Plasma and Red Blood Cell Magnesium Levels and Plasma Creatinine after a 100 km Race

R. Rama*, J. Ibáñez, T. Pagés, A. Callis** and L. Palacios

Departamento de Bioquímica y Fisiología Facultad de Biología Universidad de Barcelona 08028 Barcelona (Spain)

(Received on May 8, 1992)

R. RAMA, J. IBÁNEZ, T. PAGÉS, A. CALLIS and L. PALACIOS. Plasma and Red Blood Cell Magnesium Levels and Plasma Creatinine after a 100 km Race. Rev. esp. Fisiol., 49 (1), 43-48, 1993.

Magnesium homeostasis is critical for exercise performance. In this report the effect of long distance race on the erythrocyte and plasma magnesium concentration is determined in a group of 7 well-trained male amateur runners. After a 100 km race the plasma Mg^{2+} levels increased significantly from 0.845 ± 0.074 to 0.934 ± 0.099 mmol·l⁻¹ (p < 0.05). However, the intra-erythrocyte Mg^{2+} concentrations were not modified significantly (2.10 \pm 0.2 mmol·l⁻¹ versus 2.14 \pm 0.12 mmol·l⁻¹). Creatinine plasma levels increased significantly from 73.4 \pm 3.5 µmol·l⁻¹ to 117.6 \pm 19.4 µmol·l⁻¹ (p < 0.01), suggesting impairment of the renal function. A significant positive correlation between plasma magnesium and plasma creatinine, r = +0.65 (p < 0.01) was found. These results suggest that an increase in the magnesium plasma levels could be related to renal failure during long-distance running.

Key words: Magnesium, Creatinine, Long-distance race.

Magnesium is essential in neuromuscular transmission, muscular contraction, protein and nucleic acid synthesis and membrane transport. It also serves as a cofactor in more than 300 enzyme reactions, most notably those involving energy transfers via ATP (14). The magnesium levels in the plasma of healthy people are remarkably constant, being on average 0.85 mmol·l⁻¹ and varying less than 15 % (19, 21). Loss of intracellular magnesium can lead to muscle weakness, neuromuscular dysfunction and tetani (1, 8). Thus maintenance of intracellular magnesium is important for exercise performance.

Depending on the type of exercise, discordant findings on erythrocyte and plasma magnesium concentrations have been reported in human subjects. Various re-

^{*} To whom all correspondence should be addressed (Tel.: (93)-4021555. Fax: (93)-4110358). ** Present address: Faculté de Medecine, Université

^{**} Present address: Faculté de Medecine. Université de Montpellier. Montpellier (France)

ports have shown a decrease in plasma magnesium after prolonged heavy exercise such as marathon running (6, 15, 20). An increase in plasma magnesium concentration was observed after short-term physical exercise (11, 13, 23). Here we study the effect of a long distance race on magnesium homeostasis in well-trained male athletes during a 100-km race.

Materials and Methods

Seven well-trained male athletes, nonsmokers and in good health, aged between 27 and 41 years (mean 37), body weight of 56-74 kg (mean 65.4 kg), agreed to participate in the study. They all signed a statement of informed consent. They were experienced long-distance runners usually running between 110 and 140 km weekly.

Our survey was carried out during 100km race. It started at 6 A.M., and the average racing time was 10 hours (8-12 h). Air temperature ranged from 9 to 23 °C, and relative humidity was 65-80 %. During the race the runners were allowed to eat and drink without restriction.

All subjects were weighed before and after the race. Peripheral venous blood samples were drawn by antecubital venepuncture before the start and within 15 min of completion of the race, with the subject in the recumbent position. Aliquots of blood were anticoagulated with EDTA for determination of the erythrocyte count, haematocrit, and haemoglobin concentration, which were performed with a Coulter Counter. The percentage changes in plasma volume (% ΔPV) were calculated from resting and post-exercise haemoglobin and haematocrit measurements (22). Plasma for subsequent analyses of the metabolites and enzymes was obtained from whole blood in a refrigerated centrifuge and stored in aliquots at -40 °C until tested.

