Serum, urinary and faecal magnesium changes of endurance trained athletes during periodic and continuous hypokinesia

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Zusammenfassung

Es wird angenommen, daß periodische Hypokinesie (PHK) stärkere Elektrolytänderungen hervorruft als kontinuierliche Hypokinesie (CHK). Deshalb war das Ziel dieser Studie Magnesium (Mg)-Veränderungen zu messen bei Ausdauersportlern während verlängerter PHK und CHK.

Die Untersuchungen wurden durchgeführt während einer 30tägigen Vor-Hypokinesie (HK)-Periode und während 364 Tagen einer PHK und CHK. 30 Sportler im Alter zwischen 20 und 26 Jahren wurden als Probanden ausgewählt. Sie wurden in drei Gruppen unterteilt: nicht eingeschränkte ambulante Kontrollprobanden (UACS), kontinuierliche Hypokinese-Probanden (CHKS) und periodische Hypokinese-Probanden (PHKS). Die CHKS-Gruppe mußte durchschnittlich 4,7 km/Tag während 364 Tagen laufen; während die PHKS-Gruppe durchschnittlich eine Entfernung von 4,7 und 11,7 km/Tag an 5 resp. 2 Tagen/Woche während 364 Tagen laufen mußte. Die UACS-Gruppe wurde kontinuierlich mit einer Laufdistanz von 11,7 km/Tag gehalten. D. h. sie führten keine Veränderungen in ihrem professionellen Training und ihren Routine-Tagesaktivitäten durch.

Während der HK-Periode und während den CHK- und PHK-Perioden wurden die fäkale und Urin-Magnesiumausscheidung und die Serum-Magnesiumkonzentration gemessen. In der CHKS- und PHKS-Gruppe stiegen die Urin- und fäkale Magnesium-Ausscheidung und die Serum-Magnesiumkonzentration signifikant ($p \leq 0{,}01$) an im Vergleich zur UACS-Gruppe. Die gemessenen Parameter waren größer und traten schneller auf in der PHKS-Gruppe als in der CHKS-Gruppe. Das fäkale Urin- und Serummagnesium änderte sich nicht signifikant in der UACS-Gruppe, wenn man sie mit den Ausgangskontrollwerten vergleicht.

Daraus wird geschlossen, daß sowohl PHK als auch CHK signifikante fäkale und Urin-Magnesiumausscheidung induzieren und das Serummagnesium ansteigen lassen; jedoch waren die gemessenen Parameter größer und traten schneller auf in der PHKS-Gruppe als in der CHKS-Gruppe.

Summary

It was assumed that periodic hypokinesia (PHK) induces much greater electrolyte changes than continuous hypokinesia (CHK). Thus, the aim of this study was to measure magnesium (Mg) changes in endurance trained athletes during prolonged PHK and CHK.

Studies were done during 30 days pre hypokinesia (HK) period and during 346 days of PHK and CHK periods. Thirty athletes aged 20 to 26 years were chosen as subjects. They were equally divided into three groups: unrestricted ambulatory control subjects (UACS), continuously hypokinetic subjects (CHKS) and periodically hypokinetic subjects (PHKS). The CHKS group was maintained under an average running distance of 4.7 km.- day-1 for 364 days; while the PHKS group was maintained under an average running distance of 4.7 and 11.7 km.day-1 for five days and two days per week, respectively, for 364 days. The UACS group was maintained continuously under an average running distance of 11.7 km.day-1, that is, they experienced no changes in their professional training and routine daily activities.

During the pre HK period and during the CHK and PHK periods faecal and urinary Mg excretion and serum Mg concentration were measured. In the CHKS and PHKS groups urinary and faecal Mg excretion and serum Mg concentration increased significantly ($p \leq 0.01$) when compared with the UACS group. The measured parameters were much greater and appeared much faster in the PHKS group than in the CHKS group. Faecal, urinary and serum Mg did not change significantly in the UACS group when compared with the baseline control values.

It was concluded that both PHK and CHK induce significant faecal and urinary Mg excretion and serum Mg concentration increases; however, the measured parameters were much greater and appeared much faster in the PHKS group than in the CHKS group.

Introduction

As it is known, the muscular system, that constitues an average of up to 40 percent of body mass, hold an important place in the optimum function of different orangs and system of the human organism. Thus, any functional reduction or alteration of the muscular system may affect adversely the adaptive mechanisms of the human organism, functional condition of different organs and systems and electrolyte metabolism [1–6].

