tween rubidium-strontium and potassium-argon ages gives retentivities similar to those shown in Table 4.

In view of the fact that fairly low retentivities sometimes occur even in the case of mica, measurement of the potassium-argon age of a mica does not give a completely trustworthy value of the age. A much safer criterion for the absolute age of a sample of mica is agreement between the potassium-argon and rubidium-strontium ages. Since the daughter products, argon and strontium, have such different chemical properties, any alteration of the mineral may be expected to cause these ages to diverge. When they agree, except perhaps for a 5- to 10-percent discrepancy caused by a small loss of argon, this age is almost certainly the true age of the mineral.

There are a great many other minerals, such as the amphiboles, which contain small quantities of potassium. With modern mass spectrometric isotope-dilution and high-vacuum technique, the radiogenic argon content of these minerals should be measurable. Some work has already been done on certain sedimentary minerals, in particular glauconite (*20, 23*). The retentivity of these minerals has not yet been evaluated, and it may well be that minerals will be found which are more retentive than the micas.

The causes of the low retentivity of most feldspars and some samples of mica have not been studied as yet; they offer an interesting field for research. A number of possibilities come to mind, such as diffusion, exsolution of albite in the case of the feldspars, and slight changes in crystal structure under changing conditions of temperature and pressure. It is possible that, eventually, the radiogenic argon content of minerals which have lost argon will provide geologically useful information. However, much careful work will have to be done before this will be possible.

Using existing techniques of potassium-argon age determinations combined with rubidium-strontium age measurements, it is possible at the present time to date the time of formation of mica in a great many igneous and metamorphic rocks. This is a very exciting immediate application, and it should be possible to discern the location in space and time of the major orogenic episodes in earth history. Important contributions of this sort may be expected in the next few years, and while the problems of Precambrian geology will probably continue to be overwhelming for some time to come, a significant new approach is being made to problems left unanswered by traditional geologic techniques (*24, 25*).

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# Magnitude of Biological Hazard from Strontium-90


H. B. Newcombe


There are three main hazards to peacetime populations from radiation and radioactive materials, if we exclude major catastrophes and individual accidents. These are (i) the bone tumors (and possibly leukemias as well) which can result from ingestion of strontium-90, (ii) the shortening of the life span which results from exposure of the whole body to penetrating radiation (attributable in part to malignant diseases such as leukemia, and in part to a seemingly nonspecific acceleration of the aging processes), and (iii) the hereditary changes induced by radiation in the reproductive tissues.

In the case of i and ii, there are numerical estimates of the hazard. Thus, the extent of the loss of life expectancy caused by a given exposure can be estimated from animal data, supported to a limited extent by observations on human beings (*1*). Similarly, the probable numbers of future individuals who will suffer from serious hereditary defects as the result of a given radiation exposure can be derived from observations on the natural incidence of these defects and from estimates of the radiation dose required to double the spontaneous mutation rate in man (*1*, *2*). In spite of their limitations, such estimates are necessary if the so-called "permissible" levels of radiation for human populations are to have a rational basis.


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## Osteosarcomas

Unfortunately, comparable estimates of the strontium-90 hazard in terms of numbers of seriously affected individuals are lacking, although the importance of

the strontium-90 hazard has received considerable emphasis (see 2, p. 80). The omission might in part be remedied by using estimates based on the natural incidence of osteosarcomas, but no figure for the natural incidence is given in either of the reports cited. However, the approximate incidence is known from a survey of the province of Saskatchewan (*3*), which revealed 47 cases in a population of approximately 900,000 over the 13-year period from 1932 to 1944. For convenience, this frequency can be expressed as 12,000 cases per 100 million people per 30 years.

It is unlikely that these osteosarcomas were all caused by the natural background radiation in bone (about 7 roentgens per 70-year life-span), and it is also unlikely that the number of radiation-induced osteosarcomas increases linearly with dose (the only published response curve being sigmoid in shape, *4*). However, these two assumptions might be used to arrive at an approximate *upper limit* for the damage from osteosarcomas from a given exposure to a large population (Fig. 1, curve *A*).

