

National Cytogenetics, Inc.

**Assessment of Radiation Health Effects
of the
Resettlement of Enewetak Atoll**

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ASSESSMENT OF RADIATION HEALTH EFFECTS OF THE
RESETTLEMENT OF ENEWETAK ATOLL

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I. INTRODUCTION

The return of the people of Enewetak to their atoll will result in their being exposed to increased amounts of ionizing radiation from radionuclides remaining from atomic weapons tests. The magnitude of these increased doses, which will depend upon such variables as island of residence and sources of food supplies, have recently been assessed by a group from Lawrence Livermore Laboratory (Robison et al., 1979). The purpose of this paper is to present estimates of the possible impact of these doses upon the health of the present and future generations of the people who resettle Enewetak Atoll.

A number of sorts of health effects of ionizing radiation have been documented in human populations and experimental animals. Large, acute, whole body doses can produce fairly prompt, serious illness. However, at the low doses and dose rates of interest in connection with the resettlement of Enewetak Atoll, the only potential health effects are the induction of cancer among the exposed population and the induction of genetic effects that may be expressed as ill health in the next and in subsequent generations.

Upon the occasion of the Dose Assessment Conference held at Ujelang Atoll on September 19 and 20, 1979, officials of the U.S. Department of Energy presented to the Enewetak people a bilingual booklet (Ailin in ENEWETAK Rainin," or "The ENEWETAK Atoll Today") which also gives estimates of the risks of cancer

and of genetic effects that may result from the resettlement of the atoll. These estimates, based upon several different residence and future food supply scenarios, and utilizing different bases for both calculation and presentation, are compared with our own estimates.

II DOSE ASSESSMENT

The recent "Preliminary Reassessment of the Potential Radiological Doses for Residents Resettling Enewetak Atoll" (Robison, et al., 1979) has been reviewed both by ourselves and more extensively, by Dr. William E. Ogle, consultant to Micronesian Legal Services, Inc. on radiation dose assessment. Though some uncertainties remain, this document appears to present as good an assessment of the doses likely to be realized as can be achieved, and we adopt it as a basis for our health effects estimates.

Many factors influence the radiation dose estimates, the most important of which are islands of residence, islands upon which food (coconuts, pandanus, breadfruit) will be grown, and the future availability of imported foods. From the "Dose Reassessment," we have selected a scenario which appears to us to be both reasonable and conservative, and have calculated the ~~doses~~ to be used for health hazard estimation in the following manner.

It is assumed that 180 of the roughly 450 Enewetak people (i.e., all of the dri-Enjebi) go back to Enjebi island, while the rest (the dri-Enewetak) return to Enewetak island. The living pattern assumed for the dri-Enjebi is that called the "Enjebi Island/Northern Island" living pattern (pattern 1.b on page 26 of the Dose Reassessment). That assumed

for the dri-Enewetak is that called the "Southern Islands/Northern Islands" pattern (pattern 2.b on page 27 of the Dose Reassessment). The dose estimates for 30 years and for 50 years are given in Tables 35 and 42 of the Dose Reassessment, respectively. These give estimates for two diet patterns; one based upon the current normal diet which includes foods imported from outside the atoll, and the other, designated "famine," which assumes that no outside food is ever available. We consider the latter case unrealistic, since it assumes that no ship ever calls at the atoll bringing supplies of preferred staple foods (such as rice, sugar, and flour) during the entire 30 or 50 year period considered. In order to make a conservative, but more realistic appraisal, we have arbitrarily assumed that the famine case will persist no more than 25% of the time, or three months of each year.

The whole body doses to the Enjebi people from all sources (except natural background) are estimated to be 4.6 rem in 30 years under normal diet conditions and 8.5 rem over 30 years in the famine case (Table 35 of the Dose Reassessment) so the dose under our diet assumption would be

$$(4.6 \times 0.75) + (8.5 \times 0.25) = 5.6 \text{ rem,}$$

or an average of about 186 mrem per year. The comparable 50 year doses are 6.6 and 12 rem, respectively, so our estimate is

$$(6.6 \times 0.75) + (12 \times 0.25) = 8.0 \text{ rem,}$$

or 159 mrem per year on the average.

