Magnesium Research (1991), **4**, 1, 59–65 ORIGINAL ARTICLE

Long term effects of peak strenuous effort on serum magnesium, lipids, and blood sugar in apparently healthy young men

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Summary: Earlier findings showed a sustained lowering of serum magnesium concentration (S-Mg) which indicated the presence of Mg deficit, and a parallel, delayed rise of blood sugar and serum lipids as a sequel to strenuous effort. S-Mg was still significantly decreased 3 months after termination of peak effort. To gain further perspective, we followed the biochemical sequels of exertion over an extended period of observation, while maintaining the same experimental conditions used earlier, which mimicked those employed in the training of military recruits. We examined two groups of military recruits, n = 15 (group 1), n = 16 (group 2), mean age 18.6, SD 1.3 and 18.7, SD 0.6, years respectively, who underwent a graded training programme of 7 months' duration culminating in a 120 km forced march. Blood was sampled for estimation of S-Mg in 20 soldiers on recruitment, 6 and 10 months after the 120 km march in group 1, and 9, 11 and 15 months after the march in group 2. Blood sugar and serum lipids were screened on recruitment and up to 11 months after the 120 km march. A significant lowering of mean S-Mg was found as late as 10 months after completion of the march in group 1, and 11 months in group 2 ($P < 0.01$). Mean serum cholesterol and triglycerides showed a delayed rise, especially in group 2 ($P < 0.05$ and $P < 0.001$, respectively), whereas blood sugar decreased in group 1, but increased in group 2 ($P < 0.01$).

These results confirm that strenuous effort gives rise to prolonged Mg deficit and for the first time show the presence of concomitant long term increase of serum lipids following exertion.

Key words: Strenuous effort, magnesium deficit, lipid increase.

Introduction

In our previous studies of young trained apparently healthy Israeli volunteers after the termination of strenuous effort, we found a delayed and sustained fall in serum magnesium concentration (S-Mg)[1,2]. Since a prolonged decrease in S-Mg is accompanied by a lowering of intracellular Mg content[3], the finding signified the presence of intracellular Mg deficit. In parallel, we saw a delayed rise in blood sugar, serum triglycerides and serum cholesterol as late as 18 days after the cessation of effort[1,2].

The significant decrease in S-Mg was found to persist as long as 3 months after the termination of a forced 120 km march which constituted a culmination of a 7 months' long graded outdoor training programme modelled on that used to train military recruits.

In order to investigate the extent of the carry-over effect of exertion on S-Mg, we wished now to extend the observation time beyond 3 months after a 120 km march, in subjects matched for age, sex, and diet, and trained under the same experimental conditions. For this purpose, we examined two groups of military recruits at 6 and 9 months (group 1), and 9, 11 and 15 months (group 2) after the completion of a 120 km forced march.

In view of our previous results[1,2] and due to the importance of Mg for carbohydrate metabolism[4–6], we were interested in following blood sugar and serum lipid levels in addition to S-Mg after cessation of peak strenuous effort, for a longer period of time than in our

earlier studies. Measurements of blood sugar and lipid levels were therefore also made in the two groups of recruits, up to 10 and 11 months respectively after the termination of the 120 km march.

Material and methods

All recruits gave informed consent. Group 1 consisted of 16 recruits, mean age 18.6, SD 1.3, years; weight 66.3, SD 7.2, kg; mean maximal aerobic capacity ($\dot{V}O_2$ max), measured according to Astrand[7], 44.6, SD 5.1, $ml \cdot kg^{-1} \cdot min^{-1}$. Group 2 consisted of 15 recruits, mean age 18.7, SD 0.5, years; weight 75.0, SD 6.6, kg; $\dot{V}O_2$ max 43.7, SD 7.7, $ml \cdot kg^{-1} \cdot min^{-1}$ (Tables 1 and 2).

