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THE BIOLOGICAL HAZARDS OF A FALLOUT FIELD [1]

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BACKGROUND

THIS paper deals with the radioactive hazards associated with an acute fallout situation; that is, the type of fallout that may occur in a limited area down wind of a large nuclear detonation. Such fallout is likely when the fireball touches the ground drawing up large amounts of incinerated matter which, due to the relatively large particle size, is deposited in an area which may cover thousands of square miles and cause lethal radiation over a wide area. This is in contrast to the so-called world-wide fallout when, following a nuclear detonation at high altitude, radioactive debris of small particle size escapes into the trophosphere or stratosphere to be slowly filtered down over large areas of the globe. Due to diffusion and the time element allowing radioactive decay, the resulting contaminating situation is chronic in nature, imparting very low doses of radiation.

Acute fallout may resemble snow, powder or mist while it is falling. It seems likely that enough fallout to result in lethal accumulation would be visible; but this is not certain. The distribution is fairly uniform and contaminates trees, houses and human beings with a thin, powdery tenacious material which is highly radioactive. Human beings at distances from the detonation will not be exposed to flash burn or immediate radiation. However,

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fallout which may occur exposes human beings to the following hazards: (1) the gamma radiation is most penetrating, resulting in whole body exposure which may be sufficient to result in acute radiation syndromes; (2) the deposit of fallout on the exposed skin may produce beta radiation burns; (3) the ingestion and in-

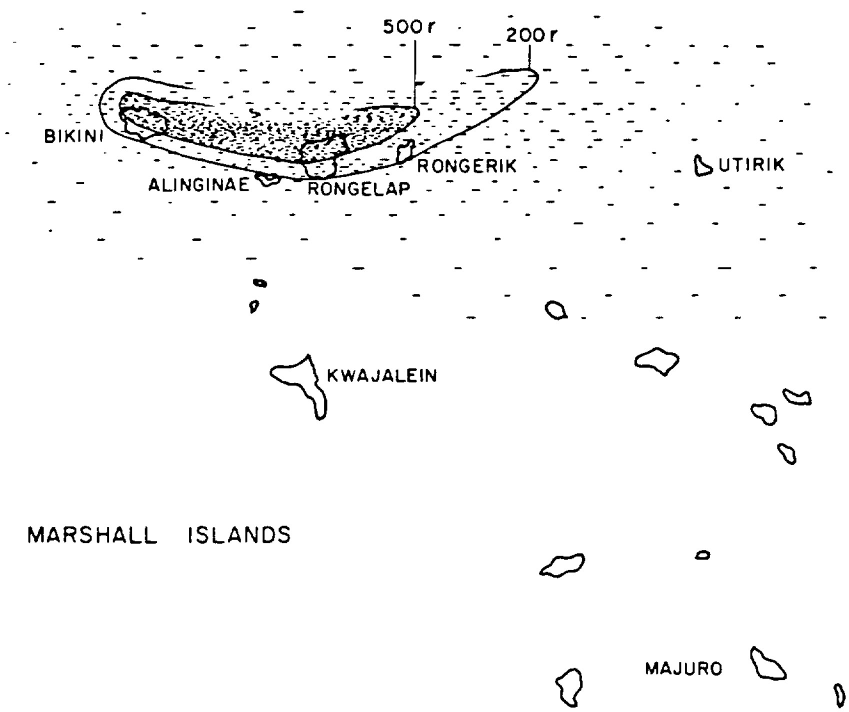

Figure 1. Map of Marshall Islands showing area of 1954 fallout.

halation of the material may result in accumulation of radionuclides internally.

We have learned much about fallout effects on human beings from our studies of the people of Rongelap Atoll in the Marshall Islands who were accidentally exposed to an acute fallout situation in 1954 (see Figure 1). The people were evacuated to another island 2 days after the accident. Annual medical surveys of these people have been carried out, the last having just been completed in March of 1960 (1–6). Although the dose was sublethal, these studies of the Marshallese offer a background for describing radiation hazards and their effects. There were eighty-two men, women and children of all ages, sixty-four of whom were exposed to 175 r and eighteen to 70 r estimated whole body gamma exposure. In addition many people suffered beta burns of the skin with epilation and showed measurable amounts of radioactive materials internally deposited. The three potential hazards will now be described—with references made to the effects on the Marshallese.[2]

