to decrease the amount in solution within the myoplasm (Wittenberg & Wittenberg, 2003). The arterial oxygen content is set by the upstream factors such as hemoglobin concentration discussed above. In general, the movement of oxygen molecules occurs in response to the relevant pressure gradient, the diffusive capability of the capillary membranes, and the distance from the capillaries to the mitochondria (Weibal, 1984).

Increased capillary density supports elevated oxygen flux in both human and non-human animals by decreasing the distance traveled by the oxygen from arterial blood to mitochondria, (Hoppeler et al., 1987; Ingjer, 1979; Saltin & Rowell, 1980). For example, with training, capillary density increased by 18% in 8 weeks (Richardson, 2000), and 28% in 24 weeks (Ingjer, 1979). Hudlicka et al. (1992) found that the primary signals for

enhancement of capillarization (angiogenesis) were increased shear stress caused by fluid dynamics of cardiac contraction and capillary blood pressure due to high blood velocities brought about by high cardiac outputs. Thus the signal to enhance capillarity depends upon intensity, (i.e., higher intensity equals a stronger signal) (Egginton, 2009; Gavin, 2009; Hudlicka, Bron, May, Zakrzewicz, & Pries, 2006; Prior, Yang, & Terjung, 2004).

In summary, pericardial flexibility is signaled to increase by volume overload, which is determined by stroke volume (Kainulainen et al., 1983). This is also true for the enhancement of capillary density, which is signaled by shear stress, which is highest at maximal stroke volumes (Hudlicka et al., 2006; Prior et al., 2004,). The evidence presented above suggests that the optimal intensity of training to increase factors influencing oxygen flux is directly related to cardiac output, but primarily stroke volume.

Evidence suggests that different absolute intensity prescriptions are necessary for sub-elite and elite athletes. Less fit individuals experience maximum stroke volumes at submaximal exercise intensities, suggesting that, for a short period of time, their minimum training intensities are closer to 40% of their VO_2 max (Astrand, 1964; Gledhill et al., 1994). For elite athletes, however, stroke volume continues to increase throughout graded exercise, thus raising their optimal training intensity to close to VO_{2max} (Gledhill et al., 1994; Warburton et al., 2002).

Applications

Classical coaching practice calls for large amounts of low intensity training (75-90% of yearly volume), coupled with lesser amounts of higher intensity training (10-25% of yearly volume), but the overall goal has been to complete as much volume as possible

(Karlson & Patterson, 1998; Rusko, 1992; Sleamaker & Browning, 1989). However, contemporary literature suggests that high intensity exercise may be more effective based on the physiological understanding of how the body adapts to supporting greater rates of oxygen uptake (Helgerud et al., 2007; Laurson & Jenkins, 2002).

A recent review of endurance performance physiology conducted by Midgley and colleagues (2007) concluded that a reason for a general lack of progress in the applicability of basic science to coaching philosophy is related to the extremely narrow focuses and methodological flaws of previous studies. However, while individual studies often do fall into these author's description, my approach for this paper has been to consider the complete body of literature as a whole, with the goal of developing scientifically justifiable training suggestions for coaches and athletes.

The training literature examining the effect intensity on whole-body oxygen consumption also points to intensity as a more effective stimulus from training enhancement of oxidative capacity (Gaskill, Serfass, Bacharach, & Kelly, 1999; Gormley et al., 2008; Helgerud et al., 2007; Kemi et al., 2005; McKay, Paterson, & Kowalchuk, 2009). Several studies have investigated the effect of high intensity exercise (HIT) as compared to low intensity exercise (LIT) on VO_2 max. The training interventions in these studies compare both short-term (weeks) and long-term (years) differences between HIT and LIT.

In order to synthesize the literature, I have examined both the basic respiratory physiology described previously, and the more applied training literature to develop suggestions for sound training interventions. The applied literature suggests that the ability of an athlete to improve VO_2 max depends primarily upon previous VO_2 max

levels (Helgerud et al., 2007; Laursen & Jenkins, 2002; Midgley et al., 2006; Swain & Franklin, 2002). In a review of 59 training studies relating changes in VO₂ max to training intensity, the amount of training related improvement in oxygen consumption was positively correlated to training intensity (Wenger & Bell, 1986). In fact, this relationship held true even when accounting for training frequency, training duration, training history, and initial VO₂ max levels (Wenger & Bell, 1986).

In single studies examining training effects, high intensity training is more effective at eliciting a positive adaptation to the body's maximum oxygen consumption than low intensity training. To investigate the differences between LIT and HIT on VO₂ max, Helgerud et al. (2007) employed 8-week training protocols matched for energy expenditure in moderately trained individuals (pre-training VO₂ max's of 50-60 mlO₂/kg/min) The authors assigned the subjects to one of four groups: (a) LIT, 45 minutes of 70% HR max; (b) LIT2, 25 minutes of 85% HR_{max}; (c) HIT1, 47x 15s at 95% HR max with 15s at 70% HR max recovery in between, and (d) HIT2, 4x4 minutes at 95% HR max with three minutes of 70% HR max between intervals. After eight weeks, only the HIT groups had significantly increased their VO₂ max's, with observed gains of 5.5% (HIT1) and 7.3% (HIT2) (Helgerud et al., 2007). Similar to Helgerud's work, Gormley et al. (2008) investigated the differences in VO₂ max response to training between 50% of Heart Rate Reserve (HRR), 75% of HRR, and 5x5 minutes intervals at 95% HRR. These authors found significant increases in VO₂ max in all groups (3.2, 4.8, and 7.2 mlO₂/kg/min respectively), with increasing intensity of exercise correlating to increased VO₂ max changes in previously sedentary individuals (Gormley et al., 2008).

