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Die Untersuchung der Beziehungen zwischen Noxen und Medikamenten mit Hilfe der Biostruktur führt zur Umreißung der theoretischen Grundlagen der Hauptprinzipien der Allo- und Homöopathie und erlaubt die Festlegung der Bedingungen, die bei der Auswahl, Dosierung und Verabreichung von Arzneimitteln zu beachten sind, wobei dem Wirkungsmechanismus der Noxen auf die Biostruktur der Kranken Rechnung getragen wird.

Summary

The Biostructure and its Relationships to Noxes and Drugs

According to a new conception, a part of the matter in living cells is integrated into a quite particular structure which breaks down at the moment of death. This life-specific structure was described as biostructure whereas the matter included in — living matter.

Both noxious substances and drugs act upon the biostructure determining the appearance of some anomalous states expressed as symptoms. At the same time they release the regulation mechanism included in the biostructure; in this way, the latter tends to abolish the noxious actions exerted upon itself.

The analysis of relationships between noxious substances and drugs in the biostructure permits the theoretical bases of the fundamental principles of allo- and homoeopathy to be outlined; it also opens some prospects to establishing the conditions of drug choice, dosage and administration, in terms of the mechanism of noxious substance action upon biostructure in patients.

Résumé

La biostructure et leur relations aux noxes et aux médicaments

Selon une nouvelle conception, une part de la matière des cellules vivantes est intégrée dans une structure tout-à-fait spéciale qui se désagrège au moment de la mort. Cette structure particulière a été, dénommée biostructure, cependant que la matière qu'elle contient — matière vivante.

Les substances nocives tout comme les médicaments agissant sur la biostructure provoquent l'apparition de certains états anormaux qui se traduisent par la manifestation des symptômes correspondantes. En même temps elles déclenchent les mécanismes de réglage contenus dans la biostructure; de cette façon elle tend à annuler les actions nocives qui agissent sur elle.

L'examen des relations entre substances nocives et médicaments dans le cadre de la biostructure permet d'ébaucher les bases théoriques des principes fondamentaux de l'allo- et de l'homéopathie; de même, il ouvre une perspective pour l'établissement des conditions du choix, du dosage et de l'administration des médicaments en fonction du mécanisme d'action des substances nocives sur la biostructure des malades.

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Vitalstoffe
Vital substances
Substances vitales

The Importance of Dietary Magnesium With Particular Reference To Humans

By J. R. Marier, Ottawa

It is not the purpose of this review to cover the full history of magnesium, dating back to its isolation as a metal. Perhaps it will suffice to make three generalizations about the statuts of magnesium only a few decades ago: To nutritionists, it was known as essential for growth; in the laboratory, it was recognized as an excellent activator of many enzyme systems; therapeutically, it had achieved world-wide fame (or notoriety) as a purgative. Much more is known about it today; some excellent reviews have appeared on the subject (e.g. 2, 58, 80, 81, 83). However, it is disconcerting to note that — even today — clinical magnesium analyses are not being performed in the great majority of our major hospitals.

Disturbances of magnesium metabolism can have serious repercussions, as we shall soon see. Before getting into the subject of metabolic pathway and function, it might be relevant to consider the overall distribution of magnesium in the human adult (Table I).

Of significance is the fact that only about 1 % of the body's magnesium is present in body fluids; the remainder is about equally divided between the skeleton (where it is associated with the calcium phosphate of the mineral phase), and the other major portion is in intracellular

Table 1 • General Distribution of Magnesium in Human Adult (83).

		% of Total
Skeleton (approx.)	11.00 g.	52.4
Intracellular	9.76	46.5
Fluids, except plasma	0.18	0.8
Plasma	0.06	0.3
	Total 21.00 g.	

compartments, mostly in muscle. The latter characteristic emphasizes the fact that magnesium — like potassium — is an avid participant in intracellular reactions.

