Baltic Journal of Health and Physical Activity

Volume 5
Issue 4 *12/31/2013*

Article 1

2013

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Anna Szczesna-Kaczmarek
Gdansk University of Physical Education and Sport in Gdansk, Poland, annaszk1@tlen.pl

Recommended Citation

Szczesna-Kaczmarek A. Blood K+ concentration balance after prolonged submaximal exercise – The role of both uptake and excretion processes. Balt J Health Phys Act. 2013; 5(4): 233-242. doi: 10.2478/bjha-2013-0021

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BALTIC JOURNAL OF HEALTH AND PHYSICAL ACTIVITY
© Gdansk University of Physical Education and Sport in Gdansk, Vol. 5, No. 4, 2013, 233-242
ORIGINAL ARTICLE
DOI: 10.2478/bjha-2013-0021

Blood K^+ concentration balance after prolonged submaximal exercise – The role of both uptake and excretion processes

Anna Szczesna-Kaczmarek

Gdansk University of Physical Education and Sport in Gdansk, Poland

Authors' Contribution:
A – Study Design
B – Data Collection
C – Statistical Analysis
D – Data Interpretation
E – Manuscript Preparation
F – Literature Search
G – Funds Collection

Key words: *contracting muscle, K^+ loss, blood $[K^+]$ increase, prolonged exercise, K^+ renal excretion.*

Abstract

Background: *A contracting muscle is a source of the plasma K^+ concentration increase during physical exercise. The flux of K^+ from contracting skeletal muscle to blood is related to the frequency of cells action potential. The elevated blood $[K^+]$ may result in the heart rate irregularities and interferes with the way nerves send signals. But plasma increased $[K^+]$ recovers rapidly to normal if a regulating mechanism takes action. The aim of this study was to evaluate the participation of processes restoring the balance in blood $[K^+]$ after prolonged submaximal exercise.*

Material/Methods: *Nineteen healthy, young, physically active men performed the 120-min submaximal cycling (intensity below individual AT). Measurements were made of urine, plasma and hemolysed whole blood collected before and after a 2-h cycloergometric exercise and after 1h, 2h and also after 24h recovery to quantify the excretion of K^+ to urine and the relative contribution of plasma and erythrocytes to the place where K^+ is released in two compartments of blood.*

Results: *The main findings in the present study are that the balance of plasma $[K^+]$ after prolonged exercise is maintained not only by the reuptake of K^+ to a non-contracting and contracting muscle and by changes in $[K^+]$ in erythrocyte and plasma, but as well by the excretion of ions into urine.*

Conclusions: *The fate of K^+ released from a contracted muscle is connected not only with the exercise intensity and acidification but also with the duration of exercise. Athletes should keep in mind different action of kidneys in case of K^+ before supplementation of electrolytes after specific exercise.*

Word count: 2,819
Tables: 1
Figures: 9
References: 40

Received: February 2013
Accepted: November 2013
Published: December 2013

Corresponding author:
Prof. dr hab. Anna Szczęsna-Kaczmarek
Gdansk University of Physical Education and Sport, Dep. of Physiology
80-336 Gdańsk, Poland, ul. K. Górskiego 1
Phone: +4858 554-72-97
E-mail: annaszk1@tlen.pl

Introduction

Muscle cells contractions begin when the action potential triggers changes in membrane permeability for sodium and potassium ions, which then causes the release of calcium ions from the sarcoplasmic reticulum. Potassium ion is the major intracellular cation – about 95% of the total potassium ion is contained in the cytoplasm. Only 2% of K^+ is located in extracellular fluid. The intracellular and extracellular potassium concentration is regulated precisely, because many cell functions are sensitive to changes in the concentration of K^+ in extracellular fluid. For instance, an increase in ion potassium concentration in plasma of only 4 mmol / liter can cause cardiac arrhythmias. The high concentration of K^+ in intracellular fluid is necessary for the proper activity of many enzymes, enzymatic processes and for the excitability of muscles and nerves or other tissues.