Plasma and erythrocyte magnesium was measured by atomic absorption spectrophotometry, and the erythrocytes were washed and lysed (15).

Plasma glucose, lactate, creatinine and urea concentrations were determined by colorimetric methods using Boehringer Mannheim kits.

Creatine kinase activities were determined by commercial kits (Boehringer Mannheim, FRG) on a Milton Roy Co., Spectronic 1201 spectrophotometer.

For statistical analysis the non-parametric test was used: the Wilcoxon test for paired values, and the Spearman rank correlation. Data are given as mean \pm SD.

Results

The athletes covered the 100-km on average in 10 h (range: 8 h to 12 h). As shown in table I, after the run, all the subjects showed a significant decrease in body weight, which represented a loss of 4.6 %.

Plasma and erythrocyte magnesium concentrations before and after race are shown in fig. 1. After a 100-km race the level of plasma Mg²⁺ showed a significant increase from 0.845 \pm 0.074 to 0.934 \pm 0.099 mmol·l⁻¹ (p < 0.05). However, the

Table I. Body weight, blood and plasma measurements (mean \pm SD) before and after the 100-km race (n = 7)

	Before	After
Body weight (kg)	65.36 ± 6.36	62.35 ± 5.42*
Haematocrit (%)	47.8 ± 2.3	50.2 ± 3.0*
Haemoglobin (g·dl ⁻¹)	15.53 ± 0.63	15.63 ± 0.64
Lactate (mmol·1 ⁻¹)	1.29 ± 0.31	3.57 ± 1.22***
Glucose (mmol·1 ⁻¹)	4.77 ± 0.88	4.44 ± 0.72
Urea (mmol·1 ⁻¹)	6.09 ± 1.00	8.35 ± 1.35**

*p < 0.05; **p < 0.01; ***p < 0.001

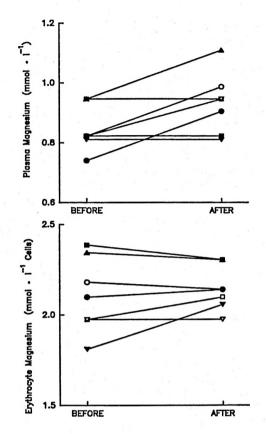


Fig. 1. Plasma and erythrocyte concentrations of magnesium expressed as single values before and after a 100-km race.

level of magnesium erythrocyte (2.10 \pm 0.2 versus 2.14 \pm 0.12 mmol·l⁻¹) were similar before and after the race; an increase in erythrocyte magnesium levels was found in three athletes, while in three others a decrease in magnesium erythrocyte levels was observed.

In order to study the renal function the levels of creatinine in plasma were determined. After the run there was a significant increase (60 %) in the creatinine concentration (73.4 ± 3.5 versus 117.6 ± 19.4 μ mol·l⁻¹ (p < 0.01) suggesting an alteration of the renal function (fig. 2). The increase in plasma magnesium correlates sig-

Rev. esp. Fisiol., 49 (1), 1993

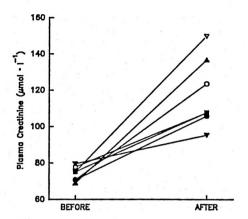


Fig. 2. Plasma concentrations of creatinine expressed as single value before and after a 100-km race.

nificantly with the increase in creatinine (r = +0.66, p < 0.01), suggesting that the high levels of plasma magnesium may be due to decrease in glomerular filtration rate.

There were no differences in erythrocyte count and haemoglobin concentration, but an increase in haematocrit values was found (table I). All the athletes showed a hemoconcentration with decrease in the plasma volume with values between 1.78 % and 10.6 % (average 5.7 %) after the race.