The 30 year whole body doses to the Enewetak people are esti-

mated to be 0.2 rem for normal diet and 0.33 rem for famine (Table 42 of the Dose Reassessment), so we assume a 30 year dose of

$$(0.2 \times 0.75) + (0.33 \times 0.25) = 0.23 \text{ rem,}$$

or an average of about 8 mrem per year. The comparable 50 year whole body estimates are 0.28 and 0.46 rem, so our estimate is

$$(0.28 \times 0.75) + (0.46 \times 0.25) = 0.33 \text{ rem,}$$

or an average of about 7 mrem per year.

It is of interest to consider the average yearly dose to the entire population of the atoll. Assuming 180 people returning to Enjebi and 273 returning to Enewetak, the average 30 year whole body dose may be estimated

$$(180 \times 5.6 \text{ rem}) + (273 \times 0.23 \text{ rem}) = 2.36 \text{ rem,}$$

or an average yearly dose of 79 mrem per year. The parallel average 50 year whole body dose may be similarly calculated to be 3.4 rem, or an average of about 68 mrem per year. It is true that in some years the estimated doses to individuals will be higher than this, and that the total 30 year doses to some individuals will likely be higher than 2.36 rem. However, because for doses and dose rates in the range of concern here all genetic and somatic health effect assessments of which we are aware assume a linear (or essentially linear) relationship between dose and effect, it is only the average population dose that is of concern in estimating the health effects for a population such as the people re-

returning to Enewetak Atoll. In other words, as far as health effects are concerned, the years in which the doses are low compensate for those in which they are high.

On the other hand, for genetic risk estimates, where the number of children that might be born to given parents must be assumed, it is nevertheless sometimes also of interest to consider the maximum dose a potential parent might receive. The largest individual 30 year doses for persons born after return occur under the Enjebi living pattern for children born eight years after the return and existing under famine conditions. Under the normal diet pattern the 30 year dose is 4.0 rem; in famine it is 7.5 rem (Table 44 of the Dose Reassessment). Again assuming famine conditions will exist 25% of the time, we have an upper credible 30 year dose of

$$(4.0 \times 0.75) + (7.5 \times 0.25) = 4.9 \text{ rem,}$$

or about 163 mrem per year on the average.

It is instructive, before even considering the possible health consequences of these radiation doses, to simply compare them with radiation dose levels that various other groups of people experience through their own choice of either residence or occupation. Everyone is, of course, exposed every day to natural background radiation from cosmic rays, from terrestrial radionuclides, and from naturally occurring radionuclides in ones own body. The average dose per year for Americans is about 80

mrem per year to either the gonads or the bone marrow (BEIR III, Table III-4). However, the actual rates experienced by individuals varies widely, depending on local geology, altitude and latitude (BEIR III, Figs. III-1 and III-3), so that persons living on the Colorado Plateau, for example, may receive more than twice the average, while persons living in some sections of the Southeastern United States may receive less than half. It should be noted that the residents of Enewetak Atoll are in an area of very low natural radiation background because of the near-equatorial latitude, sea level altitude, and the coral subsoil (3.5 μ r per hour, primarily from cosmic radiation Dose Reassessment, pg. 4). This may be compared with about 16 μ r per hour on the Colorado Plateau (BEIR III). Thus some persons in the United States receive doses from natural background radiation of the same order as the estimated average added doses to Enewetak people during the first thirty years after return under the Enjebi Island/Northern Island living pattern, and assuming no outside foods are available a full one-fourth of the time. Some populations in certain coastal areas of both India and Brazil where there are extensive alluvial deposits of the thorium-rich mineral Monazite experience terrestrial radiation background ten or more times the world average (UNSCEAR, 1972, Volume 1).

On occasion, human activities have concentrated naturally occurring radionuclides. As one example, persons living in houses built on deposits related to phosphate rock mining in Florida are

exposed to radioactive radon gas that produces dose rates far in excess of those to which the returning Enewetak people will be exposed. It has been recommended by the U.S. Environmental Protection Agency that corrective measures be taken for an estimated 4,000 houses in which about 14,000 people reside, to reduce the dose rates to that recommended for new construction in this area (Federal Register, 1979), but even if so corrected, the target dose rate is still almost 30 μ r per hour, which would result in an estimated whole body dose of about 190 mrem per year (assuming a 75% occupancy factor). In 30 years the cumulative dose would be 5.7 rem, which may be compared with the 30 year estimate of 4.9 rem for a child born on Enjebi eight years after the return.