During the skeletal muscle cells action potential, there is an intensification of the influx of sodium ions from extracellular fluid to the inside of cells, which causes cell membrane depolarization. As a result of the membrane depolarization, potassium ions from the cytoplasm flow outside. The diffusion of potassium ions outside causes the repolarization of cell membranes. The return of cell membrane potential to the recovery state requires the activity of the Na^+- K^+- pump to re-establish the sodium and potassium membrane concentration differences [1, 2]. Since this pump requires energy for operation and because the above described recurrent phenomenon lasts as long as the muscle is contracting, this process is energy absorbing. For this reason this phenomenon can be upsetting for muscle cells.

During prolonged exercise, potassium ions are released from the intracellular to the extracellular space of contracting muscle cells [3, 4]. Increased extracellular K^+ concentration can reduce the excitability of skeletal muscles [5, 6]. Prolonged or strong contraction of skeletal muscles leads to the state of muscle fatigue [7, 8, 9]. The general assumption is that an increased diffusion of potassium ions to the extracellular fluid is a very important fatigue factor. The insufficient activity of the Na^+-K^+ pump cannot restore the sodium and potassium ions free setting in muscle cells which is the cause of the above mentioned phenomenon. Increased potassium ions in extracellular fluid are next released from a muscle to blood [10, 11]. The rate of increase in ion potassium concentration in plasma is dependent on exercise intensity and its period. The peak plasma [K^+] was achieved within the first 6 min of exercise with increasing intensity [12]. However, our subjects exercised at a fixed intensity below the individual anaerobic thresholds (AT) but lasting as long as two hours. Peak plasma [K^+] returned to the standard concentration after about five minutes when exercise stopped. What is interesting is the mechanism of rapid decline in arterial plasma [K^+] during the first minutes of recovery. A number of research centres study this subject [11, 13, 14]. We have also conducted the examination of the fate of potassium ion released from contracting muscles during repeated supramaximal exercise [15]. The most important finding of our earlier study was that the rapid increase in [K^+] in plasma during repeated supramaximal exercise did not lead to the intensity of renal potassium ion excretion to urine.

The supramaximal exercise is known as a source of protons (H^+) involved in both intracellular and interstitial [K^+] regulation [12, 16, 17]. During less intense exercise, H^+ did not appear in muscles and blood in such a great quantity. Taking into consideration the differences in the effects of physical exercise at different intensity on the human body, the distribution of muscle – K^+ released to blood during and after prolonged exercise was the subject of the research in this paper.

Materials and methods

Participants

Nineteen healthy, young, physically active men – students of the Gdansk University of Physical Education and Sport (Poland) volunteered for the study. The mean (± SE) age, height and weight for the group were respectively: 22.5 ± 1.16 yr, 180.9 ± 5.8 cm and 83.4 ± 6.5 kg (Table 1). The Ethical Committee of Scientific Research at the Medical University of Gdansk approved the experimental protocol.

Tab. 1. Subjects characteristics (n = 19)

Variables	Mean ± SD
Age, yr	22.5 ± 1.16
Weight, kg	83.4 ± 6.5
Height, cm	180.9 ± 5.8
$\dot{V}O_{2\ max}$, $ml.kg^{-1}.min^{-1}$	53.52 ± 3.8
AT, $ml.kg^{-1}.min^{-1}$	33.43 ± 4.1
AT, % $\dot{V}O_{2\ max}$	63.15 ± 1.9

Exercise protocols

All experiments were conducted in the morning. Exercise was performed on a mechanical, Monark cycloergometer with automatic counting of pedal revolutions. Subjects performed the warm-up lasting 5min before the specific test, to promote specific physiologic and motor adaptation. After a 5-min recovery, subjects performed the two cycloergometric tests:

The first test – a maximal incremental test – the gradually increased intensity as far as the participant was able to continue in order to measure the maximal oxygen uptake ($\dot{V}O_{2\ max}$) and the individual anaerobic threshold (AT). The exercise test consisted of 5 minute steady-state part of ergometer cycling at 60 rpm, starting at 100 W/min and proper part of progressively increasing by 25 Watt increments. The exercise session was completed once the participant's pedal frequency dropped below 60 rpm. The highest $\dot{V}O_{2\ max}$ over a 30-s interval was defined as $\dot{V}O_{2\ max}$.

The second test – prolonged exercise. In the second visit, subjects performed submaximal cycling exercise at 85% of the individual anaerobic threshold (AT) (it was about 50% of $\dot{V}O_{2\ max}$) lasting 120 min, pedaling frequency 60 rpm. To insure adequate recovery, each test was separated by 24 h rest.

