Symposium: Dairy Product Components and Weight Regulation

Normalizing Calcium Intake: Projected Population Effects for Body Weight[1,2]

Robert P. Heaney

Creighton University, Omaha, NE

ABSTRACT Published data describing the inverse relationship between calcium intake and body weight in 564 women were evaluated for their dispersion around their means, and the fraction above any given weight or rate of weight gain was calculated from the parameters of the normal distribution for the variable concerned. At the 25th percentile of calcium intakes, 15% of young women were overweight, and that fraction fell to only 4% at calcium intakes in the range of currently recommended values. Similarly, obesity prevalence in this cohort fell from 1.4 to 0.2% across the same difference in calcium intakes. At midlife, women at the 25th percentile of intakes gained weight, on average, at a rate of 0.42 kg/y. This gain dropped to −0.011 kg/y at currently recommended calcium intakes. Although calcium intake explains only a small fraction of the variability in weight or weight gain, shifting the mean of the distributions downward by increasing calcium intake can be estimated to reduce the prevalence of overweight and obesity by perhaps as much as 60–80%. J. Nutr. 133: 268S–270S, 2003.

KEY WORDS: • *obesity* • *overweight* • *weight gain* • *calcium intake*

Recent reports have shown an inverse relationship between calcium intake and body fat mass (1–6). Although several of these studies have been observational in nature (and hence unable by themselves to establish definitively that changing calcium intake would change body weight), published reports describe at least three randomized controlled trials, all of which were positive. Hence, although much more needs to be learned, it now seems reasonably well established that high calcium intakes can reduce the risk of being obese and assist in making weight loss regimens more effective.

Obesity is recognized to be multifactorial in character, and calcium intake has been variously estimated to explain from 3% to perhaps as much as 10% of the total variation in adult weight, a relatively small portion of the total variability. Perhaps a more important question, however, is how much difference normalizing calcium intake would make in the prevalence of obesity or overweight in the population. This study presents a preliminary attempt, using published data, to answer this question.

SUBJECTS AND METHODS

The data behind the publication of Davies et al. (3) were reevaluated, focusing on the distribution of values around the regression lines relating calcium intake to body mass index (BMI) in young women and to weight gain at midlife. In both groups of women, calcium intake was obtained from 7-d diet records, and was expressed as the calcium-to-protein ratio (mg:g). This stratagem partially corrects for portion size estimation error; at the same time it adjusts for the countervailing effects of calcium and protein intakes on calcium balance in the range of calcium intakes commonly encountered (7).

The young women constituted two cohorts (total $n = 348$), one studied in 1984–1985 and the other in 1995–1997, both on entry into studies designed to test skeletal endpoints. For the studies in these women, BMI was taken as the dependent variable. The middle-aged women also constituted two cohorts (total $n = 216$), each followed prospectively without intervention, one for a mean of 8.5 y and the other for a mean of 21.7 y. In these women rate of change in weight (kg/y) was taken as the dependent variable. Both groups of women have been characterized more fully elsewhere (3).

For both data sets the distribution of values for BMI and weight gain were tested for normality and suitably transformed, as needed. The fraction of the population represented by these samples above any specified value was calculated from the integral of the normal distribution for the respective means and standard deviations. Error terms for these fractions were calculated from the confidence intervals of the slope of the relationship of the dependent variable on calcium intake, in each case at the specifically tested calcium intakes. The ap-

[1] Presented as part of the symposium "Dairy Product Components and Weight Regulation" given at the 2002 Experimental Biology meeting on April 21, 2002, New Orleans, LA. The symposium was sponsored by The American Society for Nutritional Sciences and supported by Dairy Management Inc. and General Mills, Inc. The proceedings are published as a supplement to *The Journal of Nutrition*. Guest editors for the symposium were Dorothy Teegarden, Department of Foods and Nutrition, Purdue University, West Lafayette, IN, and Michael B. Zemel, Departments of Nutrition and Medicine, The University of Tennessee, Knoxville, TN.

[2] Supported by National Institutes of Health (grants AR07912, AR39221, AR42155, AR32462), Health Future Foundation, National Dairy Council and Creighton University.

[3] To whom correspondence should be addressed.
E-mail: rheaney@creighton.edu.

