

# High Intensity Interval Training: Cardiorespiratory Adaptations, Metabolic and Performance

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**Abstract** High Intensity Interval Training (IT) involves repeating maximum and/or supramaximal sprints for short and/or long periods ( $\geq 90 - 120\%$  of the maximum oxygen consumption speed [ $v\text{VO}_{2\text{máx}}$ ];  $\geq$  the Maximal Lactate Steady State [MLSS]) separated by recovery periods, which may be passive or by performing exercises in moderate intensity (passive and active pausing, respectively). However, such cardiorespiratory, metabolic, morphological, and performance adaptations depend on manipulating acute variables which guide the continuous training process, including volume, intensity, different times and types of recovery between series, and weekly training schedule. With this in mind, the study aimed to review and discuss various results investigating the effects of IT in cardiorespiratory, metabolic, and performance parameters in athletes and physically active individuals. The most relevant original scientific studies as of September, 2015, were analyzed, using the following databases: Science Citation, Index, Scopus, Sport Discus, The Scielo, and National Library of Medicine, combining the following keywords: endurance training, running training, recovery, repeated sprint, high intensity, speed endurance, interval training, anaerobic, and rest interval. It has been concluded literature has not clearly demonstrated the best combination of training variables, which allow for better efficiency in increasing cardiorespiratory, metabolic, and performance parameters. Most studies have showing effort intensity, when close to maximum cardiorespiratory capacity, results in increased stress and, therefore, greater cardiorespiratory and metabolic adaptations

**Keywords** Endurance training, Interval training, High intensity

## 1. Introduction

High Intensity Interval Training (IT) involves repeating maximum and/or supramaximal sprints for short and/or long periods ( $\geq 90 - 120\%$  of the maximum oxygen consumption speed [ $v\text{VO}_{2\text{máx}}$ ];  $\geq$  the Maximal Lactate Steady State [MLSS]) separated by recovery periods, which may be passive or by performing exercises in moderate intensity (passive and active pausing, respectively).

It has been reported, in 1912, Olympic 10,000 meter champion runner Hannes Kolehmainen was already using IT in training: he would run in 3 to 5 repetitions of 3 minutes and 5 seconds each, for 1000 meters, at a speed of 19 km/h [1]. In 1920, Hill et al. conducted pioneering experiments with intermittent exercises [2-3] However, IT was initially described in a scientific journal by Reindell &

Roskamm [4] and Reindell et al. [5], rising in popularity after World War II in the 1950s thanks to Olympic champion Emil Zatopek.

In 1960, researcher Per Aløf Astrand et al. [6] developed the first scientific basis with several papers comparing the metabolic responses of long interval training (LIT) and short interval training (SIT) under MLSS or  $v\text{VO}_{2\text{máx}}$  [1]. Within the same group of researchers, Christensen et al. [7] showed that SIT (5 to 30 seconds) under  $v\text{VO}_{2\text{máx}}$  separated by 10 to 40 second pause intervals could reach  $v\text{VO}_{2\text{máx}}$  during subsequent sprints without significantly accumulating lactate. On the other hand, the referred authors have also showed that LIT (2 to 6 minutes) under  $v\text{VO}_{2\text{máx}}$  increase the plasmatic concentrations of lactate due to maintaining contractile effort. The authors have considered that intensity under  $v\text{VO}_{2\text{máx}}$  is an excellent tool for increasing maximum oxygen consumption ( $\text{VO}_{2\text{máx}}$ ), as all cardiorespiratory parameters are in their maximum capacities [6].

In the last few decades, IT has been the subject of scientific research and debates, due to its potential to bring

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benefits to healthy and sick individuals, and athletes, through several neuromuscular adaptations (increment in the content of energy substrates and oxidative and glycolytic enzyme activity, improvements in recruiting and synchronization of motor units, and increase in conducting speed of action potential), molecular adaptations (mitochondrial biogenesis through phosphorylation of AMPK, CaMK, and PGC-1 $\alpha$ ), metabolic adaptations (improvements in transporting protein involved in relating pH and in the efficiency in removing of lactate co-transporter and of H<sup>+</sup> of the muscle to the blood and blood tissue; phosphate, bicarbonate, protein, hemoglobin, mitochondria), immunologic improvements (increased leukocytes activity and subpopulations), and, especially, cardiorespiratory adaptations (increase in  $\dot{V}O_{2\max}$ ) [8-10], in addition to the ability to treat chronic-degenerative and inflammatory diseases, such as diabetes, hypertension, and obesity.