For example, if the first of the two assumptions is in error and a part of the osteosarcomas is not radiation-induced, or if a part is caused by clinical x-rays, the true yield per unit dose would be less than the estimated upper limit (Fig. 1, curve *B*). If, in addition, the second assumption is in error, and the response curve is really sigmoid (Fig. 1, curve *C*), or there is a "threshold," the yield per unit dose would be still less, at least at the lower doses.

It is a little more difficult to show that the two assumptions lead necessarily to an upper limit at the higher doses, but additional information can be used in this connection. Both reports (*1*, *2*) state

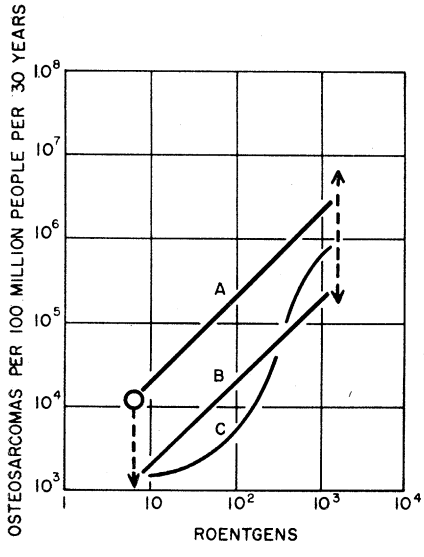


Fig. 1. Dose-effect relationships for radiation-induced osteosarcomas, based on three sets of assumptions.

Table 1. Upper limits for the hazard from strontium-90 induced osteosarcomas compared with conservative estimates of the genetic hazard.

| Item | Strontium-90 hazard (tumor induction in bone) | | | Gamma radiation hazard (genetic effects) | |
|---|---|---|---|---|---|
| | $Sr^{90}$ concn. (μμc/g of Ca) | Radiation in bone (rep per 70-yr life-span) (*8*) | Osteosarcomas (per 100 million people per 30 yr) (upper limit) | Radiation in the gonads (rep per 30 yr to average reproductive age) (*9*) | Serious hereditary defects (per 100 million people per 30 yr) (conservative estimate) |
| Nature | | 7 | (12,000) | 3 | (2,500,000) |
| Fallout from weapons tests (*10*) | | | | | |
| 1956 (milk, Canada) | 5 | 1 | 1,700 | 0.1 | 6,250 |
| Possible 1966 | 35 | 7 | 12,000 | 0.1 | 6,250 |
| Possible equilibrium | 70 | 14 | 24,000 | 0.1 | 6,250 |
| Arbitrary levels of exposure to populations above which concern is expressed | | | | | |
| U.K report (*11*) | 10 to 100 | 2 to 20 | 3,400 to 34,000 | 6 | 375,000 |
| U.S report (*12*) | 50 | 10 | 17,000 | 10 | 625,000 |

that 1500 roentgens is the lowest clinical exposure known to produce an osteosarcoma. There is no indication concerning the probable number of individuals receiving such an exposure, but a second very approximate point might be inserted on the dose-effect plot if we think of the incidence as perhaps somewhere in the range from 1 in 10 to 1 in 10,000. On our two assumptions, a dose of 1500 roentgens would be expected to produce about 1 osteosarcoma per 40 people, but the true incidence at this dose is almost certainly very much less. Further, since exposures lower than 1500 roentgens have given rise to no reported cases of bone cancer, it would seem that the response curve must drop rapidly with decreasing exposure (see *5*). Thus it is unlikely that the true response curve passes above the linear curve *A* (Fig. 1). A similar conclusion might also be drawn from the studies on radium poisoning.

Table 1 shows the probable *maximum* number of osteosarcomas, based on the afore-mentioned assumptions, for various strontium-90 levels in a hypothetical population of 100 million people over a 30-year period. The true number of osteosarcomas in each case may be anywhere in the range from zero to the number shown. For comparison, an estimate of the genetic damage which would occur at the same time has been included, based on a "doubling dose" for mutation of 40 roentgens per generation and a natural incidence of severe hereditary defects of 2.5 per 100 people. It is considered that this represents a conservative estimate for the genetic damage (*6*). The numbers are those for sustained increases in radiation background extending over many generations. About one-tenth of this damage would appear in the first generation following an increase in the radiation background, but, when long-term hazards are being considered, the number shown would seem to be the important one.

