Effect of Eccentric and Concentric Exercise on Plasma Nitric Compounds in Healthy Men

by

Stanislaw Poprzecki¹, Adam Zajac², Adam Staszkiewicz³, Jaroslaw Cholewa², Teresa Zwierko⁴

During all physical activities, including those of competitive sport different forms of muscular contractions are encountered. In some exercises concentric contractions dominate while in others there is a predominance of eccentric work. During eccentric contractions, mechanical changes in the sarcomers and sarcolemma, cause significant damage to the entire muscle cell. This phenomenon is manifested by the outflow of cell proteins, including enzymes and other metabolites. The consequences include muscle soreness, local tenderness, decreased range of motion and decreased strength.

The main objective of this work was the observation of exercise, post exercise and recovery plasma changes of uric acid, urea and ammonia in men subjected to a progressive endurance exercise protocol performed with the predominance of concentric and eccentric muscular work.

The conducted research allows to state that plasma concentration of uric acid and urea changed linearly to the increased work intensity in both concentric and eccentric exercise protocols, yet this was not true for ammonia and lactate concentration which increased exponentially. The greatest changes in plasma concentration of uric acid and urea occurred during the 24 hour recovery period, especially after eccentric muscle contractions. This confirms significantly greater muscle disruption following eccentric work.

It can be concluded that the evaluation of plasma urea and uric acid concentration during exercise and recovery is not agood marker of exercise intensity, while plasma ammonia dynamics may serve this purpose well.

Key words: eccentric and concentric exercises, nitric compounds

¹ - Department of Biochemistry, Academy of Physical Education, 40-065 Katowice, 72 Mikolowska str., Poland

² - Department of Sport Training

³ - Department of Physiology

⁴ - University of Szczecin

Introduction

Exercise induced muscle damage particularly that which follows strenuous exercise that contains intensive eccentric muscle contractions is well documented (Michaut et al. 2004, Byrne et al.2004, Balnave et all. 1993). Eccentric muscle contractions are common during many daily activities such as walking, jumping and running. Symptoms associated with muscle damage include soreness, changes in range of motion, loss and release of muscle metabolites. Changes in these symptoms are often evaluated as indirect markers of muscle damage (Balnave 1993).

Active skeletal muscles are a source of ammonia. Its synthesis occurs at the level of the nucleotide purine cycle. Ammonia is produced by deamination of AMP (adenosine-monophosphate) to IMP (inosine 5' –monophosphate) under the action of AMP-deaminase, which occurs during rapid resynthesis of an ATP molecule from two ADP molecules, areaction catalyzed by adenylate kinase (Terjung 1996, Baldwin 1999, Sahlin 1996). Ammonia is also produced in biochemical reactions catalyzed by glutamate dehydrogenase (EC 1.4.1.2), adenosine deaminase (EC 3.5.4.4), glutaminase (EC 3.5.1.2) and glutamylotransferase (EC 2.3.2.2). NH₃, AMP and ATP are important regulators of cell metabolism, especially glycolysis (Spodaryk 1989) and ventilation (Vanuxem et al. 1998). Plasma exercise dynamics of ammonia are highly correlated with the rate of glycolysis and lactate concentration (Graham et al. 1993).

Urea is the final byproduct of nitrogen excretion in the body. Over 95 % of this compound is created through deamination of amino acids, while less than 5% is derived from a different urea and ammonia molecule in the presence of the enzyme urease (Carraro et al. 1993). During short intensive physical effort blood levels of urea are stable. Its concentration rises significantly after several hours of recovery or during prolonged endurance exercise. This is most likely caused by post exercise catabolic processes in the organism (Haralambie et al. 1996, Hubner-Wozniak et al. 1986, Lemon et al. 1989).

Another compound that contains nitrogen includes uric acid. It is the end product of IMP breakdown derived through the deamination of AMP. An increase in the plasma concentration of this metabolite occurs following prolonged exercise (Jensen et al. 1989). The plasma level of uric acid may be an indirect measurement of ATP metabolism. On the other hand it can reflect the rate of free oxygen radical generation with the presence of hypoxanthine and the enzyme oxydase hypoxanthine (Sjodin et all. 1990). It is a well known fact that the deposition of insoluble uric acid salts in the body is highly undesired and may lead to serious health consequences such as: gout, arthritis, kidney stones ect. (Montoye et al. 1976, Moser 1980).