Finally, many people engage in activities or occupations that increase their exposure to ionizing radiation. It has been estimated, for example (UNSCEAR, 1977: Table 33), that one round trip between Los Angeles and Paris in a conventional jet results in an added dose of 4.8 mrad, a substantial fraction of the estimated 8 mrem average per year for the people returning to Enewetak Island. Average occupational exposures are, of course, much higher. For example, in the nuclear power industry in the United States it is estimated that the approximately 40,000 workers who receive measureable doses each year are exposed to an average of between 600 and 800 mrem per year (BEIR III, Table III-23). Since employment in the nuclear industry cannot start before age 18,

this sort of dose is not accumulated over the entire life span. A nuclear industry worker who receives an average of 700 mrem per year from age 18 to age 30 will have accumulated a 30 year dose of 8.4 rem, which may be compared with the estimated 30 year whole body dose of 4.9 rem for a child born on Enjebi 8 years after the return. If such a worker were to accumulate such an annual dose over his entire working life from age 18 to, say, 65, he would accumulate 32.9 rem, over four times the estimated 50 year dose for residents of Enjebi Island.

III. GENETIC EFFECTS

Genetic health effects result from alterations in the genetic apparatus in the germ cells or their precursors. Thus they are not expressed in the persons in whom they arise, but only in the following or in subsequent generations. They are of two general types: alterations called mutations (changes in the information encoded in the genetic material, (deoxyribose nuclei acid, or DNA), and chromosomal aberrations (changes in number or form of the chromosomes, the microscopic structures containing the cells' DNA). Such changes have long been known to arise spontaneously, without known exposure to any mutation-causing agent. Identified mutation-causing agents (*mutagens*) include many chemicals and drugs, as well as physical agents like ionizing radiation and ultraviolet light.

It is generally agreed that where possible it is best to base human health effects estimates upon human data. However, despite intensive study over the past fifty years, there is virtually no positive evidence of any genetic effects having arisen as a consequence of human radiation exposure. Human generations are simply too long, and the sizes of the populations required far too great to allow their detection. However, there is positive evidence from all of the experimental organisms tested, and because of the similarities between the genetic apparatus of all organisms including man, we may be certain that

human radiation exposure must also produce genetic effects, even though their frequency may be so low as to be undetectable.

Because of the unavailability of human data, estimates of the risk of radiation-induced human genetic health effects must perforce be based upon data from experimental animals, and all recent estimates by national and international committees have depended very heavily upon the large body of data now available for the laboratory mouse. The mouse is a mammal like man, and the negative human data, most importantly from study of the offspring of Hiroshima and Nagasaki atomic bomb survivors, is consistent with the mouse data. Though there remain uncertainties, we may thus be confident that the extrapolation from mouse to man cannot greatly underestimate the genetic health effects of human radiation exposure.

Furthermore, neither is there any direct evidence for the induction of genetic effects in animals by doses as low as those of interest in connection with the return of the Enewetak people to their atoll. The effects of doses below a few tens of rem are simply too small to be detected statistically. Nevertheless, both indirect experimental evidence and theoretical considerations strongly indicate that genetic effects are in fact induced by even very low doses of ionizing radiation, and that, in the range of dose and of dose rate that is of interest here, the numbers of effects produced is a simple linear function of dose.

Genetically related ill health is ubiquitous in human populations; almost 11% of live births in the United States are affected (BEIR III, TABLE IV-2). Such ill health spans the entire range from major congenital defects incompatible with life to relatively trivial conditions that, while eventually requiring medical attention, are of relatively minor consequence in the lives of the affected person. It is important to recognize that, as far as is known, ionizing radiation produces only the same kinds of genetic changes as occur spontaneously. No novel types, arising only from radiation, have ever been observed. It follows, then, that any genetically related ill health which might arise as a result of human radiation exposure must be indistinguishable from that which already occurs spontaneously. Thus no case of genetic ill health in any particular individual can ever be absolutely attributed to parental radiation exposure. Only the probability can be assigned. Furthermore, the smaller the dose the less likely it becomes that parental irradiation was the cause.

Mutations are generally recognized to be likely to be detrimental to the health of individuals carrying them. The degree of harm varies from mutation to mutation, and a few might even be beneficial under certain circumstances, but it is widely agreed that any increase in the human mutation rate may be expected to result in some net increase in human ill health.

