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MEDICAL SURVEY OF RONGELAP PEOPLE FIVE AND SIX YEARS AFTER EXPOSURE TO FALLOUT (With an Addendum on Vegetation)

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September 1960

BROOKHAVEN NATIONAL LABORATORY
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Addendum

A NOTE ON THE VEGETATION OF THE NORTHERN ISLETS OF RONGELAP ATOLL, MARSHALL ISLANDS, MARCH 1959

B.S. BLUMBERG* AND R.A. CONARD

Fosberg[1,2] reported changes in the vegetation of the northern islets of Rongelap Atoll (observed in 1956) which he inferred might have been associated with the radioactive fallout that occurred on this atoll in 1954. During the medical survey of the Rongelap people[3] carried out in March 1959, an opportunity arose to visit some of these islets and to re-examine the vegetation. A helicopter was available for transportation, which permitted general and detailed air examination as well as two short ground surveys. The northern islets were estimated to have received a radiation dose of $\approx$3000 r. The islets of Naen and Gegen were examined in greatest detail. The most striking feature observed from the air was the generally gray color of much of the vegetation, in contrast to its

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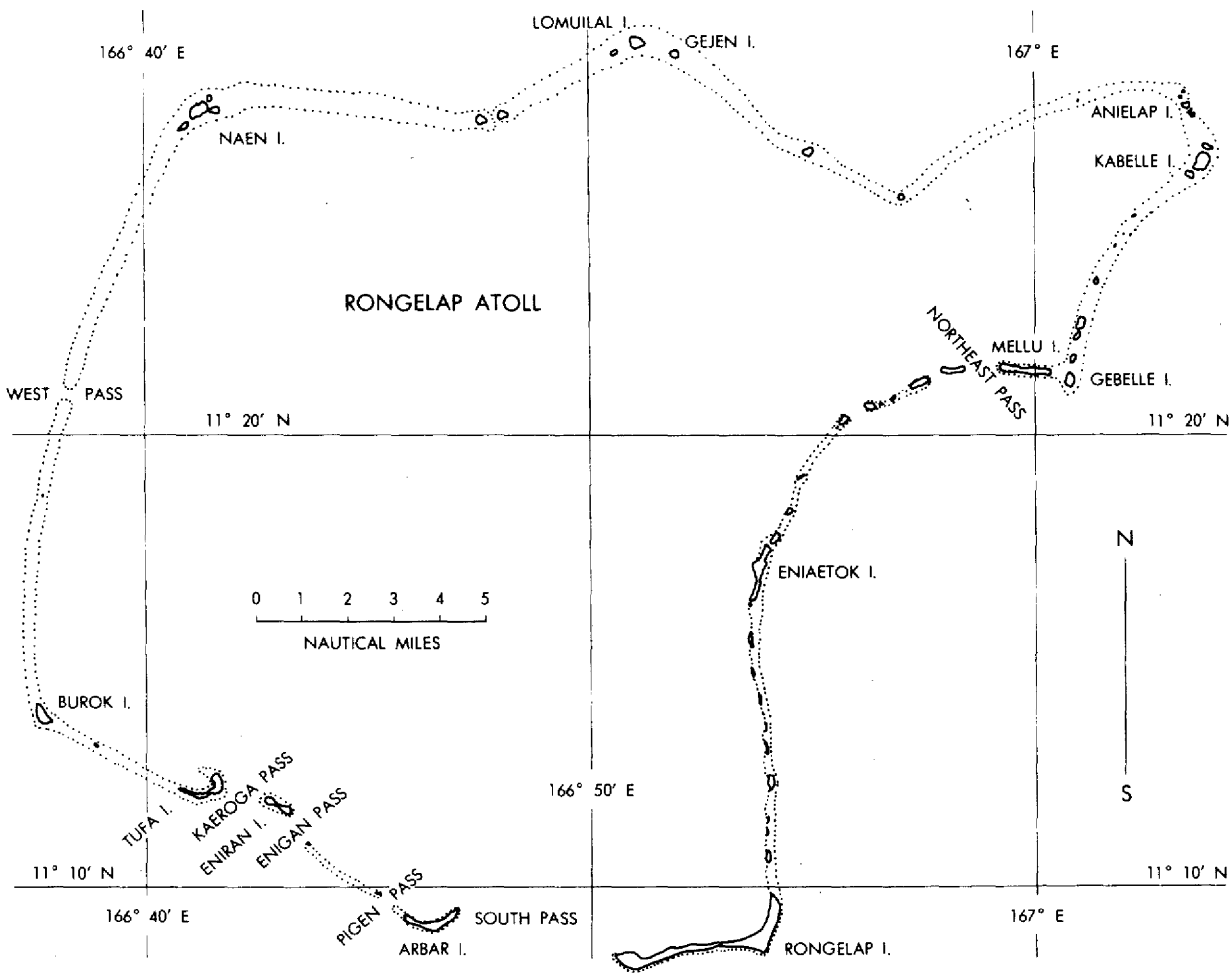

Figure A-1. Map of Rongelap Atoll showing position of major islets.

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Figure A-2. Affected *Guettarda speciosa* with normal appearing *Scaevola sericea*.

normal green color. Ground surveys revealed that *Scaevola sericea* was common and normal in appearance. Many of the *Guettarda speciosa* appeared to be in poor condition (Figure A-2). In some, all or nearly all the leaves were gone from the terminal 1 to 12 in. of the branches, and other leaves were yellowed and shriveled. In other *Guettarda*, nearly all the leaves were gone, and the bushes appeared completely dead. More than 50% of the *Guettarda* were affected in whole or part. In one area of Naen several hundred yards inland from the ocean beach, there was a field of ≃30 *Guettarda*, all of which were dead. Some young *Pisonia grandis* were seen which appeared to be in good condition. Mature *Pisonia* were seen which were partially defoliated, but these did not appear to be greatly different from those seen on Rongelap Islet on the southeast corner of Rongelap Atoll. None of the mistletoe-like clumps described by Fosberg were observed. Several *Ochrosia oppositifolia* were seen with nearly complete defoliation, which appeared dead. A small grove of coconut trees near the center of Naen Islet contained 4 to 5 dead trees within a radius of ≃300 yards, which were decapitated at heights 5 to 12 ft above the ground with no evidence of axe or machete marks. Two 2-headed coconut trees were seen, one with fronds that were mostly brown and appeared dead growing from the trunk ≃2 ft below the true crown of the tree. Several trees had dry and shriveled fronds, and ≃6 had deformed bulges 4 to 8 ft below the crown with apparently normal growth above the bulges.

Photographs of the affected vegetation were examined by Dr. Fosberg, and he stated that the changes were similar to those he had previously reported.

It is not possible to evaluate the cause of the changes from the present observations. More extensive and detailed botanical and ecological surveys will be necessary, both on the islands that received radiation and on those that did not, to determine whether the changes seen bear any relation to fallout. In particular, it should be noted that these observations were made during the dry season.

We are indebted to Professor Frank Richardson of the University of Washington for identifying the plants, and to Commander W. Lyons, USN, for his assistance in taking the photographs.

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CONTENTS

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MEDICAL SURVEY OF RONGELAP PEOPLE FIVE AND SIX YEARS AFTER EXPOSURE TO FALLOUT

Introduction

The results of medical surveys of the people of Rongelap in the Marshall Islands, carried out in March 1959 and in March 1960 at 5 and 6 years after the accident, are presented in this report. These people had been accidentally exposed to fallout radiation following a detonation of a high yield thermonuclear device during experiments at Bikini in the Pacific Proving Grounds in March 1954. An unpredicted shift in winds caused a deposition of significant amounts of fallout on four inhabited Marshall Islands to the east of Bikini (see Figure 1) and also on 23 Japanese fishermen aboard their fishing vessel, the *Lucky Dragon*. Of the inhabitants of the island of Rongelap, 105 nautical miles away from the detonation, 64 received the largest fallout exposure: an estimated dose of 175 r of whole-body gamma radiation, contamination of the skin sufficient to result in beta burns, and slight internal absorption of radioactive materials through inhalation and ingestion. Another 18 Rongelap people away on a nearby island (Ailingnae), where less fallout occurred, received only an external gamma dose of about 69 r. There were 28 American servicemen on the island of Rongerik further to the east who received about the same amount of radiation as did the Rongelap people on Ailingnae. Lastly, 157 Marshallese on Utirik Island, about 200 miles further east, received about an estimated 14 r of whole-body radiation. The fallout was not visible on this island and no skin effects developed.

The exposed people were evacuated from these islands by plane and ship about two days after the accident and taken to Kwajalein Naval Base about 150 miles to the south, where they received extensive examinations for the following three months. In view of the generally negative findings on the American servicemen, they were later returned to their duty stations. The Utirik people were also allowed to return to their home island, where radioactive contamination was considered to be of a slight enough extent to allow safe habitation. Because Rongelap Atoll was considered to be too highly contaminated, a temporary village was constructed for the Rongelap people on Majuro Atoll several hundred miles to the south, where they lived for the following 3½ years and were examined at yearly intervals by a special medical team. In July 1957, after careful evaluation of the radioactive contamination situation, Rongelap Island was considered safe for habitation. A new village was constructed, and the Rongelap people were moved there by Navy ship. The annual medical surveys have since been carried out on Rongelap Island.

A group of more than 100 Rongelap people, who were relatives of the exposed people but had been away from the island at the time of the accident, moved back with the Rongelap people to their home island and have served as an ideal comparison population for the studies. Following the initial survey of the Utirik people on Kwajalein in 1954, a repeat survey was carried out in March 1957. In addition, during the past survey, as in the previous surveys, a visit was made to Majuro Atoll to examine a group of children who represent part of the control group used for the growth and development studies of the exposed children.

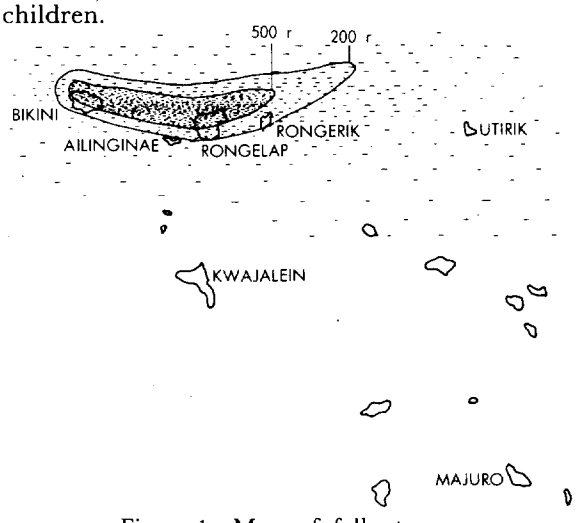

Figure 1. Map of fallout area (March 1, 1954), Marshall Islands.

The accumulation of data from these surveys is becoming increasingly voluminous. Since conditions have not been favorable for performance of extensive statistical analyses or use of electronic computing procedures to store and manipulate the data, the annual survey reports published by this Laboratory are made as complete as possible. This report, therefore, includes a considerable amount of raw data, much of it in appendices, so that others may have an opportunity to make further calculations if desired.

This report also contains a brief addendum on the vegetation of Rongelap Island.

Summary of Past Findings

Reports have been published on the findings of surveys made at the following times after exposure: initial examination,[1] 6 months,[2] 1 year,[3] 2 years,[4] 3 years,[5] and 4 years.[6] The following is a brief summary of the findings previously reported.

During the first 24 to 48 hr after exposure, about ⅔ of the Rongelap people experienced anorexia and nausea. A few vomited and had diarrhea. Many also experienced itching and burning of the skin, and a few complained of lachrymation and burning of the eyes. Following this, the people remained asymptomatic until about 2 weeks after the accident, when cutaneous lesions and loss of hair developed, due largely to beta irradiation of the skin. It was apparent when the people were first examined, a few days after exposure, that the lymphocytes were considerably depressed and that significant doses of radiation had probably been received. In addition to the whole-body dose of radiation and the beta irradiation of the skin, radiochemical analyses of the urine showed that measurable amounts of radioactive material had also been absorbed internally. The effects of the radiation can best be summarized under three headings according to the mode of exposure: penetrating irradiation, skin irradiation, and internal irradiation.

PENETRATING IRRADIATION

The changes in the peripheral blood of the more heavily exposed Rongelap people who received 175 r will be reviewed in the section on hematological examinations (see Figures 33, 35, and 38, and Appendices 1 and 2). The changes in the Ailingnae and Utirik groups were similar but less marked. Certain unexplained fluctuations have occurred from year to year in the peripheral blood levels of the comparison populations as well as of the exposed groups. Depression of the peripheral blood elements as represented by mean population levels occurred as follows.

Lymphocytes fell promptly and by the third day were about 55% of the control values in adults, and slightly lower in children. There was only slight recovery after six months. At 2 years, although further recovery was evident, the mean values of these cells were still found to be below the comparison population levels (75 to 80%). At 3 years the mean lymphocyte counts were slightly below those of the comparison population. At 4 years the mean level appeared to be about the same as that of the comparison population, but many counts remained lower.

Neutrophil levels fluctuated considerably during the first few weeks but fell gradually to a low of about 50% of comparison population levels by the 6th week after exposure. Slow recovery ensued, but at 6 months they were still slightly below the unexposed levels. However, by 1 year post exposure they had returned to the level of the comparison population and have remained so, with the possible exception of those of children <12 years old, which generally have been lower than those of the unexposed children of comparable age.

Platelets fell to about 30% of the unexposed values by the 4th week. By 6 months they had reached 70% of the controls; at 1 year the mean platelet count was still below that of the control population but higher than at the 6-month survey. Although further increases were apparent at the 2-, 3-, and 4-year examinations, the levels were still below those of the comparison population.

Changes in hematocrit were not remarkable in any of the groups.

Clinical examinations revealed no disease processes or symptoms which could be attributed to radiation effects, aside from skin lesions, loss of hair, and early symptoms. Epidemics of chicken pox and measles occurred. The diseases encountered were no more severe or frequent in the irradiated group than in the unexposed group, even during the period of greatest depression of peripheral blood elements. Three persons in the exposed population died of disease: (1) a 46-year-old man with a hypertensive heart disease which had been

present at the time of exposure, who died 2 years after the accident; (2) a 78-year-old man who died, 3 years after exposure, of coronary heart disease complicating diabetes; and (3) a 36-year-old man who died of acute varicella, 4 years after exposure, who had received only 69 r, having been on Ailingnae at the time of the fallout. There was no apparent relationship between any of these deaths and radiation exposure, and mortality in the exposed group did not appear to be greater than in the unexposed population.

It was difficult to evaluate the effects on fertility. However, a number of apparently normal babies were born during the 4-year period, and there has been no discernible fall in birth rate. A slightly higher number of miscarriages occurred in the exposed women than in the unexposed group; this will be further evaluated in the present report. No opacities of the lens or other eye changes have been found that could be related to radiation. Studies on height, weight, and bone age seemed to show a slight degree of retardation in growth and development in the exposed children. However, the small number of children involved, and a later finding that exact ages of some of the children were in doubt, has resulted in a re-evaluation of these data based on more reliable age determinations.

BETA IRRADIATION OF THE SKIN

It was impossible to get an accurate estimate of the radiation dose to the skin. Beta burns of the skin and epilation appeared about 2 weeks after exposure, largely on parts of the body not covered by clothing, About 90% of the people had these burns, and a smaller number developed spotty epilation of the scalp. Most of the lesions were superficial; they exhibited pigmentation and dry, scaly desquamation, and were associated with little pain. Rapid healing and repigmentation followed. Some lesions were deeper, showed wet desquamation, and were more painful. A few burns became secondarily infected and had to be treated with antibiotics. Repigmentation of the lesions gradually took place in most instances, and the skin appeared normal within a few weeks. However, in about 15% of the people, deeper lesions, particularly noted on the dorsum of the feet, continued to show lack of repigmentation with varying degrees of scarring and atrophy of the skin. At 4 years the only residual effects of beta radiation of the skin were seen in 12 cases which showed varying degrees of pigment aberrations, scarring, and atrophy at the site of the former burns. Numerous histopathological studies have been made,[1,4,5] and the changes found have been consistent with radiation damage. At no time have changes been observed either grossly or microscopically indicative of malignant or premalignant change. Spotty epilation on the heads was short lived, regrowth of hair occurring about 3 months after exposure and complete regrowth of normal hair by six months. No further evidence of epilation has been seen.

An interesting observation was the appearance of a bluish-brown pigmentation of the semilunar areas of the fingernails and toenails in about 90% of the people, beginning about 3 weeks after exposure. By 6 months, this pigmentation had largely grown out with the nail and had disappeared in most cases. The cause of this phenomenon has not been explained.

INTERNAL IRRADIATION

Radiochemical analyses of numerous urine samples of the exposed population showed internal absorption of radioactive materials, probably brought about largely through eating and drinking contaminated food and water and to a lesser extent through inhalation. During the first few days when the body levels were at their highest, the maximum permissible concentrations were approached or slightly exceeded only in the case of strontium-89 and the isotopes of iodine. The concentrations were believed to be too low to result in any serious effects. Body levels fell rapidly, so that by 2 and 3 years post exposure, they were far below the accepted maximum permissible level; by 6 months activity in the urine was barely detectable. The return of the Rongelapese to their home island (which after careful survey was considered safe for habitation, despite a persisting low level of radioactive contamination) was reflected in a rise in their body burdens and increased urinary excretion of certain radionuclides. Beginning in 1957, gamma spectroscopy by use of a low-level counting chamber was added to the techniques of radiochemical analysis. This, along with radiochemical analysis of urine samples, showed an increase in body burdens of cesium-137 by factors of up to 100 and of strontium-90 by a factor of 10 to 20; and some increase also in zinc-65, since the re-

turn of the people to Rongelap. The body levels of the unexposed people who returned to the island also increased, becoming indistinguishable from those of the originally exposed group. However, the levels remain well below the maximum permissible limits. Analyses of bone samples on one of the men who died showed 3.7 strontium-90 units/g calcium.

OTHER STUDIES

Other studies have been carried out on the Rongelap people which are not directly related to radiation effects. An intensive intestinal parasite survey showed that the people were infected with many types of protozoa and helminths. This finding did not entirely account for the generally high incidence of eosinophilia. Other findings that need further explanation are the general anemic tendencies, the high plasma protein levels with increased gamma globulin, and the higher than normal levels of serum protein-bound iodine and vitamin B_{12}. Another investigation is concerned with genetic studies and determination of the anthropological background of the Marshallese from genetically determined traits. Among the traits studied were blood groups and hemoglobin and haptoglobin types. The results shed some light on the origin of these people and the homogeniety of the population. Their blood groups resemble most closely those of the people from Southeast Asia and Indonesia, and the population appears to be relatively homogeneous.

DIFFICULTIES ASSOCIATED WITH THE EXAMINATIONS

As mentioned in previous reports, several difficulties were associated with carrying out the examinations as well as interpreting the findings.

1. The language barrier made examinations difficult, since very little English is spoken by the Marshallese. However, there were sufficient English-speaking Marshallese to assist the medical team in most instances.

2. The lack of vital statistics or demographic data on the Marshallese imposed a serious difficulty in interpretation and evaluation of the medical data. Records of births, deaths, etc., have been made by the health aides or magistrates of the villages and supposedly forwarded to the district administrator; however, such records have been incomplete or lost in most instances, and vital statistics are therefore inadequate. Trust Territory officials are now attempting to assemble such data.

3. There is uncertainty on the part of some of the Marshallese as to their exact ages, particularly among the older group. This imposes certain difficulties in interpreting some of the studies to be outlined.

