Failure of Magnesium Supplementation to Influence Marathon Running Performance or Recovery in Magnesium-Replete Subjects

Sonja Terblanche, Timothy D. Noakes, Steven C. Dennis, De Wet Marais, and Michael Eckert

This study examined the effect of magnesium supplementation on muscle magnesium content, on running performance during a 42-km marathon footrace, and on muscle damage and the rate of recovery of muscle function following the race. Twenty athletes were divided equally into two matched groups and were studied for 4 weeks before and 6 weeks after a marathon in a double-blind trial; the experimental group received magnesium supplement (365 mg per day) and the control group, placebo. Magnesium supplementation did not increase either muscle or serum magnesium concentrations and had no measurable effect on 42-km marathon running performance. Extra magnesium ingestion also had no influence on the extent of muscle damage or the rate of recovery of muscle function. The latter was significantly reduced immediately after the marathon but returned to normal within 1 week. Thus, magnesium supplementation in magnesium-replete subjects did not enhance performance or increase resistance to muscle damage during the race, or the rate of recovery of muscle function following the race.

Within cells, magnesium cations are present in amounts that are exceeded only by potassium. Most of the intracellular Mg$^{2+}$ is chelated to the negatively charged alpha and beta phosphate groups of ATP to form MgATP$^{2-}$ complexes, the turnover of which is inextricably linked to the rate of muscle contraction (3).

It has been suggested that endurance athletes may become magnesium deficient, either because of increased magnesium losses in sweat (8, 19) or because of increased magnesium requirements due to high habitual daily rates of energy expenditure (6). During intense exercise, a 10% reduction in serum magnesium concentration is typically observed, which is a function of both sweat losses (8, 19) and a redistribution of serum magnesium into the working muscle (8), erythrocytes (18), and adipocytes (11).

S. Terblanche, T.D. Noakes, and S.C. Dennis are with the MRC/UCT Bioenergetics of Exercise Research Unit, University of Cape Town Medical School, Observatory, 7925, Cape Town, South Africa. D.W. Marais is with the Research Institute for Nutritional Diseases, Medical Research Council, Parow, South Africa. M. Eckert is with Madaus Pharmaceuticals (Pty) Ltd, Johannesburg, South Africa.
While there is general agreement that intense exercise decreases serum magnesium concentrations, the physiological consequences of this reduction remain uncertain. In particular there are the questions of whether endurance athletes become magnesium deficient and whether that deficiency impairs their exercise performance. The only evidence to suggest that a magnesium deficiency might decrease endurance comes from studies in rats (14, 15). In humans the data are less convincing. One group has found a positive correlation between plasma magnesium concentration and exercise performance in trained athletes (18), another has observed a negative correlation (33), and a third has reported no correlation (7). Nevertheless, some researchers have suggested that magnesium supplementation could enhance the physical performance of athletes (6, 17). To date, however, there have been no carefully controlled clinical trials to test this possibility.

Accordingly, the aim of this study was to determine the effects of magnesium supplementation on running performance, muscle damage, and recovery of muscle function in a group of athletes competing in a 42-km marathon footrace. In addition we wished to determine whether athletes who do not routinely ingest a magnesium supplement are magnesium deficient, as suggested by Casoni et al. (6), and whether magnesium supplementation increases muscle or serum magnesium concentrations, or both.

**Materials and Methods**

Twenty experienced marathon runners, 16 men and 4 women ranging in age from 25 to 49, volunteered to participate in this double-blind study, the design of which had been approved by the Ethics and Research Committee of the Medical Faculty of the University of Cape Town. The subject selection criteria were that each individual (a) must have completed at least two marathons, (b) be able to run 5 km in less than 25 minutes, and (c) must not have consumed medications or magnesium supplements either immediately before or during the 10-week experimental period.

**Experimental Design**

The 10-week study was conducted over 4 weeks before and 6 weeks after a marathon race. During the 10 weeks the subjects ingested either a placebo or the magnesium supplement, described later. In order to ensure that the athletes were equally motivated to perform to their maximum capability, financial rewards were offered to runners who completed the race within 10 minutes of their previous best marathon times.

