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A SUMMARY AND EVALUATION OF THE PROBLEM WITH REFERENCE TO HUMANS OF RADIOACTIVE FALLOUT FROM NUCLEAR DETONATIONS

Hardin B. Jones

January 14, 1957

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ABSTRACT

The tolerable amount of radiation exposure to humans is probably less than formerly estimated. It is shown, however, that accumulated effects of the low-level world-wide exposure to radiation from fallout to date is relatively small. The genetic effects are not large enough to be statistically detectable. The health effects, as expressed in life expectancy, are much smaller than those of such factors as infectious or chronic disease, metabolic disturbances, smoking, obesity, lack of exercise, and environment and marital status. Predictions of strontium-90 levels to be expected in the next two decades indicates, however, that bone irradiation may become detectably harmful.

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Introduction

Nuclear detonations form radioactive isotopes in quantities so enormous that they must be reckoned in terms of many thousands of curies, the equivalent of many pounds of the element radium. A portion of this radioactivity is dispersed into the atmosphere and subsequently falls out upon the land and sea. However, the vastness of the land, air, and water of the earth provides a means of dilution so great that even these large quantities of radioactive materials are soon reduced by distance and time to exceedingly small concentrations of radioactivity. The problem of radioactive fallout in relation to human beings involves the need to know the quantity that becomes a part of human environment, and to know the effect upon man of ionizing radiation from fallout.

This summary concerns low-level world-wide fallout. It must be recognized, however, that very intense fallout may be experienced in the vicinity of an atomic detonation. For example:[1]

"On March 1, 1954, an experimental thermonuclear device was exploded at the U. S. Atomic Energy Commission's Eniwetok Proving Grounds in the Marshall Islands. Following the detonation, unexpected changes in the wind structure deposited radioactive materials on inhabited atolls and on ships of Joint Task Force 7, which was conducting the tests. Radiation surveys of the areas revealed injurious radiation levels; therefore, evacuation was ordered, and was carried out as quickly as possible with the facilities available to the Task Force.

"Although the calculated accumulated doses to the exposed human beings were believed to be well below levels that would produce serious injury or any mortality [267 Marshallese received 14 r to 175 r]... All of the exposed individuals have recovered from the immediate effects [burns, loss of hair, anemia] without serious sequelae. Nevertheless it is planned to evaluate the

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*With suggestions and critical review gratefully acknowledged to R. Lowry Dobson, John W. Gofman, John H. Lawrence, Burton J. Moyer, William Siri, Curt Stern, and Edward Teller.

[1]Quoted from Charles Dunham, A Report on the Marshallese and Americans Accidentally Exposed to Radiation from Fallout and a Discussion of Radiation Injury in the Human Being (United States Atomic Energy Commission, July 1956).

medical and genetic status of the group at appropriate intervals with a view to learning what if any of the known late effects of radiation exposure may be observed. Obviously and indeed fortunately the number of persons [92 Marshallese] receiving 75 roentgens exposure and greater is too small to make it possible to determine with any degree of accuracy the effect on life span."

Effects of Radiation Exposure in Humans

The reasonable and superficial evaluation of radiation hazard is that humans can obviously tolerate exposure to several hundred roentgens, recover from immediate effects, and remain in "normal" health and functional capacity. Recently, however, we have become aware of deleterious long-term effects of radiation which, however subtle, appear to be proportional to the total quantity of radiation exposure and may be assumed to act even at very low levels of irradiation. Except for these long-term changes, our understanding of radiation effect usually has been concerned with two important facts that dominated our thinking about these problems:

(a) For certain kinds of radiation damage and injury, there is recovery. Individuals recover from acute symptoms produced by sublethal radiation exposures even though they may show general sickness, burns, loss of hair, anemia, etc. Recovery from acute radiation effects is analogous to recovery from any other acute injury or infectious process in which damaged tissue is healed and repaired.

(b) These obvious signs of radiation effect are associated with relatively large single doses of radiation (greater than 100 r). As dose size is decreased, detectable acute effects decline, becoming disproportionately small, so that there is a true threshold of dose of irradiation, at about 100 r, below which these particular acute manifestations of radiation do not occur.

The Proportional-Effect Concept of Irradiation

Recent evidence that long-range effects of radiation simulate aging effects comes from a variety of sources and is consistent with information relating radiation effects with genetic change and changes in cell-population numbers and quality. Evidence and logic support an argument that small increments of radiation-induced morbidity persist as small permanent changes in body functional structures, which become detectable as aging, neoplastic disease, and genetic change (see Appendix A). However, it remains to be proven experimentally that these effects do occur as the result of small irradiation exposures. The testing of this question is not likely because it would involve great technical difficulties. Attempts to procure some evaluation of the problem relating small-dose (5 r to 10 r) effect to life span would involve study under uniform conditions of perhaps several million mice. It is pointed out that in order to establish the effect of the smallest doses as yet measured for genetic effect, namely 25 and 50 r, the geneticist Curt Stern and his associates worked for 6 years and examined approximately 50,000,000 individual flies. They came to the acceptable conclusion that these small exposures have the same effect per roentgen upon gene mutation as at higher exposures

to radiation. Ionizing-radiation effect, in the depression of blood-forming function and blood-forming cells, is proportional to radiation dose even down to 5 r (Hennessy and Huff). Other effects upon blood cells, leading to abnormal doubling of the cell nucleus, are now reasonably established by Dobson in the range of 0.1 to 0.3 r of single exposure, but are not yet tested for proportionality.

There is no reason to doubt the general evidence of a proportional effect of radiation; but it is also possible that linear extrapolations of higher doses to the small-dose range may not give a true representation of the problem. It is known for some kinds of cellular response to radiation that there can be no effective change in function until two or more similar critical entities within the cell are affected. Some kinds of observed injury, however, appear to depend upon the effect on one critical entity per cell; and other observed injuries may be the result of damage to any one of a number of critical functional parts. The response that depends upon a chain of two or more detrimental changes shows a lesser apparent effect in proportion to radiation exposure at the low dose ranges. Although this kind of irradiation response does not argue against extrapolation of radiation effect, it may explain a factor-of-2 or -4 buffering against detectable radiation effect in the lowest exposure ranges; or it may even have the opposite effect, because radiation effect can add onto partially initiated dysfunctional changes in structures that otherwise would have remained functional.

Radiation effect is most frequently estimated in animals that are rather uniformly irradiated over the whole body. Thus, we are usually generalizing from the observed result of whole-body exposure. In some studies (Kaplan), shielding a relatively small portion of the bone marrow from radiation may protect the animal from generation of thymic tumors. In others, local irradiation is associated with induction of cancer in that region, quite independently of exposure or shielding of the remainder of the body.

The problem of estimating radiation effect and making recommendations concerning it is not the simple problem of avoiding exposure at levels at which there is a detectable or predictable response. This is especially true when considering radiation effect through systems that allow proportional extrapolation to very small radiation exposures. It is always important to keep radiation exposure to a minimum; but it is also important in the understanding and evaluation of the relative importance of radiation effect to establish its place in the entire climate of factors that can modify health. Similar--and, at times, greater--effects upon health can be shown to result from a large number of common environmental factors.

Also, the problem is not simply that of effect of body irradiation upon health. It is necessary to evaluate the effect upon human beings of all known phenomena resulting from the onset of the atomic age, including general socio-economic factors related to our well-being, which are dependent upon progress and the development of useful energy.

Advantages Minus Cost Equals Net Gain

The sum of evidence would lead to the conclusion that radiation probably does affect man's health subtly, and--like money and time--it should be exchanged for equivalent advantages.

Since the usefulness of atomic energy--including material and energy gain and defense measures of prime importance--is a positive result, and the radiation effect upon humans generally is a negative result of the atomic age, atomic energy usefulness minus harmful radiation effect must be equated to the net gain. Therefore it is critically important to estimate hazard quantitatively, and to be mindful of other factors while doing so. However, there is no unanimity of opinion at this time as to the precise balance that should be achieved between advantages and disadvantages of use of atomic energy, because certain qualifying factors are still too poorly known. Uncertainties exist which can mean either underestimation or overestimation of the effect of radiation. This brief synopsis roughly appraises the biological costs of exposure to radiation and presents information which must largely guide decisions in the interim until more precise information on radiation effect is available.

A summary of current knowledge of radiation tolerance or hazard and fallout is provided in several major public documents that have appeared in 1956 in broad survey of the problems to man of atomic radiation and fallout.[2, 3, 4, 5, 6]

The Concept of Maximum Permissible Dose

Early estimates of that amount of exposure to ionizing radiation which constitutes a permissible occupational hazard placed the upper limit at 0.1 r per day. Such a value was exceedingly conservative in view of information available at the time it was established. It is lower by a factor of 50 than chronic exposures leading to physiological disturbances and radiation sickness, and by a factor of 1000 to 5000 than the dose which, in a single exposure, might threaten life. Also, at the time it was proposed, 0.1 r was the lower limit of radiation exposure dose known to elicit any biological response. Evidence on the magnitude of physiologic response of the individual to radiation in the range

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[2] National Academy of Science, "The Biological Effects of Atomic Radiation - Summary Report," 1956.

[3] National Academy of Science "The Biological Effects of Atomic Radiation - Report to the Public," 1956.

[4] British Report, "Radiation Hazards to Man," Cmd 9780.

[5] Willard F. Libby, "Current Research Findings on Radioactive Fallout," Proc. Nat. Academy, Dec. 1956.

[6] M. Eisenbud and J. H. Harley, "Radioactive Fallout Through September 1955," Science 124, 3215 (August 10, 1956).

of a few hundred roentgens has not changed; but extensive information on effects of lower levels of radiation has recently appeared. This knowledge requires a re-evaluation of the cost to humans of radiation exposure in terms of (a) genetic effects, (b) shortening of life span, (c) induction of cancers, (d) destruction of tissue, (e) congenital malformation, and (f) effects upon young individuals. All these effects appear to be proportional to the exposure to radiation, and have been largely responsible for a recent downward revision in maximum allowable exposure to radiation.