Studies into the role of prolonged HK on electrolyte metabolism suggest that restriction of muscular activity may be the interfering mechanism by which electrolyte metabolism is significantly affected [1–6]. Available data also suggest that exposure to prolonged HK intensifies significantly the elimination of electrolytes in urine and faeces resulting in a significant reduction in total electrolyte concentration of the body and electrolyte deficiency [1–6]. Electrolyte deficiency, including Mg deficiency, during prolonged HK is characterized by increased and not by decreased serum of plasma electrolyte concentration; while all signs, symptoms and complications referable to electrolyte deficiency are not manifested during HK but during post HK, that is, during resumption of muscular activity [1–6]. This reaction may suggest that there could be present another mechanism that is affecting the process of control and regulation of electrolyte metabolism during prolonged HK. On the other hand, Mg deficiency in clinical conditions is characterized by decreased serum or plasma Mg concentration and may develop due to three main reasons:

excessive urinary losses (e.g., diuretic therapy, diabetic ketoacidosis), decreased intestinal absorption (e.g., severe diarrhea, small bowel resection), and decreased dietary intakes (e.g., prolonged parenteral nutrition).
Meanwhile, evidence is emerging to suggest that prolonged exposure to periodic hypokinesia (PHK) induces much greater and much faster electrolyte losses in urine and faeces than continuous hypokinesia (CHK). This reaction may lead to a much greater electrolyte deficiency during PHK than CHK. Available data suggest that the type of restriction of muscular activity, that is, PHK versus CHK, may play a part in the differences between the PHKS and CHKS groups regarding the intensity and rapidity of electrolyte changes in serum, urine and faeces.
By utilizing the newly available information regarding the metabolism of electrolytes during prolonged PHK and CHK, we hypothesized that Mg metabolism may also be affected much more during prolonged PHK than CHK. Thus, the objective of this investigation was (a) to measure serum Mg concentration, urinary and faecal Mg excretion in endurance trained athletes during prolonged PHK and CHK; and (b) to establish whether some differences regarding the intensity and rapidity of Mg changes are present during PHK and CHK.
The selection of endurance trained athletes for this study was based on the following two reasons: increased muscular activity is associated with the need for a significant activation of anabolic processes, mobilization of fluid, electrolytes, lipids, carbohydrates, proteins and other elements and intensification of the functional conditions of different organs and systems of the body; this however, is the exact opposite of the need for a significant inhibition of anabolic processes and prevalence of catabolic processes that is inherent to decreased muscular activity. Any change in these conditions acts as a hypokinetic stress, and the greater the differences between these two conditions the stronger it is the hypokinetic effect on electrolyte metabolism.

Materials and methods

Subject selection

Thirty athletes ranging in age from 20 to 26 years gave informed consent to take part in the study after a verbal and written explanation of the procedures and risks involved was given. Procedures were previously review and approved by the Committee for the Protection of Human Subjects. All athletes had been trained as long distance runners for the last three to five years, at an average of 11.7 km.day-1 and had a speed of 10.3 km.h-1. Physical characteristics of the subjects are given in the tab. 1.

Experimental design

Subjects were on a metabolic diet 30 days before the start of the study and during the study. Portions of food provided were weighed and uneaten portions of the measured intake of food were also weighed. The adjusted diet was then maintained for the remainder of the study and was controlled for Mg intakes with food. Dietary composition of selected nutrients is presented in tab. 2.

Tab. 1: Anthropometric and peak oxygen uptake changes in endurance trained athletes during prolonged exposure periodic and continuous hypokinesia (mean ± SD).

Examined Parameters		Groups of Subjects: Ambulatory Control n=10	Continuous Hypokinetic n=10	Periodic Hypokinetic n=10
Age, years	Before	25.6 ± 7.0	24.8 ± 6.6	23.9 ± 6.0
Height, cm	Before	175.1 ± 6.5	174.8 ± 7.0	176.0 ± 8.0
Body mass	Before	74.0 ± 7.0	75.4 ± 6.5	73.3 ± 7.5
kg	After	74.5 ± 6.4	68.2 ± 7.7*	77.6 ± 6.0+
Body fat	Before	8.4 ± 1.2	9.1 ± 1.2	8.7 ± 1.3
%	After	8.5 ± 1.2	2.7 ± 1.4*	13.2 ± 1.5+
Fat free body	Before	67.8 ± 7.0	68.6 ± 8.5	67.1 ± 6.0
mass, kg	After	68.2 ± 6.5	66.6 ± 7.7	69.9 ± 5.5
Peak VO2	Before	65.4 ± 7.0	66.1 ± 5.5	66.7 ± 6.0
mL.kg-1.min-1	After	65.8 ± 6.4	51.9 ± 7.0*	56.8 ± 8.5*

* $p \leq 0.01$ significant differences between ambulatory control and hypokinetic groups of subjects.
+ $p \leq 0.01$ significant differences between continuous and periodic hypokinetic groups of subjects.