The requirements of magnesium for proper growth varies with the species. In the young human adult, the intake to maintain a positive balance is ca 300 mg. per day; in children and expectant mothers, the requirement is somewhat higher: 400 mg. per day (83). Seelig (80) has recently compiled data attesting to the insufficiency of dietary magnesium in the Western world, and recommends an intake above 6 mg/kg/day. The principal sources of dietary magnesium for human beings are from vegetables, blackstrap molasses, nuts and wholemeal flour; a lesser amount is available in meat and fish,

whereas eggs and dairy products make only a minor contribution (80, 83). Dairy products have a very high calcium content, relative to magnesium, which make them rather a poor source of dietary magnesium (see later). The absorption of magnesium from the gut resembles that of calcium (2). There is evidence for a common pathway, not only in the gut but also in the renal tubule, so that a deficiency of one ion leads to over-absorption of the other (18). Hanna (40, 43) has presented interesting data to show that citrate may be involved at both the intestinal and renal sites: Citrate would facilitate passage of both calcium and magnesium into the blood, and, also, from the blood into the urine. It is worth noting that hypomagnesaemia can be induced by exchange transfusion with citrated blood (4). According to Hanna, the principal function of Vitamin D in the gut would be the stimulation of citrate production. Lactose, which is known to stimulate absorption of calcium (65), might also influence citrate accumulation.

Because both ions are in competition for the same absorption mechanism, it is not surprising that large amounts of dietary calcium reduce the absorption of magnesium (55, 64). However, high levels of dietary phosphate also impede magnesium absorptivity, whereas low amounts of phosphate enhance it (12, 13). This effect of phosphate on magnesium is felt to be a "gut solubility" phenomenon, with high levels of either (or both) components favoring fecal excretion.

Once absorbed into the bloodstream, the control of plasma magnesium appears to be primarily renal (68, 83). Unlike calcium, magnesium does not seem to be controlled by parathyroid hormone (42, 58, 83), although this is still a subject open to debate (44). However, an inverse relationship has been observed between magnesium and aldosterone (11, 34, 58, 68, 83), although the primary effect of aldosterone seems to be on intracellular magnesium (83), (about which more will be said later). The maintenance of adequate serum magnesium levels, therefore, appears to depend largely on intake and subsequent absorption from the gut. Table 2 shows that the concentration of magnesium in human plasma is quite low.

Table 2 • Some Major Components of Human Blood Plasma (89).

| | Plasma | | Ultrafiltrate | % Bound |
	mg. %	mM/l	mM/l	
Calcium	9.96	2.49	1.37	45.0
Magnesium	2.36	0.97	0.66	32.0
Inorg. P	3.57	1.15	1.17	None
Citrate	2.30	0.12	0.12	None
pH		7.57	7.57	
Protein		6.63 %		None

It can be said that magnesium is serum's most vulnerable ionic component; without continual replenishment, the result is a negative magnesium balance, with or without hypomagnesaemia.

Serum magnesium concentration is usually reported in m.eq. per liter. Hanna's values (39), obtained by flame-photometry, indicate a level between 1.4 and 1.9 m.eq./l. in "normal" subjects. North American workers (e.g. 5), most of whom have used colorimetric methods, obtain a range that is about 20 % higher for their "normal" subjects (cf 41, 75). For this reason, the absolute value reported by either group may not be as comparable as the "% decrease" observed in cases of hypomagnesaemia. Reliance on such differences should more-or-less compensate for lack of uniformity in the specific values reported by different authors.

The question might be asked: "Why bother to maintain serum magnesium at a so-called normal level...?" If, for the moment, we ignore the chain-reaction that such a depletion would set in motion, and if we consider serum itself, then I will give you what (in my opinion) is a very good reason: Magnesium has the peculiar property of delaying the nucleation and precipitation of apatitic calcium phosphates (9). A graphic corroboration of this role of magnesium has been reported by Mukai and Howard (66), who found that the addition of magnesium to what

they termed "evil" urine (obtained from persons producing urinary calculus) will invariably change the urine into what they call "good" urine, i.e., one that will not calcify. As for the condition in blood it is known that additions of magnesium will prevent clotting, while calcium enhances it (36).

Various studies with animals have shown that, when the diet is depleted in magnesium, the result is a reduced level of urinary magnesium but a greatly increased output of phosphate (e.g. 27, 82). Such studies have led to an association between phosphatic urinary calculus and urines low in magnesium, but high in phosphate (22). It has been shown that dietary magnesium, supplementation protects animals against stone formation (27). In humans, oral magnesium supplements (420 mg. MgO daily) have prevented calculus formation of both the oxalate and phosphate types (63). Dietary magnesium supplements have been found to increase urinary citrate excretion (33), probably by stimulation of endogenous citrate production in the gut (43); renal calcinosis has also been associated with low-citrate levels in the urine (54).