The Genetic Evaluation of Radiation Effect

Up to about 1946, estimations of the genetic effects of radiation had placed the quantity necessary to double the mutation rate per generation in the fruit fly at about 50 r (Muller, Stern)--but with some uncertainty, so that the true value might have been 80 r or 35 r. At that time, there had been relatively little comprehensive evaluation of the range of genetic sensitivity to radiation in mammals or man. At the present time, the mutation rate per generation for the fruit fly is known to be doubled over the natural rate by about 50 r (Stern). Through genetic study of irradiated mice (Russell), the amount required to double mutation rate per generation in the mouse is partially established also at approximately 35 to 80 r. Wright has estimated from evidence now available that the mammalian mutation rate may be doubled by as little as 3 r or as much as 300 r. The best current estimates place the mammalian mutation-doubling dose of radiation at about 50 r.[4]

As an approximation, each species appears to form in natural circumstances about one new mutation in a generation time. The fruit fly lives a short time in about the same radiation environment (estimated roughly at 0.1 r/yr) as man. In its life span of 20 to 30 days, it can accumulate only the minute quantity of 0.008 r. Thus, if 50 r in the fly produces an additional number of mutations equal to those which occur naturally, radiation can account for only a part of the natural mutation frequency, namely, the fraction

$$\frac{0.008}{50} = \frac{1}{6,000} .$$

Hence, at background radiation, only one observed mutant in 6,000 is suspect of being induced by radiation. In humans, the life span up to average reproduction age is about 30 years, lived in the same environment of 0.1 r/yr, or a total of about 3 r by average reproduction age. Thus, if 50 r is estimated to double the human mutation rate, radiation from natural sources may be expected to account for

$$\frac{3}{50} = \frac{1}{17} ,$$

or approximately 6% of the naturally occurring mutations. If we accept the lowest possible value of 3 r for the mutation-doubling dose, we would have as the fraction attributable to radiation

$$\frac{3}{3} = 1 ,$$

and radiation could account for the entirety of mutation changes in humans.

The fallout of radioactive materials through 1956 has increased the radiation exposure of gonadal tissue by an amount estimated as approximately 0.004 r/yr (see Table V-D) (largely from ingested cesium-137[4] and deposits on the ground[6]). This is an increase of approximately 3% over natural radiation exposure.

The recommended limits of radiation exposure in man will be affected by information on the quantitative relationship between ionization and mutation and the understanding of the natural mutation burden. Should we estimate the level of radiation likely to double the natural mutation frequency in man as 25 r or 3 r, we will be at least 2 to 20 times as concerned about the genetic problems associated with radiation exposure as we are under the current assumption that the human mutation rate is doubled by 50 r.

Genetic studies of irradiated Japanese have been carried out by the Atomic Bomb Casualty Commission at Hiroshima and Nagasaki. A 10-year study has been analyzed by S. V. Neel and W. S. Schull.* The principal result is that no measurable increase in mutation rate was observed. They measured biological characteristics that could reflect genetic state and genetic change, such as stillbirths, male/female birth ratios, and congenital malformations. The results of all observations of this kind can be interpreted either as demonstrating no measurable increase in these events, which are associated with mutations, or as showing that, had the true congenital malformation rate been doubled, there would be only 90% probability of discovering even this increase. Thus, a small increase in congenital evidence of genetic change would not have been detected.

The results of the study of the Japanese indicate that the human genetic effect of radiation is acceptably consistent with the range of response estimated from mammalian genetic experiments; and it establishes with certainty that there are no catastrophic genetic effects at low to medium range of radiation exposure in human beings, although catastrophic effects are predicted at high levels of accumulated radiation exposures to whole populations. Many new mutations were probably produced in the Japanese exposed to the atomic bombs; but many of these may have been unobserved because of early lethality, and the rest are overwhelmingly diluted by the vast number of normal genes. This dilution was expected; and the statistical odds are known to be very greatly against the appearance of unfavorable and detectable combinations of mutant genes in any one generation of offspring.

Genetic change is, of course, basic to the concept of the Darwin principle of evolution. For this reason, it is possible that some increase in the mutation rate might be to human advantage in the long run by providing a greater pool of variance from which selection could take place, to our final

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*Reported at the First International Congress on Human Genetics, in Copenhagen in August 1956.

advantage some thousands of years from now. Some brief speculations regarding the extreme limits of variation may be offered:

(a) Humans and other long-lived animals have, as a corollary of their longevity, a less frequent natural mutation rate per gene per unit of time than short-lived species. As an approximate rule, each species appears to have about the same mutation rate per generation time. Thus, it appears that species are in some balance between generation time (or life span) and stability of genetic structure.

(b) Testing biological capacity for survival under circumstances that increase genetic variation is possible only with species having relatively short generation times. They have a common feature of a potentially great ratio of progeny per parent. These species can therefore survive even if a relatively high proportion of conceptions are incapable of survival and reproduction. Humans in natural selection are at some disadvantage in comparison with species producing a large number of offspring per generation, such as the mouse or the fly. Thus, human genetic tolerance should not be judged from effects of radiation exposures on these more fertile populations.

(c) For survival of a species, the ratio

$$\frac{\text{Reproducing offspring}}{\text{Individual}} \quad \text{must exceed 1.}$$

In humans, lowering of infectious disease toll has brought this ratio to approximately 2. As a consequence, the human population is now doubling in numbers approximately every 40 years. Thus, humans have already achieved some protective reserve against genetic changes toward lower fertility.

(d) On a scale of catastrophic genetic misfortune, humans also have the protection of vast numbers of individuals. There are 2.6 billion inhabitants of the world. While this number is small compared with numbers of insects and small mammals, it is still a very large number compared with that of any previous age in the history of man. Radiation exposure, as a cause of genetic change and increase of genetic variance, would be expected to produce that change in a random way. Thus, even with a large increase in genetic variance induced by radiation exposure, if population numbers are sufficiently great, some individuals will remain relatively unchanged. If these individuals were favored in selection, they might replace the less fit, less fertile fraction of the population. Thus, if survival of mankind were the only consideration, population numbers might, through reduction and segregation, achieve selective retention of adequately functional humans.

Several approaches to evaluation of extreme tolerance of human populations to radiation exposure with respect to health and genetic constitution are presented in Appendix B. These methods of estimation are difficult and speculative; but they indicate that an additional 2 r/yr (or 50 r per generation time) of chronic radiation exposure to the average individual in the human population would eventually cancel health and life-span gains we have achieved recently. Such estimations of impairment of health and estimations of the cost of increasing the genetic variance suggest that human population

cannot afford the biological cost of this intensity of chronic radiation exposure, and that there should be extreme caution at this time against increasing the radiation exposure to all people by ten times over its natural level.

Evolutionary Benefits?

It seems possible that human evolution is occurring in some optimal balance between mutation tendency and genetic stability. Fertility, length of life, death rate, and individual usefulness may be highly affected by the number of accumulated new genes,* which both add to favorable evolutionary drift in average human vigor and add to the pool of undesirable genes to be selected against. At low radiation levels, such as 10% or 1% above the natural radiation background (the range of fallout effect), it seems unlikely that long-range genetic disturbances can become an appreciable problem, since the natural radiation background appears to account for only 10% of the change in genetic structure per generation. One may speculate further that, in the long run, man may be beneficially affected by good genes yet to be formed, so that increasing radiation exposure and the mutation rate may operate to human advantage. Such an argument is unlikely to convince men who understand some of the dangers of too great a burden of undesirable mutants; it is analogous to an attempt to convince the experienced cook that the baking of her prize cake would be accomplished in half the time at higher oven temperature.

Penrose has evidence of indirect beneficial effect of some recessive lethal genes, which appear to enhance the effect of the functioning gene with which they are matched in individual combinations. This effect is one in which mutation may beneficially add some variance to genetic functional characteristics. On the whole, however, there is a strikingly large mass of information indicating that any genes that can disturb function should be kept to an absolute minimum.

Unfortunately, there are still many unanswered questions facing geneticists on the topic, "What is the effect of undesirable genetic burden on the quality of humans?" Fully satisfactory experimental measures have yet to be applied to this problem. One approach that has led to considerable speculation is through estimations of the numbers of undesirable mutations carried by the average person. Estimations of this burden place it within the small range of 5 to 15 undesirable genes per average individual.[4,†] This value is the equilibrium resulting from approximately one such gene gained

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*Transformed genes are, with rare exception, nonfunctional, lethal, or undesirable.

†Some individuals may have none. The fraction having none or very few diminishes steeply with increasing average numbers of undesirable mutations. Thus, doubling the burden of mutations may reduce the numbers of individuals having desirable genetic combinations to rare events.

and one lost in each generation.* Thus it has been pointed out that if, through increase in radiation exposure, the genetic gain of undesirable genes increased from one per generation to two per generation, there would be a relatively great reduction in the quality of the best 25% of individuals (assuming that reduction in quality of offspring is proportional to the number of undesirable mutations per individual). Because of speculative--but reasonable and cautious--arguments of this nature, geneticists have uniformly cautioned against allowing any major proportion of the population to accumulate radiation as high as 50 r, which is the amount estimated to double the human mutation rate.

