

## Energetic Transference Occurring in the Biosphere Part III

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### Abstract

Lactic acid under a normal physiological pH dissipates into the salt lactate and hydrogen ions. The process tends to lower pH levels if not properly cleared. Further, the lactate molecule itself can have negative effects on mechanisms responsible for contraction of sarcomere units. Mechanisms of lactate formation and clearance will be discussed in terms of supramaximal exercise performance. An interesting structure related to lactate clearance is the monocarboxylate transport system, also known as the lactate shuttle mechanism. This will be discussed in detail as well as the concept of how biological systems adapt to various training stimuli at the cellular and, more specifically, energetic level. These enzymatic adaptations will be reviewed in the context of Henry's Specificity Hypothesis (1950).

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### Introduction

The purpose of this paper was to review Lactate production and training adaptations involved in surpamaximal exercise stimuli.

### Lactate production

One of the major misconceptions in exercise science is that there exists both an aerobic and anaerobic form of glycolysis. When pyruvate is the end product of the energy pathway, glycolysis is said to be aerobic. Further, when lactic acid is the end product, the process is said to be anaerobic. The attribution of these two forms is often credited to the presence or lack of oxygen. In reality glycolysis is by its very nature an anaerobic process, and a lack of O<sub>2</sub> is seldom the cause of lactate formation. Brooks (2000) summarizes the process as follows: 1. Performance of high intensity exercise at high altitude (low O<sub>2</sub> concentration) situations results in little lactate accumulation. 2. Lower blood lactate levels do not indicate that lactic acid is not being produced, but rather that its production rate is balanced with its clearance rate. 3. Lactic Acid is always produced even at rest.

From these points, Brooks (2000) suggests that the scientific community implement new terminology for glycolysis. The term anaerobic glycolysis would be changed to fast glycolysis, while aerobic would be changed to slow glycolysis. The implications are that the faster glycolysis takes place, the more rapid the accumulation of lactic acid will be. Reasons are as follows:

1. There is a finite amount of NAD in the muscle cell. NAD serves to accept electrons (become reduced) along with two hydrogens. These are then deposited to the electron transport chain, or to pyruvate itself. When NAD is reduced to NADH + H<sup>+</sup>,

another molecule known as 3-phosphoglyceraldehyde is subsequently oxidized (loses its electrons). This is known as a coupled reaction. Glycolysis produces two ATPs from substrate level phosphorylation when glucose is the substrate, and 3 when glycogen is the substrate. However, there are a finite number of the  $\text{NAD}^+$  co-enzymes in the cell. The oxidation of 3-phosphoglyceraldehyde must continue if glycolysis itself is to continue. The ratio of  $\text{NADH} + \text{H}^+$  to  $\text{NAD}^+$  is known as Redox Potential, and is maintained by pyruvate and/or acceptance of electrons at the ETC. Consequently, pyruvate is known as a hydrogen sink. As discussed in the energy continuum, glycolysis is far more powerful than oxidative energy production, and is stimulated by muscular contraction itself. The more rapid the contractions, the faster the pathway will run, outside of fatigue mechanisms.

2. The speed of a reaction is directly correlated to the catalytic rate of the enzyme controlling the process. Lactate Dehydrogenase is the rate-limiting enzyme, which transfers two hydrogens to pyruvate to form Lactic Acid. Its rate of catalytic activity is faster than each of the glycolytic enzymes. Further it also operates at a higher rate than Pyruvate Dehydrogenase. The latter enzyme converts pyruvate to Acetyl Coenzyme A, which enters the aerobic pathways. Therefore any increase in  $\text{NADH} + \text{H}^+$  and pyruvate will inevitably increase the formation of Lactate. From this it can be seen why Brooks suggests the terms fast and slow glycolysis.

