

## 11. Magnesium

### 11.1 Tissue distribution and biological role of magnesium

The human body contains about 760 mg of magnesium at birth, approximately 5 g at age 4–5 months, and 25 g when adult (1–3). Of the body's magnesium, 30–40% is found in muscles and soft tissues, 1% is found in extracellular fluid, and the remainder is in the skeleton, where it accounts for up to 1% of bone ash (4, 5).

Soft tissue magnesium functions as a cofactor of many enzymes involved in energy metabolism, protein synthesis, RNA and DNA synthesis, and maintenance of the electrical potential of nervous tissues and cell membranes. Of particular importance with respect to the pathological effects of magnesium depletion is the role of this element in regulating potassium fluxes and its involvement in the metabolism of calcium (6–8). Magnesium depletion depresses both cellular and extracellular potassium and exacerbates the effects of low-potassium diets on cellular potassium content. Muscle potassium becomes depleted as magnesium deficiency develops, and tissue repletion of potassium is virtually impossible unless magnesium status is restored to normal. In addition, low plasma calcium often develops as magnesium status declines. It is not clear whether this occurs because parathyroid hormone release is inhibited or, more probably, because of a reduced sensitivity of bone to parathyroid hormone, thus restricting withdrawal of calcium from the skeletal matrix.

Between 50% and 60% of body magnesium is located within bone, where it is thought to form a surface constituent of the hydroxyapatite (calcium phosphate) mineral component. Initially much of this magnesium is readily exchangeable with serum and therefore represents a moderately accessible magnesium store which can be drawn on in times of deficiency. However, the proportion of bone magnesium in this exchangeable form declines significantly with increasing age (9).

Significant increases in bone mineral density of the femur have been associated positively with rises in erythrocyte magnesium when the diets of subjects with gluten-sensitive enteropathy were fortified with magnesium (10). Little is known of other roles for magnesium in skeletal tissues.

## 11.2 Populations at risk for, and consequences of, magnesium deficiency

Pathological effects of primary nutritional deficiency of magnesium occur infrequently in infants (11) but are even less common in adults unless a relatively low magnesium intake is accompanied by prolonged diarrhoea or excessive urinary magnesium losses (12). Susceptibility to the effects of magnesium deficiency rises when demands for magnesium increase markedly with the resumption of tissue growth during rehabilitation from general malnutrition (6, 13). Studies have shown that a decline in urinary magnesium excretion during protein–energy malnutrition (PEM) is accompanied by a reduced intestinal absorption of magnesium. The catch-up growth associated with recovery from PEM is achieved only if magnesium supply is increased substantially (6, 14).

Most of the early pathological consequences of depletion are neurologic or neuromuscular defects (12, 15), some of which probably reflect the influence of magnesium on potassium flux within tissues. Thus, a decline in magnesium status produces anorexia, nausea, muscular weakness, lethargy, staggering, and, if deficiency is prolonged, weight loss. Progressively increasing with the severity and duration of depletion are manifestations of hyperirritability, hyperexcitability, muscular spasms, and tetany, leading ultimately to convulsions. An increased susceptibility to audiogenic shock is common in experimental animals. Cardiac arrhythmia and pulmonary oedema frequently have fatal consequences (12). It has been suggested that a suboptimal magnesium status may be a factor in the etiology of coronary heart disease and hypertension but additional evidence is needed (16).

## 11.3 Dietary sources, absorption, and excretion of magnesium

Dietary deficiency of magnesium of a severity sufficient to provoke pathological changes is rare. Magnesium is widely distributed in plant and animal foods, and geochemical and other environmental variables rarely have a major influence on its content in foods. Most green vegetables, legume seeds, beans, and nuts are rich in magnesium, as are some shellfish, spices, and soya flour, all of which usually contain more than 500 mg/kg fresh weight. Although most unrefined cereal grains are reasonable sources, many highly-refined flours, tubers, fruits, fungi, and most oils and fats contribute little dietary magnesium (<100 mg/kg fresh weight) (17–19). Corn flour, cassava and sago flour, and polished rice flour have extremely low magnesium contents. Table 11.1 presents representative data for the dietary magnesium intakes of infants and adults.