COMPARISON POPULATIONS

During the first 2 years, two separate groups of Marshallese people were used for comparison, each of comparable size to the exposed Rongelap group and matched for age and sex. However, this population was found to be unstable, with a large attrition rate over the 2 years, which made it unsatisfactory. At the time of the 3-year survey, it was found that during the preceding 12 months the Rongelap population at Majuro Atoll had doubled because of the influx of relatives who had come back from other islands to live with them. These people had been away from Rongelap Atoll at the time of the accidental exposure. This group matched reasonably well for age and sex and was of comparable size. Since the return of the people to Rongelap, however, this group has about doubled in size.

Since the people are of the same stock genetically, they are uniquely appropriate to serve as a comparison population and have, therefore, been used since 1957.

1959 Survey

BACKGROUND MATERIAL

Organization

For the 1959 survey, 5 years after the accident, the medical team consisted of seven physicians, one dentist, two scientific specialists, and nine technicians from various institutions and laboratories in the United States. Several Marshallese practitioners and interpreters assisted in carrying out the examinations.

A group of five scientists from the University of Washington, headed by Dr. E.E. Held, accompanied the group to collect soil, marine, and plant samples for radiochemical analysis. These studies are not included in this report.

Prior to the main survey on Rongelap, several members of the team visited Majuro to examine

Figure 2. Village street scene, Rongelap Island.

Figure 3. Group of Rongelap children.

15 children who are part of the control series for the growth and development studies. Several Rongelap people of the exposed group and the comparison population group who were living in Majuro were also examined.

The Navy kindly furnished a ship, the LST *USS Duval County*. The team met at Eniwetok where the ship had put in to onload the 21-ton steel room which had been stored there and also the medical equipment and supplies. The medical team was berthed and fed aboard the ship for the expedition. The ship was beached at Rongelap and Utirik for easy accessibility to the villages.

Attitudes of Rongelap People

When the team arrived at Rongelap, the magistrate of the village indicated that there was some confusion and uncertainty in the minds of some of the people as to the necessity and significance of repeated medical examinations. He thought it wise to call a meeting of the village people in the council house so that they could ask questions to help clarify the situation. For the past 5 years during which the annual examinations have been going on there had been no problem in maintaining excellent rapport with the people, and, indeed, the relations of the team members with the Rongelapese were always cordial and friendly. It was recognized that there was slightly increasing resistance to blood sampling procedures. Also there was some discontent that, because of the high Sr^{90} content, they were forbidden to eat coconut crabs, which they consider a delicacy (Figure 4). Since the return of the people to Rongelap, copra production had not increased to the extent that the Trust Territory officials had hoped. Consequently, since copra production is the prime source of income, there was some concern over the slowness with which the people were getting back on their feet economically. It had become necessary to extend food subsistence beyond the time originally planned. Fishing was not being carried on as actively as it should have been.

At the village meeting the main questions centered around the necessity for the continued medical examinations in view of statements on the part of the medical team in the past that the people were generally in good health. It was difficult to explain to them that, though they appeared to be in good health and to have recovered from the acute effects of radiation, very little was known about the possible late effects of radiation, and continued examinations were essential in order to detect and treat any untoward effects, should they arise. The coconut crab problem was brought up again, and the reasons for prohibiting their consumption carefully explained through the interpreter. To correct a misconception that several cases of fish poisoning during the past year had been due to eating radioactive fish, it was explained that fish poisoning had been going on in these islands for years and was not connected with radioactivity. After much discussion, it seemed that the people were satisfied with answers to the questions, and preparations for the examinations proceeded. Thereafter complete cooperation and the usual friendly relations prevailed throughout the stay on the island.

During the examinations a United Nations team visited Rongelap. A meeting with the people was held in the church (Figure 5), and many aspects of the Rongelap situation were discussed. The report of the UN group was favorable toward the special medical assistance being rendered the people.

Upon completion of the 1959 survey, a meeting was held for the people, and they were advised that they were found to be generally in good health with no serious effects of their radiation exposure apparent, but that continued examinations would be necessary in order to insure continued good health. They were also advised to try to improve their oral hygiene and observe sanitary rules to control the flies on the island.

Before the team left the island, a party was held for the Rongelapese. The Navy kindly furnished a meal, and small gifts were exchanged as tokens of appreciation of mutal cooperation.

Figure 4. Coconut crab (robber crab, *Birgus latro*), considered a delicacy by the Marshallese. (Photo courtesy American Museum of Natural History, New York, N.Y.)

Figure 5. United Nations visiting group meeting with Rongelap residents in village church, 1959.

PROCEDURES

Location of Examinations

LST Tank Deck. Whole-body gamma spectroscopy was carried out with the 21-ton steel room placed in the after part of the tank deck, with the air conditioned electronics room and the shower facility close by. The people to be examined entered the ship via the forward ramp directly onto the tank deck, proceeded to the shower facility where they disrobed, took a complete soap and water shower, and donned disposable paper coveralls and slippers, and then proceeded to the 21-ton steel room, where a 10-min gamma spectroscopy count was obtained. Phonograph music was piped into the steel room during the procedure. The people, except very young children, showed no evidence of fear or claustrophobia and appeared to enjoy the procedure. Further description of the gamma spectrographic methods will be presented later.

The diagnostic x-ray machine was also set up in the tank deck.

Rongelap Village. As in 1958, the dispensary, schoolhouse, and council house were used for the examinations (Figure 6). Hematological and other laboratory procedures and ophthalmological examinations were carried out in the dispensary (Figure 7). Medical histories were taken and physical examinations were made in the schoolhouse. The council house provided areas for an administrative center, for collection of urine samples, and for laboratory use by the University of Washington group.

History and Physical Examinations

Histories were taken by a Marshallese practitioner with particular emphasis on the interval history during the past year. A special survey was conducted by the pediatrician to ascertain more accurately the birth dates of the Rongelapese, particularly the children.

Complete physical examinations were carried out including examinations of the children for growth and development (anthropometric measurements, and x-ray examinations of the left wrist

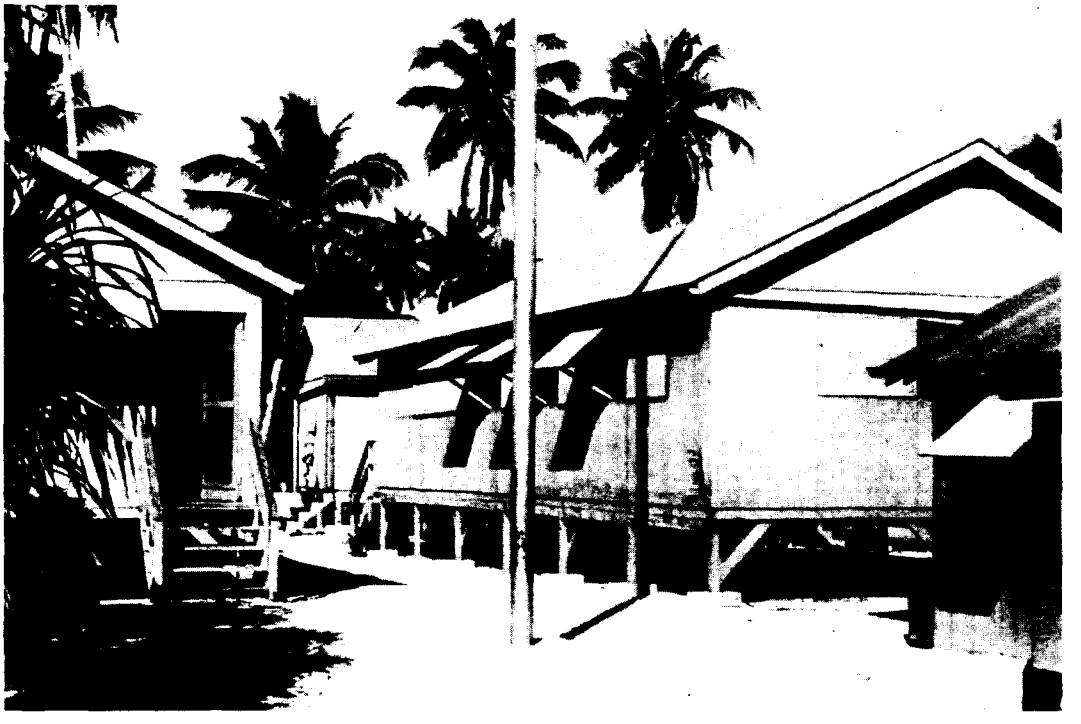

Figure 6. Dispensary and examination buildings, Rongelap Island.

Figure 7. Hematology laboratory, Rongelap Island.

and hand for bone development studies); studies of aging criteria; special examinations of the skin with color photography of selected lesions; ophthalmological studies including slit-lamp observations, visual acuity, and accommodation; audiometric examinations; cardiovascular survey; and x-ray examinations as deemed necessary.

In 1959, 96 exposed people, including their children, and 166 unexposed people were examined on Rongelap.

Cardiovascular Survey. In view of the possible association of late effects of radiation with cardiovascular disease, the following cardiovascular survey was carried out.

1. *Clinical histories* recorded present and past health and illnesses, especially cadiac and pulmonary symptoms.

2. *Physical examinations* included the usual observations in the cardiovascular system in regard to the rhythm and rate of the heart; the nature of the heart sounds and murmurs, if present; the size and shape of the heart by clinical indications; changes in the arterial walls as observed by inspection of the retinal arteries and by palpation of the radial, brachial, and dorsalis pedis arteries; and the appearance of the veins.

3. *Instrumental procedures: Systolic* and *diastolic blood pressures* were obtained with the cuff-type aeronoid sphygmomanometer. Oscillometry readings were obtained from both legs at calf level by the Collins type of oscillometer. The highest of the readings obtained at different pressure levels was used as a single reading for each subject. *Electrocardiograms* were taken on people 20 years of age and over (84 unexposed and 38 exposed individuals). The electrocardiograms were taken by a Sanborn Direct Writing apparatus which had been specially shock mounted for field use.* In almost all subjects the electrocardiograms were taken with 12 leads, to include three standard bipolar limb leads, three augmented unipolar limb leads, and six unipolar or V chest leads in accord with the standard 12-lead procedure recommended by the American Heart Association. The technical quality of the electrocardiogram was in most instances relatively good, but there were two handicaps. The electric power for the instruments was obtained from a generator on the LST by running a cable ashore to outlets in the various buildings used for the medical examinations. Induction currents often caused interference, and grounding of the instruments at times was difficult. There were also technical variations because of variable line voltage and amperage. *X-ray films* of the chest were taken on selected subjects when indicated. The number taken was kept to a minimum to avoid additional radiation of the exposed people, even though the dose involved was extremely small. The films were used chiefly for the study of the lungs and, to some extent, for measurement of heart size and shape. Chest films were obtained on 11 subjects in the unexposed group and 7 in the exposed group.

It should be pointed out that there was no standardized basal or adjusted level of physical activity, such as resting for one hour before taking the tests. The patients were seen in the course of the usual examination, as might occur in the outpatient department of a hospital.

Arthritis Survey. During the course of other studies on the Rongelap population, it was possible to obtain information on the prevalence of arthritis and allied phenomena. Since little is known of the prevalence of these diseases in tropical populations, any such data would be a contribution to the "geographic pathology" of the arthritides[7] besides being of interest in connection with the possible adverse influence of radiation exposure on degenerative diseases.

X-rays of the hands and wrists were graded for osteoarthritis on a scale of 1+ to 4+ by the method of Kellgren and Laurence[8] as modified by Blumberg et al.[9] The same x-rays were used to detect any cases of rheumatoid arthritis, other arthritides, or bony abnormalities.* A small aliquot of serum (0.3 to 0.5 ml) was used to determine the presence of "rheumatoid factor" by the bentonite flocculation test of Bozicevich et al.[10] In this test, a titer of 1/32 or greater is considered positive, 1/16 doubtful, and any titer below 1/16 negative.* A joint examination on each of the Rongelapese was performed by the medical examiners, and any individual with significant joint findings was re-examined by one of the physicians experienced in the diagnosis of joint disease. Treatment of the joint disease was undertaken where indicated.

*We are most grateful to the Medical Equipment Research Department, U.S. Army, Fort Totten, N.Y., for use of this instrument on a permanent loan basis.

*We are indebted to the following personnel of the National Institutes of Health: Drs. R.L. Black, J.J. Bunim, and E.G.L. Bywaters for reviewing the x-rays; and Dr. K. Bloch for performing the bentonite flocculation test.

Dental Survey. Intra-oral examinations were conducted as part of the physical examination. All patients were examined with mouth mirror and explorer. Illumination was by standard operating light. Salivary *p*H was taken by use of *p*Hydrion on all subjects who were not eating candy or any other foodstuff. Saliva samples were collected from these patients at the time *p*H determinations were made. Intra-oral radiograms were not available. Dental examinations were carried out on the following groups: 30 children at Majuro; all the adults and children at Rongelap who were in the physical examination group; and a number of children at Utirik.

Studies of Aging Criteria

Although the exposed group has shown no outward evidence visible by gross observation of any accelerated aging effect of radiation, it was thought desirable to have available measurable criteria of possible age changes for use during normal physical examination. Therefore the over-all objective of this study was to establish tests to evaluate any possible radiation-induced senescence in the exposed group compared with the unexposed population living on the same island. However, the data (presented in a later section on results) collected during the 1959 survey (5 years after exposure) showed no apparent differences between the exposed and unexposed groups. Therefore the data from the two groups have been pooled with the objectives of presenting the methodology, indicating the trend of changes with age of the various aging criteria chosen, and attempting to determine a biological age score for individuals and different age groups.

This study was hampered by the small number of people involved and the lack of vital statistics on the Marshallese people. Another difficulty has been the uncertainty of exact ages in some cases, particularly in older people.

Data on aging criteria were recorded only on adults 20 years of age and over. Of the 126 adults, 42 were in the originally exposed group and 84 in the larger comparison population. Table 1 shows the age and sex distribution. The ages were reasonably well distributed except for a smaller number of older people (>60 years of age).

The age criteria chosen were based on changes generally believed to be associated with physiological senescence and represent only a small number of the possible ones. They were selected with a view toward ease of assessment during routine physical examination under field conditions, time limitations, and language barrier. Therefore, unfortunately, tests of vigor and functional capacity were necessarily limited.*

Table 1

Age Distribution in Rongelap Adult Population, 1959, Used in Aging Study

	Exposed		Unexposed		
Age, yr	M	F	M	F	Total
20–24	0	5	1	6	12
25–29	2	1	12	6	21
30–34	3	2	3	5	13
35–39	1	4	4	4	13
40–44	4	2	6	3	15
45–49	1	0	2	3	6
50–54	1	0	5	5	11
55–59	2	1	3	1	7
60–64	0	6	5	3	14
65–69	0	0	2	3	5
70–74	1	2	0	0	3
75–79	0	1	0	0	1
>80	2	1	2	0	5
					126

Of the 15 criteria selected, 9 were measured directly and 6 were estimated on a 0 through 4+ scale. Five tests involved the integument: (1) skin looseness, (2) skin elasticity (retraction time), (3) senile changes in the skin, (4) greying of the hair, and (5) baldness. Four tests involved the special sense organs: (1) accommodation, (2) visual acuity, (3) arcus senilis, and (4) hearing. The cardiovascular system was tested by (1) systolic and (2) diastolic blood pressure recordings, (3) peripheral arteriosclerosis, and (4) retinal arteriosclerosis. There was one test of neuromuscular function. Vigor was measured by hand strength measurements. The test methods are described below.

Integument. A special skin caliper was designed for measuring skin looseness and elasticity (Figure 8). The legs of the caliper can be opened to any desired degree up to 5 cm on scale *A* by adjusting screw *B*. The spring tension when the caliper is closed on a fold of skin results in a pressure of about 500 g. The inner and outer surfaces of the legs are calibrated in millimeter markings (*C*).

*Several tests, such as pulse and blood pressure response to a two-step test and vital capacity, were tried but not used because they proved unsatisfactory.

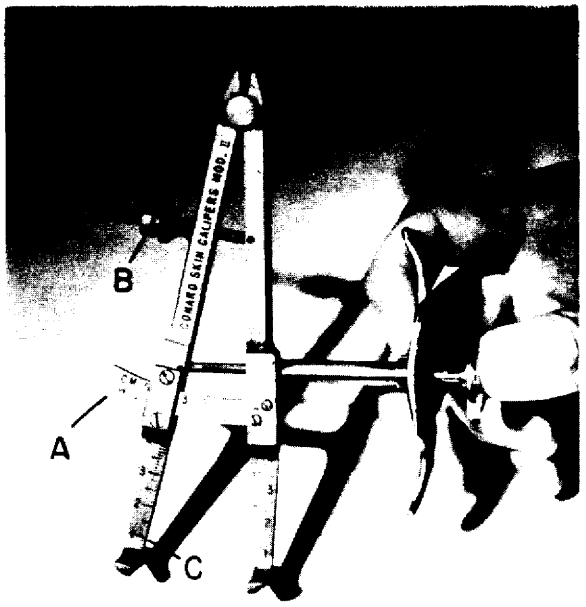

Figure 8. Skin calipers used in measuring looseness and elasticity of the skin.

1. *Skin looseness.** Preliminary investigation revealed that the skin fold at the junction of the chin and neck was the most satisfactory for the measurement of skin looseness and also seemed to give the best correlation with age. The measurement was carried out by grasping the skin with the thumb and forefinger, pulling it gently outward, and applying the caliper opened to 4 cm so that each leg was firmly against the skin lateral to the fingers. The caliper was then allowed to close by its own spring tension, and the height of the fold of skin impinged was measured in mm on scale *C*. The elongated ends of the legs impinge on a 1-cm^2 surface of skin and exert a pressure of 500 g. There is little variation in spring tension in the last 2 cm closing range of the caliper. The height of the fold represented the degree of skin looseness. The presence of excess fat in the skin probably caused the readings to err on the low side, but this was not thought to result in serious error.

2. *Skin retraction time.** The back of the hand was found most suitable for measuring the elasticity of the skin. The hand and forearm were placed at rest on a table. The caliper was opened to 2 cm (on the crossarm scale) and allowed to close on a fold of skin several centimeters proximal to the knuckles near the middle of the hand, with the long axis of the fold diagonal (45°) to the longitudinal axis of the hand to avoid natural folds of skin. The caliper was allowed to remain closed for exactly 60 sec and then removed, and the time for the skin fold to retract back to the normal skin contour was measured in seconds. The exact end point was sometimes difficult to measure in older people; if the fold had not retracted completely in 90 sec, this time measurement was used.

3. The exposed skin of the hands and face was observed for the presence of *senile changes* such as keratoses, nevi, pigmentation, etc. The degree of such change was estimated on a 0 to 4+ scale: the presence of only an occasional abnormality was scored as 1+, increasing abnormalities raised the score.

4. The degree of *greying of the hair* was expressed on a 0 to 4+ scale as follows: 0, no greying; 1+, slight "salt and pepper;" 2+, moderate "salt and pepper;" 3+, nearly complete greying; and 4+, complete greying.

5. The degree of *baldness* was expressed on a 0 to 4+ scale as follows: 0, no apparent balding; 1+, slight receding of the hair at the temples; 2+, marked receding of the hair at the temples with some thinning; 3+, marked thinning and baldness; and 4+, baldness complete to a "monk's cap" type.

Special Senses. 1. *Accommodation** was measured in diopters by use of the Prince refracting rule. The average reading of the two eyes was used.

2. *Visual acuity** was measured by Snellen's test. It was found that use of the square root of the average visual acuity (denominator) of the two eyes made the scale more compressed and more linear. Thus the best vision, 20/10, was represented as 3.2 (the square root of 10) and the worst, 20/200, as 14.1 (the square root of 200), and intermediate readings were similarly recorded.