Life-Shortening Effects

Life-shortening effects of radiation have been observed under a variety of experimental conditions. An experiment of particular significance because of the large numbers of animals and the range of exposure was the exposure of mice to nuclear detonation at "Operation Greenhouse" (Furth et al.). The fraction of life span lost per unit of radiation exposure appears to be essentially the same for a number of species, including the mouse, the rat, the guinea pig, the rabbit, and man. The largest number of experimental observations concerns the mouse. In the mouse, the fraction of the life span lost per unit of whole-body radiation exposure is acceptably constant over a wide range of variation in radiation exposure. The tentative conclusion is that radiation effect simulates aging itself, and that a unit of radiation exposure, regardless of the intensity and duration of exposure, produces approximately the same relative disturbance to body structure in adults of all mammalian species. On the human life-span scale, these effects of radiation summarized from small-animal data suggest that 1 roentgen of radiation exposure is equivalent to 5 to 15 days of physiologic aging. This prediction is confirmed directly in man (with reasonable technical reservations) by Doctor Shields Warren's recent investigation of life span of radiologists compared with physicians not using radiation in their practice of medicine. The average age at death is approximately 6 years less for radiologists than for physicians in general practice or for pathologists, both selected as being relatively unexposed to radiation. The estimation of accumulated radiation exposure in radiologists is uncertain, but has been approximated as 300 to 500 r. Thus,

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*The average mutation frequency of 1.5 spontaneous mutations of human genes per generation, as summarized by Penrose, corresponds to 30 mutations per million genes per generation, assuming that humans have about 50,000 genes:

$$\frac{30 \text{ mutations per generation} \times 50{,}000 \text{ genes per individual}}{1{,}000{,}000 \text{ genes}} = 1.5 \text{ mutations}$$

per individual per generation.

The average mutation rate may be less than this estimate, since one may suspect that the genes usually observed to mutate are perhaps ten times as mutable as the average gene.

the life-span loss, if attributed to radiation, is

$$\frac{-\ 6\ \text{yr} \times 365\ \text{days/yr}}{300\ \text{r to}\ 500\ \text{r}} = -\ 7\ \text{to}\ -\ 4\ \text{days per r of whole-body exposure .}$$

Such a number is still subject to considerable possible revision; but many different estimates give values of 1 to 30 days lost per roentgen of radiation exposure, and the probable value for humans is in the range of 5 to 10 days lost per roentgen.

A question exists whether we can justifiably extrapolate effects such as life-span lost per roentgen from measures that are mostly determined in the range of 100 to 1000 r. The evidence is that, over the range that can be tested, the effect is linearly proportional to the radiation exposure; and the information fits an extrapolation to zero shortening of life span at zero artificial radiation exposure. There is additional evidence in the effects of radiation upon cells (as distinguished from entire organisms), in which lethal damage to cells per roentgen also appears to be proportional to total radiation exposure. Such estimates agree for cells in the mouse, the rat, the rabbit, the guinea pig, and man. This experimental evidence that effect of radiation on cells is in linear proportion to radiation exposure of from 15 to several thousand roentgens provides a reasonable basis for understanding the life-shortening effects of radiation.

Furthermore, the life-shortening effects are consistent in order of magnitude with the genetic effects of radiation upon cells (2 to 3 cells affected per 1000 cells per roentgen). The genetic effect of radiation has been shown to be acceptably proportional to radiation exposure from 25 r to 8,000 r.

The sum of systematic evaluations of such effects of radiation as mutation induction, cell destruction, and life-span shortening indicates that these effects are permanent and represent the quantum interactions of radiation randomly affecting body cellular structure. The concept of quantum interactions with matter justifies extrapolation to the probability that a single quantum of radiation reacts with an individual molecule.

Although all recent evidence suggests that radiation effect is proportional to radiation exposure, such effects must be viewed together with other common environmental factors that modify health. A scheme is used here in which the effect upon health is expressed as an induction of aging (this is expressed as loss in physiologic lifetime, or minus time, written "-n years") or as a postponement of aging (expressed conversely as lifetime gained, or plus time, "+n years").* These factors all appear to have a general action upon disease

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*This estimation of life span lost or gained is in terms of relative physiologic age change. Change in life expectancy may be estimated by determining life expectancy at a given age in terms of a given age + n years' change in apparent age. Thus, a person of age 40 has a normal life expectancy of 31.1 years. If his physiologic age is 50 (because of a sum of factors predicting -10 years age over the average), his life expectancy (from life tables) is 22.8 years, or an average loss of life span of 8.3 years. Thus the life expectancy lost is somewhat less than physiologic time lost.

tendency, and the effect is about the same at any adult age. The list of relative displacements of physiologic age (Table I) is given for factors that accentuate aging or loss of life span (expressed as minus time) or retard aging (expressed as plus time). These measures are derived directly from human records. They are grouped according to whether they appear to be reversible or permanent. Most of the effects that are not partial measures of the same state are apparently additive, in the few instances that can be tested for this property.

Certain of these circumstances that modify health are partially interrelated, others may be independent of one another. Estimates of effect upon physiologic age may be additive, depending upon the extent to which they are independent. Thus country vs city dwelling may be suspected to include the factor estimated as exercise benefit. The lipoprotein test already contains information that can be estimated partially by relative overweightness, and the lipoprotein test already accounts for a portion of the smoking effect. Familial inheritance is independently estimated from each ancestor; male vs female differences are equally added to city vs country effects, and presumably each separate disease sign in the impairment study is additive.

In further support of the additive nature of effects upon health, each morbidity circumstance that can be quantitively estimated produces an effect proportional to the intensity of the circumstance. Examples of proportional change in mortality risk with morbidity severity are:

(a) Overweight -0.17 year for each percent overweight

(b) Smoking -0.45 year per cigarette used per day

(c) Radiation -5 to -10 days per r of whole body radiation
3 cells killed per 1000 cells per r (marrow and lymphatic tissue)
4 cells with chromosome breaks per 10,000 cells per r
1.4% increase in leukemia per r

(d) Atherosclerosis, Diabetes, Nephritis } end effects are proportional to severity of metabolic error

(e) Accidents are proportional to exposure risks

A somewhat similar tabulation can be made of an estimation of the cost of industrial and transporation progress in this century in terms of years of life span lost by accidental death, distributed to the average individual in the population of the United States (Table II). These values are approximately comparable to the preceding values based upon changes in physiologic age.

In about the same way, we can tabulate the effects on life span of radiation received. (Table III.)

Table I

Relative displacements of physiologic age by factors that accentuate aging or loss of life span (minus time) or retard aging (plus time).

Reversible	Years
Country vs city dwelling[1]	+ 5
Married status vs single, widowed, divorced[1]	+ 5
Overweight[2]	
25% overweight group	- 3.6
35% overweight group	- 4.3
45% overweight group	- 6.6
55% overweight group	-11.4
67% overweight group	-15.1
or, an average effect of 1% overweight	- 0.17
Occupational exercise vs sedentary occupation[3]	+ 5.0
Smoking[4]	
1 pack cigarettes per day	- 9
2 packs cigarettes per day	-18
Atherosclerosis[5]	
Fat metabolism	
In 25 percentile of population having "ideal" lipoprotein concentrations	+10
Having average lipoprotein concentrations	0

Permanent	Years
Female vs male sex[1]	+ 3
Familial constitutions:[7,*]	
2 grandparents lived to age 80	+ 2
4 grandparents lived to age 80	+ 4
Mother lived to age 90	+ 3
Father lived to age 90	+ 4.4
Both mother and father lived to age 90	+ 7.4
Mother lived to age 80	+ 1.5
Father lived to age 80	+ 2.2
Both mother and father lived to age 80	+ 3.7
Mother died at 60	- 0.7
Father died at 60	- 1.1
Both mother and father died at 60	- 1.8
Recession of childhood and infectious disease over past century in Western countries[8]	+15
Life Insurance Impairment Study:[7]	
Rheumatic heart disease, evidenced by	
Heart murmur	-11
Heart murmur + tonsillitis	-18
Heart murmur + strep infection	-13

In 25 percentile of population having elevated lipoproteins	- 7
In 5 percentile of population having highest elevation of lipoproteins	-15†
Diabetes[6]	
Uncontrolled before insulin 1900	-35
Controlled with insulin	
1920 Joslin Clinic record	-20
1940 Joslin Clinic record	-15
1950 Joslin Clinic record	-10
Antibiotics	+
Rapid pulse	- 3.5
Phlebitis	- 3.5
Varicose veins	- 0.2
Epilepsy	-20.0
Skull fracture	- 2.9
Tuberculosis	- 1.8
Nephrectomy	- 2.0
Trace of albumin in urine	- 5.0
Moderate albumin in urine	-13.5

*As measured in 1900 (Beeton and Pearson). These effects may be measurably less now, as environment is changing to produce greater differences between parents and progeny. Also, in 1900, it was a greater feat than now to live to be 80 or 90.

†This 70% difference in distribution of lipoproteins, between 25% lowest and 5% highest, is equivalent to a total of 25 years in relative displacement of physiologic age.

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Table II

Statistical distribution of lifetime shortening by travel and industrial accidents. (Calculation based on Vital Statistics of 1949, values for adult white males 20 years and older.)

All accidental deaths	-2.3 yr per individual in U.S.A.
Travel Accidents	
Accidents involving railways	-0.06 yr per individual in U.S.A.
Accidents involving ships	-0.04 yr per individual in U.S.A.
Motor-vehicle accidents involving driver and passengers	-0.67 yr per individual in U.S.A.
Assuming only half of population spends appreciable time in automobiles	-1.3 yr per individual at risk
Pedestrian motor-vehicle accidents	-0.2 yr per individual in U.S.A.
Assuming this effect largely involves the urban portion of the population	-0.4 yr per individual at risk
Aircraft accidents	-0.05 yr per individual in U.S.A.
Assuming that 1/4 of the population (actually, probably much less) uses airplanes	-0.2 yr per individual at risk
Accidents Involving Industrial Machinery	-0.04 yr per individual in U.S.A.
Assuming only 30% of males are employed using industrial machines	-0.27 yr per individual at risk

These values are based upon numbers of deaths attributed to accidents; the estimates of life span lost are actually perhaps slightly low because survivors who are maimed, and hence have reduced life expectancy, are not included in these estimates.

