Intestinal Absorption of Magnesium from Food and Supplements

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Abstract

The purpose of this study was to measure magnesium absorption over the wide range of intakes to which the intestine may be exposed from food and/or magnesium-containing medications. Net magnesium absorption was measured in normal subjects after they ingested a standard meal supplemented with 0, 10, 20, 40, and 80 mEq of magnesium acetate. Although absorption increased with each increment in intake, fractional magnesium absorption fell progressively (from 65% at the lowest to 11% at the highest intake) so that absorption as a function of intake was curvilinear. This absorption–intake relationship was almost perfectly represented by an equation containing a hyperbolic function plus a linear function. Our results are statistically compatible with a magnesium absorption process that simultaneously uses a mechanism that reaches an absorptive maximum, plus a mechanism that endlessly absorbs a defined fraction (7%) of ingested magnesium. Compared to previous studies of calcium absorption, much less magnesium than calcium was absorbed at intakes above 8 mEq/meal, apparently due to greater restriction of intestinal permeability to magnesium. We also found that magnesium from a high magnesium-containing food source, almonds, was just as bioavailable as from soluble magnesium acetate. In contrast, magnesium absorption from commercially available enteric-coated magnesium chloride was much less than from magnesium acetate, suggesting that enteric coating can impair magnesium bioavailability. (*J. Clin. Invest.* 1991. **88:**396–402.) Key words: biological availability • calcium • phosphorus • regression analysis • tablets • enteric-coated

Introduction

Normally about 7.4 mEq (90 mg) of dietary magnesium is eaten per meal (1, 2). However, when a Mg supplement is prescribed for suspected or overt Mg deficiency, 20 mEq of elemental Mg may be ingested in a single dose. Intake is even higher with use of antacids or laxatives, when Mg content per dose may be 40 or 80 mEq, respectively. Thus, oral intake of Mg at a single time during the day can vary widely.

The main purpose of this study was to measure net Mg absorption over the wide range of Mg loads to which the normal intestine may be exposed. Previous studies on this question, using the metabolic balance method, are inconclusive because most reports used only a few subjects, because patients rather than normal subjects were sometimes studied, because the range of Mg intake was usually not systematically varied, and/or because the reports used balance periods that are too short for valid results. (A 3-wk balance period is required for methodologic accuracy [3, 4].) Nevertheless, King and Stanbury pooled many previously reported metabolic balance studies, and showed that fecal Mg output was linearly correlated with Mg intake and that the regression line passed through zero (5). Converting these results to net absorption as a function of intake suggests that ~ 38% of ingested Mg is absorbed, regardless of intake. However, this analysis was limited to intake values between 100 and 500 mg/d, and its conclusion is inconsistent with some of the individual reports from which the pooled data were obtained.

The studies we report in this paper were carried out by a method wherein net absorption can be measured accurately after ingestion of a single meal (6, 7). Using this method, net Mg absorption was measured over a wide range of intakes by supplementing a standard meal with increasing amounts of a soluble Mg salt, Mg acetate (MgAc).[1] We also measured Mg absorption from an enteric-coated soluble Mg salt (Mg chloride) and from almonds (a food source high in Mg).

Methods

Subjects. Eight subjects aged 25–35 yrs old volunteered for the study, and informed consent was obtained. All volunteers were healthy males, taking no medications. Six did not drink alcohol and two averaged 45 g of alcohol (two to three beers) per wk; they did not drink during the study. Baseline serum Mg, serum 1,25-$(OH)_2$-Vitamin D, and 24-h urinary Mg excretion are given in Table I; all subjects had normal values.

Preparation of the meals and supplements. The meals and supplements given on each experimental day are listed in Table II. All meals included 10 g of polyethylene glycol (PEG), as a nonabsorbable marker (8), dissolved in 250 ml of deionized water. Food used in the study was prebought to insure uniformity between days and subjects. Portions were weighed in duplicate to the nearest milligram before cooking, and two meals were prepared in a metabolic kitchen. One meal was given to the subjects to eat and the duplicate meal was analyzed. According to dietary tables (9, 10), the standard meal contained 435 kcal and the meal with almonds (Table II) contained 676 kcal. The average measured content of Mg, Ca, and P in the duplicate meals is given in Tables II and III; the amount of ingested Ca and P was not constant because of the extra Ca and P contained in Slow-Mag® (G.D. Searle and Co., Chicago, IL) and almonds, respectively.