However, such cardiorespiratory, metabolic, morphological, and performance adaptations depend on manipulating acute variables which guide the continuous training process, including volume, intensity, different times and types of recovery between series, and weekly training schedule.

With this in mind, the study aimed to review and discuss various results investigating the effects of TI in cardiorespiratory, metabolic, and performance parameters in athletes and physically active individuals.

## 2. Main Body

The most relevant original scientific studies of September, 2015, were analyzed, using the following databases: Science Citation, Index, Scopus, Sport Discus, The Scielo, and National Library of Medicine, combining the following keywords: endurance training, running training, recovery, repeated sprint, high intensity, speed endurance, interval training, anaerobic, and rest interval. Studies investigating the effects of IT with different manipulations of the acute training variables (as well as different populations were considered inclusion criteria.

## 3. Manipulation of IT Variables

### Intensity

The primary function of the cardiorespiratory system during maximum effort is to provide a continuous flow of oxygen and nutrients to the skeletal muscle and to remove metabolites of cellular breathing [11-12]. This capacity represents a determining mechanism in performance prediction of endurance athletes and athletes of sports categories in which running is an integral part. It is also an excellent tool in the prognosis of heart and/or respiratory limitations [13-15].

One of the ways to assess the cardiorespiratory system, as established by literature, is the maximum incremental test,

considered a “golden standard” tool, due to its ability to analyze, the nervous, cardiopulmonary, and metabolic systems through submaximal (L1 and L2) and maximal ( $\dot{V}O_{2\max}$ ) ventilation parameters, and their respective speeds ( $\dot{V}L1$ ,  $\dot{V}L2$ ,  $\dot{V}O_{2\max}$ ), the reason for respiratory exchanges (R), and the maximum heart rate (FC $\max$ ). All of which may be used in prescribing intensity in individual and specific training [15]. However, the quantification of such parameters greatly depends on the exercise protocol used, and the trainability levels of the individual being assessed [16].

L1 is characterized by an increase in glycolytic participation, even if under predominantly aerobic conditions, and by the abrupt increase in the concentration of CO<sub>2</sub> compared to O<sub>2</sub>, due to the exponential increase in effort intensity and ATP hydrolysis [17], resulting a significant increase of H<sup>+</sup> in the muscle cytosol. It is responsible for decreasing the muscle pH and subsequent fatigue [17].

Such biological phenomenon promotes lactate dehydrogenase enzyme activity, which catalyzes the decrease of the pyruvate to lactate from the mitochondrial inefficiency, which, by itself in  $\dot{V}L1$ , cannot conduct tamponade [11, 17], and from the lactate's ability to tampon and co-transport the H<sup>+</sup> to the blood, where it will be reused as energy by various tissues or glucose.

Such transport of lactate is carried out through a class of monocarboxylate transporters (MCTs) in the plasma membrane of the muscle tissue [18 -23]. In addition to the lactate as a tamponing agent, other intracellular fixed tampons, such as proteins, carnosine, inorganic phosphate (Pi) and, especially, bicarbonate ion (HCO<sub>3</sub><sup>-</sup>) also take part in the tamponade process of the H<sup>+</sup> in the plasma. This reaction promotes an increase in CO<sub>2</sub> production, known as non-metabolic CO<sub>2</sub>, due to the sum of CO<sub>2</sub> produced by the Krebs cycle. Afterwards, the CO<sub>2</sub> is diffused to inside the red blood cells (RBCs), transforming into carbonic acid (H<sub>2</sub>CO<sub>3</sub>) through the carbonic anhydrase enzyme, contributing to the release of CO<sub>2</sub> in the lungs and, subsequently, the HCO<sub>3</sub><sup>-</sup> regeneration [22-23].

It is suggested this biochemical and cardiorespiratory point is marked by the beginning of the contribution of anaerobic metabolism, by the maximum capacity of the oxidative metabolism for maintaining effort, and by the efficient tamponade capacity [24]. An efficient method for detecting this point is known as V-slop, as it determines the moment the linearity of carbon dioxide ( $\dot{V}CO_2$ ) production is broken off, in intensity between 70 and 75% of the  $\dot{V}O_{2\max}$ , according to trainability level [13, 25].

