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Behaviour of Serum Magnesium during a 3-Weeks Stage Race in Cyclists

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ABSTRACT

Magnesium (Mg) plays an essential role as a regulator of membrane stability and is required for neuromuscular, cardiovascular, immune, and hormonal functions; it is also considered a potential limiting element for human performance. We examined the effects of long-term continuous endurance exercise on serum levels of Ca and Mg in professional cyclists during 21 days of competition. Fourteen male cyclists from two Pro Teams were recruited to participate in the study. The blood samples were collected at the following three points in time during the 3 weeks of La Vuelta a España: i) at rest, just before the beginning of the tour (baseline, T_0), ii) after 9 days of racing (before the beginning of the 10th stage, T_1) and iii) at the end of the tour (before the last stage, T_2). Serum levels of Ca, Mg, creatinine, lactate dehydrogenase (LDH), creatinine kinase (CK), aspartate transaminase (AST), alanine transaminase (ALT), aldolase, and total proteins (TPs) were measured. The results of the study reveal a gradually decrease of Mg and Ca mean plasma levels through the 3 weeks of La Vuelta a España and an increase of the creatinine levels. Our findings suggest that duration, intensity, and training levels of exercise play decisive roles in the regulation of serum magnesium homeostasis after endurance exercise.

Keywords: Magnesium, Calcium, Exercise, Cyclists, Creatinine.

INTRODUCTION

A poor mineral status composition leads to a decrease in endurance performance in athletes and animals [3,8-10]. Magnesium (Mg) is an important cofactor in more than 300 enzymatic reactions that are involved in energetic metabolism and acts as a physiologic regulator of membrane stability and neuromuscular, cardiovascular, immune, and hormonal functions [3,8]. Also, the major anabolic and catabolic processes are Mg dependent [15].

Exercise may increase the demand for magnesium and/or a loss of magnesium that, potentially, leads to hypomagnesaemia. Consequently, muscle weakness, neuromuscular dysfunction, and tetany may occur and may affect physical performance and/or health status. Thus, magnesium may be considered a limiting element for human performance. When studying in athletes, several studies that have shown a significant loss of magnesium after performing strenuous exercise [3,25,27,31].

Mg and calcium (Ca) are two cations closely involved in many metabolic and physiological processes that are involved in muscular performance and regulate an appropriate body function, particularly during exercise [26,28].

There are several studies that have determined compartmental shifts of Mg and Ca at rest and after exercise [9-12,32] and generally, they consist of a redistribution mechanism and their extent depend on the kind of exercise (length, training, bouts of work, etc.) [2,3,8,9,32].

The following mechanisms have pointed out to explain the mentioned redistribution. a) the acidosis caused by exercise promotes the redistribution of minerals from the bone to the extracellular fluid [11,2]. b) hormones that fluctuate during exercise such as thyroid, antidiuretic, aldosterone, ACTH, and corticosterone are the responsible of the mineral mobilization [11,12,15,16]. c) calciotropic hormone response after exercise may modify mineral distribution [5] and d) stress induced by exercise and the subsequent secretion of catecholamines may also cause mineral mobilization [15,32].

Many metabolic processes are accelerated during exercise to ensure the optimal balance of minerals in the body. As a consequence, Mg levels may fluctuate after exercise and its homeostasis may be altered for a long time. The extend of those exercise-induced shifts depend on the pathway of energy metabolism taken. Stendig-Lindberg [32] observed that serum Mg levels remained reduced after 18 days of intense exercise. Other scholars have found that sustained moderate physical exercise such 80 km march of 18 h duration and short-term high intensity (anaerobic) exercise increase the concentration of serum Mg [27,31]. According to Bohl and Volpe [3], the changes in extracellular Mg concentration suggest, partially, a body's response to exercise; in such way, a redistribution of Mg into body compartments with higher metabolic needs occurs, regardless of the nature of the effect. Energy production and counteracting oxidative stress are examples of processes with elevated metabolic needs.

Therefore, the results of the variations of Mg levels after exercise are very controversial. The existing inconsistencies may be related to differences in experimental designs, intensity and duration of exercise, timing of blood samples, environmental conditions, etc. To our knowledge there have been no studies carried out about behavior of Mg and Ca in long-term continuous endurance exercise in athletes. Thus, this article examines effects of long-term continuous endurance exercise on serum levels of Ca and Mg in professional cyclists during 21 days of competition.