Though mutations may be induced in any body cell that has a nucleus, we define as genetic effects only those that arise in germ cells or their precursor cells that actually contribute to the next generation. Consequently, the doses of concern are only those actually received by these cells, or, for practical purposes, by the gonads. Generally speaking, the gonadal dose is roughly equivalent to whole body dose. Conversion of whole body or gonadal dose to genetic effects estimates also requires an estimate of the number of children the person might subsequently have. Often, as in the present use, this requires that some assumptions be made about the parental population actually exposed and about the rate at which it will reproduce.

The available data bearing on human genetic radiation effects estimates are periodically reviewed and evaluated by various National and International committees, most notably those of the U.S. National Academy of Sciences (BEAR Report, 1956; BEIR I Report, 1972; BEIR III Report, 1979), and the United Nations Scientific Committee on the Effects of Atomic Radiation (most recently, UNSCEAR Report, 1972; UNSCEAR Report, 1977). Each report has developed and updated formulations of the probable genetic effects of human ionizing radiation exposure. The most recent effort is the report of the 1979 BEIR III Committee's Genetic Effects Subcommittee. We adopt their

findings and methodologies as the basis of our present analysis of genetic risk for the Enewetak population. We note, however, that adoption of those used in any of the earlier reports would not result in any dramatic change in our numerical assessment.

Because of differences in the circumstances under which different mutations are expressed as ill health, two types of risk estimates may be made. Some mutations, called dominant, as well as some chromosomal aberrations, are expressed in the first generation following exposure of the parental generation. Other mutations, called recessives, are not expressed in carrier individuals who inherit them from only one parent, so expression cannot occur in the first generation, but only in later generations when two carriers mate and produce an affected child who receives the mutant gene from both parents. Different estimates must thus be made for the expression of dominant genetic effects in the first generation and for the ultimate expression of both surviving dominant mutations and of recessive mutations.

The BEIR III Committee has used two separate approaches to making these kinds of risk estimates, both based very largely upon experimental data for the laboratory mouse, though taking into account the very limited pertinent human information. For estimation of effects in the first generation following parental exposure, a so-called "direct" method depending upon observations of induced heritable skeletal abnormalities in mice. The frequ-

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ency of such serious skeletal abnormalities per rem of exposure was adjusted to estimate anomalies of all organ systems. This approach provided an estimate that 5-65 induced dominant disorders would be expected during the lifetime of 1 million live-born children following parental exposure to one rem of radiation. Based largely on information from human spermatogonial irradiations and the observed frequency of chromosome abnormality, it was possible to develop in addition an estimate of the number of offspring of irradiated parents who would manifest a genetic disorder as a result of some induced chromosomal anomaly; from 0-10 affected per million offspring per rem of parental exposure. Thus the total number of cases of genetic ill health estimated to occur in the first generation is a range of from 5 to 75 per million offspring per rem of parental radiation.

The second risk estimate methodology^y employed by the BEIR III Committee (as well as the previous BEIR I Committee) is called the "indirect" method. This procedure permits aⁿ estimate of the number of genetic disorders to be expected in each generation following many generations of parental radiation, when an equilibrium has been reached between the rates at which new genetic ill health is induced in each generation and the rate of elimination in each generation through its expression in affected individuals. By definition, this equilibrium estimate is numerically the same as the total number of affected individuals to be expected over all future generations following

a single exposure of one generation, essentially the case of interest in connection with the Enewetak people. Estimates by this method integrate the number of additional disorders over literally hundreds or thousands of generations into the future.

The indirect equilibrium genetic effects estimate made by the BEIR III Committee is based largely upon extensive data on the induction of recessive mutations in mice. From these induced rates and the observed spontaneous mutations rates, a relative mutation risk (i.e., the reciprocal of the so-called "doubling dose," or amount of radiation exposure required to double the spontaneous mutation rate) is derived. The relative mutation risk factor was used together with estimates of the degree to which the frequency of human ill health is responsive to mutation frequency to derive an equilibrium estimate of from about 60 to possibly as many as about 1,100 affected individuals per million live born per generation per rem of parental exposure in each generation.

It is exceedingly important that the BEIR III Committee's numerical genetic risk estimates, and indeed any such risk estimates, be placed in proper perspective by comparison with the incidence of such effects to be expected spontaneously in the same population. The estimate of current incidence given in the BEIR III Report is 107,100 per million live births; that is 10.7% of all human live births. The estimated first generation increase

of between 5 and 75 cases per million live births per rem of parental exposure (i.e., 0.0005-0.0075%) may, then, be more meaningfully expressed as an increase from 10.7% to somewhere between 10.7005% and 10.7075% per rem of parental exposure.