Measurements

During the first and the second test, important indices characterizing cardiorespiratory activity were measured.

Blood and urine sampling. Blood samples were collected in heparinized capillary tubes at rest and immediately after submaximal exercise as well as 5 min later. Blood samples were stored on ice for analysis of: K^+ concentration in plasma and the whole blood, acid–base balance parameters, haemoglobin concentration and haematocrit. Ion concentration in plasma and parameters of blood acid-base balance were measured immediately after blood collection using ion selective electrodes. Parts of the blood samples were frozen and defrosted three times so as to lyse erythrocytes cell membranes before determination of whole blood K^+ concentration. Ion concentration in blood lysate was measured using ion selective electrodes (Ciba-Corning 644 Na^+/K^+,Cl^- Analyzer). Parameters of the blood acid-base status were determined by using pH/oxygen Ciba-Corning Analyser 238. Haemoglobin was determined by cyanmethemoglobin spectrophotometry and haematocrit was measured after centrifugation.

Whole blood and plasma ion concentration and haematocrit were used to calculate ion concentration in erythrocytes. Urine was collected during 24h before the 2h submaximal exercise and at 1h, 2h and 24h intervals of recovery.

Urine volume was measured and urinary potassium ion concentration was determined using ion-selective electrode in Ciba- Corning 644 Na^+/K^+, Cl^- Analyzer.

Statistics. Statistical analyses were undertaken using STATISTICA (Stat Soft) version 8 software package. The normality of the data distribution was verified with Shapiro-Wilk test. Significant differences between values were tested by Student's t-test and by analysis of variance (ANOVA) with repeated measures across time and between conditions (changes in blood and urine) Significance was accepted at $p < 0.05$. All data are presented as Means ± SD.

Results

Results of this study are shown in Table 1 and Figures 1-9. The changes in potassium ion concentration in blood were considered as a two-compartment system (plasma and erythrocytes). Plasma K^+ concentration increased from 5.46 ± 0.40 mmol.L^{-1} at rest to 6.65 ± 0.90 mmol.L^{-1} after a bout of exercise (Fig. 1), but after 5 min of recovery it returned to normal.

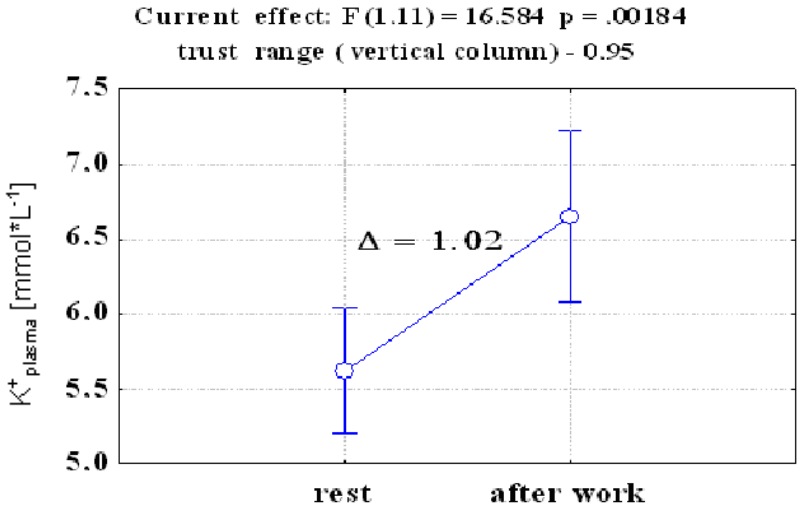

Fig. 1. Changes in plasma [K^+] after 2h submaximal exercise

The increment of plasma K^+ concentration was correlated both with blood [HCO_3^-] and its increment of blood Δ [HCO_3^-]. The levels of significant difference are: r = 0.7366 and r = 0.8274 at [HCO_3^-] and Δ [HCO_3^-], respectively.