In addition to the above work on calculus, Bunce and others have shown that a magnesium deficiency (or an increase in dietary phosphorous) promotes the accumulation of calcic deposits in the kidney (13, 54, 65). In the primary stage, these deposits led to urolithiasis; prolonged accumulation produced nephrocalcinosis in the rats studies (13). After a study of magnesium deprivation in dogs, Morris and his co-workers concluded that the generalized calcic deposits were a form of dystrophic calcification (65). Considering the fairly-normal levels of serum calcium and phosphorus in many instances of hypomagnesaemic calcification, a dystrophic phenomenon is certainly indicated. Ko et al. (50) have suggested that, in magnesium deficiency, cellular changes were brought about by precipitation of calcium phosphate in the renal tubule.

Before we leave the subject of kidney function, one more matter is worthy of attention. A few years ago, the magnesium depletion in chronic alcoholics was not too well understood. However, it has since been suggested that ethanol interferes with tubular reabsorption of magnesium in the kidney (49, 56). As we have seen that magnesium is a very "vulnerable" component of plasma, and is apparently without specific hormonal regulation, it follows that magnesium depletion is the inevitable result of chronic alcohol intake... unless, of course, dietary intake of this element is greater than usual.

In magnesium deficiency, dystrophic calcification from serum is manifested at two principal sites: One is, as we have seen, the kidney; the other is the arterial system (21, 35, 62, 65, 69, 71, 74). The significance of magnesium in atherosclerosis has not been fully appreciated during the last decade, during which people have been mainly preoccupied with cholesterol. And yet, in 1952, attention had been drawn to the interrelation of high-magnesium low-cholesterol with a low incidence of atherosclerosis (cf 23). Although some data has shown an inverse correlation between serum magnesium and cholesterol (e.g. 48) this is probably related to absorption from the gut (72, 73). The interrelation between magnesium and cholesterol warrants more detailed studies.

In much of the cardiovascular research, the pathological plaque deposits have invariably been termed "lipidic". However, Yu and Blumenthal (91) have analyzed the constituents of calcified atherosclerotic plaque from human aortas and have shown that it is composed of apatitic calcium phosphate and protein (elastin-type), with a smaller amount of mucopolysaccharide, thus accounting for over 90 % of the plaque material. Only 1.5 % of fat was found in this material. With increasing age of the subject, the calcium phosphate in aorta increased in amount and, also, in its Ca/p ratio (91). The lower degree of mineralization in "young" aortas suggests that a predisposing degenerative factor may be involved in soft-tissue calcification; such a phenomenon has been observed by Morris et al (65). In addition to its role as an inhibitor of calcium phosphate deposition, magnesium might well play a vital intracellular role in the prevention of soft-tissue degeneration. Heggveit et

al. (45) have observed mitochondrial calcification in cardiac necrosis induced by magnesium deficiency. The question of "how this all relates to the human being" must await future evaluation. Several surveys (21, 35, 61, 67) have established an inverse correlation between the total hardness of drinking-water and the incidence of cardiovascular disease. Although attempts to implicate individual components of the waters have sometimes given equivocal results (79), a comparison of three American States (Figure 1) indicates a possible inter-relation between the total hardness of drinking-water,
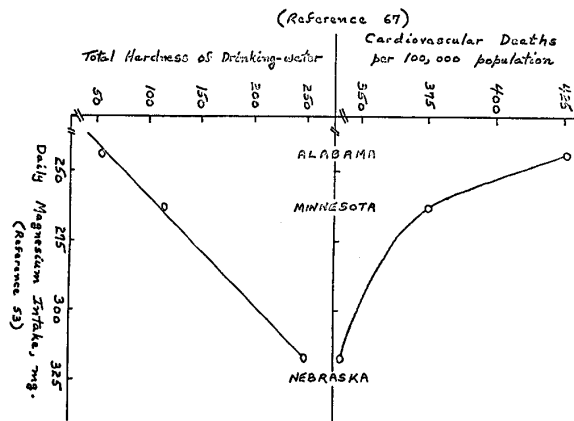
daily magnesium intake, and the incidence of cardio-vascular disease. The important role of magnesium in the prevention of cardiac disease has also been emphasized by other workers (26, 74). The N e a l s (69) have shown that the magnesium present in "hard" drinking-water can have a significant effect in the prevention of atherosclerosic lesions in rabbits, whose blood serum contained 2 % cholesterol; corroborative studies revealed a marked protective effect of magnesium, but only a slight effect of calcium. Considering the common absorption-renal pathway for calcium and magnesium, plus their antagonistic roles in various metabolic functions, perhaps the Mg/Ca ratio of drinking-water may also be a factor to be considered in relation to the cardiovascular problem.