Table III

Estimation of radiation effect upon health and life span

Radiation received (r)	Lifetime Shortening (in yr.) If 1 r = -5 days[a]	If 1 r = -10 days[a]
50	-0.7	- 1.4
100	-1.4	- 2.7
200	-2.7	- 5.5
400	-5.5	-11.0

[a]Two columns are given because of uncertainty whether 1 r = -5 days or -10 days.

Thus it is observed that, although the estimated effect of radiation upon life span is a number worth attention, its magnitude of effect at low accumulated dosage is slight compared with many public health problems. It must be remembered that major problems such as smoking and overweight and fat metabolism are so subtle that they are estimated and established not by clinical methods but rather by statistical (actuarial) researches involving large population samples. The effect of smoking 1 pack of cigarettes per day, for example, appears equivalent in reduction of health and life span to the effect of between 200 and 400 r of accumulated whole-body radiation. This is several times as great as the 50-r limit currently recommended for occupational exposure; and 50 r, in turn, is on the order of 10 times as much as the individual would accumulate through fallout. If the life-span loss is estimated as 5 to 10 days per r of whole-body exposure, the loss due to 50 r falls within the range of -0.7 year to -1.4 years of life span. This effect is greatly exceeded by the magnitude of the smoking problem; the obesity problem; the problems of atherosclerosis, diabetes, and all the chronic diseases; the benefits of marital status; etc. The effect of 50 r of whole-body exposure to the general populace can also be viewed as being in the same category of life-span loss as that which results in the population of the United States from use of the automobile. This estimation, however, does not include the problem of the mutation burden in the next generations following such radiation exposure.

Summary of the Fallout Problem on a Global Basis

On a global basis, the fallout intensity of radioactive materials is no more than one millionth of the high-level fallout that occurred by mishap in the vicinity of a thermonuclear explosion in the Marshall Islands in October 1954. Current estimations made directly in humans throughout 1953-1956 place the fallout exposure from strontium-90 as being, on the average, sufficient to produce an irradiation effect of approximately 0.004 r/yr to human bones. This is a small quantity of radiation--2% of naturally occurring bone radiation--

and estimates of effects derived from this additional tissue burden will be correspondingly small compared with other human problems.

At the present time, according to the Libby report (October 1956), there is in the stratosphere about 2.2 megacuries of Sr^{90},* and a similar quantity of cesium-137.† If all the material in the stratosphere (in the fall of 1956) were to descend upon the surface of the earth uniformly, the amount of either Sr^{90} or Cs^{137} would be about 12 millicuries per square mile. The time of retention by the stratosphere of highly dispersed fission products is on the order of many years. Measurements indicate approximately 10% fall-out per year and 2.5% radioactive decay. As about 25% has been added to the stratospheric reservoir of dispersed fission products during the past two years, the level in the stratosphere has remained nearly constant over that time. The quantity of Sr^{90} in the soil of the United States is somewhat greater than expected from the fallout estimated on an average global basis; in the far west it is 23 mC of Sr^{90} per square mile. This is due to the heavier fallout in the near vicinity of a nuclear explosion.

Strontium-90 distribution to September 1955

	mC/square mile
World-wide except U.S.A. and Pacific Islands	3.4[6]
U.S.A. except Utah, Colorado, New Mexico, and bordering regions	4.9[6] 12.5[5]
Utah, Colorado, and New Mexico	20-23

The specific ratio of Sr^{90} to normal calcium is a convenient way of expressing the Sr^{90} problem.§ This is because strontium closely follows

*Strontium-90 has a half life of 25 years and decays by emission of a β particle of 0.54 Mev maximum energy to produce yttrium-90. Yttrium-90 is short-lived (half life 65 hr); it decays to the stable zirconium by emission of a β particle having a maximum energy of 2.24 Mev. Because of the short half life of the daughter product and the probable insoluble chemical form of yttrium, the radioactivity of Sr^{90} is equivalent to both its own beta decay and that of Y^{90}.

†Cesium-137 has a half life of 33 years and decays by β emission (0.52 Mev maximum energy) with associated γ emission (0.66 Mev energy).

§A convenient concept, established by relating irradiation of bone to bone cancer, is that a maximum permissible concentration (1 MPC) of strontium-90 is equal to 1 μC Sr^{90} per 1000 grams of calcium. The concentration of calcium in the bones is such that 1 MPC can also be expressed as 1 μC Sr^{90} per 7000 grams of bone. The concentrations of radioactive strontium are usually expressed in units of 0.001 MPC; the equivalence is 0.001 MPC = 1.4×10^{-7} μC Sr^{90} g of bone, corresponding to 0.0038 r/year.

calcium in chemical behavior. The levels of Sr^{90} directly measured in young human bones during the period up to October 1956 are in the vicinity of 0.0038 r/yr to the bone. Strontium-90 is deposited preferentially in the bone by a factor of more than 100 over the soft tissues, so that only the bones need be considered with regard to this isotope.

The Libby report estimates, on the basis of a balance between accumulated fallout of Sr^{90} into the soil and uptake by cattle and man, that in America the human ratio of Sr^{90} to calcium may eventually become 10% to 30% of that observed in the topsoil. The report estimates that Sr^{90} now held by the stratosphere, in descending to the earth over the next four years, will produce a human Sr^{90} concentration of from 0.016 to 0.038 r/yr (0.004 to 0.010 MPC*), assuming that no further Sr^{90} is added. The range of this expected gain of radiation exposure is equivalent to the extra cosmic radiation exposure experienced by individuals dwelling at altitudes of 5000 feet (e.g., Denver, Colorado) compared with individuals at sea level. The estimation assumes that there is a selection factor† favoring calcium over strontium in

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*MPC = maximum permissible concentration.

†Harrison et al. have evidence that elemental strontium-to-calcium ratios, compared in food, blood plasma, and bone, are strikingly different; for man they are:

	g Sr/g Ca	Proportional Units
Food	17×10^{-4}	7
Plasma	4×10^{-4}	2
Bone	2.5×10^{-4}	1

This is confirmed by Comar in observations using radiostrontium and radiocalcium simultaneously added to the diet. In Comar's observations for milk, the discrimination achieved against strontium in the deposition ratio of Sr/Ca may be less than that for other food sources, in which strontium and calcium may have different chemical binding.

The problem of a protective discrimination for humans against the uptake of the maximum Sr^{90}/Ca ratio is presented in the Libby report. At this stage of understanding, this apparent reduction of Sr/Ca in bones of humans compared with soil, plants, or animals seems to reside partly in the large calcium pool of the adult cow's body, which constantly dilutes incoming strontium and calcium so that milk, at present, is always intermediate in Sr/Ca ratio between the cow's bones and the forage. Similarly, the human calcium pool dilutes incoming Sr/Ca (largely from milk products) so that human bones at this time always have a lower Sr^{90}/Ca ratio than cow's milk or cow or calf bones. The content of children's bones is much higher than in adult or stillbirth material. There is some evidence for atomic discrimination between strontium and calcium, but the problem needs further study to determine how much of Sr^{90} uptake by bone is lessened at fallout equilibrium. If only dilution operates, with little or no discrimination, humans will develop a higher Sr^{90} level than is now expected.

uptake from soils into the plant and into the cow and into the human bones, so that 70% to 90% of the soil strontium is rejected in favor of calcium.

Both human adults and stillborn babies have similar concentrations of Sr^{90} (i.e., similar Sr^{90}/Ca ratios). This is to be expected, since the developing child draws its calcium from the maternal calcium pool, which is in partial equilibrium with maternal bone. Both these human sources of measured Sr^{90}/Ca have been placed during 1954 and 1955 at approximately 1/6 of the value for cow's milk; the resultant adult human bone irradiation value for this period is about 0.0019 r/yr (0.0005 MPC) from the Sr^{90} content. Reported values for adults did not exceed 0.004 r/yr in the sample studied, except for one individual measured at 0.008 r/yr. This is a very small number in terms of radiation effect.

If, in the fallout to be expected, the discrimination against Sr^{90} in its course from soil to plant to human bone is by only a factor of 50% instead of a factor of 70% to 90%, Libby's estimate of the future Sr^{90} concentration would have to be increased to 0.075 r/yr (0.020 MPC), based on the present stratospheric and soil burdens. This level of Sr^{90} would represent an additional radiation exposure to the bone, equivalent to the additional cosmic radiation experienced by those who dwell at 10,000 feet in this latitude.

Libby has estimated, from soil calcium levels, that if the entire Sr^{90} burden reached the soil and humans came into equilibrium with the top 2 inches of average soil, humans would eventually approach a maximum value of 40 μμC Sr^{90}/g Ca, or about 0.15 r/yr of bone irradiation. Such a value would approximately double bone irradiation over natural radiation.

Estimation from Human Bone Assays of Future Human Bone Concentrations of Strontium-90

The uptake of Sr^{90} has been directly measured in human bones as a function of age, and of location and time of collection (Libby,[5] Kulp et al.). The following summary conclusions can be drawn from analysis of this information:

1. Strontium-90 content of the bones in human stillbirths is increasing and, on the average, is estimated from Libby as follows.