Potassium ion concentration in whole blood increased significantly from 71.93 ± 6.49 mmol.L^{-1} at rest to 79.0 ± 5.17 mmol.L^{-1} after 2h submaximal exercise (intensity 50% of $\dot{V}O_{2\,max}$). Changes were statistically significant (p = 0.001; r = -0.6235) (Fig. 2). After 5 min of recovery, the whole blood K^+ concentration decreased almost to the rest values**.**

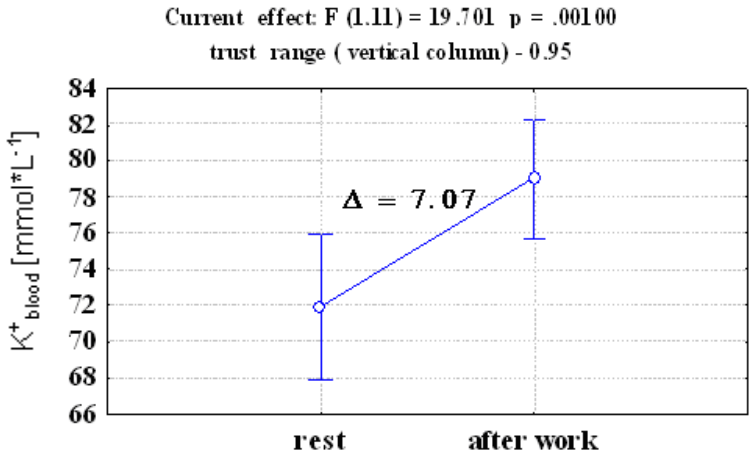

Fig. 2. Changes in blood [K^+] after 2h submaximal exercise

Potassium concentration in erythrocytes increased during exercise from 143.2 ± 11.56 mmol.L^{-1} to 157.2 ± 11.55 mmol.L^{-1}. Changes are statistically significant (p = 0.0004, r = 0.6529) (Fig. 3) and correlated with changes of [K^+] in blood at rest and after submaximal exercise (p< 0.05, r = 0.6691) (Fig. 4 and Fig. 5**).**

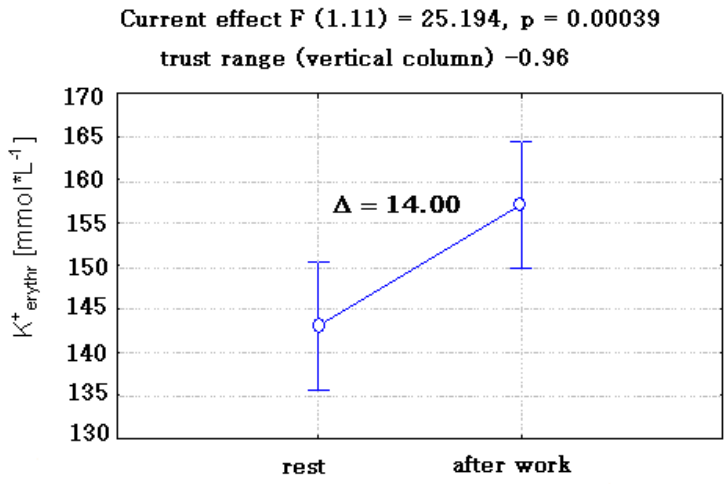

Fig. 3. Changes in erythrocyte [K^+] after 2h submaximal exercise

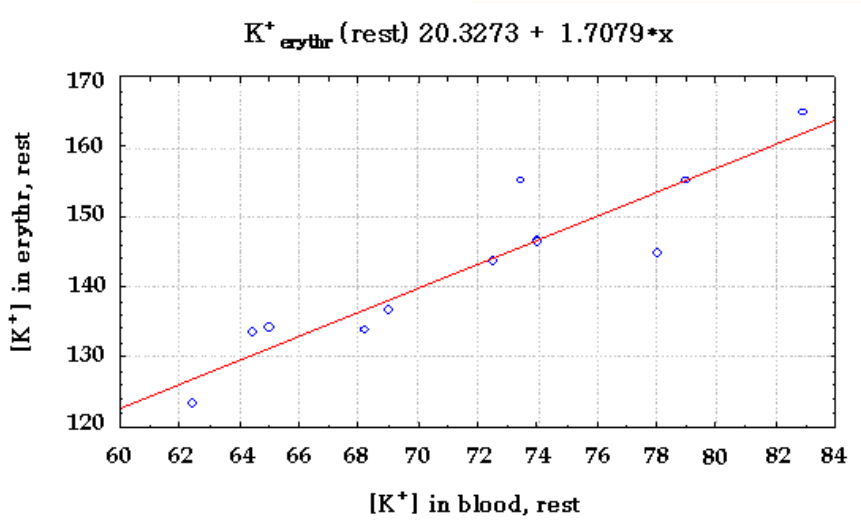

Fig. 4. Relationship between changes in blood [K^+] and changes in erythrocytes [K^+] at rest. $p < 0.05$; $r = 0.9247$.