FALLOUT HAZARDS

Penetrating Gamma Radiation

Acute Syndromes

Of the potential hazards associated with fallout, the most serious is that of exposure to gamma radiation. Such exposure may result in acute radiation syndromes and, if survival occurs, in late effects of exposure. Large doses of radiation (thousands of roentgens given in a short period of time) are known to produce a neurological syndrome characterized by disorientation, ataxia, convulsions, vomiting, diarrhea and death within hours. However, this syndrome is not likely to occur in a fallout situation since the dose accumulation (dose rate) would not be rapid enough; most likely the next syndrome to be described would predominate.

Doses of 1000 r or more result in the gastrointestinal syndrome which includes early persistent anorexia, nausea, vomiting, abdominal cramps and diarrhea with dehydration, apathy and death in 4 to 5 days. Death occurring before hemopoietic depression is a prominent feature. However, with lower doses in the lethal range (225 to 650 r?) the classical hematological radiation syndrome occurs and is marked primarily by depression of hemopoiesis with the possible development of infectious processes from leukopenia and immunological depression, hemorrhage and anemia from platelet depression and later anemia from erythropoietic depression. Gastrointestinal symptoms of nausea, vomiting and diarrhea

[2] There were also twenty eight American servicemen on Rongerik Atoll who received an estimated 70 r and one hundred and fifty-seven Marshallese on Utirik Atoll who received only about 14 r. In addition there were twenty-three Japanese fishermen on the Lucky Dragon who were exposed to about the same dose as the Rongelap people (7).

may occur early and again late in the syndrome but are not outstanding features. With sublethal doses there may occur mild, early gastrointestinal symptoms and later slight or moderate hemopoietic depression.

Acute Effects on Marshallese. In the people of Rongelap receiving a high sublethal dose (175 r) there was widespread nausea, and a few people vomited during the first day or so after exposure.

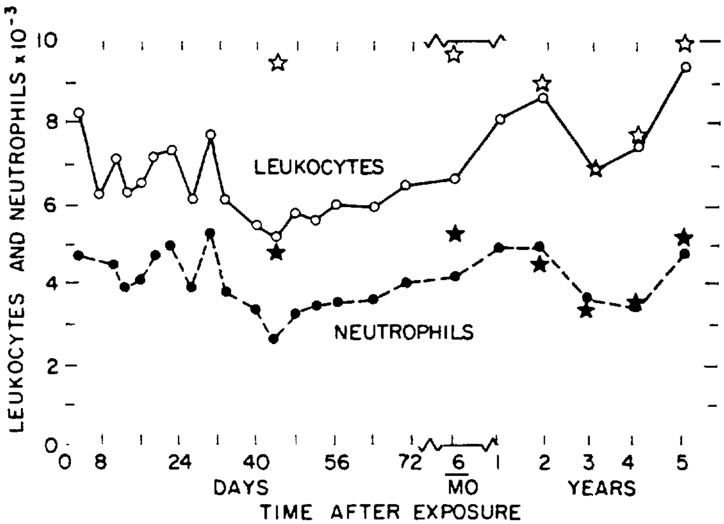

Figure 2. Mean neutrophil and total leukocyte counts of exposed Rongelap people from exposure through five years after exposure. Stars represent mean values of comparison populations.

Hemopoietic depression, though moderately severe, was not sufficient to result in clinical effects. Figure 2 shows the changes in total leukocytes and neutrophils. Following depression to about half the mean level of the comparison population at 40 to 45 days, there was gradual recovery with the mean counts reaching comparison population levels by about one year. During the period of maximum neutrophil depression, an epidemic of infections of the upper part of the respiratory tract occurred. The exposed people showed no adverse response to this epidemic, and later their responses to epidemics of measles, chicken pox and other diseases did not appear to be impaired.