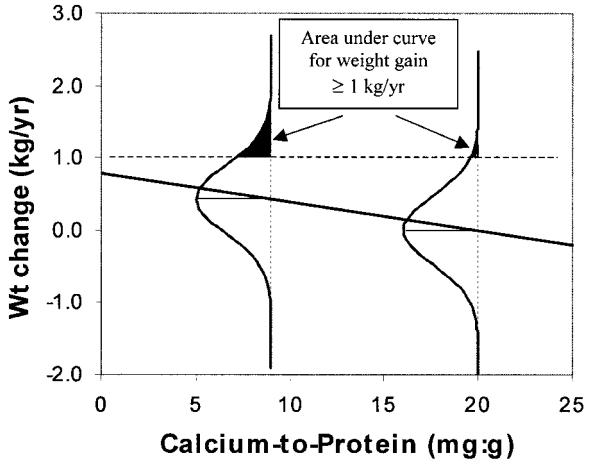

FIGURE 1 Plot of the regression line relating the dietary calcium:protein ratio to weight gain in midlife women, from the data of Davies et al. (3). Superimposed on this regression line are plots of the normal distribution with means at predicted weight gain for calcium:protein ratios of 9 and 20 mg Ca:g protein, and with standard deviations set to the standard error of the estimate for the regression. The areas under the two curves for weight gain ≥ 1 kg/y (the horizontal dashed line) are shaded. (Copyright Robert P. Heaney, 2002. Used with permission.)

proach is illustrated graphically in **Figure 1** for the data set from the middle-aged cohort.

RESULTS

Table 1 presents the area under the curve of the distribution of BMI values equal to or >26 kg/m^2 and equal to or >30 kg/m^2, respectively, both at calcium-to-protein ratios of 10 and 20 mg:g. (The former represents the approximate 25th percentile of calcium intakes for this group, and the latter is approximately the currently recommended calcium intake for third-decade women.) Because the distribution of BMI was not normal, the data were log transformed and the foregoing calculations were performed on the transformed data. As can be seen, predicted BMI at 10 mg Ca:g protein was 22.5 and the probability of having a BMI ≥ 26 was 0.146. This probability drops to 0.041 at 20 mg Ca:g protein, and the relative risk of being overweight at the approximate 25th percentile of calcium intakes, relative to the recommended intake, is thus 3.6. Similarly, the probabilities of being obese (BMI ≥ 30) are 0.014 and 0.002 for the two calcium intakes, for a relative risk of 6.9.

Table 2 presents corresponding data for weight gain at midlife, at calcium-to-protein intake ratios of 9 and 20 mg:g, respectively. (The former is the approximate 25th percentile and the latter, approximately the currently recommended intake for middle-aged women.) As can be seen, the predicted weight gain at 9 mg Ca:g protein was 0.425 kg/y, and about one sixth of the women gained at a rate of 1 kg/y or greater. By contrast, the mean predicted change at 20 mg Ca:g protein is actually slightly negative (−0.011 kg/y), and only 3.7% would be predicted to gain at a rate of 1 kg/y or more. The risk of such gain for the lower calcium intake level, relative to recommended intakes, is thus 4.2.

DISCUSSION

The data presented in this analysis suggest that the prevalence of obesity (or weight gain) in women could be reduced by 60–80% by the simple stratagem of ensuring population-wide calcium intakes at the currently recommended levels. However, it must be stressed that there is a great deal of uncertainty around such an estimate, principally because so few women were available in the two cohorts studied to provide weight or weight gain data at the recommended calcium intakes. For this reason any distributional analysis at recommended calcium intakes must involve a certain amount of extrapolation, always a risky enterprise.

Moreover, it must be stressed that, although this analysis adds new insights into the data assembled by Davies et al. (3), it does not add new information. The 564 individuals who contributed data for this analysis are the same subjects reported on by Davies et al. In this instance, however, the data from controlled trials are helpful, given that intakes in these trials are in the desired range, and the observed weight changes are at least directionally consistent with the estimates derived in this study.

Also reassuring in this regard is the analysis of the NHANES-III data earlier reported by Zemel et al. (1). After adjusting for age, sex, race and energy intake, they found a stepwise reduction in risk of obesity for each quartile of calcium intake. At the highest quartile (approximately equal to current recommendations for calcium), the risk of being in the highest BMI quartile was reduced by about 85%. Here the investigators had access to sufficient numbers of individuals at the recommended calcium intakes, and the observed reduction in their prevalence of obesity is quite similar to the estimate developed in this analysis. Consistent with this finding, also, is the recent report of the CARDIA study group (8) that dairy consumption was inversely associated with body weight in a prospective study of over 3000 young adults.