*Maximal Treadmill Test.* The athletes were matched by performance in a standardized treadmill protocol into two groups (27). Subjects ran on a Powerjog E30 treadmill (Sport Engineering, Birmingham, England) for 5 minutes at 7.5 km/hr, after which the treadmill speed was increased to 12 km/hr with further incremental increases of 0.5 km/hr every 30 seconds until the subjects could run no faster. Peak running velocity was taken as the highest speed (km/hr) maintained for the entire 30 seconds.

*Magnesium Supplementation.* On the basis of their peak treadmill running velocities, subjects were grouped into pairs and given either a magnesium
supplement (M) or a placebo (P). Both the magnesium and the placebo supplement were packaged in sachets (Madaus Pharmaceuticals [Pty] Ltd), the former containing 1.33 g of magnesium-L-aspartate hydrochloride (equivalent to 122.6 mg elemental magnesium) and the latter containing just L-aspartate hydrochloride. The supplement and placebo were identical in appearance and taste. Subjects were required to consume the contents of one sachet dissolved in approximately 200 ml of water three times a day. The code identifying which subjects were receiving the placebo or a magnesium supplementation, equivalent to the U.S. recommended 300–400 mg daily allowance (20), was held by Madaus Pharmaceuticals (Pty) Ltd and was revealed only after the trial. Despite the well-known cathartic effect of magnesium salts, none of the subjects complained of diarrhea.

**Skeletal Muscle Magnesium Content.** Two skeletal muscle biopsies were taken from the lateral portion of the quadriceps muscle using the conventional percutaneous technique as previously described from this laboratory (26). The first muscle biopsy was performed prior to the beginning of supplementation and the second was done 2 days after the marathon. Muscle biopsy samples were initially frozen in liquid nitrogen and then stored at -70°C for later magnesium determinations. Muscle magnesium content was determined by atomic absorption photometry (Pye Unicam SP9 series; Philips, Cambridge, UK) according to the method of Bergstrom (4).

**Serum Magnesium and Creatine Kinase Measurements.** Serum magnesium concentration and creatine kinase (CK) activity were analyzed in blood samples collected into plastic Vacutainers prior to the start of supplementation, after 4 weeks of supplementation immediately prior to the marathon, 1 hour after the marathon, and on the first 3 days after the race. Serum magnesium concentration was measured by atomic absorption spectrophotometry (4) and serum CK activity was measured with the CK NAC-activated method as previously described (25).

**Urinary Hydroxyproline and Creatinine Concentration.** Since connective tissue breakdown can only be measured from urinary hydroxyproline excretion (1, 2) if hydroxyproline is not ingested, all subjects ate a gelatine-free diet on the 3 days before and on the 3 days after the marathon. The assumption was that a 6-day gelatine-free diet would not alter whole-body magnesium status. For hydroxyproline measurements, early morning urine was collected on the day before and on the 3 days after the marathon. Samples (10 ml) were stored frozen in plastic vials for later hydroxyproline measurements with the Hypronosticon test (Organon Teknika, Breda, Holland) according to the method of Roberts et al. (28).

**Running Performance and Leg Muscle Function.** Three measurements were conducted: a 5-km time trial, isometric muscle function, and muscle tenderness. The subjects took part in two 5-km running time trials, the first prior to magnesium supplementation and the second after 3 weeks of supplementation, 1 week prior to the marathon. Unfortunately, weather conditions were not equal on both days. The second trial was held in wet and windy conditions, which increased the average running times from 21:40 to 22:27 minutes.

**Isometric quadriceps muscle function,** including maximal voluntary contraction (MVC) and fatigue resistance, was measured with a specially designed strain gauge dynamometer according to the techniques described by Bigland-Ritchie et al. (5). Subjects were seated in a fixed, straight-backed chair set at an
angle of 100° with the knee held at an angle slightly greater than 90°. They were strapped across the hips to maintain muscle length and to prevent substantial use of the hip extensors.

The strap from the strain gauge dynamometer was securely anchored just above the lateral malleolus of the ankle. The distance from the lateral epicondyle of the femur to just above the lateral malleolus was measured. This represented the lever arm (cm) and was specific for every subject. The amplified output from the strain gauge was recorded with a rapid response oscillograph. The torque was calculated as the force produced by the quadriceps (Newton) multiplied by the lever arm (m). Each subject’s maximum voluntary contraction values were the average of three 5-sec MVCs separated by 1-min rest intervals.