3. Glycogen phosphorylase is the enzyme responsible for the catabolism of glycogen, which is the stored form of glucose. Muscular contraction is activated by a series of neurological events. First, movement is initiated in the motor cortex. The efferent motor pathways arise out of the motor cortex and have connections with every motor unit in the body. Neurons arising out of this region are meters in length, and are known as upper motor neurons. These connect with lower motor neurons. A lower motor neuron and the muscle fibers it innervates are a motor unit. Electrical pulses travel from upper to lower motor neurons and activate musculature by transferring pulses to the membrane of the muscle cell, which then spreads inside of the cell by a series of structures known as t-tubules. A flow of ions then enters the cell and stimulates an organelle known as the sarcoplasmic reticulum to release  $\text{Ca}^{++}$ . This  $\text{Ca}^{++}$  acts as a key for muscular contraction. Consequently, it also activates the enzyme glycogen phosphorylase. Because the rate of catalytic activity is related to substrate concentration, the catabolism of glycogen increases the rate at which glycolysis runs, which inevitably results in greater lactic acid production.

4. Lactate is cleared by being transformed to pyruvate in the mitochondria. Slow twitch muscle fibers have a greater supply of mitochondria, and a greater ability to take up lactic acid into those mitochondria. The motor program, defined as the spatial and temporal elements available to the user prior to the initiation of movement, provides the pulses responsible for that movement and will activate more fast twitch motor units as force production increases. These have a lesser ability to clear the lactate produced, which will therefore enhance lactate build up.

5. The sympathetic nervous system is activated by increased movement, and in turn releases the fast acting hormones norepinephrine and epinephrine. Glucagon is also secreted. These stimulate the catabolism of glycogen, which increases the rate of glycolysis.

### **Lactic Acid Clearance**

Lactate clearance is important for numerous reasons, which are intimately related to the dissipation of hydrogen ions. Venom (2003, Active Recovery) explains the following mechanisms by which pH levels affect performance:

Problems with Lactic acid occur when the amount of free hydrogen ions (H<sup>+</sup>) surpasses your bodies buffering systems, effectively decreasing normal pH levels (acidity levels; the lower the pH is, the more acidic your blood becomes). When this occurs, the athlete will begin to feel pain, and suffer a decrease in athletic performance.

This pain is caused by an accumulation of hydrogen ions that stimulate pain nerves located in the muscle [14]. Performance decline is induced by both metabolic and muscular fatigue.

Metabolically, a decreased pH causes the inactivation of several enzymes [15], membrane nutrient transport mechanism inefficiencies [15], and energy decreased accessibility. To elaborate on energy deficiencies; glycogen catabolism is slowed by the inactivation of the enzyme glycogen phosphorylase, and lactic acid inhibits the recruitment of fatty acids, minimizing their utilization. Due to these effects, carbohydrates are used at a heightened rate, and PC catabolism is increased, which inhibits ATP regeneration. All of these factors ultimately lead to reduction in the production of ATP. Thus, decreased performance [1, 9,15].

Concerning muscular exhaustion, lactic acid promotes the restraint of the actomyosin ATPase, which breaks down ATP so it can provide energy for your body. In addition, H<sup>+</sup> interferes with calcium uptake that is essential for muscular contractions. Increased lactate may also interfere with cross bridging [16]. These factors lead to a decline in both the force and velocity of muscular contractions.

Lactic acid is mainly removed by oxidation and gluconeogenesis (Brooks et al., 2000). One of the critical issues of lactic acid clearance mechanisms concerns a family of structures known as monocarboxylate transporters (MCTs). They are also referred to as Lactate shuttles. Lactic acid travels from producing cells to consuming cells through MCTs (Brooks et al., 2000; Brooks 2000). Intracellular MCTs are able to transport lactate produced in the cytosol to the mitochondria for oxidation. Oxidation occurs by transferring hydrogens to NAD, which then enters the ETC, while the pyruvate is converted to Acetyl Co-A. Further, extracellular MCTs are able to transport lactate between tissues. In this way lactate can move from fast glycolytic fibers to slow oxidative fibers, or reach the blood stream where the substrate can be oxidized by the heart (Gladden, 2000).