Tab. 2: Dietary intakes of endurance trained athletes during prolonged exposure to periodic and continuous hypokinesia (mean ± SD).

Examined Parameters	Groups of Subjects: Ambulatory Control n=10	Continuous Hypokinetic n=10	Periodic Hypokinetic n=10
Calories, kcal	3020 ± 342	2346 ± 233*	3146 ± 327
Proteins, g	125 ± 8.4	113 ± 7.0	135 ± 6.6
Lipids, g	116 ± 11.2	99 ± 10.4	127 ± 12.0
Carbohydrates	473 ± 11.0	420 ± 12.3	497 ± 11.5
Sodium	6720 ± 8.5	5840 ± 7.4	6971 ± 6.6
Potassium, mg	4350 ± 351.0	3890 ± 257.4	4410 ± 352.0
Calcium, mg	1710 ± 142.7	1487 ± 131.2	1780 ± 148.3
Magnesium, mg	640 ± 53	565 ± 47	665 ± 35
Fluid intake, mL	3765 ± 612	2882 ± 473*	3968 ± 654

* $p \leq 0.01$ significant differences between ambulatory control and hypokinetic groups of subjects.
+ $p \leq 0.01$ significant differences between continuous and periodic hypokinetic groups of subjects.

Assignment of the subjects into three categories was done randomly. Conditions of the three groups are as follows:

Group 1 Ten athletes were experienced no changes in their professional training and routine daily activities and their muscular activity was maintained at an average of 11.7 km.day-1. They served as unrestricted ambulatory control subjects (UACS).

Group 2 Ten athletes were subjected to changes in their professional training and routine daily activities and their muscular activity was restricted continuously to an average of 4.7 km.day-1 for 364 days. They served as continuously hypokinetic subjects (CHKS).

Group 3 Ten athletes were submitted to changes in their professional training and routine daily activities and their muscular activity was restricted periodically. That is, they were maintained under an average running distance of 4.7 and 11.7 km.day-1, for five days and two days per week, respectively, for 364 days. They served as periodically hypokinetic subjects (PHKS).

Simulation of continuous hypokinesia (CHK)

Subjects were admitted to the Metabolic Study Unit at the Hospital, where the studies were done. For the simulation of the hypokinetic effect, the number of km taken per day was restricted to an average of 4.7. Activities allowed were those that approximated the normal routines of sedentary individuals. During orthostasis the subjects were allowed to walk to dining tables, lavatories and different laboratories where the tests were given. Climbing stairs and other activities that required greater efforts were not allowed. Although subjects were mobile, they were confined to the hospital and not allowed outside the hospital ground. They could receive visitors daily.

Simulation of periodic hypokinesia (PHK)

Condition for the simulation of PHK during the five days of the week were the same as those with CHK; that is, the PHKS group during the five days of the week (Monday to Friday) was on an average running distance of 4.7 km.day-1. For the other two days of the week (Saturday and Sunday) the conditions for the PHKS group were the same as those with the UACS group; that is, the PHKS group during these two days of the week was allowed to resume their professional training and routine daily activities and run an average distance of 11.7 km.day-1. Thus, muscular activity of the PHKS group was restricted only during the five days of the week while during the other two days of the week was not restricted.

Sample collection

Samples of blood were taken at rest and before any meals, on the 1st, 15th and 30th day of the pre HK period and every 60 days during the periods of continuous and periodic hypokinesia. Blood samples were drawn under the same conditions between six and 9.00 a.m., without stasis and after the subjects had been sitting for about 30 min. Sample volume was 10 to 15 mL. Blood for serum was clotted with disposable polypropylene syringes (Becton-Dickinson, Rutherford, NJ). To obtain serum, blood was collected on ice for 60 min and, after rimming, was centrifuged at 3000 x g for 10 min at room temperature. Aliquots for serum Mg measurements were stored at –20 °C. Twenty four hour urine collections were maintained refrigerated at –4 °C until needed for Mg analysis. Faeces were collected in plastic bags, then dried, weighed and refrigerated at –20 °C for later analysis of Mg.