MATERIAL AND METHODS

Subjects

Fourteen male cyclists from two professional cycling teams were recruited to participate in the study; however, four participants dropped out due to race injury. The participant anthropometric characteristics before the beginning of the 3-weeks stage race, La Vuelta a Espana, are shown in Table 1. The subjects of the study followed a similar diet and performed the same training program which were supervised by the dietitian/nutritionist and the physician of the team, respectively. Prior to onset of the study, medical examinations were conducted to ensure free-disease participants. Participants committed to use neither banned drugs nor medications which may affect body weight. Thus, we assume the participants kept an honest attitude during the length of the study. The athletes' supplementation treatment plan consisted in: iron, folic acid, B12 vitamin, glycophosphopeptical (AM3), BCAAs, glutamine, and vitamin C. Participants accepting to participate signed the informed consent after receiving an explanation of the experimental procedures, associated risks, and benefits of the study. No athletes had pre-existing injuries prior the race. This study was designed according to Declaration of Helsinki (2008) and approved by the University of Valladolid committee.

Table 1: Physical and anthropometric characteristics

	Mean ± SEM
Age (ys)	25.7 ± 1.4
Weight (kg)	69.87 ± 2.92
Height (cm)	178.2 ± 3.29
Σ 6 skinfolds	35.43 ± 3.16
Body fat (%) (Yuhasz)	8.75 ± 0.66

Max. oxygen uptake (ml. kg-1 min-1)	77.6 ± 6.48

Three weeks before the beginning of the research, each cyclist took part in an incremental maximal cycling test. A mechanically braked cycle ergometer (Monark 818 E, Varberg, Sweden) adapted with a racing saddle, drop handlebars and clip-in pedals was used. The test started with an initial resistance of 110 W, with further increments of 35 W every 3 min. Participants kept a constant 75 rpm pedal cadence with the help of a metronome. Testing ended when the cyclist was no longer able to keep the required pedal cadence. Heart rate was recorded at 5-s intervals during the test (Polar S720i, Polar Electro Oy, Finland). Gas-exchange data were continuously monitored with a breath-by-breath metabolic cart (CPX-Plus, Medical Graphics Corporation, St. Paul, Minnesota, USA). Maximal power (W-max) was determined as the highest workload that the cyclist was able to maintain for a complete 3-min period.

La Vuelta a España began in mid-August and consisted in 3 consecutive weeks of competition, with 2 rest days, one at the end of first week and the second at the end of 2º week. The participants of the study covered a distance of 3,296 km between Pamplona and Madrid. Circadian rhythms, nutrition diet, hydration levels, meal times, and sleep hours were constant or pretty similar throughout the 21 days of competition, which is a normal practice in cycling competitions.

The cyclists were tested at three specific points around La Vuelta a España: i) at rest, just before the beginning of the tour (baseline, T_0), ii) after 9 days of racing (before the beginning of the 10^{th} stage, T_1) and iii) at the end of the tour (before the last stage, T_2).

Anthropometric characteristics

Anthropometric characteristics (Table 1) were taken by a level 3 anthropometrist member of the ISAK (International Society for Advancement in Kinanthropometry), following the standard procedures at T0. The height and body mass technical error of measurement (TEM) was less than 0.02%. The skinfolds TEM was all less than 2.6%. The height (cm) was measured with a SECA measuring rod, with a precision of 1 mm (range: 130-210 cm), while body mass (BM) (kg) was assessed by a SECA model scale, with a precision of 0.1 kg (range: 2-130 kg). All skinfolds (triceps, biceps, abdominal, supraspinale, subscapular, chest, front thigh and medial calf) were taken by Harpenden skinfold caliper (CMS instruments, London, UK), with a precision of 0.2 mm. A sum of 6 skinfolds (triceps, abdominal, supraspinale, subscapular, front thigh and medial calf) was calculated.

