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Increased need for magnesium with the use of combined oestrogen and calcium for osteoporosis treatment

Mildred S. Seelig
Community and Preventive Medicine, New York Medical College, Valhalla, New York; American College of Nutrition, Wilmington, N. Carolina, USA.

Summary: Prophylactic treatment of postmenopausal osteoporosis with oestrogen and calcium, often in combination, disregards the likelihood that an excess of each agent may increase magnesium requirements and decrease serum Mg levels. Relative or absolute Mg deficiency, which is likely in the Occident where the Mg intake is commonly marginal, can militate against optimal therapeutic bone response, Mg being important for normal bone structure, and can increase the risk of adverse effects. Although oestrogen has cardiovascular protective effects (expressed by the lower incidence of heart disease in premenopausal women than in men, and also in postmenopausal women given low dosage oestrogen replacement treatment), high dosage oestrogen oral contraceptives have caused increased intravascular blood clotting with resultant thromboembolic cardio- and cerebrovascular accidents. This might be contributed to by the oestrogen-mediated shift of circulating Mg to soft and hard tissues, which in persons with marginal Mg intakes may lead to suboptimal serum levels. If the commonly recommended dietary Ca/Mg ratio of 2/1 is exceeded (and it can reach as much as 4/1 in countries with low to marginal Mg intakes), relative or absolute Mg deficiency may result, and this may increase the risk of intravascular coagulation, since blood clotting is enhanced by high Ca/Mg ratios. Mechanisms by which Ca activates the various steps in blood coagulation that are also stimulated by oestrogen are considered here, as are the multifaceted roles of Mg that favourably affect blood coagulation and fibrinolysis, through its activities in lipoprotein and prostanoid metabolism.

Key words: Alcoholic, bone structure, calcium therapy, Ca/Mg ratio, combination therapy, diabetic, intravascular coagulation, malabsorption, Mg deficiency, Mg lipoproteins, Mg requirements, oestrogen therapy, osteoporosis prophylaxis, postmenopausal, prostanoids.

Introduction

Far less appreciated than calcium in the treatment or prevention of osteoporosis is the importance of magnesium. Osteoporotic bone has subnormal Mg content, such as is found in Mg deficiency. Many factors interact in the vulnerability to relative or absolute Mg deficiency of those prone to the development of osteoporosis. Low Mg intake, decreased Mg absorption, and hormonal changes that influence the distribution of magnesium and its effects on vitamin D metabolism are major factors in the elderly. In postmenopausal women, among whom are found most of the cases of osteoporosis, loss of oestrogen – which affects distribution of both Ca and Mg – is the pre-dominant factor. The marginal Mg content of many Western diets can be contributory. Other conditions in which osteoporosis has developed, such as chronic alcoholism, disease- or therapy-induced Mg malabsorption or renal wasting, and poorly controlled diabetes mellitus increase Mg deficiency, the latter by increasing urinary Mg output and decreasing its tissue uptake. Since Mg deficiency causes changes in bone structure, the effect on Mg of agents used in prevention and treatment of osteoporosis should be considered. Oestrogen replacement delays onset and retards progression of postmenopausal osteoporosis, partly by antagonizing mobilization of both Ca and Mg by parathyroid hormone (PTH). The effect of oestrogen in lowering serum Mg by shifting Mg to
Factors that increase magnesium inadequacy in those at risk of osteoporosis

**Diabetes mellitus** — Diabetes mellitus is a disease that is characterized by Mg depletion, caused by complex mechanisms. The extent of the Mg loss in diabetes is related to the severity of the disease. Osteoporosis is not infrequent in insulin-dependent diabetes13,14. Severe hypomagnesemia has long been associated with diabetic acidosis13,14. Less profound reductions in serum Mg have been demonstrated in juvenile diabetics15,16. Contributory to the Mg depletion in diabetic acidosis, which increases Mg output in the urine even in normal subjects given glucose loads17,18. Insulin is important in cellular uptake of Mg19,20, in its long term inadequacy or with refractoriness to its activity, diminished tissue Mg — including that of bone — is likely.

