Calcium balance and calcium requirements in middle-aged women$^{1,2}$

Robert P. Heaney,$^3$ M.D., Robert R. Recker, M.D., and Paul D. Saville, M.D.

ABSTRACT Calcium balance performance was evaluated in 130 normal perimenopausal women ages 35 to 50, studied on their usual, self-selected dietary calcium intakes. Two distinct balance methods were used, one based on customary intake and output measurements, and the other based on absorption and clearance of two simultaneously administered calcium isotopes. Both methods yielded essentially identical results. Under study conditions as closely approximating normal living as possible, these women averaged $-24$ and $-31$ mg Ca/day by the two balance methods (exclusive of unmeasured dermal losses). More significantly, there was a positive correlation between intake and balance, with women on higher self-selected intakes exhibiting more positive balance than women selecting lower intakes. The calcium intake predicted from our data which would be required to produce an average balance of zero was 1.241 g/day. This is significantly in excess of the current recommended dietary allowance for calcium. The implications and significance of this discrepancy are explored in detail. *Am. J. Clin. Nutr.* 30: 1603-1611, 1977.

The currently recommended adult dietary allowance (RDA) for calcium is 0.8 g/day (1). The preceding (seventh) edition of Recommended Dietary Allowances (1968) (2) had presented a balanced argument for intakes both higher and lower than the 0.8 g figure, and indicated that possibly even more might be required. However, in the deliberations that led to the latest series of recommendations there was considerable pressure within the panel on foods and nutrition of the National Research Council to reduce this requirement to as low as 0.6 g/day, and the suggestion that 0.8 g might be too low was eliminated from the eighth edition.

It is true that no generally accepted epidemiological evidence exists which would support an adult requirement significantly greater than the National Research Council recommendations. Whereas poor absorption and subnormal intakes have often been reported in patients already osteoporotic (3, 4), and whereas calcium deficiency has conclusively been shown to produce osteoporosis in a variety of experimental animals (4), the occurrence of human osteoporosis has not been persuasively shown to be related to dietary calcium, either within ethnic groups, or across ethnic boundaries (5). Nevertheless this matter is of distinctly more than academic importance, since disorders such as osteoporosis take a profound economic, social, and human toll, and if dietary factors are in any way contributory, the benefit potentially available from their understanding and control would be significant.

The results to be described in this communication flow from early cross-sectional data obtained from a long-term study of the population-at-risk for osteoporosis. The underlying purpose of the study is to delineate factors that either contribute to osteoporosis risk or which predict development of osteoporosis. This paper describes the calcium balance behavior of early middle-aged women when studied on their customary calcium intakes.

Materials and methods

Research subjects

The subjects in this study were all white Roman Catholic nuns, related to five religious motherhouses

1 From Creighton University School of Medicine, Omaha, Nebraska.
2 Supported in part by United States Public Health Service Research Grant AM-07912, from The National Institute of Arthritis, Metabolism, and Digestive Diseases.
3 Address reprint requests to: Robert P. Heaney, M.D., Creighton University, Omaha, Nebraska 68178.
with which the Creighton University Bone Research Program had been able to establish a stable relationship. The only entry requirements were that the nun had to be between the ages of 35 and 45 at the time of onset of study, and be willing to return to the Bone Research Unit every 5 years for repeat studies. This group of women had been chosen because of its relative stability, accessibility, and dedication, and also because it had been established that they exhibited at least as high a risk of osteoporosis as did the general white female population. Subjects with intercurrent medical disorders (either previously known or discovered during the course of the research investigation) were not excluded, precisely because of our basic desire to study a complete cross-section of the population-at-risk without making any a priori assumptions about what might be relevant. Self-selection of a biased sample of the target age cohort was minimized by careful explanation both to the Mother Superior (obtaining her endorsement) and to each subject, of the nature and importance of the study. As a result we were able to achieve an overall volunteer rate of nearly 85% from the nuns in the 35 to 45 age cohort of all five motherhouses.