During all physical activities, including those of competitive, sport different forms of muscular contractions are encountered. In some exercises concentric contractions dominate while in others there is apredominance of eccentric work. During eccentric contractions, mechanical changes in the sarcomers and sarcolema, cause significant damage to the entire muscle cell. This phenomenon is manifested by the outflow of cell proteins, including enzymes and other metabolites. The consequences include muscle soreness, local tenderness, decreased range of motions and decreased strength.

The main objective of this work was the observation of exercise, post exercise and recovery plasma changes of uric acid, urea and ammonia in men subjected to aprogressive endurance exercise protocol performed with the predominance of concentric and eccentric muscular work.

Material and methods

Subjects

Ten non-athletes, students of physical education, aged 22,4±1,4 years, with a body mass of 75,5±1,5 kg and body height 177,0±5,7 cm, volunteered to take part in the study. All subjects were informed of the purpose and the nature of the study before giving their written consent to participate in the experiment, which had been approved by the Ethics Committee at the Medical University of Silesia in Katowice.

Exercise protocol

Students performed two different kinds of physical effort with graded intensity until volitional exhaustion. Tests were performed on atreadmill (Jaeger) with a one week period between sessions. The first test – concentric work of muscles (Conc) was based on uphill running (15° angle) with the initial speed of 5 km/h. The speed was increased every 3 min by 1 km/h. In the second test – eccentric work (Ecc), the subjects ran downhill (-15° angle) with the initial speed of 9 km/h. The speed was increased every 3 min by 3 km/h. Intervals lasting 1 min between successive loads were adapted in both tests. Oxygen uptake was monitored during exercise using agas analyzer (Beckman, Comp.) and expressed in ml/min/kg.

Biochemical analysis

Blood samples were taken from the antecubital vein to heparynized testtubes before and during every test, during intervals between successive loads and after 2, 7 and 24 hours of recovery. Urea (U) and uric acid (UA) concentration was evaluated in plasma, without haemolysis in a temperature of 37°C using the spectrophotometric method (Shimadzu UV-VIS 1200) with Analco kits (Poland), while plasma ammonia (AMM) and lactic acid (LA) concentration using the Boehringer Mannheim kits (Germany). Urea, uric acid, ammonia and lactic acid concentration corresponding to increases of 5 ml/min/kg values of oxygen uptake, were read from the above variables of the co-dependence graph.

Statistics

Average values (X) and standard deviations (SD) were calculated. The Student-T test for dependent and independent variables was used for the examination of significance of differences and regression analysis. Differences were accepted as significant at the level of p<0.05. The data was analyzed using Statistica 5.0 computer program. (StatSoft, Inc 1997).

Results

The concentric exercise protocol caused no significant changes in the concentration of urea (U). During the eccentric muscular work significant changes (p<0.05) in the level of this metabolite occurred at intensities corresponding to oxygen uptake of 30-40 ml/min/kg. At higher exercise intensities the concentration of U decreased to initial level. During the recovery period a tendency for increased concentration of Uoccurred, yet it was not statistically significant. There were no significant differences in the concentration of this metabolite during recovery between the two types of exercise protocols (fig. 1).

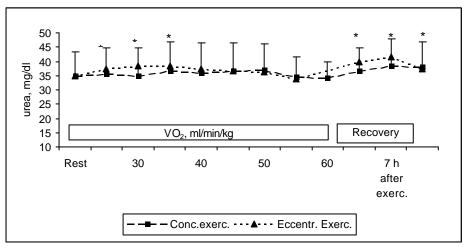


Fig. 1. Exercise and recovery plasma urea concentration during concentric and eccentric work. * different p<0,05 from rest.