In a more recent study, McKay et al. (2009) compared the differences seen in VO_2 max enhancement between long duration (60-120 minutes at 65% of VO_2 max) and HIT (8-12x 1 minute at 120% of VO_2 max). While this intervention was only two weeks long, enhancements in VO_2 max were observed in both cases (4.5% for HIT, and 7% for LIT), possibly due to the fact that the total workloads were very different. For instance, the LIT group's total training volume was over 10 times higher than the HIT group (McKay et al., 2009).

In contrast to the relatively short term studies discussed above, Gaskill et al. (1999) looked at whether elite cross country skiers (VO_2 max values over 64 $\text{mlO}_2/\text{kg}/\text{min}$) who had reached performance plateaus in the conventional high-volume method would be able to improve by increasing the amount of high intensity work done in a year. By decreasing their volume of LIT training by 22% and increasing their HIT training from 17% to 35% of total volume, these athletes significantly increased maximal aerobic capacity by 9% in one year (VO_2 max values over 69 $\text{mlO}_2/\text{kg}/\text{min}$).

Thus, for elite athletes, higher intensities may not only be optimal, but necessary for enhancement of whole body oxygen flux. Anatomical work has shown that low training intensities produced little to no enhancement of factors affecting oxygen flux in highly fit individuals (Midgley et al., 2006). In addition, elite athletes do not experience the same plateau in stroke volume as less fit individuals (Gledhill et al., 1994). As noted earlier, the ability to improve VO_2 max is negatively related with current VO_2 max (i.e., those with high maximal values have a harder time continuing to increase). HIT, however, can improve VO_2 max in those athletes thought to have reached their genetic potential (Gaskill et al., 1999; Midgley et al., 2006).

A possible reason for elite athletes continuing to train at high volume is to maintain a higher training load than with HIT alone. Total training load is determined not only by intensity, but also by duration and frequency (Pollack, 1977). Although HIT stimulates enhancement of the respiratory system more effectively than LIT, the physical stress of HIT limits the volume of training that can be performed. Thus, elite athletes can achieve a higher total training load by supplementing high intensity training with low intensity training. However, if an athlete's main focus is to accumulate as much volume as possible, the overall training load can lead to residual fatigue which compromises the higher intensity workouts, leading to a lower achieved intensity and resulting in less than optimal signaling for positive adaptation.

In conclusion, there is a disagreement between coaching dogma and the scientific literature on the optimal training intensity for the enhancement of oxygen flux. For less fit individuals, intensities as low as 40% of VO_2 max can elicit maximum stroke volumes, and thus high intensity training is not necessary to stimulate the growth of new capacity. However, due to the presence of highly developed oxidative pathways, and the fact that stroke volume does not plateau in elite athletes, a higher intensity of training is needed in order to maximize oxygen extraction from the atmosphere and delivery to the working muscle.

Implications

Based on the literature reviewed, I would recommend a program of training focused on HIT training, with overall volume being a secondary concern. These suggestions are gender-neutral. The planning for this program would be similar to a classical volume based program, with a 12-month season (macrocycle), broken down into months (mezocycles), which in turn are broken down into weeks (microcycles). I would recommend breaking the macrocycle into only four phases, *Recovery*, *Build*, *Condition*, and *Sharpen* (see Table 1). At the beginning of the training season, the *Recovery* period needs to be long enough to ensure adequate physical and mental recovery from the previous season (one mezocycle). After the athlete has sufficiently recovered, the *Build* period focuses on raising the weekly workload up to that of the *Condition* period (one to two mezocycles). Once goal workload has been achieved, it is maintained throughout the *Condition* period (length depends upon the length of the total season). Beginning 1-2 mezocycles before the goal performance time, the *Sharpen* period should be of sufficient time to enable the athlete to reach peak performance. The *Sharpen* phase focuses on developing the anaerobic capacity and familiarity with high speeds required for racing.

Table 1

Phase	Length	Focus
Recovery	1 Mezocycle	Physical and mental rejuvenation
Build	2 Mezocycles	Returning to goal training workload
Condition	7 Mezocycles	Maintaining goal training workload
Sharpen	2 Mezocycles	Build anaerobic capacity and comfort at high speed

Recovery Phase. As mentioned above, the key focus of the *Recovery* phase is mental and physical recovery. Athletes should remain active, but do so in ways unlike their usual training modes (i.e., runners should cycle and swim). Workload should be less than half than that during the *Sharpen* phase (7.5 hours/week vs. 15 hours/week, same intensity level). This should also be the planning time of the year. Coaches and athletes should meet and perform goal setting and detailed training planning for the next season.