All subjects were active, employed individuals, with occupations ranging from teaching, to nursing, to administration, to institutional cooking. Our protocol prescribed osteoporosis-related interference with our subjects. Accordingly each subject was studied essentially "as we found her." We used her own dietary choices, and we continued during our study all prescribed medications, for whatever indication. The only exceptions to this rule of noninterference were situations in which our evaluation uncovered previously unrecognized intercurrent disorders which required medical attention. These included such problems as hypothyroidism, rheumatic cardiac disease, diabetes mellitus, breast and uterine tumors, and gluten-sensitizing enteropathy. In all such instances, after the initial study, we referred the subject to her own physician for definitive treatment of the disorder. Otherwise, dietary and/or hormonal manipulations were left entirely to the patient and to her own physician, and were essentially uninfluenced by the fact of participation in this project.

Study protocol

Each study consisted of an 8-day metabolic balance regimen divided into two 4-day periods, together with one half-day for special studies before starting and another after finishing the balance portion of the protocol. Subjects were active, out of bed, dressed, and in general participated in a variety of ward and hospital activities during the course of the study. In advance of admission, each nun was questioned by a trained dietitian concerning her usual dietary intake. This involved responses to a standardized survey, as well as a detailed week-long food diary. Although the nuns were active while on the Bone Research Unit, their level of energy expenditure was often less than in the setting from which they had come, and for this reason their caloric intake was adjusted downwards so as to avoid food rejection. Sometimes this maneuver involved calcium-containing foods, and when this occurred, calcium intakes departed from usual levels. However for the data described in this report, all cases have been excluded in which actual calcium intake during study varied by more than 25% from the historical intake.

A single daily menu was used throughout the study, and foods were prepared in advance and frozen in individual portions until use. Usual metabolic balance precautions were used, including the eating of meals together under supervision of dietary and nursing personnel, cooking of foods in the dishes in which they were consumed, the rinsing of all dishes and glassware by the subjects, with consumption of the rinsewater, and so forth. All excreta were collected, feces in two 4-day pools, and urines in variously timed collections, usually 24-hr volumes, but during the early phase of the isotope portion of the study, in 8-hr periods. Each meal during the 8-day metabolic study was labeled by the injection of a precisely weighed quantity of an inert marker. During the first 3 years of the project this marker was chromium sesquioxide, and in all subsequent studies, polyethylene glycol (PEG, Carbowax 4000). Fecal analyses, in addition to those directly relating to mineral balances, included measurement of this marker content. These results were used both to relate the fecal pools to corresponding intake intervals of equivalent duration, and to call our attention to the presence of factors which would invalidate balance calculations in a study as short as ours (see below).

On the second day of the metabolic balance study, each subject received with her breakfast a one-time-only calcium supplement consisting of 10 g of a calcium gluconate syrup, containing 169 mg of calcium, together with 10 µCi of 46Ca. Two hours later, 10 µCi of high specific activity 46Ca as CaCl2 in sterile isotonic saline were given intravenously. Frequent plasma samples were obtained throughout the remainder of the study and analyzed for stable calcium and for both radioactive calcium isotopes.

Tracer methods

The ratio of the oral tracer content to the intravenous, each measured as a fraction of its administered dose in urine and serum samples, was used to calculate fractional calcium absorption according to a modification of the method of DeGrazia et al. (6) as described elsewhere (7). The extra carrier calcium was added to the oral tracer dose to insure that the absorption measurement represented intestinal transport capacity under load conditions and was not simply an "index" of absorption, as is the case with many of the published calcium absorption methods which employ only negligible calcium carrier loads.

The fecal recovery of intake marker was used to time the conclusion of the second fecal pool, relative to the time of isotope administration. This was necessary in order to measure endogenous fecal calcium excretion rate, which is defined as the clearance of extra-cellular fluid calcium by the intestine, and is calculated as the quotient of fecal content of the parenteral tracer and the time integral (0 → t) of the plasma calcium specific activity for that same tracer (8). In long-duration studies the plasma integral is relatively insensitive to timing errors, but in studies of this length, timing remains important. The marker recovery was employed for this purpose by subtracting backwards from the external time of last defecation 24 hr (or fraction thereof) for
every day's worth of unrecovered diet marker in pools one and two combined. The resulting time was then used for computing the plasma integral which corresponded with the measured fecal excretion of the parenteral tracer. Balances were calculated in two largely independent manners. The first was the traditional, whole-body balance, i.e.,

\[ \text{Ca balance} = \text{dietary Ca intake} \]