Slow oxidative fibers as well as the heart have a greater concentration of mitochondria. Further, the mitochondria contain MCT I proteins. These are specialized at transporting lactate from the cytosol into the mitochondria for oxidation or consumption. Fast twitch muscle fibers contain a higher concentration of MCT 4 proteins, which are concentrated on the sarcolemma of the muscle cell, and are specialized to transport lactate to oxidative muscle cells, or to the blood stream itself. Once in the blood stream, lactate can circulate to the liver or the heart. Interestingly enough, the heart uses lactate as its main fuel sources during exercise.

Because fast twitch fibers do not contain a high density of mitochondria, they facilitate the storage of glycogen from lactate in a reversible action, in which the lactate is used to synthesize glucose (called gluconeogenesis). This occurs with some of the lactate that does not diffuse out of the cell after exercise has ceased. Lactate that travels to the liver is converted to glucose, and then can be released by the bloodstream and used by working musculature. This process is known as the Cori Cycle.

## **Training Adaptations**

Training adaptations will be viewed as specific to imposed demands placed on participants. Henry (1950) proposed the specificity hypothesis, suggesting that the attributes that underlie an activity are specific to that activity and not transferable (task-specific). Sawyer et al. (2002) suggests that an attribute is the underlying capacity within an individual, which allows for the expression of skill (these are presently viewed as genetically predisposed and typically unaffected by practice). The statistical evidence highly supports these concepts (Sawyer et al., 2002).

It is important to understand that greater transfer, even at the level of energy systems, will be realized when training is specific to the criterion task. For example, riding the stationary bike will produce cardiovascular adaptations, but they will not enhance the extraction of the extra oxygen delivered when training the upper extremities (known as arterial venous difference). Maximum oxygen uptake by an organ is described by Fick's principle. Fick's principle states that the amount of oxygen utilized by a tissue is defined as the product of blood traveling to that tissue and the extraction of the oxygen delivered. Therefore, adaptations from a physiological level occur centrally, peripherally, and at the cellular level itself. These adaptations occur through increased and specific capillarization, increased mitochondria number, as well as specific enzymatic activity.

Therefore if a participant seeks to increase mitochondrial density, and therefore enhance the arterial venous difference, they will need to train the upper extremities in an aerobic fashion.

Further, it is important to also understand that these adaptations are also specific to the actual task itself. Riding a bike while standing will activate the motor neuronal pool, as well as various other musculatures, in a different manner than riding a bike while seated will. Moreover, running on a horizontal surface will activate musculature in a different pattern than running on an incline. It is for this reason that coaches will benefit by training their athletes for the event that they will have to face. If a cross-country team is used to running horizontal, and then are faced with running on an incline type of hilly surface, they will be seriously under matched. Therefore, the following recommendations and adaptations will be heightened when done specific to the task. This factor should be assumed throughout the remainder of the paper.

## **Energy System Specificity**

The overload principle will have to be applied in order to achieve adaptations in energy system pathways. The energy-time-continuum should be used in this case.

### **A Lactic Anaerobic Metabolism**

The A Lactic or phosphagen system is best stressed at 10-30 seconds. It clearly dominates at 10 seconds, however. Further it is important to realize that ATP-PC stores are fully recovered by 2 minutes, while the half-life is 30 seconds.

Therefore, participants can train 10-30 seconds on, with 30 seconds of rest, and still recover half of their CP stores. At 1 minute these stores will have recovered to a greater extent. The theory is to continually stress the ATP-PC system, while not allowing full recovery so as to force a change in its total capacity.