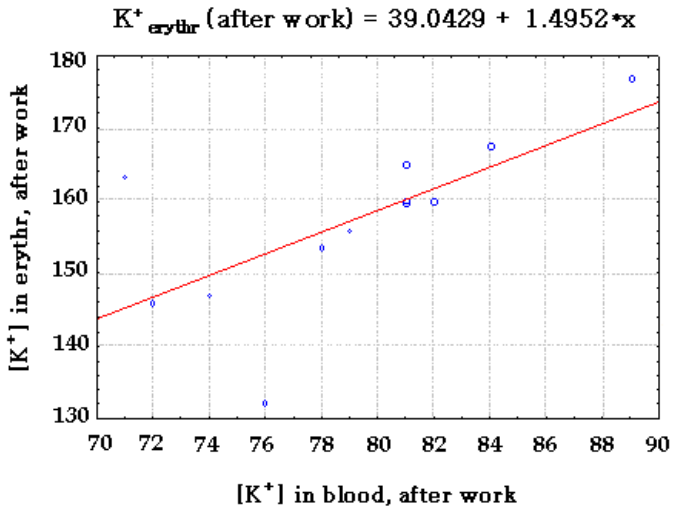

Fig. 5. Relationship between changes in blood [K^+] and changes of erythrocyte [K^+] after submaximal exercise, $p < 0.05$; $r = 0.6691$

Blood acid-base balance:

Submaximal exercise carried out for two hours caused only insignificant changes in acid-base balance parameters (Fig. 6). After the exercise period, blood pH slightly decreased from 7.39 to 7.36. Only a small range of changes in blood acid-base balance are caused by 2h submaximal exercise, assuming that the same conditions exist in contracting muscle cells. Furthermore, these results testify to the intensity of 2h exercise being below the anaerobic threshold.

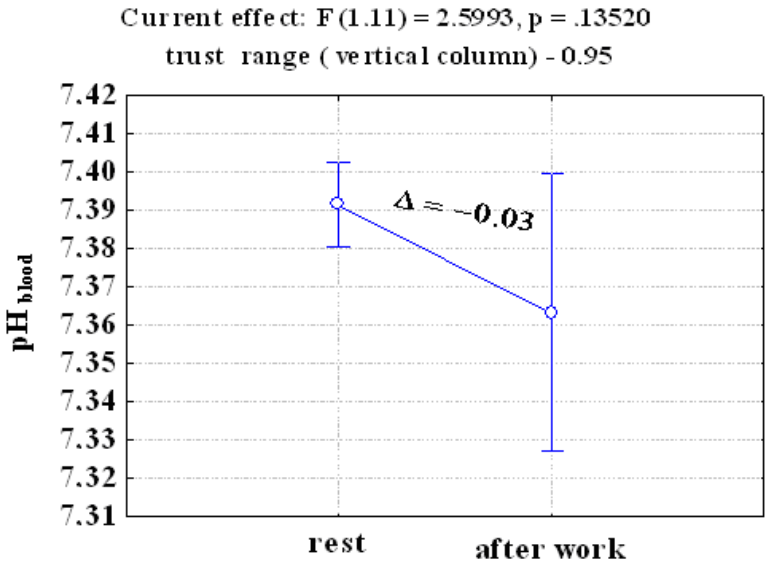

Fig.6. Changes in blood pH after 2h submaximal exercise

Potassium ion concentration in urine:

The concentration of urine potassium ion increased from 67.33 ± 16.79 mmol x L^{-1} to 96.75 ± 41.30 mmol x L^{-1} during 2h recovery from exercise (increment is statistically significant (r = 0.9180). The increase in urine [K^+] which took place during sustained work did not return to the pre-exercise concentration within the 24h of recovery (Fig. 7). Concentration of K^+ after 24h recovery ran to 77.00 ± 11.74 mmol x L^{-1} (Fig. 7) and the difference was still statistically significant (r = 0.7508). During the 2h and 24h of recovery after a dynamic exercise, an increment of [K^+] in urine did not show a significant correlation either with blood [HCO_3^-] or blood BE changes. However, we found a relationship between the changes in plasma [K^+] and the changes in [K^+] in urine during 2h and 24h recovery after submaximal exercise (Fig. 8).