- (total fecal Ca + urine Ca) (1)

The second method was based on the absorption measurement, and focused on the intake of absorbed calcium, as contrasted with ingested calcium, i.e.,

\[ \text{Ca balance} = \text{absorbed Ca} \]

- (endogenous fecal Ca + urine Ca) (2)

Absorbed calcium was measured as previously described (7). These methods have only two measurements in common: diet calcium and urine calcium, both of which can be made with good precision. The approach of equation 1 must contend with the problems of timing measured fecal calcium accurately; whereas the method symbolized in equation 2 avoids this problem entirely, using instead isotopic clearance methods and data.

**Validity criteria**

The 8-day study format was a necessary compromise, insofar as we could not obtain a sufficient volunteer rate for a longer study. Because an 8-day study is extremely short for purposes of measuring calcium balance, special methods were developed for testing balance values for validity. We imposed two conditions which had to be met for a traditional balance value to be accepted: 1) the first fecal pool content of inert marker had to exceed 25% of the first period's marker intake; and 2) the total of \(^{47}\text{Ca} \) excretion ( fecal plus urinary) and extra-intestinal \(^{47}\text{Ca} \) retention (calculated from retention of the parenteral tracer), had to equal the administered \(^{47}\text{Ca} \) ± 3%. If both conditions were met, then the second period fecal value, adjusted for its fecal marker content, was used for balance calculations. If not, no balances were derived from that subject. The rationale for these criteria was as follows. Balance calculation depends upon the ability to compare output with intake during an equivalent period of time. But because of intestinal transit time and intracolon mixing, externally timed fecal pools always follow their corresponding intake intervals by a period of time usually exceeding 1 day, and often amounting to several days. Thus, since the first period fecal collection inevitably contains prestudy intake residues ( when intake was not controlled or measured), it could never itself be used for balance calculations. But additionally, if the marker content of the first fecal pool was less than 25% of the first period marker intake, we then assumed that prestudy residues had not sufficiently cleared the colon by the start of the second fecal pool, and hence were contaminating that pool as well as the first. Fecal excretory lags of 3 to 4 days were not uncommon in our subjects.) The second condition related to the possibility of sufficient intra-intestinal mixing to carry prestudy residues into the second fecal pool, even if the first condition had been met. If the oral calcium tracer could not be accounted for either in the measured excreta, or by extra-intestinal retention, then it was presumed to be still within the colon. Since the oral calcium tracer had been given 7 days prior to completion of the study, we presumed that prestudy intake residues might also still be retained, and hence we accepted no balance data from such studies. While both validity conditions applied to traditional measured balances, only the second was required to validate computed balances.

When both the close match of study intake with historical intake and the internal validity criteria are applied to 8-day balance studies, we find that we reject about half of all our measured balances. This might be thought an unacceptably high rejection rate, but it should be borne in mind that these research subjects are the object of an extensive, multiphasic evaluation, that Ca balance is only one of more than 80 measured variables, and that loss of this one datum (because of uncertain accuracy) does not invalidate all the other measured variables.