TABLE 11.1

**Typical daily intakes of magnesium by infants (6 kg) and adults (65 kg), in selected countries**

Group and source of intake	Magnesium intake (mg/day) <sup>a</sup>	Reference(s)
<i>Infants<sup>b</sup></i>		
Human-milk fed		
Finland	24 (23–25)	17
India	24 ± 0.9	20
United Kingdom	21 (20–23)	21,22
United States	23 (18–30)	11,23
Formula-fed		
United Kingdom (soya-based)	38–60	24
United Kingdom (whey-based)	30–52	24
United States	30–52	11,23
<i>Adults: conventional diets</i>		
China, Changle county	232 ± 62	25
China, Tuoli county	190 ± 59	25
China, females	333 ± 103	25
France, females	280 ± 84	26
France, males	369 ± 106	26
India	300–680	27
United Kingdom, females	237	28
United Kingdom, males	323	28
United States, females	207	29,30
United States, males	329	29,30

<sup>a</sup> Mean ± SD or mean (range).

<sup>b</sup> 750ml liquid milk or formula as sole food source.

Stable isotope studies with <sup>25</sup>Mg and <sup>26</sup>Mg indicate that between 50% and 90% of the labelled magnesium from maternal milk and infant formula can be absorbed by infants (11, 23). Studies with adults consuming conventional diets show that the efficiency of magnesium absorption can vary greatly depending on magnesium intake (31, 32). One study showed that 25% of magnesium was absorbed when magnesium intake was high compared with 75% when intake was low (33). During a 14-day balance study a net absorption of 52 ± 8% was recorded for 26 adolescent females consuming 176 mg magnesium daily (34). Although this intake is far below the United States recommended dietary allowance (RDA) for this age group (280 mg/day), magnesium balance was still positive and averaged 21 mg/day. This study provided one of several sets of data that illustrate the homeostatic capacity of the body to adapt to a wide range of magnesium intakes (35, 36). Magnesium absorption appears to be greatest within the duodenum and ileum and occurs by both passive and active processes (37).

High intakes of dietary fibre (40–50 g/day) lower magnesium absorption. This is probably attributable to the magnesium-binding action of phytate

phosphorus associated with the fibre (38–40). However, consumption of phytate- and cellulose-rich products increases magnesium intake (as they usually contain high concentrations of magnesium) which often compensates for the decrease in absorption. The effects of dietary components such as phytate on magnesium absorption are probably critically important only when magnesium intake is low. There is no consistent evidence that modest increases in the intake of calcium (34–36), iron, or manganese (22) affect magnesium balance. In contrast, high intakes of zinc (142 mg/day) decrease magnesium absorption and contribute to a shift towards negative balance in adult males (41).

The kidney has a very significant role in magnesium homeostasis. Active reabsorption of magnesium takes place in the loop of Henle in the proximal convoluted tubule and is influenced by both the urinary concentration of sodium and probably by acid–base balance (42). The latter relationship may well account for the observation drawn from Chinese studies that dietary changes which result in increased urinary pH and decreased titratable acidity also reduce urinary magnesium output by 35% despite marked increases in magnesium input from vegetable protein diets (25). Several studies have now shown that dietary calcium intakes in excess of 2600 mg/day (37), particularly if associated with high sodium intakes, contribute to a shift towards negative magnesium balance or enhance its urinary output (42, 43).

#### **11.4 Criteria for assessing magnesium requirements and allowances**

In 1996, Shils and Rude (44) published a constructive review of past procedures used to derive estimates of magnesium requirements. They questioned the view of many authors that metabolic balance studies are probably the only practicable, non-invasive techniques for assessing the relationship of magnesium intake to magnesium status. At the same time, they emphasized the great scarcity of data on variations in urinary magnesium output and on magnesium levels in serum, erythrocytes, lymphocytes, bone, and soft tissues. Such data are needed to verify current assumptions that pathological responses to a decline in magnesium supply are not likely to occur if magnesium balance remains relatively constant.