The plasma creatine kinase activity is used to estimate the amount of muscle damage because this enzyme is muscle specific (12). After the run we found a significant increase in the plasma creatine kinase (91.1 \pm 25 versus 2843 \pm 941 IU·l⁻¹, p < 0.01), suggesting a muscular tissue damage produced during the run.

Several parameters were tested in plasma that reflect the metabolic activity. As shown in table I, at the end of the race plasma lactate (+176 %), urea (+37 %), had increased significantly (p < 0.001 and p < 0.01), while no significant differences were found for the values of plasma glucose. Values below 4 mmol·l⁻¹ for plasma lactate at the end of the 100-km race suggest that the effort performed by the runners can be considered aerobic.

Discussion

The present study shows that plasma Mg^{2+} concentration in well trained runners increased after a 100-km race. The increase in plasma magnesium correlate with the increase in the creatinine levels suggesting that a transitory renal failure could alter the homeostasis of plasma magnesium during long distance races.

Our study contrasts with previously reported data on prolonged heavy exercise. During marathon running a decrease in plasma magnesium has been described (6, 15, 20, 24). In subjects performing a 25 km race no differences in plasma magnesium levels during the race were found (3).

Several mechanisms could be proposed to explain the different results. Depending on the type of exercise the magnesium ion shifts from the erythrocytes which act as a reservoir, to the plasma and from there to the tissues. After prolonged heavy exercise it has been proposed that a significant decrease in magnesium in plasma may occur, probably caused by an exhaustion of the erythrocyte reservoir (7).

The increase in the plasma magnesium found in our study may be due to several mechanisms. It could be due in part to the hemoconcentration which is reflected by the increase in the haematocrit and the decrease in plasma volume.

In our athletes a significant increase in creatine kinase activity was found in plasma after the race. Rises in plasma levels of creatine kinase has been reported in strenuous exercise due to changes in the permeability of the skeletal muscle membranes (18). Such changes are considered as evidence of skeletal tissue damage. Due to the higher intracellular magnesium concentration the increased levels found in plasma may be related to muscular damage.

An increase in the levels of creatinine in plasma that correlates with the concentrations of magnesium has been found. This may be related to a temporary renal failure that has been described during prolonged exercise (4), which could explain the high levels of plasma magnesium. In normal subjects hypermagnesemia is unusual. The principal path of excretion of magnesium is by urinary elimination. Therefore, primary regulatory mechanisms are active in urinary excretion (16). If magnesium increases after a high dietary supply or bone release, the kidney eliminates magnesium excess by reduction of the proximal tubular reabsorption (2, 5). During acute renal failure in oliguric phase a situation of hypermagnesemia is found. However, during the diuretic phase, the levels fell to normal (16).

Recently, in marathon runners a decrease to 50 % urine output and a reduction of the urinary magnesium excretion rate by 83 % was found (15). In long term physical exercise the magnesium loss by the kidney is lower than during rest (10) probably due to a decreased glomerular filtration rate as suggested by the evolution of plasma creatinine levels. The magnesium excretion rate fell on average from 1.83 to 0.65 mg \cdot h⁻¹ (reduction of 64 %) at 2 hours of ergometer exercise at 65 % Vo_{2max} (9). These data support the hypothesis that the increased levels could be directly related to the transitory renal failure that occurs after a long-term run.

Acknowledgements

Supported by National program for Scientific Research and Technological Development, grant DEP233/89 from «Ministerio de Educación y Ciencia» (Spain).

