乳 酸 及 運 動
LACTATE AND SPORTS PERTORMANCE

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中文摘要

本篇文獻回顧旨在探討乳酸在生理上所扮演色。血液乳酸廣泛地被認爲是無氧代謝之指標。乳酸是Embden-Meyerhof 路徑的最終產物。大部分乳酸(50%-70%) 會在肌肉組織中被氧化。乳酸在運動肌肉之濃度和FT及ST股纖維分佈有關。乳酸堆積是乳酸製造量大於清除是之結果。乳酸代謝下只影響到醣解中之速率限制步驟，也會抑制脂肪細胞中移出。肌力會隨著乳酸濃度的上升而下降。激發－收縮聯合也會被乳酸中所含氫離子所抑制。

高強度運動後進行中強度運動可以經由血液循環及氧化酵素（如乳酸脫氫酵素）活性機轉促進乳酸清除。從事冰上曲棍球這項間歇運動時在如每回下場後實施動態恢復可以加速降低乳酸濃度。

本篇文獻探討中提出影響競技表現之生理參數，而利用運動訓練克服這些參數之弱點，可改進運動成績。

1.Abstract

The prupose of this review article is to study the physiogical role of lactate and effect of lactate on sports performance. Blood lactate is widely accepted as indicator of anaerobic glycolytic metabolism. Lactate is the end product of the Embden-Meyerhof pathway. Most of lactate (50%-70%) is oxidixed by muscle tissue. Lactate concentration in exercising muscle is found in association with the FT and ST fiber distribution. Accumulation of lactate is evident when lactate production excesses the removal rate. Lactic acid not only influence several of the rate-limiting steps in the glycolytic process but it is also a potential inhibitor of fat mobilization from adipose tissue. Muscle strength linearly decreased as lactate levels increased, The E-C coupling was inhibited by concen-

trated hydrogen ion from lactic acid. A moderate exercise following intense physical effort may enhance lactate clearance by accelerated circulation and activated enzyme (e.g. lactate hydrogenase). Performing intermittent exercise as ice hockey, active recovery between bout is recommended as to reduce lactate at a faster rate.

After this review study, physiological parameters that influence athletic performance may determine weaknesses so that the athlete can modify the training program to correct the weaknesses.

2.Introduction

As early as the late 19th century, scientists recorded lactate production in animal and human muscle as well as in the circulation following exhaustive exercise (karlsson,1971). Short duration of high intensity muscle contractions had been postulated to be the primary factor causing a rapid lactate build-up. High lactate concentration directly or indirectly impairs muscle function (Asmussen et al.,1948; Bang,1936; Karlsson et al.,1975). The exercise potential of individuals is strongly associated with lactate concentration in muscle tissues (Jacobs,1986).

Althoug the accumulation of excess lactate is an inevitable metabolic response to heavy muscular work, this acidic metabolite can be used by exercising muscle at the same time as it is being produced (Stamford et al.,1981). Many scientific reports (Belcastro & Bonen,1975;Boone,1984; Kaczynski,1989; Newman et al.,1937; Stamford et al.,1981; Waston & Hanley 1986) show that if a moderate level of exercise is performed following intense physical exertion, lactate concentration decreases at a faster rate. This is of practical importance for sports involving intermittent and short duration, high intensity exercise, since lactate reduction may aid athlete in delaying the onset of muscular fatigue and thereby permit exercise for longer periods of time and perform at optimal levels.

3.Gylcolysis and Lactate Production

In all living cells energy metabolism is a very complex procedure in which chemically bound substrates are converted into mechanical energy. Glycolysis is one of the metabolic processes in which glucose is enzymatically catabolized to resynthesize high-energy ATP. The end product of this energy yielding system is pyruvate. In aerobic conditions with adequate mitochondrial activity, pyruvate is oxidized to water and carbon dioxide via acetyl-coenzyme A and the citric acid cycle (Kreb's cycle). Under anaerobic conditions, pyruvate is catalyzed to lactic acid by the enzyme lactate dehydrogenase (LDH) in the biochemical reactions whereby a 6-carbon glucose molecule is broken down into two 3-carbon molecules of pyruvate, with the aid of specific enzymes. Pyruvate can then be further catalyzed into lactate (Fig.1) This process has been fully studied and extensively elaborated in many scientific research papers. Lehninger (1982), McArdle et al. (1986), and Orten and Neuhaus (1982), have described this process (Fig.2) in their textbooks. The glycolytic process is briefly described in the next parahraph.

The first step in the metabolism of glucose is its phosphorylation which yields the phosphate ester alpha-D- glucose 6-phosphate. This reaction is catalyzed by a group of four distinct enzymes: type I to IV hexokinase. Glucose 6- phosphate is then converted into fructose 6-phosphate. This reaction is controlled by an enzyme specific for glucose 6- phosphate called phosphoglucoisomerase. Fructose 6-phosphate is further phosphorylated to fructose 1,6-diphosphate. This reaction is catabolized by a specific kinase, phosphofructokinase. The phosphorylation of fructose 6- phosphate is physiologically irreversible, since a separate enzyme is required for this reaction. Fructose 1,6-diphosphate is hydrolyzed to fructose 6-phosphate by the action of fructose diphophatase. The following step in glycolysis is the break-down of fructose 1,6-diphosphate by the enzyme aldolase to form two phosphorylated molecules with three carbon chains (glyceraldehyde 3-phosphate, and dihydroxyacetone phosphate). The glyceraldehyde 3-phosphate is then oxidized to 3- phospho-