Biochemical measurements

All measurements were done in duplicate: Serum, urinary and faecal magnesium was measured using an atomic absorption spectrophotometer (Perkin-Elmer, Model 2380).

Anthropometric measurements

Height (cm) and weight (kg) were measured using a beam scale with attached stadiometer. Subjects wore nylon shorts during the measurements. Percent of body fat was calculated using the following skinfolds: biceps, triceps, subscapular, abdominal, chest, suprailiac as has been described previously [9]. Body fat was calculated using the equation of *Brozek* [10].

Peak oxygen uptake measurements

Respiration parameters were recorded continuously during the tests using an automated system (Beckman MMC). Room temperature was controlled at 18–20 °C. Oxygen uptake was calculated over min times intervals. The highest value during the tests was taken as maximum oxygen uptake. Peak oxygen uptake tests were done on a treadmill. The graded treadmill test protocol began with 10 min warmups at a work load that elicited a heart of 110 to 130 bpm. Treadmill speed increased at five min intervals until a speed of 10 to 13 mph was reached, after which the grade was increased 1% every five min until the subject fatigue was reached.

Statistical analysis

Data were analysed by performing a one or two-way analysis of variance (ANOVA) corrected for repeated measures. Post hoc analyses were done using the Tukey-Kramer multiple range tests. A format analysis was done to determine the shape of

changes. In all comparisons differences were considered statistically significant when $p \leq 0.01$.

Results

Prehypokinetic reactions

During the pre HK period, faecal and urinary Mg excretion and serum Mg concentration did not change significantly in the control and hypokinetic groups of subjects (tab. 3). Peak oxygen uptake, anthropometric measurements (tab. 1), food and water intakes (tab. 2) remained stable in the control and hypokinetic groups of subjects. The values of the corresponding parameters were typical to the values found in trained subjects on the same dietary intakes of Mg and in an analogous situation.

Hypokinetic reactions

During the hypokinetic period all subjects were in a satisfactory condition and none of the subjects were complaining of any serve discomfort or manifested any serious disorders. However, the subjects who were submitted to PHK and much less the subjects who were submitted to CHK experienced chest pain, heart burns, dyspepsia, dizziness, and heaviness in the head. Most subjects also complained of pain in the joints of the upper and lower extremities.

Anthropometric and peak oxygen uptake changes

In the UACS group peak oxygen uptake, body weight, body fat and fat free body mass did not change significantly when compared with the baseline control values (tab. 1). By contrast, in the CHKS group body weight, body fat and peak oxygen uptake decreased significantly ($p \leq 0.01$), while fat free body mass did not decrease significantly when compared with the UACS group (tab. 1). On the other hand, body weight and body fat ($p \leq 0.01$) increased significantly in the PHKS group, while fat free body mass did not increase significantly when compared with the CHKS group (tab. 1). Peak oxygen uptake decreased significantly ($p \leq 0.01$) in the PHKS group when compared with the UACS group (tab. 1).

Food and water intakes

In the UACS group food and water intakes remained stable when compared with the baseline control values (tab. 2). The CHKS group shows a significant decrease in their food and fluid intakes during the initial stages of the CHK period (tab. 2); then, as the duration of the CHK period increased food and water intakes increased progressively, but they stabilized below the values observed in the UACS group. By contrast, the PHKS group showed a much greater increase in their food and fluid intakes than the CHKS group (tab. 2); while during the days of resumption of muscular activity food and water intakes increased significantly ($p \leq 0.01$) in the PHKS group when compared with the CHKS group.

Serum, urinary and faecal magnesium

Urinary and faecal Mg excretion and serum Mg concentration remained stable in the UACS group when compared with the baseline control values (tab. 3). By contrast in the CHKS and PHKS groups urinary and faecal Mg excretion and serum Mg concentration increased significantly when compared with the UACS group (tab. 3). However, the increased serum Mg

Tab. 3: Serum, urinary and faecal magnesium of endurance trained athletes during prolonged exposure to periodic and continuous hypokinesia (mean ± SD).

