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The 3rd Annual International Seminar On Trends In Science And Science Education 201

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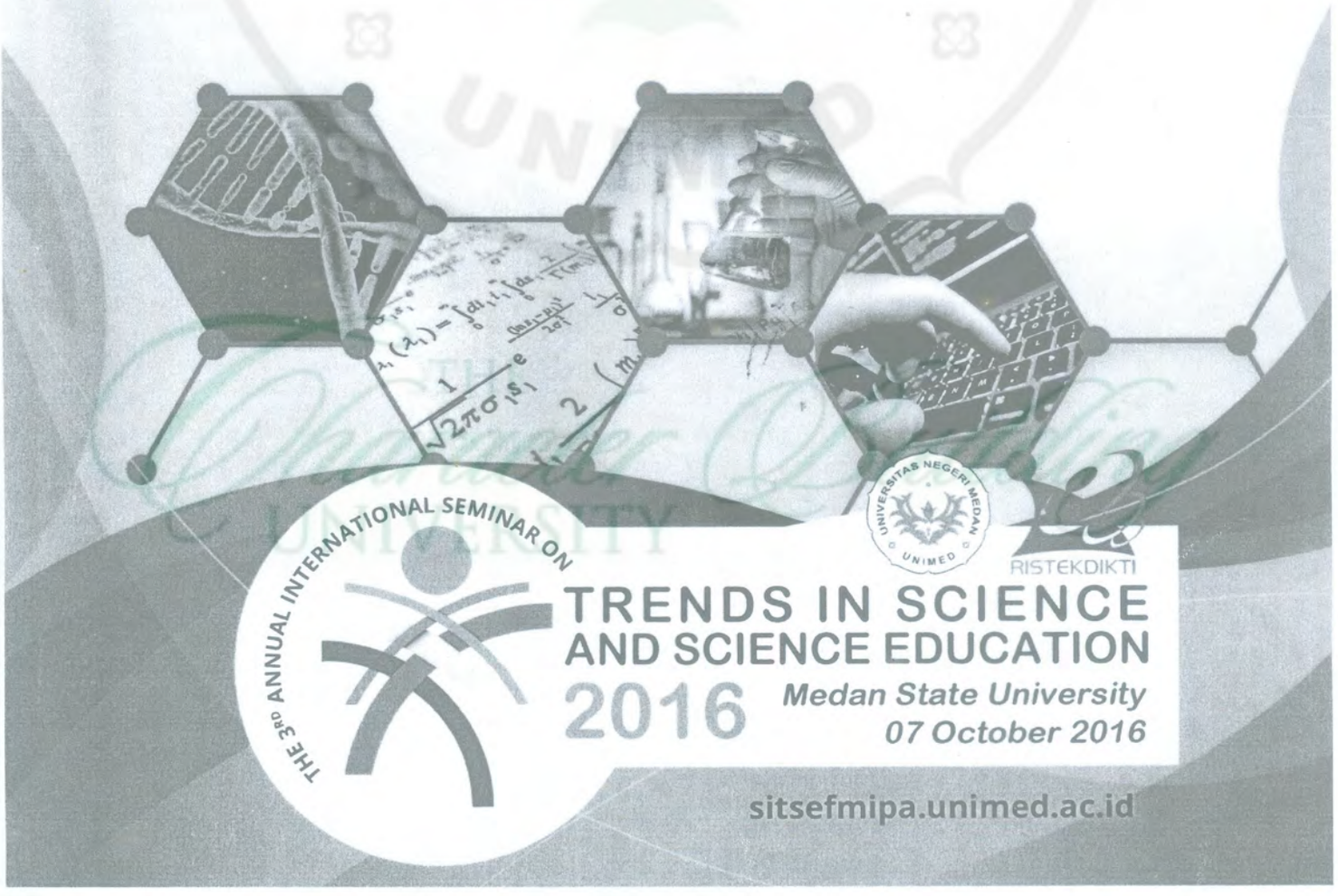
Faculty of Mathematics and Natural Science
Medan State University

October 7th, 2016



TRENDS IN SCIENCE AND SCIENCE EDUCATION
2016 State University of Medan 07-08 October 2016

ISBN: 978-602-60343-2-8

AISTSSE 2016

Annual International Seminar on Trends Science and Science Education

Proceedings

The 3rd AISTSSE
Trends in Science and Science Education

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7 October 2016

Organized by:
Faculty of Mathematics and Natural Sciences
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Publisher: Research Institute of Medan State University
(Lembaga PenelitianUNIVERSITAS NEGERI MEDAN)
Jln. Willem Iskandar, Psr V Medan 20222; Telp (061) 6636757; Fax. (061) 6613319-6614002 – Medan, Indonesia

