

Comment

## IS PHYSICAL WORKING CAPACITY DETERMINED BY OPTIMAL MAGNESIUM CONCENTRATION?

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### ABSTRACT

Two lines of research originated in the mid-seventies; one attempted to gauge the effect of strenuous effort on serum magnesium concentration (S-Mg) and the second, the effect of S-Mg on the indices of physical working capacity.

In apparently healthy trained young Israeli men, long-term studies of the effect of strenuous effort on S-Mg showed that after a moderately strenuous effort (70 km forced march), there was a decrease of S-Mg which became statistically significant after 72 h, whereas after a severely strenuous effort (120 km forced march), the decrease was biphasic, being significant after 1 h, with a second fall after 72 h. In repeated experiments, the decrease of S-Mg was found to persist over time /1/. Since chronic lowering of S-Mg signifies a concomitant lowering of intracellular Mg content, these findings showed that Mg deficiency was a sequel to strenuous effort.

Studies of the effect of S-Mg on the indices of physical working capacity showed that: (1) S-Mg determines the maximal voluntary muscle contraction force (MVC); (2) S-Mg affects, among other things, the maximal aerobic capacity ( $VO_2\max$ ).

The estimated daily Mg intake in the Israeli population is inadequate and a widely prevalent marginal to overt Mg deficiency is found in apparently healthy Israelis.

It is proposed that an optimal physical working capacity in the Israeli population will be achieved only under conditions of Mg saturation of metabolism. Studies to validate this hypothesis are under progress.

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**KEY WORDS**

serum magnesium, physical working capacity

**INTRODUCTION**

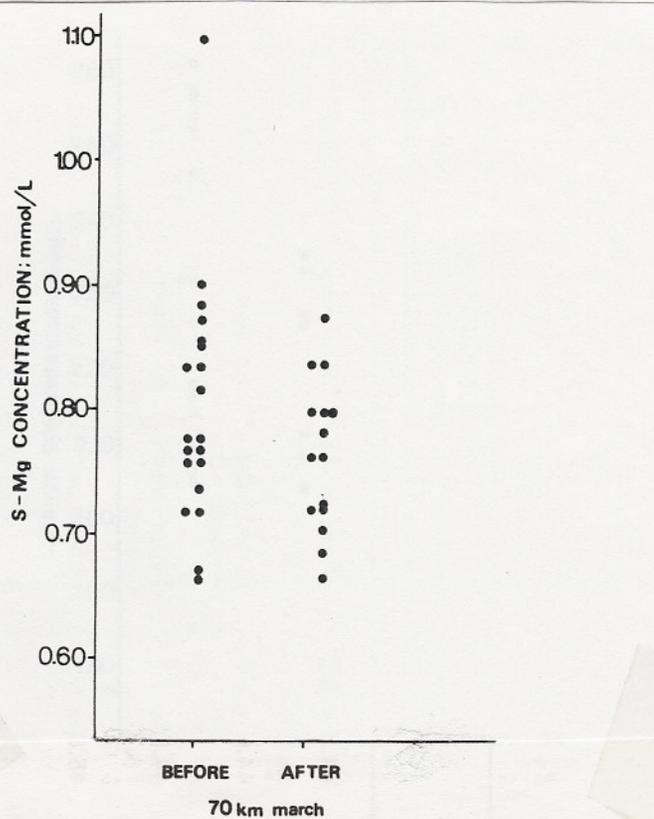
Two lines of investigation have been pursued since the mid-seventies. One tried to gauge the effect of strenuous effort on serum magnesium concentration (S-Mg) and the other, the influence of S-Mg levels on the maximal voluntary muscle contraction force (MVC) and other indices of physical working capacity.

**THE EFFECT OF STRENUOUS EFFORT ON S-Mg**

A transient fall of S-Mg has been reported following marathon running by Rose *et al.* /2/, Cohen and Zimmerman /3/, Linjen *et al.* /4/ and Casoni *et al.* /5/; after competitive skiing by Refsum *et al.* /6/; and after treadmill exercise in thermoneutral and a hot, dry environment by Beller *et al.* /7/.