**Interrelations between magnesium and calcium**

High calcium intake with marginal or low magnesium intake

To compensate for the loss of Ca from osteoporotic bone, oral treatment with Ca and vitamin D as a calcemic agent is common, without taking into consideration the effect of these agents on Mg requirements, or the importance of Mg in maintaining normal bone matrix11. High dietary Ca/Mg ratios interfere with Mg absorption, partially because Ca and Mg share a common intestinal transport pathway21,22. Vitamin D favors Ca over Mg absorption23. Metabolic studies have shown interference by high Ca diet on the serum levels of normal young women24, and of patients with osteoparthisis (Fig. 2)25,26. Moderately high Ca intake (1270 to 2500 mg/day) had little effect on the Mg balance of elderly women27. Similarly, addition of 900 mg Ca/day to young men's diets containing 700 mg Ca had little effect on Mg balance28. However, even though the Mg balance was not affected by up to 2 g/day of Ca in normal young men, their plasma Mg levels fell29.

**Effect of magnesium supplementation on hypocalcemia**

**Interference of Mg absorption with parathyroid hormone —** Low Ca levels, despite adequate dietary Ca, may result from Mg deficiency, as well as from Ca deficiency. It has long been hypothesized that Mg deficiency can be a manifestation of severe Mg deficiency in adults30,31,32,33, as well as in infants with convulsive hypocalcemia34,35,36. Their refractoriness to calcitriol is caused by impaired release of PTH and target organ (bone and kidneys) resistance to PTH37,38,39. However, early or less severe Mg deficiency increases PTH secretion, which increases bone mineral mobilization not only of Ca but also of Mg in human32,33,34. Studies of adults with hypercalcemia secondary to Mg deficiency showed that most had been impaired PTH secretion and renal resistance to PTH40,41. Mg deficiency is required for activity of adenylate cyclase in parathyroid tissue42, in kidney43,44. Enzyme converts ATP to cyclic AMP (cAMP)45, which is needed for PTH secretion46. Defective cAMP generation may thus be responsible for both hypocalcemia and hyperparathyroidism and for its impaired secretion in severe Mg deficiency47.

**Vitamin D Interrelations with magnesium —** Vitamin D deficiency impairs not only Ca absorption, but that of Mg25,26,27. Formation of the active hormonal metabo-

![Fig. 1. Factors affecting magnesium supplements, of particular significance in the aged (from M.S. Seelig2).](image)

**Fig. 1.** Factors affecting magnesium supplements, of particular significance in the aged (from M.S. Seelig2).

**Magneeum deficiency and decreased bone magnesium in osteoporosis**

Significantly increased retenion of Mg after a parenteral Mg load in osteoporotic patients2. The most reliable tests for Mg deficiency1,18—20, has been found in postmenopausal patients with osteoporosis13,14. A study of Mg levels in serum and bone specimens of 10 elderly women (58.8 ± 2 years) with osteoporosis, compared with serum Mg of age-matched women free of bone disorder and with necropsy bone of women who had died suddenly, showed markedly lower serum Mg in the controls (1.74 mg/litre) and low Mg (1.69 mg/litre) in the osteoporotic women23. The bone Mg of the osteoporotic women (1.54 ± 0.29 mg/g) was lower than that of sudden death victims (1.73 ± 0.23 mg/g). There was low Mg absorption in 12 of 20 postmenopausal osteoporotics13. Loss of trabecular bone (in which the bone crystals are abnormal24—25) is associated with trabecular bone Mg in postmeno-

![Fig. 2. Effect on magnesium balance of calcium supplementation in patients with hypoparathyroidism (adapted from D. Ammou, D.J. Hoorn, J. Durlach: J. de Med., Bruxton S, 371—378, 1969, in M.S. Seelig4)].(image)

**Fig. 2.** Effect on magnesium balance of calcium supplementation in patients with hypoparathyroidism (adapted from D. Ammou, D.J. Hoorn, J. Durlach: J. de Med., Bruxton S, 371—378, 1969, in M.S. Seelig4).
Mildred S. Seelig

MAGN+S+NM AND OSTE+GEN THERAPY

Table 1. Plasma, bone and muscle magnesium and calcium levels in rats treated with ethinyl estradiol +/- progesterin (adapted from Charoen, G romance & Paireis119)