### **Lactic Anaerobic Metabolism**

This system dominates from 30 seconds to 2 ½ to 3 minutes. In order to change its capacity the athlete will want to train within this range. However, unlike the A Lactic system, the half-life for the LA system is much longer, ranging from 15-20 minutes in duration. Therefore, a one to three minute rest between sets would be prudent, as this would allow for full recovery of CP stores to assist maximizing workload. The system will again be stressed to both increase its ability to clear lactic acid, and also to increase glycogen storage capacity. For example, Macdougall et al. (1979) showed that 10 weeks of heavy external resistance training increased concentrations of muscle creatine by 39%, Creatine Phosphate by 22%, and ATP by 18%.

Adaptations will occur when a specific workload is able to be handled without as much homeostatic disruption, or an even greater work load is able to be handled. When this occurs a further overload should be placed on the body.

Overload can come in the form of a greater intensity, increased volume, or increased

frequency, and each should be utilized. However, intensity appears to be the factor that causes the greatest regression in the individual when lowered. That is, both frequency and volume are vital when overloading the system, but intensity appears to be more important when maintaining that adaptation (Mujika and Padilla 2000 Part I, Mujika and Padilla 2000 Part II).

### **Anaerobic Training Adaptations in Specific Enzymes and Receptors**

It appears that enzymatic activity is enhanced more than enzymatic concentration in terms of adaptations involved in anaerobic training.

MacDougall et al. (1999) investigated the effect of intense interval training on glycolytic enzyme activity. Participants consisted of 12 healthy males, with an average age of  $22 \pm 2$  years. The apparatus consisted of a bicycle ergometer. Training consisted of the Wingate protocol with 2-4 minutes of rest between sets, three days a week, for 7 total weeks. The effect of 7 weeks of intense cycling on glycolytic enzyme activity resulted in an increase in activity in hexokinase, and in phosphofructokinase. The effect of training on cycling performance found significant increases in peak power output, total work over 30 s, and  $\dot{V}O_{2\max}$ . The implications are that high intensity specific training can increase glycolytic enzymes, and that the enzymatic activity is highly correlated to peak power output, and total work. Further, these results in glycolytic enzyme activity do not appear to be prevalent in endurance training (Holloszy, 1976).

The adaptations seen were in Hexokinase and phosphofructokinase. Hexokinase is an important rate-limiting enzyme in glycolysis, and serves to phosphorylate glucose and prepare it for extraction of energy. Further, Phosphofructokinase is the most important rate-limiting enzyme in glycolysis. As its activity increases, glycolysis increases.

In another interesting study, Costill et al. (1979) investigated the effect of 7 weeks of isokinetic strength training on muscle enzyme activities. Two experimental conditions were implemented. In the first, one of the participants' legs was trained using 10 repeated 6-s maximal work bouts. The second condition consisted of repeated 30-s maximal knee extension exercise. The effect of various maximal work bout conditions on enzymatic activity, found that the 30-second condition produced a significant increase in glycolytic enzymes, while the 6-second condition found no significant increase in glycolytic enzymes. The suggestion is that training the glycolytic pathway is time continuum specific.

Cadefau et al. (1990) investigated the effects of 8 months of a specific and controlled sprint training program on three groups of young athletes (two groups of males and one of females). Glycogen content and the activities of the enzymes of glycogen metabolism (glycogen synthase and glycogen phosphorylase), glycolysis (phosphofructokinase, pyruvate kinase) were measured. The effect of 8 months of sprint training resulted increased glycogen content, and increased activities of glycogen synthase, glycogen phosphorylase, phosphofructokinase, pyruvate kinase.

Glycogen synthase is the rate-limiting enzyme involved in the storage of glycogen. As its activity increases, so too does the participant's ability to store glycogen. Glycogen is the preferred fuel of glycolysis. Moreover, an increase in substrate

availability would also directly increase catalytic rate due to concentration and availability of binding.

Glycogen Phosphorylase is the enzyme responsible for introducing glycogen into the glycolytic process. As the activity of GP increases, so too does the activity of the pathway itself. It is highly correlated to bodybuilding type activities.