Blood was collected into polypropylene test tubes between 08.00 and 10.00 hours, using brief stasis and after an overnight fast, for S-Mg, serum cholesterol, triglycerides, and blood sugar, as well as for routine laboratory screening. S-Mg was estimated soon after sampling in a Perkin Elmer atomic absorption spectrophotometer No 305A, according to Stendig-Lindberg *et al.*[8] 6 and 10 months (group 1), and 9, 11 and 15 months (group 2) after the 120 km march.

Blood sugar, serum cholesterol and serum triglycerides were examined on recruitment and at 6 and 10 months (group 1) and 6, 9 and 11 months (group 2) after the march, using an autoanalyser.

Standardized diet was consumed throughout containing 270–340 mg food Mg content daily.

Following the 120 km march, no further bouts of strenuous effort took place; however, the soldiers continued to carry out daily physical activity. Total energy expenditure was about 4000 kcal/d (16.7 mJ).

Means and standard deviations were computed for age, $\dot{V}O_2$ max and laboratory variables. Independent *t* test was used to compare the mean S-Mg with that of 20 age- and sex-matched Israeli soldiers (age 19.3, SD 1.4, years) examined at the same laboratory on recruitment, and also to compare the means of blood sugar, serum lipids, and $\dot{V}O_2$ max before and after the peak strenuous effort. In this control group (n = 20), S-Mg was 0.834, SD 0.166, $mmol \cdot litre^{-1}$.

Results

Group 1

The mean S-Mg was significantly lower 6 and 10 months after the 120 km forced march than in the untrained, matched controls ($P \leq 0.01$). By 6 months after the march, mean S-Mg had already fallen below the lower limit of the reference interval (0.73–0.96 $mmol \cdot litre^{-1}$), to 0.701, SD 0.083, $mmol \cdot litre^{-1}$, n = 13 ($P \leq 0.01$), and it decreased further 10 months after the march, to 0.677, SD 0.060, $mmol \cdot litre^{-1}$, n = 12, $P \leq 0.01$), see Fig. 1. In parallel with this fall, there was a gradual, although non-significant, rise of mean serum cholesterol (Fig. 2).

Mean serum triglycerides, after an initial significant fall ($P \leq 0.05$), rose between 6 and 10 months after the 120 km march, to reach a mean value 7% higher than the baseline value ($P \geq 0.05$) (Fig. 3).

The mean blood sugar fell significantly 6 months after the 120 km march ($P \leq 0.001$) and remained

Table 1. *Age, weight and $\dot{V}O_2$max of subjects in group 1. Values are means ± SD.*

Subjects (n = 16)	Prior to the 7 months of training	Prior to the 120 km march	6 months after	10 months after
Age (years)	18.6 ± 1.3	19.5 ± 0.7	19.9 ± 0.9	20.1 ± 0.7
Wt (kg)	66.3 ± 7.2	68.6 ± 5.8	72.9 ± 6.1**	70.5 ± 5.7*
$\dot{V}O_2$max ($ml \cdot kg^{-1} \cdot min^{-1}$)	44.6 ± 5.1	44.6 ± 5.4	44.0 ± 8.2	44.2 ± 6.7

*$P < 0.05$; **$P < 0.01$ *vs* initial value.

Table 2. *Age, weight and $\dot{V}O_2$max of subjects in group 2. Values are means ± SD.*

Subjects (n = 15)	Prior to the 7 months of training	Prior to the 120 km march	6 months after	9 months after	11 months after
Age (years)	18.7 ± 0.5	19.3 ± 0.5	19.7 ± 0.5	20.4 ± 0.5	20.1 ± 0.7
Wt (kg)	75.0 ± 6.6	75.3 ± 8.1	77.3 ± 7.3	75.5 ± 6.6	77.3 ± 6.7
$\dot{V}O_2$max ($ml \cdot kg^{-1} \cdot min^{-1}$)	43.7 ± 7.7	45.8 ± 8.2	47.0 ± 7.1	41.7 ± 7.0	44.7 ± 6.7

There were no significant differences from initial values.