Because of the importance of metabolic acidosis as a mechanism for the exercise blood hiperkalemia, we earlier performed research concerning the fate of K^+ released from contracting muscles during interval supramaximal exercise. Exercise of this kind is associated with a large efflux of muscle cell K^+ and lactic acid into the blood [15]. This earlier obtained result supplied us with important knowledge of the role of red blood cells and the renal system in exercise-released K^+ conservation after intermittent supramaximal exercise (after three bouts of Wingate 30s An Test).

We observed that the concentration of potassium ions in urine after intermittent supramaximal exercise was lower than at rest. On the basis of these results, we concluded that potassium ion concentration in the body fluid during supramaximal exercise and following recovery is conserved.

In the current study we observed, contrary to the above-mentioned, an increase in [K^+] in urine after 2h-lasting submaximal exercise i.e. we can notice a loss of potassium from blood to urine. The different excretion of blood K^+ to urine after both comparing exercise conditions, i.e. after submaximal and intermittent supramaximal exercise is presented in Figure 9.

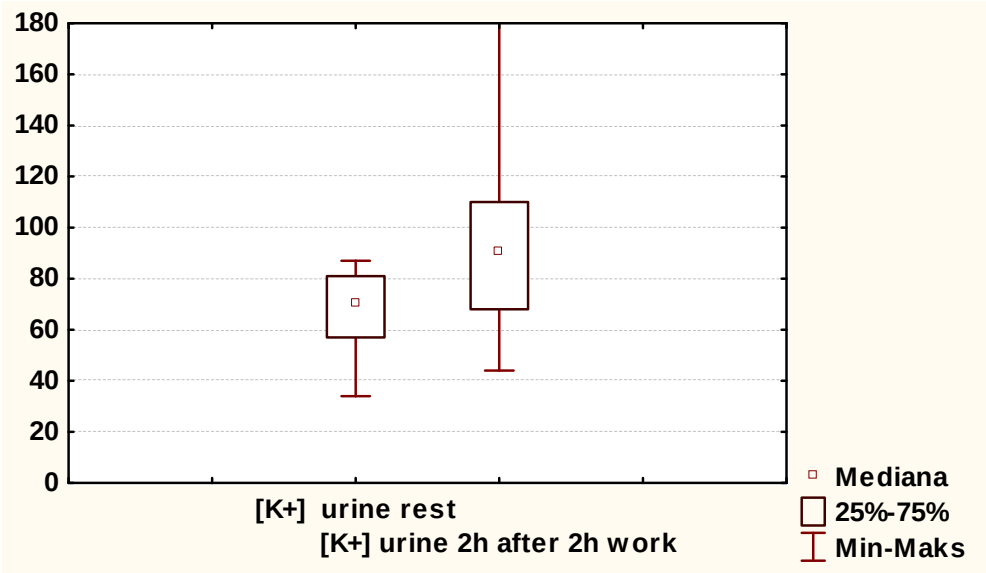

Fig. 7. Changes in urine K^+ concentration during 2h rest after submaximal exercise