The "all time" (i.e., equilibrium) estimate of from 60 to 1,100 cases per million per rem of parental exposure is more difficult to put in perspective, simply because neither the total number of future generations nor the future population dynamics is known. However, the number of persons that will eventually descend from most human population groups is clearly very large, so the "all time" estimates would certainly be diluted out over many, many generations, in each of which we might expect a very large "background" of spontaneously arising cases of genetic ill health.

IV. SOMATIC EFFECTS

Of the somatic effects of ionizing radiation, cancer induction is that of the greatest concern. Unlike the case for genetic effects, there is much direct positive evidence for statistical increases in human cancer incidence and mortality following radiation exposures. Leukemias and various cancers of solid organs are known to be increased following exposure to high doses of ionizing radiation, and to occur in the ratio of approximately one leukemia to four cases of other forms of cancer. Good information relating the risk of cancer induction to radiation is available from human studies involving many different kinds of exposures, and there is surprisingly good agreement between the different large studies for doses above approximately 50 rads.

Cancer occurs spontaneously, of course, and with an unfortunately high frequency in all human populations. There is furthermore no known means of distinguishing a cancer induced by radiation from a spontaneously arising one. Thus no case of cancer may be definitely attributed to a prior radiation exposure; only the probability that it might have been can be assessed. And the lower the dose, the less likely it becomes that a given cancer was radiation induced.

Knowledge concerning the molecular and cellular mechanisms by which radiation induces cancer is unfortunately not as complete

as for genetic effects. The process may vary depending on the type of tumor. Though there are human data, they are limited to dose ranges above those with which we are usually concerned, and in considering the possible consequences of population exposures like those estimated for the people of Enewetak it is, therefore, necessary to extrapolate below the range at which positive effects have been documented. The three types of dose-response curve usually considered in making such extrapolations can be described by a single mathematical function which has terms both linear and quadratic in dose, and this relationship has been accepted as best fitting the available human leukemia and the available animal tumor data. The predictions of such a model fall in between those derived from fitting the data with either of the extreme limiting cases, the purely linear one or the pure quadratic which have been used to describe radiation dose-effect curves for breast cancer and for skin cancer, respectively. In the calculations presented later in this report, we use the linear-quadratic model to provide what we believe to be the best estimates of cancer risk, and the linear model predictions in order to place an upper credible bound on these estimates.

In estimating the possible number of cancers that may be induced by radiation exposure, the following observations are pertinent:

1. As already noted, cancers induced by radiation cannot be distinguished from those that occur naturally. Their existence may only be inferred from observations of statistically significant increases above the natural incidence. Tissues and organs of the body vary considerably in their sensitivity to the induction of cancer by radiation.

2. The natural incidence of cancer varies significantly in magnitude, depending on the type and site of the neoplastic growth, age, sex, and other factors.

3. The time elapsing between irradiation and the appearance of the detectable neoplasm is characteristically long, i.e., years or even decades.

4. This long latent period is taken into consideration in our risk calculations by assuming that risk observed during the first 30 years following radiation can be projected into the future either at a fixed level (absolute projection model) or at a rate which increases with natural cancer rates (relative risk projection model).

5. Some of the existing human and animal data on radiation-induced cancers come from populations exposed to internally deposited radionuclides for which dose-incidence relationships are influenced by marked nonuniformities in the temporal and spatial distribution of radiation within the body.

From the large human experience now accumulated, a clear-cut increase in cancer mortality with increasing radiation dose

has been documented. Most of the information now available is reasonably consistent from one irradiated human population to another; this suggests that it can be used for estimating risks to the general population.

It is generally acknowledged that there is no good evidence that dose rates of gamma or x-irradiation of the order of a few hundreds of mrem per year are in anyway detrimental to the exposed people. It is important to recognize that the absence of sound evidence for any effect at these very low doses means that it is possible that either there might be no effect at all, or there might be an effect equal to the upper range of risk estimates. The fact that knowledge is not precise at these low doses arises from the fact that if excess risk is truly proportional to dose, and if 1,000 exposed and 1,000 control subjects are required to adequately statistically test for the cancer excess at a dose of 100 rem, then about 100,000 in each group would be required at 10 rem, and 10,000,000 in each group would be needed for a dose of one rem.