Reagent grade MgAc tetrahydrate (Aldrich Chemical Co., Inc., Milwaukee, WI) was measured in duplicate to the nearest milligram and manually "loaded" into gelatin capsules (Eli Lilly and Co., Indianapolis, IN). One set of capsules was ingested with the meal (immediately before the first bite); the other was analyzed for Mg content. A similar protocol was followed, on appropriate days, for Slow-Mag® and almonds.

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1. *Abbreviation used in this paper:* MgAc, magnesium acetate.

Table I. Baseline Laboratory Values

Subject	Serum Mg	Serum 1,25-(OH)$_2$-Vitamin D	24-h urinary Mg excretion
	mEq/liter	pg/ml	mEq/d
Normal range	1.4–2.1	15–60	>2 mEq/d
1 (JL)	1.9	36	9.6
2 (EF)	1.6	41	3.2
3 (AR)	1.7	30	17.0
4 (DD)	1.6	25	8.2
5 (JC)	1.6	22	6.0
6 (CC)	1.7	40	14.0
7 (MG)	1.8	40	6.2
8 (JM)	1.6	27	7.2

Study design. Each subject underwent eight experiments (Table II) in random order and separated by a minimum of 1 wk. On each experimental day, the subject came to the laboratory after an 8-h fast. He underwent gastric intubation by swallowing a small caliber polyvinyl tube with a mercury weighted tip, and gastrointestinal lavage was performed using a poorly absorbable solution (11). This lavage is similar to that routinely used to cleanse the colon for colonoscopy.

After this lavage was complete, the subject removed his tube (time —zero). 4 h later, the subject voided urine (which was discarded) and a 10-h urine collection then began; immediately thereafter, he ingested the supplement and began eating the meal. 10 h after ingesting the Mg load, sufficient time for unabsorbed contents to reach the colon (12, 13), the subject voided to complete a 10-h urine collection, another small caliber tube was placed in the stomach, and a second lavage was undertaken for a 4-h period. The rectal effluent from this lavage was collected for subsequent analysis.

This experimental protocol was approved by the Institutional Review Board for Protection of Human Subjects of Baylor University Medical Center, Dallas.

Analysis. The meals were homogenized in a household blender, and a 50-g aliquot was added to a Kjeldahl flask for acid digestion (wet ashing) with nitric and perchloric acids. When digestion was complete, Mg and Ca were measured by atomic absorption spectroscopy and P was measured colorimetrically (14). The MgAc in gelatin capsules was dissolved in deionized water (acidified with nitric acid) and analyzed for Mg content by atomic absorption spectroscopy. After manually breaking two Slow-Mag® tablets into two pieces (to break the enteric coating) they were dissolved in deionized water (acidified with hydrochloric acid) and measured for Mg, Ca, and P in the manner mentioned above. The meal containing almonds was homogenized and measured for Mg, Ca, and P.

The rectal effluent collected after the second gastrointestinal lavage was homogenized in a commercial blender (model 34BL22; Waring Products, New Hartford, CT) and a 50-g aliquot was acid digested for determination of Mg, Ca, and P concentration (as above). Content was derived by multiplying concentration by the volume of rectal effluent collected in 4 h (usually about 4 liters). This sample was also measured for PEG content (15).

The 10-h urine collection was mixed by manual agitation of the container, and a 25-ml aliquot was acidified with hydrochloric acid and measured for Mg and Ca concentration by atomic absorption spectroscopy.

Calculation of absorption and urinary excretion. The amount of PEG recovered in the effluent from the second gastrointestinal lavage was expressed as a percentage of the 10 g ingested on each experimental day. This was done to confirm full recovery of unabsorbed gastrointestinal contents. PEG recovery was consistently near 100% (Table II).