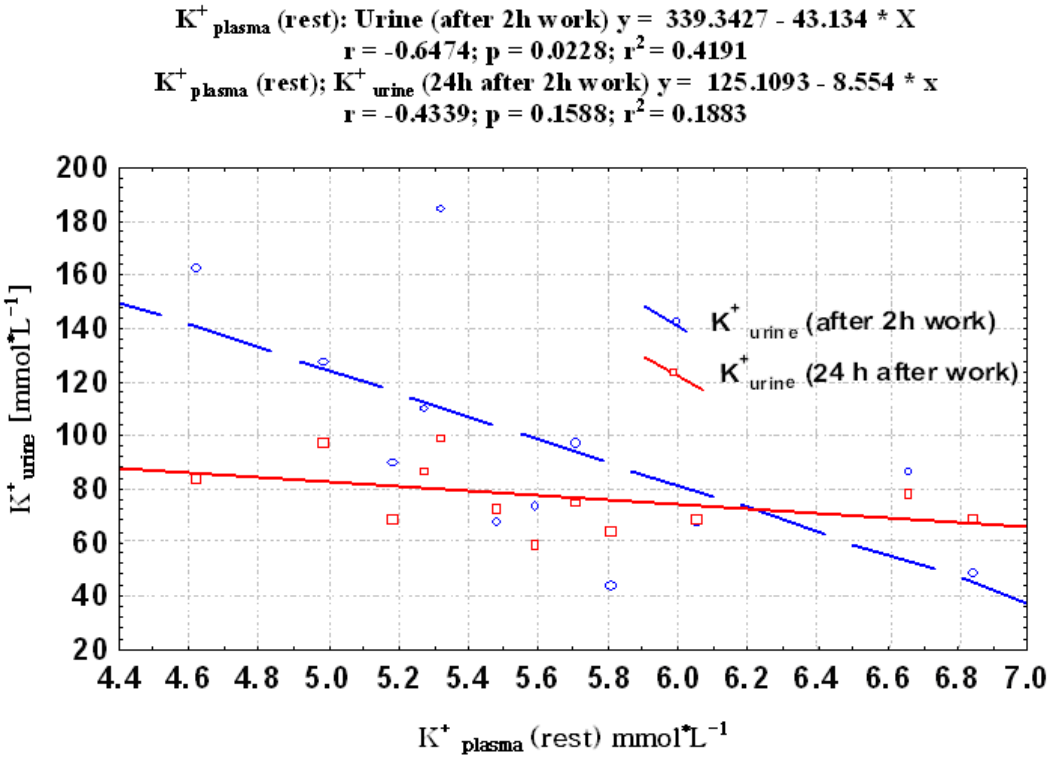

Fig. 8. Relationship between changes in plasma [K^+] and changes in urine [K^+] during 2h and 24h rest after submaximal exercise

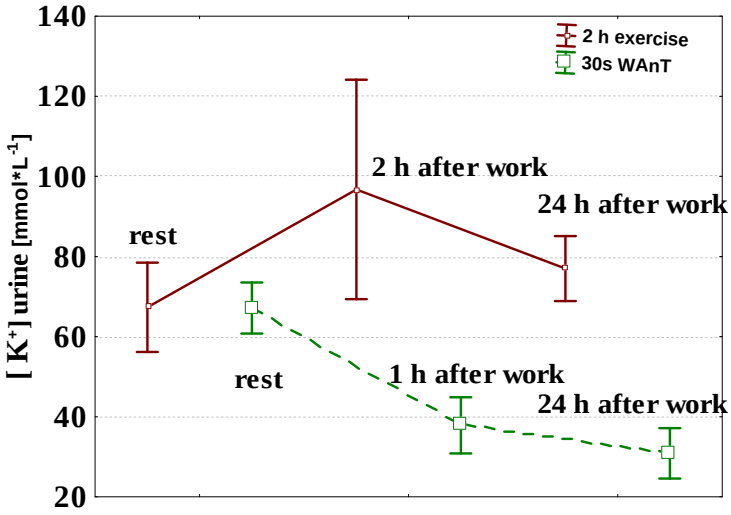

Fig. 9. Comparison of changes in [K^+] in urine during 2h and 24h rest after submaximal and supramaximal exercise (30s Wingate An Test) [15]

Discussion

The main findings in the present study are that the balance of blood K^+ concentration after 2h-submaximal exercise is maintained not only by the reuptake of K^+ to non-contracting and contracting muscle, but also by changes in [K^+] in erythrocyte and plasma as well as by the excretion of ions into urine. In the present research, the reuptake of K^+ released from muscles to the bloodstream by contracting and non-contracting muscles and also by inactive tissue was not measured. We drew on unquestionable results of many research centres [9, 14, 18, 19, 20]. We, like other researchers, have mainly based our work on the comparable results of the Norway group [11]. In their experiments authors applied continuous recordings of changes in plasma K^+ concentration during bicycle exercises of different intensity and duration. They made use of a K^+- sensitive microelectrode inserted into a femoral vein and artery of the subjects in their measurements. A lot of research centres have been interested in potassium fluxes in contracting skeletal muscles and plasma due to the regulating role of [K^+] in muscle cell function and also in the cardiovascular and respiratory function [21]. It is known that a significant increase in K^+ concentration in extracellular fluid in muscles and blood leads to a decrease in the contractile force and muscle fatigue [4, 22, 23, 24, 25, 26] and causes abnormalities in the behaviour of the heart [27, 28].