3. *Arcus senilis* was estimated on a 0 to 4+ scale. Only slight limbic clouding was scored as 1+, and increasing clouding raised the score.

4. *Hearing** was tested in a tent in a quiet location with a rugged screening-type audiometer.**

*The reliability of the skin looseness and skin retraction time measurements was tested by having two examiners take data on 20 hospital patients of various ages with the skin calipers. The mean values with their standard deviations were: for skin looseness in mm, 20.85±0.71 and 20.90±0.70; for skin retraction time in seconds, 58.2±8.13 and 58.75±8.18. No significant difference was found between the means of the two examiners, who had previously ascertained that their techniques for using the calipers were alike.

*The tests were carried out under standardized conditions, but, in view of the necessity of using an interpreter under field conditions, it was not feasible to repeat them with a different examiner. The data are thought to be sufficiently reproducible to be of relative value, although not so accurate perhaps as those obtained under more desirable conditions.

**The authors are grateful to the Armed Services Medical Procurement Agency, Fort Totten, N.Y., for loan of the audiometer.

Impairment of hearing was averaged for the two ears as follows: the decibel loss for each of seven frequencies (200, 500, 1000, 2000, 3000, 4000, and 7000) in each ear was averaged to give a mean frequency loss in decibels for the two ears.

Cardiovascular Changes. 1. Systolic and diastolic *blood pressures* were obtained with the standard aeronoid cuff-type sphygmomanometer. Two readings were obtained, and the average value was used. There was no basic or adjusted level of physical activity such as resting for a standard period prior to the readings. Pressures were taken on the left arm with the subject supine during the course of the physical examination.

2. The degrees of *peripheral arteriosclerosis* and *retinal arteriosclerosis* were scored on a 0 to 4+ scale. The former was estimated by palpation of the peripheral arteries, the latter by viewing the retina with an ophthalmoscope.

Neuromuscular Function. Neuromuscular function was measured by having the subject depress the key of a hand tally type of blood cell counter as many times as possible in the period of one minute. The total number of depressions represented the score.

Hand Strength. Hand strength was measured by a Smedley hand dynamometer.* The spring tension of the hand grip was measured in kilograms. The maximum squeeze strength in the dominant hand in three tries was recorded.

Handling of Data. Because of sex differences, some of the above criteria were evaluated separately for the two sexes; these were baldness, neuromuscular function, and hand strength.

In order that the estimated and measured data could be compared and combined, both types of data were converted to a percentage scale. The estimated values 0, 1+, 2+, 3+, and 4+ were presented also as 0, 25, 50, 75, and 100%, respectively. In the case of the measured data, the values associated with least aging were taken as zero percent (sometimes the highest reading, as with hand strength; sometimes the lowest, as with hearing loss), and those indicating most aging as 100%.

The data were examined on both an individual basis and a population basis. A mean age score was obtained for each individual by averaging all his percent test values. In studying population trends, means were calculated for each criterion by 5-year age groups (in most cases) including combined exposed and unexposed populations; these were plotted, and a curve was drawn according to the best fit by eye. A curve was obtained in the same way of the 5-year group means of the individual mean age scores. The combining of scores into 5-year age groups was done to reduce possible errors due to the uncertainty of the exact age of some individuals.

*C.H. Stoelting Company, Chicago, Ill.

Laboratory Procedures

Hematological Examinations. Two complete routine blood counts were done, about a week apart, and a third was done on persons showing abnormalities. White blood counts, red blood counts, and Price-Jones curves (for determination of red cell size distribution) were obtained with the electronic Coulter, which proved very satisfactory and time-saving for this type of field examination. Differential counts were performed in the usual manner after staining with Wright's fluid. Platelet counts were done by phase microscopy, and hemoglobin was determined by the cyanhemoglobin technique with the Lumitron colorimeter. Serum proteins were determined with the Hitachi refractometer. Blood and serum samples for the studies described below were collected in the field, kept under refrigeration, and shipped by air to the various laboratories in the United States for analysis.

Urine Analyses. Routine urine analyses were carried out on all people receiving physical examinations. These included determinations of protein and hyperglucosuria by reagent paper strips.* In all cases showing positive findings, the urine was centrifuged and the sediment examined microscopically. In the four cases showing positive urine sugar tests, fasting blood specimens were obtained and analyzed for blood sugar at the Naval Dispensary on Kwajalein.

Serum Cholesterol. Serum cholesterol was determined in blood samples drawn from exposed and unexposed Rongelap people, and also in about 70 blood samples from people at Utirik Island and 63 from people at Majuro Atoll. These analyses were done at the National Institutes of Health by Dr. J.H. Bragdon and Mr. J.C. Lauter.

Complement Fixation Tests. Serum samples obtained from 163 exposed and unexposed Rongelap people were frozen and sent to the National Institutes of Health for various examinations. Com-

*Clinistix, Ames Company, Inc., Elkhart, Indiana.

plement fixation tests were carried out by Mr. H. Turner and Dr. R. J. Heubner for the following diseases: para-influenza 1, 2, and 3; respiratory syncitial; psittacosis group; and Q fever. The modified Bengtson method was used.[11,12]

Sodium and Potassium Levels. Determinations of sodium and potassium urinary excretion and dietary levels were carried out by Dr. L.K. Dahl of Brookhaven National Laboratory. This study was made to see whether there was any correlation between salt consumption by the Rongelapese and blood pressure levels. Both spot and 24-hr urine collections on about 200 people were tested for sodium and potassium levels by flame photometry. Several sample meals from the Rongelap people were also analyzed.

Thyroid Metabolism. Since the largest dose to any part of the body had been received by the thyroid glands in the Marshallese, studies of the metabolic state of the thyroid gland have been of interest. These studies have been made by Dr. J.E. Rall at the National Institutes of Health. Samples from the previous years' surveys had shown surprisingly high protein-bound iodine levels. In order to determine whether this was a true finding or due to contamination of glassware, 14 samples were again collected, with very carefully cleaned glassware used. In addition to protein-bound iodine, butanol-extractable iodine and thyroxin binding proteins were determined. Several urine samples were analyzed for total iodine content.

Serum Vitamin B_{12}. Determinations of serum vitamin B_{12} concentrations done during the previous year's survey gave values higher than normal in the majority of the Marshallese people. Therefore, 15 samples from the 1959 survey (7 from persons tested the year before) were analyzed by Dr. D.W. Watkin of the National Cancer Institute, National Institutes of Health.

Studies of Genetically Inherited Characteristics

Studies of genetically inherited characteristics of blood components and urine were continued on samples brought back to laboratories in the United States. Such studies, although not directly related to radiation effects, are of interest in understanding the anthropological background of the people and in establishing a base line of genetic characteristics for detection of possible genetic effects of radiation in future generations. These studies included the following: blood groups ABO, MN, Rh-Hr, and Duffy, Kell, and Diego by Dr. Leon N. Sussman, Beth Israel Hospital, New York, N.Y.; and haptoglobins by Dr. B.S. Blumberg and Zora Gentile of the National Institutes of Health. Blood samples were obtained from 176 individuals representing 70% of the inhabitants of Rongelap for the above studies. Aliquots of urine samples from 65 exposed and 119 unexposed people collected for routine analysis were used for determination of β-amino-*iso*-butyric acid. Eighteen urine samples from Utirik were also analyzed.

Blood Groupings. To complement studies begun in 1958, blood grouping studies were carried out on 57 of the blood samples mentioned above. In addition, 64 blood samples were collected at Utirik, and 65 at Majuro for this purpose. Dr. Sussman tested these for ABO, MN, Rh-Hr, and Duffy, Kell, and Diego factors.

Haptoglobin and Transferrin. Further analyses for haptoglobin by the method of Smithies were carried out on these samples at the National Institutes of Health. Transferrins were determined by the discontinuous buffer starch gel method of Poulik[13] on 66 Rongelapese sera, and on an additional 40 sera by the borate buffer, horizontal method.[14] No transferrin types other than CC were seen.

Hemoglobin Types. Further starch gel electrophoretic studies of hemoglobin types were also made on these samples by Dr. R.L. Engle, Jr., and Dr. G. Castillo of the Cornell University Medical Center, New York, N.Y.

Glucose-6-phosphate Dehydrogenase Activity of Red Cells. Various studies have indicated that a deficiency of the enzyme of red cells is transmitted by a sex-linked gene. Individuals with this deficiency can develop a hemolytic anemia after the ingestion of certain drugs (e.g., primaquine or fava beans[15-18]).

In the present study the blood samples were collected in ACD solution, refrigerated (4°C), and sent by air to Seattle, Washington, where the tests were done by Dr. Arno G. Motulsky, Department of Medicine, University of Washington. All tests were performed within one week of collection. Tests were done on 151 individuals: 75 males, 75 females, and one not classified.

β-Amino-iso-butyric Acid (BAIB) Excretion. Differential excretion of β-amino-*iso*-butyric acid (BAIB) is under genetic control, and family data indicate that a single major gene pair is responsible for most of the variations.[19,20] High excretors are homozygous for a single recessive gene, and

low excretors either heterozygous or homozygous for the dominant allele. Striking differences in the incidence of high excretors in various populations have been demonstrated, and it appears that this genetic polymorphism may be of considerable value in anthropo-genetic investigations.[21,22]

As a part of the study of the genetic relationships and origins of various Pacific peoples, this report extends observations on BAIB excretion to the Micronesians inhabiting the Marshall Islands. The fact that some of the Micronesians were exposed to considerable radiation in 1954 is of special interest here, since it is known that radiation exposure can, temporarily at least, increase BAIB excretion,[23] and studies on the Marshallese population may elucidate possible long-term effects.

Urine samples from 65 exposed and 119 unexposed people (75.7% of the population of the village) were collected in plastic bottles containing thymol preservative. The sexes were equally represented, and the ages varied from 3 to >70 years. In some cases several members of the same family were included, and some kinships suitable for genetic analysis were available. Eighteen samples collected on Utirik were also studied. Urines were kept at 4°C and shipped in refrigerated containers to Seattle for analysis. These studies were carried out by one of us (B.S. Blumberg) and Dr. S.A. Gartler of the Department of Medicine, School of Medicine, University of Washington. Studies were completed 2 to 3 weeks after collection of the specimens. Determinations of BAIB were carried out by high voltage electrophoresis on paper,[24] and creatinine was determined by the alkaline picrate method.

Radionuclide Body Burden Evaluation

The methods used in the radionuclide body burden evaluation are described later in a separate section.

1960 Survey

BACKGROUND MATERIAL

The 1960 survey was reduced in size and scope and limited to a very brief examination of the exposed people only. Several factors brought about this change. The people had recovered to the extent that certain special examinations previously carried out every year need be done only once every two to three years. In addition, as pointed out before, the Trust Territory officials were concerned about the slowness of economic recovery of the Rongelapese and felt that the numerous visiting scientific teams, particularly those with large ships and crews, were partly responsible for the unrest of the people and therefore requested that the size of the surveys be kept to a minimum. It was decided to defer the gamma spectrographic analysis until 1961. The Trust Territory officials agreed to greater participation of their medical personnel in future surveys and to the use of one of their cargo ships (Figure 9), which routinely made the rounds of the islands for gathering copra, for carrying out the survey at Rongelap. Accordingly, for the 1960 survey the team consisted of only one physician and one technician from Brookhaven National Laboratory, and the remainder of the medical group, arranged by the Trust Territory, included its Director of Public Health, two Marshallese medical officers, and two Micronesian laboratory technicians. The Director of Dental Services and one of his dental officers also accompanied the team to carry out treatment of the people. The District Administrator of the Marshall Islands accompanied the team in order to consult with the people on their agricultural program.*

As in the previous year, several of the exposed people now living at Kwajalein and Majuro Atolls were examined at these atolls prior to the Rongelap visit.

PROCEDURES

As in previous surveys, examinations were carried out in the dispensary and the schoolhouse in Rongelap village. Interval medical histories and complete physical examinations were carried out

*Again in 1960, when the team arrived at Rongelap, the magistrate requested a meeting with the people. The line of questions and discussion was about the same as that reported for the previous year. Resistance toward the examinations was expressed by only one or two of the people. Objections were again raised against the ban on eating coconut crabs, which selectively concentrate Sr^{90} to such an extent that their consumption had to be prohibited (Figure 4). The people were assured that these crabs were being repeatedly examined and that, when it was safe to eat them, immediate notification would be given. Fish poisoning apparently had been less of a problem during the past year since only one case had been noted, and the subject was not brought up again. Following this meeting, cooperation by the people in the examinations was almost complete.

Figure 9. Trust Territory ship used for 1960 survey, anchored in the lagoon off Rongelap village.

Table 2

Percent Distribution of Population by Age Groups

Age, yr	Rongelap control (206 people, 1959)	Rongelap exposed (102 people, 1959)	Marshall Islands (1948-50)	U.S. (1940)
<15	45.6%	49.0%	33.8%	25.1%
15–24	11.2	12.7	18.9	18.2
25–44	23.8	20.6	25.9	30.1
45–64	15.0	10.8	15.5	19.8
>65	4.4	6.9	5.9	6.8
Median age, yr	19.0	16.2	23.6	29.0

on the exposed population only. The histories were taken by one of the Marshallese doctors. Complete physical examinations were done, but special examinations, such as slit-lamp studies of the lens and anthropometric measurements on the children (except for height and weight), were not made. One hematological examination was carried out on the exposed population which included WBC by electronic counting technique (Coulter), differential smears, smears for alkaline phosphatase staining, and basophil counts. About thirty 24-hr urine samples were collected from exposed and unexposed people for radiochemical analysis to determine body burdens of Sr^{90}.

Results and Discussion

The results of the 5- and 6-year post-exposure surveys will generally be reported together.

INTERVAL MEDICAL HISTORY

The census of Rongelap as of 1959 was 308 people, of whom 82 had been exposed, 20 were children born of the exposed, and 206 were unexposed. A census was not taken in 1960, but the number of people appeared to be about the same. Table 2 shows the percentage distribution in the population for the Rongelap exposed and unex-

posed groups, compared with that for the Marshall Islands as a whole in 1948-50 and for the United States in 1940. The table also shows the median ages. The lower median age of the Marshallese would seem to support the impression that their life span is shorter than that in the continental United States.

During the interval between the surveys of 1958 and 1959 the people of Rongelap suffered no major epidemics. There were the usual bouts of upper respiratory infections. A few cases of chicken pox developed in April 1958, two of great severity, one of which resulted in the death of a 36-year-old man (#31) from the exposed group. During the year Navy evacuation planes removed these two cases to Kwajalein Hospital (150 miles away) for treatment, and also the following cases: perforated appendix, threatened abortion, retained placenta, complicated delivery, pyelonephritis, and acute diarrhea. Three of these patients were in the exposed group. Four cases of fish poisoning occurred during the year with the usual symptoms of nausea, vomiting, diarrhea, fever, double vision, and tingling sensations in the limbs. Cancer of the ovary was found in a 61-year-old woman in the exposed group, and the diagnosis was confirmed by biopsy.

During the interval between the 1959 and 1960 surveys the medical history of the people on Rongelap Island was generally uneventful. However, an epidemic of influenza occurred in the Marshall Islands in the spring of 1960, and, though Rongelap Atoll was spared, the epidemic reached serious proportions on Kwajalein Atoll. About 20 of the unexposed Rongelap control population had previously moved to Kwajalein Atoll for employment by the Navy, and among this group 10 cases of influenza developed, two of which became complicated by pneumonia resulting in death – one in a 55-year-old man (#933) and the other in a 64-year-old man (#927). Both these men had complicating diseases prior to influenza; one had suffered a cerebral hemorrhage with hemiplegia several months earlier, and the other a urinary tract infection. The 61-year-old woman with cancer of the ovary, discovered in 1958, died in May 1959. Unfortunately, no autopsies were obtained on these people. This brings the deaths to four for the exposed population. One other death occurred in the unexposed control population on Rongelap, that of a 54-year-old woman (#854) who died of infection complicating diabetes. The deaths in the unexposed population now number four. Only one case of fish poisoning occurred in 1959 on

Table 3

Yearly Incidence of Births and Fetal Deaths*

Year	Women aged 14–45	Total pregnancies	Live births	Children M	Children F	Miscarriages*	% Pregnancies terminating in miscarriage
			Exposed				
1954**	19	1	0	0	0	1	100
1955	20	6	5	4	1	1	17
1956	20	6	4	0	4	2	33
1957	21	5	2	2	0	3	60
1958	22	14	8	4	4	6	43
1959	22	5	5	2	3	0	0
Total		37	24	12	12	13	
			Unexposed				
1956	18	8	6	5	1	2	25
1957	18	8	6	2	4	2	25
1958	18	4	3	2	1	1	25
1959	17	6	5	1	4	1	17
Total		26	20	10	10	6	

*Includes children dying during first few hours after birth.
**Includes only children conceived after March 1, 1954.

Rongelap. During that year the health aide was visited frequently for upper respiratory infections (nearly everyone had at least one cold during the year). Gastroenteritis was also a frequent complaint.

REVIEW OF DATA ON MORTALITY AND PREGNANCY TERMINATION OVER THE PAST SIX YEARS

Mortality

The four deaths that have occurred in the exposed Rongelap people during the past 6 years give an annual rate of 8.1 per 1000 population. The Marshall Islands annual rate is reported as 6.8 per 1000. The unexposed Rongelap population has had four deaths also, which gives a rate of 8.3 per 1000. These figures do not include fetal and infant mortality. The people of Utirik Island, who received about 14 r of radiation in the 1954 accident, have shown a death rate of about 10 to 11 per 1000 population. Their population has varied between 160 and 213 people since the event.

Pregnancy Terminations

During 1958 six miscarriages and stillbirths were recorded in the exposed group, but none was reported in this group for 1959. Only one was reported for each of these years in the unexposed women. Pregnancy terminations in the exposed group are compared with those in the unexposed group in Tables 3 and 4. Since any radiation-induced genetic imperfections that might result in nonviable offspring might be present in the germ plasm of the father as well as the mother, two unexposed women mated to exposed men are included in the exposed group. Four children born in 1954 were excluded from the list because they had been conceived before the accident.

Table 3 and Figure 10 show the yearly incidence of live births and miscarriages and the sex of babies born of women of child-bearing age in the exposed and unexposed groups. Under the category of miscarriages are included stillbirths and babies dying a few hours after birth. It was unfortunate that in most cases it was not possible for physicians to inspect the products of miscarriage. Figure 10, a plot of the percentage incidence of miscarriage in the two groups, indicates that it is somewhat greater in the exposed group. The data on pregnancy terminations, summarized in Table 4, also show an increased incidence of miscarriage in the exposed group.

The birth rate in the Marshall Islands in 1957 was 37.3 per 1000 population. The 24 live births

Table 4

Summary of Pregnancy Termination Data, Rongelap Exposed (1954–1959) and Rongelap Unexposed (1956–1959)

	% Exposed	% Unexposed
Women giving birth to living children	64	66
Women with miscarriages* but no live births	18	11
Women with no recorded pregnancies	18	22
Women with 1 or more miscarriages	41	28
Women with 2 or more miscarriages	14	11
Pregnancies terminating in miscarriages	35	23

*Includes children dying first few hours after birth.