Blood collection and analysis

Blood samples were collected from all cyclists at T_0, T_1, and T_2. All samples were collected in basal conditions after an overnight fast and were taken at 8:30 am. Blood samples were collected after the subject was taken in a comfortable seated position from the antecubital vein using a Vacutainer system (10 mL to serum tubes, 5-mL and 3-mL tubes with EDTA). Serum was separated from blood cells and stored at 20°C until analysis.

Red blood cell, white blood cell and platelets count, hemoglobin (Hb), and hematocrit (Hct) were determined on a Coulter Counter (model MAX-M). The percentage of change in plasma volume (%PV) was calculated in function of hematocrit changes [34].

Serum Ca and Mg were determined with a Perkin Elmer 272 “Flame atomic absorption spectrometry (FAAS)” device in flame emission mode. Erythrocytic Mg was determined as follow: whole blood was hemolyzed by dilution with deionized water, mixed with a vortex, and then frozen. After Mg determination in whole blood and plasma, erythrocytic Mg concentration was calculated as: (whole blood Mg - plasma Mg)x(1 - Hct))/Hct. Serum levels of creatinine, creatin kinase (CK), lactate dehydrogenase (LDH), aspartate transaminase (AST), alanine transaminase (ALT), aldolase and total proteins (TPs) were measured at each control point (T_0, T_1, T_2). The biochemical parameters were measured using coupled enzyme reactions on an automatic autoanalyzer (Hitachi 917, Japan). Serum C levels were measured with an enzyme-linked fluorescent assay technique, a combination of ELISA and a fluorescent final lecture, with the aid of a multipara metric analyzer (Minividas, Biomerieux), using a substrate 4 methylumbelipherone capable of fluorescent emission at 450 nm, after stimulation at 370 nm. Myoglobin (Mb) assessment was performed using a technique based chemiluminescence reaction enzyme immunoassay "sandwich" of two points.

Statistical data analyses

The Statistical Package for the Social Sciences 22.0 (SPSS) was used. Data were expressed as mean ± standard error of the mean. The Shapiro-Wilk test was used to assess the non-normality OD the distribution and an one-way repeated measure ANOVA was carried out for all biochemical parameters (minerals, biochemical, muscle damage, and stress). Greenhouse-Geisser test was used to determine whether statically significant differences exist between mineral parameters at the different 3 phases of the study (T_0, T_1, y T_2). Bonferroni test post hoc was performed to determinate differences between the periods of study. Bivariate correlations between changes in Ca and Mg during the length of the study $\Delta(T_0–T_2)$ were tested using Pearson rank order correlation test after calculating $\Delta(T_0–T_2)=((T_2-T_0)/T_0) \times 100$. Likewise, bivariate correlations between changes in Mg and hematological, muscle damage, and stress during the length of the study $\Delta(T_0–T_2)$ were tested using Pearson. A value of $p<0.05$ was considered as significant.

RESULTS

The results of serum Mg and Ca levels at the three points in time during the 3 weeks of La Vuelta a España are shown in Table 2. The Serum Mg and erythrocytic Mg levels show a decreasing tendency (NS) through the 3 weeks of competition. However, serum Ca levels at the end of the race are statistically significant lower than at the beginning of the race ($p<0.05$). A significant high positive correlation ($p<0.001$, $R^2=0.8385$) between Ca and Mg behavior is observed through the 3-weeks stage race. This finding implies that those cyclists who have higher decrease of the serum Ca concentration also have higher decreased of serum Mg concentration and vice versa.

Table 2: Serum calcium and magnesium levels of professional cyclists during "Vuelta a España".

		Mean ± SEM	95% confidence interval of ICC		p
Ca (mg/L)	T0	121.25 ± 2.99	114.19	128.31	0.05
	T1	111.88 ± 5.36	99.2	124.55	
	T2	105.50 ± 5.31a	92.95	118.05	
Mg (mg/L)	T0	24.25 ± 0.86	22.214	26.286	NS
	T1	22.63 ± 0.78	20.786	24.464	
	T2	22.75 ± 0.80	20.867	24.633	
Mg-e (mg/L)	T0	66.65 ± 0.25	60.75	72.55	NS
	T1	61.14 ± 0.32	53.47	68.81	
	T2	62.36 ± 0.17	58.36	66.36	

Mg-e: eritrocytic magnesium.
Significant differences among period of study by Bonferroni´s test:a: Vs. T0.