The absolute prevalence of obesity in our sample of young women was relatively low, only nine out of 348 individuals, or slightly <3%. These women were entered into their respective trials 18 and 7 y ago, respectively, and it is known that obesity prevalence in this age range has increased substantially since then (9,10). This means that the distribution of weight has shifted upward and that both the predicted mean values and the population fraction above any given BMI level will probably be higher today than the values presented in Table 1. Whether the slope of BMI on calcium intake will have changed since then cannot be determined from the data analyzed here. However, the response to calcium supplementa-

TABLE 1

BMI and calcium intake in young women

Ca-to-protein ratio (mg:g)	Predicted BMI	Fraction ≥ 26 kg/m^2	Confidence interval	Fraction ≥ 30 kg/m^2	Confidence interval
10	22.5	0.146	0.106–0.152	0.014	0.0087–0.0153
20	19.3	0.041	0.032–0.051	0.002	0.0014–0.0029

TABLE 2

Weight gain and calcium intake at midlife

Ca-to-protein ratio (mg:g)	Predicted weight change (kg/y)	Fraction gaining ≥ 1 kg/y	Confidence interval
9	+0.425	0.154	0.124–0.189
20	−0.011	0.037	0.027–0.049

tion in contemporary trials (2) indicates that the inverse relationship found in these women remains operative today.

The observation, both evident here and previously noted (4), that mean weight gain at midlife is effectively zero if calcium intake is at currently recommended levels is a fortuitous confirmation of the approximate adequacy of those recommendations. It is fortuitous in the sense that the currently recommended intakes were pegged to a skeletal endpoint, and there is no a priori reason to expect that all systems would exhibit the same requirement. It is also interesting to note that, despite the established bone protective benefit of an adequate calcium intake, the data presented here suggest that the effect on obesity prevalence–unrecognized until recently–is likely to be as large as, or larger than, the corresponding effect on osteoporosis prevalence.

Both the skeletal and the weight benefits are manifestations of the pleomorphic effects of dietary calcium, the bases for which are only now becoming clear and which were described in detail elsewhere (11). At the same time, they illustrate, tangentially, a point made by Geoffrey Rose nearly 20 y ago (12) that, when the bulk of the population is exposed to any given, but unrecognized, harmful influence, usual studies of apparent causation are able to identify, not the true etiology, but only predisposing factors (i.e., the reasons why some succumb to the disease and others do not). Low calcium intakes in this case are so widespread in the North American population today that virtually everyone is exposed to that influence. If, as seems increasingly likely, these low intakes are inadequate, then correcting calcium intake at a population level would produce benefits for many body systems. Furthermore, some of the factors currently considered to be causative of the diseases concerned will likely turn out to be only predisposing or triggering factors, operating by exaggerating or uncovering the effects of the real cause, inadequate calcium intake.

LITERATURE CITED

1. Zemel, M. B., Shi, H., Greer, B., DiRienzo, D. & Zemel, P. C. (2000) Regulation of adiposity by dietary calcium. FASEB J. 14: 1132–1138.

2. Zemel, M. B., Thompson, W., Zemel, P., Nocton, A. M., Milstead, A., Morris, K. & Campbell, P. (2002) Dietary calcium and dairy products accelerate weight and fat loss during energy restriction in obese adults. Am. J. Clin. Nutr. 75: 342S–343S (abs.).

3. Davies, K. M., Heaney, R. P., Recker, R. R., Lappe, J. M., Barger-Lux, M. J., Rafferty, K. & Hinders, S. (2000) Calcium intake and body weight. J. Clin. Endocrinol. Metab. 85: 4635–4638.

4. Heaney, R. P., Davies, K. M. & Barger-Lux, M. J. (2002) Calcium and weight: clinical studies. J. Am. Coll. Nutr. 21: 152S–155S.

5. Carruth, B. R. & Skinner, J. D. (2001) The role of dietary calcium and other nutrients in moderating body fat in preschool children. Int. J. Obes. 25: 559–566.

6. Lin, Y. C., Lyle, R. M., McCabe, L. D., McCabe, G. P., Weaver, C. M. & Teegarden, D. (2000) Dairy calcium is related to changes in body composition during a two-year exercise intervention in young women. J. Am. Coll. Nutr. 19: 754–760.

7. Heaney, R. P. & Recker, R. R. (1982) Effects of nitrogen, phosphorus, and caffeine on calcium balance in women. J. Lab. Clin. Med. 99: 46–55.

8. Pereira, M. A., Jacobs, D. R., Jr., Van Horn, L., Slattery, M. S., Kartashov, A. I. & Ludwig, D. S. (2002) Dairy consumption, obesity, and the insulin resistance syndrome in young adults. J. Am. Med. Assoc. 287: 2081–2089.

9. Deckelbaum, R. J. & Williams, C. L. (2001) Childhood obesity: the health issue. Obes. Res. 9(suppl. 4): 239S–243S.

10. Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S. & Koplan, J. P. (2001) The continuing epidemics of obesity and diabetes in the United States. J. Am. Med. Assoc. 286: 1195–1200.

11. Heaney, R. P. (2002) Ethnicity, bone status, and the calcium requirement. Nutr. Res. 22: 153–178.

12. Rose, G. (1985) Sick individuals and sick populations. Int. J. Epidemiol. 14: 32–38.