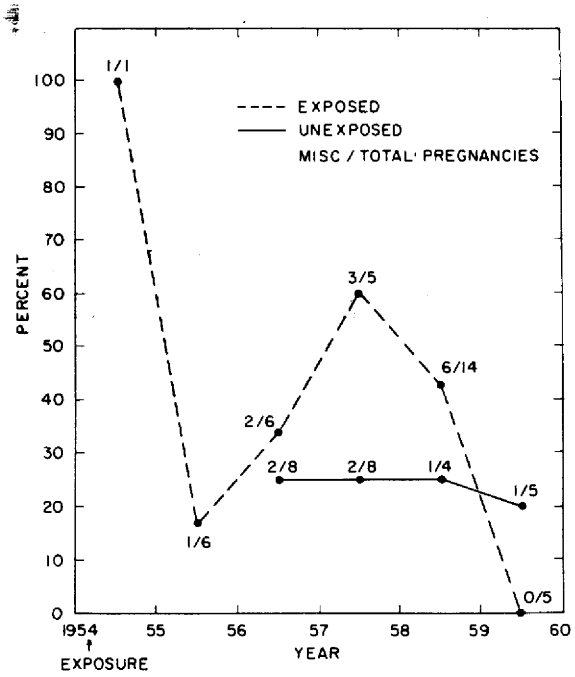

Figure 10. Incidence of miscarriages and stillbirths in exposed Rongelap women. Stillbirths include babies living only a few hours after birth. Data on unexposed women are incomplete prior to 1956.

over a 6-year period in the exposed population represent 48 per 1000, and the 20 births over a 4-year period for the unexposed population represent a rate of 62 per 1000. The four *in utero* irradiated children did not show any abnormalities nor have they shown any impairment of growth and development.

PHYSICAL EXAMINATIONS

At the times of the 1959 and 1960 surveys of the Marshallese, the people appeared to be generally in a state of good health and nutrition. There was no indication of vitamin deficiencies such as had been observed in 1957 in the children, when about 10 had night blindness associated with vitamin A deficiency.[6] The improvement may be due in part to the argicultural program on the island resulting in the availability of papaya and squash. The usual number of skin infections and fungus diseases was noted, as was the usual incidence of impetigo in the children.

PEDIATRIC EXAMINATIONS

The numbers of children seen during the 1958, 1959, and 1960 surveys are summarized in Table 5. In the limited 1960 survey, only the body meas-

Table 5

Numbers of Children Examined in 1958, 1959, and 1960

	In 1960	In 1959	In 1958	In 1958 but not in 1959	In 1959 but not in 1958	In 1959 but not in 1960
Exposed	35	34	39	5 (5)*	0	0
Nonexposed with exposed parents	10	20	13	0	7	10
Nonexposed, Rongelap	51	82	88	16 (3)	10	34 (2)
Nonexposed, Majuro	0	15	20	6 (2)	1	15
Utirik	0	60	0	0	60	60

*Numbers in parentheses are numbers of children not examined who had become adults or near-adults since the preceding survey.

Table 6

Summary of Physical Findings on Children, 1959

	Exposed Rongelap (34)*	Nonexposed with exposed parents (20)	Nonexposed Rongelap (82)	Nonexposed Majuro (15)
Chronic impetigo (active)	10	7	25	0
Molluscum contagiosum	0	1	6	0
Tinea versicolor	2	0	0	2
Chronic otitis media	0	0	4	0
Palpable liver				
Under 3 cm	4	1	15	2
Over 3 cm	5	2	11	2
Cervical nodes	14	6	36	8
Axillary nodes	8	2	13	3
Rheumatic heart disease, inactive	1	0	0	0
Vitiligo	1	0	0	1
Cheilosis	0	0	0	1
Asthma	0	0	1	0
Loss of hearing, unilateral	0	0	1	0
Elevated blood pressure**	0/20		0/36	
Obesity in girls >10 years of age**	1/4		0/7	0/8

*Number examined.

**Utirik group showed no elevated blood pressure in 58 children examined, and an incidence of obesity in girls >10 years of age of 7/16.

urements were determined in children. Comparisons have been made between the numbers examined each year. Excluding those who were shifted into the adult study, the total over-all attrition rate between 1958 and 1959 was 10%. Of the 18 children not seen in 1958 but examined in 1959, 14 were babies born in the interval between the two examinations.

The age distribution of children examined in 1959 was as follows:

	Age, yr			
	<4	4 to 9	⩾10	Total
Exposed		19	15	34
Nonexposed with exposed parents	20			20
Nonexposed, Rongelap	30	26	26	82
Nonexposed, Majuro		6	9	15
Utirik	?	?	?	60

The occurrence of various medical conditions is summarized in Table 6. There seemed to be no concentration of clinical abnormalities in the exposed groups. Since the incidence of many of these conditions could have been related to the ages of the children at the time of examination, two of the most frequent findings, active chronic impetigo and palpable liver over 3 cm in size, were tabulated separately for several different age groups (Tables 7 and 8). The ages were based on the best available birth date estimations. A tendency for these findings to occur more frequently in the younger children was noted. The incidences, however, did not appear to be related to exposure to radiation.

During the physical examinations, the following congenital anomalies were recorded:

Patent ductus arteriosus (repaired)	1 (Subject #805)
Deformity of the hip	1 (Subject #896)
Pectus excavatum	1 (Subject #2265)
Bifid uvula	1 (Subject #2269)

In addition, the incidence of congenital hypoplasia of the middle phalanx of the fifth finger was determined from inspection of the roentgenograms of the left hand. The incidence in relation to various exposure categories was as follows:

	Incidence
Exposed, Rongelap	2/33
Nonexposed with exposed parents	0/4
Nonexposed, Rongelap	5/53
Nonexposed, Majuro	0/14
Utirik (low exposure)	5/51

Hypoplasia of the middle phalanx has been reported as occurring in the normal population in incidences ranging from 0.5% to 5.2%. The anomaly was found to have considerably greater frequency (25%) in Japanese children.[25] The over-all incidence in the Marshallese children was 0.77%.

Pediatric dispensary work was carried out preceding and following the regular examinations. In all, 36 children were treated. The presenting complaints are shown in Table 9.

Table 7

Incidence of Chronic Impetigo (Active)

	Age, yr			
	<4	4–9	⩾10	Total
Exposed		7/19	3/15	10/34
Nonexposed with exposed parents	7/20			7/20
Nonexposed, Rongelap	13/30	7/26	5/26	25/82
Nonexposed, Majuro		0/6	0/9	0/15

Table 8

Incidence of Palpable Liver (Over 3 cm)

	Age, yr			
	<4	4–9	⩾10	Total
Exposed		5/19	0/15	5/34
Nonexposed with exposed parents	2/20			2/20
Nonexposed, Rongelap	6/30	5/26	0/26	11/82
Nonexposed, Majuro		1/6	1/9	2/15

Table 9

Pediatric Dispensary Visits During 1959 Survey

Presenting complaint	Number of patients
Cough	18
Back or limb pain	5
Skin sores	3
Otitis	3
Diarrhea	3
Anorexia	3
Headache	2
Worms	1
Loss of hearing	1
Laceration	1
Abdominal pain	1

Growth and Development Studies

In evaluating the growth and development data on these children, serious inconsistencies in birth date information have been uncovered. Official written birth records did not exist for most of the children. The parents actually had no realistic perspective of time. No local or regional events, tragic or otherwise, were remembered to serve as reference points. The births of some children had been registered at Majuro, but even among these a few instances showed conflict between the recorded date and the available circumstantial evidence.

Since almost all analyses of growth data depend basically on the use of chronological ages, the painstaking task of improving the validity of the age data was undertaken. This amounted to a virtual reconstruction of the biological history of the childhood population of the island. Interviews were held with the parents, relatives, and village elders. Cross-examinations were conducted to obtain all relevant information. In spite of these efforts, a significant lack of accurate information remained in many cases. Further attempts to check birth dates are necessary before classification of the children into age groups can be done with reasonable validity.

An earlier analysis of the skeletal ages of the Marshallese children had indicated possible retardation in development among the exposed group.[5] Since such comparisons required reference to accurate chronological ages, further detailed analyses of this type were deferred. It was noted, however, that in the 6-year chronological age group three boys and one girl out of five boys and two girls exposed to radiation were markedly retarded in skeletal maturation (Table 10). The birth dates of these particular children seemed firmly established. The boys showing most retardation (#2, 3, and 5) were 16 to 17 months old and the girl (#65) 15 months old at the time of expo-

Table 10

Skeletal Ages in 6-Year-Old Children

Subject No.	Sex	Age at exposure, mo	Chronological age in 1959, yr	Skeletal age* in 1959, yr
2	M	16	6 4/12	4 6/12
3	M	17	6 6/12	2 10/12
5	M	16	6 5/12	3 6/12
6	M	16	6 5/12	5 6/12
65	F	15	6 3/12	3 6/12
33	F	20	6 8/12	7 6/12
54	M	12	6 1/12	†
955	F	**	6 10/12	†
962	F	**	6 4/12	†
980	F	**	6 5/12	6 10/12
996	F	**	6 3/12	†
814	M	**	6 11/12	5 6/12

*Greulich-Pyle standards.
**Control.
†No film.

Table 11

Height and Weight of 6-Year-Old Children

Subject No.	Height, cm	Weight, lb	Skeletal age peers: Height, cm	Skeletal age peers: Weight, lb
2	108.3	41.5	99.3	32.0
3	102.2	39.5	95.3	32.5
5	98.8	36.0	104.8	36.1
6	106.3	41.0	109.0	41.0
65	98.4	33.0	—	—
33	115.8	43.8	118.2	47.4
54	112.5	47.5	—	—
955	117.5	47.5	—	—
962	108.3	42.3	—	—
980	112.8	43.8	—	—
996	108.0	35.0	—	—
814	111.7	43.0	—	—

Table 12

Comparison of Stature (1958, 1959, and 1960) of Children With Retarded Osseous Development With That of Their Next Younger Sibs

	Sex	Born	Stature, cm: 1958	1959	1960
Subject (#5)	M	10/20/52	95.7	98.8	102.2
Sib (#85)	M	9/ 7/54	95.5	100.9	108.0
Subject (#2)	M	10/23/52	103.0	108.3	115.6
Sib (#91)	M	1/ 3/55	89.8	97.1	104.1
Subject (#3)	M	9/11/52	98.5	102.2	106.7
Sib (#83)	M	6/ 8/54	97.6	98.6	113.0
Subject (#65)	F	12/ 4/52	93.0	98.4	102.9
Sib (#86)	F	10/17/54	90.6	97.0	103.5
Subject (#6)	M	10/14/52	100.4	106.3	111.8
Sib (#84)	M	5/31/54	94.2	98.6	104.8

Table 13

Comparison of Anthropometric Data (1959) on Children With Retarded Osseous Development With Those of Their Next Younger Sibs

Subject No.	Age in 1959, yr	Weight, lb.	Stature, cm	Sitting height, cm	Head circumference, cm	Biacromial width, cm	Bi-iliac width, cm	Calf circumference, cm
5	6 5/12 (2)*	36 (2)	98.8 (1)	54.6 (1)	48.3 (2)	21.6 (1)	17.0 (1)	22.0 (2)
85	4 6/12 (1)	33 (1)	100.9 (2)	56.0 (2)	46.0 (1)	22.0 (2)	17.8 (2)	20.8 (1)
2	6 4/12 (2)	41.5 (2)	108.3 (2)	60.3 (2)	52.7 (2)	22.8 (2)	18.0 (2)	22.6 (2)
91	4 3/12 (1)	34.5 (1)	97.1 (1)	56.3 (1)	49.5 (1)	21.6 (1)	16.8 (1)	21.4 (1)
3	6 6/12 (2)	39.5 (2)	102.2 (1)	57.4 (1)	49.3 (1)	22.3 (–)	16.9 (1)	22.6 (1)
83	4 9/12 (1)	38.3 (1)	104.7 (2)	59.5 (2)	50.0 (2)	22.3 (–)	17.0 (2)	23.6 (2)
65	6 3/12 (2)	33.0 (2)	98.4 (2)	55.8 (2)	47.2 (1)	20.8 (1)	17.5 (2)	20.1 (–)
86	4 5/12 (1)	29.8 (1)	97.0 (1)	54.5 (1)	48.4 (2)	22.0 (2)	16.6 (1)	20.1 (–)
6	6 5/12 (2)	41.0 (2)	106.3 (2)	59.3 (2)	49.3 (2)	23.0 (2)	17.0 (2)	22.4 (2)
84	4 9/12 (1)	35.5 (1)	98.6 (1)	55.0 (1)	48.3 (1)	21.6 (1)	16.5 (1)	21.3 (1)

*Numbers in parentheses refer to ranking of each item, (1) indicating the younger child or the smaller measurement of the pair and (2) the older child or the larger value.

sure to radiation. One boy (#6) showed less retardation. One boy and one girl, also about the same age, were exposed to radiation but did not show any retardation in bone development.

The height and weight of the one exposed girl with retarded osseous maturation were considerably below those of chronological age peers (Table 11). However, measurements on the one exposed girl with normal bone development (#33) were not inferior to those of control chronological age peers. She was slightly smaller than her control skeletal age peers. For the boys, unfortunately, there were insufficient control chronological age peers for calculation of means. Comparison with skelatal age peers indicated that two of the boys with skeletal retardation were taller and one shorter than the controls (Table 11).

Comparison of the physical sizes of the children with retarded skeletal maturation with the physical sizes of their sibs brought out another significant finding. Three (subjects #3, 5, and 65) of the five children with skeletal age retardation were shorter in stature in 1960 than their next younger sibs (Table 12; see also Figure 11). Increment data indicated that these three children failed to show satisfactory statural gain during the past two years, even though in 1958, at the age of ≈5½ years, all three had been taller than their younger sibs. The difference in age between sib pairs

Figure 11. Brothers. Left, #5, age 6; right, #85, age 4 (1960).

Table 14

Physical Findings (Excluding Ophthalmological) in Marshallese Adults 1959 and 1960

	Exposed (43 examined)		Control (84 examined)	
	Subject Nos.	%	Subject Nos.	%
Acne			833, 865, 882	3.6
Arteriosclerosis, peripheral, mild	11, 52	4.7	850, 851, 852, 854, 855, 858, 859, 871, 873, 878, 884, 886, 894, 898, 899, 908, 910, 917, 956, 957, 969, 970, 973, 982	28.6
Arteriosclerosis, peripheral, moderate to severe	13, 28, 29, 30, 43, 46, 55, 56, 57, 60	23.3	853, 860, 861, 862, 927, 933, 947, 964	9.5
Asthma			953	1.2
Auricular fibrillation with myocardial damage	80	2.4		
Blindness	28, 29, 55, 56,	9.3		
Bronchitis, chronic	52	2.4		
Carcinoma, ovarian	62	2.4		
Cardiac enlargement	30, 43, 60	7.0	853, 859, 862, 942, 964,	6.0
Cyst, perineal			958	1.2
Colloid goiter			858	1.2
Deafness	1, 43	4.7	853, 862, 910, 916, 964	6.0
Diabetes mellitus			853, 854, 893, 991	4.8
Emphysema			853, 855	2.4
Ganglion, left wrist			970	1.2
Hallux valgus	50	2.4		
Heberden's nodes	52, 57	4.7	908, 928	2.4
Hemiplegia, partial	46	2.4		
Hemorrhoids, mild			859, 860	2.4
Hypertension (>140/90)	11, 28, 30, 46, 56, 58, 60	16.2	850, 851, 852, 853, 858, 859, 862, 878, 884, 894, 908, 933, 947, 957, 964, 982, 991	20.2
Hypotension	49, 51	4.7		
Impetigo	79	2.4	873	1.2
Inguinal hernia			1005	1.2
Kyphoscoliosis	13, 43, 56, 57	9.3	854	1.2
Leprosy	77	2.4		
Lipoma, chest			875, 964	2.4
Myocardial damage or insufficiency (EKG)	46	2.4	844, 851, 858, 861, 878, 884, 893, 917, 933, 947, 956, 957, 969, 970	16.7
Osteoarthritis	13, 29, 46, 56, 57, 60	14.0	854, 859, 860, 862, 884, 886, 894, 898, 899, 915, 927, 933, 935, 964	16.7
Otitis media, chronic	49	2.4	832, 973	2.4
Prolapse of vaginal wall	45, 59, 63	7.0		
Prostatic hypertrophy	29, 46, 82	7.0	856, 862, 864, 868, 873, 899, 910, 915, 920, 933, 944, 953, 964	15.5
Proteinuria	22, 30, 71, 79	9.3	855, 862, 895, 993	4.8
Rheumatoid arthritis (?)			858, 878	2.4
Senility	55, 56	4.7		
Shortened left thumb, congenital	57	2.4		
Tonsilar hypertrophy	1, 64, 77	7.0	864, 867, 914, 928, 934, 944	7.1
Traumatic arthritis			975	1.2
Trichomonas of urinary tract	9	2.4		
Tumor, left ear (benign)			882	1.2
Tumor, vulva (benign?)	13	2.4		
Upper respiratory infection	27, 28, 40	7.0	873, 875, 908, 935, 938, 973, 1001	8.3
Varicose veins	13	2.4	881	1.2

ranged from 20 to 26 months, with an average of 22.4 months.

To determine whether or not some pattern in physique characterized these children with retarded osseous development, several physical measurements (from the 1959 study) on them and on their sibs were compared (Table 13). These anthropometric data suggested two trends. First, the weight rankings corresponded to chronological age rankings and not to statural rankings. Second, the lack of synchrony in rankings of several measurements was noticeable in those children who were shorter than their younger sibs. This contrasted with the uniform ranking of all measurements in those whose statural ranking corresponded with the chronological age ranking.

It might be speculated from these limited observations that these children were exposed to radiation at a particularly vulnerable age and that the resulting retardation in osseous development led to failure in statural growth. On the other hand, it is not possible to exclude completely the possibility that some process unrelated to radiation damage was responsible for the retardation in skeletal development.

ADULT ABNORMALITIES

Table 14 is a compilation of the various physical abnormalities noted in the adult group during the 1959 and 1960 surveys. No abnormalities are included for 1960 in the unexposed group because this group was not examined in the 1960 survey. This table does not show any significant differences between the abnormalities noted in the exposed and in the unexposed populations. Results of special examinations are discussed below.

CARDIOVASCULAR SURVEY

The cardiovascular findings may be found in the table of physical abnormalities (Table 14). The incidence of various electrocardiographic abnormalities is shown in Table 15. The population was divided into a younger group, aged 20 through 49, and an older group, aged $\geqslant 50$. In the exposed population, the younger group of 24 people showed no major abnormalities, but of the 14 older people 29% showed one or more abnormalities. In the unexposed population, among the younger group 15% had one or more abnormalities, and in the older group 41%. Specific findings may be summarized as follows.

Electrocardiographic Findings

1. Rhythm. In the younger group of exposed subjects, all had normal rhythm. One abnormality of rhythm was seen in a member of the younger unexposed group. In the older groups, arrhythmia occurred in 3 of 14 exposed individuals and in 4 of 29 unexposed.

2. Conduction Times. Few abnormalities were seen. No individual in either the exposed or the unexposed group had prolonged auriculoventricular conduction time (P-R interval) above normal. Several subjects had the shorter conduction time of 0.12 sec; this is considered normal. The intraventricular conduction time (QRS interval) was prolonged in several subjects. In the younger unexposed group, the QRS interval was 0.10 to 0.11 sec in one subject, sufficient to be considered right bundle branch block. In the exposed population the intraventricular conduction time was prolonged to 0.12 sec in only one individual, age 81, who had a marked degree of hypertensive and arteriosclerotic cardiovascular disease and cardiac enlargement. Among 29 individuals in the older unexposed group, two showed intraventricular conduction times of 0.12 sec without the typical QRS complex of bundle branch block. In all other subjects the intraventricular conduction time ranged from 0.06 to 0.09 sec in the younger groups and 0.08 to 0.09 in the older.