Table 3 shows hematological parameters at the three points in time of the study. No statistically significant differences are shown in RBC and TP but an increasing tendency at the end of the 3 weeks of competition is observed. Additionally, WBC levels at the end of the study (T^2) are statistically significant higher than at the baseline (T0) ($p<0.05$). On the other hand, Hb values ($p=0.003$) and platelets count ($p<0.01$) are statistically significant lower at T^1 than at T^0.

Table 3: Hematological parameters of professional cyclists during "Vuelta a España".

		Mean ± SEM	95% confidence interval of ICC		P
WBC (103 µL-1)	T0	5.97 ± 0.57	4.64	7.31	0.01
	T1	6.89 ± 0.43	5.88	7.89	
	T2	9.33 ± 0.51a	8.13	10.53	
Platelet (103 µL-1)	T0	244.75 ± 17.47	203.44	286.06	0.01

	T1	200.25 ± 12.69a	170.25	230.25	
	T2	249.75 ± 24.39b	192.07	307.43	
RBC (106 µL-1)	T0	5.36 ± 0.70	5.2	5.52	
	T1	4.80 ± 0.12	4.51	5.08	NS
	T2	4.83 ± 0.30	4.23	5.63	
Hb (g.dL-1)	T0	16.68 ± 0.15	16.32	17.03	
	T1	14.76 ± 0.32a	14.01	15.52	0.01
	T2	15.19±0.52	14.25	16.72	
Htc (%)	T0	48.64 ± 0.43	49.23	51.25	
	T1	46.15 ± 0.87	44.69	48.81	NS
	T2	44.04 ± 2.62	37.85	50.23	
ΔPV (%)	T0-T1	4,3			
	T1-T2	4,2			
	T0-T2	5,9			

WBC: White blood cells; RBC: Red blood cells; Hb: Hemoglobin; Htc: Hematocrit; %ΔPV: Plasma volume.

*Significant differences calculated by Wilcoson test.

Significant differences among period of study by Bonferroni´s test:

a: Vs. T0.

b: Vs. T1.

Table 4 shows biochemical enzymes and hormonal parameters at the 3 phases of the study. No statistically significant differences are shown in CK, LDH, and aldolase but an increasing tendency at the end of the race (T_2) is noticed. Regards to cortisol levels, no statistically differences are determined but an increasing tendency at the end of the 3 weeks of competition (T_2) is observed. Additionally, creatinine ($p<0.01$), Mb ($p<0.01$), GOT ($p<0.05$) and GPT ($p<0.01$) values are statistically significant higher at the end of the study (T_2) than at the baseline (T_0) whereas total protein values are statistically significant lower ($p<0.01$).

Table 4: Muscle damage data of professional cyclists during "Vuelta a España".

		Mean ± SEM	**95% confidence interval of ICC**		**P**
Creatinine (mg/dl)	T0	0.85 ± 0.02	0.79	0.9	0.01
	T1	0.93 ± 0.04	0.84	1.01	
	T2	$1.05 \pm 0.04_a$	0.95	1.15	
CK (U/l)	T0	107.4 ± 2.3	101.9	112.8	0.05
	T1	115.0 ± 5.4	102.2	127.7	
	T2	144.4 ± 5.8	106.8	134	
Mb (ng/ml)	T0	34.43 ± 2.46	28.62	40.24	0.01
	T1	34.75 ± 1.67	30.81	38.69	
	T2	$42.03 \pm 3.38_a$	34.04	50.03	
LDH (U/l)	T0	299.5 ± 39.9	196.85	402.15	NS
	T1	355.3 ± 42.2	246.83	463.84	
	T2	425.7 ± 57.5	277.76	573.57	
GOT (U/l)	T0	29.88 ± 3.42	21.789	37.961	0.05