	Examined Parameters		
Testing Days	Serum Magnesium mmol/L	Urinary Magnesium mmol/Day	Feacal Magnesium mmol/Day
	Ambulantory Control Subjects, n=10		
Base Line	0.84 ± 0.04	5.5 ± 0.6	40.1 ± 2.4
60th	0.83 ± 0.07	5.3 ± 0.4	38.0 ± 4.0
120th	0.84 ± 0.03	5.5 ± 0.3	41.1 ± 3.3
180th	0.82 ± 0.05	5.2 ± 0.6	37.5 ± 2.5
240th	0.84 ± 0.03	5.4 ± 0.5	39.4 ± 3.0
300th	0.85 ± 0.05	5.6 ± 0.4	36.8 ± 2.5
364th	0.04 ± 0.06	6.3 ± 0.6	38.9 ± 3.3
	Continuous Hypokinetic Subjects, n=10		
Base Line	0.86 ± 0.05	5.7 ± 0.3	43.0 ± 2.2
60th	0.96 ± 0.06*	7.5 ± 0.6*	65.5 ± 3.0*
120th	0.93 ± 0.03*	6.7 ± 0.4*	56.7 ± 4.4*
180th	0.99 ± 0.05*	8.6 ± 0.7*	73.4 ± 2.6*
240th	0.97 ± 0.04*	7.9 ± 0.6*	65.8 ± 3.3*
300th	1.04 ± 0.07*	9.5 ± 0.5*	77.1 ± 5.0*
364th	1.01 ± 0.05*	8.6 ± 0.6*	68.4 ± 2.5*
	Periodic Hypokinetic Subjects, n=10		
Base Line	0.84 ± 0.04	5.6 ± 0.5	45.2 ± 2.6
60th	0.99 ± 0.05*	8.3 ± 0.7*	77.5 ± 3.4*
120th	0.96 ± 0.04*	7.7 ± 0.5*	61.2 ± 3.0*
180th	1.04 ± 0.07*	9.5 ± 0.8*	89.6 ± 4.4*
240th	1.01 ± 0.04*	8.6 ± 0.5*	70.0 ± 2.6*
300th	1.06 ± 0.05*	10.6 ± 0.7*	99.5 ± 3.7*
364th	1.02 ± 0.06*	9.7 ± 0.6*	85.2 ± 2.6*

* $p \leq 0.01$ significant differences between ambulatory control and hypokinetic groups of subjects.
+ $p \leq 0.01$ significant differences between continuous and periodic hypokinetic groups of subjects.

concentration and urinary and faecal Mg excretion was much greater and appreared much faster in the PHKS group than in the CHKS group. Nevertheless, between the CHKS and PHKS groups were not observed any significant differences regarding urinary, faecal and serum Mg changes. In the CHKS and PHKS groups maximum urinary and faecal Mg excretion was always corresponded to the maximum serum Mg concentration. During the CHK and PHK periods urinary and faecal Mg excretion and serum Mg concentration increased progressively in both the hypokinetic groups while they showed a pattern of a wave like changes (tab. 3). Although urinary and faecal Mg excretion and serum Mg concentration was fluctuating throughout the HK period never reverted to the values observed in the UACS group (tab. 3).

Discussion

General hypokinetic reactions

Since none of the hypokinetic subjects complained of any severe discomfort or any serious disorders, it may be assumed that the hypokinetic subjects tolerated well the experimental procedures. It may also be assumed that faecal, urinary and serum Mg changes in the CHKS and PHKS groups could not have been induced either due to severe discomfort or serious disorders.

Anthropometric reactions

Unfortunately the reasons responsible for the significant body weight losses during prolonged CHK are not completely understood. However, available data [11–14] have shown that body weight losses during prolonged CHK may be attributable to several factors and primarily to the prevalence of catabolic processes over anabolic processes [11–14]. Since the CHKS group ended losing significant amounts of their body weight, it may be assumed that it was not in energy balance but in a catabolic condition that could definitely have influenced significantly urinary, faecal and serum Mg.

The possible reasons responsible for the body weight gains during prolonged exposure to PHK are much more complicate than the reasons responsible for the weight losses during prolonged CHK. It was assumed, however, that body weight gains during prolonged PHK may be attributable to several factors, while the induction of an additive effect of PHK and changes in energy metabolism should be considered first [7–8]. Since the PHKS group ended gaining significant amounts of their body weight, it may be assumed that the PHKS group, in contrast to CHKS group, was in an anabolic condition.

