20. Magnesium

Physiology

The adult male contains 20-28 g magnesium; 60-65 % of this is in the skeleton; only about 1 % is in the extracellular fluid, and is distributed similarly to calcium in the plasma; the rest is intracellular where its activity concentration is tightly regulated [1]. Magnesium is essential for mineralisation and skeletal development and for the maintenance of the transmembrane electrical potentials in nerves and muscle. It also serves as a cofactor for the phosphorylated purine nucleotides and hence all related enzyme activities. It is involved in the structure and replication of nucleic acids, and the ribosomal binding of mRNA. Thus magnesium is vital for the regulation of cellular metabolism and the synthesis of proteins.

Magnesium is absorbed by a carrier-mediated mechanism and by a non-specific diffusional process [2]. Reported intakes range from 132 to 350 mg for women and 157 to 595 mg for men with respective means of 234 and 310 mg [3]; in the UK dietary survey corresponding means of 237 mg (9.8 mmol) and 323 mg (13.3 mmol) were recorded [4]. At such intakes there is great variability in the efficiency of absorption and retention: average net absorptions of 21 % and 27 % have been reported in men and women respectively [5]. Absorption of magnesium is adaptive; at intakes in excess of 2 g/d the element is poorly absorbed. Phytate, calcium phosphate and long chain triacylglycerols may impair the intestinal absorption of magnesium but since there is very little evidence of magnesium deficiency arising from the diet, there is very little definitive information on factors influencing its bioavailability. Systemic homoeostasis of magnesium is achieved primarily through renal excretion; at low dietary intakes there is increased absorption and renal conservation of the element.

The endocrine control of magnesium homoeostasis and metabolism is understood poorly. The parathyroid hormone responds to acute changes in extracellular (serum) concentrations of magnesium in the same way as it does to alterations in ionised calcium activity; thus, in response to a sudden drop in serum magnesium, the production of parathyroid hormone increases, thereby enhancing both the renal conservation of the element and its release from the skeletal pool [1].

Deficiency and excess

Current dietary intakes of magnesium are adequate but systemic magnesium deficiency can result from other conditions such as intestinal and pancreatic malabsorption syndromes, the use of diuretic drugs and increased requirements arising from rapid tissue synthesis, e.g. during recovery from malnutrition, for which as an intracellular cation the element is essential.

Deficiency is manifested by altered metabolism of calcium, sodium and potassium, which is reflected in altered function of skeletal and cardiac muscle, muscle weakness, and fits. Tetany may develop with a resistant hypocalcaemia arising from reduced secretion of parathyroid hormone and end organ hyporesponsiveness.

Large intakes (e.g. 3-5 g of the element) induce intestinal secretion and diarrhoea. Since renal excretion of excess magnesium is so efficient, such ingestions usually have minimal direct systemic effects, but systemic excess, such as may arise from large intakes of magnesium salts by individuals with renal insufficiency or from intravenous administration, can cause central nervous system depression, with muscular paralysis and death [6].

Requirements

Adults

Magnesium is ubiquitous in the diet; both plants and meats are good dietary sources. Early balance studies suggested that adult requirements may be as high as 700 mg/d [7] but interpretation of these is difficult because of analytical difficulties and the long time needed to achieve equilibrium [8]. Additionally the improved efficiency, on restricted intakes, of both intestinal absorption [9] and increased renal conservation of magnesium make it difficult in the absence of more definitive studies to propose any PRI with confidence. One study [10] has suggested that normal adults can achieve positive balance on 3 mg/kg/d over a 6-9 day period. Evaluation of the variance of such balance data suggests that intakes of 3.4 mg/kg would be associated reliably with a net balance [11]. Actual requirements may well be below this; and, in the absence of better physiological data with which to establish reliable reference intakes, an acceptable range of intakes of 150-500 mg /d is proposed on the basis of observed intakes.

This range of intakes will also cover pregnancy and lactation.

Children

The difficulties in proposing a reliable PRI for adults are more pronounced in children, for whom data are even more scarce. If however some guidance is required, rough estimates can be made by calculating average requirements for groups on the basis of body weights. Factors can be used ranging from 7 mg/kg body weight/d at 6-11 months (slightly higher than the intake from breast milk at 6 months) to 4.2 mg/kg body weight/d at 15-17 years (slightly higher than the 3.4 mg/kg/d which appears adequate for adults). An extra 30% is added to allow for individual variations in growth. The quasi - PRIs thus calculated are (mg/d): 6-11 months, 80; 1-3 years, 85; 4-6 years, 120; 7-10 years, 200; 11-14 years, 280; 15-17 years, 300. These guesses should be treated with caution. The amounts would certainly be adequate, and it is highly likely that they are over-generous, but the data are too sparse to support good estimates.

Summary

Acceptable Range of Intakes for Adults: 150-500 mg/d.

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