glycerate and is catalyzed by glyceraldehyde phosphate kinase. In the presence of phosphate glycerate kinase, the free-energy change in the conversion of 1,3 disphosphoglycerate to 3-phosphoglycerate is used to form ATP from ADP at the substrate level. The next step in the glycolytic pathway involves the transference of the phosphate group from carbon-3 to carbon-2 of glyceric acid. This reaction is catalyzed by phosphoglyceromutase. The product, 2- phosphoglycerate, is then dehydrated to yield a high-energy phosphate compound, phosphoenolpyruvate (PEP). This step is catalyzed by enolase. Pyruvate kinase catalyzes the transfer of the high-energy phosphate from PEP in order to resynthsize ATP with a spontaneous formation of pyruvate. At moderate levels of energy metabolism, sufficient oxygen is available to the cells. The hydrogens (electrons) are stripped from the substrate and carried by the reduced form of nicotinamide-adenine dinucleotide (NADH). NADH is oxidized within the mitochondria and combines with O_2 form H_2O. Phruvate molecules are irreversibly converted to form acetyl-coenzyme A. These molecules are degraded to carbon dioxide and hydrogen atoms in the Kreb's cycle within the mitochondria. Under anaerobic conditions, the process of oxidation and phosphorylation of 3-phosphoglyceraldehyde soon consumes the available supply of NAD^+. In this event, the accumulating NADH in the cytoplasm can be oxidized by the reaction which degrades pyruvate to lactate and which is catalyzed by the specific isozyme lactate dehydrogenase.

4.Lactate Removal

High concentrations of lactate in the exercising muscle may be catabolized by the muscle itself (Bang, 1936; Bonen et al., 1978; McGrail et al.,1978; Stamford et al.,1981; Welch & Stainsby,1967) or may diffuse into the blood stream. There are many organs other than muscle which have the capability of consuming lactate. These include the heart (Keul et al.,1972), Spleen, liver (McGrail et al.,1977), Kidneys (Jorfeldt,1970) and brain (Belcastro & Bonen,1975). Most of the lactate (50-75%) is

oxidized (Brooks,1986; DePocas et al.,1969; Donovan & Brooks,1983; Eldridge,1975; Issekutz,1984; Issekutz et al.,1976; Jorfeldt,1970) by muscle tissue. This evidence has been provided by studies using labelled lactate on rats (Brooks et al.,1973 Donovan & Brooks,1983; Freminet et al.,1969; Eldridge,1975; Issekutz,1970; Issekutz,1978; Issekutz,1984) rabbits (drury & Wick, 1956), dogs (DePocas et al., 1969; Eldridge, 1975; Issekutz, 1970; Issekutz, 1978; Issekutz, 1984), and humans (Hubbard, 1973). The data demonstrate that oxidation is the major pathway to dispose of lactate. At an exercise level of 50 to 75 % of $\dot{V}O_2$ max, the rate of lactate turnover was positively correlated (r=0.92) to the rate of oxidation expressed as $\dot{V}O_2$ uptake (Brooks et al.,1984). The majority (75%) of blood lactate produced during exercise is metabolized by the working (Minaire & Forichon, 1975) as well as the non-working muscle (Poortmans et al.,1978). Approximately 25% of the lactate is resynthesized to glycogen in the liver during rest. This percentage is reduced (12-20%) during exercise. Heart tissue is capable of taking up about 10% of the lactate during exercise, while small fractions of blood lactate are removed by the kidneys, brain, and spleen (McGrail et al.,1977). Under aerobic conditions, the lactate can be oxidized to form H_2O and CO_2 (55-70%) (Mayes,1985). In addition, lactate can also be converted into protein constituents (5%-10%), glucose,or other substances such as amino acids, and TCA intermediates (10%) (Gaesser & Brooks,1984).

Following high intensity exercise, exess lactic acid diffuses from the working muscle into the blood (DeCoster et al.,1969). Blood lactate is removed faster by maintaining the metabolic rate at an elevated level (Belcastro & Bonen, 1975). Results from tracer studies, using isotopically labelled carbon (C^{14}), demonstrated that there is a close relationship between the rate of lactate turnover and metabolism (Brooks et al.,1973; Brooks & Divine-Spurgeon, 1982; Eldridge,1975; Issekutz et al.,1976; Jorfeld,1970; Mazzeo et al.,1982). As the metabolic rate increases, lactate production and catabolism increase (Donovan & Brooks,1983). However, lactate reduction is expected when the removal rate is equal to or

greater than the rate of lactate diffusion into blood (Jorfeldt et al.,1978; Karlsson et al.,1968; Stegmann & Kindermann, 1982; Wasserman et al., 1967). A higher rate of lactate catabolism was also exhibited in rats after endurance training. Following biochemical analysis of infused $U^{-14}C$, investigators reported an increased lactate disposal instead of production in trained rats (Freminet et al.,1975). The combination of an increase in lactate removal and a decrease in production resulted in a lower lactate level following exercise training.

Lactate concentration in exercising muscle is associated with the FT fiber distribution (Aunola & Rusko,1986; Baldwin et al.,1977; Tesch, 1978). The FT muscle fiber may enzymatically stimulate lactate formation. According to Gollnick et al.,(1986), the maximal rate of lactate production appears to be about 0.5 mmol/g wet weight/s in muscle with high concentration of FT fibers and was estimated to be approximately 50% less in muscle with a predominance of ST fiber. This finding is in agreement with a previous investigation (Bonen et al.,1978), which reported a good correlation (r=0.544) between the percentage of ST fibers and the rate of lactate reduction. Two assumptions are made with respect to the influence of muscle type on lactate levels. First, ST fibers are surrounded by more capillaries than FT fibers (Saltin & Gollnick, 1983) which appears to increase the rate of lactate diffusion out of the muscle (Andersen,1975). Second, ST fibers contain heart specific LDH enzyme (Sjodin,1976) and myoglobin (Jacobs et al.,1987; Jansson et al., 1978; Nemeth & Lowry,1984) which favors lactate re-conversion to pyruvate and further oxidation to water and carbon dioxide. Along this line, many investigations have shown that exercise which predominantly recruits ST fibers may promote lactate removal (Bonen et al.,1978; Bulbulian et al.,1987; Gisolfi et al.,1966; McGrail et al.,1978; Stamford et al.,1978; Stamford et al.,1981; Weltman et al.,1979).