The PCR or L2 is the point where the greater dependence in lactic anaerobic metabolism occurs to form ATP, due to the constant increase of effort intensity [23], which leads to increased production of H<sup>+</sup> due to continuous contractile work.

This reaction leads to a decrease in blood pH, which is promptly detected by peripheral chemoreceptors (aortic bodies and carotid bodies) and central chemoreceptors,

leading to an increase in breathing rate (VE) by the respiratory centers, as well as hyperventilation. This aims to reestablish pH and decrease partial pressure of carbon dioxide (PCO<sub>2</sub>) [26-27]. Literature suggests the intensity of effort in vL2 is to be set between 75 and 85% of VO<sub>2</sub>max [25]. Such effort can be observed by the loss of linearity between VE in relation to VCO<sub>2</sub>.

Awareness of the speeds corresponding to these variables is vital for prescribing training intensities accurately, and to assess the effects of different methods in the increment of endurance performance.

With this in mind, Lucia *et al.* [16] established and classified three distinct training intensity zones, based on what is determined as L1 and L2: Zone 1 represents low intensity (below L1); Zone 2 represents moderate intensity, fluctuating between L1 and L2; Zone 3 represents intensities above L2. Nevertheless, after the point corresponding to L2, upon continuous increase of effort intensity, obtaining, transporting, and using oxygen (VO<sub>2</sub>max) reaches its maximal capacity, this being one of the major determining factors for endurance performance, as well as being of particular interest to researchers, coaches, and athletes [12]. This reaction may be observed through a steady state in the VO<sub>2</sub> under maximal exercise intensity (vVO<sub>2</sub>max) [15], and it is also speculated as a possible fourth zone.

For many years, it had been widely accepted that the efforts submaximal intensities (L1 and L2) in certain regularity, or maximal intensities in shorter periods (SIT) were the most efficient way to increase VO<sub>2</sub>max [28].

However, in regards to high level runners, cardiorespiratory adaptations stemming from these intensities are likely to have occurred already [29]. Consequently, this leads to a need for greater gains in aerobic strength and anaerobic capacity. For this reason, many researchers agree and recommend that, in order to increase VO<sub>2</sub>max, it is important to use TI protocols allowing for speeds close to VO<sub>2</sub>max, at vVO<sub>2</sub>max, or over vVO<sub>2</sub>max [30-32], as well as maintaining this intensity for longer periods [33].

vVO<sub>2</sub>max has been used to determine the remaining time in maximal effort until voluntary exhaustion (Tlim) [1, 12], this being a parameter correlated to performance in long distance running, such as 1500 to 5000 meter, 10 kilometers, and 21 kilometers races [3, 34]. Both vVO<sub>2</sub>max and Tlim have been used to indicate, respectively, aerobic strength and anaerobic capacity to individually prescribe the duration, the amount of stimuli in interval session, as well as athlete performance [35]. However, there are severe difficulties of possible comparisons of Tlim values both between studies or otherwise, due to the wide variability of training protocols and of individuals' trainability level [35].

Billat *et al.* [1] and Smith *et al.* [36] manipulated interval training sessions in close intensities or in vVO<sub>2</sub>max, in order to check how much Tlim had increased, as well as in the remaining time in VO<sub>2</sub>max or over its percentages (90 or 95% vVO<sub>2</sub>max). Furthermore, other experiments

have been reporting variations between 2.5 minutes [35] and 10 minutes [28] of Tlim in vVO<sub>2</sub>max. This shows great variation in different endurance athletes' performance. Based on this idea, [35] assessed the Tlim in vVO<sub>2</sub>max for different sports (cycling, kayaking, swimming, and running) using specific ergometers for each sport. The results revealed significant differences between cyclists (222 seconds) kayak rowers (376 seconds), highlighting differences between sports.

It is speculated this respective intensity is strongly recommended for reaching increased VO<sub>2</sub>max, due to the amount of effort, which allows for high VO<sub>2</sub>max percentages during exercise and cardiorespiratory (cardiac output), metabolic (tamponing capacity), and neuromuscular (motor unit recruitment) disorders [37].