**Analytical methods**

Diets, excreta, and serum samples were analyzed for stable and radioactive calcium by methods previously described in detail (7, 9, 10). In summary these were as follows: diets were homogenized in a commercial Waring Blender, made up to volume, and sampled volumetrically. Feces were mixed without added water by 2 to 4 hr shaking in the collection can on a commercial paint shaker, and were then immediately sampled gravimetrically. Both diet and feces aliquots were then dried and ashed at 450 to 500°C and the ash made up to a suitable volume with HCl. Urine was acidified, made up to convenient volumes, and sampled volumetrically. Stable calcium in all samples was determined by atomic absorption spectrophotometry. \(^{47}\text{Ca} \) was measured in a gamma scintillation detector set to exclude radiation from \(^{57}\text{Sc} \); and \(^{47}\text{Ca} \) was measured as the dry calcium oxalate powder in a thin end-window gas-flow detector system. All samples counted for their \(^{47}\text{Ca} \) content were allowed to age three months before counting to allow the beta activity of the \(^{47}\text{Ca}-^{57}\text{Sc} \) to decay. Furthermore, each sample for \(^{47}\text{Ca} \) was counted against two standards, one a known fraction of the \(^{47}\text{Ca} \) dose, and the other a fraction of the \(^{45}\text{Ca} \) dose. The latter was necessary because commercial \(^{47}\text{Ca} \) contains a small, but variable contaminant of \(^{45}\text{Ca} \). The \(^{47}\text{Ca} \) standard, at the time of \(^{47}\text{Ca}-\)counting, contained only the contaminating \(^{45}\text{Ca} \) component. Its presence in the \(^{47}\text{Ca} \) sample was then corrected for on the basis of the previously determined \(^{45}\text{Ca} \) content of the same sample.

**Results**

The intake, output, and balance data for the 130 women who are the subjects of this report are presented in summary form in Table 1. Diet calcium averaged 0.661 g/
day, somewhat lower than the RDA of 0.800 g/day, but above the sometimes recommended level of 0.600 g/day. Absorption from this intake averaged 32.5%, or 0.195 g, which was not quite sufficient to offset endogenous losses, thus leading to an average negative balance. By usual methods, this balance averaged -0.024 g/day, and by the computed balance method, -0.031 g/day. Neither balance estimate differed significantly from the other, but both were significantly different from zero ($P < 0.001$). In view of the fact that neither balance method measures dermal loss, estimated to be on the order of 0.015 to 0.020 g/day under sedentary conditions (11, 12), it may be stated that, on average, these perimenopausal women were in negative balance to an extent of 0.040 to 0.050 g/day. Of some interest is the smaller standard deviation of the computed balance ($P < 0.05$), a reflection of the greater independence of this measure from the problems of timing fecal collections.

Of more interest than the finding of negative balance, was the observation that balance was dependent upon intake. In other words, balances were more negative at low calcium intakes, and more positive at higher intakes. Table 2 presents the relevant linear regression statistics for intake, output, and balance. There was, of course, a strong positive correlation between intake and output. For measured balance, much of this was trivial, since most of the ingested calcium, being unabsorbed, reappears in the fecal calcium without effectively entering the body. This explains the very high value for the correlation coefficient ($r = 0.982$) for total output as a function of total intake. However there was nearly as impressive a correlation between absorbed intake and endogenous output ($r = 0.698; P < 0.001$). Here the X- and Y-variables are quite independent and share no common measurement or component.

In both cases the slope of the regression line of output on intake was less than unity. Figure 1A depicts this relationship for our subjects graphically. The point at which the computed regression line crosses the line of the identity function represents the intake at which predicted balance would average zero; i.e., at lower intakes predicted balances are all negative, and at higher intakes, all positive. The same data are replotted as Figure 1B to illustrate this point more clearly. In this case it is balance which is plotted as a function of intake. It is immediately evident from inspection that there were no positive balances at all at the low absorbed intakes.

Table 3 presents the predicted zero-balance intake values for the two balance meth-

**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>Diet Ca</th>
<th>Absorption fraction</th>
<th>Absorbed Ca</th>
<th>Urine Ca</th>
<th>Endogenous fecal Ca</th>
<th>Measured balance</th>
<th>Computed balance</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>130</td>
<td>130</td>
<td>130</td>
<td>130</td>
<td>130</td>
<td>106</td>
<td>130</td>
</tr>
<tr>
<td>Mean</td>
<td>0.661</td>
<td>0.325</td>
<td>0.195</td>
<td>0.118</td>
<td>0.109</td>
<td>-0.024^</td>
<td>-0.031^</td>
</tr>
<tr>
<td>SD</td>
<td>±0.328</td>
<td>±0.092</td>
<td>±0.067</td>
<td>±0.055</td>
<td>±0.034</td>
<td>±0.061</td>
<td>±0.051</td>
</tr>
<tr>
<td>SEM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.00593</td>
<td>0.00447</td>
</tr>
</tbody>
</table>

* N is less for measured balance than for computed because of the application of validity criteria (see text). Absorption fraction is dimensionless, and all other variables are expressed as grams Ca/day. ^ Denotes value different from zero ($P < 0.001$).