The available data on human cancer induction by ionizing radiation have been reviewed most recently by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1977) and by the U.S. National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiations (BEIR III, 1979). The estimates provided in these Reports have been used in making

the present estimates of the risk of induced cancer that will result from the return of the Enewetak people to their Atoll. Based upon their comprehensive review of current knowledge of radiation induced cancer, the BEIR Committee calculated the risk of cancer mortality in accordance with different models that relate dose to observed effects. The highest risk estimates are derived from linear (L) models, and the lowest from dose-squared (D^2) models. The Committee is presently attempting to define an intermediate model, for all cancers, like the mixed, linear quadratic (LQ) model adopted for leukemia analyses. The computations presented in this document are based on the LQ model, which many believe produces the most reasonable results, and the upper limit is represented by the L model.

V. SPECIFIC RISK ESTIMATES

A. Genetic Effects

As already noted, any estimation of possible genetic effects arising as a consequence of population exposure to ionizing radiation requires some assumptions regarding population dynamics, birth rate, generation time, etc.

For most purposes, a generation is taken to be 30 years. In the United States, it is currently a bit shorter, but 30 years is still taken as a convenient simplification. For the Marshall Islands, the Five Year Comprehensive Health Plan gives a breakdown by age of mother (Table V-3, Page 120) that shows the age of mothers at the "average" or "middle" birth to be only 23-24 years. However, no information is available on the age of fathers, who seem likely to be older than the mothers, and in any case the usual 30 year interval is used here. It should be noted that to the extent the Enewetak generation is actually shorter, this tends to overestimate dose to the parents of the average child, and thus the genetic health risk estimate.

While genetic risk estimates may be expressed per live birth, thus avoiding any assumptions about future birth rates, it is helpful to attempt to estimate the total risk for the entire next generation of the Enewetak people. As a minimum, we might simply assume a "replacement" birthrate of 453 live births over the next 30 years, or about 15 births per year. As a maximum, we might

assume that the average birth rate in the entire Marshall Islands for the 20 years from 1955 to 1975 might apply to the Enewetak population for the next 30 years. From Table III-1 of the final draft of the Five Year Comprehensive Health Plan, one can calculate that the average yearly birth rate for the period (it seems remarkably stable over this period) is 39.7 ± 4 births per 1,000 of population; for practical purposes 40 per 1000 or about 11 per year for the 273 people assumed to return to Enewetak and about 7 per year for the 180 people assumed to return to Enjebi.

Of course, should the present birth rate and current population growth rate of the order of 3-4% per year continue, the absolute numbers of births will grow during the coming 30 years. Assuming a 4% growth rate, the Enewetak population may include 816 people 15 years from now, and about 33 births might be expected that year, while in thirty years there would be almost 1,500 persons, with well over 60 births per year. It seems unlikely that the population will grow to this extent; in view of the uncertainties involved, perhaps a reasonable assumption would be that there will be not more than roughly 1000 births in the population during the next 30 years.

With exponential population growth, roughly one-half of the births expected over 30 years will occur during the first 20 years; the remainder will occur during the final 10 years.

In view of the uncertainties involved, it seems reasonable to assume as an upper credible limit that there will be 1000 births, the average accumulated parental dose for which will be that accumulated for the first twenty years. However, the doses were calculated for 30 years, and since these are not enormously larger than the 20 year integral doses (see Fig. 3 of Dose Re-assessment), they are used here as upper bound estimates of the doses of genetic significance in calculating genetic risk.

The population dose estimates described earlier give the average 30 year dose for all members of the Enewetak people that return, allowing for 180 returning to Enjebi Island, as 2.36 rem. The BEIR III estimates are for a population receiving one rem in thirty years (BEIR III, 1979, Table IV-2). The first generation estimate for the Enewetak people, which may be obtained by adjusting by the ratio of doses (2.36/1) the range of risk from BEIR III. Thus the increased risk is from (2.36x5) to (2.36x75), or 11.8 to 177 per million live births.

As a minimum estimate, we assumed the present population might just replace itself in 30 years; i.e., 453 births. The risk, then, would be

$$(11.8 \text{ to } 177) \times 453/1,000,000 = 0.0053 \text{ to } 0.0802$$

additional cases. Assuming a 10% spontaneous risk, we would expect 48.5 cases to occur naturally during the same period. Thus the upper bound risk in this case is that the normally expected 48.50 cases arising during the next 30 years might conceivably

increase to as much as 48.58, an increment of less than two tenths of one percent.