Net Mg absorption from the meal and MgAc supplements was calculated with the previously derived formula (7):

Net Mg absorption from meal and supplement

= Mg ingested in meal and supplement

− [Mg in effluent after meal and supplement

− Mg in effluent after water].

For example, if the lavage effluent contained 8 mEq after ingestion of a meal and supplement containing 14 mEq, and 1 mEq after ingestion of only water, then net Mg absorption = [14 − (8 − 1)] = 7 mEq. Similar measurements for net Ca and P absorption were also carried out. Intake and absorption from the meal can be approximately converted to daily intake and absorption by multiplying meal intake and meal absorption by 3 (16). Fractional absorption was calculated as (net absorption/intake × 100).

Mg absorption from the three different 10-mEq Mg supplements (MgAc, Slow-Mag®, and almonds) was calculated with the following formula:

Mg absorption from supplement

= Measured amount of magnesium in supplement

− [Mg in effluent after meal and supplement

− Mg in effluent after meal and placebo].

This method of calculation removes absorption of meal Mg from the net absorption equation, allowing a more clear-cut comparison of Mg absorption from the three supplements.

10-h urinary Mg and Ca excretion was calculated by multiplying cation concentration by 10-h urine volume. Percent of absorbed Mg excreted in urine in 10 h was calculated with the following formula:

Percentage of absorbed Mg excreted in urine

$$= \frac{\text{(Urinary Mg excretion after meal and supplement} - \text{Urinary Mg excretion after fast)} \times 100.}{\text{Net Mg absorption from meal and supplement}}$$

Statistical analysis. A computerized analysis of variance combined with a two-tailed t test was used to test for significance; results were deemed significantly different when $P \leq 0.05$.

Results

Net magnesium absorption and urinary magnesium excretion. The data used to calculate net Mg absorption under various experimental conditions are given in Table II. Net Mg absorption increased with increasing Mg intake, but fractional Mg absorption fell progressively (from 65 to 11%) as Mg intake was increased from 3 to 80 mEq. Thus, Mg absorption was not a linear function of Mg intake.

Several curve-fitting methods were applied to the data from experiments 1–5, using individual results of net Mg absorption from each subject at all levels of intake (rather than mean results). Analysis by the double reciprocal (Lineweaver-Burk) method (17), as well as single reciprocal methods (Hanes and Eadie-Hofstee, and their reciprocals [18]) found Michaelis-Menten equations and curves inconsistent with the observed data. Because these reciprocal methods can be inaccurate and/or imprecise (18), computerized nonlinear least squares regression analysis (18–22) was used to find the Michaelis-Menten equation that best fit the data in terms of the lowest mean squared error (software was from SAS Institute, Inc., Cary, NC). The resultant equation (and curve), however, was also inconsistent with the data. Thus, statistically our results did not

*Table II. Net Magnesium Absorption and 10-h Urinary Magnesium Excretion**

Experiment	Meal	Supplement	Measured magnesium intake			Magnesium in Effluent	Net magnesium absorption	Fractional magnesium absorption	10-h urinary magnesium excretion	Percent of absorbed magnesium excreted in urine in 10 h	Percent of PEG intake recovered from 2nd gastrointestinal lavage
			Meal	Supplement	Total						
			mEq	mEq	mEq	mEq	mEq	%	mEq	%	%
1	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	Placebo	3.0±0.05	0	3.0±0.05	2.0±0.2	2.0±0.08	65.0±4.1	2.5±0.3	46.7±11.2	99.9±0.6
2	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	MgAc 1130 mg	3.1±0.04	10.2±0.08	13.3±0.08	10.4±0.3	4.0±0.3‡§	30.0±2.4‡	2.9±0.2	31.4±5.0	98.8±1.1
3	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	MgAc 2145 mg	3.0±0.02	19.4±0.2	22.5±0.2	18.7±0.5	4.8±0.5‖	21.1±2.2‖	3.0±0.4	28.9±4.1	100.0±0.4
4	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	MgAc 4289 mg	3.0±0.04	39.0±1.0	42.0±0.2	36.5±0.6	6.5±0.5‖	15.4±1.1‖	4.4±0.2	43.8±4.1	100.8±0.5
5	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	MgAc 8578 mg	3.1±0.02	77.2±0.6	80.2±0.6	72.0±0.9	9.1±1.1¶	11.4±1.3**	5.8±0.6	51.5±9.6	100.1±0.3
6	Standard meal: 80 g steak, 45 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	Slow-Mag® 2 tablets	3.0±0.04	10.5±0.07	13.5±0.06	12.0±0.3	2.6±0.3	19.6±2.2	2.0±0.2	21.7±13.7	100.8±0.3
7	Alternate meal: 80 g steak, 20 g Swiss cheese, 100 g rice, 250 ml water, 10 g PEG	Almonds 55 g (~30 whole)	2.4‡‡	12.2±0.2	14.6±0.2	11.7±0.3	4.0±0.3‡§	26.9±2.3	2.7±0.4	27.7±2.5	100.5±0.4
8	250 ml water, 10 g PEG	None	0	0	0	1.0±0.06	—	—	1.6±0.3	—	99.4±0.4