**TABLE 2**

Linear regression statistics for various measured intake and output variables. The first member of each variable-pair is treated as the Y-variable; the second member as the X-variable.

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Correlation coefficient</th>
<th>Slope</th>
<th>Intercept</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total output vs total intake</td>
<td>106</td>
<td>+0.982</td>
<td>0.951</td>
<td>+0.057</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Endogenous output vs absorbed intake</td>
<td>130</td>
<td>+0.968</td>
<td>0.643</td>
<td>+0.101</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Measured balance vs intake</td>
<td>106</td>
<td>+0.261</td>
<td>0.049</td>
<td>-0.057</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>Computed balance vs intake</td>
<td>130</td>
<td>+0.252</td>
<td>0.039</td>
<td>-0.057</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>Computed balance vs absorbed intake</td>
<td>130</td>
<td>+0.476</td>
<td>0.357</td>
<td>-0.101</td>
<td>$P &lt; 0.0001$</td>
</tr>
</tbody>
</table>
done by averaging the two balance estimates in each subject, and recomputing the regression. This new estimate of zero balance intake, also included in Table 3, is 1.241 g/day, with the 95% confidence interval from 1.166 to 1.316 g/day.

This range is greatly in excess of the 0.8 g RDA and is even more at variance than might first seem, because this value is the predicted intake at which average balance would be only zero, whereas the RDA is purportedly selected to be adequate for the vast majority of the population, i.e., to be in excess of need for most persons (2). That this estimate is not an excessive extrapolation from limited data is suggested visually, from the data in Figure 1B, in which many of even those women with intakes in excess of the current RDA exhibited negative balances.

Discussion

To our knowledge this is the largest group of calcium balances in women of this age reported to date. The finding that, on average, such women have negative balances of about 0.03 g calcium per day is itself not surprising, and is consistent with what is known from population studies concerning the rate of skeletal loss in this age (i.e., 5 to 10% per decade) (13). Nordin et al. (14) had earlier noted a correlation between intake and balance in a series of studies collected from the literature, performed on men and women over a broad age span. Whedon (4) has also called attention to such a correlation. Thus our finding the same general relationship in perimenopausal women is not in itself surprising, but the balance level at which the relationship occurs in these women is much more negative than had been expected. This finding raises several questions. First, relating to this set

TABLE 3
Calcium intake at which predicted balance equals zero limits

<table>
<thead>
<tr>
<th>Predicted intake (g/day)</th>
<th>95% confidence limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measured balance</td>
<td>1.154 ± 0.097</td>
</tr>
<tr>
<td>Computed balance</td>
<td>1.460 ± 0.116</td>
</tr>
<tr>
<td>Composite</td>
<td>1.241 ± 0.075</td>
</tr>
</tbody>
</table>
of data: Is the correlation somehow artifactual? Does the correlation imply a cause-and-effect relationship between intake and balance? Would balances improve in most of these women if their calcium intakes had been raised? And second, relating to more philosophical matters: What are the implications of this finding for normal calcium intake requirements? Is it conceivable that human evolution and the subsequent ascent of man could have occurred in the face of frank, widespread dietary calcium deficiency? We will deal with these matters one at a time.

Balances are always difficult to perform, for a variety of reasons, and one must always must worry about artifacts, not least of which is the metabolic response to the peculiar constraints of being studied. We have previously alluded to our own a priori validity criteria (close match of study intake with usual intake, together with objective evidence of "good" fecal stream characteristics). These have as their purpose both the detection of unreliable results and the elimination of results that might reflect perturbations produced by altered intake levels. Our confidence in our results is based on several considerations: 1) on the use of these stringent criteria; 2) on the fact that these patients were well and hence were without the anxiety which may accompany research observations in other groups of patients; 3) on the fact that the subjects were ambulatory and reasonably active during study; and 4) most importantly, on the fact that both balance methods produced essentially the same conclusion. The latter point dispels the problem of measurement-related artifacts, since, from the standpoint of major analytical or systematic pitfalls, the two methods are nearly completely independent. Hence we conclude that both the average balance performance of these women and the correlation of balance with intake were real.