5.Lactate Accumulation

A rapid elevation in lactate levels is commonly seen following short term, heavy isometric comtractions (Ahlborg et al.,1972; Karlsson & Ollander,1972) and intense isotonic contractions (Karlsson,1971; Knuttgen & Saltin,1972) requiring the progressive recruitment of many muscle fibers (Clausen,1976).

Although Karlsson (1971), using muscle biopsies, observed a negligible variation in lactate distribution in different locations in a muscle, the lactate production was greatly influenced by fiber type, enzyme levels, and enzymatic activity. When exercise is performed at high intensity, the fast twitch fibers are the major contracting unit. This type of fiber generates greater tension per unit of time and produces more lactate than slow twitch fibers (Aunola & Rusko, 1986; Tesch,1980). FT muscle fibers contain a greater concentration of the isozyme lactate dehydrogenase that is specific to muscle (LDH-5 or M-LDH) (Sjodin,1976). M-LDH isozyme facilitates the reduction of pyruvate to lactate (Sund, 1968). Research has shown that FT fibers have a higher total LDH activity, which in turn may promote lactate production (Tesch,1978). Further support for the contribution of FT fibers to lactate production is provided by research involving patients with McArdle's disease. These patients cannot produce lactate. This syndrome is attributed to an enzymatic disorder occurring at the fast twitch fiber level (Kula et al.,1977).

Muscle glycogen content is another factor that may influence lactate concentration. Maughan and Poole (1981) have shown that following a high carbohydrate diet, resting lactate was elevated (2.20 mmol/l;$P<0.05$) more than after a normal diet (1.54 mmol/l). A lower level was found following a low carbohydrate diet (0.98 mmol/l;$P<0.01$). During a two minutes exercise, the peak lactate values were 8.60, 11.66, 12.86 mmol/l for the low, normal, and high carbohydrate diets, respectively. These results support the conclusion that if muscle glycogen conentration is reduced to a critical level, then muscle lactate will be decreased during intense exercise (Jacobs,1981a; Jacobs, 1981b; Hermansen,1969).

Evidence has shown that endurance training results in an enhanced ST fiber proportion and a lower rate of lactate production. When an individual trains one leg then performs exercise with both legs, a lower muscle and blood lactate concentration is found in the trained leg than the untrained leg (Ahlborg et al.,1975; Saltin et al.,1976). This finding concurs with studies of Asmussen et al.,(1974), and Hermansen (1971), indicating that during submaximal exercise, there is a lower muscle and blood lactate concentration in endurance trained individuals when compared to their untrained counterparts. Previous studies have shown that endurance trained muscles demonstrate a lower FT/ST ratio than untrained muscles (Jansson,1975; Jansson et al.,1978; Jansson & Kaijser, 1977; Nygaard,1976; Tesch,1980). Sahlin and Henriksson (1984) found a higher percentage of ST fibers and enzymatic activities in the trained subjects than in the untrained subjects. The trained subjects were able to sustain an isometric contraction at 60% of maximum voluntary contraction for a longer period of time while producing less lactate. The effect of sprint and endurance training on lactate production has been examined by many investigators (Medbϕ & Sejersted,1985; Sejersted et al.,1982; Sharp et al.,1986; Tesch & Karlsson,1984). Results from these studies have shown that sprint trained muscle demonstrates a higher FT/ST ratio, higher glycolytic enzyme activity, and an increased lactate concentration after hard exercise as compasred to endurance trained muscle.

The classic concept regarding lactate metabolism during exercise postulates that a deficit in oxygen uptake and delivery during anaerobic conditions stimulates glycolysis, glycogenolysis and lactic acid production (Hill,1924). However, findings of tracer studies (Brooks & Donovan, 1983; Brooks et al.,1973; Brooks et al.,1985; DePocas et al.,1969; Donovan & Brooks,1983; Eldridge et al.,1975; Freminet et al.,1974; Issekutz et al.,1976; Issekutz,1984; Terblanche et al.,1981), suggest that the classic concept is incorrect. Instead, lactate is formed not only during the exercising state but also at rest when adequate oxygen is available. In fact , lactate formation is an on-going activity (Kreisberg et al.,1970;

Mazzeo et al.,1982) in a constant state during rest. Data from many sources indicate that approximately 1-2 mmol of lactate per kilogram of muscle or per liter of blood exists at rest in normal subjects (Fujitsuka et al.,1979; Ivy et al.,1987; Karlsson,1970; Karlsson,1971; Poortmans et al.,1978). This small amount of lactic acid is manufactured due to basal metabolism of tissues in a resting state.

Investigations have also examined the rate of lactate accumulation. Margaria et al.(1963) and Saiki et al.(1967) suggested that lactate production terminates when steady state is reached. In contrast, recent studies using isotopic techniques in animals (Brooks & Gaesser, 1980; De-Pocas et al.,1969; Eldridge,1975) and humans (Hubbard,1973; Mazzeo et al.,1982), have found that lactate accumulation is a function of lactate production and removal rates. When the rate of production exceeds that of removal, accumulation occurs. When no lactate accumulation is found. it was postulated that production and removal of lactate were matched (Eldridge,1975). Hence, it appears that lactate accumulation is primarily dependent on the balance between production and removal (Brooks & Divine-Spurgeon.1982: Gollnick & Hermansen, 1973; Jacobs, 1986).