U.S.A.	μμC Sr^{90}/g Ca	% increase per year
December 1953	0.14	
December 1954	0.30	114
December 1955	0.66	120
December 1956	(1.3) (extrapolated)	(100)

2. The bones of stillborn humans have a much lower Sr^{90} content than those of year-old children. The Sr^{90} content of children's bones, which may be averaged from the Libby report, is given in Table IV. This table is representative of the Sr^{90} concentration observed in children of early ages at two study intervals, namely 15 September 1954 and 1 August 1955, average collection dates. Newborns (stillbirths) have a much lower Sr^{90} concentration, because the uterine source of Sr^{90}/Ca has some intermediate value between dietary Sr^{90}/Ca and adult tissue-bone Sr^{90}/Ca. The value for stillbirths, as of January 1955, is 0.31 μμC Sr^{90}/g Ca; at this same time, growing children, age 0 to 5 years, are laying down Sr^{90} at 2 μμC Sr^{90}/g Ca. Thus, the fetal tissues appear to have available to them only 0.31/2 = 0.16 as much Sr^{90} as the growing child. This is a reasonable fraction, considering the lesser relative amount of milk products consumed by the average mother and the fact that her tissue stores of calcium are largely from the pre-fallout era. The growing child at each interval of growth (i.e., 0–1 year, 1–2 years, etc.) dilutes the entering Sr^{90}/Ca by the existing quantity of Sr^{90}/Ca already present in the body. However, analysis of the increment increase in Sr^{90} content shows that children of all ages are consuming and laying down equivalent concentrations of Sr^{90}/Ca, and that in January 1955 this concentration was approximately 2 μμC Sr^{90}/g Ca.

On this date, three sources of milk showed the following ratios:

Radiostrontium content of milk samples, January 1955

	(μμC Sr^{90}/g Ca)
Foreign cheese[5]	2.0
Chicago milk[5]	1.9
New York milk[7]	1.6

Since growing children have milk as their chief source of Sr^{90}, it is as expected that the value of milk closely approximates the concentration of Sr^{90}/Ca being deposited in growing bones. These values imply that, should milk remain as it was in January 1955, all children born close to this date will eventually have in their bones an average concentration of Sr^{90} of 2 μμC Sr^{90}/g Ca.

However, the milk Sr^{90}/Ca is increasing, and has been increasing since monitoring of milk was begun in 1953. Eisenbud's report[7] gives the following.

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[7] Merril Eisenbud, Global Distribution of Radioactivity from Nuclear Detonations with Special Reference to Strontium-90, Washington Academy of Sciences, Fall Symposium, November 15, 1956, Washington, D. C.

Table IV

Strontium-90 content of children's bones (from Libby Report)

Age (yr)	Weight: Av. at measurement (kg)	Weight: Δ/yr (kg)	Sr^{90} content: 8/1/54 to 11/1/54 ($\mu\mu C\ Sr^{90}/g\ Ca$)	Sr^{90} content: 6/1/55 to 10/1/55 ($\mu\mu C\ Sr^{90}/g\ Ca$)	Sr^{90} content in newly formed bone[a] (corresponding to Jan. 1955) ($\mu\mu C\ Sr^{90}/g\ Ca$)
birth	3.3	-	0.25	0.53	-
1	7.2	3.9	0.54	1.16	2.2
2	9.6	2.4	0.43	0.87	2.1
3	11.5	1.9	0.39	0.68	2.2
4	13.4	1.9	0.35	0.54	1.7
5	15.1	1.7	0.33	0.44	1.3
				Av.	1.9

[a] $$\frac{\Delta\ \mu\mu C\ Sr^{90}}{\Delta\ g\ Ca} = \left[\frac{Sr^{90}/Ca_{(t_2)} \times wt_{(t_2)} - Sr^{90}/Ca_{(t_1)} \times wt_{(t_1)}}{\Delta\ wt_{(t_2 - t_1)}}\right] \times \frac{12.0}{10.5}.$$

(The 12.0/10.5 is the correction factor for 10.5-month time interval 9/15/54 to 8/1/55.)

Sr^{90}/Ca content of milk in the New York area

Date	μμC Sr^{90}/g Ca
June 1954	1.2
January 1955	1.6
June 1955	2.0
January 1956	2.7
September 1956	5.0

should be corrected

The minimum estimate of future average human burden of Sr^{90}, then, is that 5 μμC Sr^{90}/g Ca will be present in the bone. This corresponds to the latest reported value for milk concentration and to the fact that bone acquisition of Sr^{90}/Ca in growing children is very similar to milk Sr^{90}/Ca.

A difficult current problem is the estimation of future Sr^{90}/Ca in milk. The level of Sr^{90}/Ca in milk is increasing, and, by linear extrapolation, may be expected to raise the Sr^{90} concentration in a year's time (by September 1957) to about 7 μμC Sr^{90}/g Ca. At this date, accumulated fallout of Sr^{90}, based upon the quantity estimated at the time of the Libby report, may be about 25%[6] to 50%[5] of the amount initially dispersed in the atmosphere. Since the Libby report was written, other nuclear detonations have occurred, so that it would be very reasonable to assume that fallout, by some 10 years from now, should have increased milk levels significantly. For lack of better information, we may assume a factor of, say, 3 to 5 times as much as September 1957 (allowing for residual hold-up in the atmosphere and for decay of Sr^{90}). Thus, milk levels and human bone levels by 1967 may be 20 to 35 μμC Sr^{90}/g Ca.

An additional factor must be considered, which may require that these future estimates be even higher. Cows, in body content of Sr^{90}/Ca, may be expected to lag several years behind the plant and soil levels. This is because of the large calcium reservoir in their bones and other tissues, and because the start of growth to milk-producing stage preceded current time by 4 or more years; moreover, the food consumed by dairy cows is customarily stored for many months before it is eaten. It is difficult to estimate that point in fallout time that corresponds to current milk values; it seems likely, however, that the Sr^{90}/Ca content of the bones of pasture-fed calves approximates the Sr^{90}/Ca level that adult cows would secrete in their milk, were they in more rapid equilibrium with fallout. The following table, derived from values averaged from Libby's and Eisenbud's reports, shows that calf bones are approximately 60% higher in Sr^{90}/Ca content than milk. Thus, future estimations of Sr^{90} levels should be at least 60% higher than the 20 to 35 μμC Sr^{90}/g Ca estimate, or, say, 30 to 50 μμC/g, in round numbers.

Strontium-90 contents of various materials (in μμC/g Ca)

	Mid-1953	Mid-1955	Mid-1956
Milk	1.1	2.1	3.6
Calf bones	1.4	3.5	5.7
Alfalfa (Wisconsin)	6.7	18	-
Soil (Wisconsin)			
2 to 6 in. depth	-	9	-
0 to 2 in. depth	-	35	-

It appears that the Sr^{90}/Ca of cow's milk is a close index of the concentration of strontium in newly acquired human bone. Current milk levels suggest that children's bones in the next decade will approach an average concentration of approximately 50 μμC Sr^{90}/g Ca. This is in close agreement with the estimation by Libby of a minimum average concentration of Sr^{90} of 10 to 40 μμC Sr^{90}/g Ca. These estimates do not consider local variance in the United States, nor, with respect to future concentrations, the special problem of high-rainfall or low-calcium areas.

The upper value of approximately 40 μμC Sr^{90}/g Ca has been set by Libby upon the consideration that this is the projected specific concentration ratio when all the fallout is complete and mixed with the average calcium content of 2 inches of topsoil. There does not seem to be a way of independently confirming the upper average limit of radiostrontium concentration from observation of milk or bone. The biological concentrations are increasing rapidly with respect to time, approximately following the level of total accumulated fallout, and 40 μμC Sr^{90}/g Ca may not truly be a limit.

Whatever the speculation concerning future levels of Sr^{90} in humans, we can be certain that current values (1956) represent a low level. If we translate a small dose such as 0.0038 r/yr (0.001 MPC) into numbers predicting an increase in leukemia mortality (an estimate may be based upon tentative data that leukemia tendency may be doubled by 50 r whole-body exposure*), an increment of

$$\frac{0.0038 \text{ r/yr} \times 50 \text{ years mean life span}}{50 \text{ r/tumor doubling}} = 0.004 ,$$

or 0.4% increase in leukemias, is estimated. Since there are only approximately 8,000 cases of leukemia deaths reported in the population of the United

*This number may be high, since it is based upon whole-body radiation exposure, while induction of leukemia by Sr^{90} exposure is the result of direct irradiation of bone and marrow, the specific tissues involved in the leukemia change.

States per year (plus 2,000 cases of bone-tumor deaths, which may be similarly affected by radiation), such a radiation burden is equivalent to an increase of 40 cases per year after fifty years' equilibration with this level of fallout. If radiation fallout and uptake of Sr^{90} in human bones were to increase by a factor of 10, one could estimate 400 additional cases of bone tumor and leukemia induced per year after a 50-year period, in comparison with 1,000,000 deaths from all causes and 10,000 expected deaths from leukemia and bone tumors. Both above numbers are small in comparison with over-all public health problems.

Although there are some sizable uncertainties regarding Sr^{90} burdens during the next 10 to 20 years, it seems from the average human values that Sr^{90} may increase and become a public health problem if levels should rise to 50 $\mu\mu$C Sr^{90}/g Ca (equal to about 0.2 r/yr to bone). There is time--but not much time--for a re-evaluation of many unsatisfactorily estimated aspects of this problem, including the extent to which radiation exposure induces leukemia and bone tumors, and more precise estimation of the strontium levels in humans. At the reference level of 1 MPC of Sr^{90} burden, which is 4 r/yr to bone, an estimated increase in bone tumors and leukemia is

$$\frac{4 \text{ r/yr} \times 50 \text{ years}}{50 \text{ r per doubling of incidence of tumors}},$$

or an approximately fourfold increase in natural expectancy of these neoplasms with respect to the radiation-related component of their origin. This level may be reached by humans as a result of Sr^{90} fallout. At some such value, reason argues against further exposure. The 1-MPC value based on radium exposure is consistent with a prediction of a fourfold increase in natural incidence of tumors. It would be difficult to observe a fourfold increase above natural incidence of bone tumors in animal-colony studies with radium, but not at all difficult in large human populations.