Figure 3 shows the changes in the lymphocytes which were depressed early to half the level of the unexposed population and were very slow in recovering, not completely reaching the level of the comparison population until about five years after exposure. The platelets (see Figure 4) became depressed to about 30% of the comparison population by four weeks after exposure with a slow recovery pattern thereafter. Even after five years, the mean platelet level was still slightly below the comparison population level. No

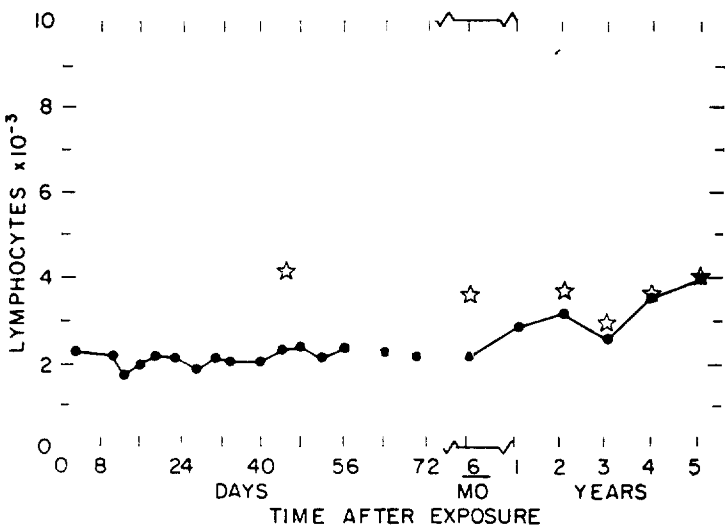

Figure 3. Mean lymphocyte values for exposed Rongelap people from exposure through five years after exposure. Stars represent mean values of comparison populations.

bleeding was associated with low platelet levels. In eleven people counts dropped to a low of thirty-five thousand to sixty-five thousand. Erythropoietic changes were not remarkable. Since there were no clinical signs associated with hematological depression, no specific treatment was required or given. No illnesses or deaths have occurred that appeared to be related to radiation exposure. Had the dose received been higher, there would almost certainly have been complications of infections and bleeding with possible mortality. During the first six weeks over half of the exposed people lost a few pounds in weight even though their diet was satisfactory during this period. This possibly reflects an effect of radiation exposure on general metabolism, but factors associated

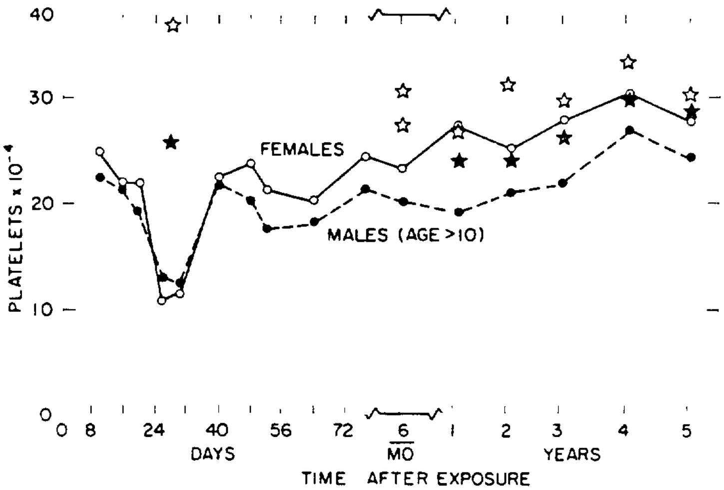

Figure 4. Mean platelet values for exposed Rongelap people from exposure through five years after exposure. Stars represent mean values for comparison populations.

with change in environment cannot be ruled out. Four babies irradiated *in utero* appeared normal at birth.

Late Effects

It has been learned, from animal studies and limited experience with human beings, that certain late effects of radiation may occur in those surviving acute gamma radiation. Some of these are: shortening of life span (8, 9, 10, 11); premature aging and early development of degenerative diseases (12, 13); earlier appearance and possibly increased incidence of cancer and leukemia (14, 15, 16); radiation cataracts (17); impairment of reproduction (18, 19); impairment of growth and development (20, 21); and genetic effects (22).

Late Effects in the Marshallese. The only possible effects noted in the Marshallese have been: (1) the incidence of miscarriages and stillbirths appears to have been somewhat higher in the exposed women than in the unexposed—but a deficiency of vital statistics and the small number of women involved preclude definite conclusions; (2) evidence suggestive of a slight lag in growth and development in the exposed children during the first two years after exposure is being re-evaluated on the basis of better age data which are being obtained. None of the other aforementioned late changes have been observed in the Marshallese.

