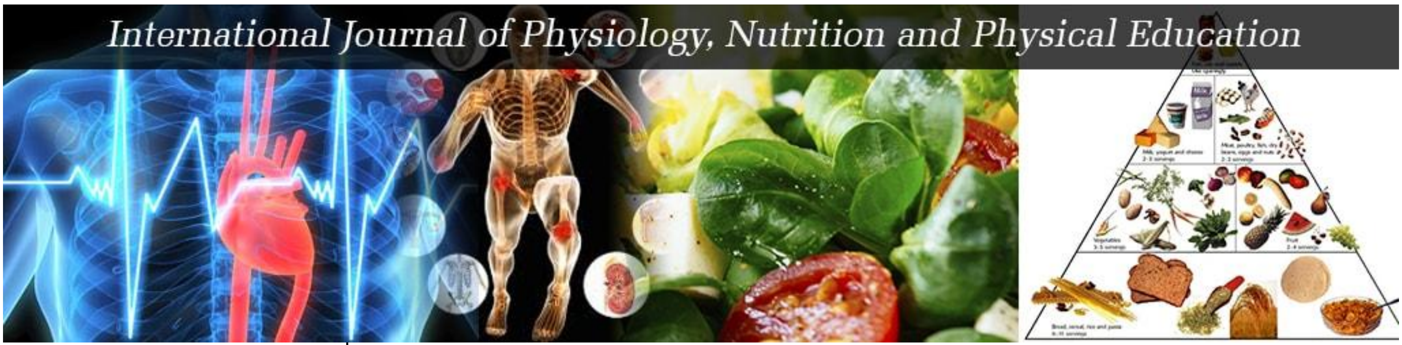

ISSN: 2456-0057
IJPNPE 2018; 3(1): 196-198

www.journalofsports.com
Received: 08-11-2017
Accepted: 09-12-2017

Anantarup Sen Sarma
Research Scholar, Department of Physical Education, University of Kalyani, West Bengal, India

Correspondence
Anantarup Sen Sarma
Research Scholar, Department of Physical Education, University of Kalyani, West Bengal, India

Lactate threshold training

Anantarup Sen Sarma

Abstract
Lactate threshold (LT) training Lactate threshold (LT) training is a popular method of improving high intensity endurance performance. While V02 Max may indicate an athlete's genetic potential and natural ability, their lactate threshold can be increased substantially with the right training program. Athletes often use their lactate threshold to determine how to train and what sort of a pace they can maintain during endurance sports. Because the lactate threshold can be increased greatly with training, many athletes and coaches have devised complicated training plans to increase this value. This article will explain and discuss how lactate threshold training principles can be incorporated into your client’s training program.

Keywords: Lactate threshold, training, performance

Introduction
The terms lactic acid and lactate, despite biochemical differences, are often used interchangeably. Fitness professionals have traditionally linked lactic acid or ‘the burn’ with an inability to continue an intensive exercise bout at a given intensity. Although the conditions within the exerciser’s muscle cells have shifted towards acidosis, lactate production itself does not directly create the discomfort (acidosis) experienced at higher intensities of exercise. It is the proton (H+) accumulation, coinciding with, but not caused by lactate production, that results in acidosis, impairing muscle contraction, and ultimately leading to the ‘burn’ and associated weariness (Robergs, Ghiasvand, Parker 2004) [9]. The increased proton accumulation occurs most notably from the splitting of ATP (the body’s energy liberating molecule) by the muscle protein filaments, in order to sustain vigorous muscle contraction. Interestingly, the lactate production is proposed to be a physiological event to ‘neutralize’ or ‘retard’ the exerciser’s muscle acidic environment (Robergs, Ghiasvand, Parker 2004) [9]. Thus, lactate accumulation, which for years has been associated with the cause of the burn, is actually a beneficial metabolic event aimed at diminishing the burn. Scientists denote conditioning at this physiological state as lactate threshold training. Fitness professionals can utilize this knowledge to enhance the cardiovascular endurance performance of their students and clients. All world and Olympic endurance athletes incorporate lactate threshold training into their workouts. This article will explain and discuss how lactate threshold training principles can be incorporated into your client’s training program.

Lactate Threshold and Endurance Performance
Traditionally, maximal oxygen uptake (VO2max) has been viewed as the key component to success in prolonged exercise activities (Bassett & Howley 2000) [2]. However, more recently scientists have reported that the lactate threshold is the most consistent predictor of performance in endurance events. Studies have repeatedly found high correlations between performance in endurance events such as running, cycling, and race-walking and the maximal steady-state workload at the lactate threshold (McKardle, Katch, & Katch 1996) [8].