	T1	45.50 ± 3.35$_a$	37.581	53.419	
	T2	46.50 ± 7.34	29.141	63.859	
GPT (U/l)	T0	25.13 ± 3.09	17.817	32.433	0.01
	T1	34.88 ± 2.61$_a$	28.708	41.042	
	T2	51.75 ± 7.89$_a$	33.105	70.395	
Total proteins (g/dl)	T0	7.35 ± 0.11	7.083	7.617	0.01
	T1	6.94 ± 0.09$_a$	6.723	7.052	
	T2	7.07 ± 0.07$_b$	7.255	7.27	
Aldolase (U/l)	T0	4.05 ± 0.58	2.565	5.535	NS
	T1	5.55 ± 0.37	4.596	6.504	
	T2	4.72 ± 1.13	1.802	7.631	
Cortisol (µg/dL)	T0	19.42 ± 0.83	17.45	21.39	NS
	T1	20.67 ± 0.87	18.61	22.72	
	T2	21.55 ± 0.65	20.03	23.08	
Significant differences among period of study by Bonferroni´s test: a: Vs. T0. b: Vs. T1.					

Figure 1A shows a statistically significant negative correlation ($p<0.003$, $R2=0.787$) between Mg and creatinine behavior during the 3-weeks stage race (T_0-T_2) and Figure 1B shows a similar negative correlation but not statistically significant between erythrocytic-magnesium (Mg-e) and creatinine. This result implies that those cyclists who have higher decrease of Mg level have higher increase on creatinine level and vice versa.

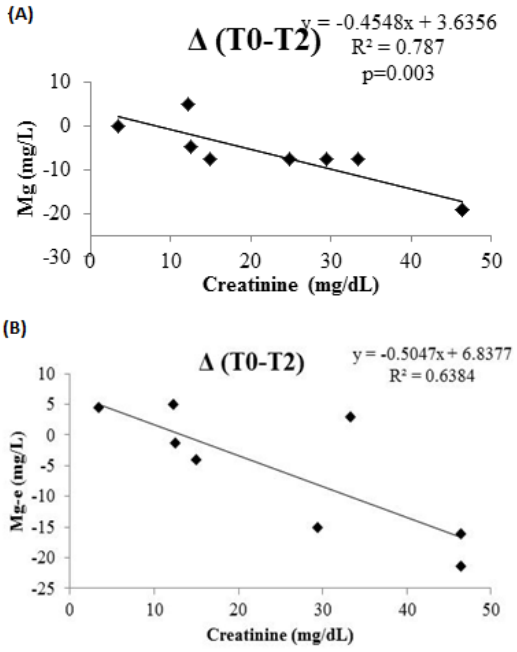

Figure 1: Correlations between serum Mg and creatinine during stage race (start and finish of 3 weeks of race). A) serum Mg, and B) eritrocytic Mg (Mg-e)

DISCUSSION

Poor dietary magnesium intake may lead to Mg deficiency status which results in serious impaired physiologic function and athletic performance. In addition, exercise increases the demand of magnesium and/or increases magnesium loss which contribute to the impair exercise performance [3,8,23]. Most of the existing studies focus on the relation between the serum magnesium concentration and the type of exercise performance. However, and to our knowledge, there are no studies that examine the behavior of serum Mg during 21 days of competition.

A considerable number of studies have examined the Mg variations induced by exercise, however, the resulting findings are very inconsistent. Whereas some scholars have found significant increases of serum Mg concentrations induced by exercise, several have found decreases, and others found no substantial variations [3,4,10,11,13,25,31]. This lack of consistency may be explained due to the different exercise performed by the participants of the studies. According to Hsu et al. [20] the behavior of Mg depends of the type, intensity and duration of exercise.

Based on findings from previous studies performed in athletes, short term high intensity exercise induce hypomagnesaemia, while long term moderate exercise is frequently associated with hypomagnesaemia. For instance, Deuster et al. [13] pointed out that acute anaerobic exercise may induce hypomagnesaemia, whereas prolonged submaximal exercise may lead hypomagnesaemia. Hypomagnesaemia results in muscle weakness, neuromuscular dysfunction, and tetany [22], which has negative consequences for athletes' performance and/or their health status.