6.Lactate Levels and Performance

Performance of intense muscular exercise results in a rapid increase in lactate levels to approximately 20-30 mmol.kg-1 of muscle (Hermansen,1971; Hermansen & Stensvold.1972; Hermansen & Vaage,1977; Hermansen & Osnes,1972; Karlsson,1971). This source of lactate has been described as an energency process for energy yield (Fujitsuka et al., 1980) when aerobic metabolism is insufficient to meet the energy demands. The anaerobic metabolism of glycogen is therefore used to supplement aerobic metabolism (Hill et al .,1924; Meyerhof,1920). Theoretically, under anaerobic conditions, the lack of oxygen in the mitochondria inactivates oxidative phosphorylation by reducing the electron exchange process. This reaction results in a decline in both the [NAD]/[NADH]

ratio and the [ATP]／[ADP]・[Pi] ratio in the mitochondria and cytoplasm (Stainsby,1986). NAD^+ is an important coenzyme for optimal efficiency of the glycolytic process (Boxer & Devlin,1961; Edington,1970; Edington, et al.,1973). The coenzyme NAD^+ accepts hydrogen ions generated by anaerobic glycolysis. A high concentration of hydrogen ions interferes with calcium which is released from the sarcoplasmic reticulum (Hermensen & Osens,1972; Hultman et al.,1986). Calcium binds to troponin C (Fuchs et al.,1970; Hermansen & Osnes,1972; Hultman et al., 1986; Portzehl et al.,1969) and affects the action potentials of muscle fibers by altering the permeability of the membrane to Na^+ and K^+ (Nocker,1964). Muscle force decreases due to the lack of excitation-contraction coupling (Nagata et al.,1981). Anaerobic type of work degrades NAD^+ to NADH due to the high glycolytic activity. Lactic acid not only influences several of the rate-limiting steps in the glycolytic process (Danforth,1965) but it is also a potential inhibitor of fat mobilization from adipose tissue (Boyd et al.,1974; Issekutz et al.,1966). When fat oxidation decreases in the muscle, there is an increased demand on the limited muscle glycogen stores as the fuel for muscle contractions. As muscle stores diminish, there is an earlier cessation of work (Wenger & Reed,1976).

Lactic acid accumulation is a source of muscular fatigue and a cause for decreased exercise potential. Researchers using supra-maximal exercise (Jones & Green,1984; Karlsson et al.,1975; Weltman et al.,1977; Yates et al.,1983) as well as other experimental treatments (e.g. pharmacological administration, atmospheric manipulation, and dietary modification), have demonstrated that as lactate levels increase, power output decreases linearly. In contrast, the utilization of fatty acids reduces lactate production due to decreased glycolytic activity. The influence of lactate on fatty acid utilization and beta-oxidation is slowed by blocking the enzyme specific for the process (Wenger & Reed,1976). At an exercise intensity below 40% of maximum oxygen uptake, there is a negligible or no change in lactate concentration when compared to resting

values (Gollnick et al.,1986; Stamford et al.,1981).

Many physiological investigations which aimed at achieving high lactate production or accumulation have used exercise intensities from 75% to 120% of $\dot{V}O_{2\ max}$ (Bonen et al.,1979; Bonen & Belcastro,1976 Bonen & Belcastro,1977; Dykstra et al.,1973; McLellan & Skinner,1982; Siebers & McMurray,1981). It is difficult for the subject to complete a given physical test if the exercise intensity is too high. A work load of 275 watts has been shown to be an appropriate exercise intensity for an anaerobic performance test (Wasserman et al.,1986). For a 30 to 60 second cycling test, the workload is related to the braking force of the cycle ergometer. Resistances of 0.095 kp/per kg body weight (Evans & Quinney,1981; LaVoie et al.,1984; Patton et al.,1985; Vandewalle et al., 1985) and 0.086 kp/per kg body weight (Dotan & Bar-Or,1983; Vandewalle et al.,1985) have been recommended for well trained male and female adults, respectively. The determination of an optimal exercise intensity for monitoring a marked lactate response depends on the fitness level of the subject and the duration of the exercise test.

7.Recovery Activity and Lactate Removal

The resting period of intermittent exercise permits the removal of the lactate metabolite. Evidence from many sources indicates that exhaustive exercise followed by active recovery reduces lactate more than passive recovery. The effectiveness of lactate removal during active recovery is a function of the exercise intensity, the mode of recovery, and the duration of recovery (Watson et al .,1986).

There is much discussion about the optimal intensity for recovery activity. Many investigators (Belcastro & Bonen,1975; Bonen et al.,1978; Davies et al.,1970; Dodd et al.,1984 McGrail et al.,1978) have postulated that exercise at 30-40% of $\dot{V}O_{2\ max}$ following high intensity physical effort decreases lactate concentration at a greater rate than passive recovery alone. In addition, there seems to be less lactate removed at an exercise recovery between 60-80% of $\dot{V}O_{2\ max}$. Results from a study by Stamford

et al.(1981) disagree with this conclusion. They studied recovery exercise above and below the anaerobic threshold following maximal exercise. Six males performed 40 minutes of passive recovery and cycling recovery at 40% and 70% of $\dot{V}O_{2\ max}$. Blood samples were taken at rest, immediately after maximal exercise, and at 1, 2, 3, 4, 5, 7, 10, 15, 20, 30, and 40 minutes of cycling recovery. The investigators found, while not statistically significant, a lower post-exercise lactate concentration following 4-5 minutes of recovery at 70% of $\dot{V}O_{2\ max}$ (12.8 mM) as compared to 5 minutes of recovery exercise at 40% $\dot{V}O_{2\ max}$ (13.3 mM). The investigators suggested that high intensity recovery exercise, in the early stage of recovery, is probably suitable for a faster reduction in lactate.