Assumption of the higher number of 1000 births in the next 30 years simply increases the absolute numbers proportionately: The risk becomes

$$(11.8 \text{ to } 177) \times 1,000/1,000,000 = 0.012 \text{ to } 0.18$$

additional cases in 30 years, against a spontaneous total of 107 cases.

To provide an absolute upper limit to credible risk of genetic ill health, we might consider a child born to a couple born on Enjebi eight years after the return. They would receive as much as 4.9 rem in 30 years, so the risk to a child born to them at age 30 would be roughly 5 times the BEIR II risk for 1 rem, or (5×5) to $(5 \times 75)/1,000,000 = 25$ to 375 per million or roughly 3 chances in 100,000 to 3 chances in 10,000. This is of course, in addition to the 10.7 chances per hundred normal risk.

B. Cancer

Approximately 15% of the U.S. Population, at birth, are destined to develop cancer during their lifetime. The risk of several forms of cancer is high in young children, falls in middle age and thereafter the risk of cancer increases with increasing age. There are many causes of cancer in addition to radiation, and as already noted, there is no characteristic that allows one to distinguish radiation-induced from "spontan-

eous" or natural cancers. Mortality data for the Marshall Islands (final draft of the Marshall Islands Five Year Comprehensive Health Plan) suggest that reported mortality from cancer is less frequent among the Enewetak people than among the United States population. This is surely in part due to poorer diagnosis of cancer among the Marshallese. However, the structure of the Enewetak population, and indeed that of Marshall Islanders in general, is also markedly different from that of the United States, there being relatively more young people and proportionately fewer old people. Since the risk of dying of cancer increases with increasing age, one would expect a lower current death rate from cancer for the younger Enewetak population. For these reasons, and in order to be conservative, we will assume that the spontaneous cancer risk for this population is in fact like that for the United States population, or about 15% at birth.

Leukemia is the most well understood cancer that can be induced by ionizing radiation. Following large doses in large populations, the incidence can be observed to increase in 2 years, peak in 5 to 10 years, and fall to background levels in 25 years. For this cancer we know the total expressed risk and can compute the number of cases expected with precision. For other cancer the latent period is longer, and the length of time the risk remains elevated is less well known. Two models are used to make risk calculations for these cancers--

absolute and relative risk projection models. The absolute risk projection model assumes that the risk observed in the first 25 years persists throughout life at that observed rate. Thus, if the risk falls in later years, as it does for leukemia, the expected risk would be overstated. The relative risk model assumes that the increased risk increases with the spontaneous cancer rates. Thus, as the cancer risk rises with age, so does the expected radiation-induced risk. This model results in the highest risk estimates, and when compared to calculations by the UNSCEAR Committee (UNSCEAR 1977) exceeds their risk estimates by 2 to 4 times.

Thus, it seems reasonable to accept the linear model and the relative risk projection model, as furnishing maximum credible risk estimates, and the linear-quadratic as providing the best estimate under either risk model.

The risk coefficients for these models are given in Table 2. These coefficients, when multiplied by the normal cancer incidence observed, give the increase expected from lifetime exposure (from birth) to 1 rem/year. Thus, assume 500 people are exposed to 0.25 rem per year through life. In the U.S., at current cancer rates, 15% of the population at birth, are expected to die ultimately with cancer; i.e., $500 \times .15 = 75$ cancers are expected normally. If the risk coefficient is

1.8% per rem for lifetime exposure, then

$$75 \times \frac{1.8 \times 0.25 \text{ rem/yr}}{100} = 0.34$$

added or new cancers are expected from radiation above the 75 expected ordinarily in the lifetime of the population.

TABLE 1
RISK COEFFICIENTS (PERCENT) FOR INCREASE IN CANCER MORTALITY
FOLLOWING 1 REM PER YEAR FOR LIFETIME*

DOSE-RESPONSE MODEL	RISK PROJECTION MODEL	
	ABSOLUTE	RELATIVE
LINEAR-QUADRATIC	1.95	3.65
LINEAR	6.65	14.35

*Percent increase in cancer risk (as a multiple of normal cancer risk) from lifetime exposure to 1 rem/year. From BEIR III May 1979 Report and from the most recent unpublished draft.

Using the above described data and computational methods, and the dose estimates already discussed, the number of new cases of cancer expected in the residents of Engebi and the Southern Islands are presented in Table 2.

