Chapter I
Introduction
Chapter-I
INTRODUCTION

Today more than ever before it is necessary for physical educators, coaches, trainers, and fitness instructors to recognize the vital part ‘Science’ plays in the successful conduct of physical education, athletics, and activity program. Over the past thirty years the number of Exercise Physiology laboratories has increased tremendously. As a result much knowledge now dealing with how best to train athletic teams and to develop fitness for health has appeared in the scientific literature.

We are aware that all human activity centers around the capability to provide energy on a continuous basis. Without a continuous source of energy cells, including muscles cease to function and die. Energy is provided through the metabolic degradation of principally, two foods carbohydrates and fats. Carbohydrates are metabolized through glycolysis and the Krebs cycle. Fats are also metabolized through the Krebs cycle but begin with a process called Beta Oxidation.

As indicated, the production of energy is an ongoing activity requiring both aerobic and anaerobic processes.

ATP (Adenosine Triphosphate) is the immediate source of energy for muscle cell. There are two chemical or metabolic pathways by which ATP is formed. They are; (a) Anaerobic pathway (commonly referred to as glycolysis) which does not require oxygen and uses only carbohydrates in its production of ATP. This pathway results in an incomplete breakdown of glucose to pyruvate and eventually to lactic acid. (b) The
aerobic pathway (Krebs, tricarboxylic acid, or citric acid cycle and the electron transport enzyme system) requires the presence of oxygen and it can use all three food stuffs such as carbohydrates, fats, and protein in its production of ATP. This pathway involves a complete breakdown of carbohydrate to carbon dioxide and water.

Carbohydrates are breakdown into glucose where it is transported by the blood and stored in the muscles and liver in the form of glycogen. It is generally believed that somewhere between 350 to 150 grams of glycogen are stored in the human body.

While each glucose molecule is made up of 6 carbon atoms, glycogen molecules are merely clusters of glucose sugar molecules that are linked to each other in chain-like structures. The actual process of breaking down glycogen involves the removal of a glucose molecules from the chain-like structure at a time. (Process called glycogemolysis)

Anaerobic metabolism (in the absence of oxygen) involves a series of chemical reactions starting with the 6-carbons glucose molecule being broken down partially from glycogen into two 3-carbon molecules of pyruvic or lactic acid. This process is referred to as anaerobic glycolysis.

Anaerobic glycolysis takes place entirely in the sarcoplasm of the cell since all the enzymes that catalyze these reactions are located in this area. Note that it is generally believed that glycogen phosphorylase and phosphofructokinase are the two major enzymes that actually determine the rate of glycogen breakdown during the early stages of anaerobic
metabolism. Strictly speaking, anaerobic metabolism proceeds in the absence of oxygen, but when we consider the exercising athlete there appears to be a paradox. A quick examination of the data from a maximal oxygen uptake test reveals three things. The first is that even at rest, when oxygen is in plentiful supply, blood lactate levels are not zero but typically lie between 0.5 and 0.7 mmol L$^{-1}$. The second is that increasing oxygen uptake and, eventually, increasing levels of lactate in the blood accompany incremental exercise. Finally, blood lactate levels increase rapidly as exercise approaches maximal intensity. Indeed, one of the second-order criteria for determining whether an individual has reached maximal oxygen uptake is a blood lactate concentration – written $[\text{La}_{b1}]$ – of more than 8 mmol L$^{-1}$.

Elite endurance athletes routinely perform at 70-80 per cent of their maximal oxygen uptake for long periods with lactate levels of around 4 mmol L$^{-1}$. Thus, a more reasonable description of the anaerobic metabolism entailed in the exercising human is biochemical activity taking place in individual muscle cells where oxygen is either lacking or transiently unavailable for example, a sustained contraction of a muscle group – a timed bent-arm pull-up on the beam for example – compress the capillary walls and interferes with the flow of blood; this prevents access to the oxygen in the blood. The muscle then switches to the oxygen stored in the myoglobin [MgbO$_2$], but it depletes rapidly; the pain that accompanies high lactate concentrations increases, followed by the inability to maintain the isometric contraction.
So when we analyse the physiology of sports performance we must recognize that during activities involving whole body movements i.e. running, cycling, swimming, rowing, ATP production is the outcome of an interaction between aerobic and anaerobic metabolism. If we take the two extremes of the athletics running events, the 100 m run in under 9.8 s, and the Marathon [42195 m] run in under 2 hours 6 minutes – the ratio of anaerobic: aerobic metabolism that provides the fuel for adenosine triphosphate resynthesis is roughly 9:1 for the 100 m, and 0.1:9.9 for the marathon. The suggestion that the marathon entails a small measure of anaerobic metabolism may be unsurprising. on the other hand, the fact that aerobic metabolism partly fuels the 100 m sprint, even though the race is over before the oxygen delivery system had time to respond to the demands of the event, at first sight seems surprising.