In view of Shils and Rude's conclusion that many estimates of dietary requirements for magnesium were "based upon questionable and insufficient data" (44), a closer examination is needed of the value of biochemical criteria for defining the adequacy of magnesium status (13). Possible candidates for further investigation include the effects of changes in magnesium intake on urinary magnesium–creatinine ratios (45), the relationships between serum

magnesium–calcium and magnesium–potassium concentrations (7, 8), and various other functional indicators of magnesium status.

The scarcity of studies from which to derive estimates of dietary allowances for magnesium has been emphasized by virtually all the agencies faced with this task. One United Kingdom agency commented particularly on the scarcity of studies with young subjects, and circumvented the problem of discordant data from work with adolescents and adults by restricting the range of studies considered (21). Using experimental data virtually identical to those used for a detailed critique of the basis for United States estimates (44), the Scientific Committee for Food of the European Communities (46) proposed an acceptable range of intakes for adults of 150–500 mg/day and described a series of quasi-population reference intakes for specific age groups, which included an increment of 30% to allow for individual variations in growth. Statements of acceptable intakes such as these leave uncertainty as to the extent of overestimation of derived recommended intakes.

It is questionable whether more reliable estimates of magnesium requirements can be made until data from balance studies are supported by the use of biochemical indexes of adequacy that could reveal the development of manifestations of suboptimal status. Such indexes have been examined, for example, by Nichols et al. (14) in their studies of the metabolic significance of magnesium depletion during PEM. A loss of muscle and serum magnesium resulted if total body magnesium retention fell below 2 mg/kg/day and was followed by a fall in the myofibrillar nitrogen–collagen ratio of muscle and a fall in muscle potassium content. Repletion of tissue magnesium status preceded a three-fold increase in muscle potassium content. Furthermore, it accelerated, by 7–10 days, the rate of recovery of muscle mass and composition initiated by restitution of nitrogen and energy supplies to infants previously deficient.

Neurologic signs such as hyperirritability, apathy, tremors, and occasional ataxia accompanied by low concentrations of potassium and magnesium in skeletal muscle and strongly negative magnesium balances were reported by many other studies of protein calorie deficiency in infants (47–49). Particularly noteworthy is evidence that all these effects are ameliorated or eliminated by increased oral magnesium, as were specific anomalies in the electrocardiographic T-wave profiles of such malnourished subjects (49). Evidence that the initial rate of growth at rehabilitation is influenced by dietary magnesium intake indicates the significance of this element for the etiology of the PEM syndromes (31, 50).

Regrettably, detailed studies have yet to be carried out to define the nature of changes resulting from a primary deficiency of dietary magnesium. Defin-

ition of magnesium requirements must therefore continue to be based on the limited information provided by balance techniques, which give little or no indication of responses by the body to inadequacy in magnesium supply that may induce covert pathological changes, and reassurance must be sought from the application of dietary standards for magnesium in communities consuming diets differing widely in magnesium content (27). The inadequate definition of lower acceptable limits of magnesium intake raises concern in communities or individuals suffering from malnutrition or a wider variety of nutritional or other diseases which influence magnesium metabolism adversely (12, 51, 52).

### **11.5 Recommended intakes for magnesium**

The infrequency with which magnesium deficiency develops in human-milk-fed infants implies that the content and physiological availability of magnesium in human milk meets the infants' requirements. The intake of maternal milk from exclusively human-milk-fed infants 1–10 months of age ranges from 700 to 900 ml/day in both industrialized and developing countries (53). If the magnesium content of milk is assumed to be 29 mg/l (11, 54, 55), the intake from milk is 20–26 mg/day, or approximately 0.04 mg/kcal.

The magnesium in human milk is absorbed with substantially greater efficiency (about 80–90%) than that of formula milks (about 55–75%) or solid foods (about 50%) (56), and such differences must be taken into account when comparing differing dietary sources. For example, a daily intake of 23 mg from maternal milk probably yields 18 mg available magnesium, a quantity similar to that of the 36 mg or more suggested as meeting the requirements of young infants given formula or other foods (see below).