From these calculations, it is clear that persons living on the Southern Islands, eating 10% Northern Islands foods when they become available, might have as high as .05 of an added cancer above the $.15 \times 273 = 40.95$ expected in the lifetime of the 273 persons expected to return to the Southern Islands. It should be noted that the risk figures used assume that the population is exposed from birth, and since this is an obvious overestimate, even fewer cases would actually be expected. This can be balanced out against the increasing population that will be born to residents on the island, at later times, when doses on the average are lower.

For persons who return to Engebi, the estimated number of additional cancer cases above the $180 \times .15 = 27$ expected, are between 0.10 and 0.66 and again the upper estimates are 2-4 times higher than those that would result if the risks produced by the UNSCEAR Committee were used instead. In any case, radiation-induced cancer mortality in the lifetime of the population is estimated to be less than a single case.

TABLE 2

NO. OF ADDED CANCERS DUE TO LIFETIME EXPOSURE (50 YRS) TO
INHABITANTS OF ENJEBI AND SOUTHERN ISLANDS

POPULATION GROUP	ABSOLUTE RISK PROJECTION MODEL		RELATIVE RISK PROJECTION MODEL	
	LINEAR-QUAD. DOSE RESPONSE MODEL	LINEAR DOSE RESPONSE MODEL	LINEAR-QUAD. DOSE RESPONSE MODEL	LINEAR DOSE RESPONSE MODEL
ENJEBI/NORTHERN ISLES	.09	.30	.17	.62
SOUTHERN ISLES	.01	.02	.01	.04
TOTAL GROUP	.10	.32	.18	.66

VI. COMPARISON WITH DEPARTMENT OF ENERGY RISK ESTIMATES

In the booklet "Ailin in Enewetak Rainin" (The Enewetak Atoll Today) the U.S. Department of Energy presented their own estimates of the numbers of genetic effects and of cancers that might occur as a consequence of the return of the people of Enewetak according to a number of different living and dietary patterns. Because the BEIR III Report was available only in draft form, their estimates were based upon the risk factors contained in the Committee's earlier report (BEIR I, 1972). In contrast, our own estimate uses the more recent BEIR III draft Report risk factors. In addition, the DOE estimates were made for either the continuous availability of outside food or the "famine" case doses from the Preliminary Reassessment, so no case is strictly comparable to those we used. Because their mode of presentation of both radiation doses and risk estimates is somewhat different from those we favor, it might appear at first glance that the DOE estimates are much higher than our own. However, such is not actually the case.

The Enjebi Island/Northern Island living pattern is considered for the two dietary patterns on pages 22 and 23 of the booklet, for example. The 30 year whole body doses listed are the same ones we obtained from the Preliminary Reassessment: 4.6 and 8.5 rem. The cancer risk estimates given, however, consider only these 30 year doses, whereas we have used the 50 year doses. Thus for comparison we may calculate parallel

30 year dose cancer risks as 14.35 percent for a one rem per year radiation dose (under the linear dose-response and the relative risk models), times either .153 rem per year or 0.283 rem per year average over the 30 year period, or either a 2% or a 4% increase in natural risk. These are, by coincidence, precisely the same numerical values presented in the DOE booklet.

The BEIR III genetic effects upper bound estimate of 75 cases per million live births per rem of parental exposure is .07% of the spontaneous incidence, so for the 50 year integral doses listed the upper bound increases expected would be 0.32% and 0.6% respectively. The DOE estimates of 0.92% and 1.7%, though larger, are only about three times the comparable estimates from the BEIR III risk factors, and this is simply a reflection of the larger BEIR I genetic risk estimates.

VII. CONCLUSION

Even using conservative assumption, the average yearly radiation doses likely to be experienced by the people of Enewetak following reoccupation of the atoll, including residence of the dri-Enjebi on Enjebi Island, are relatively small, and comparable to those experienced by many other populations elsewhere in the world through their choices of either residence or occupation. It is entirely possible that this radiation exposure will never result in even a single case of disease among either the returning population or their descendants. It is estimated that the upper credible limit of genetic risk is 0.18 additional case in 1,000 live births over the next 30 years, which may be compared with some 100 cases that are expected to occur naturally among these 1,000 births, without any added radiation exposure. Furthermore, using very conservative estimation models, the upper credible limit on the number of cases of radiation-induced cancer among the returning people is only 0.66 case, which may be compared with more than 60 cases than are expected to occur naturally during the lifetime of the population. Though presented in somewhat different form, our upper credible risk estimates are in substantial agreement with those presented to the Enewetak people on September 19, 1979, by the U.S. Department of Energy.

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