The reasons responsible for the significant body fat losses in the CHKS group may be attributable to the lipid metabolic changes inherent to prolonged CHK [16–18]. A significant decrease in both lipid reserves and tissues total lipids concentration has been observed during prolonged CHK [15–18]. A significant mobilization of lipids from fat depots and increased lipolytic activity of fatty tissue by 45 to 75% has been also observed during prolonged CHK [15–18]. Decreased fat free body mass in the CHKS group may be attributable to the muscle wasting that is inherent to prolonged exposure to CHK [19–22].

The possible reasons responsible for the body fat gains during prolonged PHK are much less clear than the reasons responsible for the body fat losses during prolonged CHK. However, it was assumed that body fat gains during PHK may be attributable to several factors: for instance; activation of lipogenetic processes and inhibition of lipolytic processes [23], increased lipid reserves and tissue total lipid concentration [15–18]. Increased fat free body mass in the PHKS group may be attributable to the decreased muscle wasting inherent to periodic restriction of muscular activity [19–22].

Peak oxygen uptake reactions

Decreased peak oxygen uptake in the PHKS and CHKS groups may be attributable to several factors and in particular to the decreased synthesis of hem proteins [24–27]. Decreased synthesis of haemoglobin and myoglobin could definitely have influenced significantly the oxygen carrying capacity to transport oxygen in blood muscles of hypokinetic subjects. Thus, the decreased peak oxygen uptake in the hypokinetic subjects may be attributable to the decreased synthesis of hem proteins and consequently to the decreased carrying capacity of blood to transport oxygen.

Serum, urinary and faecal magnesium changes

It is known that when endurance trained athletes receive large amounts of electrolytes with their food, serum or plasma electrolyte concentration does not increase significantly because significantly greater amounts of the given electrolyte loads are deposited in different organs and systems of the body; this reaction in turn protects systemic circulation from a rapid and excessive hyperelectrolemia by playing the role of a buffer system [1–3]. However, when endurance trained athletes were submitted to prolonged HK and electrolyte supplements were administered, serum or plasma electrolyte concentration increased significantly leading in significant faecal and urinary electrolyte loses and thus in electrolyte deficiency. It was assumed, therefore that the conditions during decreased muscular activity were much less favourable for the deposition of electrolytes in the body than the conditions during increased muscular activity.

Since serum Mg concentration increased significantly in the CHKS group and much more in the PHKS group, it may be assumed that they were deficient in Mg; this is because electrolyte deficiency during prolonged HK is characterized by

increased and not by decreased plasma or serum electrolyte concentration [1-6]. Furthermore, all signs, symptoms and complications referable to electrolyte deficiency are showing up during the post HK period and not during the HK period. Electrolyte deficiency during prolonged HK was established by performing electrolyte loading tests [3, 28, 29] during the post HK period, that is, during resumption of muscular activity. It was found that the hypokinetic subjects displayed a significant decrease in urinary and faecal electrolyte excretion in the presence of a significant decrease of plasma or serum electrolyte concentration. It was shown that the higher the retention of the given electrolyte load, the greater the electrolyte deficiency in the hypokinetic subjects. Increased faecal and urinary excretion of Mg, against the background of a significant increase of serum Mg concentration, may be attributable to several factors and in particular to the decreased ability of the body to retain Mg [1–6]. Faecal Mg and urinary Mg excretion appeared to be much greater and much faster during PHK than CHK presumably due to the type of restriction of muscular activity to which the athletes were subjected. As was shown in previous studie [1–6] serum or plasma electrolyte concentration increased significantly because significantly few amounts of electrolytes could be deposited by the body resulting in a significant faecal and urinary electrolyte excretion and thus electrolyte deficiency [1-6]. Decreased ability of the body to retain electrolytes during prolonged CHK and PHK may be attributable to several factors; for instance, changes in bone and muscles where most electrolytes are deposited, decreased electrolyte body deposition capacity, decreased utilization and assimilation of electrolytes for synthetic processes and several other reasons that are inherent to prolonged restriction of muscular activity [1–6].