BETA BURNS

Cutaneous irradiation from the less penetrating components of the fallout may result in beta burns and epilation of varying severity. This is due largely to the fallout in direct contact with the skin with a lesser contribution from the ground source. As the name implies, beta radiation accounts for most of the skin dose causing lesions since its energy is almost entirely absorbed in the skin as compared with only a small percentage of the gamma energy. The gamma dose alone would have to be supralethal to

Figure 5. Spotty epilation in boy, age 13, at 28 days after exposure. Note scalp lesions in area of epilation.

Figure 6. Hyperpigmented plaques and bullae on dorsa of feet and toes 28 days after exposure. One lesion on left foot shows deeper involvement. Feet were painful at this time.

produce significant skin burns. The hazard of beta burns is not so serious as the whole body gamma radiation hazard, but resulting lesions may be quite disabling and, if coupled with severe radiation neutropenia, lesions may become badly infected, possibly enhancing the development of septicemia. The prophylatic measures of avoidance of contact by taking shelter or by prompt cleansing of the skin with water (not necessarily complete decontamination) would practically eliminate this hazard.

Description of Lesions

Beta burns are not very different from X-ray or other penetrating radiation burns except that they tend to be more superficial. Early symptoms of burning, itching and tingling with possible erythema a few hours after exposure may be accompanied by some edema and blanching in severely irradiated skin. These symptoms usually subside to be followed by a generally asymptomatic latent period of days or weeks, depending on the dose (the larger the dose the shorter the latent period). Then pigmentation and erythema may develop in the irradiated area with desquamation and, in more severe burns, ulceration and pain of varying de-

Figure 7. Same case as in Figure 6, six months later. Foot lesions have healed with repigmentation except depigmented scars persist in areas where there were deeper lesions.

gree. Patchy epilation may also occur at this time. Healing with re-epithelialization and repigmentation is likely except with deeper burns, in which case non-pigmentation, atrophy and scarring may remain with possible development of chronic radiation dermatitis and the risk of malignant change.

Beta Burns in the Marshallese. In the Rongelap people white, powdery fallout material clung tenaciously to their skin causing itching and burning with irritation of the eyes and lachrymation during the first 24 to 48 hrs. It was over a week before complete decontamination was possible. It was impossible to calculate the skin dose with any degree of accuracy. About 90% of the people developed spotty beta burns on exposed parts of their skin (not covered by clothing) with a large number (90% of the children and 30% of the adults) developing patchy epilation (see Figure 5). These lesions appeared about 12 to 14 days after exposure. No erythema was noted in the Rongelap people, possibly due to the darkness of their skins. Characteristically, lesions first appeared as dark pigmented macules, papules or patchy thickening of the skin followed by desquamation leaving depigmented areas. Vesiculation was only seen on the dorsa of the feet (see Figure 6). Most of the burns were superficial and had completely re-epithelialized and repigmented in a few weeks. However, fourteen people had deeper burns which took longer to heal with some scarring, atrophy and varying degrees of pigment aberration remaining (see Figure 7). Some of these changes still persist six years after exposure. However, no chronic radiation dermatitis or malignant changes have been noted. Regrowth of hair was noted beginning about three months after exposure and was complete and normal by six months after exposure.

Internal Hazard

Acute Exposure

Exposure to an acute fallout field is not likely to result in sufficient internal absorption of radioactive materials to produce an acute radiation syndrome. Indeed, an individual would receive a lethal dose of gamma radiation long before he could accumulate sufficient radioactive material internally for the dose to be lethal. Therefore, the internal hazard is secondary to both whole body penetrating exposure and beta radiation of the skin. The effect of chronic, long-term exposure to internal fallout is another story.

The internal absorption of fallout material occurs largely through ingestion with relatively small amounts via inhalation since the particle size of fallout is not likely to be small enough to get into the alveoli. That which gets into the bronchial tree is brought up by ciliary action and swallowed. Therefore, from an acute point of view, the gastrointestinal tract and the thyroid gland probably receive the largest dose of radiation of any organ system. As with irradiation of the skin, the principal hazard from internally absorbed fission products comes primarily from beta irradiation. The short-lived radionuclides, such as $I^{131-135}$, Sr^{89}, Ba^{140} and La^{140}, are important during the early period. Iodine is probably the most important isotope in the early period since it is selectively absorbed by the thyroid gland.