For the measurement of fatigue resistance, subjects repeatedly maintained a 50% MVC for 6 seconds with a 4-sec rest between contractions. Time to fatigue was defined as the point at which the subject could no longer achieve the displayed 50% MVC target force for a full 6 seconds.

Muscle tenderness was measured as the force (kg) required to elicit tenderness at fixed sites measured with a spring-loaded, round-ended probe (2-cm diameter) specially constructed for the purpose. Test sites were spaced over the quadriceps and marked with indelible ink (10, 22, 23). The subjects were tested before, and daily for 3 days after, the marathon.

**Statistical Analyses**

Group mean values were compared with a Student’s unpaired *t* test (32). Changes over time were analyzed using a one-way repeated-measures analysis of variance (32). *P* values of <0.05 were regarded as statistically significant.

**Results**

The age, mass, height, peak treadmill running velocity, and marathon racing times of the magnesium supplement (M) and placebo (P) groups are listed in Table 1. There were no significant differences in any of these variables between

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Anthropometric Data and Peak Treadmill Running Velocity for the Magnesium Supplementation and Placebo Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>Mass (kg)</td>
</tr>
<tr>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Magnesium</td>
<td>32.4</td>
</tr>
<tr>
<td>Placebo</td>
<td>32.5</td>
</tr>
</tbody>
</table>
Figure 1 — Skeletal muscle magnesium concentrations in two matched groups of runners receiving either placebo or magnesium before and after a 42-km marathon footrace. The concentrations were not influenced by magnesium supplementation.

Figure 2 — Serum magnesium concentrations before and for 72 hours after a 42-km marathon footrace in two matched groups of runners receiving placebo or magnesium supplementation. The concentrations were not influenced by magnesium supplementation.

the groups. Skeletal muscle magnesium concentrations in the two groups before and after the marathon are summarized in Figure 1. Again there were no differences either before or after the marathon; nor did magnesium supplementation alter muscle magnesium concentration.

Serum magnesium concentrations prior to supplementation and for the 3
days after the marathon in both groups are presented in Figure 2. The concentrations were not different at any time between groups, nor did they alter with supplementation. However, serum magnesium concentrations did fall significantly in both groups immediately after the race, and here the between-group differences were apparent. Whereas the postrace serum magnesium concentrations returned to prerace values within 24 hours in the supplemented group, in the placebo group they only returned to control values 48 hours after the race.

As expected, serum CK activity rose significantly in both groups after the marathon, but there was no significant difference between groups at any time (Figure 3). Peak serum CK activities occurred after 24 hours and then fell over the next 2 days to values that were still significantly elevated above resting levels. Although there was no correlation between racing time and serum CK activity, a significant correlation ($r=0.56, p<0.01$) was found between serum CK activity and the time difference between the previous best marathon time and the finishing time in this marathon. Thus, runners who ran closest to their previous best marathon times had the highest peak serum CK activities.

Changes in the urinary hydroxyproline/creatinine ratios in both groups on the day before and for 3 days after the race are shown in Figure 4. There were no differences between groups. Urinary hydroxyproline/creatinine ratios rose for the first 2 days after the marathon but returned to prerace values within 72 hours after the marathon. Peak hydroxyproline/creatinine ratio correlated positively ($r=0.53, p<0.05$) with the duration of exercise (marathon time). The time course of the changes in maximum voluntary contraction (MVC) for the two groups from 2 weeks before to 5 weeks after the marathon is presented in Figure 5. MVC fell significantly immediately after the race but thereafter it increased. This increase
Figure 4 — Urinary hydroxyproline/creatinine ratios 24 hrs before and for 72 hrs after a 42-km marathon footrace in two matched groups of runners receiving either placebo or magnesium supplementation. There were no differences at any time between groups.

Figure 5 — Maximum voluntary contraction (MVC) measured weekly for 2 wks before and for 5 wks after a 42-km marathon footrace in two matched groups of marathon runners receiving either placebo or magnesium supplementation. Although MVC first fell and then rose significantly in both groups after the marathon, there was no difference at any time between groups.
Magnesium Supplementation and Marathon Running

exceeded prerace values at 2 weeks and became significant by 5 weeks after the marathon ($p<0.05$). However, there were no significant between-group differences in MVC and time to fatigue before and after the race.