3. Electrical Axis Deviation and Electrical Position of the Heart. There were few variations. The

Table 15

Electrocardiographic Abnormalities
(Percent Incidence in Younger and Older Age Groups)

	Exposed		Unexposed	
Abnormality	Age 20–49 (24)*	Age $\geqslant 50$ (14)	Age 20–49 (55)	Age $\geqslant 50$ (29)
Rhythm	0	21	2	14
A-V	12	7	2	3
I-V	0	7	4	7
RST	0	7	2	14
T wave	0	21	11	34

*Number examined.

axis deviation was normal or to the right in most individuals. Left axis deviation, and only of moderate degree, was present in six individuals, two in the younger unexposed group, three in the older unexposed group, and one in the older exposed group. The electrical position of the heart in a great majority of both the unexposed and exposed subjects was vertical. In 22 subjects it was intermediate and in 9 (all unexposed) it was moderately horizontal. Rotation of the heart electrically in the combined groups was clockwise in 25 subjects and counterclockwise in 36, with about equal distribution between the exposed and unexposed groups.

4. RST and T Waves. Abnormal variations were carefully noted for indications of coronary or myocardial insufficiency and/or myocardial disease or damage. Deviations of the RST segment from the isoelectric line were found to be minimal; they occurred in only five of the 84 unexposed subjects and in only one of the 38 exposed subjects. T wave abnormalities were of appreciable degree in only a few instances in the older group. The abnormalities were chiefly in lower T waves and were related to hypertensive disease. T wave changes were most marked in a 51-year-old male (#80) of the exposed group, who had arteriosclerotic heart disease and auricular fibrillation. Marked RST and T wave changes were noted in an 81-year-old male (#46) of the exposed group, who had marked hypertensive and arteriosclerotic cardiovascular disease, and had prolonged auriculoventricular conduction time as noted above. Among the unexposed population there was evidence of myocardial insufficiency according to RST and T wave changes in a 51-year-old male (#947), a 64-year-old female (#861), a 55-year-old male (#993), a 64-year-old female (#858), and a 64-year-old male (#884); the last had changes sufficient to indicate a healed anterior septal cardiac infarct.

Other Cardiovascular Observations

1. Peripheral Vascular Findings. Clinical estimation was made of the degree of arterial changes in the peripheral vessels (radial, brachial, and dorsalis pedis). The degree of abnormality was graded by 0 to 4+ sclerosis (see section on aging studies). Arteriosclerosis was seen chiefly in subjects >50 year of age in both exposed and unexposed groups. It was not marked except in four individuals >70 years of age in the exposed group, and then to a degree compatible with age. Since there were no individuals examined in the unexposed group >70 years of age for comparison, any assumption that the arteriosclerosis was increased in these four individuals by their exposure to fallout radiation is unwarranted. Otherwise the presence and the amount of arteriosclerosis was fairly well distributed between both populations, as can be seen in Table 14. It was not observed in a degree premature or advanced for the given age.

2. Arterial Blood Pressures. Blood pressures greater than 140 systolic and 90 diastolic were considered abnormal. On this basis in the exposed group none of the 24 subjects <50 of age and seven of the 16 subjects >50 had hypertension, but mostly of mild degree. There were 17 cases of hypertension in the unexposed group, 2 in those <50 years of age and 15 in those >50. The diastolic blood pressure showed only infrequent increase above the level of 90 mm; as would be expected, this occurred chiefly in the older group. The overall incidence of hypertension for the total population examined was 9.2%.

3. Oscillometry Readings. Observations were taken on the legs at the mid-calf. In almost all subjects the pulsation was sufficient to indicate normal blood flow. Although the readings were lower in the Marshallese than might be expected in Caucasians, it appeared that peripheral vascu-

Table 16

Ophthalmological Findings (% Incidence)

	Exposed	Unexposed
Pterygium	23.0	21.0
Pinguecula	24.0	22.0
Corneal pigment	12.0	2.6
Corneal scars	9.0	3.4
Arcus senilis	38.0	26.0
Phthisis bulbi	1.3	0.0
Nystagmus	1.3	0.7
Strabismus	8.3	0.7
Argyll-Robertson pupil	1.3	0.0
Cataracts	19.0	10.3
Aphakia	1.3	1.3
Vitreous opacities	4.1	0.0
Retinal arteriosclerosis	11.0	12.3
Chorioretinal scars	1.3	0.7
Macular degeneration	0.0	0.0
Drüsen	2.6	0.0
Congenital anomalies	3.9	0.0

lar disease was probably of lower incidence in the Marshallese. There were two low readings in the exposed group, one in a 63-year-old woman with abdominal carcinosis and ascites of ovarian origin and the other in an 81-year-old man (#46) with marked hypertensive and arteriosclerotic cardiovascular disease.

4. *Veins.* There was nearly a total absence of peripheral venous disease – thrombophlebitis, phlebothrombosis, varicose veins, or hemorrhoids – in both men and women. Only one individual showed varicose veins in the lower legs, in minimal degree.

5. *Other Cardiac Conditions.* No patients were observed with luetic, metabolic, or nutritional forms of heart disease. One evident case of inactive rheumatic heart disease occurred in a boy of 14 years (#76) with typical signs of mitral valvular involvement and moderate cardiac enlargement. He was symptom-free and his electrocardiogram was normal.

Table 17

Incidence of Individuals With X-Ray Evidence of Osteoarthritis in Rongelap, by Age and Sex (Only those rated as 2+ or higher are included)

Age, yr	No. in age group	Persons with 2+ osteoarthritis	
		Number	%
	Males		
0– 9	28	0	
10–19	23	0	
20–29	17	0	
30–39	9	2	
40–49	16	1	
50–59	8	5	
>60	5	4	
Total	106	12	
	Females		
0– 9	23	0	
10–19	17	0	
20–29	16	1	
30–39	12	0	
40–49	3	0	
50–59	6	4	
>60	11	8	
Total	88	13	
	Males Plus Females		
0– 9	51	0	0
10–19	40	0	0
20–29	33	1	3.0
30–39	21	2	9.5
40–49	19	1	5.3
50–59	14	9	64.3
>60	16	12	75.0
Total	194	25	12.9

OPHTHALMOLOGICAL EXAMINATIONS

Table 16 shows the major ophthalmological findings. No major differences were found between the exposed and the unexposed groups except, as has been noted in the past, for a slightly greater incidence of pterygii, pingueculae, corneal scars, and pigmentation in the exposed group. As a whole both groups showed vision and accommodation levels above the average in the United States. The absence of glaucoma was also notable. The incidence of retinal arteriosclerosis, squints, and congenital diseases was very low. No cases of basal cell carcinoma of the eyelids or of retinitis pigmentosa were seen.

The degree of changes in accommodation, visual acuity, arcus senilis, and retinal arteriosclerosis have been plotted against age. The plots show no obvious differences between the exposed and unexposed populations. (See Figures 19 to 22 and further discussion in the section on aging.)

Slit-lamp examinations of the lens showed no opacities in the exposed Marshallese resembling those that had been noted in the irradiated Japanese.

ARTHRITIS SURVEY

1. Osteoarthritis

The incidence of osteoarthritis, as indicated by the hand and wrist x-ray survey, is shown, by age and sex, in Table 17. The small number of individuals in the susceptible age groups precludes an accurate estimation of the prevalence; however, it does not appear to be grossly different from that found in the United States white population studied by Dr. Alice Waterhouse and cited by Blumberg et al.[9] In accordance with general clinical experience, there was no close correlation between x-ray findings and clinical symptoms of arthritis, i.e., subjects with quite advanced osteoarthritis as determined by x-ray often had no complaints referable to their joints.

Table 18

Results of Bentonite Flocculation Test

Age, yr	No. in age group	BFT titer 0	¼	⅛	1/16	1/32	1/64	1/256
		Males						
0– 9	14	9	3	2	–	–	–	
10–19	24	21	3	–	–	–	–	
20–29	17	15	1	1	–	–	–	
30–39	9	9	–	–	–	–	–	
40–49	13	12	–	–	–	–	1	
50–59	11	9	–	1	–	–	1	
>60	11	9	2	–	–	–	–	
Total	99	84	9	4	0	0	2	
		Females						
0– 9	12	8	2	1	1	–	–	–
10–19	13	10	–	2	1	–	–	–
20–29	18	14	–	3	–	–	–	1
30–39	12	11	1	–	–	–	–	–
40–49	6	5	–	–	1	–	–	–
50–59	8	7	1	–	–	–	–	–
>60	16	11	2	1	2	–	–	–
Total	85	66	6	7	5	0	0	1

2. Rheumatoid Factor in Sera

The prevalence of sera positive for "rheumatoid factor" by the bentonite flocculation test (BFT) is shown in Table 18. Individual data are listed in Appendix 6 according to age and sex. The total prevalence of positive sera is not greatly different from that reported for white American populations. There was no apparent difference in arthritis incidence between the exposed and unexposed groups. Three individuals, all in the unexposed group, had definitely positive sera (Table 19), but with no evidence of joint disease. One (#953) had bronchial asthma and prostatic hypertrophy, but these findings, since they were common in the population, were probably coincidental. There is no apparent explanation for the presence of rheumatoid factor in these individuals.

3. Clinical Evidence of Arthritis

A 59-year-old male (#878) had some clinical findings compatible with rheumatoid arthritis. He had complained of pain and swelling in his hands, wrists, and knees for 5 to 10 years, but with no limitation of function. Physical examination showed enlargement of the proximal and distal interphalangeal joints and ulnar deviation of the hand. No rhuematoid nodules were present. The x-ray changes were consistent with a diagnosis of osteoarthritis of the hands, but there was no evidence for rheumatoid arthritis. The BFT was negative. This is probably a case of osteoarthritis, but rheumatoid arthritis cannot be completely ruled out.

4. Unusual Skeletal Findings

As noted previously, hand and wrist x-rays had shown congenital hypoplasia of the middle phalanx in some children. The same condition was noted in some of the adults (Figure 12). The middle phalanx of the fifth finger was shortened and slightly broadened. This was found both unilaterally and bilaterally and was occasionally associated with lengthening of the ulna. Twenty subjects showed definite changes, and some additional ones had only moderate shortening of the phalanx.

DENTAL SURVEY

Majuro Children

Thirty Majuro children were examined, ranging in age from 2 to 13 years and selected at random. Widespread caries were present in 75% of the children <12 years old. Many erupting permanent teeth showed retained root fragments in the adjacent gingival crevice. Although the oral hygiene could be rated fair to good, the incidence of caries remained high. Bunching of the lower incisors was a common finding in males and females in the 7 to 9-year age group. Eruption time of the bicuspids appeared advanced, possibly because of premature loss of the deciduous molars. All the children in this age group showed signs of retarded jaw development in comparison with children in the continental United States.

Rongelap Children

No significant difference was found in caries incidence, eruption time, and morphology of the teeth, or growth of the jaws, between the exposed and the unexposed children. In both groups about 20% showed retarded jaw development which had resulted in narrowing of the arches plus bunching of the lower anterior teeth. The Rongelap children had a much lower incidence of caries than

Table 19

Subjects With Positive Bentonite Flocculation Test

Subject No.	Age	Sex	BFT titer	X-ray evidence of arthritis	Clinical findings
841	41	F	1/256	None	Neg. in 1957, '58, and '59.
886	49	M	1/64	Not tested	Mild osteoarthritis in 1959.
953	42	M	1/64	None	No joint findings; bronchial asthma; 2+ prostatic hypertrophy.

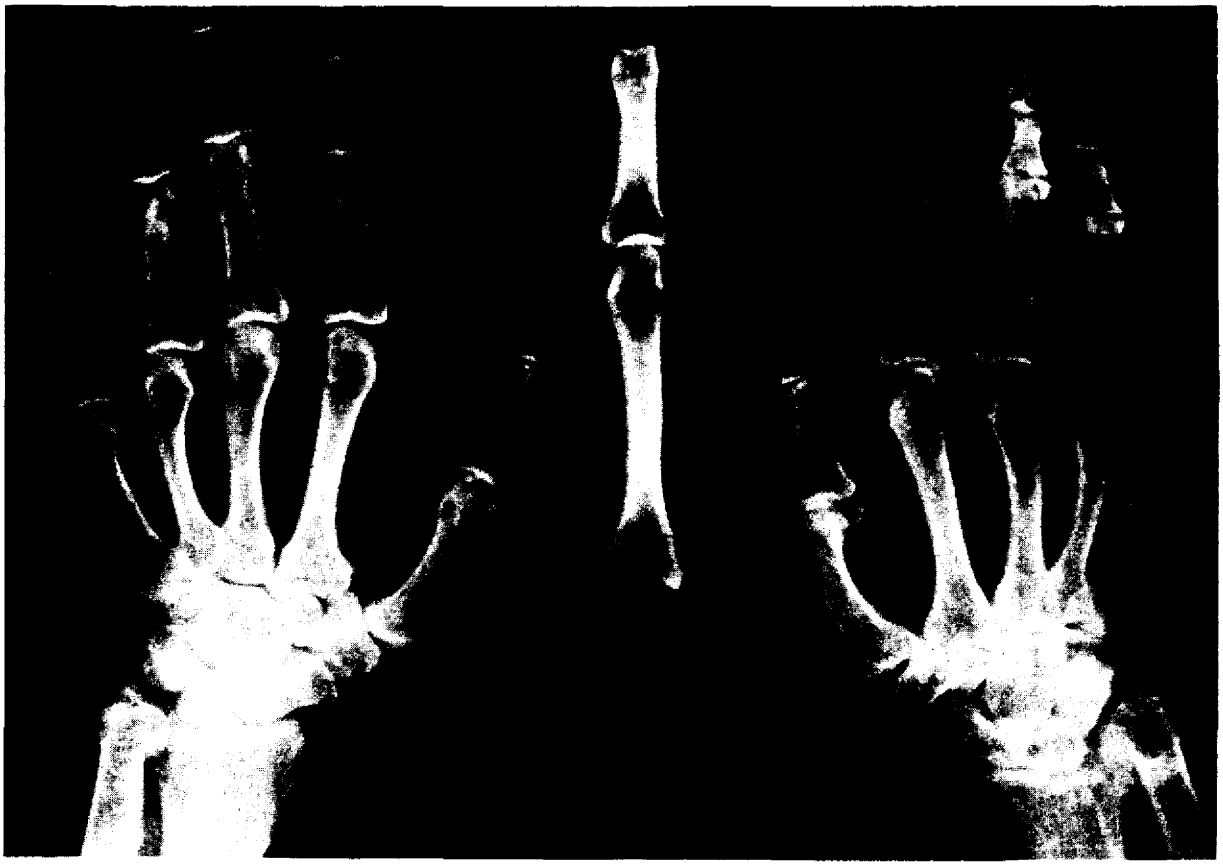

Figure 12. Roentgenograph of hands showing congenital hypoplasia of the middle phalanx, 5th finger.

the Majuro group, although the general impression was that their standards of oral hygiene were lower. In both the exposed and unexposed Rongelap children, areas of hypoplastic enamel in deciduous teeth were not an unusual finding.

Adults

Only about 10% of the Rongelap people practiced good oral hygiene. Peridontal disorders may have been slightly higher in the exposed group, although there was no significant difference in the incidence of caries. In young adults, subgingival calculus was usually present in the form of cervical ringlets accompanied by marginal gingivitis, and very little supragingival deposition was observed. In subjects >35 years of age, loss of alveolar bone was quite apparent, and supragingival calculus was more prevalent than subgingival. Only 2% of

those >35, exposed and unexposed, were free of clinically detectable peridontal disease. In the aged, many of the remaining teeth were simply held in position by the gingival attachment.

Almost all the Rongelapese exhibited marked xerostomia. The oral mucosa felt damp but not wet. During the dental examination, manipulation of the tissues by mouth mirror and explorer failed to stimulate the flow of saliva. For *p*H readings, the paper strip had to remain in contact with the floor of the mouth for 25 to 30 sec in order to absorb sufficient saliva. In most instances it was difficult for the subject to produce the 5 ml of saliva necessary for *p*H determination in a period <10 to 15 min. The *p*H values ranged from 6.0 to 7.5, with an average of 6.3

Utirik Population

The Utirik population exhibited oral findings similar to those of the Rongelapese. Their standards of oral hygiene were somewhat superior, but the incidence of peridontal disease and caries in adults was practically identical. In the Utirik children less evidence was seen of bunching of the lower anterior teeth, and jaw development was in harmony with general body growth.

Conclusions

These examinations led to the following conclusions.

1. There was no significant difference in either caries rate or incidence of peridontal disease between the exposed and the unexposed people of Rongelap. The level of oral hygiene appeared to be somewhat better in the children examined at Majuro and at Utirik, but the incidence of caries was about the same in the two groups, and higher than in the Rongelapese children.

2. The poor oral hygiene generally observed in the Marshallese people had its usual results: a high caries rate in teen-age children; severe peridontal lesions in adults (heavy calculus, loss of alveolar bone); and edentulous mouths in the aged.

3. No difference was perceptible in the clinical appearance of developing dentition among the exposed children, the nonexposed, and those born to irradiated parents.

STUDIES OF AGING CRITERIA

The results of aging criteria studies are plotted in Figures 13 to 31. Individual readings are plotted (open circles, exposed people; closed circles, unexposed people) to show the spread of the data, and the mean values are also plotted (squares) for each 5-year age group with exposed and unexposed people combined. The trend of each criterion with increasing age is represented by a line of approximate best fit by eye. The values of the criteria generally either increase or decrease with increasing age. Many changes, such as greying of the hair, balding, arteriosclerosis (peripheral and retinal), and skin looseness and retraction, are not appeciably manifest in the Marshallese until after age 35 to 40. There is a tendency for values of criteria such as blood pressures, greying of the hair, loss of visual acuity, and accommodation to level off, or even to show slightly reduced values in the oldest groups. The number of people in these groups is too small to make this observation certain. However, American statistics on blood pressure (systolic and diastolic) also show a plateau effect beyond about 60 years of age and even a slight tendency to lowering of blood pressures beyond this age.[26] By the age of 65 to 70, arcus senilis in all Marshallese showed a 4+ change and, therefore, the plateau was at a maximum. Some curves showed lower maxima. For example, baldness in women showed only a 35% maximum, and arteriosclerosis of the retina only 45%. The differences in maxima probably were due largely to differences in the degree of change associated with the various criteria but also partly to the arbitrary nature of the scoring. The sexual differences in hand strength and baldness were expected. In the neuromuscular function test, slight muscle fatigue may have been a factor in the lower female scores, necessitating separate evaluation.

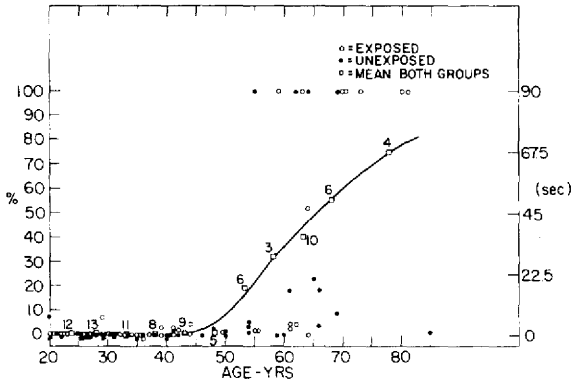

Figure 13. Skin retraction time, 1958-1959.