Initial Internal Absorption in the Marshallese. In the Marshallese who lived for 2 days under heavily contaminated conditions, urine analyses showed accumulation of isotopes to near maximum permissible levels for lifetime exposure (6, 23) in a few cases. Radioiodine produced an estimated 100 to 150 reps to the thyroid glands of the Rongelap people—but with no apparent effect. Damage to the gastrointestinal tract by passage through it of 3 mc. of fission product activity is not believed to have added materially to the symptomatology. The remarkable thing in the Marshallese was that, in spite of their heavy exposure to fallout without serious attempts at avoidance, urine analyses showed rapid dwindling of activity so that by one or two years radionuclides, including long-lived ones such as Sr^{90} and Cs^{137}, were hardly detectable (see Figure 10). This leads one to the conclusion that an acute exposure to fallout which is not lethal will probably not result in acute effects or long-term effects from internally deposited isotopes. Even so, simple precautions such as reasonable care in eating covered or canned foods or drinking protected water at such a time would greatly decrease the internal accumulation of isotopes.

Chronic Exposure Effects of Residual Contamination of the Environment on the Marshallese. Exposure to residual environmental contamination which may persist long after fallout occurs is exemplified by the situation at Rongelap Island. By 1957 complete surveys of the island showed such low levels of persisting contamination that the island was considered quite safe for habitation, and at that time the people were returned to Rongelap village which had been reconstructed for them. (Persistent background gamma radiation as of March 1959 averaged about 0.04 mr per hour which represents a dose of about 0.35 r per year.) The influence of this light contamination on the people's body burdens of radionuclides has been measured by radiochemical urine analyses and the very sensitive whole body gamma spectrographic techniques using a steel room to lower the background radiation level. Before their return to the island in 1957, gamma spectrography carried

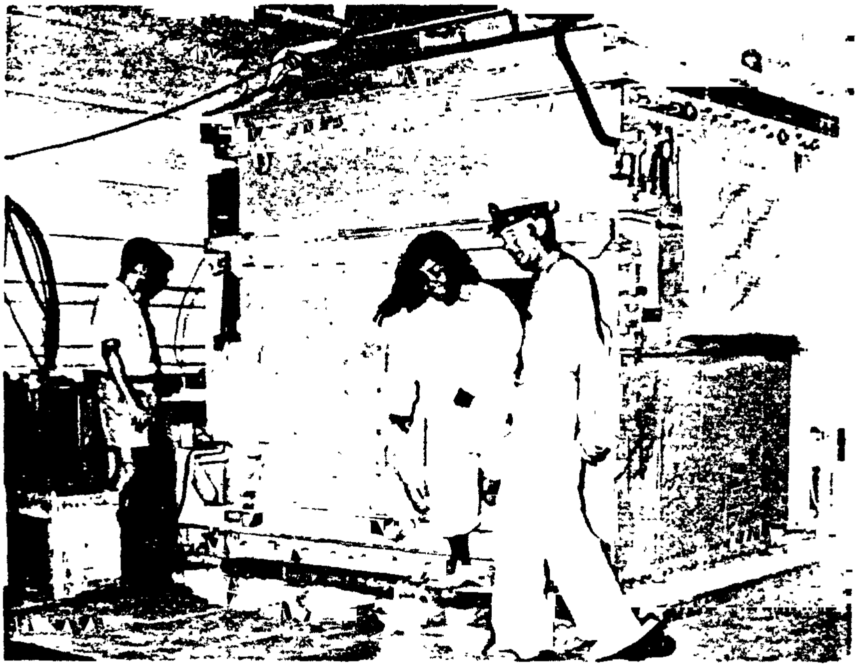

Figure 8. Steel room used for whole body gamma spectroscopy.

out at Argonne National Laboratory on four Rongelap people (24) showed the presence of Cs^{137} and Zn^{65}, the latter due to the eating of fish which selectively concentrate this isotope.