In both groups, muscle soreness peaked 48 hours after the race (Figure 6). Again there was no significant difference between groups nor any interaction effect over time. The peak soreness ratings at 48 hours after the race coincided with peak changes in the hydroxyproline/creatinine ratios (Figure 4) but not with peak serum CK activities (Figure 3).

**Discussion**

The possibility that magnesium supplementation may aid athletic performance is frequently claimed in the lay literature; yet the scientific evidence for such an effect is more tenuous (19). In this double-blind, placebo-controlled trial we found that neither muscle (Figure 1) nor serum (Figure 2) magnesium concentrations were increased by magnesium supplementation in a group of trained marathon runners. This failure of magnesium supplementation to raise circulating magnesium concentrations in subjects who were not deficient in magnesium is not surprising. Unlike most minerals which are primarily excreted in urine, excess dietary magnesium is not absorbed from the intestine and that is why large doses of magnesium have a cathartic effect.

The only effect of magnesium supplementation was that it increased the rate of return to prerace levels of serum magnesium following the race (Figure 2), but this relatively minor effect is unlikely to be of any major physiological benefit because muscle magnesium contents were unaffected by either the race.
or magnesium supplementation. That serum magnesium concentrations fall after exercise, including marathon racing, is well established (11).

Since muscle magnesium concentrations were not increased by magnesium ingestion, it is perhaps not surprising that neither marathon running performance (Table 1), nor the extent of muscle damage (Figure 3), nor the rate of recovery of muscle function was influenced by magnesium supplementation (Figure 5). Whether higher doses of magnesium or the presence of magnesium deficiency would produce a different result is not answered by this study. However, the finding that muscle and serum magnesium concentrations in these subjects were within the normal range (9, 16, 30, 31) in both the supplemented and nonsupplemented groups argues against the concept that most athletes eating an adequate diet may become magnesium deficient because of large magnesium losses during exercise (6, 19). Accordingly, we conclude that for magnesium-replete athletes, magnesium supplementation produces no significant advantage in terms of muscle performance.

Other interesting findings of this study were the fall in MVC after the marathon with full recovery within 7 days (Figure 5). This is consistent with the studies of Sherman et al. (29) which showed that both peak quadriceps muscle isokinetic torque and isokinetic work capacity were reduced for up to 7 days after a similar marathon footrace. We also found that peak serum CK activities occurred at 24 hours postrace, as expected (24), whereas peak ratings of muscle soreness (Figure 6) and peak hydroxyproline/creatinine ratios (Figure 4) were only reached 48 hours after the race. This finding is in agreement with those of Abraham (1, 2), who also showed peak urinary hydroxyproline excretion on the day that subjects reported their greatest postrace muscle soreness. This suggests that postexercise muscle soreness may be related to connective tissue damage, as has also been proposed by others (1, 2, 12, 13, 21, 22, 23).

Interestingly, there was a poor correlation between the exercise intensity and the peak postrace urinary hydroxyproline/creatinine ratio \((r=0.2)\), but there was a significant correlation between the marathon time and the peak postrace urinary hydroxyproline/creatinine ratio \((r=0.53; p<0.02)\). This suggests that connective tissue damage could be related to the duration of exercise rather than to its intensity, as was also found for changes in serum CK activity (25). In summary, magnesium supplementation did not increase either serum or muscle magnesium concentrations in trained marathon runners. Magnesium concentrations in these runners did not differ from those measured in a control group receiving the placebo. Accordingly, we conclude that not all marathon runners are necessarily magnesium deficient. In addition, magnesium supplementation did not alter muscle function or running performance, or the extent of muscle damage as measured with changes in serum CK activity, urinary hydroxyproline/creatinine ratios, and muscle soreness. Neither did magnesium supplementation increase the rate of recovery of muscle function. Whether these findings apply to higher magnesium doses in exercising populations at risk of magnesium deficiency is not addressed by this study but is worthy of further investigation.

**References**


**Acknowledgments**

This research was funded by Madaus Pharmaceuticals (Pty) Ltd with additional support from the Medical Research Council and the Nellie Atkinson and Harry Crossley Staff Research Funds of the University of Cape Town.