Up to now, mainly results of research concerning the effect of exercise on potassium balance in muscles and blood have been gathered. In this paper we show that the mechanisms that modulate the distribution of potassium between the extra- and intracellular compartment are rapid, within minutes. In this study, a short-lived increase in potassium ion concentration in plasma and distribution of these K^+ into erythrocyte and plasma was observed. Results of this research are consistent with other authors [29, 30, 31, 32].

The results of many authors have proved that K^+ released from contracting muscle to blood was returned to an active muscle and an inactive tissue within minutes. The reversal from release to K^+ uptake requires an increase in sarcolemmal Na^+ -K^+ pump activity. It is known that skeletal muscles and other tissues also possess other system mediating K^+ fluxes, such as Na^+- K^+- $2CL^-$ co-transport and NaCl co-transport [14], which aid the Na^+- K^+ pump action. As is known, strong stimulation of activity of the Na^+- K^+ pump is caused by stress hormones, i.e. epinephrine and norepinephrine and their concentration increase after exercise [2, 23, 25, 33]. In our research, we take into consideration the role of the renal regulation of potassium concentration in extracellular fluid. Extracellular fluid potassium concentration is regulated at about 4.2 ± 0.3 mmol/liter. The maintenance of balance of potassium concentration requires kidneys to adjust their excretion precisely to variations in the intake of these ions [34, 35, 36]. The mean part of ion potassium is secreted in the late distal tubules and cortical collecting tubules of the nephron. The cells in these regions are called principal cells. Secretion of potassium from blood into the tubular lumen is a two-

step process. In the first one - potassium ions are transported into the cell by the Na^+,K^+ - (ATP-ase) pump in the basolateral membrane of the cell; the second step of the process is passive diffusion of potassium from the cytoplasm of the cell by a special channel into the tubular fluid. The rate of potassium secretion is stimulated by increased concentration of potassium in extracellular fluid. The second step of potassium ion passive diffusion is stimulated by aldosterone [37]. It is known that the aldosterone secretion is stimulated by prolonged exercise [38, 39, 40]. Thus, increased plasma potassium concentration as a consequence of muscle contraction directly raises potassium secretion by the cortical collecting tubules and indirectly increases potassium secretion by increasing plasma aldosterone concentration. This mechanism explains the increase in potassium concentration in urine - as stated in our research - after two hours of exercise. It is known that one factor that decreases potassium secretion is increased hydrogen ion concentration [H^+], i.e. acidosis [16, 17]. Increased hydrogen ion concentration was affirmed after interval supramaximal exercise but did not appear after 2h of submaximal exercise. Under these circumstances, it is clear why in our experiments potassium ions were lost from blood to urine after prolonged submaximal exercise and were conserved after supramaximal exercise. Differences in blood K^+ turnover during and after muscle exercise may result from exercise intensity differences in the blood aldosterone increase to exercise and/or the H^+ levels during exercise.

Conclusion

In conclusion, it is worth emphasizing the significance of the above mentioned opposite results for the practice. It is common knowledge that fluid and electrolytes are supplemented after exercise by athletes in order to restore to plasma and muscle hydration status. Unfortunately, the consumption of fluids after exercise does not always take into consideration the intensity and duration of the exercise.

Athletes should supplement ion potassium in the body through a suitable diet after prolonged exercise and after supramaximal exercise in varied amounts. Because postexercise plasma K^+ fate is connected with the intensity and duration of exercise, varying strategies of rehydration connected with electrolyte supplementation can be used to enhance fatigue removal.

Acknowledgments

The author wishes to express gratitude to dr Marcin Łuszczyk for his statistical analysis. Subjects' participation in this study is also acknowledged.

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