No correlation can establish causality. But in this situation we know of no plausible alternative hypothesis other than that a higher intake tended to produce a more positive balance and a lower intake a more negative balance. This conclusion is consistent with the results of many other studies in both animals and man (2, 15, 16), and is, in fact, a specification for calcium of a general nutritional principle applicable to nutrients which are stored within the body. These include such substances as protein, calories (in the form of fat), potassium, magnesium, and many others. Even more to the point, this positive correlation between intake and balance is, in all cases except for calories, a specific characteristic of their feeding at suboptimal intakes.

This point leads naturally to the next question: would the balance in these individuals have been more positive if they had been ingesting higher calcium intakes? This study provides no data bearing directly on this question, and our long-term study protocol precludes interference with the dietary or therapeutic regimens of these women; hence we could not directly test this question in them. But the fact remains that those of our women who normally consumed higher intakes had more positive (or less negative) balances than did those on lower intakes. It seems reasonable to conclude that at least those on lower self-selected intakes would have done better on higher intakes. It is less clear, however, that those on high intakes could be pushed into more positive balance by still higher levels. There is no existing evidence that skeletal mass is greater than normal in persons on very high calcium intakes, nor that a parallel phenomenon ever occurs in animals. (The curious syndrome observed in bulls fed a milking ration (17) may be an exception to this statement, but this disorder probably presents a more complex etiology than simple dietary calcium excess.)

As mentioned earlier, the correlation between nutrient balance and nutrient intake is a phenomenon that is observed principally at suboptimal intakes. We suggest that, rather than being a straight line, the theoretical regression line relating balance to intake would instead be a curve approaching zero balance asymptotically. The number of our subjects on high self-selected intakes is not sufficient for us to evaluate this hypothesis. However, considerable experience with high calcium intakes as treatment in patients with frank osteoporosis suggests that, after an initial period of calcium retention, the body adjusts to the new intake and then assumes a balance close to zero (15).

Thus, to synthesize, we propose that our
data can be interpreted to mean only one of two things: 1) that women at this age are in negative balance and that diet has nothing to do with this behavior; or 2) that current RDA’s are grossly inadequate for this age group. Because of the positive correlation, already discussed, we reject the first alternative, and are forced to confront the possibility that, for this group of women, the current RDA of 0.8 g is low by at least 0.4 g. We say “at least” because the RDA for any nutrient is usually set so as to be adequate for the vast majority of the population, and is thus in excess of actual minimum requirements for most individuals. Our estimate of 1.241 g represents the intake required to produce an average zero balance, and may, thus, be still inadequate for roughly half the group. While it is evident that balance performance in perimenopausal women may not be typical of other adult groups, it must also be said that, until now, except for pregnancy and lactation, no distinctions whatsoever have been made with respect to adult calcium RDA’s.

A requirement has meaning only in terms of a deficiency. Is there in fact a calcium deficiency state in the adult human? This is not a simple question. If such a deficiency state exists, it is undoubtedly osteoporosis, but it is not at all certain that clinical osteoporosis can be produced in man by calcium deficiency, at least within the range of calcium intakes found in adult diets. This uncertainty is complicated further by the fact that osteoporosis is now generally recognized to be a multi-factorial disorder, and even if calcium deficiency is proved to be one of those factors, it almost certainly is not the only one. i.e., not the limiting factor in many cases. While this fact complicates retrospective analysis of groups of osteoporotic patients; nevertheless it must be pointed out that Garn (5) failed to find any difference in bone mass, as measured by metacarpal cortical thickness, between persons habitually receiving very low and very high calcium intakes.

But even if such does come to be shown, is it reasonable to think that the dietary calcium requirement might be so much larger than we have up till now believed? Could emerging man, without modern dairy products, have ascended to present levels of civilization despite a continuing, universal dietary calcium deficiency? (He could not have done so, presumably, in the face of chronic protein or B-vitamin deficiency.)