Body reserves of magnesium reside in the skeleton and soft-tissues (1, 58, 83). The skeletal system can certainly act as a reserve in cases of need. However, in the case of magnesium, a specific regulation by parathyroid function has not been found (42, 58, 83). Nevertheless, parathyroid disorders can affect magnesium requirements (39, 41, 58, 83). Primary hyperparathyroidism causes an increased urinary loss of magnesium, and a resulting negative balance (23, 42). Parathyroidectomy or hypoparathyroidism produces an increased uptake of magnesium by bone, resulting in hypomagnesaemia (42, 47). Vitamin D cannot cope with the latter conditions because it also promotes renal excretion of magnesium (40, 43). However, post-operative magnesium supplements have been shown to be extremely beneficial in cases of parathyroid surgery (47).

It would seem that availability of bone magnesium largely depends on the "youth" of the bone, being more available from short-lived species, in which the bone is more reactive for ion-exchange (52, 62). M c A l e e s e and F o r b e s (55) have shown that young rats can lose 80 % of their skeletal magnesium while still gaining in femur calcium. Nevertheless, considering the relatively high level of calcium in bone, its overall mobilization can hardly be expected to cope with a specific deficiency in serum magnesium (See Table 3). Adult human bone is probably quite unreactive in terms of supplying plasma requirements of magnesium. This was demonstrated by M a c I n t y r e et al. (59) in a study of a patient with steatorrhoea and hypomagnesaemia, whose intracellular muscle magnesium had been reduced by two-thirds while bone magnesium was little affected. F o u r-m a n and M o r g a n (29) have shown that — even with normal plasma magnesium levels — severe depletion of muscle magnesium can occur in older animals. These

Table 3 • Composition of the Mineral Phase of Human Adult Bone (92).

	% by wt.	X 10/MW: Molar equiv.
Calcium	37.82	9.45
Magnesium	0.50	0.21
Sodium	0.73	0.32
Potassium	0.18	0.05
Phosphorus	17.52	5.65
Carbonate	5.31	1.21
Citrate	1.95	0.10
Fluoride	0.10	0.05

findings illustrate that muscle magnesium may well be the most easily available reserve of magnesium in adult subjects. However, studies of intracellular magnesium have shown that membrane diffusability of magnesium is very gradual (59, 88).

O' D e l l et al. (71) have stated that the symptoms of magnesium deficiency in the guinea-pig are: Gross kidney damage and calcification, retarded growth, hypertrophic skeletal exostoses, with muscular degeneration and calcification. They point out that guinea-pigs do not usually show the hyperaemia, epidermal lesions, hyperirritability or convulsions exhibited by the rat. Of these two animal species, man most resembles the rat. Clinical features due purely to magnesium deficiency (Table 4) have been compiled by several authors (3, 7, 10, 41, 84). These features all point to a serious depletion in intracellular muscle magnesium, combined with serum electrolyte disturbance.

Table 4 • Metabolic Features of Magnesium Deficiency in Humans.
(Source: ref. 41)
1. Outright tetany absent, if serum calcium normal.
2. Positive Chvostek sign, without a concomitant Trousseau sign.
3. A low-voltage E. C. G.
4. Epileptiform convulsions and E. E. G. changes, sometimes suggesting a focal cerebral lesion.
5. Ataxia, and muscular weakness.
6. Irritability, and sometimes depression.
(Source: ref. 3, 7, 10, 84)
7. Tremor, twitching.
8. Hypertension, and muscle rigidity.