The terms lactic acid and lactate, despite biochemical differences, are often used interchangeably. Fitness professionals have traditionally linked lactic acid with 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). 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). 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 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 athletes training program.

Historically, lactic acid is a metabolite that has received a lot of bad press. We grew up hearing about all the bad things that it causes 1) muscle soreness, 2) cramps, 3) fatigue, and 4) over training. Most of this negative information came from coaches, physicians, and athletes. Unfortunately, there is still a need for education on this subject, since the majority of educators, athletes, physicians and coaches still practice this doctoring. Although there is not much doubt that at high lactic acid levels muscle contraction is inhibited; because specific proteins in the muscle cells can only function at a certain range of acidity. Once lactic acid has risen to this level and the muscle cells become saturated; cellular reactions from within the cells simply stop. The result is acute muscular fatigue. Forcing us to greatly reduce the exercise intensity, or to stop exercise altogether. This point of saturation of lactic acid in the muscle
cells is different for each individual, and is regulated by the physical fitness of that individual, duration and intensity of exercise. But in general the limitation of its concentration is 30 mmol per cubic decimeter (a standard of measurement).

To fight off this muscular fatigue, caused by lactate acid; it is very important to teach your muscles how to cope with large concentrations of it.

For a long time, it's been recognized that lack of oxygen increases the rate at which lactic acid is formed, and it was also thought that high, intense, muscular contractions, could result in high muscle lactate levels.

Recent studies on lactate and its role show that lactic acid is constantly made and removed frequently at very fast rates, even during rest. Certainly, lactic acid can be formed in muscles with adequate oxygen, and a rise in the concentration simply means that its production rate exceeds its removal rate. Even at rest lactic acid is formed in the liver, red blood cells, intestines and kidneys. The breakdown of blood glucose seems to be the major source of its formation.

Lactic acid is not just a useless by-product of incomplete metabolism. The breakdown of lactic acid is one of the body’s most important sources of energy. In highly oxidative fibres such as the ones found in cardiac muscle and Type II muscle fibres (Fast twitch) it is the preferred fuel source for contraction. Lactate also produces blood glucose and liver glycogen. There are four possible fates of lactic acid.
1. Excretion in urine and sweat: Lactic acid is known to be excreted in the urine and sweat. However, the amount of lactic acid removed in this manner during recovery from exercise is negligible.

2. Conversion to glucose or glycogen: Since lactic acid is break-down product of carbohydrate (glucose and glycogen), it can be reconverted to either of these compounds in the liver and in muscle given the required ATP energy. However, as previously mentioned, glycogen resynthesis in muscle and liver is extremely slow compared with lactic acid removal. In addition, the magnitude of the changes in the blood glucose level during recovery is also minimal. Therefore, conversion of lactic acid to glucose and glycogen accounts for only a minor fraction of the total lactic acid removed.

3. Conversion to protein: Carbohydrates including lactic acid, can be chemically converted into protein within the body. However, once again, only a relatively small amount of lactic acid has been shown to be converted to protein during the immediate recovery period following exercise.

4. Oxidation/ conversion to CO2 and H2O: Lactic acid can be used as a metabolic fuel for the oxygen system, mostly by skeletal muscle, but heart muscle, brain, liver, and kidney tissues are also capable of this function. In the presence of oxygen, lactic acid is first converted to pyruvic acid and then to CO2 and H2O in the Krebs cycle and the electron transport system, respectively. Of course
ATP is resynthesized in coupled reactions in the electron transport system.

Cardiac muscle from within the heart is made up of highly oxidative fibres. These fibres extract lactate from the bloodstream and use it for energy. This occurs especially during very intense exercise where large amounts of lactate are released into the bloodstream from skeletal muscle.

Type II muscle fibres oxidize lactate at a very fast rates. When muscle contraction produces a significant amount of lactate, it is then released into the central circulation of the blood; and within seconds it is made available to muscle for energy. Therefore, 75% of the lactate produced from high intensity exercise is made available for energy production in Type II muscle fibres. The remaining 25% of lactic acid is used for energy in the heart, the make up of liver glycogen, and the supply of energy to inactive muscles. A good example of this would be a runner who is exceeding his or hers planned race pace in 10 km. The excess lactic acid accumulated in the contracting muscle from insufficient oxygen is then made available to inactive muscles (e.g. arms) from the central circulation of blood.