Establishing the reasons responsible for the inability of the body to retain electrolytes during prolonged HK experimental studies were done using electrolyte loading tests. During these studies large amounts of electrolytes were administered in control and hypokinetic groups of subjects [1–6]. It was found that the hypokinetic subjects showed a much greater and a much faster electrolyte excretion in urine and faeces than the control subjects despite the presence of electrolyte deficiency and decreased total electrolyte concentration of the body. The results obtained from electrolytes loading tests also showed that the more electrolyte supplements the hypokinetic subjects receive the more deficient become and the more efficient electrolytes are cleared from the blood stream and the less likely it is to benefit the hypokinetic subjects and prevent electrolyte negative electrolyte balance and normalize total electrolyte concentration of the body. This type of reaction resembles a positive feedback, that is, the higher the electrolyte intake with food the greater the electrolyte loses in urine and faeces of the hypokinetic subjects. The failure to increase total electrolyte concentration of the body and prevent electrolyte deficiency in ahtletes with electrolyte supplements made it possible to formulate the following hypothesis; during prolonged exposure HK electrolyte deficiency and decreased total electrolyte concentration of the body is not so much a matter of shortage of electrolytes in the diet as the inability of the body to retain electrolytes due to several reasons [1–6].

It is known that Mg is one element necessary for protein synthesis. However, during prolonged HK, total Mg concentration of the body decreased significantly [1–6]. This reaction may also be attributable to the decreased use of Mg for synthesis of proteins because protein synthesis is significantly decreased during prolonged HK [11-14]. The shortage and imbalance of proteins may affect Mg metabolism; however, most probably the impossibilitiy of the body to retain Mg is the primary cause of an even lower assimilation and utilization of Mg by the body: decreased uptake of Mg on the one hand and increased urinary and faecal Mg excretion on the other. Then, the reaction apparently develops like a vicious circle, due to which an even greater shortage and imbalance of Mg develops during prolonged HK. The failure to normalize Mg deficiency and decreased total Mg concentration of the body by means of Mg supplements, as has been shown in previous studies [1–6], may be used as confirmation of this hypothesis.

Evidently, prolonged exposure to PHK induces a much greater serum Mg concentration and faecal and urinary Mg excretion than exposure to CHK. Moreover, serum, faecal and urinary Mg changes appeared much faster in the PHKS group than in the CHKS group. The reason responsible for this type of reaction may be attributable to several factors and in particular to the form of restriction of muscular activity, that is, PHK versus CHK. Perhaps it is this reason for which PHK exerted a much greater effect on serum, urinary and faecal Mg values than CHK. All trained subjects responded to PHK with a much greater and much faster serum, urinary and faecal Mg changes than to CHK. Since the PHKS group showed a much greater and much faster serum, urinary and faecal Mg changes then the CHKS group, it may be assumed that when physically conditioned subjects are submitted to PHK have a much lower electrolyte stability than when they are subjected to CHK. Apparently, physically conditioned subjects have a much more labile and a much less responsive Mg metabolic control system when they are exposed to PHK than to CHK. The reasons for this reaction remain unclear. However, evidence is emerging to suggest that when physically conditioned subjects are exposed to PHK have a much lower retention capacity for electrolytes than when they are submitted to CHK. This reaction suggests that alteration in muscular activity may affect significantly the metabolism of

electrolytes in individuals who do not maintain a stable muscular activity level.

Conclusion

It was concluded that prolonged exposure to CHK and PHK induces a significant increase of Mg excretion in urine and faeces of endurance trained athletes against the background of a significant serum Mg concentration. However, serum, faecal and urinary Mg changes were much greater and appeared much faster in the PHKS group than in the CHKS group. Differences between the PHKS and CHKS groups regarding the intensity and rapidity of serum, faecal and urinary changes of Mg may be attributable to several factors. Type of restriction of muscular activity and the additive effect of PHK on serum, faecal and urinary values of Mg should be considered first. Significant serum, faecal and urinary Mg changes in the PHKS and CHKS groups may also be associated with the decreased ability of the body to retain Mg and changes in bone and muscle tissues where most Mg is are deposited [1–6]. Unfortunately, the actual mechanisms responsible for the much greater and much faster serum, faecal and urinary Mg changes during exposure to PHK than CHK remain unclear. Understanding of the mechanisms responsible for the much greater and much faster serum, faecal and urinary Mg changes during PHK than CHK might come by studying Mg metabolism under different forms of HK and in different individuals. Investigating changes of Mg that might be present in individuals who are subjected to PHK and CHK due to various reasons may also contribute to the further understanding of Mg metabolism. For the present, however, it may be concluded the prolonged PHK induces a much greater and a much faster serum, faecal and urinary changes of Mg in endurance trained subjects than CHK.

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