**S-Mg FOLLOWING STRENUOUS EFFORT IN APPARENTLY HEALTHY TRAINED ISRAELIS**

Contrary to other reports, we found in our early studies no significant immediate decrease of S-Mg on termination of moderately severe effort, i.e. 40 km and 70 km forced marches, in apparently healthy Israeli trained young men (Fig. 1). However, we did find a significant decrease of S-Mg 1 h after very strenuous exertion, i.e. a 120 km forced march. Subsequently, we extended the observation time and carried out serial measurements in four groups of Israeli trained young volunteers after the termination of strenuous effort; to our knowledge this has not been done before. As a model of strenuous effort we used brisk 120 and 70 km marches /1, 8, 9/. The age, weight and mean maximal aerobic capacity ( $VO_2max$ ) of the volunteers, who gave informed consent, are given in Table 1. The climatic conditions were: ambient temperature 20-27°C; relative humidity 50-75%. S-Mg was estimated in an atomic absorption spectrophotometer as described previously /10/.



**Fig. 1:** S-Mg concentration before and immediately after a 70 km forced hike. Initial mean  $0.803 \pm 0.10$ ; after  $0.764 \pm 0.06$ ;  $n = 15$ ;  $p \approx 0.05$ .

The estimated daily Mg intake during the 70 km march was 270 mg from food and 226 mg from water (8 l at 28.2 mg/l), and during the 120 km march studies the estimated intake was 340 mg from food and 361 mg from water (13 l at 28.2 mg/l) daily /11/.

After the 70 km march we found a delayed, highly significant fall of mean S-Mg (Figs. 1, 2), which first occurred 3 days after termination of the effort, when 95% of the probands were hypomagnesaemic, a phenomenon which had not been previously reported. The decrease of S-Mg persisted on reexamination 18 days later /9/. After more intense effort, a 120 km march, we found there was an immediate significant fall of mean S-Mg, followed by a second, more profound, decrease occurring at 72 h (Fig. 3) when 89% of the probands

TABLE 1

	$\bar{X}$ age $\pm$ SD, n	Weight, kg	VO <sub>2</sub> max ml. kg <sup>-1</sup> .min <sup>-1</sup>	Type of effort	Observation time after termination of strenuous effort	Year of Publication
Study 1	19.5 $\pm$ 0.5, n=20	74.9 $\pm$ 6.6	44.5 $\pm$ 6.2	120 km forced march	3 months	1987
Study 2	19.3 $\pm$ 1.4, n=20	68.5 $\pm$ 6.2	44.0 $\pm$ 4.0	70 km forced march	18 days	1989
Study 3	18.6 $\pm$ 1.3, n=16	66.3 $\pm$ 7.2	44.6 $\pm$ 5.1	120 km forced march	10 months	1991
Study 4	18.7 $\pm$ 0.5, n=15	75.0 $\pm$ 6.6	45.8 $\pm$ 8.2	120 km forced march	15 months	1991

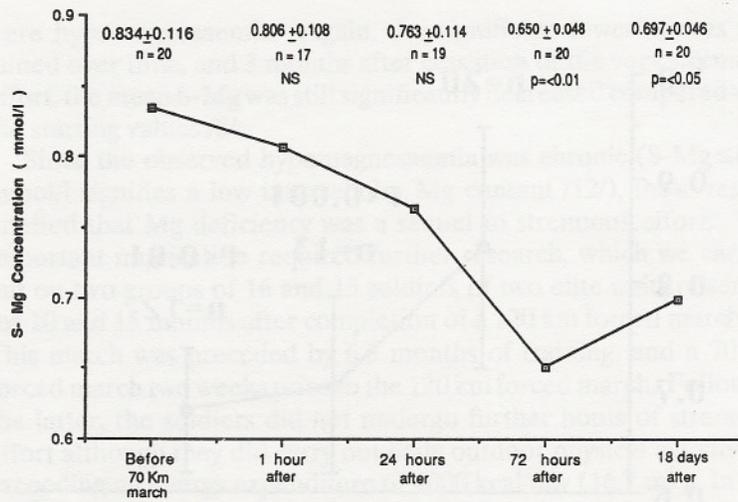


Fig. 2: S-Mg concentration before and up to 18 days after a 70 km forced hike.