Proceedings
The 3rd AISTSSE (Trends in Science and Science Education)
7 October 2016Organized by:
Faculty of Mathematics and Natural Sciences
Medan State University - North Sumatera-Indonesia :
Research Institute Medan State University Publisher (Penerbit Lembaga Penelitian Universitas Negeri Medan)

V, 251 pages, 27 cm

ISBN: 978-602-60343-2-8

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Cetakan I: 2017

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Preface I

Welcome to the 3rd Annual International Seminar on Trends in Science and Science Education 2016. This is the third time we are hosting this seminar and we are proud to inform you that this seminar is an annual event in our calendar and will be held every year since 2014. We are inviting international recognized speakers from several countries to share their latest discoveries to all of us in Biology, Chemistry, Physics, Mathematics and Science Education fields. Well known researchers in science and science education will share their experiences and knowledge so we can have up to date the information. This is one of the goals of this seminar.

As science researcher we realize the importance of information exchange among us. The new information enlighten our mind and give us ideas on what to do next in our research and how to do it. This new informationoften become the basic for our next project in particular, and become the upcoming year research trends in general. Information exchange also keeps us updated, allow us to give and receive suggestions and critics which will lead us to better results. Thus, we need a forum where we can share and exchange information. Seminar, conference and other scientific gathering are media for us to do so.

Wewould like to thanksto all the researchers who responded to our call for papers and participant of this seminar. Let us share information about our latest discoveries in science and science education and set the trends for the upcoming year. Let us collaborate and create new opportunities for a better and more holistic research.

Finally, we convey our thanks to the Rector of State University of Medan, Prof. Dr. SyawalGultom, M.Pd and all the vice rector for the support and attention to this seminar and also to all of the committee members for their work in ensuring the run of this seminar. Once again, welcometo the 3rd Annual International Seminar on Trends in Science and Science Education 2016.

Medan, 7 October 2016

Dr. Asrin Lubis, M.Pd.
DeanFaculty of Mathematics and Natural Sciences
State University of Medan

Preface II

First, let us be thankful to the one and all-powerful God that on this fine morning we are still given bodily and spiritual health and can gather together in this room, on our beloved capital city of North Sumatra, Medan.

A warm and special welcome goes to our keynote speakers, Dr. Mohd. Sazali Khalid (from University Tun Hussein Onn Malaysia), Prof. Dr. Janchai Yingprayoon (Suan Sunandha Rajabhat University, Thailand), Rabeta bt. Mohd. Salleh, Ph.D (University Sains Malaysia), Dee-Jean Ong (R.E.A.L. Education Group Malaysia) and Dr. Anna Ratna Wulan (from Universitas Pendidikan Indonesia).

The special welcome also goes to all invited speakers from top Universities all over Indonesia.

This seminar, The 3rd International Seminar on Trends in Science and Science Education 2016 is an annual seminar organized by Fakultas Matematika dan Ilmu Pengetahuan Alam, Universitas Negeri Medan. This is the third year seminar following the succesful first and second year seminar held in 2014 and 2015. This year seminar is focusing on the contribution of research to the development of technology. The committee expects the information exchange among researchers in this seminar will encourage collaboration among the different actors in science and science education community so as to achieve a better result for the benefit of the community. This third annual international seminar will be held from October 7 – 8, 201.

The committee are really honored to have attention from approximately 200 speakers and participants from three different countries. They come from Thailand, Malaysia and of course Indonesia. About 20 universities from all over Indonesia participate in this event. It is expected that those who participate in the seminar will afterwards be familiar and able to interact with their international counterparts in their scientific area. This is in line with the vision of Universitas Negeri Medan to become a world class and character building university.

The committee recieved more than 100 seminar abstracts and full papers from science education, biology, chemistry, physics, and mathematics sciences. Most of the abstract have been edited and bound into an abstract collection book which is a part of the seminar kit.The seminar full papers are now in editing stage by the committee before publish in seminar proceeding that will be available in

both printed and on-line forms, in the next January 2017. Please, remind the committee if you want to get the copy of the seminar proceeding.

This year seminar is a special event because it is held together with the annual meeting of all mathematics and natural science faculties from LPTK in Indonesia or Forum MIPA LPTK Indonesia. The meeting will be held from October 7-9, 2016, in Medan and Parapat. This forum is intended to built collaboration among LPKT's in Indonesia.