<table>
<thead>
<tr>
<th></th>
<th>Plasma</th>
<th>Bone</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>mg/dl</td>
<td>Mg</td>
<td>Ca</td>
<td>Mg</td>
</tr>
<tr>
<td>Control</td>
<td>2.5</td>
<td>8.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Ethinyl estradiol</td>
<td>2.4</td>
<td>8.1</td>
<td>1.7</td>
</tr>
<tr>
<td>Northionate</td>
<td>2.3</td>
<td>8.0</td>
<td>1.6</td>
</tr>
<tr>
<td>Combination</td>
<td>2.2</td>
<td>7.9</td>
<td>1.5</td>
</tr>
</tbody>
</table>

Table 2. Contrast between Ca and Mg effects in blood coagulation; comparison with oestrogen effect

<table>
<thead>
<tr>
<th>Factor VII</th>
<th>Factor X</th>
<th>Prothrombin</th>
<th>Antithrombin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Dependent</td>
<td>Inhibited</td>
<td>Increased</td>
</tr>
<tr>
<td>Ca</td>
<td>Dependent</td>
<td>Inhibited</td>
<td>Increased</td>
</tr>
<tr>
<td>Mg</td>
<td>Dependent</td>
<td>Inhibited</td>
<td>Increased</td>
</tr>
</tbody>
</table>

Table 3. Plasma magnesium in postmenopausal women (adapted from Taylor, Paireis & Seelig117)

<table>
<thead>
<tr>
<th>Calcium</th>
<th>Magnesium</th>
<th>High</th>
<th>Osteogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor VII:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>Normal</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Factor X:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activated</td>
<td>Dependent</td>
<td>Dependent</td>
<td>Inhibited</td>
</tr>
<tr>
<td>Prothrombin:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activated</td>
<td>Increased</td>
<td>Decreased</td>
<td>Increased</td>
</tr>
</tbody>
</table>

Fig. 3. Serum and bone Mg, Ca, P, bone strength and MLI in estrogen-naive (E+) and low, optimal and high Mg breakers (derived from E.R. Miller et al., reproduced from M.S. Seelig, page 296).

Fig. 4. Urinary magnesium of young women using oral contraceptives X and of postmenopausal women (decreased in hyperparathyroid women). Oestrogen antagonizes Mg-induced bone resorption in rats and mice, and bone of osteoporotic rats is more sensitive to PTH as measured by Ca and hydroxyproline content and decreased bone thickness. Loss of estrogen at menopause causes negative Ca balance and greater bone responsiveness to the Ca-mobilizing effect of PTH, and decreased formation of calcitriol. These findings, and the evidence that oestrogen inhibits the bone-resorbing effect of PTH, support the clinical use of oestrogen in osteoporosis. In addition, the loss of the protective effect of oestrogen on bone – mediated both directly by PTH release and indirectly by decreased mineralization – is the cause of postmenopausal osteoporosis. Both of these effects may be influenced by the effects of oestrogen on Mg.

Oestrogen effect on vitamin D metabolism – role of Mg

Low calcium intake and low activity of enzymes involved in its formation, are associated with old age and with involutional osteoporosis. The response of the disease to its administration is inconsistent. The need for more parathyroid stimulation for normal vitamin D metabolism by women with osteoporosis, and the lesser response of the elderly to calcium, has been correlated with possible Mg deficiency. The level of calcium has been raised during physiologically increased oestrogen, for example during pregnancy, and during use of oestrogen. It has been suggested that impaired activation of 1,25-dihydroxy-calcitriol might be responsible for the oestrogen-deficient induced decreased formation of the active vitamin D metabolite. On the grounds that low levels of the active form of vitamin D were found in Mg deficiency, and that oestrogen increases Mg reabsorption, it is suggested that the increase in calcitriol formation caused by oestrogen might be mediated by increased tissue Mg. Worth exploring is the relationship to osteoporosis of the lower serum Mg and decreased urinary Mg loss by women on oestrogen therapy in comparison with those not treated. Does this reflect a shift to bone, as in rats? Does increased urinary Mg of postmenopausal women reflect their inability to retain Mg? It has been noted that low trabecular bone mass, and abnormal bone crystals, seen in pelvic crest biopsies of osteoporotics, are different in postmenopausal women receiving oestrogen.