The remaining lactic acid that is not directly oxidized for fuel is sent to the liver, where it is stored as glycogen. In the process of exercise, glycogen is released into the blood stream to form glucose.

Endurance athletes require a continual supply of energy for their working muscles. This energy, under aerobic conditions is supplied by
the breakdown of fat and glycogen (stored carbohydrates). Fat is the primary fuel source for continuous muscular contractions under aerobic conditions. However, in order for fat to be used for fuel, it must mix with glucose. Glucose is made from the breakdown of glycogen (stored carbohydrates). Together, these two types of fuels provide energy to contracting muscles under aerobic conditions.

As a coach and athlete one must learn how to teach the body to handle lactic acid. It is imperative, if you want success in today’s highly competitive field of athletics to train your muscles, body and mind to accomplish gains in performance even in the presence of lactic acid. Coaches and athletes should design training programs with this being a primary focus. This is done by two basic components of training.

Long Slow Distance (LSD) training beyond the normal racing distance, will develop tissue enzyme adaptations that will rely upon the use of free fatty acids for energy production, which will result in less lactic acid being produced. LSD training will also increase the rate of lactic acid removal from the blood and muscles. During continuous steady state exercise, you increase capillary density and mitochondria function in skeletal muscle. These two peripheral adaptations brought on by LSD training will enable your body to handle lactic acid much more efficiency.
High intensity training will develop the cardiovascular system to increase the rate of oxygen transport to the contracting muscles so there is less reliance on carbohydrate breakdown to lactic acid. High intensity training such as intervals, fartlek and variable pace workouts, will increase your functional capacity (Max VO2). This means that in actual competition you will produce less lactic acid, because your muscles are relying mostly on the use of free fatty acids for fuel. The tissues that can use it as fuel, such as the heart and Type II muscle fibres will remove the lactic acid that is produced.

B. F. Visser, Joh. Kreukniet and A. H. J. Maas in 1964 made an attempt to establish whether it is possible to predict the change in the lactic acid concentration of the arterial blood during exercise from the calculated metabolic change in the bi-carbonate concentration of the arterial blood during exercise. The changes in the HCO$_3^-$ and the lactic acid concentration averaged —4.15 and 4.41 mEq./l, respectively.

It was found possible to predict the measured change in lactic acid concentration from the change in HCO$_3^-$ concentration calculated from the pH and the $P\text{ CO}_2$. A respiratory correction in the calculation ensured the best correlation coefficient (0.81).

The regression equation was $\Delta\text{lactic acid}=0.29-0.99 \Delta\text{HCO}_3^-$ metabolic.

An equally good approximation was attained in calculating the change of lactic acid concentration from the total change in bi-carbonate
concentration, with application of a simplified respiratory correction: \( \Delta \text{lactic acid} = 0.33 - 0.97 (\Delta \text{HCO}_3^- \text{ total} - 0.18 \Delta P \text{CO}_2) \).

The correlation coefficient remained 0.81.

Lactic Acid Tolerance training on the other hand allows an athlete to experience even higher levels of intensity by increasing the rest period to twice that of the work period. This type of training will maximize bout speed and allow an athlete to develop their application of power with quick muscle movement. The emphasis should be to achieve extreme levels of lactate concentration of 12-13mM/l, also pushing an athlete's heart rate to their maximum levels.

Very short intervals of 10-15 seconds flat out work will increase the rate of glycolysis 100% above that in a long distance run and will help to recruit muscle fibre, increasing strength. The down side, however, is that very short intervals will over time tend to decrease the number or capillaries bringing blood into the muscle, reducing aerobic conditioning.

Slightly longer intervals of 15-20 seconds will improve the use of phosphocreatine (PC) as an energy source to be used gradually over the duration of the race ie. enabling the power normally reserved for the 'start' or end 'kick' to provide a small but continuous contribution. It is important that the rest interval be kept long, close to 2 minutes, so that the work interval is kept alactic and sufficient time is provided to build-up PC stores. It only takes about 22 seconds to replenish 1/2 of PC stores and 44 seconds to replenish 3/4 total capacity. Work must be to absolute
intensity so that the training effects will be confined to expenditure of PC and not glycogen. This type of training is ideal the week prior to race day since it avoids the longer term effects of fatigue.