An indication of a likely requirement for magnesium at other ages can be derived from studies of magnesium–potassium relationships in muscle (57) and the clinical recovery of young children rehabilitated from malnutrition with or without magnesium fortification of therapeutic diets. Nichols et al. (14) showed that 12 mg magnesium/day was not sufficient to restore positive magnesium balance, serum magnesium content, or the magnesium and potassium contents of muscle of children undergoing PEM rehabilitation. Muscle potassium was restored to normal by 42 mg magnesium/day but higher intakes of dietary magnesium, up to 160 mg/day, were needed to restore muscle magnesium to normal. Although these studies show clearly that magnesium synergized growth responses resulting from nutritional rehabilitation, they also indicated that rectification of earlier deficits of protein and energy was a prerequisite to initiation of this effect of magnesium.

Similar studies by Caddell et al. (49, 50) also illustrate the secondary significance of magnesium accelerating clinical recovery from PEM. They indicate that prolonged consumption of diets low in protein and energy and with a low ratio ( $<0.02$ ) of magnesium (in milligrams) to energy (in kilocalories) can induce pathological changes which respond to increases in dietary magnesium supply. It is noteworthy that of the balance trials intended to investigate magnesium requirements, none has yet included treatments with magnesium–energy ratios of  $<0.04$  or induced pathological responses.

The relationship  $Mg = (kcal \times 0.0099) - 0.0117$  ( $SE \pm 0.0029$ ) holds for many conventional diets (58). Some staple foods in common use have very low magnesium contents; cassava, sago, corn flour or cornstarch, and polished rice all have low magnesium–energy ratios ( $0.003$ – $0.02$ ) (18). Their widespread use merits appraisal of total dietary magnesium content.

It has been reported with increasing frequency that a high percentage (e.g.  $<70\%$ ) (26) of individuals from some communities in Europe have magnesium intakes substantially lower than estimates of magnesium requirements derived principally from United States and United Kingdom sources (21, 29). Such reports emphasize the need for reappraisal of estimates for reasons previously discussed (44).

Recommended magnesium intakes proposed by the present Consultation are presented in Table 11.2 together with indications of the relationships of each recommendation to relevant estimates of the average requirements for dietary protein and energy (19). These recommended intakes must be regarded as provisional. Until additional data become available, these estimates reflect consideration of anxieties that previous recommendations for magnesium are overestimates. The estimates provided by the Consultation make greater allowance for developmental changes in growth rate and in protein and energy requirements. In reconsidering data on which estimates were based cited in previous reports (21, 29, 46), particular attention has been paid to balance data suggesting that the experimental conditions established have provided reasonable opportunity for the development of equilibrium during the investigation (34, 60–62).

The detailed studies of magnesium economy during malnutrition and subsequent therapy, with or without magnesium supplementation, provide reasonable grounds that the dietary magnesium recommendations derived herein for young children are realistic. Data for other ages are more scarce and are confined to magnesium balance studies. Some studies have paid little attention to the influence of variations in dietary magnesium content and of the effects of growth rate before and after puberty on the normality of magnesium-dependent functions.

TABLE 11.2

**Recommended nutrient intakes (RNIs) for magnesium, by group**

Group <sup>a</sup>	Assumed body weight (kg) <sup>b</sup>	RNI (mg/day)	Relative intake ratios		
			(mg/kg)	(mg/g protein <sup>c</sup> )	(mg/kcal/day <sup>d</sup> )
<i>Infants and children</i>					
0–6 months					
Human-milk-fed	6	26	4.3	2.5	0.05
Formula-fed	6	36	6.0	2.9	0.06
7–12 months					
1–3 years	9	54	6.0	3.9	0.06
1–3 years	12	60	5.5	4.0	0.05
4–6 years	19	76	4.0	3.9	0.04
7–9 years	25	100	4.0	3.7	0.05
<i>Adolescents</i>					
Females, 10–18 years	49	220	4.5	5.2	0.10
Males, 10–18 years	51	230	3.5	5.2	0.09
<i>Adults</i>					
Females					
19–65 years	55	220	4.0	4.8	0.10
65+ years	54	190	3.5	4.1	0.10
Males					
19–65 years	65	260	4.0	4.6	0.10
65+ years	64	224	3.5	4.1	0.09

<sup>a</sup> No increment for pregnancy; 50 mg/day increment for lactation.