Bulbulian et al.(1987) compared the effectiveness of three modes of recovery activity on lactate removal. Following three separate maximal graded exercise tests, one female and five male subjects performed either running recovery on a treadmill at 35% of $\dot{V}O_{2\ max}$ or rested by assuming either a supine or a seated positions of a possible hemodynamic contribution to lactate removal by assuming different body positions during recovery. No significant difference was found in lactate removal between the supine and the seated position. In contrast, treadmill running at 35% of $\dot{V}O_{2\ max}$, resulted in a significantly greater $\dot{V}O_{2}$ and there was a significantly higher lactate reduction (56 and 28.1 mg.100 ml-1 blood)at 15 and 25 minutes into recovery when compared to the supine condition (61.2 and 41.1 mg.100 ml-1 blood) and sitting condition (65 and 44.9 mg.100 ml-1 blood). Active recovery enhances the circulation of lactate from high glycolytic areas to high respiratory tissues. Gollnick et al.(1986) pointed out that as blood flow is augmented through the muscle, there is an increase in oxidation, efflux, and transportation of muscle lactate following heavy exercise. This observation concerning lactate removal, is in agreement with that of Newman et al.(1937) and Rammal and Strom (1949). They found that increased blood flow aided the rate of lactate removal. This viewpoint has been supported by numerous investigators. (Belcastro & Bonen,1975; Boone,

1984; Dodd et al.,1984; Dykstra et al.,1973; Kaczynski,1989; Koutedakis & Sharp,1985; McLellan & Skinner,1982; Stamford et al.,1981; Timson, 1976).

There is, however,little agreement with respect to the optimum intensity of recovery activity for lactate removal. One group of researchers suggested that the optimal exercise intensity for active recovery should be 25-45% of $\dot{V}O_{2\ max}$ (Belcastro & Bonen,1975; Davies et al.,1970; Dykstra et al.,1973; Koutedakis & Sharp,1985; McLellan & Skinner,1982). In contrast, another group of researchers,postulated that greater lactate disposal occurs when recovery exercise is performed at higher intensities (50-75% of $\dot{V}O_{2\ max}$) (Hermansen & Stensvold,1972; Spath,1974; Stamford et al.,1981; Timson,1976).

Davies and associates (1970) compared the effect of four exercise intensities on recovery lactate. Four subjects performed six minutes of physical exercise on a cycle ergometer at 80% of $\dot{V}O_{2\ max}$ followed by active recovery at 15%, 30%, 45%, and 60% of $\dot{V}O_{2\ max}$. Lactate removal was greaer at exercise levels of 30% and 45% of $VO_{2\ max}$ and lower at 15% and 60% of $\dot{V}O_{2\ max}$. Dykstra et al.(1973), Koutedakis and Sharp (1985), and McLellan and Skinner (1982), were in agreement with these results.

Dykstra and colleagues chose 25%, 40%, 55%, and 70% of $\dot{V}O_{2\ max}$ as recovery intensities following eight minutes of exercise at 90% of $\dot{V}O_{2\ max}$. An increased lactate disappearance was found during exercise between 25% and 47% of $\dot{V}O_{2\ max}$. Belcastro and Bonen (1975) utilized a similar protocol in an attempt to determine the most effective recovery intensity for lactate disposal. Following six minutes of exercise at 89% of $\dot{V}O_{2\ max}$, the subjects recovered either passicely or actively at 30%, 45%, 60%, and 80% of their $\dot{V}O_{2\ max}$. Exercise between 29.7% and 45.3% of $\dot{V}O_{2\ max}$ contributed to the highest rate of lactate reduction.

Weltman et al., (1977) combined varying durations of recovery exercise and the inhalation of oxygen in their experimental design to examine the effect of active recovery on subsequent performance. Subjects

began with an all-out pedalling task on a cycle ergometer against a 5.5 kg resistance for 60 seconds and was followed by one of eight randomly assigned recovery patterns and a repeat of the all-out exercise task at the end of each experimental condition. The eight experimental sessions consisted of passive and active recovery, breathing room air or 100% oxygen, for a 10 or 20 minute duration. The results indicated highly significant main effects for active vs passive recovery, and for 10 vs 20 minutes of recovery, which lead to both increased lactate removal and greater post-recovery exercise performance on a cycle ergometer. Oxygen inhalation was found to have no effect on both lactate removal and subsequent performance. Since the resulting correlation was low (r=-0.19), these investigators suggest that other factors apart from lactate removal were critical for subsequent performance.

Using aerobic threshold as a criteria to determine the optimal recovery intensity, McLellan and Skinner (1982) reported that the most expedient lactate removal occurred when recovery exercise was performed at an intensity expressed as the aerobic threshold minus 10%. This intensity was equal to 43% of $\dot{V}O_{2\ max}$. The researchers also postulated that a recovery intensity between 27%-58% of $\dot{V}O_{2\ max}$ may elicit marked lactate removal. The subjects for this investigation performed 10 minutes of exercise at 90% of theit $\dot{V}O_{2\ max}$ on order to produce high lactate. Recovery conditions included rest or exercise at the aerobic threshold, -10%, -20%, -30%, and +10% of aerobic threshold.

A recent study by Koutedakis and Sharp (1985) examined the effects of recovery intensity on lactate. Subjects (rowers) sprinted 2000 meters and were told to recover by resting or by continuous stroke exercise at 40% and 60% of $\dot{V}O_{2\ max}$. Results were similar to those of prevous investigations. Greater lactate turnover was found after active recovery compared to passive and low intensity recovery. Recovery at 40% of $\dot{V}O_{2\ max}$ was more effective than at 60% $\dot{V}O_{2\ max}$.