The inverse secretion of aldosterone and plasma magnesium has caused M a c I n t y r e to wonder if aldosterone might not be the regulatory mechanism for magnesium homeostasis (58). One thing is certain: A high level of aldosterone (e. g. primary aldosteronism) is known to exert depletion of intracellular magnesium in muscle; the effect on potassium is only secondary to that on magnesium (cf 83). This may well explain the muscle weakness often displayed in sucjects with magnesium deficiency. However, the intolerance for magnesium in myasthenia gravis (5) suggests that, under certain conditions, the effect of the thymus gland might be antagonistic to that of the adrenals, at least at the muscle site. Recent data indicates that the pineal gland may have a direct effect on the regulation of serum magnesium level (68). Whatever the situation concerning endocrine control of magnesium, the mechanism is probably not a simple one. It has recently been suggested that the hormonal regulation of magnesium is independent of that for calcium (11). Considering the important role of magnesium in enzymatic reactions, it is possible that magnesium exerts a crucial influence on many endocrine processes, and is itself regulated by some of them (e. g., aldosterone).

Perhaps the earliest recognition of the role of magnesium in neurological disturbances was in the case of Delirium Tremens (28). However, in 1934, H i r s c h f e l d e r had described the clinical syndrome of low plasma magnesium in humans, where he specifically noted muscular twitching and convulsions (46). In a 1955 study with humans, S u t e r and K l i n g m a n (84) described the manifestations of magnesium depletion in man as consisting of tremor, muscular twitching, delerium, hallucinosis, apprehensive behavior, and convulsions. It must be stressed that most of their patients were chronic alcoholics. Symptoms of hypokalemic alkalosis were recently observed in a magnesium-depleted patient given ten ounces of whiskey daily (24). R y a n (77) has recently observed that magnesium depletion can interfere with normal regulation of intracellular potassium. The hypertension, muscle rigidity, twitches, tremor, and convulsions

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have also been observed in magnesium-deficient children (3) and in the new-born (10). It has also been found that magnesium levels are low in hypertensive men (7), and that magnesium therapy alleviates hypertension in rats (17).

The tremor, twitching, and muscular tension are probably the result of reactions affecting the myoneutral junction and the motor-end-plate (cf 6, 83). Apparently, an adequate ratio of Mg/Ca is essential to ensure proper control of the muscles (11). However, the role of phosphate cannot be disregarded, especially as Martindale and Heaton (62) have shown that — at about the time of a convulsive seizure — a three fold increase in serum phosphate is observed. Although the antagonistic effects of calcium and magnesium on acetylcholinesterase activity have been cited (83), the large increase in serum phosphate at the time of convulsion suggests an impediment in phosphate utilization, which is probably also enzyme-related, considering the well-established role of magnesium in transphosphorylation processes (cf 70). Magnesium insufficiency probably inhibits oxidative phosphorylation (1, 45).

One of the diseases most associated with tremor and twitching is, of course, Parkinson's disease. Barbeau et al. have demonstrated that Parkinsonian patients have a serum magnesium level which is about 30 % lower than normal subjects (5, 6). Barbeau also reported a reduced urinary excretion of serotonin and serotonin derivatives in this disease (6). However, the latter observation may reflect the urinary status in advanced Parkinson's disease; information on urinary excretion of these metabolites during the early stages of this degenerative ailment might be of value on assessing the sequence of events. Other muscular ailments might be related to magnesium metabolism. Tollersrud (85) has found that magnesium supplements prevent the occurrence of muscular dystrophy in Vitamin E deficient calves. It remains to be seen whether magnesium plays a significant role in this, and other, muscular diseases.