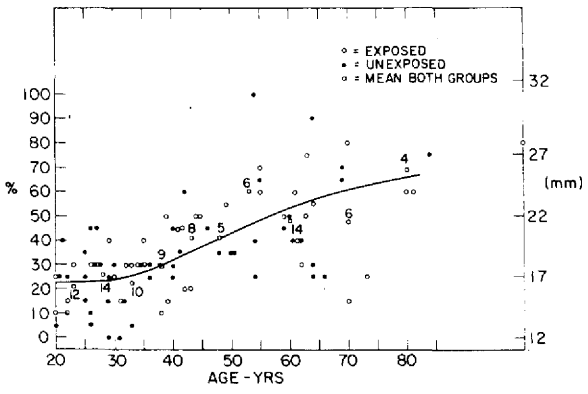

Figure 14. Skin looseness, 1958-1959.

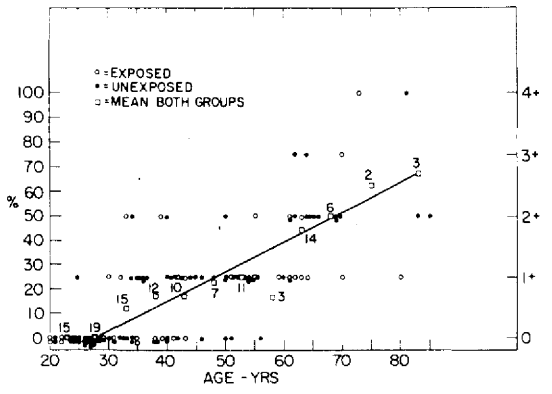

Figure 15. Senile changes of skin, 1959.

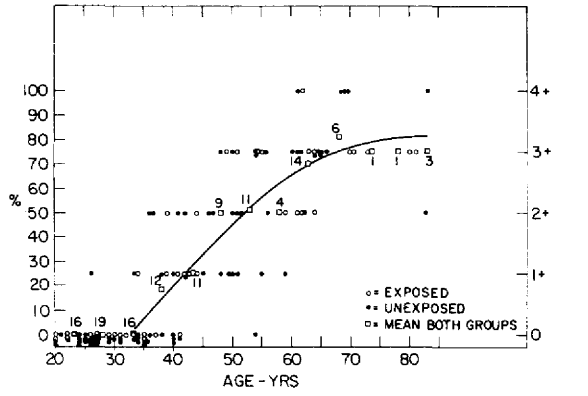

Figure 16. Grayness of hair, 1959.

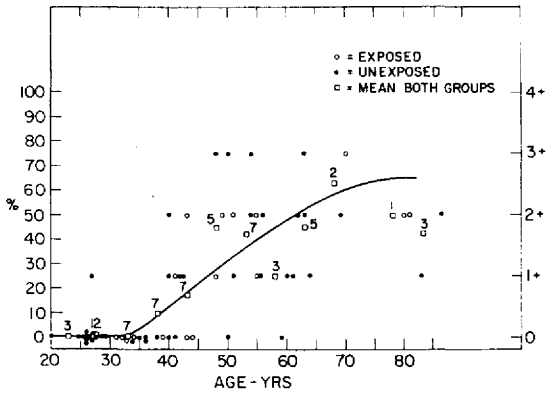

Figure 17. Baldness in males, 1959.

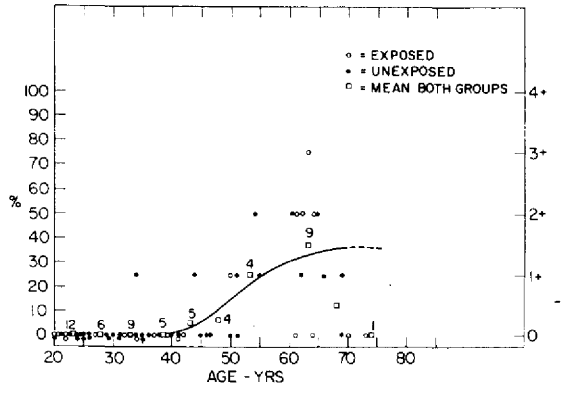

Figure 18. Baldness in females, 1959.

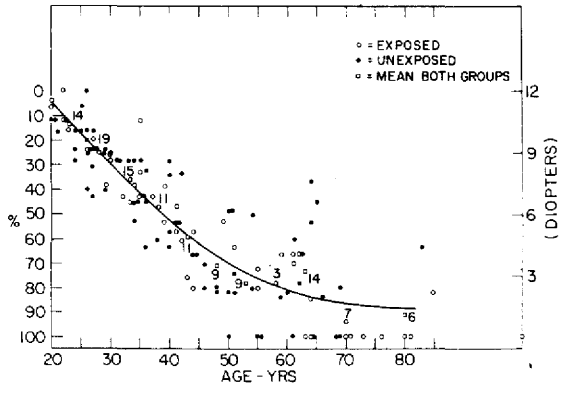

Figure 19. Accomodation, 1959.

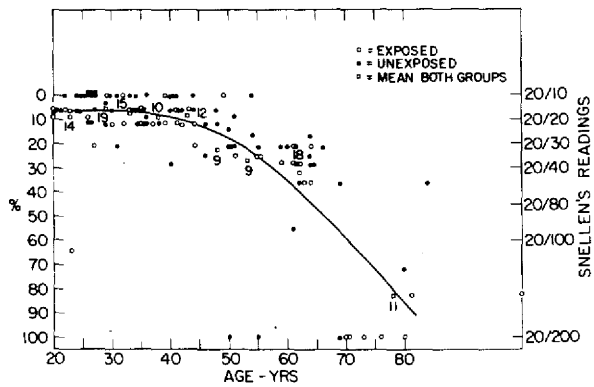

Figure 20. Visual acuity, 1959.

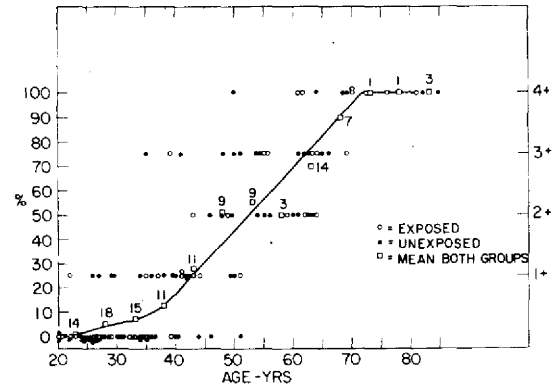

Figure 21. Arcus senilis, 1959.

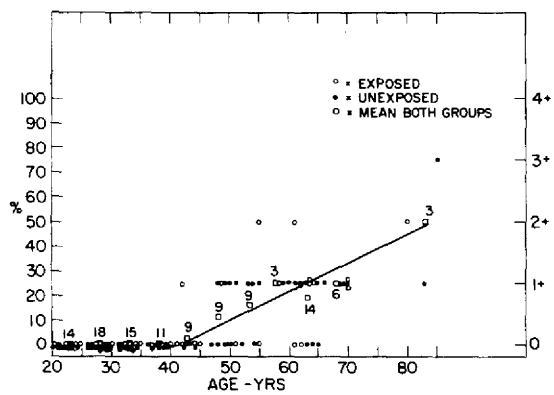

Figure 22. Arteriosclerosis of retina, 1959.

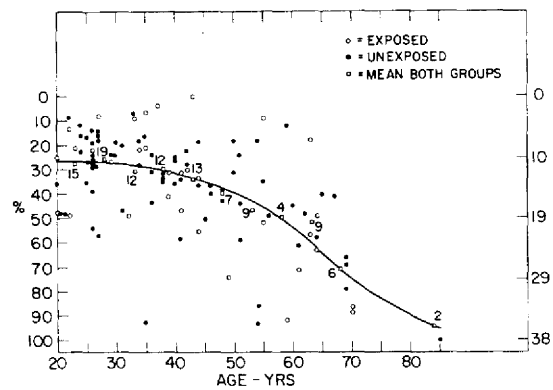

Figure 23. Hearing loss, 1959.

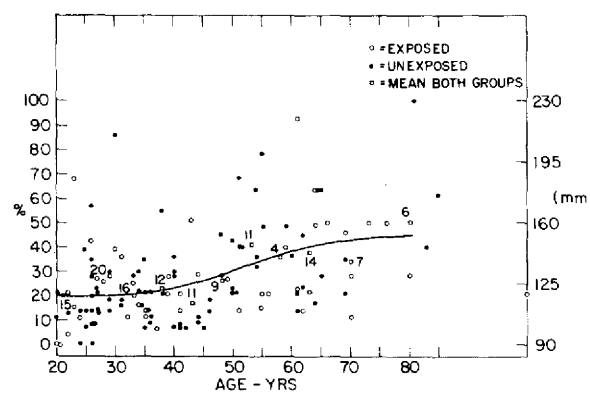

Figure 24. Blood pressure, systolic, 1959.

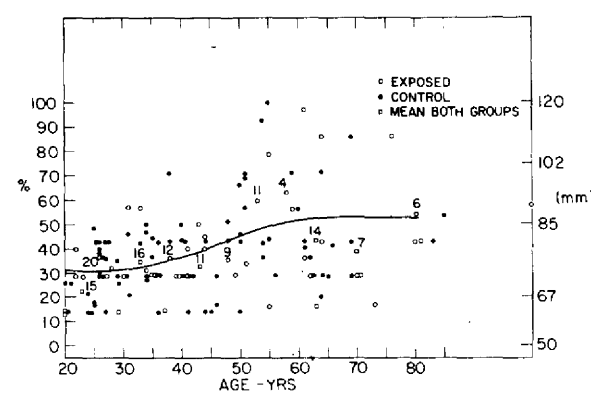

Figure 25. Blood pressure, diastolic, 1959.

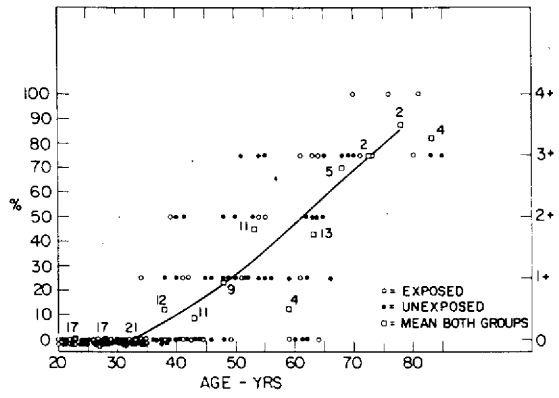

Figure 26. Peripheral arteriosclerosis, 1959.

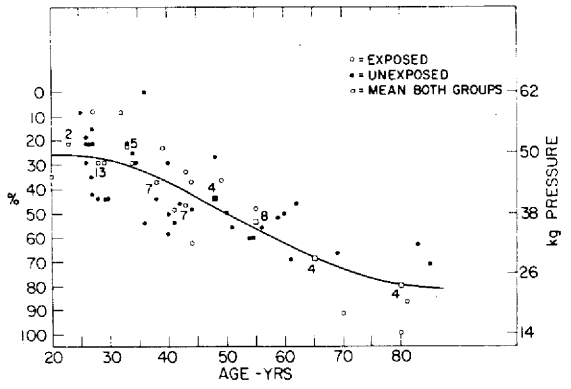

Figure 27. Hand grip, males, 1959.

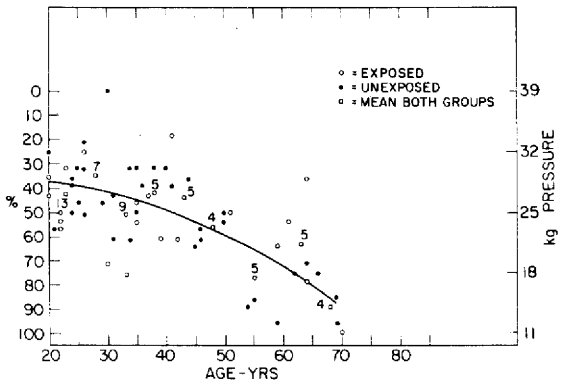

Figure 28. Hand grip, females, 1959.

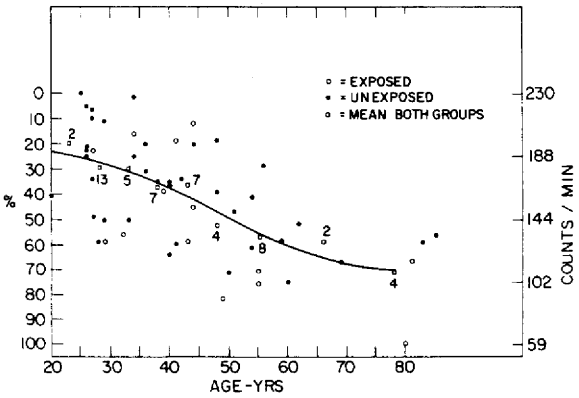

Figure 29. Neuromuscular function (hand tally count), males, 1959.

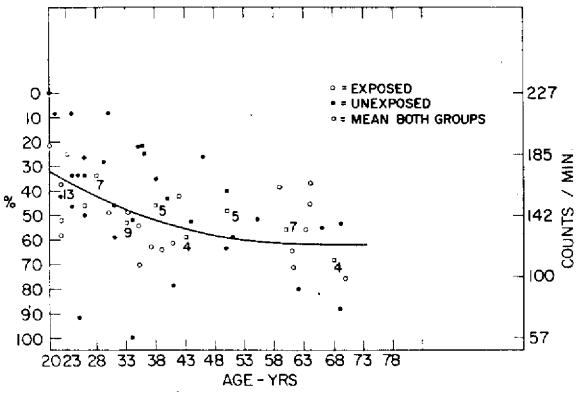

Figure 30. Neuromuscular function (hand tally count), females, 1959.

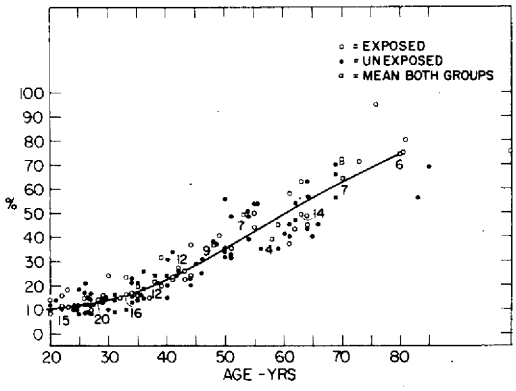

Figure 31. Average age scores, 1959.

The various changes generally associated with physiological senescence are known to show wide variability among individuals of the same age, and this was borne out by these data. However, some of the criteria appeared to be better indices of aging than others, judging by the degree of change and the variability of the data observed. (This statement is not based on statistical analysis.) Some of the better criteria appeared to be accommodation of the eyes, visual acuity, skin retraction, arcus senilis, greying of the hair, and hand strength. The measured criteria would be expected to be more reliable than the estimated ones in view of the subjective element and less precise scoring of the latter. This appeared generally to be the case, but the estimated values for greying of the hair and arcus senilis correlated surprisingly well with age. The amount of subcutaneous fat probably influenced measurements of skin looseness, but, since loss of subcutaneous fat is somewhat age dependent, probably in the right direction. It is hoped that in the future statistical treatment of the data may furnish a weighting factor for each criterion based on the degree of age-associated change and the variability of the data. It is not always possible to run the complete battery of tests on each individual, and use of such weighting factors would tend to minimize the disparity due to the omissions.

The data presented must be considered as preliminary in nature and to represent only a small fraction of the many varied changes associated with the aging process. Under the conditions of examinations of the Marshallese, the battery of tests employed is necessarily limited. As more experience is gained, some of the tests may be eliminated and new ones added. The tests of vigor are thought to be extremely important in assessing aging, and it is hoped that more tests of this nature may be added. At this time the data are presented to introduce an approach to a feasible means of assaying "biological age" which might be of use in studying the possible effects of radiation on the aging phenomenon in human beings. In the case of the exposed Marshallese, results of further aging surveys will be carefully evaluated, and the results

Table 20

Residual Beta Burns, 1960

Subject No.	Sex	Age	Description
17	F	9	Slight depigmented scars, left antecubital fossae.
24	F	19	Mottled spots of pigmentation and depigmentation, dorsum of feet.
26	M	18	Marked scarring between first and second toes, right foot, with binding to subcutaneous tissues. Areas of lesser involvement peripheral to this area. No evidence of chronic radiation dermatitis.
39	F	20	Slight mottled pigmentation-depigmentation with little scarring, dorsum of feet. Mottled hyperpigmented spots persist on back of neck.
49	F	21	Black moles believed to have increased in number over right side of neck, also several noted in left antecubital fossa.
59	F	40	Mottled roughening and pigment variation, back of neck.
63	F	42	Mottled pigmentation, left side of neck, with slight roughening of skin.
67	F	20	Depigmented scarred areas with slight atrophy of skin over dorsum of feet, particularly at site of deeper scar on left foot. Some areas adjacent to scars show increased pigmentation.
78	F	43	Pigmented moles appear to have increased in number over left side of neck and to lesser extent on right side at sites of rather deep beta burns.
79	M	45	Back of left ear shows depigmented scar with some binding down to cartilage. No evidence of breakdown.

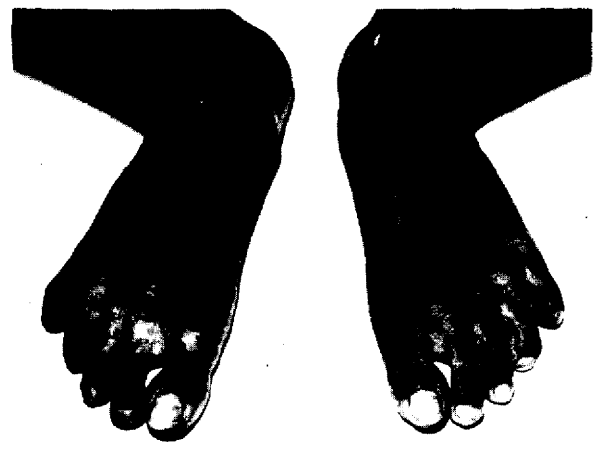

Figure 32. Residual scarring between first and second toes, right foot, from beta burns, 5 years post exposure (Subject #26).

in the exposed population will be compared with those in the unexposed for differences that might indicate possible premature aging effects. As pointed out, such differences have not been apparent thus far.

RESIDUAL BETA BURNS

Ten people continued to show residual skin damage from beta burns sustained 6 years previously. Most of the residua consisted of very mild changes, such as varying degrees of pigment alteration giving a mottled appearance, sometimes accompanied by a roughening of the skin (increased rugosity). Some showed more pronounced changes, such as atrophy and scarring. None of the more severe residual lesions showed any gross evidence of breakdown with the development of chronic radiation dermatitis or any premalignant or malignant change. No biopsies were taken. Several women who had sustained more severe neck lesions seemed to be showing an increase in pigmented moles in and around the affected areas. However, this is not certain, and these moles will be observed carefully in the future for any suspicious changes. In Table 20 are listed descriptions of the residual beta burns seen in 1960. Figure 32 shows healed beta burns of the feet 5 years after exposure.

LABORATORY EXAMINATIONS

Hematological

In Appendices 1 and 2 are presented summaries of the mean blood counts of the exposed population and various comparison populations since exposure in March 1954. In Appendices 3 and 4 are listed the individual blood counts for 1959 and 1960. The data are also summarized graphically in Figures 33, 35, and 38. The blood data have been classified as in the past according to age and sex. The results of blood counts in the 1959 and 1960 surveys may be summarized as follows.

White Blood Counts. The mean WBC in 1959 showed a further trend upward in both the exposed and unexposed populations, being slightly lower in the former (see Table 21 and Figure 33). The 1960 WBC level of the exposed people showed a considerable drop from a mean level of 9500 in 1959 to 6500 in 1960 as compared with the trend during the previous two years (no counts were made on unexposed people in 1960). The explanation for this is not clear. The percentage distribution of the various white cell levels was not very different, however, from that of the previous values.