## Future Levels

Table 1 deals in part with possible future levels for strontium-90 and penetrating radiation, on the assumption that weapons testing in the future will continue as it has over the past 5-year period. The projected strontium-90 levels cannot be considered as "estimates," because too many of the variables are unknown. They are, however, values which have been discussed as "possible" and are thus important in the absence of anything more reliable. It should be noted that the combined natural incidence of all types of bone malignancy is in the vicinity of 2 to 3 times that of osteosarcomas alone; thus, if strontium-90 is thought to induce other types of bone tumor, the upper limits for the numbers of seriously affected individuals will be greater than those indicated in Table 1 by as much as two- or three-fold. Also, leukemias have been omitted so far from the present discussion because they appear to have been much less common as a cause of death, both in animal experi-

ments with radioactive strontium (*1*) and in cases of radium poisoning in man (*2*), possibly because only a part of the hemopoietic system is exposed to the radiation from bone-seeking materials. Others have estimated the effect of fallout on leukemia, on the assumption that strontium-90 is as effective as the equivalent penetrating radiation, at least with regard to the induction of those kinds of leukemia which are believed to originate in the bone marrow (*7*). However, the estimates others have obtained are very similar to the numbers indicated in Table 1 for osteosarcomas and do not greatly alter the present comparisons. It should be noted also that the estimation of genetic damage neglects all except the serious hereditary defects, although the less serious defects tend to be inherited over a larger number of generations, and thus affect more people before they are eliminated.

Remembering that Table 1 compares an approximate *upper limit* for the strontium-90 induced osteosarcomas with a conservatively chosen estimate for the serious hereditary defects, two conclusions might be drawn from the comparisons.

First, fallout in the future might perhaps result in more osteosarcomas than serious genetic defects, but the reverse could equally well be true, and there is no certainty that there will be any osteosarcomas at the strontium-90 levels considered in Table 1. Thus, in spite of assertions to the contrary (see *2*, p. 80, paragraphs 4a and 4b) there are, as yet, no objective grounds for deciding which will be the greater of the two hazards.

Second, the "permissible" levels for strontium-90 exposure in large populations would seem to have been chosen with greater caution than those for penetrating radiation to the reproductive tissues. The numerical discrepancy in the estimated damage in the two cases as shown in Table 1 is in the range from ten-fold to 100-fold, and the true discrepancy may be much greater still. If we wish to be equally cautious with respect to both kinds of hazard, it would seem (i) that future revisions of the "permissible" levels for populations must be based on an attempt to assess the two kinds of damage in comparable terms and (ii) that there must be a common guiding principle in deciding how large an effect is acceptable in each case.

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# H. E. Sigerist, Social Historian of Medicine

For more than 2000 years the history of medicine has been studied and interpreted in some form. During this period, the purposes that motivated those who concerned themselves with the past of medicine and the evaluations derived from the materials available to them have varied considerably. Throughout antiquity and indeed far into modern times, such activity was motivated by a doxographic interest—by a desire to learn and to present the opinions and methods of previous medical generations. The essential purpose behind these writings is perhaps closer to that of the modern writer of a medical paper, who cites his immediate predecessors in the particular field of interest, than it is to that of the historian.

Within the present century, a more sophisticated approach to medical historiography has become increasingly prominent and influential. The keynote of this approach is the proposition that medicine is an activity whose development can be most fully understood only when it is considered in relation to the network of social interaction within which it occurs. Taking the social character of medicine as a point of departure, its history becomes the history of human societies and their endeavors to cope with problems of health and disease. While a number of medical historians, both in this country and abroad, have studied the development of medicine in terms of social factors and institutional structures, the foremost proponent of a need for reinterpretation of medical history from this broader viewpoint was Henry Ernest Sigerist, commonly recognized as the leading medical historian of his generation. Consequently, it was an occasion of distress and sorrow for the many who had known him personally or