Further studies have shown improvements in VO<sub>2</sub>max, vVO<sub>2</sub>max, anaerobic performance, and blood flow through intensities exceeding vVO<sub>2</sub>max (120 – 150% vVO<sub>2</sub>max; zone 5) [38-40].

As it was discussed in the above topic, IT in intensities under vVO<sub>2</sub>max is an excellent tool for improving aerobic strength and anaerobic capacity in runners of all levels [6, 34]. Recently, several studies have assessed the metabolic and cardiorespiratory changes caused by IT with different times and fixed intensities (15 seconds until Tlim; 80% and 140% of vVO<sub>2</sub>max) [36, 41]. However, there are still methodological inconsistencies in manipulating the acute variables guiding the continuous physical training process and that directly influence the extent of VO<sub>2</sub>max, and in the time elapsed in high percentages of VO<sub>2</sub>max (t90% - t95% VO<sub>2</sub>max).

This does not explain clearly the best combination of intensity, effort duration, and recovery, as well as the recovery intensity when actively performed, and the amount of effort inherent to the protocol [39].

The pioneering experiments by Astrand *et al.* [6] demonstrated the main physiological change between SIT and LIT. This group of researchers reported SIT (15 – 30 second effort in VO<sub>2</sub>max spread by 30 second recovery) can reach VO<sub>2</sub>max without significantly accumulating lactate (2.2 mmol  $\pm$ 1), while LIT (2 to 6 minutes in vVO<sub>2</sub>max spread by 2 to 6 minutes of recovery) in vVO<sub>2</sub>max increases plasma concentration of lactate due to maintaining contractile effort, which, until then, was considered the cause of peripheral fatigue [42].

With this in mind, several experiments with SIT and fixed durations were conducted with the aim to analyze the capacity to reach VO<sub>2</sub>max and the time elapsed in high percentages of VO<sub>2</sub>max without significantly accumulating lactate [1, 32].

However, the concepts of cause and effect between the production of lactate, lactic acidosis, and muscle fatigue were dismissed with the article by Robergs [17]. Several experiments have been showing high production of lactate would only indicate a particular magnitude of ATP production through lactic anaerobic metabolism [19] with no concomitant reductions in strength and performance

levels [43].

Moreover, high concentrations of muscle lactate stimulate its removal through MCT4 [1, 44], which is one of the most required tamponing mechanisms for maintaining pH. Additionally, literature shows SIT cannot reach VO<sub>2</sub>max right during the initial effort, but rather through continuous repetitions, as well as maintaining high percentages of VO<sub>2</sub>max for short periods, due to stimulation time [3].

Based on this hypothesis, it has been suggested Tlim is the most adequate variable for it can also handle increased times of high VO<sub>2</sub>max percentages aside from its ability to reach VO<sub>2</sub>max right at the initial effort [1, 28, 30, 33].

Billat et al. [1] Billat et al. [34], and Smith et al. [36] used 3 to 5 repetitions based on the Tlim percentages in well-trained runners, and discovered an improvement in time elapsed in high percentages of VO<sub>2</sub>max, in vVO<sub>2</sub>max, and running performance in 3,000 meters in a few weeks' training (2 to 4 weeks).

Furthermore, Franch et al. [45] investigated the effects of 6 weeks' (3 sessions per week) continuous running under 90% of the vVO<sub>2</sub>max, of SIT (15 seconds of effort divided by 15 seconds of passive recovery), and LIT (4 minutes of effort divided by 2 minutes of passive recovery) in intensities of over vVO<sub>2</sub>max. Researchers found an increase in vVO<sub>2</sub>max, VO<sub>2</sub>max and in economic running in continuous training (9%, 5.9%, and 3.1% respectively), both under LIT (10%, 6%, and 3%, respectively), and SIT (3.6%, 4%, and 0.9%, respectively).

Such results from the aforementioned studies substantially support the notion that SIT under vVO<sub>2</sub>max has decreased physiological effects, especially in athletes with higher Tlim [46]. On the other hand, LIT in vVO<sub>2</sub>max, through severe contractile work, has greater capacity to stress the cardiorespiratory (VO<sub>2</sub>max and cardiac output), metabolic (tamponing capacity), neural (higher motor unit recruitment), molecular (mitochondrial density) mechanisms and the energy supply systems, resulting in more pronounced adaptations [1, 33, 41].