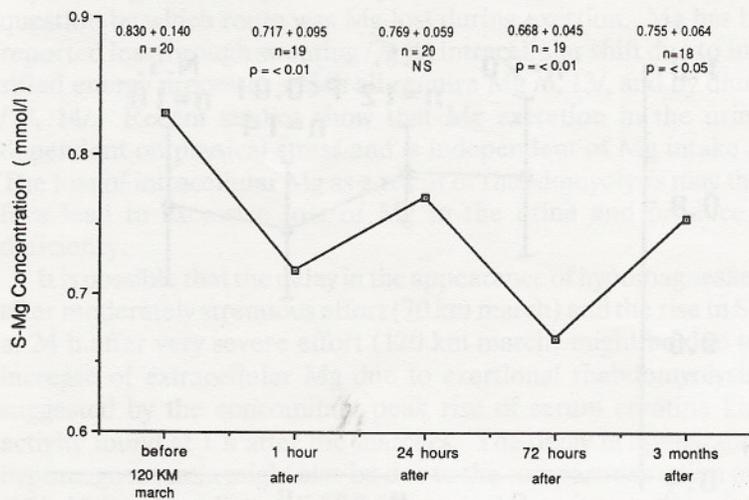


Fig. 3: S-Mg concentration before and up to 3 months after a 120 km forced hike.

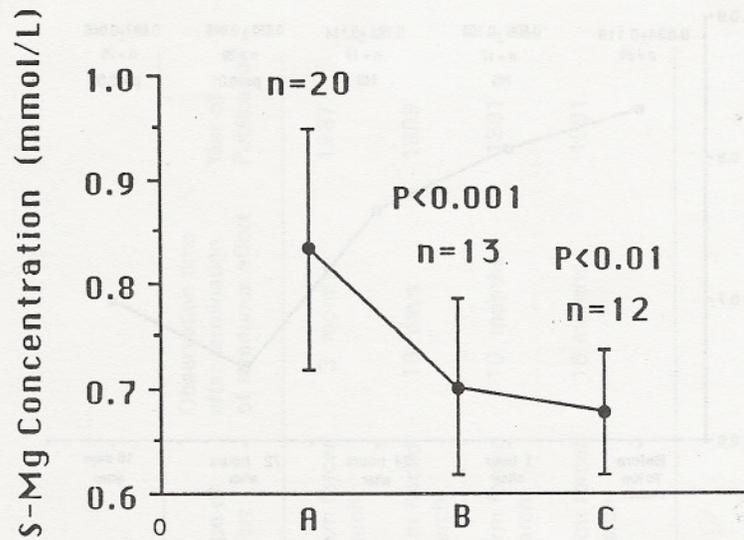


Fig. 4: S-Mg concentration 6 (B) and 10 (C) months after a 120 km forced march, compared with the baseline value (A).

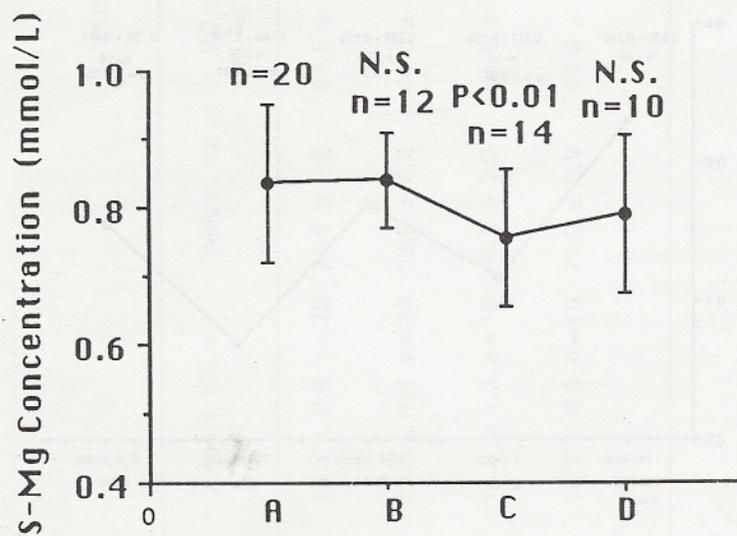


Fig. 5: S-Mg concentration before (A) and 9 (B), 11 (C) and 15 (D) months after a 120 km force march.

were hypomagnesaemic. Again, the significant lowering was sustained over time, and 3 months after cessation of the very strenuous effort, the mean S-Mg was still significantly decreased compared with the starting values /8/.