These questions are not as difficult to deal with as they might at first seem. To begin with, if osteoporosis is the result of a calcium deficiency, it is one that has a long latent period, and which manifests itself principally in old age. It would thus not be expected to have a natural selective impact on human evolution in an era when life expectancy was short. Furthermore, occurring in postmenopausal women, neither would it have produced an effect upon mate selection.

Secondly, our assumption that primitive man, without access to modern dairy products, had a low calcium intake represents a kind of temporal provincialism. It has been argued that the loss of lactase from the digestive secretions of adult members of many races indicates that adult calcium requirements cannot be large. For if milk is indigestible, then it follows that a nutrient found principally in milk cannot be an essential dietary component. However this argument is germane only if one accepts the premise that milk is the principal source of calcium in the human diet. While it is true that dairy products are the principal dietary source of calcium for modern, civilized western man, this is self-evidently not the case for other cultural situations. After all, the dairy cow herself gets the calcium she gives us from exclusively non-dairy sources! Ubiquitous, fresh milk is a product of high technological civilization (and of refrigeration). For less technologically advanced peoples, milk is preserved primarily as cheese, in which lactose is pre-digested. Furthermore, non-dairy dietaries provide significant calcium in forms such as bone meal, roots, tubers, seeds, and other vegetable sources, especially the greens of root vegetables such as turnips, beets, and carrots, as well as many other vegetables themselves. Simple calculation, even from our civilized diet tables, reveals that a predominantly vegetarian intake adequate to meet protein requirements would have provided an abundance of calcium—significantly in excess of the current RDA. Furthermore, when such vegetarian sources are supple-
mented by nuts, seeds, and by animal sources such as locusts, termites, grubs and caterpillars of various sorts, all of which are staples even of twentieth century primitive man, the calcium content of the diet may rise to quite respectable levels (18). Add to this small birds, rodents, and fish, often eaten bones and all, and it becomes clear that calcium intake for most of the human race has not been predominantly determined by dairy products.

At a more civilized level, stews of birds, fish, game, and other meats, cooked with the bones, contain significant amounts of calcium even when the bones are removed before eating. A series of random turkey and chicken carcass stews analyzed in our laboratory contained from 40 to 124 mg calcium per 250 g serving (median: 82 mg). Even in modern civilized India, bone sections are commonly served with curries, and are nibbled upon after the curry has been eaten. (This practice is generally omitted when westerners are served, presumably out of deference to western tastes, but it remains a feature of Indian domestic dining.) Hence, we conclude that the suggestion that the human adult RDA for calcium might be as high as 1.24 g/day is not as outlandish as might first have seemed. Nor is this the first time evidence for a higher requirement has been presented. Whedon has independently estimated intake requirements at 1.09 g/day, for both males and females, by calculations from published normal values for absorption, excretion, and dermal losses (19).

What is known of the calcium requirement of other mammals? The same National Research Council responsible for the human RDA's has panels concerned with the nutritional requirements of laboratory and domestic animals. The published calcium requirements for a variety of these animals (20, 21), when expressed as a function of body size, fit an inverse power function, which when plotted on full-logarithmic coordinates can be conveniently represented by a straight line. Figure 2 graphically portrays these data for animals ranging in size from rodents to bulls, and shows how closely the RDA's of nonhuman species approximate this relationship. The only departure of any magnitude is for Homo sapiens. Curiously the human RDA has been set at a level only one-fifth of what would have been predicted from this curve. Lutwak (16) has previously called attention to this same inconsistency.

It must be admitted that the underlying reason, if any, for this tidy mathematical relationship between calcium requirement corrected for body size, and body size itself,

FIG. 2. Full logarithmic plot of recommended calcium intakes expressed as a function of body size. The number following each label is the actual recommended value for the species concerned. The value for monkeys is from Harris et al. (21); values for all the other animals are from the National Research Council (20).
is not at all apparent; and it must also be admitted that the evidence behind many of these animal RDA's is not very firm. Hence it would be inappropriate to conclude that the human RDA is too low on this basis alone. On the other hand, these nonhuman standards certainly provide no support whatsoever for the level at which the human RDA is presently set.

References