Table 5 • Conditions Where Hypomagnesaemia May Be Found in Human Beings.
(Source: ref. 58)
1. Malabsorption syndrome
2. Prolonged or severe loss of body bluids
3. Chronic alcoholism
4. Diuretic therapy (especially large thiazide doses)
5. Renal tubular acidosis or necrosis
6. Portal cirrhosis
7. Primary aldosteronism
8. Primary hyperparathyroidism
9. Hypercalcaemia from any cause
(From other sources)
10. Post-operative hypoparathyroidism (47)
11. Epileptiform seizures (3, 10, 25, 84)
12. Malnutrition (3, 10, 84)
13. Hypertension (7)
14. Leukemia (57)

Hypomagnesaemia has been observed in connection with several human ailments (Table 5). These ailments have been alleviated by intravenous, intramuscular or oral magnesium therapy (3, 10, 26, 29, 39, 41, 42, 47, 59, 84). The above list does not include the muscular diseases previously alluded to; neither does the list include several "curio" findings, one of which concerns cattle exhibiting "grass tetany staggers". This phenomenon occurs when cattle graze on early spring grass, and is associated with hypomagnesaemia, even though the magnesium intake is normal (15, 75). The only abnormality appeared to be the high organic content of the early spring grass (15). Recently, Burau and Stout (14) have found that such grasses can contain up to 12 % of aconitate, which is an isomer of citrate and has very similar properties. If one recalls Samir Hanna's "absorption-renal" function of citrate (40, 43), it is not difficult to follow the sequence of events, in which the high intake of aconitate would stimulate absorption of both calcium and magnesium, but would also stimulate renal excretion of both. However, a recent study indicates that this "spring grass syndrome" is caused by a reduced intestinal absorption of magnesium (16). This might explain why dietary supplements of magnesium have been the only effective means of coping with the affliction (75).

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Another "curio" finding concerns the interrelation of magnesium and fluoride. When very high levels of both components were fed to growing chicks, it caused noticeable leg weakness (30, 37), and reduced the mineral content of bone, i. e., total ash, calcium, phosphorus, and citrate (38), but increased the magnesium content of bone (37). The leg weakness symptoms were not noticed in older subjects, which suggests a transient stage in growth during which the subjects would be particularly vulnerable. Interference with vital enzymatic processes has been suspected (30, 37, 38). However, the situation in bone (38) suggests a rachitic condition, which may be the result of a high-magnesium law-citrate condition, compounded by the presence of fluoride. Belanger et al. (8) have reported that high fluoride supplements can produce a rachitic-like condition. Also, rachitic bone has an increased magnesium content (cf 29).

Another aspect of the magnesium and fluoride interrelation was reported in studies of experimental hypomagnesaemia by Phillips et al. (12, 19, 20, 85). In magnesium-deficient dogs, fluoride supplements prevented soft-tissue calcification, but not the muscle weakness and convulsions. In rats, fluoride aggravated the hypomagnesaemia which, as might be expected, intensified the convulsive seizures in these rats. Again, we must note the somewhat different effects in the two species. Nevertheless, the symptoms of magnesium deficiency are remarkably similar to those of fluoride intoxication. The reason for this is most probably due to a fluoride-induced increase in the uptake of magnesium by bone (cf 60). Even small increases in bone magnesium can have serious effects, because bone can contain up to 63 % of body magnesium (1), whereas body fluids account for

Table 6 • Symptoms Common to Both Fluoride Intoxication and Magnesium Deficiency.

Symptom	Fluoride Intoxication	Magnesium Deficiency
Leg cramps, or "pins and needles"	Reference 78	Reference 29
Muscular twitching	Reference 51, 87	Reference 41, 84
Tetaniform convulsions (with normal serum Ca)	Reference 51, 87	Reference 29, 62
2 to 3-fold increase in serum P at time of convulsion	Reference 51, 87	Reference 62
Cataracts (optical neuritis)	Reference 31	Reference 29
Bone exostoses and/or soft-tissue calcification	Reference 90	Reference 71

Note: With the exception of references 62, 71, and 90, all of the above observations were made on human subjects.

only about 1.0 % (83). Table 6 lists some of the symptoms common to both magnesium deficiency and fluoride intoxication. It may be that nonskeletal magnesium depletion will someday be recognized as one of the "key" symptoms of fluoride intoxication. The interrelation of fluoride intake and magnesium deficiency remains to be elucidated in the human being.

The final "curio" phenomenon concerns the recent observation that magnesium deficiency can induce leukemia in the rat (57). Thus, we see that there are many clinical conditions in which magnesium can be involved. More attention should be paid to the role of magnesium, especially since the daily requirement for this important mineral is probably much higher than previously supposed (80).