Since the observed hypomagnesaemia was chronic (S-Mg  $\leq 0.73$  mmol/l signifies a low intracellular Mg content /12/), these results implied that Mg deficiency was a sequel to strenuous effort. This important implication required further research, which we carried out on two groups of 16 and 15 soldiers of two elite units observed for 10 and 15 months after completion of a 120 km forced march /1/. This march was preceded by 6.5 months of training, and a 70 km forced march two weeks prior to the 120 km forced march. Following the latter, the soldiers did not undergo further bouts of strenuous effort although they did carry out daily outdoor physical activity not exceeding an energy expenditure of 4000 kcal/day (16.7 mJ). In one group, the decrease of mean S-Mg was apparent 6 months after the march, the mean still remaining below the reference interval after 10 months (Fig. 4), while in the other group the decrease of the mean S-Mg was still apparent 11 months after the 120 km march (Fig. 5).

The long-term decrease of S-Mg shown by these studies, which imply a Mg deficiency as a sequel of strenuous effort, posed the question by which route was Mg lost during exertion. Mg has been reported lost through sweating /7/, by intracellular shift due to intensified energy processes which all require Mg /6, 13/, and by diuresis /13, 14/. Recent studies show that Mg excretion in the urine is dependent on physical stress and is independent of Mg intake /15/. The loss of intracellular Mg as a result of rhabdomyolysis may therefore lead to excessive loss of Mg in the urine and produce Mg deficiency.

It is possible that the delay in the appearance of hypomagnesaemia after moderately strenuous effort (70 km march) and the rise in S-Mg at 24 h after very severe effort (120 km march) might be due to an increase of extracellular Mg due to exertional rhabdomyolysis, as suggested by the concomitant peak rise of serum creatine kinase activity found at 1 h after the marches. The delay in appearance of hypomagnesaemia might also be due to the compartmentation of Mg /16/. If the immediate fall of S-Mg were due to intracellular influx, perhaps only the more intense bouts of exertion would be able to cause such a shift, considering how important is the maintenance of a steady state of S-Mg (i.e. Mg saturation of metabolism) /11, 17/.

This could explain why the immediate decrease was seen only after the forced 120 km march and not after more moderate exertion. Further studies to identify the routes of Mg loss in exertion more precisely are needed.

#### THE EFFECT OF S-Mg ON VOLUNTARY MUSCLE CONTRACTION FORCE AND OTHER INDICES OF PHYSICAL WORKING CAPACITY

Patients with chronic hypomagnesaemia, i.e. serum magnesium concentration (S-Mg) of  $< 0.73$  mmol/l, were found to have a significantly reduced mean maximal voluntary muscle contraction force (MVC) of the quadriceps femoris muscle, compared with normomagnesaemic subjects, while their muscle-Mg content and the intracellular content of high energy phosphates (measured using muscle tissue biopsy of the quadriceps femoris muscle) were significantly lowered compared with apparently healthy controls /12/. The suggested association of S-Mg and MVC was investigated by screening 106 subjects (93 patients recruited from diagnostic groups in which hypomagnesaemia has been reported, and 13 apparently healthy controls) for MVC, anthropometric data, degree of training and 19 laboratory variables: a total of 60 variables. The following variables were significantly associated with MVC: Age, Weight, Height, Length of tibia, Sex; four laboratory variables: S-Mg, S-sodium, B-standard bicarbonate, and S-iron; and the dummy variables: Moderate degree of training, and two diagnostic groups /18/.

When these potentially explanatory factors were simultaneously considered, by means of a Ridge regression analysis /19/, only three factors proved statistically significant: Weight, Age and S-Mg. The results showed that the higher the S-Mg within the range measured (which was 0.43 - 0.91 mmol/l), the greater the MVC, and inversely, the lower the S-Mg, the lower the MVC. Since MVC constitutes one of the indices of physical working capacity, the important conclusion of this study was that in addition to Weight and Age, S-Mg was a determinant of physical working capacity.

Simultaneously, evidence from experimental data of several researchers showed the importance of Mg for sarcoplasmic reticulum Ca transport in skeletal muscle /20/, and indicated a possible regulatory role for Mg in skeletal muscle excitation-contraction coupling /21-24/. Borikov and Ledvitsky /25/ assume that Mg binding to myosin and phosphorylation of light chain 2, associated with myosin

heads, produce structural changes in myosin filaments of muscle fibres. This causes change in the orientation of myosin heads and the conformation of myosin rods. Dulhunty and Gage /26/ reported that Mg activated, as well as inactivated, tension in small bundles of rat soleus muscle, and this effect could not be explained by a change in surface charge.