### **Different times and types of pauses in IT**

Continuous contractile work during high intensity efforts leads to changes in intra-muscle and cardiorespiratory biochemical homeostasis, resulting in an increase in the production of muscle metabolites. Consequently, this also leads to physiological disorders, which are largely responsible for decreasing performance during maximal repetitive efforts [47].

Therefore, other than intensity and duration of effort, manipulating and combining different times and types of pauses, as well as the intensity of the pause when it is active, have been considered particularly important by sports researchers. They may be seen as essential in adaptive metabolic IT processes [48].

The type and time of pause may directly impact the severity of metabolic disorders, such as: ability to resynthesize PCr, maintain intracellular and extracellular

pH, prevent the accumulation of metabolites, maintain synaptic and neural properties, as well as conducting the oxidation of lactate following exercise, through the production of mitochondrial ATP by means of oxidative phosphorylation. This contributes to the continuous production of strength and speed of the shortening of muscle tissue [10] and IT performance [49].

The literature has been demonstrating active pauses are more efficient in removing lactate by increasing blood flow [50-52]. This could lead to improved regulation of tamponing mechanisms and the resulting positive effect in performance. Dorado et al. [53] found improved performance from active recovery between maximal efforts, in relation to passive paused, through a possible increase in exercise yield.

Gupta et al. [51], and Taoutaou et al. [52] discovered that active pause between 30 and 60% of vVO<sub>2</sub>max, for at least 20 minutes, leads to high lactate oxidation. However, faster lactate oxidation does not necessarily imply faster recovery, with subsequent improvements in repeated maximal efforts' performance.

Bonen & Belcastro [50], compared the effects of the type of pause in removing blood lactate and in strength testing. One group of individuals underwent 1 minute of effort at 150% of vVO<sub>2</sub>max divided by active recovery during 20 minutes, at 30% of vVO<sub>2</sub>max. While another group underwent passive pause. There were no differences in performance (maximum strength, potency output, and fatigue) between the different types of pause, although there was an increase in the removal of lactate by active paused, compared to passive pause.

Toubekis et al. [54] also found increased removal of blood lactate by active pause (120 s) after 25 meter swimming sprints, with subsequent decrease in performance.

Based on these studies, many researchers have been demonstrated increase or better potential of performance maintenance through passive pause [55-56]. Passive pausing appears to provide better recovery of the main energy substrates (PCr) between the maximal efforts, in the oxidative capacity and regulation systems of pH, and in ventilator parameters [54].

This leads to an increase or better maintenance of high intensity continuous effort performance. Nevertheless, the magnitude of such metabolic and adaptive responses also differ based on manipulating different passive pause times (which is to say, the longer the pause, the better will performance maintenance and resynthesize rate is. At the same time, the shorter the pause, the more physiological disorders may occur alongside decreased performance maintenance).

Toubekis et al. [54] showed that long passive pause (120 second pause) between maximal 25 meter swimming efforts has better capacity to improve and maintain performance compared to short passive pause (45 seconds), and compared to long (120 seconds) and short (45 seconds) active pauses. This is because of increased PCr resynthesis

during long passive pause, which key for improved performance.

A possible explanation for these performance differences between passive and active pausing is that, during active pause, more energy is required, which decreases oxyhemoglobin reoxygenation and, consequently, loss of balance between oxygen supply and demand. As such, PCr resynthesis is impaired, and such resynthesis depends on the oxidative pathway [57].

Aside from metabolic and performance parameters, literature has also shown several acute studies which investigated the impacts of the different pauses in the VO<sub>2</sub>max range, in the time elapsed in high VO<sub>2</sub>max percentages, and in Tlim [58].

Most of these studies have suggested the use of active pauses allows for better time to reach VO<sub>2</sub>max and for better remaining time between 90% and 95% of VO<sub>2</sub>max compared to passive pause (46, 59-60).