On the other hand, conflicting results exist with regards to the effectiveness of recovery intensity on lactate removal. A greater or faster

lactate removal has been seen following recovery exercise at 50%-70% of $\dot{V}O_{2\ max}$. Hermansen and Stensvold (1972) investigated the optimal recovery intensity for effective lactate disposal. They examined the effect of recovery exercise (30%, 60%, 70%, and 80% of $\dot{V}O_{2\ max}$), on lactate removal rate. Results showed that 0.8 mmol/min of lactate would be removed at 63% of the subjects $\dot{V}O_{2\ max}$. Timson (1976) showed that lactate reduction following 10 minutes of exercise was 0.78 mmol/min when the subjects performed active recovery at 57.1-70.0% of $\dot{V}O_{2\ max}$. Spath (1974) observed a higher lactate reduction rate (1.03 mmol/min) when the subjects performed exercise reconvery at 59.2% of $\dot{V}O_{2\ max}$. Results from these studies propose higher (50-70% of $\dot{V}O_{2\ max}$) recovery intensities. Using the baseline lactate concentration as the criteria to verify the effect of recovery intensity on lactate concentration, Stamford et al.(1981) studied the contribution of recovery intensities for the half time reduction of lactate. Base line lactate was determined to be 0.9 mM for the resting state, 1.3 and 3.5 mM for 40% and 70% of $\dot{V}O_{2\ max}$, respectively. Six subjects exercised on a cycle ergometer with an initial workload of 50 watts with increments of 25 watts for each work bout until volitional exhaustion. The work bouts consisted of 3 minutes of cycling with 5 minute trcovery intervals between bouts. Recovery conditions were rest, exercise at 40% of $\dot{V}O_{2\ max}$, and 70% $\dot{V}O_{2\ max}$. In the first 5 minutes of recovery, lactate reduction was best for the 70% condition. However, when the half time reduction was analyzed, 40% recovery exhibited a significantly faster half time than either 70% recovery or resting recovery. These investigators hypothesized that there is no single optimal recovery exercise intensity for eliciting lactate disposal. They recommended to start the recovery with a higher intensity and gradually decrease the exercise intensity. Two subsequent studies tested this hypothesis.

Boone (1984) and Dodd et al.,(1984) implemented Stamford's thinking by utilizing recovery exercise with a high initial intensity, then the workload was progressively decreased. In the former study, the subjects

performed 50 seconds of maximal cycling exercise followed by one of four recovery treatments for 40 minutes: (1) passive recovery, (2) cycling at 35% of $\dot{V}O_{2\ max}$, (3) cycling at 65% of $\dot{V}O_{2\ max}$ (4) cycling at 65% for 7 minutes followed by cycling at 33% of $\dot{V}O_{2\ max}$ for 33 minutes. The rate of lactate disappearance was significantly higher ($P<0.05$) when recovery was at 35% of $\dot{V}O_{2\ max}$ and the 65%-35% combination for recovery compared to either passive or the 65% recovery condition. No significant difference ($p>0.01$) occurred between 35% and the combination recovery treatment. The latter finding failed to support the hypothesis of Stamford et al.(1981). However, Boone (1984) found conflicting results with respect to the results found by Dodd et al.(1984) and supported Stamford's hypothesis. In Boone's study, following cycling exercise, the subjects were randomly assigned to four 20-minute recovery groups:(1) resting, (2) 40% of $\dot{V}O_{2\ max}$, (3) 80% of $\dot{V}O2$ max, (4) 80% of $\dot{V}O_{2\ max}$ for the first 5 minutes then 40% of $\dot{V}O_{2\ max}$ for the last 15 minutes. Results demonstrated that the combination of 80%-40% active recovery produced the most effective lactate disappearance (0.65 mmol/min) when compared to the other recovery protocols.

Bonen et al.(1979), identified three factors that influence the lactate removal rate. These factors were (1) ST fiber proportion, (2) blood lactate levels at the end of exercise, and (3) the intensity of the recovery exercise. To predict optimal recovery intensity, the investigators developed a regression equation that included these factors. To validate the equation, the subjects exercised for six minutes at 90% of $\dot{V}O_{2\ max}$. Recovery exercise was then performed at 40% of $\dot{V}O_{2\ max}$. Statistical analysis indicated that the observed value was significantly correlated (0.91) with the predicted value.

Bonen and Belcastro (1976) reviewed previous articles concerned with recovery intensity on lactate removal. They noted that the optimal intensity occurred from 55% to 70% of subjects $\dot{V}O_{2\ max}$ for running recovery. The following factors may influence the rate of lactate metabolism:(1) recovery activity, (2) exercise intensity, (3) individual

variations of LDH isozyme level and LDH enzymatic activity, (4) FT/ST fiber ratio, (5) physical fitness level (6) initial lactate concentrations, and (7) muscle glycogen content (Bonen & Belcastro,1977; Bonen et al.,1978; Bonen et al.,1979; Hermansen,1969; Jacobs, 1981a; Jacobs, 1981b; Mann & Garret, 1978; Tesch et al.,1978).

8.Glycogen Utilization and Exercise Performance

Substrate utilization during sport participation has been a major topic in many research studies. Many investigators credit the utilization of carbohydrates as a substrate source which is one of essential peripheral factors affecting muscular activity (Essen et al.,1977; Gollnick et al., 1974; Saltin & Karlsson,1971).

Results from previous histochemical studies have shown that the store of glycogen substrate is equally distributed within FT fibers and ST fibers (Ess'en et al.,1975) even though these two types of muscle possess distinctive characteristics. The potential mechanism for glycogenolysis during physical effort is evident as the work intensity approaches maximal and supramaximal work levels. Following high intensity exercises, Saltin and Karlsson (1971) determined that more than 10% of the muscle's normal glycogen store was used per minute, this utilizastion was in the order of 3.4 glucosyl units/kg.min at 100% of $\dot{V}O_{2\ max}$. The resultant findings suggest that an exponential increase in the rate of glycogen utilization occurs as a function of exercise intensity in terms of $\dot{V}O_{2\ max}$.