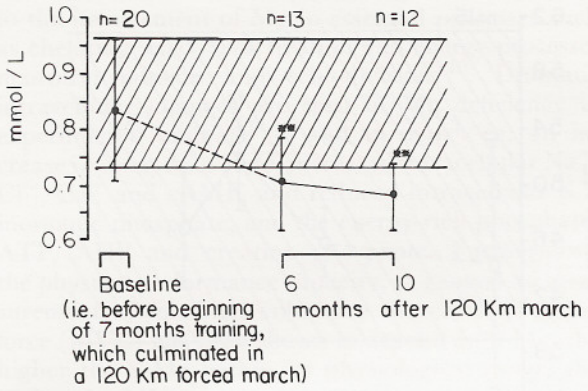

Fig. 1. Serum magnesium concentrations in group 1. Values are means, bars = SD. Shaded area marks the reference interval.
$^{**}P < 0.01$.

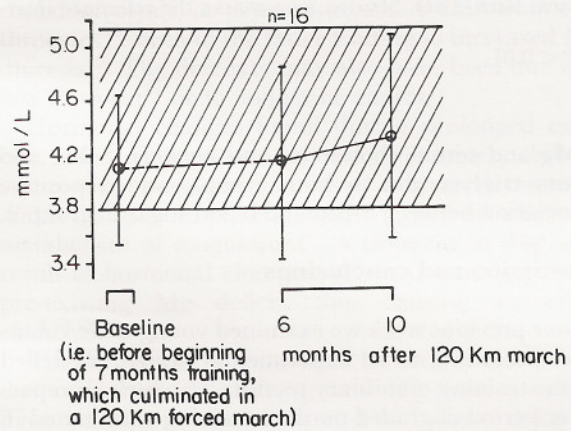

Fig. 2. Serum cholesterol concentrations in group 1. Values are means, bars = SD. Shaded area marks the reference interval.

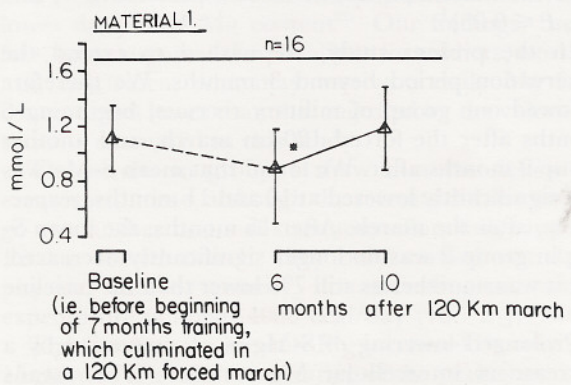

Fig. 3. Serum triglyceride concentrations in group 1. Values are means, bars = SD. The upper line marks upper order of reference interval.
$^{*}P < 0.05$.

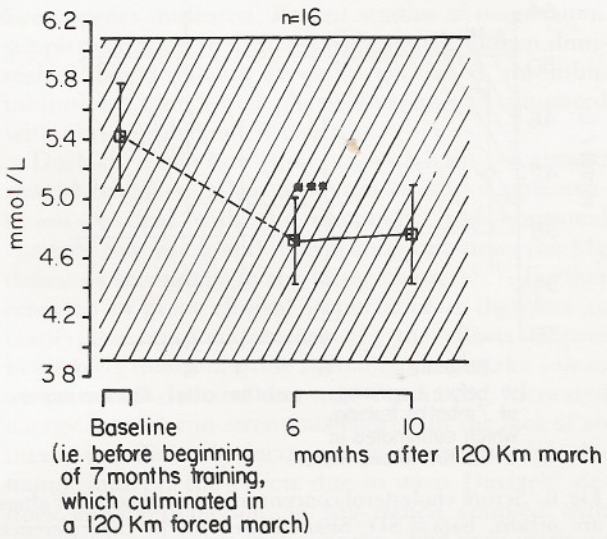

Fig. 4. Blood sugar concentrations in group 1. Values are means, bars = SD. Shaded area marks the reference interval.
$^{***}P < 0.001$.

decreased 10 months after, although by this time it was no longer significantly different from the baseline value (Fig. 4).