Build Phase. The goal of the build phase is to transition from the *Recovery* period to the *Condition* period. This should be done smoothly and easily to avoid overtraining or injury. However, since injury has been associated with volume of training, it is possible that the injury load is less in a high intensity training plan of the same workload (Brill & Macera, 1995; Marti, Vader, Minder, & Abelin, 1988) The increase in training load should be accomplished by first raising volume to goal levels, then intensity (see Table 2). For example, if the goal workload for the conditioning phase is 15 hours/week with 20% HIT, then athletes would begin with perhaps 10% HIT, build to 15 hours/week, and then begin to introduce more HIT over the following three weeks. If it takes more than two months to make this transition, the athletes goal workload is too high.

Table 2

Workload During Build Period

Week	Volume	Intensity
1	Low	Low
2	Medium	Low
3	Goal	Low
4	Medium	Low
5	Medium	Medium
6	Goal	Medium
7	Goal	Goal
8	Low	Medium

Condition phase. This phase is the focus of the entire year, and much thought should go into what the goal workload is. The premise behind this phase is to train hard at a moderate volume in order to allow adaptations to the speed with which the athlete completes the training. Thus, this period is what separates the high volume program from the high intensity program. A high volume program would continue to build in volume throughout the condition period with the idea that volume is the most important training stimulus. For example, during an off season fitness building period, historically the prescription could be 20 hours of training with 90% of that LIT (see Table 5). However, a high intensity program based on the review of the scientific literature would focus on maintaining a similar volume through the condition period and increasing the workload by increasing the speed at which each workout is completed (see Table 3).

Table 3

<i>Workload During Condition Period</i>		
Week	Volume	Intensity
1	Goal	Goal
2	Goal	Goal
3	Goal	Goal
4	Medium	Medium

The weekly training in this period should focus on maintaining a moderate volume of high intensity training in order to signal the oxygen pathways to adapt positively. An example of an athlete training 10.5 hours per week, but with a high amount of HIT is shown in Table 4.

Table 4

Weekly Workouts, Condition Phase

Day	Training
Monday AM:	Off
PM:	Off
Tuesday AM:	0:30 @ 60-70% HRmax
PM:	1:30 @ 75-85% HRmax
Wednesday AM:	0:30 @ 60-70% HRmax
PM:	1:00 @ 60-70% HRmax +0:30 @ 90-100% HRmax
Thursday AM:	0:30 @ 60-70% HRmax
PM:	Off
Friday AM:	0:30 @ 60-70% HRmax
PM:	2:00 @ 75-85% HRmax
Saturday AM:	2:00 @ 75-85% HRmax
PM:	Off
Sunday AM:	2:30 @ 75-85% HRmax
PM:	Off
TOTAL HOURS:	10.5

Sharpen phase. The foundation of this phase must be 2-3 HIT sessions per week, with LIT workouts in between solely to aid in recovery. Other than these recovery workouts, all the focus between workouts is recovering sufficiently to be able to perform at maximum levels for the next training session. The goal of the *Sharpen* phase is to increase anaerobic capacity, while maintaining aerobic fitness. Volume should be much lower, and percentages of HIT work should increase. Since athletes usually do (and should) race often during this phase of training, great care should be taken not to exhaust them in any given workout, as this could lead to residual fatigue in their next race.

Table 5

Weekly Workouts, Volume-Based vs. Intensity-Based

Day	Volume-Based Training	Intensity-Based Training
Monday AM:	Off	Off
PM:	Off	Off
Tuesday AM:	2:00 @ 60-70% HRmax	0:30 @ 60-70% HRmax
PM:	2:00 @ 60-70% HRmax	1:00 @ 60-70% HRmax + 0:30 @ 85-95% HRmax
Wednesday AM:	0:30 @ 60-70% HRmax	0:30 @ 60-70% HRmax
PM:	1:00 @ 60-70% HRmax + 0:30 @ 90-100% HRmax	1:00 @ 60-70% HRmax
Thursday AM:	2:00 @ 60-70% HRmax	1:00 @ 60-70% HRmax + 0:30 @ 85-95% HRmax
PM:	2:00 @ 60-70% HRmax	0:30 @ 60-70% HRmax
Friday AM:	2:00 @ 60-70% HRmax	0:30 @ 60-70% HRmax
PM:	1:00 @ 60-70% HRmax + 1:00 @ 75-80% HRmax	0:30 @ 60-70% HRmax
Saturday AM:	2:00 @ 60-70% HRmax	1:00 @ 60-70% HRmax + 0:30 @ 85-95% HRmax
PM:	Off	Off
Sunday AM:	4:00 @ 60-70% HRmax	2:00 @ 75-85% HRmax
PM:	Off	Off
TOTAL HOURS:	20	10

Based on the reviewed articles, I have presented a training plan that is both scientifically based and easy to access by coaches and endurance athletes alike. My hope is that this paper will help to bridge the gap in knowledge between those who study the science of endurance athletics, and those who practice it as their livelihood.

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