* Results from eight subjects expressed as mean±SE. ‡ Significantly different than Experiment 1 ($P \leq 0.0001$). § Significantly different than Experiments 1 and 2 ($P \leq 0.03$). ‖ Significantly greater than Experiments 1, 2, 3, and 4 ($P \leq 0.02$). ¶ Significantly greater than Experiment 6 ($P \leq 0.006$). ** Significantly different than Experiments 1, 2, and 3 ($P \leq 0.01$). ‡‡ Measurement of this meal without almonds before the study.

Table III. Net Calcium and Phosphorus Absorption and 10-h Urinary Calcium Excretion*

Experiment	Mg source	Calcium intake	Net calcium absorption	Fractional calcium absorption	10-h urinary calcium excretion	Phosphorus intake	Net phosphorus absorption	Fractional phosphorus absorption[‡]
		mEq	*mEq*	*%*	*mEq*	*mmol*	*mmol*	*%*
1	Standard meal + placebo	15.4±0.1	4.6±0.4	29.9±2.9	3.0±0.5	13.3±0.1	9.7±0.4	72.5±2.9
2	Standard meal + 10.2 mEq MgAc	15.5±0.1	4.0±0.4	25.9±2.9	3.1±0.6	13.4±0.1	7.5±0.4[§]	55.8±2.8[§]
3	Standard meal + 19.4 mEq MgAc	15.4±0.1	3.7±0.5	24.5±3.7	3.3±0.6	13.3±0.1	6.3±0.5[§]	47.1±3.4[§]
4	Standard meal + 39.0 mEq MgAc	15.4±0.1	3.9±0.6	25.4±3.8	3.9±0.5	13.2±0.1	4.8±0.7[‖]	37.0±5.4[‖]
5	Standard meal + 77.2 mEq MgAc	15.4±0.1	3.7±0.7	24.0±4.2	4.9±0.5	13.3±0.1	3.8±0.6[¶]	28.1±4.8[¶]
6	Standard meal + 10.5 mEq Mg in 2 Slow-Mag® tabs	26.7±0.1[‡]	7.2±1.0[‡]	27.0±3.4	3.1±0.7	13.2±0.1	9.5±0.6	71.9±4.9
7	Alternate meal + 12.2 mEq Mg in almonds	14.3±0.2	2.0±0.3**	14.5±2.4**	3.1±0.6	17.4±0.2	8.2±0.6	47.0±3.5[§]
8	Fast	0	—	—	1.5±0.3	0	—	—

* Results from eight subjects expressed as mean±SE. [‡] Higher Ca intake and absorption due to 11 mEq of Ca in two Slow-Mag® tablets. [§] Significantly lower than Experiment 1 ($P \leq 0.01$). [‖] Significantly lower than Experiments 1 and 2 ($P \leq 0.003$). [¶] Significantly lower than Experiments 1, 2, and 3 ($P \leq 0.004$). ** Significantly lower than Experiment 2 ($P \leq 0.0005$, see text).

conform well to a hyperbola derived from the Michaelis-Menten equation.