Zusammenfassung

Bedeutung des Magnesiums in der Nahrung mit besonderen Hinweisen auf den menschlichen Körper

Magnesium wird von verschiedenen metabolischen Standpunkten aus besprochen, wie z. B. seine Verteilung im menschlichen Körper, Ernährungserfordernisse, Verfügbarkeit in Nahrungsmitteln, Resorption, Hormonregelung, Ausscheidung und die Wechselwirkungen mit Kalzium, Kalium, Phosphor, Zitrat und Vitamin D. Ferner geht der Autor auf die Symptomotologie des Magnesiummangels ein, wobei die Möglichkeit seiner Beteiligung an mehreren metabolischen Störungen erörtert wird, z. B. Kalzinose der weichen Gewebe, erhöhter Blutdruck, „delirium tremens", Hypercholesterinämie, Muskelschwund, Parkinsonsche Krankheit, Fluoridvergiftungserscheinungen, Krampfanfälle und Leukämie.

Summary

The Importante of Dietary Magnesium with Particular Reference to Humans

This review article outlines various metabolic aspects of magnesium, i. e., its distribution in the human body, dietary requirements, availability in foods, absorption, hormonal control, excretion, and the interrelations with calcium, potassium, phosphorus, citrate and vitamine D. The symptomatology of magnesium deficiency is discussed, along with its possible involvement in several metabolic derangements, i. e., soft-tissue calcinosis, hypertension, Delirium Tremens, hypercholesterolaemia, muscular dystrophy, Parkinson's disease, fluoride toxicosis, convulsive seizures and leukemia.

Résumé

L'importance du Magnésium Alimentaire en Rapport surtout au Genre-Humain

Cet article traite de divers aspects métaboliques du magnésium, c'est-à-dire, sa distribution dans l'organisme humain, la teneur recommandée dans la diète, son absorption, le processus de contrôle endocrinologique, l'excrétion, ainsi que les interrelations envers le calcium, le potassium, le phosphore, le citrate et la vitamine D. La symptomatologie de la carence magnésienne est discutée, en rapport de son rôle possible dans plusieurs troubles métaboliques, tels que la calcinose des tissus mous, l'hypertension, le «delirium tremens», l'hypercholestérolémie, la dystrophie musculaire, la maladie de Parkinson, la toxicité fluorique, les convulsions, et la leucémie.

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Ernährung und Gesundheit
Nutrition and health
Alimentation et santé

Die anschauliche Darstellung von Nahrungsbrennwert und Nährwertträger — Relationen durch Nomogramme

Von A. von Klein-Wisenberg, Freiburg

Eine Graphik kann eine sehr anschauliche Darstellung eines abstrakten, in Zahlen tabellarisch gegebenen Sachverhaltes bieten. Die üblichen Kurvenschaubilder zeigen im allgemeinen die Abhängigkeit einer Größe von einer anderen, die Wirkung einer Ursache. Man kann dasselbe, weniger sinnfällig, mit einer Tabelle zeigen. Die sogenannte Nomographie* gibt uns ein Mittel an die Hand, mit den graphisch versinnbildlichten Größen auch zu rechnen.

Rechenhilfsmittel

Die menschliche Arbeitskraft von ermüdender mechanischer Tätigkeit zu entlasten und dadurch für eine ordnende Steuerfunktion freizumachen, ist das erklärte Ziel aller Rationalisierungsbemühungen. Bei den Rechenhilfsmitteln läuft das hinaus auf eine Verminderung des Schwierigkeitsgrades oder auf eine Mechanisierung des Rechnens. Mit Hilfe der Rechenbretter werden die Ergebnisse von Additionen oder Subtraktionen nach einem Zählvorgang ersichtlich, die logarithmischen Rechentafeln führen Multiplikationen auf Additionen und Divisionen auf Subtraktionen zurück — die Verminderung des Schwierigkeitsgrades der Rechnung ist zuerst ersichtliches Leitziel. Das Rechenbrett, bei dem auf vorgezogernen Linien Rechenmünzen, Steine oder Muscheln verschoben wurden, wird Abakus genannt.

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* gr. nomos = Gesetz, graphein = zeichnen