Group 2

The mean S-Mg showed virtually no change 9 months after the 120 km march, but by 11 months it decreased significantly, to 0.754, SD 0.998, mmol·litre^{-1}, n = 14, $P \leq 0.01$. At a follow-up 15 months after the march, the mean S-Mg showed a slight (3%) rise, yet it still remained 7% below the baseline value, although this difference was not significant (Fig. 5).

Mean serum cholesterol was initially only slightly

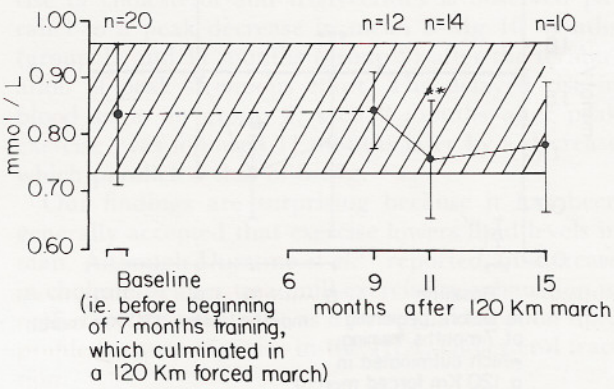

Fig. 5. Serum magnesium concentrations in group 2. Values are means, bars = SD. Shaded area marks the reference interval.
$^{**}P < 0.01$.

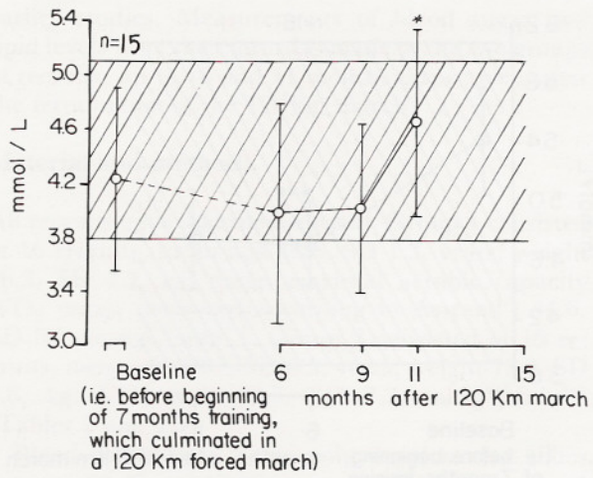

Fig. 6. Serum cholesterol concentrations in group 2. Values are means, bars = SD. Shaded area marks the reference interval.
*$P < 0.05$.

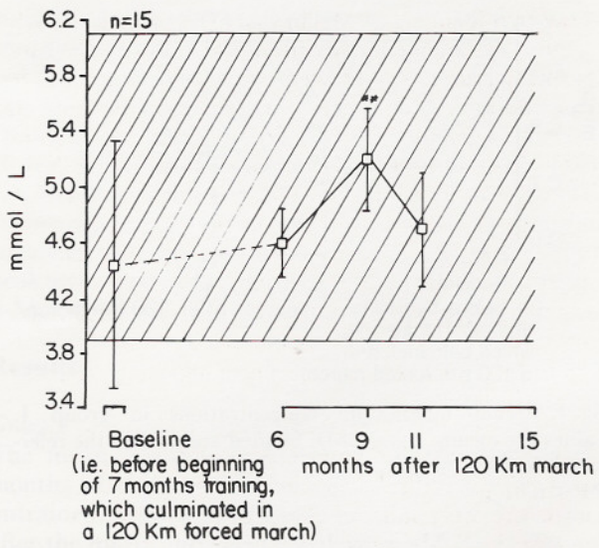

Fig. 8. Blood sugar concentrations in group 2. Values are means, bars = SD. Shaded area marks the reference interval.
**$P < 0.01$.

decreased (NS), and remained virtually unchanged between 6 and 9 months, but by 11 months after the march it had increased by 14% ($P < 0.05$) (Fig. 6).