Magnesium deficiency-associated thromboses; mechanisms

Magnesium/calium in blood coagulation

That Ca activates blood clotting, and that Mg antagonizes this effect, has been known since 1944; it was assumed when corrected for haemodilution, serum Mg by Ar A. Goulding & McChesney, reproduced from M.S. Seelig.
platelet aggregation were unaffected. Hypercoagulability of blood of Mg-deficient rats, produced by feeding diets rich in saturated fat to animals (see below), decreased coagulation time and increased prothrombin consumption and could be prevented by adding Mg to the diet.\textsuperscript{171,172}

\textit{Prostacyclin and thromboxane} — There is some evidence that Mg deficiency inhibits synthesis of prostacyclin (a vasodilating, anti-platelet-aggregating, anti-hypertensive prostaglandin), and decreases release of thromboxane A2 and B2 (TXB2), which are vasoconstrictive, pro-aggregating prostaglandin from platelet aggregates. It has been hypothesized that the accelerated platelet clumping produced by platelet-activating collagen (as a model of the bare subendothelial collagen of damaged vascular intima) in Mg-deficient lambs was mediated by diminished production of prostacyclin.\textsuperscript{173}

The release of increased amounts of arachidonic acid, the precursor of prostaglandins, from thromocytes of Mg-deficient rats\textsuperscript{174} led to study of the effect of Mg deficiency on these derivatives of phospholipid metabolism in blood.\textsuperscript{175} Prostacyclin, as measured by its stable metabolite 6-keto-PGF1\textsubscript{a}, was increased two-fold; PGI2 was increased three-fold, but TXB2 was increased more than ten-fold. These increases were attributed to increased Ga levels in Mg-deficient rats, since the enzyme responsible for liberation of arachidonic acid from phosphatidylethanolamine is Mg-dependent. The depression of cyclic AMP seen in Mg deficiency may also participate directly in the markedly increased TXB2 synthesis of Mg deficiency, since cyclic AMP inhibits TXB2 production by platelets.\textsuperscript{176} Fatty acid metabolism is altered in Mg deficiency, with arachidonic acid production diminished as a result of slowed conversion of linoleic acid to arachidonic acid,\textsuperscript{177} a finding pertinent to the role of prostaglandins in blood coagulation.

Dietary Mg depletion of humans has recently been shown to increase platelet aggregation and TXB2 release, effects that were reversed by Mg infusion.\textsuperscript{178} A nine-fold increase in serum TXB2 was seen in a marathon runner immediately after racing, when his serum Mg was lowest (1.02 mg/100ml), as compared with the pre-race value.

Increasing Mg intake, in experimental Mg deficiency, increases Mg requirements, as expressed by the anti-coagulative and cardiovascular-protective effects of adding Mg to thrombogenic, hyperlipidemic, or infarctoid diets.\textsuperscript{179,180} Experimental Mg deficiency has long been known to affect serum lipoproteins.\textsuperscript{181-183} Mechanisms involved in Mg lipid interrelations, as they affect coagulation and vascular disease, are being elucidated. Mg deficiency of weaning rats has caused hypertenproproteinemia with increases in very low density lipoprotein (VLDL), and low density lipoprotein (LDL) fractions, with increased cholesteryl in those fractions, but decreased cholesteryl in the high density lipoproteins (HDL).\textsuperscript{184-186} (Fig. 5). Defective triglyceride removal from the blood in Mg-deficient rats\textsuperscript{187} is associated with reduction of plasma lecithin cholesteryl ester transferase (Fig. 6), and of lipoprotein lipase.\textsuperscript{188}

\textbf{Magnesium deficiency-associated thromboses: clinical aspects and treatment}

\textbf{Merital clinical magnesium deficiency}

Patients with latest stage of marginal Mg deficiency have developed thromboses and emboli; new events were prevented by Mg supplements, and recurred when supplements were discontinued.\textsuperscript{189-192}