The concentration of uric acid (UA) did not change during the concentric exercise. A significant increase in the concentration of UA (p<0,05) occurred during the eccentric exercise protocol at intensities equal to 25, 30, 40, 45 and 55 ml/min/kg. During the 24h recovery period a significant increase in the plasma concentration of this metabolite was registered after eccentric work (p<0,05), while marginal changes were observed during the same time period following concentric work. After 24h of recovery the plasma concentration of UA continued to increase following the eccentric exercise protocol and was significantly (p<0,05) greater than that observed after concentric work (fig. 2).

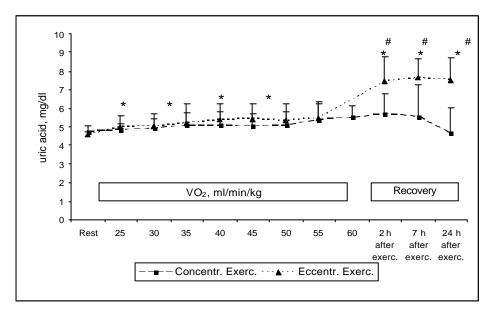


Fig. 2. Plasma uric acid concentration during concentric and eccentric work of muscles and at recovery. * different p<0,05 from rest, # different from concentric work of muscles.

The plasma concentration of ammonia and lactate increased significantly (p<0,05) in both exercise protocols along with the increase of exercise intensity. Beginning at exercise intensity corresponding to oxygen uptake of 45 ml/min/kg the LA concentration registered during concentric exercise differed significantly (p<0,05) from the values observed during eccentric work (fig. 3, 4, 5). Higher exercise values of ammonia in relation to lactate concentration were registered during concentric exercise while the opposite was true during eccentric muscular contractions (fig. 4, 5).

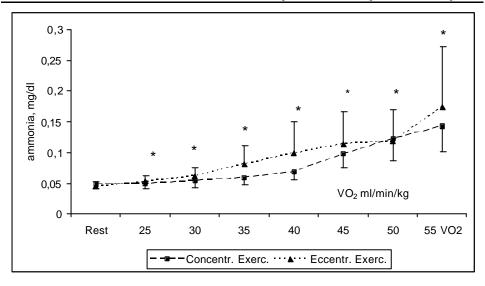


Fig. 3. Plasma ammonia concentration during concentric and eccentric work of muscles. * different (p<0,05) from rest.

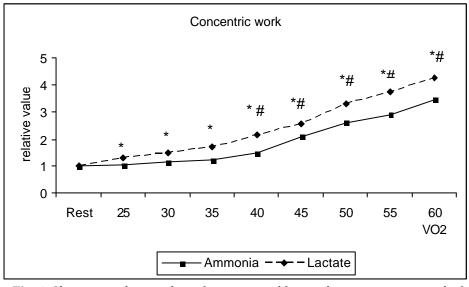
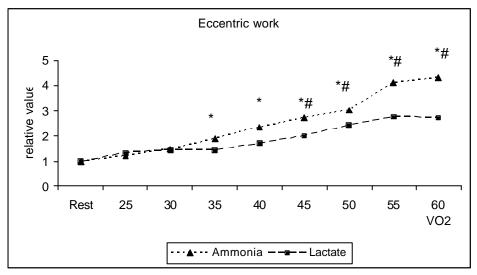


Fig. 4. Changes in relative values of ammonia and lactate during concentric work of muscles. * different (p<0,05) from rest, # different (p<0,05) between concentric and eccentric work of muscles.



*Fig. 5. Changes in relative values of ammonia and lactate during eccentric work of muscles. * different (p<0,05) from rest, # different (p<0,05) between concentric and eccentric work of muscles.*

The regression analysis of relative LA and ammonia values indicate a high relationship r=0.68 (p<0.001) in case of concentric exercise as well as in the eccentric one r=0.74 (p<0.001).

Discussion

It is a well known fact that intensive exercise, especially with a predominance of eccentric muscle contractions causes muscular damage. The most obvious symptoms include soreness, local tenderness, restricted range of movement, decreased strength and an increased outflow of cell met abolites into the blood (Nosaka et al. 1995, 1997, 2002). These changes are often referred to as indirect markers of muscle damage. Research indicates that repeated exercise significantly diminishes muscle disruption do to adaptation (Nosaka et al. 1995).