As a result of this study a steel room with 4 in. thick walls, weighing 21 T., was constructed at Brookhaven National Laboratory and transported to the Marshall Islands for use in subsequent surveys carrying out whole body gamma spectroscopy (see Figure 8). The increase in body burden of these isotopes after the aforementioned people had been living back on Rongelap one and one-half years can be seen in Figure 9. Cs^{137} values increased over one hundredfold. However, it appears from spectrographic analysis in 1959 that equilibrium of body level with environmental contamination for the two gamma emitting isotopes has been attained. The levels of 0.57 μc Cs^{137} and 0.44 μc Zn^{65} present in 1959 represent only a small percentage of the maximum permissible dose

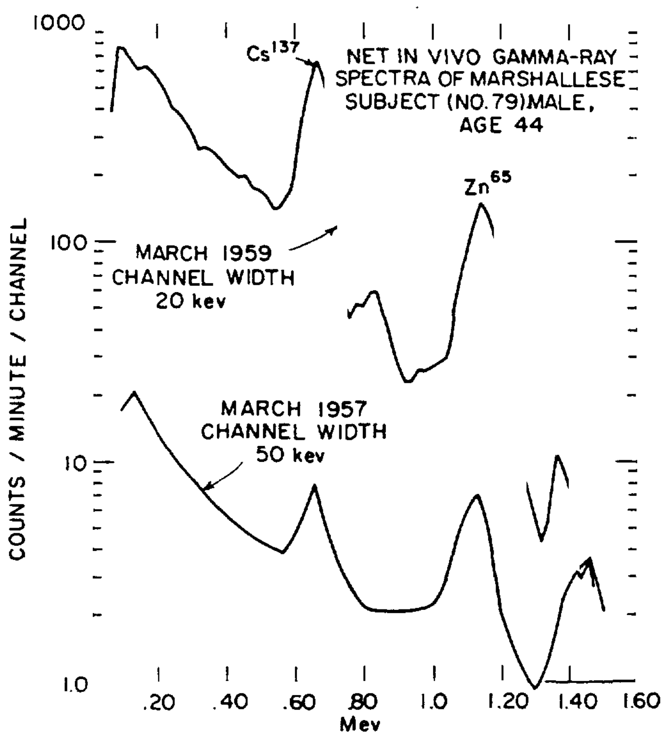

Figure 9. Gamma ray spectrograph of Rongelap man in March of 1957 and March of 1959 before and after his return to Rongelap.

(see Figure 10). Sr^{90} excretion rates increased by a factor of about twenty during the first year after the return of the Rongelap people. Analysis of bone samples of an exposed man who died after living on Rongelap about eight months showed 3.7 $\mu\mu c$ Sr^{90}/Gm. Ca. Further slight increase occurred during the following year as evidenced by urinary excretion increase of from mean values of 3.2 $\mu\mu c$/l. to 5.7 $\mu\mu c$/l. (Based on an estimated body burden of .002 μc Sr^{90} after a return of one year, the following year's value