When subjects performed six, 60-second bouts of exercise separated by 10 minute rest periods, Gollnick et al.(1973) visualized the glycogen depletion pattern in FT and ST fibers of the vastus lateralis muscle during bicycling exercise. At a workload corresponding to 150% of $\dot{V}O_{2\ max}$, there was a linear decline in glycogen content as a function of the number of completed sprints. The mean pre and post-exercise glycogen concentrations were 132 and 49 mM glucose units/kg, respectively. The PAS staining intensity determined the proportion as 46% dark, 16%

moderate, 26% light, and 12% negative for FT fibers, while ST fibers were rated 76% dark, 2% moderate, 13% light, and 9% negative. These values indicate greater glycogen sparing in FT muscle fibers as a result of high intensity exercise.

In order to determine the muscle glycogen sparing pattern in FT fiber subgroups during submaximal and supramaximal exercise, Thomson et al.,(1979) employed an experimental protocol consisting of ten, one-minute bouts of supramaximal (120% of $\dot{V}O_{2\ max}$) exercise interposed with five minute rest periods. The resulting PAS intensity indicated higher negative rating (76%) for the FTb fibers than that for FTa fibers (25%). The glycogen was reduced to half (52%) of its total concentration following the supramaximal exercise.

Essen (1978) observed a higher glycogen depletion rate in the type I fibers (277 mmol/kg d.w.) than the type II fibers (113 mmol/kg d.w.) during continuous submaximal exercise. Yet, the glycogen depletion rate was parallel between type I (213 mmol/kg d.w.) and type II fibers (203 mmol/kg d.w.) after intense intermittent exercise. The performance tests for this study were cycling at 50-60% of $\dot{V}O_{2\ max}$ for 60 minutes for the continuous protocol and a work: recovery ratio of 1:1 (15s:15s) at 100% of $\dot{V}O_{2\ max}$ for the intermittent protocol.

The difference between continuous and intermittent exercist was determined in terms of glycogen sparing pattern. Green (1978) noted an overall glycogen reduction of 29% and 70% of the pre-exercise concentrations for continuous and intermittent skating, respectively. In addition, glycogen depletion was greater in the type I fibers during the continuous skating condition and in the type II fibers during the intermittent condition.

In light of the above studies, it appears that the rate of glycogen utilization increases exponentially as a function of exercise intensity with respect to $\dot{V}O_{2\ max}$, and increases linearly as a function of exercise duration. Furthermore, maximal or supramaximal exercise elicits a predominant muscle depletion in FT fibers with a greater depletion in FTb fibers.

9.Implications of Lactate Removal for Intermittent Exercise

Information regarding lactate metabolism has been widely utilized in many sports. High intensity, short duration exercise results in a rapid rise in lactate levels which is detrimental to performance. Intermittent exercise produces greater stress on the body. The team coach may choose short work bouts in order to avoid excessive lactate production as well as to slow down the rate of glycogen depletion. Recovery periods can be used to lower lactate levels and replenish glycogen stores.

Ice hockey is a popular game in North America. It is characterized by high intensity intermittent skating. A typical hockey player performs for 15 to 20 minutes during a 60-minute game. Each shift lasts from 30 to 80 seconds with 4 to 5 minutes of recovery between shifts (Montgomery,1988).

High intensity bursts of skating during the on-ice shift of a hockey player results in 3 to 11 mmol/L of lactate production in the blood (Green et al.,1976; Green et al.,1978; Wilson & Hedberg, 1976). If lactate production exceeds the rate of lactate removal, the substrate reaches a level where it interferes with performance (Hogan & Welch,1984; Karlsson et al.,1975; Klausen et al.,1972; Tesch & Wright,1983; Yates et al.,1983).

During the periods of continuous play, hockey players exercise close to their $\dot{V}O_{2\ max}$, with a significant energy contribution from glycolysis. ATP and CP stores are completely depleted in appoximately 6 seconds of maximal exercise (Bergstrom et al.,1971; McArdle et al.,1986). As a result of glycolysis and depending on the biochemical and physiological profiles of the energy systems, pyruvate will either be reduced to lactate or oxidized to form acetyl-coenzyme A (Lehninger,1982).

The study of hockey playing intensity has some implications regarding the effect of shift length on lactate levels. With a trend towards shorter and fewer play stoppages, higher intensity work output should

produce higher lactate levels at the end of a shift. Bouts of six intermittent repetitions of all-out skating produced blood lactate values between 10.7 to 12.1 mmol/L (Montgomery,1988; Watson & Sargeant,1986). Following six 45 second maximal skating trials, subjects were found to have lactate levels of 12.1 mmol/L (Watson & Hanley,1986).

If each shift on the ice is terminated prior to excessive accumulation of lactate, recovery characteristics are much faster. The recovery period can be used to replenish myoglobin stores, resynthesize phosphocreatine and lower lactate (Montgomery,1988).

Bonen and Belcastro (1977) concluded that the half-time for lactate removal is 20 minutes in a passive condition. Hockey players have only 3.5 to 4 minutes of rest between shifts (Green & Houston,1975). Hence, lactate will accumulate more rapidly than it can be removed from the system. This may cause a decrement in performance over time. If lactate levels are used to evaluate work output (Jacobs,1986) then a performance decrement is demonstrated by the fact that lactate production at the end of the third period is usually lower than those measured at the end of the first or second period of play (Green et al.,1976; Green et al.,1978).