The adaptation process seems complex, and takes place at the cellular, tissue and organelle level (Chen et al. 2001). Muscle degradation can also be a consequence of decreased homeostasis of Ca⁺⁺ ions which activate many proteolytic and lipolytic enzymes as well as a decreased ATP concentration during eccentric exercise (Nosaka e al. 1995).

Another phenomenon that accompanies exercise includes blood distribution in particular organs. It is mainly directed to the working muscles, thus organs such as kidneys may have a limited supply of blood during exercise. The distribution of water in the body is also significant during exercise since it plays a vital role in thermoregulation (Balnave et al. 1993).

During this research project the tested subjects performed a graded endurance exercise protocol until volitional exhaustion with the predominance of concentric and eccentric muscular work. The stress placed on the organism was significant. The exercise protocol caused changes in blood and water distribution of the body what influenced the concentration of metabolites per unit of blood. From the considered metabolites containing nitrogen (urea, uric acid and ammonia) the highest exercise induced increase in plasma concentration occurred in case of ammonia while the lowest in urea and uric acid. These compounds are connected with protein and ATP metabolism, which during exercise are subjected to catabolic processes (Baldwin 1999, Carraro et al. 1993, Plante et all. 1984). The rather stable plasma concentration of urea during concentric and eccentric exercise may be explained by a decreased kidney filtration and a simultaneous liberation with sweat and in the lungs as well as a decreased supply of water (Wolfe et al. 1984). It seems that the exercise induced changes in blood urea concentration are not related to increased production but most likely with distribution of blood in the body (Carraro et al. 1993). On the other hand the exercise uptake of BCAA may slightly increase the production of urea (Harper et al. 1984).

The post exercise increase in plasma ammonia and lactate concentration registered in this work was a consequence of hypoxia. These two variables were significantly correlated, what has been confirmed by Casasa et al. (2001), Ogino et al. (2000), Ringa et al. (1999).

The intensified catabolism of purines during exercise, when the rate of ATP degradation exceeded its synthesis was the main cause of increased plasma uric acid concentration. Similar observations were performed by Sjodin (1990).

The significant rise in plasma concentration of uric acid and urea during post exercise recovery, especially after eccentric work may be related to the well documented muscle damage and the increased outflow of metabolites outside the cell (Klapcinska et al. 2001).

The conducted research allows to state that plasma concentration of uric acid and urea changed linearly to the increased work intensity in both concentric and eccentric exercise protocols, yet this was not true for ammonia and lactate concentration which increased exponentially. The greatest changes in plasma concentration of uric acid and urea occurred during the 24 hour recovery period, especially after eccentric muscle contractions. This confirms significantly greater muscle disruption following eccentric work. It can be concluded that the evaluation of plasma urea and uric acid concentration during exercise and recovery is not a good marker of exercise intensity, while plasma ammonia dynamics may serve this purpose well.