Mean serum triglycerides rose significantly 6 months after the march ($P \leq 0.001$), decreased 9 months after ($P \leq 0.001$), and increased again significantly 11 months after, by 42% ($P \leq 0.001$), reaching the upper limit of the reference interval for the age group examined (1.70 mmol·litre^{-1}), see Fig. 7.

Mean blood sugar was only slightly raised at 6 months (NS), but at 9 months it had risen by 12% ($P \leq 0.01$); it then declined by 10% 11 months after the march, paralleling the fall in S-Mg (Fig. 8).

The results suggest a negative association between S-Mg and serum cholesterol and between S-Mg and serum triglycerides in both groups, and a positive association between S-Mg and blood sugar in group 2.

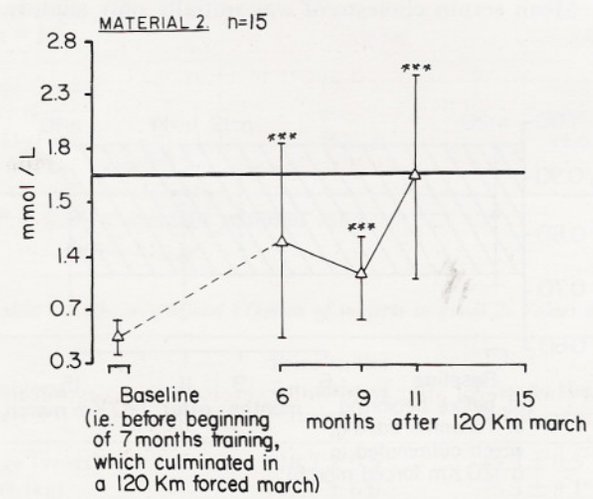

Fig. 7. Serum triglyceride concentrations in group 2. Values are means, bars = SD. The upper line marks upper border of reference interval.
***$P < 0.001$.

Discussion and conclusions

In our previous work we examined young male volunteers according to an experimental design, modelled on the training of military recruits, in which a preparatory period of graded outdoor training culminated in a forced 120 km march. We found a transient fall in S-Mg 1 h after the march, followed by a rebound and then a second sustained significant fall 72 h after the termination of the peak strenuous effort. The significantly decreased S-Mg was still present 3 months after the march (0.755, SD 0.064, mmol·litre^{-1}, n = 18, $P \leq 0.05$)[1].

In the present study, we wished to extend the observation period beyond 3 months. We therefore followed one group of military recruits, beginning 6 months after the forced 120 km march, and another group 9 months after. We found that mean S-Mg was still significantly lowered at 10 and 11 months, respectively, after the march. After 15 months, the mean S-Mg in group 2 was no longer significantly decreased, but it was nonetheless still 7% lower than the baseline value.

Prolonged lowering of S-Mg is accompanied by a decrease in intracellular Mg content[3]. This means that the prolonged hypomagnesaemia following strenuous effort may signify the presence of intracellular Mg deficit[3,6]. Prolonged intracellular deficit of magnesium is bound to have deleterious effects on health due

to the involvement of Mg in essential processes such as chelating, enzyme activation and energy processes neurotransmission and cell division[5,10]. Dramatic intracellular changes are seen in Mg deficiency in experimental animals[11,12] and in man[3]: eg, an increase in interstitial H_2O, increased intracellular Na^+, Cl^-, Ca^+ and cAMP, and reduced intracellular K^+, inorganic phosphate, and the energy-rich phosphates ATP, ADP and creatine phosphate. Furthermore, the physical performance capacity, as gauged by measurement of maximal voluntary muscle contraction force (MVC) has been shown to depend on S-Mg: the higher the S-Mg within its physiological range, the higher the MVC[12].