In summarizing their opinion for the British Report Cmd 9780, Mayneord and Mitchell write, "It appears however that each unit quantity of radiostrontium absorbed by bone confers a certain probability of bone-tumour formation, the tumour development time perhaps decreasing and the tumour incidence increasing with the dose. On the whole, the experiments seem in favour of a proportionality between the frequency of tumours produced in a given length of time and the amount of radioactive material in the body even at low dose levels."

The problem in the experimental animal is that the frequency of bone tumor appearance is so slight that statistically significant increases in the frequency are not to be expected as a result of irradiation. The human problem is similar in that osteogenic sarcoma and leukemia are relatively unlikely occurrences, together causing about 1% of adult deaths in the United States, so that a small percentage change in incidence caused by radiation could not be distinguished from random fluctuation, and a relatively large fractional increase in the number of these cancers would not appreciably increase the total death rate.

No gross evidence of osteogenic sarcomas has been observed following administration of P^{32} (approximately 100 rep to bones) to polycythemia vera patients. However, these patients do have a high incidence of leukemia. This leukemia tendency is probably attributable to both the radiation exposure and the nature of the basic disease of the blood-forming system in these patients.

Special phases of the Sr^{90} problem need additional examination:

(a) In several areas of the world, Sr^{90} concentration exceeds the average world values by more than a factor of 10.[4,5] This excess poses questions as to the origin of the enhanced concentration. To a reasonable extent, it is explained by Libby as calcium deficiency of soils in such areas. Rainfall variation also leads to variation in fallout. It will be useful to know more about these anomalous effects. Current world-wide sampling is perhaps far from representative of the world as a whole, because special effort was made to seek out low-calcium high-rainfall areas.

(b) There may be a factor-of-8 difference between Sr^{90}/Ca concentrations in soil and in humans, resulting from discrimination in favor of calcium (Libby); this must be further studied.

(c) Some factor of uncertainty must be allowed for in the prediction of levels today and in the early future of Sr^{90} in humans, considering that the most recent of these measures are based on early 1956. These uncertainties may amount to a factor of somewhat more than 10.

(d) Although it is unlikely that all these factors would reach their maximum, nevertheless, the total uncertainty in the estimated human burden of Sr^{90} throughout the world could mean an upper limit of 10 x 8 x 10 x Libby's lower estimate of exposure in the near future, 0.02 r/yr, which works out to about 15 r/yr or 4 MPC. This possibility indicates that the Sr^{90} fallout problem urgently calls for further attention.

Cesium-137 Fallout

The Cs^{137} problem is quantitatively similar to that of Sr^{90}. These two fission products are present in the air and in fallout in approximately equivalent quantities,[5] and they have similar decay rates. Whereas strontium is a bone-seeker, cesium is found in approximately equal quantities throughout the body, though less in bone than in soft tissues. Its distribution roughly approximates that of potassium. Furthermore, cesium is not retained by the body. Thus, the cesium burden at any given time rapidly reaches equilibrium with the rate of fallout, in the potassium pool in plants and animals.

Marley, in the British report,[4] writes (page 124), "The highest body-activity detected so far in the United States is found to be 4 x 10^{-3} μC. This activity if maintained would produce a total body irradiation of 0.0006 r per year or about 1/30 of the dose due to naturally occurring potassium-40 in the body." Since this time in early 1956, the fallout level and fallout rate of Sr^{90} have been increased only slightly, so that we may assume that the Cs^{137} level in man, which is more reflective of immediate fallout, may have risen by as

much as a factor of 2. It should remain nearly at this level, estimated as a maximum of 0.0012 r/yr, for an indefinite period.

Cesium-137 body burden at 0.001 r/yr is certainly not to be considered an adult hazard. With a linear relation between effect and dosage, 0.001 r/yr over a lifetime would be less than 0.1 r, and irreversible accumulative effects of radiation, such as leukemia, might be increased by less than

$$\frac{0.1\ \text{r}}{50\ \text{r leukemia-doubling dose}} = 0.002, \text{ or } 0.2\% .$$

Stated in terms of life span lost or of the total tendency toward disease, 0.1 r Cs^{137} dose x -10 days of life span per r amounts to 1 day lost from the life span. A loss of 1 day is very small compared with health-modifying factors that are measured in years instead of days. Thus, in comparison with the smoking problem, the long-term effect of Cs^{137} is approximately 1/40,000 as deleterious. Only this extraordinary method of estimation by extrapolation of effect can convince the human reason that there is any such effect at all; even the best statistical procedures could not detect it through study of the most accurate data on the 160,000,000 people in the United States. A 0.2% increase in leukemia (which is approximately 0.002 x 8,000 cases per year) is just 16 additional cases. This 8,000 expected normal incidence can fluctuate by random interplay of chance factors by plus or minus 1%, equal to 80 cases per year; thus, 16 cases of increased incidence cannot be detected.

The Level of Radiation Exposure from Fallout

The total increase in background radiation on a global basis, as a consequence of radioactive fallout, has been very slight. In the preatomic age, natural sources of radiation produced an average radiation exposure of 0.1 to 0.2 r/yr.* The variation is due to slight geographic differences, to differing radioactive content of earth and buildings, and to the variation of cosmic radiation with altitude. At 5000 feet above sea level, cosmic-ray intensity (measured by numbers of ionizations produced in matter) is increased to 1.5 times the sea-level intensity of cosmic rays; at 10,000 feet, the cosmic-ray ionization is 3 times that at sea level.

The increased human-tissue irradiation due to fallout and ingestion of radioisotopes is approximately as follows:

	Soft tissue irradiation (r/yr)	Bone irradiation (r/yr)
1955-1956		
Cs^{137}	0.0009	<0.0004
Sr^{90}	0	(0.002 (adult) (0.004 (young)
Predicted future values		
Cs^{137}	0.0012	<0.0006
Sr^{90}	0	0.04 to 1.5

Table V lists human radiation exposures from a number of sources.

* [illegible] 15 r/yr [illegible]

Table V

Human radiation exposure
(r/yr)

Natural radiation exposure		Exposure from fallout from nuclear detonations	
A. External Exposure to Whole Body			
1. Cosmic radiation		1. Fallout to earth,[6] Oct. '52 to Sept. '56	
at sea level	0.038	Salt Lake City	0.05
at 2,000 feet	0.043	All other U. S. cities and other countries	0.003
at 5,000 feet	0.056		
at 10,000 feet	0.112		
at 15,000 feet	0.214		
2. Radiation from the earth			
England	0.04		
Berkeley hills	0.12		
Sweden	0.09-0.16		
Average	0.10		

B. Internal Exposure

	To gonads and soft tissue	To bone		To gonads and soft tissue	To bone (1955)
Potassium-40	0.020	0.005	Strontium-90	a	0.002 (adult) 0.004 (young)
Carbon-14	0.001	0.001	Cesium-137	0.001	a
Hydrogen-3	a	a	Iodine-131	a	a
Radium	0.002	0.120			
Average	0.023	0.126	Average	0.001	0.002 (adult) 0.004 (young)

C. Total Exposure

Prefallout			Now		
At sea level	0.164	0.264		0.165	0.269
At 5,000 ft (Denver)	0.179	0.282		0.183	0.287

D. Internal Exposure Under Other Conditions

1955		Predicted for individuals born now (if no additional nuclear detonations)	
Strontium-90 (av. value, USA)	0.0019	(min. av. value predicted by Libby--0.004 MPC)	0.016
(highest value reported)	0.0075	(max. av. value predicted by Libby for USA--0.010 MPC)	0.038
Certain low-calcium areas			0.16 to 0.38
If humans at equilibrium should approach the Sr/Ca ratio of plants rather than 10% to 30% of plant Sr/Ca ratio			0.10 to 1.5

1956			
Iodine-131 (thyroid) (max. measured by Van Middlesworth)	0.004[b]	(probably)	<0.001 0.0002
(other than thyroid) (estimated)	<0.000001	(estimated)	<0.000001

[a] too small to be considered in this tabulation

[b] possibly the true value is 0.001 or less

The problem of radioactive fallout may also be examined in comparison with other ways of acquiring exposures to radiation (English values for radiological exposure are generally much less than in America[4]). See Table VI.

Thus, it is possible that, from common use of X-ray-generating devices, the average person in the United States has already begun to accumulate an exposure to radiation effect that is sizable compared with the fallout problem. That no gross evidence of disease has become evident during these past few years of increasing radiation exposure does not disprove the existence of slight average effects of radiation. For example, at current estimation of leukemia induction by radiation, about 20% of the relatively rare cases of leukemia (0.5% of adult fatalities) may be attributable to natural radiation. There is no difficulty in believing that supplementary radiation resulting from our modern activities may have been responsible for the other 80% of known cases of leukemia; the average additional artificial radiation exposure per year would only have had to be 0.8 r to account for this difference. Considering the generous use of unshielded and unfiltered X-ray equipment in dental offices and shoe stores alone, and the lack of public and professional appreciation of need to minimize radiation exposure, it is even reasonable to conjecture that the addition of artificially created radiation exposure to natural irradiation may essentially account for leukemia. Faber has analyzed 828 cases of leukemia registered in Denmark in the period 1950-53 with regard to the amount and type of irradiation each patient received for 20 years prior to development of leukemia. The incidence of previous incidental X-ray or radiation exposure for the chronic lymphatic leukemia cases was 18%, for myeloid leukemia, 30%, and for acute leukemia, 32%. It appears that both acute leukemia and myeloid leukemia can be induced by radiation; and the traceable X-radiation induction may account for a sizable percentage of current cases in Denmark. Faber's information does not rule out that lymphatic leukemia may also be induced by radiation. The analysis of leukemia incidence in follow-up of three groups of individuals who had had varying exposures to X-rays or other radiation strongly suggests that the radiation induction of leukemia is proportional to the radiation exposure, and that for whole-body radiation exposure the number would be entirely consistent with an estimation that 50 r doubles the chance of development of leukemia.*

Radioiodine Fallout

Of all the problems that we can currently evaluate, the radioiodine fallout problem is disposed of most readily. Radioiodine is produced in thousands of curies by some of the nuclear detonations; and, in falling to the earth's surface, it contaminates grass and is eaten by foraging animals. In its fallout, it is greatly diluted and does not at any time become a human problem. The herbivorous animal, however, eats large quantities of grass; and in the cow, for example, essentially all the iodine-131 ingested accumulates in the thyroid gland. Over a few days' time, several hundred pounds of grass may be eaten, and all the iodine contained becomes concentrated in the 15 to 30 grams of

*Court-Brown and Doll, Summary of Leukemia Induction, British Report,[4] pp. 84-89.