<sup>b</sup> Assumed body weights of age groups are derived by interpolation (59).

<sup>c</sup> Intake per gram of recommended protein intake for age of subject (21).

<sup>d</sup> Intake per kilocalorie estimated average requirement (21).

It is assumed that during pregnancy, the fetus accumulates 8 mg magnesium and fetal adnexa accumulate 5 mg magnesium. If it is assumed that this magnesium is absorbed with 50% efficiency, the 26 mg required over a pregnancy of 40 weeks (0.09 mg/day) can probably be accommodated by adaptation. A lactation allowance of 50–55 mg/day for dietary magnesium is made for the secretion of milk containing 25–28 mg magnesium (21, 63).

It is appreciated that magnesium demand probably declines in late adulthood as requirements for growth diminish. However, it is reasonable to expect that the efficiency with which magnesium is absorbed declines in elderly subjects. It may well be that the recommendations are overgenerous for elderly subjects, but data are not sufficient to support a more extensive reduction than that indicated. An absorption efficiency of 50% is assumed for all solid diets; data are not sufficient to allow for the adverse influence of phytic acid on magnesium absorption from high-fibre diets or from diets with a high content of pulses.

Not surprisingly, few of the representative dietary analyses presented in Table 11.1 fail to meet these recommended allowances. The few exceptions,

deliberately selected for inclusion, are the marginal intakes ( $232 \pm 62$  mg) of the 168 women of Changle County, People's Republic of China, and the low intake ( $190 \pm 59$  mg) of 147 women surveyed from Tuoli County, People's Republic of China (25).

### 11.6 Upper limits

Magnesium from dietary sources is relatively innocuous. Contamination of food or water supplies with magnesium salt has been known to cause hypermagnesaemia, nausea, hypotension, and diarrhoea. Intakes of 380 mg magnesium as magnesium chloride have produced such signs in women. Upper limits of 65 mg for children aged 1–3 years, 110 mg for children aged 4–10 years, and 350 mg for adolescents and adults are suggested as tolerable limits for the daily intake of magnesium from foods and drinking water (64).

### 11.7 Comparison with other estimates

The recommended intakes for infants aged 0–6 months take account of differences in the physiological availability of magnesium from maternal milk as compared with infant formulas or solid foods. With the exception of the Canadian recommended nutrient intakes (RNIs), which are 20 mg/day for infants aged 0–4 months and 32 mg/day for those aged 5–12 months (63), other countries recommend intakes (as RDAs or RNIs) which substantially exceed the capacity of the lactating mother to supply magnesium for her offspring.

Recommendations for other ages are based subjectively on the absence of any evidence that magnesium deficiency of nutritional origin has occurred after consumption of a range of diets sometimes supplying considerably less than the United States RDA or the United Kingdom RNI recommendations, which are based on estimates of average magnesium requirements of 3.4–7 mg/kg body weight. The recommendations submitted herein assume that demands for magnesium, plus a margin of approximately 20% (to allow for methodological variability), are probably met by allowing approximately 3.5–5 mg/kg body weight from pre-adolescence to maturity. This assumption yields estimates virtually identical to those for Canada. Expressed as magnesium allowance (in milligrams) divided by energy allowance (in kilocalories)—the latter based upon energy recommendations from United Kingdom estimates (21)—all of the recommendations of Table 11.2 exceed the provisionally estimated critical minimum magnesium–energy ratio of 0.02.

### 11.8 Recommendations for future research

There is need for closer investigation of the biochemical changes that develop as magnesium status declines. The responses to magnesium intake, which

influence the pathological effects resulting from disturbances in potassium utilization caused by low magnesium, should be studied. They may well provide an understanding of the influence of magnesium status on growth rate and neurologic integrity.

Closer investigation of the influence of magnesium status on the effectiveness of therapeutic measures during rehabilitation from PEM is also needed. The significance of magnesium in the etiology and consequences of PEM in children needs to be clarified. Claims that restoration of protein and energy supply aggravates the neurologic features of PEM if magnesium status is not improved merit priority of investigation. Failure to clarify these aspects may continue to obscure some of the most important pathological features of a nutritional disorder in which evidence already exists for the involvement of a magnesium deficit.

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