I would like to take this opportunity to acknowledge the important role of the honorable Prof. Dr. Syawal Gultom, M.Pd, rector of Universitas Negeri Medan for giving us his full support and attention and for providing his precious time to be with us and to honour us by opening this seminar.

Our sincere thanks also goes to the honorable Dr. Asrin Lubis, M.Pd, Dean of Fakultas Matematika dan Ilmu Pengetahuan Alam, who havelead and encourage all the committeemembers to be always focused and worked hard even in a very short period of time to prepare the seminar.

My sincere thanks also goes to all members of the committee and to all staff of Fakultas Matematika dan Ilmu Pengetahuan Alam for their continuous support and hard work because without their assistance this seminar may not have taken place today.

Finally, I conclude my speech by kindly inviting Honourable Prof. Dr. Syawal Gultom, M.Pd, Rector of Universitas Negeri Medan, to give special direction and officially open the seminar.We wish you good luck and success in this endeavor. Thank you very much

Prof. Dr. Herbert Sipahutar, MS., M.Sc.
Chairman AISTSSE 2016

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Table of Content

Lactate Metabolism and the Effect for Sprinter Performance

Novita Sari Harahap

Faculty of Sport Science, State University of Medan
Medan, Sumatera Utara

ABSTRACT : The ability to achieve and maintain speed is the main determinant of success in sprinter. The energy needed by sprinter from both phosphocreatine and anaerobic glycolysis, with each source contributing about 50%. Lactate is produced in muscle cells during a run will diffuse into the blood. In most cases, the diffusion rate is slower than the rate of production, so that after running intensive, lactate concentration increased in the 5th minute until the 10th. The high concentration of lactate during 10 minutes restitution shows the strong involvement of anaerobic glycolysis. Increased levels of lactic acid of high occurred during practice or game on a sprint runner can affect the performance of athletes. Furthermore, the lactic acid in the muscles will dissociate into H^+ ions and lactate ions. Increased H^+ ions causes a decrease in pH in the extra and intracellular fluid. This decrease in pH will affect and inhibit the binding of oxygen by hemoglobin in the lungs, and can inhibit the activity of the enzyme phosphofructokinase and myofibril ATPase enzyme in the muscle that plays a role in the synthesis of ATP thus providing energy during muscle contraction becomes impaired. Disruption of energy supply will reduce the ability of muscle contraction. If the decreased ability of muscle contraction, it will lower the running speed of sprinter.

Keywords : *metabolisme, glycolysis anaerobic, lactate, sprinter*

INTRODUCTION

Lactate metabolism is an integral pathway in physical exercise. Numerous contrasting views exist regarding the physiological effects of lactate and its roles post production. This paper attempts to clarify and highlight the significance of lactate in exercise. Lactate production is associated with muscular fatigue; and is a major limitation in athletic performance. This fatigue is partially due to the production of H^+ ions which depresses muscle functions. Lactate is transported in the skeletal muscles through plasma monocarboxylate transport (MCT) system and is utilized by muscles such as the heart and red muscles. It is also very important that the lactate produced to satisfy high energy demands is cleared from the muscles and metabolized by the liver or be utilized as an energy substrate [1].

Many coaches and athletes feel that the lactic acid as an end product that is less favorable to the performance of sprinter. The ability to achieve and maintain the speed is the primary determinant of success sprinter. The energy required by the runners 200m comes from phosphocreatine and anaerobic glycolysis, with each source contributes about 50% [2].

ATP is the primary source of energy during muscle work. During a sprinting effort, the amount of ATP increases even as much as 1000 times relative to the level of restitution and its resources in the muscles already become depleted during the 2 seconds into the run. Re-synthesis of ATP initiates a creatine kinase reaction immediately after the start of the effort. A small amount of ATP is produced as a result of the adenylate kinase reaction occurring in the cytoplasm. Another source of ATP re-synthesis is also anaerobic glycolysis activating immediately after the start of an effort [3].

Lactate is produced in muscle cells during a run will diffuse into the blood. In most cases, the diffusion rate is slower than the rate of production, so that after running intensive, lactate concentration increased in the 5th minute until the 10th. The high concentration of lactate during 10 minutes restitution shows the strong involvement of the anaerobic glycolysis [3].