To explore the possibility that the net absorption results might conform to a hyperbolic function plus a linear function, nonlinear regression analysis was used to find an equation of format $y = e^{a + b/(x + 1)} + cx$ that best fit the data (statistically). The derived equation, $y = e^{1.286 - 2.889/(x + 1)} + 0.0710x$, (mean squared error = 2.993) depicts a curvilinear increase in net Mg absorption versus intake that almost exactly matches the mean results at all intakes tested (Fig. 1). Our results are therefore statistically compatible with two simultaneously functioning Mg absorptive processes: a mechanism that reaches an absorptive maximum beginning at an intake of 10–12 mEq, and a mechanism that endlessly absorbs 7.1% (0.0710) of ingested Mg.
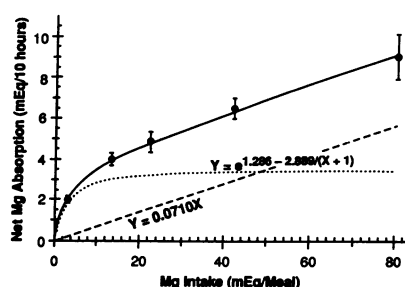
Fig. 2 compares net Mg absorption (Table II, experiments 1–5) with net Ca absorption from similar standard meals supplemented with varying amounts of Ca carbonate; the Ca data were obtained by the same method in normal subjects of similar age, and were published previously (7, 23). At very low levels of intake, more Mg than Ca is absorbed. At intakes greater than ~ 8 mEq, increasingly more Ca than Mg is absorbed.

Fig. 3 shows a comparison of individual and mean (±SE) Mg absorption from the three supplements MgAc, Slow-Mag®, and almonds. (The reader is referred to Methods to distinguish "Mg absorption from supplements" from "net Mg absorption.") Mg absorption from MgAc and almonds was similar, while absorption from Slow-Mag® was less by an average of 67%.

Figure 1. Net Mg absorption versus intake from experiments 1–5 (Table II) as derived by nonlinear regression analysis. The equation for net absorption, $y = e^{1.286 - 2.889/(x + 1)} + 0.0710x$ represents a hyperbolic function plus a linear function. These separate components and their respective equations are shown.
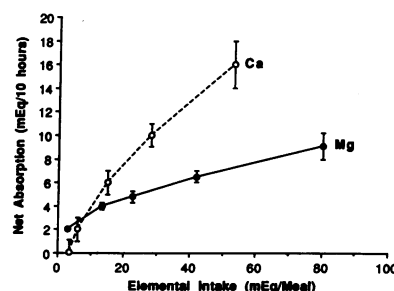

Figure 2. Comparison of net Mg absorption from experiments 1–5 (Table II) with net Ca absorption measured previously from similar meals with and without Ca carbonate. (Mean±SE values are shown).
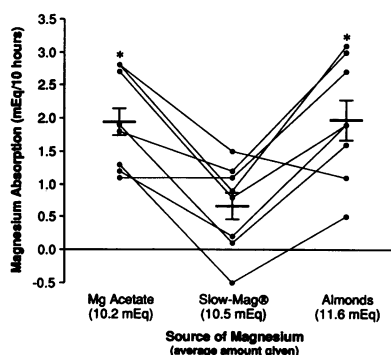
Figure 3. Comparison of Mg absorption from Mg acetate, Slow-Mag®, and almonds. The amount of ingested Mg was ~ 10 mEq with each supplement. *Significantly greater absorption compared with Slow-Mag® ($P \leq 0.003$).

Fig. 4 shows 10-h urinary Mg excretion as a function of net Mg absorption while the subjects fasted and after they ingested the standard meal without and with MgAc supplementation. Despite moderate individual variation, there was a significant linear correlation ($y = 0.43X + 1.4$, $r = 0.84$, $P < 0.001$) between these two parameters. The slope of this line (0.43) indicates that the average percentage of absorbed Mg excreted in urine in 10 h was 43% (see also Table II, "percent of absorbed Mg excreted in urine in 10 h"). This linear regression did not include values measured with Slow-Mag® and almonds because Ca and P intake on these days was different. When all eight experiments were considered together, similar results were obtained ($y = 0.45x + 1.3$, $r = 0.82$, $P < 0.001$).