References

- Baldwin J., Snow R.J., Carey M.F., Febbraio M.A. 1999. Muscle IMP accumulation during fatiguing submaximal exercise in endurance trained and untrained men. Am. J. Physiol. Regul. Integr. Comp. Physiol. 277(1):R295-R300.
- Balnave C.D., Thompson M.W. 1993 Effect of training on eccentric exercise induced muscle damage. J. Appl. Physiol. 75(4):1545-1551.
- Byrne C., Eston R.G., Edwards R.H.T. 2004. Characteristics if isometric and dynamic strength loss following eccentric exercise-induced muscle damage. Scand. J. Med Sci. Sports 11(3):134.
- Carraro F., Kimbfough T.D., Wolfe R.R. 1993. Urea kinetics in human at two levels of exercise intensity. J. Appl. Physiol. 75(3):1180-1185.
- Casas H., Murtra B., Casas M., Ibnez J. et al. Increased blood ammonia in hypoxia during exercise in men. J. Physiol. Biochem. 57(4):303-312.
- Chen T.C., Hsieh S.S. 2001. Effects of 7-day eccentric training period on muscle damage and inflammation. Med. Sci. Sports Exerc. 33:1732-1738.
- Graham T.E., Rush J.W.E., MacLean D.A. 1995. Skeletal muscle amino acid me tabolism and ammonia production during exercise. In Exercise metabolism, M. Hargreaves (ed), Human Kinetics pp. 131-175.
- Hübner-Wozniak E., Najmar E., Górski J., Gasiewska W. 1986. Zmiany stezenia mocznika w osoczu w cyklu treningowym zapasników. Sport Wycz., 7(259):33-38. (in Polish).
- Haralambie G., Berg A. 1976. Serum urea and amino nitrogen changes with exercise duration. Eur. J. Appl. Physiol. 36:39-48.
- Harper A.E., Miller R.H., Block K.P. 1984. Branched chain amino acid metabolism. Ann. Rev. Nutr. 4:409-454.
- Jenssen G.M., Degenaar C.P., Menheere P.P., Habets H.M., Geurten P. 1989. Plasma urea, creatinine, uric acid, albumin, and total protein concentrations before and after 15-, 25-, and 42 km contests. Int. J. Sports Med. 10(Suppl. 3.):132-138.
- Klapcinska B., Iskra J., Poprzecki S., Grzesiok K. 2001. The effects of sprint (300 m) running on plasma lactate, uric acid, creatine kinase and lactate dehydrogenase in competitive hurdlers and untrained men. J. Sport Med. Phys. Fitness 41(3):306.
- Lemon P.W., Deutsch D.T., Payne W.R. 1989. Urea production during prolonged swimming. J. Sports Med. 7:241-246.
- Michaut A., Babult N., Pousson M. 2004. Specific effects of eccentric training on muscular fatigability. Int. J. Sports Med. 25(4):278-283.

- Montoye H.J., Mikkelsen W.M., Metzner H.L., Keller J.B. 1976. Physical activity, fitness, and serum uric acid. J. Sport. Med. Phys. Fitness 16:253-260.
- Moser B. 1980. Zur Harnsäure bei Sportaktiven. Dt. Z. Sportmed. 31: 237-244.

40

- Nosaka K., Clarkson P.M. 1995. Muscle da mage following repeated bouts of high force eccentric exercise. Med. Sci. Sports Exerc. 27:1263-1269.
- Nosaka K., Clarkson P.M. 1997. Influence of previous concentric exercise on eccentric exercise-induced muscle damage. J. Sport Sci. 15:477-483.
- Nosaka K., Newton M. 2002. Concentric or eccentric training effect on eccentric exerciseinduced muscle damage. Med. Sci. Sports Exerc. 34(1):63-69.
- Ogino K., Kinugawa T., Osaki S., Kato M. et al. 2000. Ammonia response to constant exercise: differences to the lacta te response. Clin. Exp. Pharmacol. Physiol. 27(8):612-617.
- Plante P.D., Houston M.E 1984. Effects of concentric and eccentric exercise on protein catabolism in man. Int. J. Sports Med. 4: 174-178.
- Ring S., Mader A., Mougios V. 1999. Plasma ammonia to sprint swimming. Int. J. Sport Med. Phys. Fitness 39(2):128.
- Sahlin K., 1996. Ammonia metabolism I humans during exercise. In Biochemistry of Exercise, Ed. R.J. Maughan, S.M. Shirreffs, Human Kinetics pp. 497-510..
- Sjödin B., Hellsten W.Y. 1990. Changes in plasma concentration of hypoxantine and uric acid in man with short-distance running at various intensities. Int J. Sports Med. 11: 493-495.
- Spodaryk K. 1989. Rola amoniaku w rozwoju zmeczenia. Sport Wycz. 295(7-8):71-74. (in Polish).
- Terjung R.L. 1996. Ammonia metabolism in muscle. In Biochemistry of Exercise, Ed. R.J. Maughan, S.M. Shirreffs, Human Kinetics pp. 485-491.
- Vanuxem D., Delpierre S., Fauvelle E., Guillot C., Vanuxem P. 1998. Blood ammonia and ventilation at maximal exercise. Arch. Physiol. Biochem. 106(4):290-296.
- Wolfe R.R., Wolfe M.H., Nadel E.R., Shaw J.H.F. 1984. Isotopic determination of amino acid -urea interactions in exercise in human. J. Appl. Physiol. 56: 221-229.