The present study was mostly performed under aerobic conditions, approximately 80% of the overall time of the race, and only a 20% was performed under anaerobic circumstances such as sprinting and climbing. This fact is essential to interpret the findings of the study that reveal a decrease of the levels of Mg and Ca in the mean plasma of the cyclists after completing 21 days of competition and riding more than 3,000 km in La Vuelta a España. However, the levels of serum Mg determined ranged within normal values. The normal serum Mg concentration may be explained due to the well-balanced diet and good eating habits followed by the participants of the study (they were under supervision of a professional dietitian/nutritionist). The decrease of the levels of Mg presented by the cyclists after 3 weeks of competition may be explained due to the redistribution of Mg from one storage area (release) to an active site (exercising muscle, erythrocytes, or adipose tissue) as previously have been documented [3,6,24,29,31,32].

According to Lijnen et al. the decrease of Mg levels may be explained because as exercise is prolonged and fatty acid metabolism is increased, magnesium can be taken up into adipocytes, thereby reducing the vascular magnesium concentration [22]. These authors concluded that at the end of a marathon race, as fatty acids are mobilized for muscle energy, Mg can be taken into fact cells, causing a decrease in plasma Mg. However, Swaminathan [33] did not agree with Lijnen [22] because cyclists and triathletes usually have very low percentages of body fats. For Golf [18] the explanation of the decrease in Mg concentration may be explained because, as the exercise duration increases, the magnesium shifts from an erythrocyte reservoir into the plasma and then shifts to the working muscles.

The concentration of creatine and total CK activity has also been object of study in athletes because of its importance in muscle damage and kidney function. Firstly, the concentration of creatine in serum is the most widely used and also commonly accepted measure of renal function in clinical medicine and reflects in part the muscular activity. Secondly, in sports medicine, creatine is used to assess the general health status of athletes, particularly in events in which hydroelectrolytic balance is crucial for the developing of the activity. In this sense Banfi [1] saw that the serum creatinine concentrations in athletes were higher than in sedentary people. They also saw a significant increase in the concentration of muscular enzymes such aminotransferases, LDH, and myoglobin protein that confirms the damage and destruction of skeletal muscle. In a similar research line, Stendig-Lindberg [30] measured total CK activity and magnesium concentration in subjects who completed a 120-rnile hike and found an increase of both CK activity and magnesium concentration 24 h post-exercise. Because CK is released from damaged skeletal muscle after exercise [7], these scholars suggested that the increase of magnesium concentration post-exercise may indicate a release from damaged tissue.

According to the mentioned long term exercise studies, we found an increase in creatinine levels in our study. Creatinine concentration (a breakdown product of creatine phosphate produced in muscle) increases generally after strenuous exercise events such as Ironman, ultra-triathlon, or marathon [17,35]. The increase in plasma creatinine concentration is probably the result of a release of creatinine from the working muscles, dehydration, and/or a reduction in renal blood flow and glomerular filtration rate.

We also found a gradually decrease in the levels of serum Ca through the 21 days of competition. According to Grimston et al. [19] the Ca concentration decreases due to an increase in calcitonin level as consequence of a bone resorption and Ca absorption from the small intestine or urinary Ca output.

On the other hand, stress has been demonstrated that affects generally the redistribution of minerals and specifically affects the redistribution of Mg. At the same time, catecholamines are elevated during strenuous effort and they facilitate entry of magnesium into soft tissue [14]. In this line, Joborn et al. [21] observed that the infusion of adrenaline (5 μg/min for 30 min followed by 10 μg/min for 30 minutes) significantly reduced the plasma Mg levels in healthy males. They conclude that both the beta-adrenergic system and muscular activity by itself affect Mg homeostasis. Since catecholamines were not measured in this study, future research is needed to clarify the mechanisms that enhance the entry of magnesium into skeletal muscle in response to exercise. Lastly, we want to mention that although stress level of the participants was increasing as the end of race was closer; the cortisol levels remained within normal range.

In conclusion, the performance of a prolonged endurance exercise is associated with several potential biochemical changes. Our findings suggest that exercise duration and intensity and training status play critical roles in the regulation of serum magnesium homeostasis after performing exercise. The results of the study support our hypothesis that long-term continuous endurance exercise in professional cyclists during 21 days of competition induces losses of magnesium and small decrease in Ca concentration.