Effect of Mg on net calcium absorption and urinary calcium. Net Ca absorption from the standard meal was unaffected by increasing amounts of MgAc (Table III). Net and fractional Ca absorption from the almond meal were significantly lower ($P \leq 0.0005$) than from the standard meal plus an equivalent amount of Mg as MgAc; this was true even though Ca intake on these two days was approximately the same.

10-h urinary Ca excretion after the standard meal increased with MgAc supplementation (Table III), and correlated significantly with Mg intake ($r = 0.41$, $P < 0.01$), net Mg absorption ($r = 0.41$, $P < 0.01$), and urinary Mg excretion ($r = 0.39$, $P < 0.05$). The rise in urinary Ca excretion occurred despite a constant Ca intake and unaltered net Ca absorption. After the highest supplemental dose of MgAc (experiment 5), 10-h urinary Ca excretion exceeded net Ca absorption (Table III).

Effect of Mg on net phosphorus absorption. As shown in Table III, increasing amounts of MgAc ingested with the standard meal caused progressive and significant decreases in net and fractional P absorption ($r = -0.73$, $P \leq 0.001$). By contrast, net and fractional P absorption from the standard meal was unaffected by supplementation with Slow-Mag®.

Fractional absorption of P from the almond meal was significantly lower than from the standard meal and placebo ($P \leq 0.0005$). In other words, ingested P was not as efficiently absorbed when Swiss cheese was partially replaced by almonds.
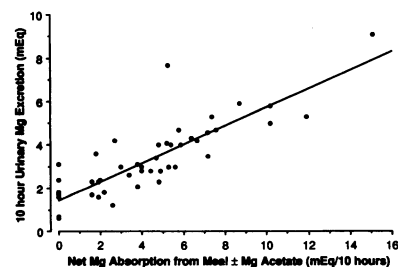

Figure 4. Correlation of 10-h urinary Mg excretion with net Mg absorption. The data are from experiments 1-5, and 8 (Table II). The equation of the linear regression is $y = 0.43x + 1.4$.

Discussion

In this study, net Mg absorption was measured in normal subjects after they ingested a standard meal supplemented with 0, 10, 20, 40, and 80 mEq of MgAc. This allowed a determination of the relationship of Mg intake and Mg absorption in normal subjects who had previously been on a free-choice Mg intake. If the intestine were to undergo "adaptation" in response to chronic ingestion of a low or a high Mg diet, the relationship we observed between intake and absorption would change in a quantitative sense, but would probably not change in a qualitative sense. As far as we are aware, there is no strong evidence that the intestine does in fact adapt to a chronic high or low Mg intake, as it does with calcium.

In our studies of normal subjects we found that net absorption rose with each increment in Mg intake, but that fractional absorption declined in such a way that net Mg absorption versus Mg intake was curvilinear (curval at low intakes and linear at high intakes). There are two main ways in which this relationship might be explained mechanistically. The first is that Mg absorption is entirely passive, with absorption mediated by electrochemical gradients between luminal fluid and plasma; the decrease in fractional absorption at high intakes could be explained if the percentage of ingested Mg remaining soluble in intestinal fluid fell as intake was increased. This explanation seems unlikely, however, because in other experiments we have shown that even very high Mg intakes remain nearly completely soluble in fecal fluid (24) and therefore presumably are soluble in intestinal fluid as well. The second possible explanation is that Mg absorption is mediated by both a saturable carrier and by passive diffusion, with the carrier primarily responsible for mediating absorption when Mg intake is normal or only slightly elevated and with passive absorption accounting for the continual rise in absorption when intake is elevated further. Evidence that a Mg transport carrier does exist in the human intestine comes from in vitro and in vivo studies of human ileum that showed saturation of net Mg absorption as Mg concentration in the perfusate was increased (25, 26). The existence of a recessively inherited disorder of Mg absorption (27-29) also supports the existence of carrier-mediated Mg absorption in normal subjects.