The sustained decrease in S-Mg which we found as a sequel to strenuous effort in the present study and in our previous studies[1,2] may account for the absence of rise of the VO_2 max in the soldiers studied[13]. The decrease in S-Mg may have been due to an intracellular shift[14,15], or to an extracellular shift due to rhabdomyolysis, or to an extracellular shift from erythrocytes[16], both of the latter leading to increased Mg diuresis[15]. The decrease may also have been due to a loss in sweat[17] or to endocrine causes.

Hormonal changes found during prolonged exercise, such as increases in catecholamines, thyroid stimulating hormone, glucagon, and corticosteroids and a decrease in plasma insulin[18], will all affect the metabolism of magnesium[6]. A decrease in Mg as a result of hormonal changes will in turn enhance the pre-existing Mg deficit, thus causing a vicious circle[6,19,20].

The deficit was not alleviated by dietary means (the diet contained 270–340 mg food Mg content daily). This confirms our previous reports[1,2] where, using an identical diet, we saw that an estimated food Mg content of 270–340 mg daily was not sufficient to replace the Mg loss due to strenuous effort[1,2].

It is important to stress that the food Mg content of the volunteer recruits was close to the current RDA[21] and that the majority of the population receives a lower daily food Mg content[20]. Our findings, therefore, have important implications for soldiers, workers and sportsmen, who may receive a daily Mg food content even lower than that ingested by the volunteer recruits examined in this study.

The results in group 1 show that the deficit increases with time, although the recruits did not undergo any further bouts of strenuous effort following the 120 km forced march; they only carried out daily physical outdoor activity, not exceeding an energy expenditure of about 4000 kcal/day (16.7 mJ).

The fact that the mean S-Mg increased by only 3% 15 months after the termination of the peak strenuous effort shows that without active treatment of the Mg deficit, it may persist for an extremely long time, the soldiers' dietary Mg intake being insufficient to alleviate it. Active supplementation with magnesium therefore appears indicated. Recent studies of magnesium supplementation in athletes have indeed shown diminished blood lactate levels[22], decreased thrombin inhibition[23], and better physical capacity[24] compared with non-supplemented controls.

Durlach[6] suggests that concomitant treatment should be given as an adjuvant to Mg supplementation to deal with the metabolic and hormonal changes that are found in exertion, and views the Mg deficit as secondary to the latter changes[6,19]. Further research of great clinical importance is therefore to clarify the relation between (1) the effects of prolonged Mg deficiency due to inadequate intake – or to an increased demand due to, for example, increased energy turnover in strenuous effort – in the face of an intake insufficiently increased to meet the high demand, and (2) the effects due to what Durlach[6] defines as Mg depletion, ie Mg deficit coupled with metabolic and hormonal changes. The very intricate relation between cause and effect in the latter states requires extensive long term research because we are not certain at present which of the hormonal changes are primary and which are in part or wholly a result of changes of ionic concentrations, of substrate concentration, or are influenced by substrate-sensitive receptors[19].

Of great clinical importance also is the continuation of the pioneering research of Durlach[6] on the clinical treatment methods to be used as an adjuvant to Mg replacement in states of Mg deficit accompanied by metabolic and hormonal changes.

In our previous studies[1,2] a delayed rise in serum lipids and blood sugar was reported for the first time after strenuous effort, in parallel with a sustained fall in S-Mg[2]. Our findings in the present study confirm our earlier results[1,2] and document the long term detrimental effect of strenuous effort on serum lipids, expressed as a gradual rebound rise over time of both cholesterol and triglyceride levels and, in one group, a transient rise of blood sugar level as well. The peak rise of cholesterol and triglycerides is observed parallel to a peak decrease in mean S-Mg 10 months (group 1) and 11 months (group 2) after the termination of peak strenuous effort. The delayed rise in blood sugar in group 2, seen 9 months after peak exercise, was followed 11 months later by a decrease which paralleled that of S-Mg.