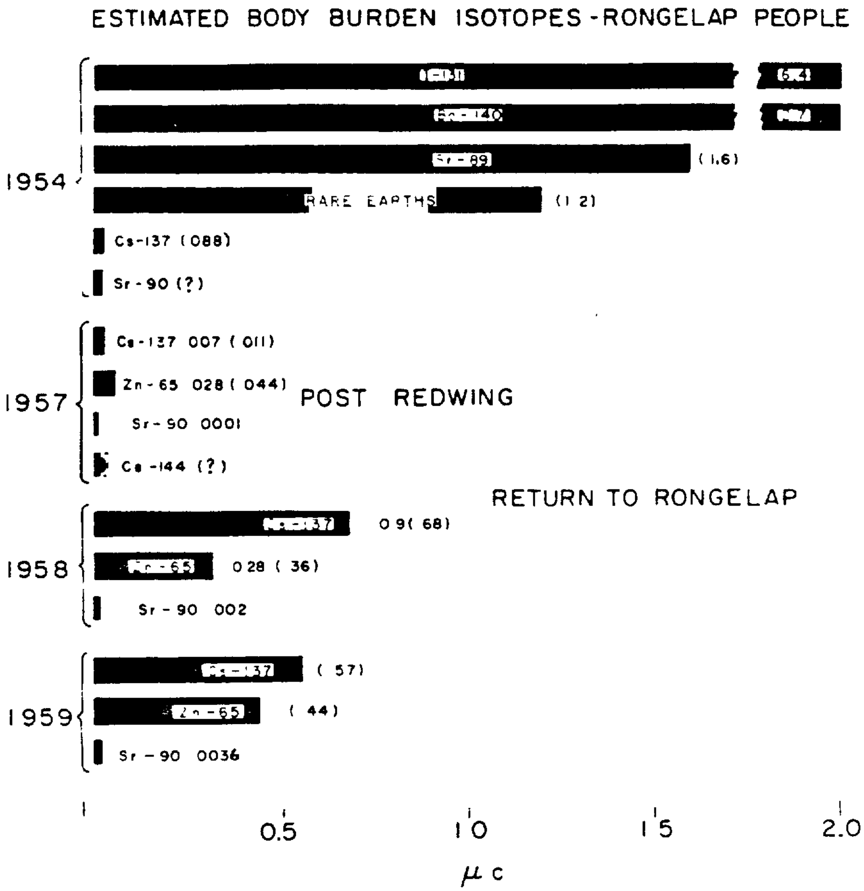

Figure 10. Estimated body burdens of Rongelap people since exposure to fall-out, based on radiochemical urine analyses and gamma spectrographic analyses.

would be about .0036 μc). Analyses for Sr^{90} *units* (Sr^{90}/Ca) for the 1959 urine samples are not complete.

The changes in isotope levels of marine, plant and animal life as related to the body burdens of the Marshallese is being investigated. Coconut crabs, considered a great delicacy by the people, have been found to selectively concentrate Sr^{90} up to levels of about five thousand units and consumption has therefore had to be prohibited. Coconut and pandanus show slight Cs and Sr activity but are considered safe to eat. No untoward effects have been observed in the Rongelap people as a result of living on their lightly contaminated island.

SUMMARY

Exposure to an acute fallout field results in three potential types of radiation hazards to man: penetrating whole body gamma radiation; superficial beta irradiation of the skin, mainly from contact with fallout material on the exposed skin and to a lesser extent from material on the ground; and internal absorption of radioactive materials largely through ingestion and to a lesser extent from inhalation. All these types of exposure occurred in the people of Rongelap Island in the Pacific as a result of accidental fallout on their atoll in 1954 following experimental detonation of a nuclear device.

The most serious hazard associated with acute fallout comes from penetrating gamma exposure which may result in acute radiation syndromes. In the Marshallese people, the gamma dose was sublethal but sufficient to result in (1) early nausea and vomiting and (2) moderate depression of blood elements. There were no clinical effects noted from the hemopoietic depression. There was questionable effect on (a) metabolism as suggested by temporary weight loss; (b) growth and development in children as suggested by slight lag in height and weight gain during the first two years (being re-evaluated on the basis of better data); (c) normal termination of pregnancy as represented by increased incidence of miscarriages and stillbirths in exposed women. No other late effects have been noted.

Beta burns may be moderately incapacitating but the hazard is not considered as serious as whole body gamma exposure. Simple prophylactic measures will greatly reduce this hazard. The Marshallese developed extensive beta burns, most of which were superficial and healed rapidly. In some cases, there were deeper burns with persisting changes (scarring, atrophy and pigment aberrations) but with no evidence of chronic radiation dermatitis or malignant change as yet.

The hazard from internal absorption is the least serious of the three, and it is not likely that sufficient material could be internally

absorbed to produce an acute radiation syndrome before an acute lethal gamma dose had been received. Ingestion of radioactive materials exposes the gastrointestinal tract and the thyroid gland to the largest radiation doses of any organ systems. Radioiodine probably is the most important radioelement present early in fallout in view of its selective concentration in the thyroid gland. The Marshallese people received near maximum permissible levels of some isotopes early, but there was rapid excretion so that by one or two years barely detectable amounts remained.

The return of the Rongelap people to their slightly contaminated island in 1957 has caused a measurable increase in body burdens of radionuclides. Cs^{137} and Zn^{65} increased by factors up to one hundredfold but, even so, present levels represent only a fraction of the maximum permissible levels and equilibrium appears to have been attained. Sr^{90} excretion increased twentyfold during the first year after the return of the Rongelap people, with a slight further increase noted the following year. Marine, plant and animal studies are part of the radiative ecological aspects of these studies.