Neutrophils. The neutrophils in 1959 (Table 21 and Figure 33) showed an increase corresponding to the increase in WBC in both exposed and unexposed populations, the mean level also being slightly lower in the exposed. A scattergram (Figure 34) of the age distribution of the counts in 1959 shows a greater tendency for the younger groups to have lower mean counts in the exposed than in the unexposed populations. This was noted also in 1958. In 1960 the average of neutrophil counts was 3500 for the exposed group compared with 4800 in 1959. This decrease corresponded with the decrease in the total white count.

Table 21

Mean Leukocyte and Platelet Counts, 1959 and 1960, by Age and Sex

	Rongelap exposed		Ailingnae exposed		Unexposed
	1959	1960	1959	1960	1959
WBC ($\times 10^{-3}$), age >5	9.5± 2.3* (60)**	6.5±1.8 (54)	9.7±2.4 (15)	7.3±4.3 (14)	10.1± 3.1 (123)
Neut. ($\times 10^{-3}$), age >5	4.8± 1.9 (60)	3.5±1.5 (54)	5.1±2.1 (15)	3.6±1.6 (13)	5.2± 2.1 (123)
Lymph. ($\times 10^{-3}$), age >5	4.0± 1.3 (60)	2.7±0.8 (54)	3.7±1.1 (15)	3.0±1.2 (13)	4.1± 1.4 (123)
Mono. ($\times 10^{-2}$), age >5	2.0± 1.8 (60)	0.6±0.2 (54)	3.2±2.5 (15)	0.6±0.6 (13)	2.4± 2.3 (123)
Eosin. ($\times 10^{-2}$), age >5	5.0± 3.3 (60)	2.7±0.8 (54)	6.0±4.2 (15)	4.0±0.2 (13)	6.0± 9.9 (123)
Baso. ($\times 10^{-2}$), age >5	0.4± 0.6 (60)	0.4±0.11 (54)	0.5±0.8 (15)	0.4±0.4 (13)	0.5± 0.8 (123)
Plate. ($\times 10^{-4}$), Males age 5–10	32.3±10.0 (7)	–	40.9 (2)	–	34.6±10.8 (11)
>10	24.4± 9.9 (20)	–	26.3±6.0 (4)	–	28.0± 8.4 (54)
Females >5	27.6±10.5 (29)	–	26.8±3.7 (9)	–	31.1± 7.2 (52)

*Standard deviation.
**The numbers in parentheses are the numbers of people in the groups.

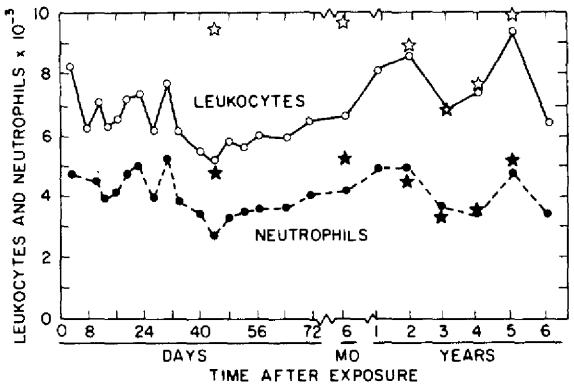

Figure 33. Mean neutrophil and white blood cell counts of exposed Rongelap people from exposure through 6 years post exposure. Stars represent mean values of comparison population.

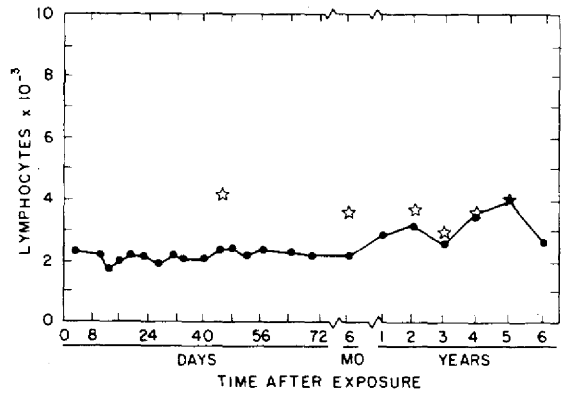

Figure 35. Mean lymphocyte values of exposed Rongelap people from exposure through 6 years post exposure. Stars represent mean values of comparison population.

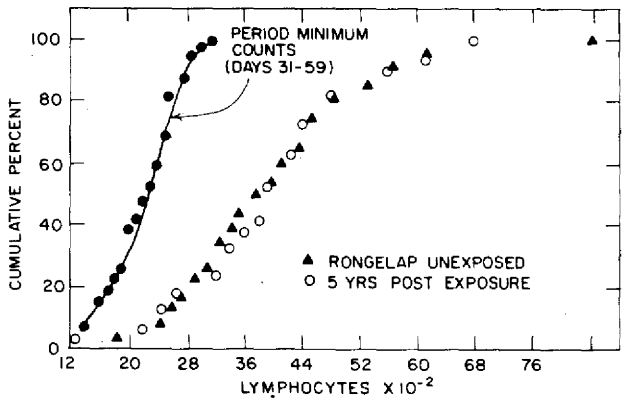

Figure 37. Cumulative distribution curve, Rongelap lymphocytes, 1959.

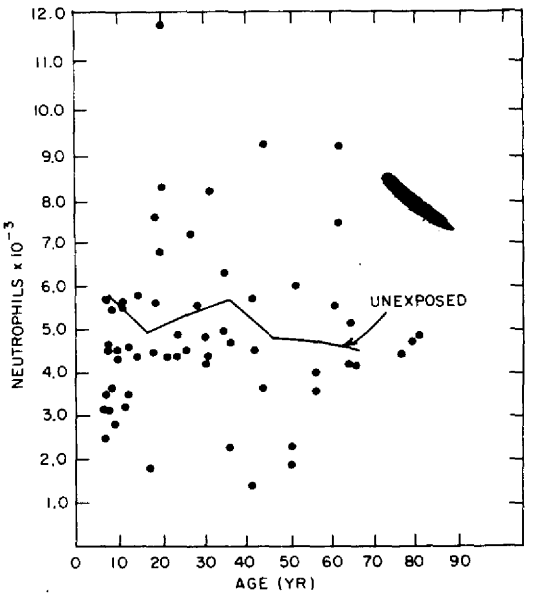

Figure 34. Neutrophil counts of exposed Rongelap individuals plotted against age at 5 years post exposure, 1959. Solid line represents mean level of comparison population.

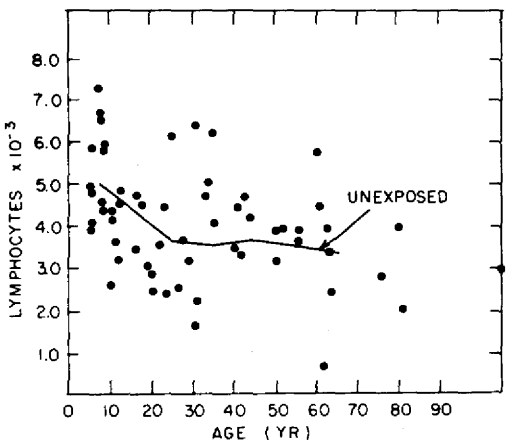

Figure 36. Lymphocyte counts of exposed individuals plotted against age at 5 years post exposure, 1959. Solid line represents mean counts of comparison population.

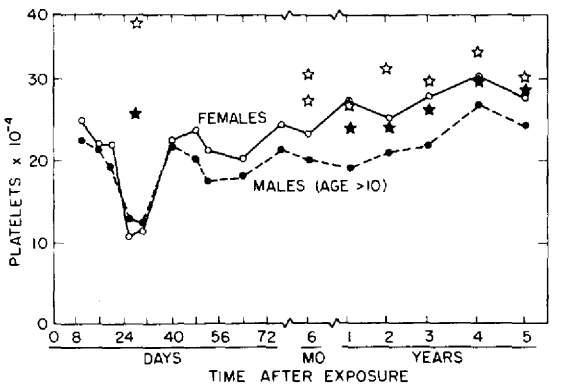

Figure 38. Mean platelet values of exposed Rongelap people from exposure through 5 years post exposure. Stars represent mean values of comparison population.

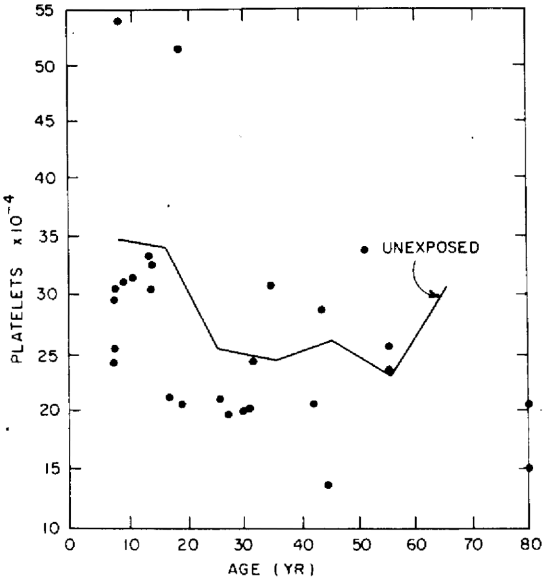

Figure 39. Individual platelet counts of exposed males plotted against age, 1959. Solid line represents mean level of comparison male population.

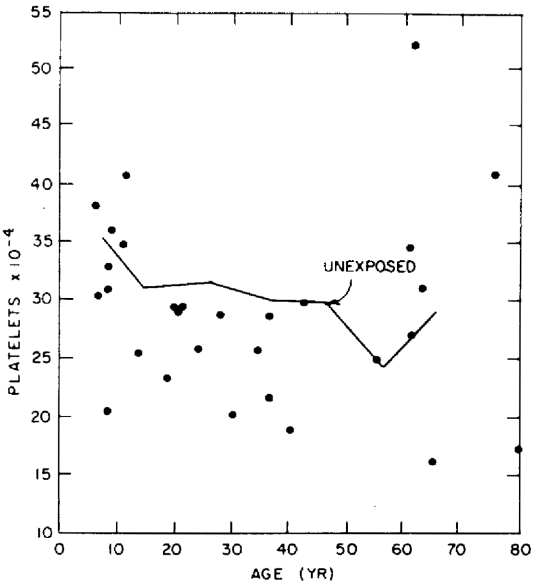

Figure 40. Individual platelet counts of exposed females plotted against age, 1959. Solid line represents mean level of comparison female population.

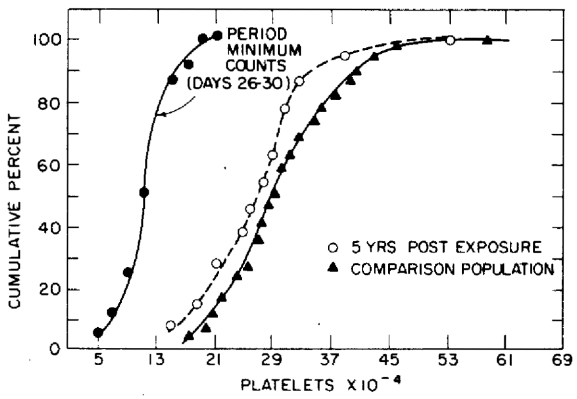

Figure 41. Cumulative distribution curve, Rongelap platelets, 1959.

Lymphocytes. The mean level of the lymphocyte counts (Table 21 and Figure 35) in 1959 showed a slight increase over the 1958 values. The mean levels were about the same in the exposed and unexposed populations. A scattergram (Figure 36) of the age distribution of the lymphocyte counts in the exposed group for the first time showed about an equal distribution in the exposed population of counts above and below the mean levels of the unexposed people. The cumulative percentage distribution curves of the exposed and unexposed (Figure 37) showed close approximation. The 1960 absolute lymphocyte counts also showed a decrease corresponding to the WBC decrease, the mean dropping from 4000 in 1959 to 2700 in 1960.

Eosinophils and Monocytes. Eosinophil and monocyte counts showed a slight increase in 1959 over the 1958 levels and were slightly greater in the exposed population. As noted in 1958, a large percentage of the population had eosinophil counts >5% of the total white count (1959, 44% of exposed population and 39% of unexposed; 1960, 46% of exposed, no data on unexposed). The levels of eosinophils and monocytes in 1960 were not very different from the 1959 levels. (Basophils are discussed below in connection with leukemia.)

Platelets. Mean platelet counts in 1959 (Table 21 and Figure 38) were slightly lower than in 1958 in both the exposed and unexposed populations. The mean deficit in platelets in the exposed population was about the same as last year (−9.3% for the males and −11.3% for the females). Age distribution scattergrams for the individual platelet counts in both males and females of the exposed population showed more counts below than above the unexposed mean curve (Figures 39 and 40). This was also borne out by comparison of the cumulative percentage distribution curves for the exposed and unexposed populations: the latter showed continued displacement to the left (Figure 41). The significance of the continued platelet depression in the exposed population is also indicated by the finding of levels <250,000 in 37% of the exposed group but in only 24% of the unexposed.

Erythropoetic Function. Because of technical difficulties, the hematocrit levels were not considered reliable for the 1959 survey. Samples containing ethylenediaminetetraacetic acid as an anticoagulant appeared to have a lower hematocrit than untreated venous or finger stick blood.

Table 22
Mean RBC, Hemoglobin, MCH, and Reticulocyte Levels by Age and Sex, 1959

		Rongelap exposed	Ailingnae exposed	Rongelap control
RBC ($\times 10^{-6}$),	Males age 5–15	4.45±0.5* (10) **	4.46 (2)	4.6 ±0.5 (22)
	>15	4.71±0.4 (19)	5.15 (4)	4.8 ±0.5 (44)
	Females >5	4.21±0.4 (31)	4.31±0.36 (9)	4.4 ±0.4 (53)
Hgb., g,	Males age 5–15	12.3 ±0.5 (10)	12.2 (2)	13.1 ±1.1 (22)
	>15	14.4 ±1.3 (19)	15.4 (4)	14.8 ±1.8 (44)
	Females >5	12.5 ±0.95 (31)	13.2 ±0.87 (9)	12.9 ±2.1 (52)
MCH, μμg,	Males age 5–15	28.0 ±3.3 (10)	27.7 (2)	28.5 ±1.9 (22)
	>15	30.8 ±2.5 (19)	30.0 (4)	30.7 ±3.4 (44)
	Females >5	29.8 ±3.3 (31)	30.5 ±1.9 (9)	29.4 ±2.7 (52)
Retic., %,	Males age 5–15	0.13 (9)	0.05 (2)	0.29 (17)
	>15	0.31 (15)	0.3 (4)	0.43 (42)
	Females >5	0.21 (23)	0.17 (7)	0.41 (48)

*Standard deviation.
**The numbers in parenthese are the numbers of people in the groups.

The explanation is not clear. However, it was felt that the erythrocyte counts done on the Coulter electronic counter, and the hemoglobin and reticulocyte counts, were sufficient indices for evaluation of erythropoetic function.

Erythrocytes and Hemoglobins. The mean levels of erythrocytes and hemoglobins (Table 22) were generally somewhat below those accepted as average for Americans and were only slightly lower in the exposed Rongelap population than in the unexposed group. Scattergrams (Figures 42 and 43) of the individual red cell counts plotted by age for exposed people of both sexes show more of the counts below the average level of the unexposed group than above it, and a plot of percentage cumulative distribution of counts (Figure 44) shows the curve for the exposed group distinctly displaced to the left.

Ailingnae Blood Counts. Counts in the Ailingnae people (a group of 15 who had received an estimated 69 r from fallout) are summarized in Tables 21 and 22 and in Appendix 2, and the individual counts are shown in Appendices 3 and 4. These counts generally averaged slightly higher than in the exposed Rongelap people but lower than in the unexposed people.

Price-Jones Curves. Price-Jones curves for determination of red cell size distribution on 17 Rongelap people (exposed and unexposed) were averaged and compared with an averaged curve for 53 Americans of the same age group (Figure 45). The Marshallese curve is displaced slightly to the left, which indicates a slight tendency toward microcytosis.

Serum Iron. Serum iron levels were <100 μg/100 ml serum in only 5 persons, all unexposed.

Serum Protein. Serum protein levels in 1959 were again higher than normal in many cases. The range in the exposed group was 6.6 to 8.4 g with a mean of 7.45, and in the unexposed group from 6.6 to 9.0 g with a mean of 7.55.

Comments. The peripheral levels of blood elements have shown considerable fluctuation from year to year. The explanation is not known. One might speculate that, since upper respiratory and gastrointestinal infections are common, the temporal relationship of the hematological examinations to periods of bacterial infection might strongly influence the general level of certain elements, particularly the leukocytes. It is not known whether the drop in 1960 leukocyte levels was so influenced. As pointed out, the influenza epidemic in early 1960 apparently spared Rongelap Atoll. Since hematological examinations were not done on the unexposed group in 1960, it was not possible to evaluate exposed levels with relation to radiation effects.

The only blood elements at 5 years post exposure that showed slightly lower levels in the exposed group were the platelets and erythrocytes. Erythrocyte counts had not been done before 1959. Re-examination of earlier hematocrit levels by construction of scattergrams and cumulative distribution curves showed a slight tendency for

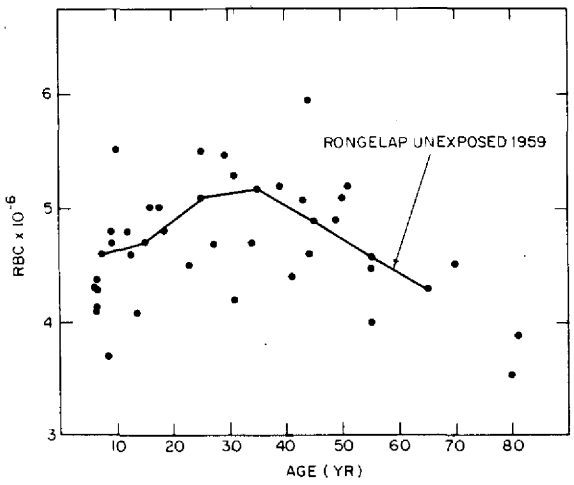

Figure 42. Individual red blood counts of exposed males plotted against age at 5 years post exposure, 1959. Solid line represents mean values for comparison male population.

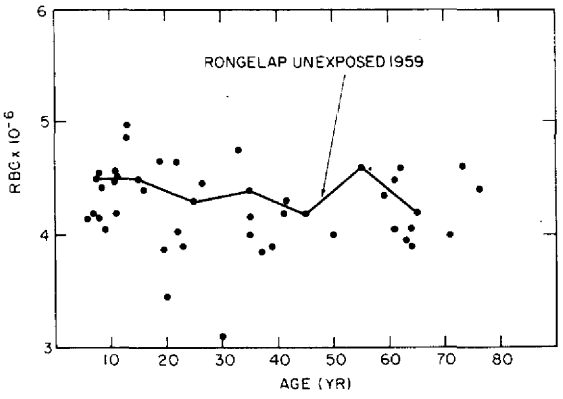

Figure 43. Individual red blood counts of exposed females plotted against age at 5 years post exposure, 1959. Solid line represents mean values for comparison female population.