Resumen

La homeostasis del magnesio es crítica durante la realización de ejercicio. Se determina el efecto que producen las carreras de larga dis-

tancia sobre la concentración de magnesio en eritrocitos y plasma en un grupo de 7 corredores aficionados bien entrenados. Después de 100 km de carrera, los niveles de Mg²⁺ plasmático se encuentran aumentados significativamente (p < 0,05), no modificándose significativamente la concentración de Mg²⁺ intraeritrocitario. Los niveles de creatinina en plasma se incrementan significativamente (p < 0,01), sugiriendo un deterioro temporal de la función renal. Se encuentra una correlación positiva significativa entre los niveles de magnesio y creatinina del plasma (r = 0,65, p < 0,01). Estos resultados sugieren que el aumento de los niveles de magnesio en plasma pueden estar relacionados con una insuficiencia renal durante carreras de larga distancia.

Palabras clave: Magnesio, Creatinina, Carrera de larga distancia.

References

- 1. Brautbar, N. and Carpenter, C.: Magnesium, 3, 57-62, 1984.
- Brunette, M., Wen, S. F., Evanson, R. L. and Dirks, J. H.: Am. J. Physiol., 216, 1510-1516, 1969.
- Casoni, L., Guglielmini, C., Graziano, L., Reali, M. G., Mazzotta, D. and Abbasciano, V.: Int. J. Sports. Med., 11, 234-237, 1990.
- Castenfors, J.: Am. N. Y. Acad. Sci., 301, 151-159, 1977.
- 5. Dibona, G. F. and Sawin, L. L.: Magnesium, 1, 104-109, 1980.
- Franz, K. B., Rüddel, H., Todd, G. L., Dorheim, T. A., Buell, J. C. and Eliot, R. S.: J. Am. Coll. Nutr., 4, 187-194, 1985.

- Golf, S. W., Happel, O. and Graef, V.: J. Clin. Chem. Clin. Biochem, 22, 717-721, 1984.
- Gunther, T.: Magnesium Bull, 3, 91-101, 1981.
 Haralambie, G.: In «Metabolic adaptation to prolonged physical exercise». (Howald, H. and Poortmans, J. R., eds.) Birkhäuser-Verlag. Basel, 1975, pp 340-351.
- 10. Heaton, F. and Hodgkinson, A.: Clin. Chim. Acta, 8, 246-254, 1963.
- Hespel, P., Lijnen, P., Fiocchi, R., Lissens, W. and Amery, A.: J. Hypertens, 4, 767-772, 1986.
- 12. Hortobagyi, T. and Denahan, T.: Int. J. Sports Med., 10, 69-80, 1989.
- 13. Joborn, H., Ackerstrom, G. and Ljunghall, S.: Clin. Endocrinol., 23, 219-216, 1985.
- Levine, C. and Coburn, J. W.: N. Engl. J. Med., 19, 1253-1254, 1984.
- Lijnen, P., Hespel, P., Fagard, R., Lysens, R., Vanden Eynde, E. and Amery, A.: *Eur. J. Appl. Physiol.*, 58, 252-256, 1988.
- Massry, S. G.: Ann. Rev. Pharmacol., 17, 67-82, 1977.
- 17. Mordes, J. P. and Walker, W. E. C.: Pharmacol. Rev., 29, 273-300, 1978.
- 18. Noakes, T. D.: Sports Med., 4, 245-267, 1987.
- 19. Reinhart, R. A.: Arch. Intern. Med., 148, 2415-2420, 1988.
- Rose, L. I., Carroll, D. R., Lowe, S. L., Peerson, E. W. and Cooper, K. H.: J. Appl. Physiol., 29, 449-451, 1970.
- Seelig, M. S. and Berger, A. R.: N. Engl. J. Med., 290, 974-975, 1974.
- 22. Strauss, M. B., Davis, R. K., Rosenbaum, J. D. and Rossmeisl, E. C.: Clin. Invest., 30, 862-868, 1951.
- 23. Szadkowska, D., Suhr, D., Lehnert, G. and Schaller, K.: Med. Welt., 21, 821-824, 1970.
- 24. Wolfswinkel, J. M., Van der Walt, W. H. and Van der Linde, A.: S. Afr. J. Med. Sci., 19, 37-38, 1983.