\textbf{Postpartum: magnesium-sparking anasarca}

An early study showed that blood taken from surgical patients, who had been anesthetized with an Mg-containing preparation, clotted but then liquefied completely within 2 hours; blood of such patients at autopsy 12 hours after death was liquid.\textsuperscript{193} Because there were no cases of phlebothrombosis or embolism among 120 survivors of major surgery so anesthetized, the investigator undertook clinical trials of the presumed thrombotic effect of Mg. Patients with deep vein phlebothrombosis responded to parenteral Mg therapy; oral Mg therapy was prescribed prophylactically, with resultant decreased platelet aggregation and inconsistent thrombolysis. In a confirmatory study of the improved recovery of patients with thrombophlebitis or phlebothrombosis treated with intramuscular or oral Mg (providing 80 mg/mo/ampule and 50 mg/tablet), blood coagulation indices ( clot formation, prothrombin times, antithrombin) were unaffected, but fibrinolysis increased substantially.\textsuperscript{194}

\textbf{Pregnancy-associated thrombotic disease}

\textbf{Pregnancy and puerperium} — Pulmonary embolism is a major cause of death during pregnancy and the puerperium, which suggests that the naturally high oestrogen secretion at that time increases intravascular coagulation. Comparison of incidence of postpartum phlebothrombosis, prothrombin time, and antithrombin Mg, followed by oral Mg (200 mg three times/day for 3-10 days postpartum), provided after delivery, was compared with the phlebitic events during a prior year in which that regimen was not followed. Those treated with Mg exhibited substantial reduction in postpartum thromboses, after spontaneous or operative delivery (Table 5)\textsuperscript{195}. The common denominator of all of these magnesium-depletion syndromes is the Mg deiniciency described above, as a result of intracellular coagulation.\textsuperscript{196} (See above for interaction with prosta-

<p>| Table 3. Effect of magnesium on lipids in rats on high fat diet (Adapted from Vitalae, Hollestein, Nakamura et al. 1954) |
|-----------------|----------------|----------------|
| Fat intake      | Cholesterol   | Lipoproteins   |</p>
<table>
<thead>
<tr>
<th>Sr</th>
<th>Low Mg</th>
<th>High Mg</th>
<th>Low Mg</th>
<th>High Mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>20% Saturated</td>
<td>15</td>
<td>97</td>
<td>15</td>
<td>97</td>
</tr>
<tr>
<td>20% Un saturated</td>
<td>110</td>
<td>102</td>
<td>81</td>
<td>83</td>
</tr>
</tbody>
</table>

| Table 4. Lipids in experimental magnesium deficiency in dogs |
|-----------------|----------------|----------------|
| Mg intake       | Fat intake     | Reference      |
| (0.00%) of diet | (8%) of diet   |                |
| Butter fat      | Corn oil       |                |
| Kg                | Kg              |                |
| 0                | (5%) of diet    |                |
| Mg                | No change in total serum lipids |                |
| 80 ppm           | No change in blood cholesterol |                |
| 100 ppm          | Aortic lesions  |                |
| No change in total serum lipids |                |
| 0                | Cardiogenic    |                |
| Also free of K | Serum cholesterol |                |
| High in vitamin D; Ca, P04, protein |                |

Fig. 5. Effects of magnesium deficiency on triglycerides and cholesterol (in rats) (reproduced from Y. Raynsquier).
Table 6. Blood lipid changes in 50 patients with ischaemic heart disease after treatment with 2 ml 50% MgSO₄, at 5 days intervals x 12 derived from R.S. Parissis, I.C. Butler & E.P. Sellers.***

<table>
<thead>
<tr>
<th>Reduced</th>
<th>No change</th>
<th>Increased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>67%</td>
<td>12%</td>
</tr>
<tr>
<td>Lecithin/cholesterol</td>
<td>0%</td>
<td>14%</td>
</tr>
<tr>
<td>α-lipoproteins</td>
<td>60%</td>
<td>28%</td>
</tr>
<tr>
<td>β-lipoproteins</td>
<td>60%</td>
<td>28%</td>
</tr>
</tbody>
</table>

Changes in fibrinolytic potential after Mg treatment:

- Plasmin activation: 10% increase
- Plasmin inhibition: 14% increase

MAGNESIUM AND OSTEORENOSIS THERAPY

Table 7. Platelet adhesives and serum Mg in myocardial infarction (Adapted from C. Prakash et al., Proc. Intl. Mg Symposium, 1971/1973)

<table>
<thead>
<tr>
<th>Mg (mg/ml)</th>
<th>Platelet adhesives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normals</td>
<td>3rd d</td>
</tr>
<tr>
<td>70</td>
<td>1.93</td>
</tr>
</tbody>
</table>

The anti-atherosclerotic effect of Mg is implicated in the improved survival of post-myocardial infarction patients treated with Mg infusions; its antithrombotic effect may also participate.