In the physiological evaluation of athletes, it is imperative that the testing protocol be specific to the demands of the sport. The cycle ergometer is frequently used to evaluate the aerobic and anaerobic capacities of hockey players in a laboratory setting (Montgomery,1988). Several studies (Brayne,1985; Daub et al.,1983; Watson & Sargeant, 1986) hav examined the specificity of on-ice testing versus laboratory testing of hockey players. Some research has indicated that the glycogen depletion patterns and muscles used in cycling are similar to those used in skating (Geijsel, 1979; Geijsel,1980; Green et al.,1978).

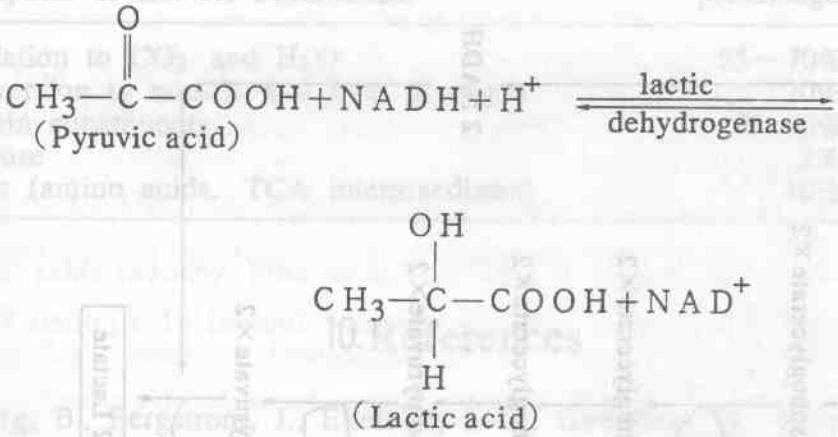

Fig. 1. Pyruvate acied is reversibly converted into lactic acid by enzyme lactic dehydrogenase

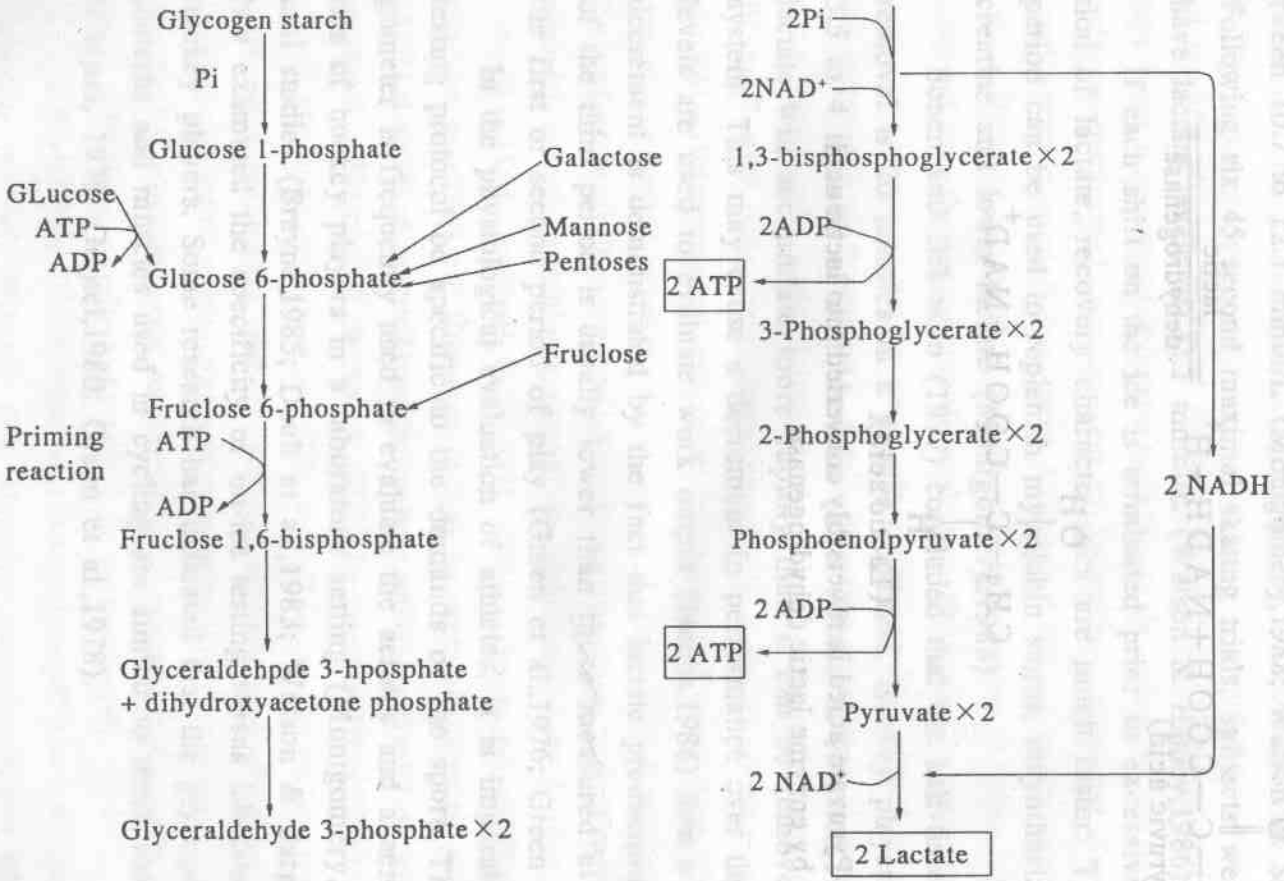

Fig. 2. Glycolysis pathway in which 5-carbon glucose is catabolized into two 3-carbon pyruvate.

Table 1. End point of Lactate Metabolism

Endpoint of Lactate Metabolism	percentage
- Oxidation to CO_2 and H_2O	55－70%
- Conversion to muscle and liver glycogen	20%
- Protein constituents	5－10%
- Glucose	2%
- Other (amino acids, TCA intermaediates)	10%

10. References

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