Our findings are surprising because it has been generally accepted that exercise lowers lipid levels in man. Although Durstine *et al.*[25] reported an increase in cholesterol after treadmill exercise to exhaustion in man, they interpreted it as a beneficial effect on lipid profile because of a rise in the HDL-cholesterol fraction.

Mg occupies a pivotal role in intermediary metabolism. The importance of Mg for carbohydrate metabolism has been reviewed by Durlach & Rayssiguier[3,4] and by Durlach[5]. Evidence derived from

animal experiments on the effect of Mg on plasma lecithin-cholesterol acyltransferase was presented by Gueux *et al.*[26], and on lipoprotein lipase activity by Gueux & Rayssiguier[27].

A possible modification by Mg deficiency of membrane phospholipids affecting the cellular membrane has been suggested by Sinclair[28]. In view of these reports, our findings are most significant, although published reports on the association between Mg and lipid metabolism in man are still very scanty.

Although we did not study cholesterol fractions, the findings of the present study reinforce those reported by us earlier[1,2], and show a deleterious long term effect of strenuous effort on the lipid and carbohydrate profile in apparently healthy, young military recruits. The sustained Mg deficit following strenuous effort found by us in our two previous studies and confirmed by the present study may constitute one of the causes of the gradual delayed rise in serum lipids[29]. Furthermore, the hormonal changes reported to favour mobilization of fatty acids and hepatic glucose production in prolonged exercise[18] may in turn increase the demand for Mg.

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Effets à long terme d'un effort épuisant extrême sur les magnésium, lipides et glucides sériques d'hommes jeunes apparemment sains

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Résumé: Nos résultats precedents ont revele qu'un effort épuisant provoquent une diminution de la concentration du magnésium sérique (S-Mg) (indiquant sans doute un deficit magnésique) ainsi que l'augmentation retardée du taux du sucre sanguin et des lipides sériques. Le S-Mg demeurait encore bas 3 mois après l'acmé d'un effort épuisant. Pour élargir notre comprehension, nous voulons etudier les consequences biochimiques de l'épuisement pendant une longue periode de temps tout en utilisant les mêmes conditions expérimentales employées auparavant, et qui elles-memes reproduisent celles qui sont en vigueur dans l'entrainement des recrues militaires. Dans ce but nous avons examiné deux groupes de recrues militaires, groupe 1, n = 15, et groupe 2, n = 16, d'un age moyen de 18.6 ± 1.3 et 18.7 ± 0.6 ans qui ont subi un entrainement gradue de 7 mois, culminant en une marche forcée de 120 km. La teneur en S-Mg dans le sang de 20 recrues a été évaluée pendant l'entrainement et dans le groupe 1 6 et 10 mois après le marche forcée et dans le groupe 2, 9, 11 et 15 mois après. Le taux en sucre et lipides a été évalué au moment du recrutement et pendant les 11 mois qui suivirent. Une diminution significative du taux de S-Mg a été observée jusqu à 10 mois apres la marche forcée dans le groupe 1, et 11 mois après dans le groupe 2 ($P < 0.01$). Le taux moyen du cholestérol et des lipides sériques a montré une élévation progressive et retardée, specialement dans le groupe 2 ($P < 0.05$ et 0.001 respectivement), tandis que le taux en sucre a diminue dans le groupe 1 et augmente dans le groupe 2.

Les résultats présentés ici confirment le fait qu'un effort épuisant cause un déficit magnésique prolongé et pour la première fois démontrent l'augmentation concomitante et prolongée des lipides.

Mots clés: Déficit magnésique, effort épuisant, hyperlipidémie.

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