Table VI

Common means of exposure to radiation

Source	Exposure	
	Directed to the specific body region	Scattered to the whole body (dose per use)
Routine chest X-ray[a]	0.05 to 2.0 r/exposure	
Fluoroscopic examination[b]	10 to 20 r/min	1/200 to 1/1000 of local dose
Cinefluorography[b]	25 r per examination	
Dental X-rays[a]	10 to 150 r per whole-mouth series	0.01 to 1 r
Shoe store fluoroscopy[a, b] shoe-fitting unit	50 to 150 r/min to feet	1 to 10 r/min
Radium-dial watch[a] 1μC/watch	7.0 r/yr to the wrist	0.01 r/year
Radium and X-ray[a, b, d] technicians (throughout the world)		0.1 to 0.3 r/week 5 to 15 r/yr
AEC maximum permissible dose for 20 years' exposure		15 r/yr at 0.3 r/week
Average accumulated exposure of 10 most highly exposed individuals over 5-year period–U. C. Radiation Laboratory[c]		0.1 r/week 5 r/yr

[a] William Nolan

[b] AEC Report to Congress

[c] University of California Radiation Laboratory records

[d] Jones

thyroid tissue. Following nuclear detonations of the last two years, the thyroid concentrations of radioactive iodine in pastured cattle reached as high as 0.001 to 0.003 mμC/g (depending upon the quantity of fallout); and the average radiation exposure, as measured over 3 years, was about 1 r/yr to the thyroid tissue. This would be of genuine concern to man at similar human burdens of I^{131}, because it is now known that thyroid tissue is especially sensitive to radiation induction of tumors. However, cattle fed principally in feed-lots have only 1/100 (or less) as much I^{131} as range-fed cattle. Further careful measurement of fresh human thyroid material has been routinely made during the last two years by techniques that are sensitive and reliable for estimation of I^{131} content. Direct measurement shows that human thyroid, at any time of high uptake of I^{131} by bovine thyroid, has less than 1/5000 of the bovine I^{131} content. It is possible that human thyroids had less than 0.0006 mμC/g during the latter part of 1956, when range cattle had 1 to 2 mμC/g. It is certain that the human thyroid exposure during the 1956 period did not exceed 0.001 r/yr, and the probable value is 0.00016 r/yr or even less. (Interestingly, one human thyroid showed an activity comparable with bovine thyroid content of I^{131}; the case, when traced to its source, proved to be from a man who had previously been given a small tracer dose of I^{131} in the Donner Laboratory. The observed quantity of I^{131} was accounted for by the magnitude of the dose, the estimated excretion, and the radioactive decay.)

Up to this time, radioiodine from world-wide fallout is not a problem of concern to humans; and it is not expected that it will become a problem in the future.

Summary

1. This paper reports a broad examination of the levels of radiation exposure incurred from fallout. The discussion is limited to Sr^{90}, Cs^{137}, and I^{131}, the only radioactive isotopes reported to become associated with human environment in detectable quantities.

2. The world-wide effect of radiation from fallout is now far less than that of naturally occurring radiation from cosmic rays and from radioactive elements normally contained in earth, buildings, and body tissue. The inescapable minimum of natural radiation exposure, for all people, is about 0.1 r/yr. The average person at sea level in the United States is probably receiving about 0.16 r/yr.

3. During 1954-55 the Sr^{90} concentration in human bones (both in adults and in stillborn infants) produced an average exposure to the bones themselves of 0.002 r/yr. (Only the bones--not the soft tissues--are exposed to measurable levels of Sr^{90} irradiation.) At current fallout trends, the irradiation of human bone by Sr^{90} will increase to 0.016 r/yr, perhaps even to 0.038 r/yr (Libby). The maximum value projected in this discussion is 0.2 r/yr. (These are average predictions for the northern hemisphere and for the major population densities of the earth.)

4. Radioiodine (I^{131}) activity has been measured in humans during periods of likely fallout exposures. Radiation exposure from fallout I^{131} is essentially nil for humans.

5. Any analysis of the fallout of radioactive materials on a world-wide basis shows that it does not even remotely approach the threshold for acute radiation effects, which cannot be recognized below 100 r in a single exposure. Radiation predicted from future fallout is still far less than natural radiation background. Increases in the internal radiation exposure of 0.1 r/yr are not meaningful in comparison with acute radiation damage. Attempted comparisons are responsible for most misunderstanding of the fallout hazard to humans.

6. Life-span changes, cancer or leukemia induction, and cell changes appear to be proportional--as are genetic effects of radiation--to radiation exposure. Although these effects are not measurable in any individual exposed to fallout, they can be estimated, in terms of very small risks. The effects are dwarfed in comparison with the adverse environmental hygienic factors that most persons regard as commonplace. For example:

Factor	Life-span loss per person (days)
Smoking 1 pack cigarettes per day *	3,000
Being 25% overweight	1,300
Having 25% elevated lipoproteins	2,500
Living in United States as a driver of an automobile	470
Working in industry (industrial hazard)	100

7. The evidence indicates that Sr^{90} may eventually cause a world-wide increase in leukemia, accounting for about 2% of all deaths. Compared with the current accident rate, a 2% leukemia increase distributed throughout the entire population would be a life-span loss of about 0.02 year per person in the United States; all accidents account for a 2.3-year life-span loss per person, automobile use for 0.87 year. Thus the Sr^{90} induction of leukemia is comparable with some of the mechanical mishaps we risk as a partial cost of the "advantages" of our mechanized and energized age.

8. The sum of evidence is that radiation has a deleterious effect upon man's health, but that the effects are extremely small at such slight radiation exposures as are involved in the world-wide fallout. Nevertheless, since radiation probably does affect man's health and progeny--even though minutely for minute exposures--incurring it should be treated as the equivalent of the spending of money or time, and should be allowed only for necessary gainful advantages.

* The author feels that the evidence regarding the amount of life span loss from smoking is just as strong as is the evidence for the much less serious life span loss from fallout.

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Dean Franklin
At John's
I read your, request article.
Would be glad to discuss comments.

I think this is two reports in one

(1) A general review of protection, p1-18 etc.

(2) A detailed technical consideration of the Sr and CS problem, for the expert.

Suggest to separate the two; it would help the person interested in the technical details.

Toby

$ Jones paper

p 14 Suggest to define, rigorously

"Physiologic age"

"death rate"

and their connections

everything else is defined, but that

p 14 table (a) add references

(b) prefer values given by different groups of investigators (eg. smoking) with range of predictions given

e.g.

	Jones	Smith	Graham
	~~0.5y~~		
	0.45yr	( )	( )

p 15 The listed probabilities correlate with others, they are not independent.

e.g. country vs city dwelling & smoke, smog are interrelated & some of the same factors are involved. - This should be discussed. —

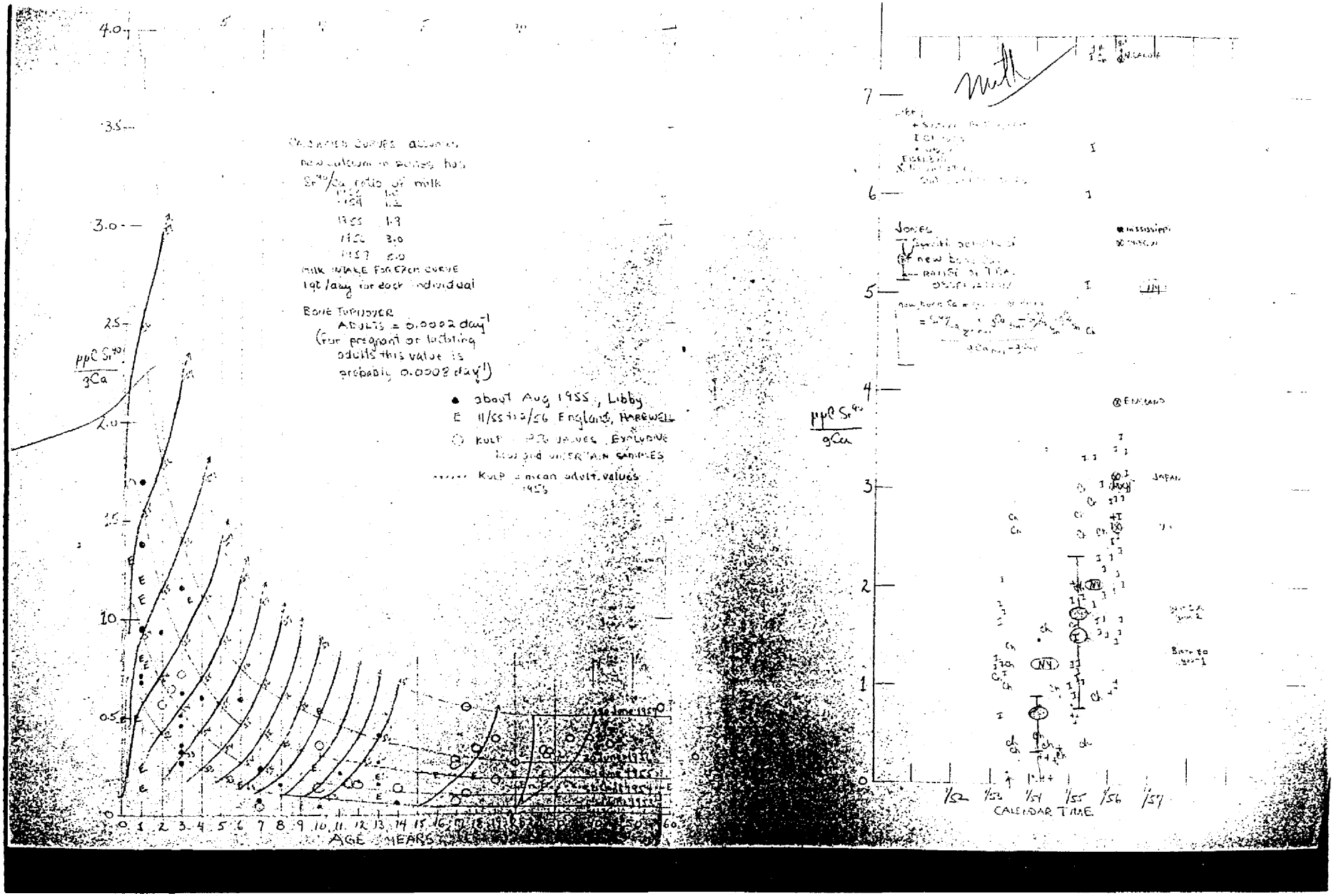
p 18 Actually p 1-18 is a condensation that could be omitted. —