DISCUSSION

Lactic acid is the result of glucose metabolism through anaerobic glycolysis reaction laktasid. Lactic acid is the chemical name Lactic Acid, 2-hydroxypropanoic acid with the chemical formula C3H6O3. Lactic acid is produced from the reduction of pyruvic acid, this process occurs in muscle tissue is starved of oxygen, for

example at the time of exercise intensity is relatively heavy [4,5]. n healthy individuals there is a continuous cycle of metabolism and the production of lactic acid in a sustainable manner so that the levels of lactic acid in normal circumstances. High lactic acid levels when production is higher than elimination, while capacity decreased elimination, or more often both occur simultaneously. Blood lactic acid levels in the resting state about 1-2 mmol/L [6].

In hypoxia, glycogen is converted into glucose, the glucose is converted further lactic acid. The lactic acid in the bloodstream goes to the liver, in the heart, lactic acid is converted into glucose through gluconeogenesis. Glucose back into the blood that will then be used in the muscle. In the muscle, glucose is converted back into glycogen. It is known as lactic acid cycle or cycles Cori [5].

Glycogen early cut into 1-phosphate glucose units and each unit is divided into two three-carbon fragments. The end product of the metabolism of glucose is pyruvic acid. Useful energy from glycolysis is 3-Adenosine diphosphate (ADP) and phosphorylated back to earn a 3-Adenosine triphosphate (ATP), and four hydrogen ions (H^+) per molecule of glucose 1-phosphate in the break up of glycogen. In anaerobic conditions, hydrogen ions are released in glycolysis, but the tricarboxylic acid cycle or the Krebs cycle can not combine with oxygen at a sufficient speed so that tends to accumulate in the muscles. Excess hydrogen ions is then used to convert pyruvic acid into lactic acid [7].

In aerobic conditions, the ion received by the carrier compound H^+, Nicotinamide adenine dinucleotide oxidation form (NAD^+) and transport H^+ into the mitochondria for phosphorylation back so as to produce four molecules of ATP. Furthermore, pyruvic acid enters the Krebs cycle and reformed into carbon dioxide and hydrogen ions. Carbon dioxide then diffuses into the bloodstream as a result of metabolic waste, while the hydrogen ions are accepted by NAD^+ to form a compound NADH (NAD in the form of reduction). The products of the metabolism of fatty acids and protein, may also enter the Krebs cycle and is converted into energy [7].

The next pyruvic acid diffuses into mitochondria and metabolized to carbon dioxide through the Kreb cycle. The metabolism of glucose into pyruvate may also occur as a result of reduction of enzyme cofactor that mengoksigenasi form of NAD^+ to NADH, the reduced form. Lactic acid is produced through the process of glycolysis and is formed in the cytosol catalyzed by the enzyme lactate dehydrogenase. NADH / NAD^+ is a cofactor exchange of hydrogen atoms that are released or dipakai.Oleh Therefore, the ratio of lactate / pyruvate is always proportional to the ratio of NADH / NAD^+ in the cytosol [7].

High concentration of lactic acid can also be accompanied by a high concentration of pyruvate or NADH disitosol, or both. This is a reversible reaction that helps the synthesis of lactic acid by normal ratio of lactate to pyruvate acid is 25:1. Lactic acid synthesis is increased when the formation of pyruvate in the cytosol exceeded its use by the mitochondria. This occurs when found to increase metabolic fast or when the delivery of oxygen to the mitochondria decline, as in hypoxia jaringan.Sintesis lactic acid can also occur when glucose metabolism exceeds the mitochondrial oxidative capacity [8].

Lactic acid is formed in the muscle cells will diffuse out of the cell, then the lactic acid extracellular will enter the cell when will be used as an energy source or as an ingredient in gluconeogenesis. To enter the cells of lactic acid in two ways, namely simple diffusion and diffusion facilitated. Only a small proportion of lactic acid that goes into the cell by simple diffusion through the cell membrane. While most of the lactic acid into the cell with the easy diffusion path through conjugation with the cations H^+, Na^+, and K^+. Muscle and blood pH gradient influence the direction of transport of lactic acid. If the pH of the muscle cell is lower than the pH of the blood, then the lactic acid out of the muscle cells are active and will enter the cell oxidative and cell adjacent inactive. However, if the blood pH is lower than the muscle cells, the lactic acid in the blood will go to the liver, heart, kidney, and muscle inactivity [9].