Plasma Mg was significantly correlated with  $VO_2$ max among athletes; in untrained males, there was a weak association /27/. In young swimmers  $VO_2$ max was positively associated with S-Mg in young males, but not in the young females /28/. Rayssiguier *et al.* /29/ suggest that the association found in athletes between S-Mg and  $VO_2$ max, which depends on the delivery of oxygen to the working muscle, may represent a cellular adaptation of Mg metabolism to physical training. Adequate S-Mg may facilitate oxygen delivery to the working muscle by aiding erythrocytic production of 2,3-diphosphoglycerate /27/. Evidence that Mg deficiency can result in a significant reduction of physical performance has recently been reviewed by McDonald and Keen /30/ and Rayssiguier *et al.* /29/.

#### Mg SUPPLEMENTATION AND PHYSICAL PERFORMANCE

Ahlborg *et al.* /31/ were the first to test the effect of Mg administration on physical capacity in apparently healthy males. Oral potassium magnesium aspartate was administered during one day prior to and during a 4-day long test consisting of exercise training to exhaustion each day. In the Mg treated subjects there was an increase of nearly 50% in the volunteers' capacity for prolonged exercise compared with placebo treated controls. Sen Gupta and Srivastava /32/ confirmed these findings, although they found only a 23% increase in endurance. Wodick and Grunert-Fuchs /33/ showed that a 4-week Mg supplementation increased physical performance as measured by means of  $VO_2$ max and bicycle ergometry. Golf *et al.* /34/ reported on ergometer test, after a four-week long Mg-aspartate-hydrochloride supplementation in 14 rowers, a decrease in maximal ventilation and total oxygen uptake as well as a decrease of  $VO_2$ max by 11%, and a decrease of the oxygen content of whole blood. Lactate elimination rate from plasma was accelerated as well. However, they found no change in work capacity. Rasmussen *et al.* /35/ also did not find any difference in maximal working capacity, in 9 healthy volunteers given

a 10 mmol infusion of  $MgCl_2$ , i.v., compared with placebo treated controls. In submaximal effort, Ripari *et al.* /36/ reported that supplementation with Mg pidolate improved cardiorespiratory performance.

The results quoted vary, probably due to the different doses and duration of supplementation and the different Mg vehicles used. It is not widely realized that it may take a very long time before Mg deficit is abolished and a steady state is achieved by means of Mg substitution. It is proposed that only after saturation of Mg metabolism has been achieved /37/ and an optimal Mg vehicle defined, will a final assessment of the effect of Mg substitution on physical performance become possible.

#### PUBLIC HEALTH IMPLICATIONS OF THESE FINDINGS

It is important to stress that the total daily intake of Mg in food and water of 496 subjects, namely 707 mg /1, 8, 9/, during strenuous effort was not enough to offset the appearance of Mg deficiency as a sequel to exertion.

In Israel the average daily intake of Mg in the diet is low, as in other industrialized countries: 200-300 mg daily /11/. The majority of researchers /38/ recommend much higher daily requirements, e.g. for adolescent men: 10-16 mg/kg/day. It is therefore apparent that large sections of the Israeli population who engage in strenuous effort will be at risk of developing Mg deficiency. Such a deficiency has a deleterious effect on health, since Mg is essential for energy processes, for catabolism and anabolism of carbohydrates, fats, proteins and nucleic acids, for neurotransmission, receptor binding and cell division /17, 39/.

Dramatic changes are seen within the cell in Mg deficiency, both in experimental animals /40, 41/ and in man /12/. There is an increase in interstitial  $H_2O$ , in intracellular  $Na^+$ ,  $Ca^{++}$ ,  $Cl^-$  and cAMP, and a lowering of  $Mg^{++}$ ,  $K^+$ ,  $PO_4^{--}$  and the energy-rich compounds: ATP, ADP and creatine phosphate. Cronin /41/ rightly defines a magnesium-deficient cell as a "sick cell".

The importance of Mg for energy processes and several indices of physical working capacity has been documented. Thus in addition to the threat posed by Mg deficiency to health, it constitutes also a threat to physical capacity, even in the presence of a moderate or a marginal

deficiency. The resultant lowering of physical working capacity may have broad socioeconomic implications. Some studies to date suggest that physical performance may improve as a result of Mg replenishment.

#### CONCLUSION

The available evidence suggest the hypothesis that the individual, as well as the national, level of physical performance will improve significantly once the widely prevalent marginal or overt Mg deficiency in apparently healthy Israelis is adequately treated. Studies to prove this hypothesis are being carried out.

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