Cardiovascular protection by estrogen

The lower cardiovascular disease death rate in premenopausal women has long been thought to indicate that estrogen protects against cardiovascular disease. However, it is now known that estrogen reduces the risk of myocardial infarction even in postmenopausal women. The protective effect of estrogen is further enhanced when combined with aspirin or other antithrombotic agents.

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may develop relative or absolute Mg deficiency, with suboptimal serum Mg levels. Combined Ca and oes-
trogen treatment of such patients might lower serum Mg, thereby increasing the risk of intravascular
coagulation. Hypocalcaemia has been ascribed to a common use of low dose replacement oestrogen to prevent or slow osteoporosis is not considered to be at risk of thromboembolic events. It has been shown that As the acti-
Deactivates many blood coagulation factors, with a coagulation, and even has some fibrinolytic activity. Furthermore, it has been shown to participate in actives that may function to improve the cutaneous and prostatic abnormality associated with Mg deficiency and that increase the risk of thrombosis. Thus Mg supplementation is suggested to reduce the risk of clot formation, and possibly to improve the anti-thrombotic therapeutic effect.

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Besoins magnésiés traduit par l'utilisation d'estrogènes et de calcium associés dans le traitement de l'ostéoporose

M.S. Seelig (Wilmingtom, N.C., USA).

Résumé: Le traitement prophylactique de l’ostéoporose post-ménopausique: estrogène et calcium (Ca), souvent associés, dissimule la probabilité qu’un excès de l’un ou de l’autre agent accroît le risque de besoins magnésiés accrus et d’hypercalciuries sériques. La déclivité magnésiée relative ou absolue qui est probable en Occident où l’apport magnésiée est fréquemment marginal peut intervenir en mal être et de réponse thérapeutique optimale, le magnésiée étant important comme statut normal de l’os et doit atténuer le risque de défaut secondaire féminin. Bien que les estrogènes puissent avoir des effets protecteurs cardiovasculaires (important à souligner le plus faible incidence des affections cardiovasculaires chez la femme avant la ménopause que chez l’homme ou chez les femmes agées sous traitement par de faibles doses palliatives d’estrogènes), les traitements contraceptifs par de faibles doses normalement d’estrogènes ont provoqué une augmentation de la coagulation sanguine intravasculaire avec comme résultat des accidents thromboemboliques cardio- et cérébrovascularis. Ils peuvent dépendre de l’âge apparent des estrogènes entre magnésiée circulante et tissus mous et durs, ce qui face à des apports magnésiés marginaux peut amener à des concentrations sériques suboptimaux. En dépassant le rapport ingéré habituellement recommandé de 2/1 pour Ca/Mg (et qui d’ailleurs peut atteindre 4/1 dans les régions à faible ou marginal ingesta magnésiée), on peut augmenter le risque de coagulation intravasculaire, la coagulation sanguine étant augmentée par des taux élevés du rapport Ca/Mg ingéré. Les mécanismes de coagulation multiples étapes de la coagulation sanguine sont aussi stimulés par les estrogènes. Ils sont analysés comme le sont les rôles à multiples facettes dûe Mg favorables sur la coagulation sanguine et la fibrolyse, à travers ses activités sur les métabolismes des lipoprotéines et des prostanoïdes.

Mots-clés: Alcoolisme, besoins magnésiés, calcithérapie, coagulation intravasculaire, déficience magnésiée, diabète, lipoprotéines, magnésiée, malabsorption, postménopause, prévention de l’ostéoporose, progestatif, rapport Ca/Mg, structure de l’ostéoporose traitement.

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