In the resting state simultaneously form the skeletal muscles and consume lactic acid. At the time of an increase in muscle contraction and the formation of lactic acid consumption more. In the resting state, the lactic acid produced by red blood cells, brain, muscle cells and intestinal mucosa. Erythrocytes was instrumental in bringing the results of glycolysis; however these cells do not have mitochondria and can not use oxygen to produce ATP, therefore red blood cells produce lactic acid through the regeneration of ATP during anaerobic glycolysis but not be able to use lactic acid [9].

Lactic acid is metabolized by the liver, kidneys and heart. In the resting state, the kidneys and the heart using lactic acid as energy, while the liver using lactic acid as a raw material in gluconeogenesis. Heart have more of the enzyme lactate dehydrogenase as compared to muscle tissue, it allows the heart to have a better ability to use lactic acid as raw material for energy. The energy source used by the heart muscle is derived from free fatty acids 60-80%, 10-20% glucose and the rest comes from the lactic acid [7,9].

Lactic acid is one of the parameters other than ACTH response to stress and adrenalin. Since more than 150 years ago has been known lactic acid is produced in the sheer number of the lot during muscle activity as a result of the process glikolisisanaerobik. Physical exercise causes a variety of physiological effects depending

on the intensity of physical exercise. Running or treadmill tests led to increased metabolic needs that go beyond the limits of rest (resting level), then the lactic acid production will increase over the limit lactate threshold (4 mmol/L). Lactic acid is the result of the reduction of pyruvate, the lactate dehydrogenase enzyme activity. Lactic acid can then be stacked oxidized back to pyruvate in the cytosol, this situation lasts physiologically [5.9].

The lactic acid is excreted through urine and skin in small amounts. Lactic acid levels in the blood veins in the resting state is 0.63-2.44 mmol / l or 5.7-22.0 mg / 100ml, whereas the largest source of lactic acid during isitrahat comes from the breakdown of glucose in the blood cells. Rapid lactic acid that is formed will be eliminated continuously. One's ability to eliminate lactic acid is highly dependent on the physical condition of athletes. What happens when the elevation of lactic acid levels in the blood circulation, means there is an increased entry of lactic acid into the blood circulation exceeds the rate of elimination of lactic acid [4.9].

When lakat acid concentration in the blood increases, the muscles at rest consume lactic acid as an energy source by way of oxidation up to 50%. While the active muscle is the tissue that can eliminate lactic acid in the greatest quantities by means of oxidation up to 90%. The ability of the muscles in lactic acid metabolism is due, skeletal muscle fibers consist of fibers delayed type (type I) is an aerobic-type fibers (red muscle fibers) and fast type (type II) is a type of anareobik fibers (white muscle fibers). Type I fibers contain more myoglobin, mitochondria and oxidative enzymes compared with type II fibers that have oxidative capacity. Whereas type II fibers (white muscle fibers) contains more creatine phosphate, glycogen, myosin ATPase and glycolytic enzymes compared with type I fibers, so that it has the capacity glycolytic, or anaerobic capacity. At low intensity activity is more dominant type I fibers are used, while in the high-intensity activity type I and II fibers used [10]. A study indicated that a linear relationship between the increase NAD +, lactic acid with an increased intensity of physical activity. At the time of the test ergometer bike with 50-70% VO2 maximum intensity will occur a sharp increase lactic acid levels in the blood and muscle sarcoplasmic. Occurrence of lactic acid in the muscles due to muscle tissue hypoxia [9].

The Process Of Formation Of Lactic Acid In Runners 200 M

Sports sprint classified as predominantly anaerobic exercise at a high intensity so that it will produce lactic acid with a high amount in muscles and sarcoplasmic. Increased levels of lactic acid of high occurred during practice or game on a sprint runner can affect the performance of athletes.

Furthermore, the lactic acid in the muscles will berdissosiasi into H + ions and lactate ions. Increased H^+ ions causes a decrease in pH in the extra and intracellular fluid. This decrease in pH will affect and inhibit the binding of oxygen by hemoglobin in the lungs, and can inhibit the activity of the enzyme phosphofructokinase and myofibril ATPase enzyme in the muscle that plays a role in the synthesis of ATP thus providing energy during muscle contraction becomes impaired. Disruption of energy supply will reduce the ability of muscle contraction. If the decreased ability of muscle contraction, it will reduce the running speed sprint runner [4.9]. Presence of acid in the muscle will interfere with the mechanisms associated with muscle contraction [11], namely:

1) Inhibit the enzyme activity of aerobic and anaerobic, this situation will reduce the capacity of aerobic endurance and anaerobic capacity,
2) Inhibit the formation of creatine phosphate (CP) and will interfere with motor coordination,
3) Inhibits the enzyme phosphofructokinase,
4) Inhibiting the release of $Ca2^+$ from the vesicles and thus the activity of troponin C has decreased, and resulting in disruption or cessation of contraction of muscle fibers,
5) Inhibit the activity of mATPase especially in fast muscle fibers, because mATPase in fast muscle fibers are sensitive to acid. From the above description may explain the mechanism causes a decrease in the athlete's performance runners sprint

Low intracellular pH due to lactic acid buildup will cause a bottleneck Ca2 + binding to troponin, thus inhibiting the formation of cross-bridge actin-myosin complex. The decline in the number of cross-bridge will lead to a decrease in muscle contraction. If the situation lasts longer, then the ability of muscle contraction will be reduced or even lost her ability to continue activities which will eventually lead to fatigue [12, 13]. Lactic acid levels in the blood can also be used as a parameter athlete's fitness level. Through Conconi test, anaerobic threshold of 4 mmol who dikolerasikan with a pulse can be used to determine the dose of aerobic exercise and anaerobic exercise [14].

Lactic acid production is very dependent on the intensity of physical activity. Production of lactic acid in people not trained with people who are trained, that is different is the process of elimination of lactic acid. In people who trained the elimination process is faster than in people who are not trained. In addition, people

who are trained to increase anaerobic threshold is correlated with the pulse, the pulse rate is higher and less pain than people who are not trained [11].

Elimination of lactic acid in the organs of the body in a way, kidneys and heart. Heart and kidneys can use lactic acid as an energy source. Hearts can use lactic acid as a raw material for the formation of glucose in gluconeogenesis. For the elimination of lactic apart by the ability of the body's organs can also be done with Massase. Massase increases blood flow so that areas that experienced an increase in lactic acid would be transported to another place in the body that have enough oxygen so that the elimination of lactic acid can be improved [11].

To get an accurate lactic acid levels, blood sampling performed 10 minutes after stopping exercise. This is because 10 minutes after activity nearly all the lactic acid that is formed diffuses from the blood circulation to the muscles or in other words the peak levels of lactic acid in the blood 10-12 minutes after activity [15, 16].
Research conducted to determine the physiological responses during exercise performed to exhaustion in exercise intensity corresponding to the maximal lactate steady state (MLSS). Subject of the study consisted of 11 men trained, MLSS tests performed on cycle ergometer until exhaustion. The results showed that the lactate concentration arteries and arterial carbon dioxide tension ($PaCO_2$) decreased significantly while the pH and base excess increased significantly ($p < 0.05$) [17].

Research to analyze changes in blood lactate concentration at the Polish elite runners. The study sample consisted of 9 runners male and 8 female runners, a finalist of the Polish national championships. Measurements were taken before the 100 m and at minute 1, 5th and 10th after a run of 100 m. The results showed that the average concentration of blood lactate before running 100m in male runner was 1.40 ± 0.24 and 1.23 ± 0.24 mmol/L in women runners. Blood lactate concentration after a run of 100 m in the first minute of recovery rose to 9.38 ± 2.18 mmol / L for male runners and 2:48 ± 6.73 mmol / L for women runners, the 5th minute was 11.01 ± 2:14 mmol / L for women runners and 10:11 ± 0.99 mmol / L for male runners, minute-10 reached 9.51 ± 1.23 mmol / L for male runners and 9.09 ± 1.46 mmol / L for female runners. The conclusion showed that the pattern of the recovery process is different for male runners and female runners. Blood lactate concentration should not be measured immediately after the activity or after the 10th minute. Changes in blood lactate concentration were observed in Poland after the elite runners run 100 m provides new information on the dynamics of the recovery which could be useful in designing training sprint runner [16].

CONCLUSIONS

Sprinter performance is affected by muscle fatigue is believed to be associated with the production of lactate. There are many conflicting views on the metabolism and the effects of lactate. This needs further study to provide conclusive results that would describe the controversy lactate. It is interesting that, during exercise lactate can be fuel for the brain, heart and skeletal muscle. Thus the increase in lactate concentration can be seen as an advantage the athlete. However, lactate relationship with muscle fatigue due to accumulation of lactate is a big loss. Energy system determines the quality of performance of the athlete. A clearer understanding of the interaction of the energy system will be very helpful in optimizing the performance of athletes. The accumulation of lactate in the muscles can be used as an indicator of fitness levels of athletes.

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