Millet *et al.* [46], and Tardieu-Berger *et al.* [56] used efforts in vVO<sub>2</sub>max or over vVO<sub>2</sub>max, alternating with active pause (50% of vVO<sub>2</sub>max), and showed active pause allows for increased speed in reaching VO<sub>2</sub>max (movement of VO<sub>2</sub>max). This can be explained by the fact that VO<sub>2</sub>max and FCmax are kept under increased values when pausing [59] improved Tlim [1], as well as the time elapsed in high VO<sub>2</sub>max percentages ( $t_{90\%} \text{ vVO}_{2\text{max}} = 338$  seconds) [46], ( $t_{95\%} \text{ vVO}_{2\text{max}} = 178$  seconds) [60].

Also, Gorostiaga *et al.* (1991) showed that 30 seconds in vVO<sub>2</sub>max divided by passive pause do not let individuals reach VO<sub>2</sub>max.

In contrast, certain studies do not clash with the previous results. Dupont *et al.* [61] compared the effects of 15 second effort under 120% of the maximal aerobic speed (MAS) divided by passive and active pause (50% of MAS) in Tlim. The researchers theorized Tlim would be higher with the usage of active pause in relation to passive. However, this hypothesis was rejected, as Tlim was significantly higher with passive pause. One possible explanation is the high energy demand during active pause, while passive pause appears to have increased PCr resynthesis and O<sub>2</sub> availability [38].

Dupont *et al.* [56] compared the effects of the different types of pauses in the time elapsed for high VO<sub>2</sub>max percentages during 15 second efforts under 120% of MAS. It was suggested active pause could allow for increased elapsed time in high VO<sub>2</sub>max percentages, but results showed that the time elapsed in high VO<sub>2</sub>max percentages was not significantly different between active and passive pause. On the contrary: higher time limits in passive pause were detected, as well as increased number of repetitions, leading to speculations that passive pause may promote better recovery quality [57].

According to the referred studies, Thevenet *et al.* [62] investigated 30 seconds of effort under 105% of MAS, divided between passive and active pause (50% of MAS), based on the idea that active pause leads to increased

VO<sub>2</sub>max x movement speed and, consequently, between the 90% and 95% of VO<sub>2</sub>max range, as well as increased remaining time in these intensities. Results were not in favor of this theory, as Tlim was significantly higher during passive pause in relation to active pause. Moreover, there were no major changes between the pauses in the time elapsed in these percentages [62].

Based on the topics discussed so far, it is clear that a number of protocols have been proposed, aiming to increase VO<sub>2</sub>max and the ability to support high VO<sub>2</sub>max percentages, due to their key factors in endurance sports, as well as a various sports competitions [1]. Nonetheless, literature has not clearly demonstrated the best combination of training variables, which can provide effective increase in such parameters. Most cross-section studies comparing the effects of different pauses in metabolic, performance, VO<sub>2</sub>max range, and time elapsed parameters in high VO<sub>2</sub>max percentages used SIT as basis (90% - 140% of vVO<sub>2</sub>max; 15 to 30 seconds [39].

Germano [63] acutely verified the influence of different times (2 and 8 minutes) and types of recovery (active x passive) in IT in cardiorespiratory, metabolic, and performance parameters. Training sessions were made up of 5 EM on a treadmill up until the time limit (Tlim) interspersed by different recovery models.

Results show that, when comparing intra-recovery, long and passive (8 minutes) was responsible for maintaining performance throughout the 5 EM, while short and passive (2 minutes), and both active recoveries (2 and 8 minutes) led to significant decrease in performance [63].

On the other hand, on the very same study conducted by Germano [63], when comparing passive recoveries, no differences between the two passive recoveries were observed. Additionally, no significant changes were detected in the concentrations and removal of lactate between none of the recoveries.

## 4. Conclusions

It has been concluded literature has not clearly demonstrated the best combination of training variables, which allow for better efficiency in increasing cardiorespiratory, metabolic, and performance parameters. Most studies have showing effort intensity, when close to maximum cardiorespiratory capacity, results in increased stress and, therefore, greater cardiorespiratory and metabolic adaptations. Also, manipulating passive recoveries between maximum efforts appears to have better effects on performance. On the other hand, active recoveries seem to lead to poorer performance. With this in mind, there needs to be broader studies with different acute training variable manipulations in different individuals, so that the extent of what is currently known about the effects of IT may be expanded.

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