Pyruvate Kinase is important because it is responsible for substrate level phosphorylation of ATP. In this step a phosphate is removed from phosphoenolpyruvate to ADP, in turn producing Pyruvate and ATP.

### **Adaptations to Lactate Accumulation**

As with other enzymes, various species of Lactate Dehydrogenase (LDH) exist. The enzyme is of course responsible for the production of lactate from pyruvate. Comparison of LDH found in the myocardium (heart musculature) and skeletal muscle results in the cardiac LDH having a lower affinity for pyruvate than skeletal musculature. In a review of the effect of endurance training on enzymatic activity Abernethy et al. (1990) found that skeletal muscle could acquire the cardiac form of LDH through training. Therefore pyruvate would be more likely to enter into the Krebs cycle directly. This would tend to decrease lactate build up during exercise.

Pyruvate dehydrogenase is the rate-limiting enzyme for pyruvate entry into the Krebs cycle. Its activity therefore affects the oxidation of pyruvate, as well as lactate accumulation (Heigenhauser and Parolin, 1999). The catalytic rate of lactate dehydrogenase is much higher than pyruvate dehydrogenase. It follows that an increase in pyruvate will ultimately lead to an increase in lactate accumulation. It is for this reason that higher work loads facilitate the build up of LA. However, endurance training may enhance the activity of Pyruvate dehydrogenase. It would therefore accept more pyruvate, which would lower overall lactate levels, especially at submaximal exercise intensities (Heigenhauser and Parolin, 1999).

Interestingly enough, high intensity and endurance training lowers epinephrine and nor epinephrine levels at absolute workloads. These sympathetic messengers speed the rate of glycogenolysis. If lower, then not as much lactate will build up, and training can last longer. However, this is for absolute and submaximal relative workloads. When training maximally, sympathetic hormones will actually increase (Wilson, 2004).

Other avenues for management of lactate occur at the Monocarboxylate level (MCT). For example, mitochondria increase preferentially to more endurance than weight training (Brooks, 2000). More mitochondria would afford a greater supply of MCT proteins, just by sheer concentration of this organelle itself, leading to increased oxidation of lactate. Such an adaptation would be beneficial for recovery between high intensity sets, but may not enhance performance directly during those sets. MCT 1 proteins may increase in concentration as well overall (Brooks, 2000). This would facilitate lactate removal. At maximal workloads, lactate levels are actually higher in the trained individual, for reasons such as increased sympathetic activity as mentioned, as well as greater glycogen storage and catalytic activity. Yet, these greater levels of lactate can be tolerated. Galbo (1983) suggests that this is due to psychological aspects of training, in that the athlete is more prepared mentally for higher levels of lactate than the untrained.

That is in completely maximal workloads, however. When submaximal, lactate levels do significantly drop, which is highly beneficial to the bodybuilder who trains at submaximal levels during many sets for hypertrophy reasons. Reynolds et al. (1997) investigated the effect of resistance training on blood lactate and rate of perceived exertion to a set of exhaustive squats. Exhaustive squats at 50 and 70 percent of 1 repetition maximum were performed before a 10-week training program. The effect of 10 weeks of resistance training on blood lactate levels found that the same workloads performed at pre training had significantly lower blood lactate levels. Further, when new workloads were performed (participants had increased their 1-RM) at 50 and 70 percent 1 RM, there was no significant difference between pre- and post-training lactate levels. This suggests that lower lactate levels will accumulate at greater absolute workloads as a result of resistance training. This may reflect the increase in the cardiac isoform mentioned by Abernathy (1990), as well as changed mentioned previously.

## Implications

Training should be specific to the activity. Specificity includes task-specific training, especially if in a highly specialized sport, as well as energy continuum specificity. For the bodybuilder, a number of skills will need to be increased in capacity if success is to be achieved. That is, differing tasks will preferentially recruit slow or fast twitch muscle fibers. The bodybuilder will therefore benefit by training in a range of repetition ranges. Further areas which need improvement should be targeted with specific training regimens.

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