Regardless of the mechanism of Mg absorption and the explanation of the curvilinear relation between Mg absorption and Mg intake, the observed results are of interest from several perspectives. One of these is a comparison of Mg and Ca absorption. At very low levels of intake, there is net secretion rather than net absorption of Ca (23). By contrast, there is no suggestion that net secretion of Mg would occur if Mg intake was extremely low. On the other hand, at high levels of intake, Ca absorption is much higher than Mg absorption; this occurs in spite of the fact that ingested Ca tends to precipitate in the lower small intestine (30) (and thus becomes unabsorbable) and Mg tends to remain highly soluble in physiological intestinal fluids (24; Fine, K. D., and J. S. Fordtran, unpublished observations). It thus appears that the normal intestine is somewhat protective against net loss of Mg when Mg intake is low and against hyperabsorption of Mg when intake is high. A relatively restricted passive diffusion across the mucosa of Mg in comparison with Ca would explain such behavior. Assuming Mg and Ca are passively absorbed via a common pathway of fixed size, lower intestinal permeability to Mg might be due to

the fact that the effective ionic radius in aqueous solution is larger for Mg (8 Å) than for Ca (6 Å) (31). Alternatively, a Ca-specific membrane ionophore (i.e., a Ca channel) might allow passive permeation of Ca but not Mg.

The results are also of interest in regard to the use of oral Mg supplements to treat or prevent Mg deficiency. Although a number of Mg supplements and doses are in current use, the best agent and dose has been heretofore unknown because intestinal absorption of these agents has not been compared in humans. We chose to study MgAc in gelatin capsules so that Mg would be quickly presented to the intestine in a soluble (absorbable) state. The data in Fig. 1 suggest that a 10-mEq dose of MgAc would be optimal because fractional absorption falls off fairly steeply with higher doses, leaving a larger fraction of ingested Mg unabsorbed with the likely consequence of osmotic diarrhea. We also found that Mg absorption from enteric-coated Mg chloride (Slow-Mag®) on average was 67% less than from MgAc in gelatin capsules. Since it is not likely that chloride and acetate influenced Mg absorption directly, our results suggest that the enteric coating on Slow-Mag® tablets (cellulose acetate phthalate) reduced Mg bioavailability. This could be explained by the fact that cellulose acetate phthalate requires a 3–5-h exposure to gastrointestinal contents before pill contents are completely expelled (32); this delay would presumably reduce the small bowel absorptive area to which ingested Mg is exposed, and consequently reduce absorption (33).

One of the richest natural food sources of Mg is almonds, and we found Mg absorption from almonds to be equal to that from soluble MgAc. In contrast, Ca and P were much less bioavailable from almonds than from Swiss cheese (Table III), perhaps because one element interfered with the absorption of the other by formation of insoluble $Ca_3(PO_4)_2$. Alternatively, almonds may contain a substance that reduces Ca and P absorption by a separate but unknown mechanism.

As Mg intake increased, P absorption progressively decreased. This presumably occurred because Mg and P precipitated in the intestinal lumen as $Mg_3(PO_4)_2$. The observed reduction in dietary P absorption suggests that MgAc would be an effective P binder in the treatment of chronic renal failure. Compared to 50 mEq of Ca acetate, which in a previous study reduced the fractional absorption of dietary P from 77% to 26% (34), 50 mEq of MgAc (extrapolated from the measured reduction with 40 mEq and 80 mEq of MgAc) would reduce fractional P absorption from 77% to 34%. While there is slightly more P bound by Ca acetate per mEq of cation ingested, because much more Ca than Mg is absorbed at a 50 mEq intake (Fig. 2), there is more P bound per mEq of cation absorbed with MgAc, a desired characteristic of P binders used in this setting. Unfortunately, the risk of hypermagnesemia and diarrhea from MgAc ingestion would likely limit the clinical usefulness of MgAc as a P binder.

In contrast to the marked inhibition of P absorption, increasing Mg intake had no effect on Ca absorption. This suggests that Mg does not inhibit active or passive Ca absorption, even when Mg is present in great excess. However, there was an increase in urinary Ca excretion with increasing Mg intake and absorption. This effect has been noted previously in metabolic balance studies (35–37) and after intravenous injection of Mg salts (38, 39); possible mechanisms have been discussed elsewhere (40–42).

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