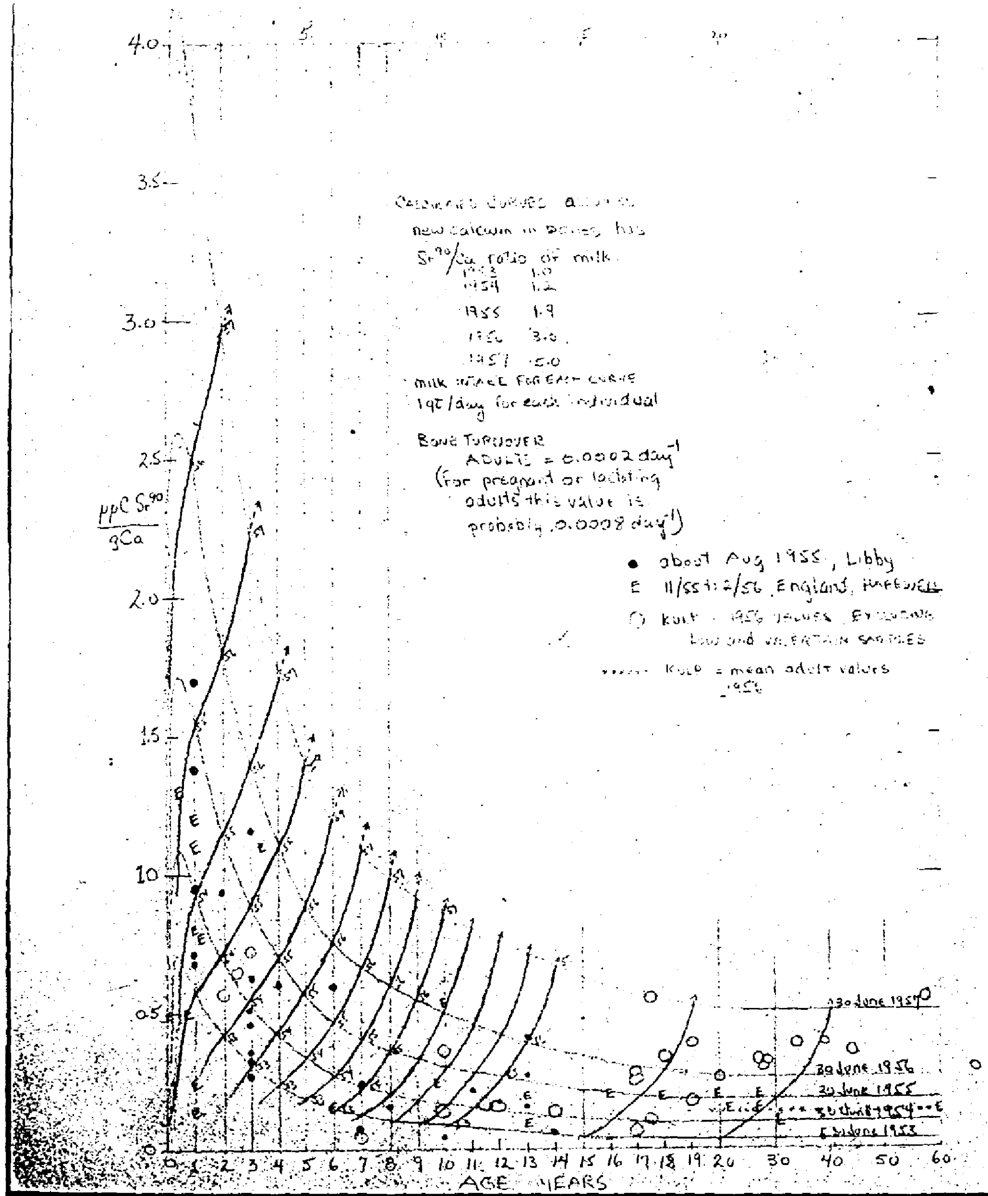
0.004 T/yr to bones

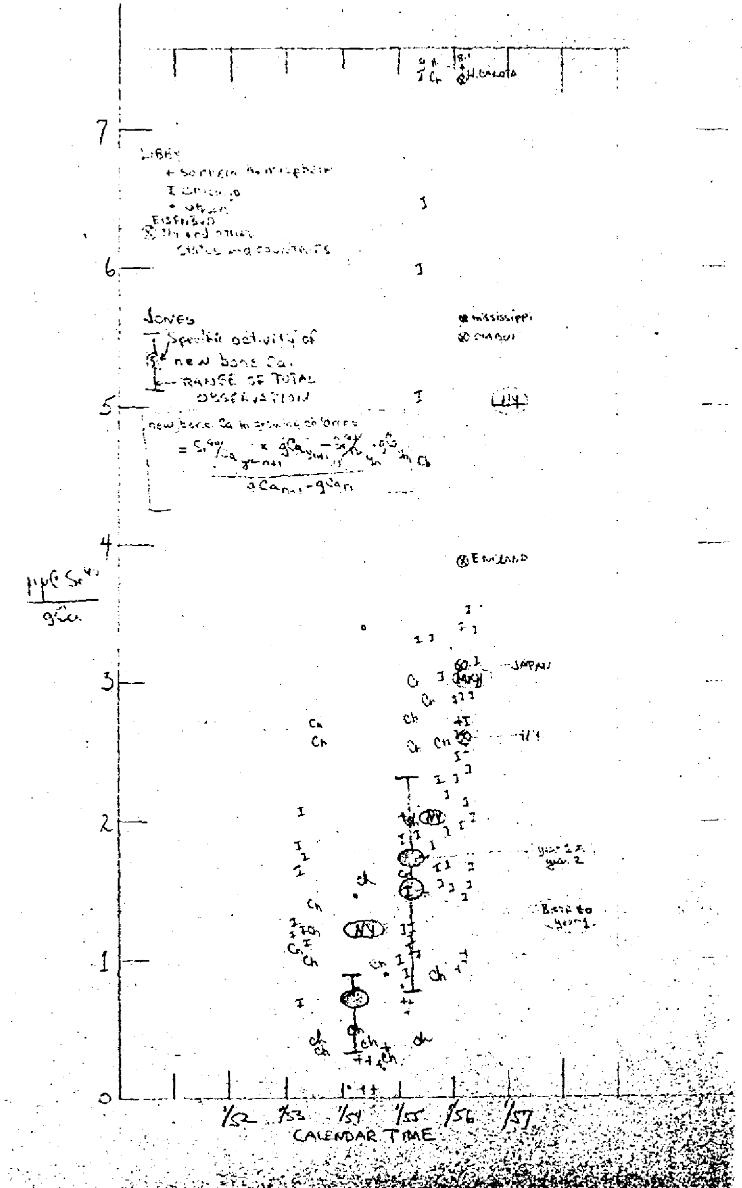
p 22 milk Sr/Ca same as what is deposited in bones.

p 24 humeon bone is only represented for 1 year by this situation. —

p 27


ppC Sr90/gCa
AGE YEARS
CALENDAR TIME
Jones
new calcium in bones has Sr90/Ca ratio of milk
1954 1.2
1955 1.9
1956 3.0
1957 5.0
MILK INTAKE FOR FROZEN CURVE
1qt/day for each individual
Bone Turnover
ADULTS = 0.0002 day-1
(For pregnant or lactating adults this value is probably 0.0003 day-1)
about Aug 1955, Libby
11/55 to 2/56 England, HARWELL
KULP 23% JARVES EXCLUSIVE
KULP = mean adult values 1955
JAPAN


Calculated curves assume new calcium in bones has Sr90/Ca ratio of milk
1953 1.0
1954 1.2
1955 1.9
1956 3.0
1957 5.0
milk intake for each curve 1qt/day for each individual
Bone turnover
Adults = 0.0002 day-1
(for pregnant or lactating adults this value is probably 0.0008 day-1)
about Aug 1955, Libby
E 11/55-2/56 England
O Kulp - 1956 values
Kulp = mean adult values 1956
30 June 1957
30 June 1956
30 June 1955
30 June 1954
30 June 1953
μμC Sr90 / gCa
AGE YEARS


Libby
Jones
Specific activity of new bone Ca
RANGE OF TOTAL OBSERVATION
Mississippi
England
Japan
μμC Sr90 / gCa
1/52 1/53 1/54 1/55 1/56 1/57
CALENDAR TIME