REFERENCES

[1]. Banfi, G., del Fabbro, M, and Lippi, G., *Br J Sports Med*, **2006**. 40: p.675-678.
[2]. Barzel, VS. and Jowsey, J., *Clin Sci*, **1969**. 36: p.517-524.
[3]. Bohl, CH. and Volpe, SL., *Magnesium and exercise. Crit Rev Food Sci Nutr*, **2002**. 42: p.533-563.
[4]. Casoni, I., et al ., *Int J Sports Med*, **1990**. 11: p.234-237.
[5]. Chilibeck, P.D., Sale, D.G, Webber, CE., *Sports Med*, **1995**. 19: p.103-122.
[6]. Clarkson, PM. And Haymes EM., *Med Sci Sports Exerc*, **1995**. 27: p.831-843.
[7]. Clarkson, PM., Nosaka, K, and Braun, B., *Med Sci Sports Exerc*, **1992**. 24: p.512-520.
[8]. Clarkson, PM., *J Sports Sci*, **1991**. 9: p.91-116.
[9]. Córdova, A., Giménez, M, Escanero, JF., *J Trace Elem Electrolytes Health Di*, **1990**. 4: p.189-192.
[10]. Córdova, A., Giménez, M, Escanero, JF., *Physiol Behav*, **1990**. 48: p.595-598.
[11]. Córdova, A., Giménez, M, Escanero, JF., *Magnes Res*, **1992**. 5: p.23-27.
[12]. Córdova, A., *Physiol Behav*, **1992**. 52: p.819-821.
[13]. Deuster, PA., *J Appl Physiol*, **1987**. 62: p.545-550.
[14]. Durlach, J., and Durlach, V., *Magnesium*, **1984**. 3: p.109-131.
[15]. Durlach, J., *Magnesium in Clinical Practice. John Libbey*, London, **1988**. p.13-17.
[16]. Galbo, H. and Kjaer, M., *Can J Sport Sci*, **1987**. 12: p.102-107.
[17]. Gastmann, U., *J Sports Med Phys Fitness*, **1988**. 38: p.18-23.
[18]. Golf, SW., Bender, S, Grüttner. J., *Cardiovasc Drugs Ther*, **1998**. 12: p.197-202.
[19]. Grimston, S.K., *J Clin Endocrinol Metab*, **1993**. 76: p.867-872.
[20]. Hsu, M.H., *J Biomed Lab Sci*, **2007**. 19: p.25-29.
[21]. Joborn, H., Akerstron, G, Ljunghall, S., *Clin Endocrinol*, **1985**. 23: p.219-226.
[22]. Lijnen, P., *Eur J Appl Physiol*, **1988**. 58: p.252-256.
[23]. Lukaski, H.C. and Nielsen, F.H., *J Nutr*, **2002**. 132: p.930-935.
[24]. Navas, F.J., Martin, J.F, and Córdova, A., *Med Sci Sports Exerc*, **1997**. 29: p.882-891.
[25]. Nielsen, F.H. and Lukaski, H.C., *Magnes Res*, **2006**. 19: p.180-189.
[26]. Rayssiguier, Y., Guezennecc, C.Y, and Durlach, J., *Magnes Re*. **1990**. 3: p.93-102.
[27]. Rayssiguier, Y., Mazur, A, and Durlach, J., *Advance in Magnesium Research: Nutrition and Health. John Libbey Company, London*. **2001**.
[28]. Richardson, J., Palmertson, T, Chenan, M., *J Sports Med Phys Fitness*, **1980**. 20: p.149-151.
[29]. Schwellnus, M.P., et al ., *Br J Sports Med*, **2004**. 38: p.488-492.
[30]. Stendig-Lindberg, G., *J Am Coll Nutr*, **1987**. 6: p.35-40.
[31]. Stendig-Lindberg, G., *Trace Elem Electroly*. **1999**. 16: p.56-161.

[32]. Stendig-Lindberg, G., *Magnes Res*, **1989**. 3: p.211-218.
[33]. Swaminathan, R., *Clin Chem*, **2000**. 46: p.1695-1696.
[34]. Van Beaumont, W., *J Appl Physiol*, **1972**. 32: p.712-713.
[35]. Whiting, P.H., Maughan, R.J, Miller, J.D., *Eur J Appl Physiol*, **1984**. 52: p.183-187.