REFERENCES

1. Conard, R. A., Meyer, L. M., Robertson, J. S., Sutow, W. W., Wolins, W., and Hechter, H.: Effects of fallout radiation on a human population, Rad. Res. Suppl., 1:280, 1959.
2. Conard, R. A., Meyer, L. M., Rall, J. E., Lowery, A., Bach, S. A., Cannon, B., Carter, E. L., Eicher, M., and Hechter, H.: March 1957 medical survey of the Rongelap and Utirik people three years after exposure to radioactive fallout. BNL 501 (T-119) :1 (Mar.) 1957.
3. Conard, R. A., Huggins, C. E., Cannon, B., Lowery, A., and Richards, J. B.: Medical survey of the Marshallese two years after exposure to fall-out radiation, J.A.M.A., 164:1192 (July 13) 1957.
4. Cronkite, E. P., Dunham, C. L., Griffin, D., McPherson, S. D., and Woodward, K. T.: Twelve month post-exposure survey on Marshallese exposed to fallout radiations, BNL 384 (T-71) (Aug.) 1955.
5. Bond, V. P., Conard, R. A., Robertson, J. S., and Weden, E. A.: Medical examination of Rongelap people six months after exposure to fallout, Operation Castle Addendum Report 4.1A, WT-937 (Apr.) 1955.
6. Cronkite, E. P., Bond, V. P., and Dunham, C. L.: Some effects of ionizing radiation on human beings, United States Government Printing Office, Washington, D. C., TID 5358.
7. Koyama, Y., et al.: Iryo (published by Iryo Dokokai, Medical Affairs Bureau, Ministry of Health and Welfare, Tokyo, Japan) 9:5, 1955.

8. Brues, A. M., and Sacher, G. A.: Analysis of mammalian radiation injury and lethality, Symposium on Radiobiology, New York, J. J. Nickson, John Wiley and Sons, 1952, pp. 441–465.
9. Blair, H. A.: A formulation of the injury, life span, and dose relations for ionizing radiations, University of Rochester A.E.C. Report, UR-206, 1952.
10. Dublin, L. I., and Spiegelman, M.: Mortality of medical specialists, 1938–1942, J.A.M.A., 137:1519 (Aug. 21) 1948.
11. Warren, S.: Longevity and causes of death from irradiation in physicians, J.A.M.A., 162:464 (Sept. 29) 1956.
12. Alexander, P.: Accelerated ageing; a long term effect of exposure to ionizing radiations, Gerontologia, 1:174, 1957.
13. Comfort, A.: Natural ageing and the effects of radiation, Rad. Res. Suppl., 1:216, 1959.
14. March, H. C.: Leukemia in radiologists, J.A.M.A., 135:179 (Sept. 20) 1947.
15. Moloney, W. C., and Kastenbaum, M. A.: Leukemogenic effects of ionizing radiation on atomic bomb survivors in Hiroshima City, Science, 121:308 (Feb. 25) 1955.
16. Simpson, C. L. and Hempelmann, L. H.: Association of tumors and roentgen-ray treatment of the thorax in infancy, Cancer, 10:42 (Jan.–Feb.) 1957.
17. Sinskey, R. M.: The status of lenticular opacities caused by atomic radiation, Am. J. Ophth., 39:285 (Mar.) 1955.
18. Brown, F. T. and Osgood, A. T.: X-rays and sterility, Am. J. Surg., 18:179, 1905.
19. Casarret, G. W.: The effects of ionizing radiations from external sources on gametogenesis and fertility in mammals. A review. University of Rochester A.E.C. Report, UR-441, 1956.
20. Greulich, W. W., Crismon, C. S., and Turner, M. L.: The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, J. Pediat., 43:121 (Aug.) 1953.
21. Reynolds, E. L.: The growth and development program of the Atomic Bomb Casualty Commission. Analysis of observations on maturation, body build and posture taken in 1951 on 1800 Hiroshima children. NYO-4459, 1952.
22. Neel, J. V., and Schull, W. J.: The effect of exposure to the atom bomb on pregnancy termination in Hiroshima and Nagasaki, National Academy of Sciences, National Research Council Pub. No. 461, 1956.
23. National Bureau of Standards Handbook 69, United States Government Printing Office, Washington, D. C., (June) 1959.
24. Miller, C. E., and Steingraber, D. J.: Measurements on some residents of the Marshall Islands, ANL-5755:53, 1957.