What is the Lactate Threshold
At rest and under steady-state exercise conditions, there is a balance between blood lactate production and blood lactate removal (Brooks 2000) [4]. The lactate threshold refers to the intensity of exercise at which there is an abrupt increase in blood lactate levels

(Roberts & Robergs 1997) [10]. Although the exact physiological factors of the lactate threshold are still being resolved, it is thought to involve the following key mechanisms (Roberts & Robergs 1997) [10]:

Decreased lactate removal

1) Increased fast-twitch motor unit recruitment
2) Imbalance between glycolysis and mitochondrial respiration
3) Ischemia (low blood flow) or hypoxia (low oxygen content in blood)

1) Lactate Removal

Although once viewed as a negative metabolic event, increased lactate production occurring exclusively during high-intensity exercise is natural (Robergs, Ghiasvand, Parker 2004) [9]. Even at rest a small degree of lactate production takes place, which indicates there must also exist lactate removal or else there would be lactate accumulation occurring at rest. The primary means of lactate removal include its uptake by the heart, liver, and kidneys as a metabolic fuel (Brooks 1985) [5]. Within the liver, lactate functions as a chemical building block for glucose production (known as gluconeogenesis), which is then released back into the blood stream to be used as fuel (or substrate) elsewhere. Additionally, non-exercising or less active muscles are capable of lactate uptake and consumption. At exercise intensities above the lactate threshold, there is a mismatch between production and uptake, with the rate of lactate removal apparently lagging behind the rate of lactate production (Katz & Sahlin 1988) [7].

2) Increased Fast-Twitch Motor Unit Recruitment

At low levels of intensity, primarily slow-twitch muscles are recruited to support the exercise workload. Slow-twitch muscle is characterized by a high aerobic endurance capacity that enhances mitochondrial respiration, which is the aerobic ATP energy production system. With increasing exercise intensity there is a shift towards the recruitment of fast-twitch muscles, which have metabolic characteristics that are geared towards glycolysis (an anaerobic energy pathway). The recruitment of these muscles will shift energy metabolism from mitochondrial respiration more towards glycolysis, which will eventually lead to increased lactate production (Anderson & Rhodes 1989) [1].

3) Imbalance between Glycolysis and Mitochondrial Respiration

At increasing exercise intensities, there is an increased reliance on the rate in the transfer of glucose to pyruvate through the reactions of glycolysis. This is referred to as glycolytic flux. Pyruvate, which is the final product of glycolysis, can either enter the mitochondria for further biological breakdown (for eventual synthesis of energy) or be converted to lactate. There are some researchers who believe that at high rates of glycolysis, pyruvate is produced faster than it can enter into the mitochondria for mitochondrial respiration (Wasserman, Beaver, & Whipp 1986) [11]. Pyruvate that cannot enter the mitochondria will be converted to lactate, which can then be used as fuel elsewhere in the body (such as the liver or other muscles).

4) Ischemia and Hypoxia

For years, one of the primary causes of lactate production was thought to include low levels of blood flow (ischemia) or low levels of blood oxygen content (hypoxia) to exercising muscles (Roberts & Robergs 1997) [10]. However, there is no experimental data indicating ischemia or hypoxia in exercising muscles, even at very intense bouts of exercise (Brooks 1985) [5].

What is the Anaerobic Threshold?

The term anaerobic threshold was introduced in the 1960's based on the concept that at high-intensity levels of exercise, low levels of oxygen (or hypoxia) exist in the muscles (Roberts & Robergs 1997) [10]. At this point, for exercise to continue, energy supply needed to shift from the aerobic energy system (mitochondrial respiration) to anaerobic energy systems (glycolysis and the phosphagen system).

However, there are many researchers who strongly object to the use of the term anaerobic threshold, believing it is misleading. The main argument against using the term anaerobic threshold is that it suggests oxygen supply to muscles is limited at specific exercise intensities. However, there is no evidence that indicates muscles become deprived of oxygen, even at maximal exercise intensities (Brooks 1985) [5]. The second main argument against using anaerobic threshold is that it suggests at this point in exercise intensity, metabolism shifts completely from aerobic to anaerobic energy systems. This interpretation is an overly simplistic view of the regulation of energy metabolism, as anaerobic energy systems (glycolysis and the phosphagen system) do not take over the task of ATP regeneration completely at higher intensities of exercise, but rather augment the energy supply provided from mitochondrial respiration (Robergs, Ghiasvand, Parker, 2004) [9].

Lactate Threshold Training Programs and Workouts

Although the optimal training for lactate threshold improvement has yet to be fully identified by researchers, there are still some excellent guidelines you can follow in generating training programs and workouts in order to optimize the endurance performance of clientele. Research has indicated that training programs that are a combination of high volume, maximal steady-state, and interval workouts have the most pronounced effect on lactate threshold improvement (Roberts & Robergs 1997, Weltman 1995) [10, 12].

High Volume Training

Initially, the best way to improve the lactate threshold levels of your clients is to increase their training volume, regardless of the cardiovascular mode of exercise. Let's create a case study as we discuss lactate threshold training to fully see how to design an effective program. For our case study, let's assume the client is presently doing 100 minutes of cardiovascular exercise a week, with the goal of increasing the total time to 200 minutes per week. Increased training volume should be gradual and in the order of approximately 10-20% per week (Bompa 1999) [3]. For our case study, if the client progressed at 20% per week, it would take approximately four weeks to safely progress to the target weekly volume of 200 minutes per week. The Rating of Perceived Exertion (RPE) scale should be used to prescribe cardiorespiratory exercise intensity during this period. For this high volume training, your client should train at an RPE of 11-12, which subjectively is a light exercise intensity level. Mix up the total time per session of cardiovascular exercise throughout the week, however it works best for the client. However, the minimum bout of cardiovascular exercise should be 10 minutes in duration. The major benefit of increased training volume is an increased capacity for mitochondrial respiration, which is imperative to improvements in lactate threshold.

