the percentage retention of Ca^{45} about twofold, while having no significant effect on Sr^{90} retention.

The last column of Table 1 shows the total calcium derived from the diet, which is deposited and retained during the course of the experiment. These figures are calculated from the measured Ca^{45} concentration in the bone and the known ratio of Ca^{45} to stable calcium in the diet. While rather close physiological control of dietary calcium utilization is evidenced at the two lower dietary levels, this control is no longer very effective at the 2-percent calcium level.

Blood levels of Sr^{90} and Ca^{45} at different times of sacrifice were reasonably constant within a given dietary group. Between dietary groups, the blood levels of Sr^{90} and Ca^{45} varied in essentially the same manner as their concentrations in the femur. In general, for a given group of animals, the ratio of Sr^{90}/Ca^{45} in the blood was not significantly different from the ratio of Sr^{90}/Ca^{45} in the bone; this suggests, as others have pointed out (*2*), that the major discrimination between dietary calcium and strontium does not occur in the specific processes of deposition on the bone. Because total calcium analyses on blood were not obtained, and in view of the limited time period studied, closer intercomparison of the blood and bone data with a view toward elucidating discrimination mechanisms would not seem to be justified.

Wasserman *et al.* have recently described an experiment in which Sr^{90} and Ca^{45} were chronically administered with diets varying in total calcium content from 0.5 to 2.0 percent (*4*). The skeletal deposition of both Sr^{90} and Ca^{45} in their experiment was inversely proportional to the calcium content of the diet. A number of differences between the experiments of Wasserman *et al.* and those which we report may have contributed to the different results obtained. Perhaps the most significant of these differences is the constant Ca/P ratio which was maintained in all diets by Wasserman *et al.* The increase in phosphate levels may have contributed very significantly in decreasing the availability for absorption of both the Sr^{90} and Ca^{45}. Changes in Sr^{90}/Ca^{45} ratios with time were not observed by Wasserman *et al.* (*4*). Although they varied the period on the experimental diets from 15 to 45 days, Sr^{90} and Ca^{45} were present in the diets for only the final 7 days of each exposure period. Effects due to differing turnover rates of Sr^{90} and Ca^{45} in bone would not, therefore, have been observed.

Should it develop that the ratio of strontium-to-calcium retention in bone does indeed vary with dietary calcium level under conditions which would normally apply in the human being, this would have an important effect on the evaluation of the Sr^{90} fallout hazard. Results from the present experiment suggest that the variation in hazard due to uneven distribution of fallout may be more closely related to actual Sr^{90} concentrations than to the much wider range of Sr^{90}/Ca ratios (*5*).

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References and Notes

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2. C. L. Comar, R. S. Russell, R. H. Wasserman, *Science* 126, 485 (1957).
3. The basic diet consisted of: Sucrose, 62 percent; casein 20 percent; alphacell, 10 percent; butterfat, 4 percent; vitamin mixture, 2 percent; Ca-free salt mixture, 2 percent. The calcium content was adjusted by addition of calcium lactate.
4. R. H. Wasserman, C. L. Comar, D. Papadopoulou, *Science* 126, 1180 (1957).
5. This paper is based on work performed under contract No. W-31-109-Eng-52 for the U.S. Atomic Energy Commission. The technical assistance of Joan Hess is gratefully acknowledged. A more detailed account of this and related studies is in preparation.

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Acanthamoeba: Observations on Animal Pathogenicity

The occurrence of *Acanthamoeba* in tissue cultures of trypsinized monkey-kidney cells has been observed by Jahnes *et al.* (*1*). We have isolated, from a similar source, an amoeba tentatively classified as *Acanthamoeba* which we have found to be pathogenic for mice and monkeys.

The amoeba first was recognized in the lesions of animals dead from the inoculation of a tissue-culture fluid thought to contain an unknown virus, but which later was shown to contain only *Acanthamoeba.*

Following intracerebral and intraspinal inoculation into cortisonized monkeys, extensive choriomeningitis and destructive encephalomyelitis occurred, causing death of the monkeys in from 4 to 7 days. Intracerebral injections into mice produced destructive encephalitis, causing death in 3 to 4 days. Many amoebae were found in the lesions at the site of inoculation, but it was obvious that the amoebae had migrated through the meningeal spaces and also through the tissue spaces of the brain and spinal cord.

To date, a few experimental observations have been made by injection of the cultures of amoebae into mice by other routes. Intravenous injection of the amoebae results in perivascular granulomatous lesions about amoebae that evidently escaped from the pulmonary capillaries. Intranasal instillation in mice lightly anesthetized with ether results in a fatal infection in about 4 days. These mice exhibit ulceration of the nasal mucous membrane with direct invasion of the adjacent base of the skull and involvement of the frontal lobes of the brain. The presence of many amoebae in the lungs is associated with a severe pneumonic reaction, with extensive fibrinopurulent exudate containing polymorphs and monocytic cells. Hemorrhage is a common feature. Pulmonary veins are invaded, and there are numerous thrombi containing many amoebae. Sections of the kidney show that the amoebae are present in the glomerular capillaries.

These observations suggest a possible explanation for the presence of the amoebae in the monkey-kidney culture and indicate that further inquiry into the possible role of the *Acanthamoeba* in disease processes is desirable.

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