Intervals of 60-90 seconds result in an increase in the amount of glycolytic enzymes improving the rate of glycogen mobilization and the muscles ability to tolerate the products of anaerobic metabolism. This duration of work interval will also help to smooth out the edges between the effects of different shorter distance training.

Exercise physiologist of many countries are trying to evolve an effective programme of training which may lead to utmost increase in the lactic acid tolerance capacity which can lead to enhancement of performance by pushing back the fatigue.

Lactic acid can be removed from blood and muscles more rapidly following heavy to maximal exercise rather than by resting throughout the recovery period. When lactic acid, the product of anaerobic glycolysis, accumulates in blood and muscles, fatigue sets in. therefore; full recovery from exercises in which maximal amounts of lactic acid have accumulated involves the removal of lactic acid from both blood and the skeletal muscles that were active during the preceding exercise period.

As lactic acid has detrimental effects on exercise performance, it is important to consider the factors which effect its removal from the blood
stream. The rate at which lactic acid leaves the muscle and enters the blood stream is determined by the rate at which blood flows through the tissues. Lactic acid may then be taken up by the heart or other skeletal muscles and converted back into pyruvic acid and further metabolized in order to generate energy in the form of ATP. Some of the lactic acid not consumed in this manner is, in the period immediately after exercise, converted back into glycogen by the liver.

At the completion of an exercise session, or between repetitions during interval training, one has the choice of employing an active or a passive recovery. An active recovery involves exercising at a low intensity whilst the passive mode of recovery involves total rest. If lactic acid accumulates during exercise there is a distinct advantage to employing an active recovery because the blood flow, and therefore the lactic acid dispersal from muscle, is greater than that during a passive recovery. Furthermore, the rate at which lactic acid is utilized as an energy source by the heart and skeletal muscle is greater during low intensity exercise than at rest. The optimal exercise intensity for active recovery depends on each subject's fitness level, but for most individuals it occurs at heart rates of approximately 15-30 beats per minute below that of the anaerobic threshold. Even with an active recovery it may take as long as 30 minutes for 95% of the accumulated lactic acid to be removed after extremely intense anaerobic exercise. However, lactic acid
concentrations may remain elevated above resting levels for 60 minutes or more if a passive recovery is employed.

It is obviously inappropriate to wait for lactic acid to completely disperse before completing another repetition during an interval training session. However, lactic acid levels do drop quite significantly in the first few minutes of recovery and it may take as little as five minutes of active recovery for 50% of the accumulated lactic acid to be removed from the blood stream. As a result a significant recovery occurs when five to 10 minutes are taken between intervals.

**Purpose of the Study**

The purpose of this research work was to assess the effectiveness of three training programmes of varying intensities in significantly enhancing the lactic acid tolerance capacity. Another pertinent area of this study was to find out an effective relaxation programme during recovery period which is best in speedy lactic acid removal after exhaustive bouts of exercise.

**Statement of Problem**

The objectives of this study had led the researcher to state the problem as

"Comparative effect of different training programmes on lactic acid tolerance capacity and its rate of removal"
Delimitation

The purpose of this study and resources available at our hand had lead us to confine the study in following delimitation:

(1) Lactic acid tolerance capacity.
(2) Excess lactic acid removal rate.
(3) Boys in the age group of 16-22 years were selected as subjects of the study.
(4) The three different training programmes were
   (a) Fast continuous method.
   (b) Intensive interval method.
   (c) Extensive interval method.
(5) The three relaxation programmes were
   (a). Aerobic exercise
   (b). Yogic technique
   (c) Rest.

Hypothesis

The objectives of the study had led the researcher to set following hypotheses.

1. It is hypothesized that the lactic acid tolerance capacity shall be differently affected by the three training programmes.
2. It is further hypothesized that the lactic acid tolerance capacity will be increased through all the training programmes.
3. It is hypothesized that various relaxation programmes shall differently affect the speed of lactic acid removal from the blood.
Significance of the Study:

Lactic acid tolerance capacity is an indicator of energy production. Hence a very important determinant of sports performance. This study is an attempt to assess the effect of various training programmes on this capacity.

Speedy lactic acid removal from muscles and blood is again a matter of great concern for the exercise physiologists. Lactic acid can be removed from blood and muscles more rapidly following heavy to maximal exercise rather than by resting throughout the recovery period. To develop an appropriate relaxation programme for its fast removal from the body is another concern of this study.

Thus this study shall help the coaches, sports trainers and athletes in utilizing lactic acid phenomenon for their performance enhancement.