Maximal Steady-State Training

Steady-state training at the lactate threshold is often referred to as “maximal steady-state” exercise or “tempo runs.” Research has shown that the lactate threshold occurs at 80-90% of heart rate reserve (HRR) in trained individuals and at 50-60% HRR in untrained individuals (Weltman 1995) [12]. Without access to an exercise physiology laboratory to get actual lactate threshold measurements for your clients, the RPE scale will be the most accurate way to determine training intensity for maximal steady-state exercise sessions. Research has shown that RPE is strongly related to the blood lactate response to exercise regardless of gender, training status, type of exercise being performed, or the intensity of training (Weltman 1995) [12]. Findings from studies have indicated that the lactate threshold occurs between 13 and 15 on the RPE scale, which corresponds to feelings of ‘somewhat hard’ and ‘hard’ (Weltman 1995) [12].

Following the build-up in training volume described above, your client may begin maximal steady-state exercise sessions. Collectively, these sessions should consist of no more than 10% of the total weekly volume (Foran 2001) [6]. In our case study, 10% of 200 minutes is 20 minutes, which is the upper limit of total time accumulated during maximal steady-state exercise sessions in one week. While this approach may appear conservative, it will help to prevent over training and injuries and is a wonderful starting place.

Interval Training above the Lactate Threshold

Interval training workouts are high-intensity training sessions performed for short durations of time at velocities or workloads above the lactate threshold. Although you can design the interval workout however you wish, for our case study let’s choose to alternate a 4-minute, high-intensity bout with a 4-minute, low-intensity aerobic recovery bout. During the high-intensity bouts above the lactate threshold, have the client exercise above a 15 RPE (subjectively training at a HARD or VERY HARD intensity), but below an all-out effort (19 or 20 RPE). Encourage the client to workout at a very light intensity (less than 12 RPE) during the recovery bout. Similar to maximal steady-state sessions, the total interval training workout time should not exceed 10% of weekly training volume. With our case study, 10% of 200 would be 20 minutes of interval training sessions per week. Important training prescription recommendation! Avoid scheduling the interval training workouts and maximal steady-state exercise sessions in back-to-back workouts.

Final Thoughts

Lactate threshold is the most important determinant of success in endurance-related activities and events, and the main goal of endurance training programs should be the improvement of this parameter. Factors such as training status, age, gender, body mass, goals, and training time availability will all help determine the actual training intensities and volumes your client is capable of achieving. Utilization of a lactate threshold training program may add much excitement and interest to you client’s cardiorespiratory training program. By performing lactate threshold training, your clients are directly increasing their caloric expenditure during this type of exercise program. Educate them that this type of training is also highly recommended to enhance weight loss and weight management. Progress slowly and be creative with this program. Good luck with your lactate threshold training!

References

1. Anderson GS, Rhodes EC. A review of blood lactate and ventilatory methods of detecting transition threshold. Sports Medicine, 1989; 8(1):43-55.
2. Bassett DR, Jr, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. Medicine and Science in Sport and Exercise. 2000; 32(1):70-84.
3. Bompa TO. Periodization: Theory and Methodology of Training, 2nd Ed., Champaign, IL: Human Kinetics, 1999.
4. Brooks GA. Intra- and extra-cellular lactate shuttles. Medicine and Science in Sport and Exercise. 2000; 32(4):790-799.
5. Brooks GA. Anaerobic threshold: review of the concept and directions for future research. Medicine and Science in Sport and Exercise. 1985; 17(1):22-34.
6. Foran B. (edited by). High-Performance Sports Conditioning, Champaign, IL: Human Kinetics, 2001.
7. Katz A. Sahlin K. Regulation of lactic acid production during exercise. Journal of Applied Physiology. 1988; 65(2):509-518.
8. McArdle WD, Katch FI, Katch VL, Exercise Physiology: Energy, Nutrition, and Human Performance. Baltimore, MD: Williams & Wilkins, 1996.
9. Robergs RA, Ghiasvand F, Parker D. Biochemsitry of exercise-induced metabolic acidosis. American Journal of Physiology: Regulatory, Integrative and Comparative Physiology. 2004; 287:R502-R516.
10. Robergs RA, Roberts S. Exercise Physiology: Exercise, performance, and clinical applications. St Louis, MO: Mosby, 1997.
11. Wasserman K, Beaver WL, Whipp BJ. Mechanisms and patterns of blood lactate increase during exercise in man. Medicine and Science in Sport and Exercise. 1986; 18(3):344-352.
12. Weltman A. The Blood Lactate Response to Exercise. Champaign, IL: Human Kinetics, 1995.