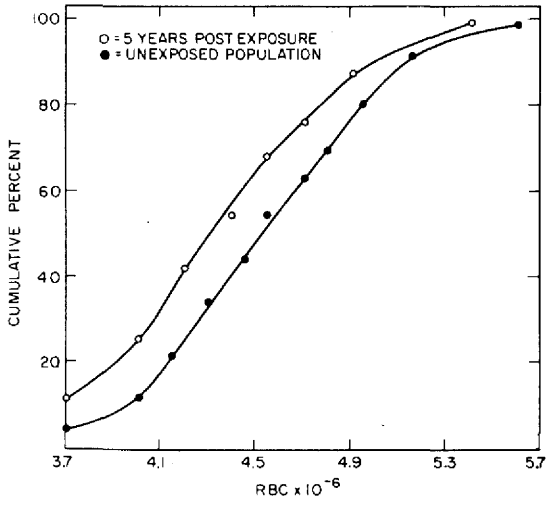

Figure 44. Cumulative distribution curve, Rongelap red blood counts, 1959.

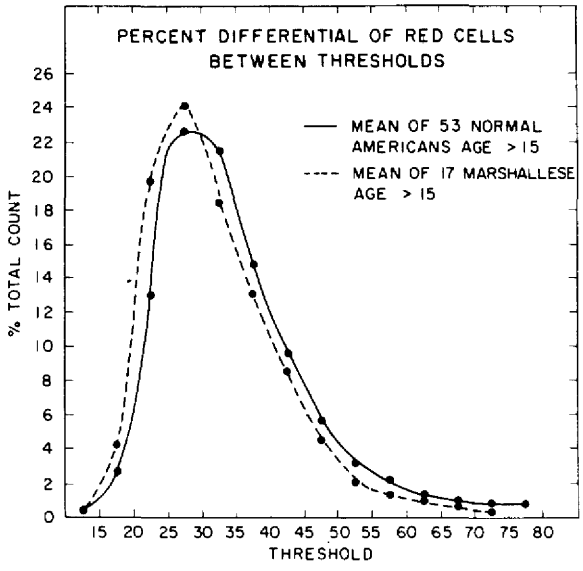

Figure 45. Price-Jones sizing of red cells showing mean of 17 Marshallese curves (from both exposed and comparison groups) compared with mean curve of 53 Americans.

erythropoetic depression compared with the unexposed levels, but not as distinct as indicated by erythrocyte counts in 1959. It would appear from these findings that some bone marrow damage persists at 5 years post exposure.

The Rongelap people generally have a slight anemic tendency (very slightly microcytic) compared with Americans. The cause is unknown. Apparently it is not due to iron deficiency, since serum iron levels are generally normal. It may possibly be based on another type of nutritional deficiency. The serum proteins and serum vitamin B_{12} levels, however, tended to the high.

The continued high level of eosinophils is unexplained. It was not believed that the types of intestinal parasites noted on a previous survey could account for it.

Hematological Leukemia Survey

Differential counts showed no increase in immature leukocytes that would cause one to suspect leukemia in either the 1959 or 1960 survey. *Alkaline*

phosphatase staining of neutrophils showed a generally normal distribution of positive cells, as in the previous year. *Basophil counts of 4000 cells* showed no increase in levels above the normal percentage in any subjects, either exposed or unexposed, in the 1959 and 1960 surveys, except for one 6-year-old exposed boy (#3).* The mean percent basophil count (counting 4000 white cells) for the exposed Rongelap group was 0.42% in 1959 and 0.93% in 1960; in the Ailingnae group, 0.29% in 1959 and 0.80% in 1960. Basophil counts on the unexposed group are not yet complete for 1959. The individual values for alkaline phosphatase and basophil counts are presented in Appendix 5.

Complement Fixation Tests

The results of the complement fixation tests are shown in Table 23. In discussing these results Dr. R. J. Heubner made the following comments:

"I suppose the most interesting thing is that there is evidence of infection with almost all the groups of viruses that we tested for. It is important to point out that complement-fixing antibodies are generally less persistent than are hemagglutination inhibition and neutralizing antibodies. Therefore, the high levels of adenovirus and HA 1 complement-fixing antibodies reflect either recent infection or multiple infections with various members of the parainfluenza family of viruses. The low levels obtained for other agents, such as influenza A, influenza B, HA 2, CA, mumps, LGV, and Q fever may reflect low levels of infection with these agents, but more likely might reflect the fact that such agents have not been prevalent in recent years. Thus, one might conclude from the data that the Asian influenza virus has not yet seriously involved the Marshall Island populations, or else, of course, that the complement-fixing antibody produced by the Asian influenza has not persisted.

"I would rather suspect from the age distribution that Asian influenza still has not reached these people, and that one might look forward to see if they are involved at some future date. Similarly, the age distribution of influenza B antibodies suggests that it has not occurred for at least 6 to 10 years. The absence of HA 2 antibodies suggests that the virus has not been prevalent in recent years. However, the total absence of antibodies even in persons over 40 suggests the possibility that it has not occurred in this population at all, but the first interpretation is equally good. The same comments would refer to the CA virus or parainfluenza 2, but here the more likely interpretation is that the virus has not been present in the recent past, but the presence of antibodies may suggest that it had been present 16 or more years earlier. Since there is some overlap in antigens and antibodies between mumps and the parainfluenza group, the CA reactions and/or the mumps reactions could conceivably merely reflect infections with other members of the parainfluenza group.

"It would be interesting to find out to what extent the psittacosis group and Q fever represent a threat to this population, and of course in the birds and domestic animals as well."

Dr. Heubner's forecast of Asian influenza in the Marshall Islands from the Rongelap data is interesting in that an epidemic of serious proportions did occur at Kwajalein Atoll in January 1960 resulting in significant mortality.

The average antibody titers for the different age groups in the exposed people were, in nearly all cases, lower than in the corresponding unexposed age groups. It is not known whether this is a significant finding; further studies will be done to determine this. At three years after exposure, the primary response to tetanus antitoxin averaged slightly less in the exposed people than in the unexposed.[5] However, because of the small numbers of people studied, this finding could not be considered significant.

Sodium and Potassium Levels, 1959

Potassium intake appeared to approximate that of Americans, namely, ≃40 to 100 mEq/day. Salt intake varied widely as judged by urine excretion, ranging from 2 to 18 g/day. The relatively high salt consumption of some individuals is undoubtedly related to the irregular consumption of canned C rations, to which 2% NaCl is added routinely during preparation. The median intake of salt appeared to approximate that of male employees at Brookhaven National Laboratory, namely, ≃10 g/day. This undoubtedly represents an increase over the intake in earlier years when only native foods were eaten. It will be of interest to note whether the incidence of hypertension also will begin to increase. At present the prevalence of hypertension (B.P. greater than 140/90) is 9.2%

*In 1959 his basophil count was 2.1%, WBC 8600, and alkaline phosphatase negative cells 79%. In 1960 his basophil count was 3%, WBC 6100, and alkaline phosphatase negative cells 70%. No abnormal forms were seen.

for all age groups, a relatively low figure compared with that for Japanese and American societies.

Thyroid Metabolism

Table 24 shows results of protein bound iodine, total iodine, and butanol extractable iodine determinations. The 12 samples obtained in 1959 showed values definitely lower than the 1958 values, although several readings were still somewhat high. It was thought that the high 1958 levels must have been due to contaminated glassware in spite of the great care taken to maintain strict cleanliness.

Glucosuria

On routine urine analysis during the 1959 survey one male and four females (all in the unexposed group) were found to have glucosuria; all but one of these also had an elevated fasting blood sugar level* (see Table 25). These individuals were considered to have mild diabetes mellitus. Later during the year one of these cases (#854, F, age 54) died of a genitourinary infection complicating diabetes even though she (along with the other cases) had been placed on oral therapy with Orinase (tolbutamid tablets). (It was later found that these patients did not take the tablets consistently.)

Table 26 compares the mortality from diabetes in the United States[27] and the Marshall Islands. The incidence appears higher in the Marshall Islands and on Rongelap Island (based on its small population). It should be noted that, since only routine urine examinations were done on the Rongelap people, the incidence of diabetes may actually be higher. The greater incidence of deaths due to diabetes in the Marshall Islands than in the United States in 1956 and 1957[27] is statistically significant ($0.01 > p > 0.001$).

Serum Vitamin B_{12}

Analysis of the 15 serum samples obtained in 1959 again showed, as in 1958, vitamin B_{12} levels slightly higher than normal. The mean value was 714 $\mu\mu$g/ml, with a range of 312 to 1500. A scattergram of levels for both years was plotted against white blood count, since it has been shown that in leukemia the white cells have a higher B_{12} binding

*We are grateful to Dr. W.W. Fennel at Kwajalein for having the blood sugar analyzed.

Table 23

Results of Complement Fixation Tests on Rongelap People, 1959, by Age and Sex
(Positive Reactions of Serum Dilutions 1-10)

Age, yr	No. in age group	Flu A		Flu B		HA 1 (Parainfluenza 3)		HA 2 (Parainfluenza 1)		CA (Parainfluenza 2)		Long (Respiratory syncitial, CCA)		Adeno		Mumps		LGV (Psittacosis group)		QF (Q fever)	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Females																					
1–5	0																				
6–15	17					8	47					6	35	14	82	2	12	1	6	2	12
16–40	24	4	17	9	38	19	79			1	4	14	58	16	67	6	25	3	13	2	8
>40	20	4	20	7	35	16	80			1	5	6	30	10	50			1	5	1	5
Total	61	8	13	16	26	43	70	0		2	3	26	43	40	66	8	13	5	8	5	8
Males																					
1–5	1													1	100						
6–15	12	1	8	2	17	11	92					4	33	11	92			1	8	1	8
16–40	21	4	19	8	38	18	86			2	10	6	29	11	52	3	14	1	5		
>40	17	6	35	12	71	14	82			1	6	5	29	9	53	5	29	3	18		
Total	51	11	22	22	43	43	84	0		3	6	15	29	32	63	8	16	5	10	1	2
Females Plus Males																					
Total	112	19	17	38	34	86	77	0		5	4	41	37	72	64	16	14	10	9	6	5

Table 24

Protein Bound Iodine, Total Iodine, and Butanol Extractable Iodine in Marshallese Sera, 1959

Subject No.	μg % Iodine PBI	Total	BEI
	Exposed		
10	7.0	5.0	5.0
19	4.1	2.0	2.7
26	6.2	—	4.9
39	9.7	—	8.7
44	6.7	—	3.4
Mean	6.7	3.5	4.9
	Unexposed		
818	9.2	—	7.5
830	6.0	—	4.4
863	6.7	—	4.9
869	5.9	—	4.7
874	5.2		4.0
887	6.2		5.0
815	5.3	—	3.3
Mean	6.3		4.8

Table 25

Subjects From Rongelap With Glucosuria

Subject No.	Sex	Age	Fasting blood sugar, mg/100 ml	Weight, lb	Height, cm	ABO blood group
853	M	54	200	176	150	O
854	F	54	172	72	130	B
893	F	41	312	—	—	O
956	F	50	103	134	151	O
991	F	51	278	184	155	A

Table 27

ABO Frequency Among Marshallese and Polynesians

	Marshallese		Polynesian
	Present report (310 persons)	Simmons et al.[28] (678 persons)	Simmons and Graydon[29] (138 persons)
Percent			
O	60.7	52.2	39.1
A	19.7	21.4	60.9
B	15.1	21.1	0
AB	4.5	5.3	0
Gene frequency			
r_c	0.768	0.723	0.626
p_c	.128	.135	.374
q_c	.103	.134	0

Table 28

MN and Kell-Diego Frequency Among Marshallese and Polynesians

	Marshallese		Polynesian
	Present report	Simmons et al.[28]	Simmons and Graydon[29]
Percent			
M	8	10	19.6
MN	22	19	47.8
N	70	71	32.6
Gene frequency			
m	0.194	0.220	0.435
n	0.806	0.780	0.565
Others			
Kell (K)	0		0
Diego (Di^a)	0		0

Table 26

Deaths Reported as Due to Diabetes in the U.S. and in the Marshall Islands District of the Trust Territory of the Pacific Islands (1956 and 1957 Data Combined)*

	Population	Total deaths	Deaths per 1000	Diabetes deaths	Diabetes deaths per 100,000 population	Diabetes deaths per 1000 deaths
U.S.	337,552,000	3,197,604	9.5	53,364	15.8	16.7
Marshall Islands	27,215	160	5.9	10	36.7	62.5
					$0.01>p>0.001$	$p>0.001$

*The popualtions, total deaths, and diabetes deaths for 1956 and 1957 were totaled to get the average deaths per 1000 and diabetes deaths per 100,000 for these two years. Data for the U.S. are from reference 27 and for the Marshall Islands from Dr. H.E. Macdonald (see text).

capacity. No correlation was seen. The explanation for the higher B_{12} levels is not apparent at this time.

Studies of Genetically Inherited Traits

Blood Groups. The laboratory analysis of blood groups was conducted by Dr. L.N. Sussman and colleagues and reported as follows. The results of the 1958 and 1959 studies were combined, making a total of 310 individual bloods. Care was taken to avoid duplication. The results of this broader sampling, compared to findings of Simmons et al. for the same area[28] and for the Polynesians[29] are presented in Tables 27, 28, and 29. Data on all individuals tested are given in Appendix 6. The findings may be summarized as follows.

1. *ABO system.* The high frequency of the B gene is again demonstrated, in contrast to the absence of B genes in the Polynesians. The absence of A_2 gene in this area has been noted repeatedly.[30] In the present series a single individual of group A_2B was confirmed. The χ^2 value in this system is 5.18 ($p=0.15$). The excess of AB persons (expected 8, observed 14) contributes the major part of the χ^2 deviation.

2. *MN system.* The extremely low frequency of M gene has been noted in many studies of this population and area, in contrast to its high frequency in Polynesians. The χ^2 value in this system is 23.7 ($p=0.001$), which is statistically invalid. The error lies in the finding of 25 M persons whereas only 11.6 could be expected. Thus it appears that the N gene in the heterozygote escapes detection. If this were corrected for, the result would be an even greater frequency of N gene.

3. *Rh-Hr system.* The marked frequency of the R^1 gene is again demonstrated, higher than reported in any other study. The failure to demonstrate any rh negative persons suggests that the probable genotype of the heterozygous Rh_1 people is R^1R^0. This is further supported by the finding of two persons of phenotype Rh_0. The χ^2 value in this system is 13.7. Again the statistical value is diminished because 2 Rh_0 people were found whereas 0.26 were expected. It can be seen that a major change in χ^2 value can be caused by a single individual of "unusual" grouping.

4. *Other systems.* The failure to find in this group of 310 a single person with a Diego or Kell factor is noteworthy. The Marshallese, Maoris,[31] and Polynesians are similar in this respect, in contrast to the Amerindians, Mongolians, and Eskimos,[32] among whom some Diego positive people are found.

The following blood group characteristics of 310 Marshallese represent significant differences from those of their eastern neighbors (Polynesians) and suggest a relationship with Southeast Asians and Indonesians.

1. A relatively high B gene frequency.
2. A high N gene frequency.
3. Extremely high R^1 gene frequency.
4. Total absence of Kell and Diego factors.

Haptoglobins and Transferrins. The distribution of the haptoglobin types in the 176 Rongelapese tested is shown in Table 30. Data on all individuals tested may be found in Appendix 6. This sample included some families with two or more offspring; in these, all siblings but one were removed by random selection to give a sample of 124 individuals in which the families included, at most, parents and one child. The distribution of the haptoglobin types in this group did not differ significantly from that in the total group. In each case, agreement with the Hardy-Weinberg predictions was good, suggesting that the population was homogeneous for this trait. Omitting the two sera with no haptoglobins, the frequency of the Hp^1 gene is 0.58 and of the Hp^2 gene 0.42. The frequency of the Hp^1 gene is higher than in the West European populations so far tested.

Four Rongelapese had no detectable haptoglobin either in 1957 or 1959. In addition, in many sera only very small amounts of haptoglobin

Table 29

Rh-Hr Frequency Among Marshallese and Polynesians

	Marshallese		Polynesian
	Present report	Simmons et al.[28]	Simmons and Graydon[29]
Phenotype percent			
Rh_1Rh_1	90.9	90.6	19.6
Rh_1rh	4.2	0.7	0.7
Rh_1Rh_2	3.9	8.0	50.0
Rh_2	0.3	0.3	29.7
Rh_0	0.6	0.12	
Gene frequency			
R^1	0.950	0.951	0.449
R^2	.020	.04	.543
R^0	.030	.006	.007

Table 30

Distribution of Haptoglobin Types in Micronesians From Rongelap Atoll

Group	No. in group	Type 1–1 Observed %	Type 1–1 Observed No.	Type 1–1 Expected, No.	Type 2–2 Observed %	Type 2–2 Observed No.	Type 2–2 Expected, No.	Type 2–1 Observed %	Type 2–1 Observed No.	Type 2–1 Expected, No.	0 and rare types, Observed %	0 and rare types, Observed No.
All Rongelap	176	33.5	59	59	18.2	32	31.5	47.2	83	85.5	1.1	2
Selected* Rongelap	124	33.1	41	–	15.3	19	–	50.0	62	–	1.6	2

*Family groups include only one child (see text).

were visible; two of these were very faint 2–2's, but the fastest-moving haptoglobin band was not seen in them.

The Micronesian sera studied were all transferrin type CC, which is the common European type.

Considerable caution must be exercised in extrapolating to an entire population the gene frequencies obtained from a small sample. This is particularly true when studying societies made up of small isolated or semi-isolated groups which compartmentalize the breeding community. Thus, the Rongelapese appear to have a fairly high frequency of type 1–1, consistent with the general (though not exclusive) rule that in Europe-Africa and America the frequency of type 1–1 is higher in populations living near the equator than in those remote from it.

It is clear from the Micronesian studies that, at least in some cases, an individual may have no haptoglobin at one time, but have sufficient haptoglobin to permit typing at another time. (In this case, the interval was 2 years.) Examination of the ahaptoglobinemic individuals gave no significant findings. With $\approx$¾ of the Rongelap population sampled, all the ahaptoglobinemic individuals fell into one family grouping. This did not help to elucidate the genetic pattern, except to make it appear unlikely that the Hp^0 allele, if such exists, is not at the same locus as the Hp^1 and Hp^2 alleles. One of the sibs of an ahaptoglobinemic was a type 2–1.

β-Amino-iso-butyric Acid (BAIB) Levels. Figure 46 shows the frequency distribution of BAIB excretion for the Rongelapese and that of a New York City white population for comparison. The two distributions are essentially complementary to one another, and, if superimposed, exhibit bimodality with the antimode in the neighborhood of 1.75. With this point used as the division between high and low excretors, it is estimated that nearly 90% of the Rongelapese are high excretors. A breakdown of this population into exposed and unexposed groups shows almost identical mean BAIB excretion values for the two (exposed, 2.07±

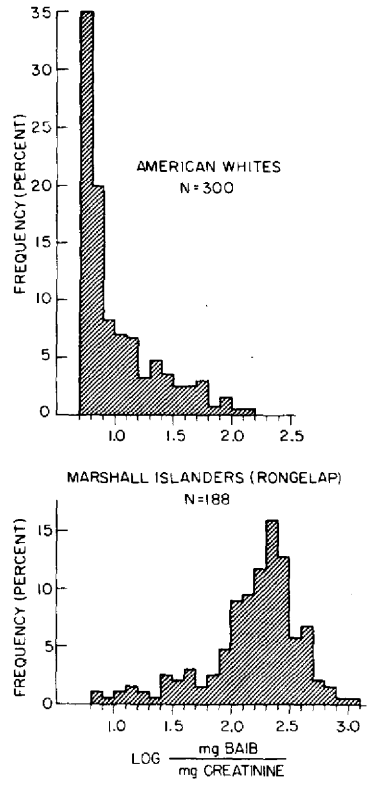

Figure 46. Frequency distributions of urinary excretion rates of β-amino-*iso*-butyric acid of Marshall Islanders (Rongelap) and U.S. whites.

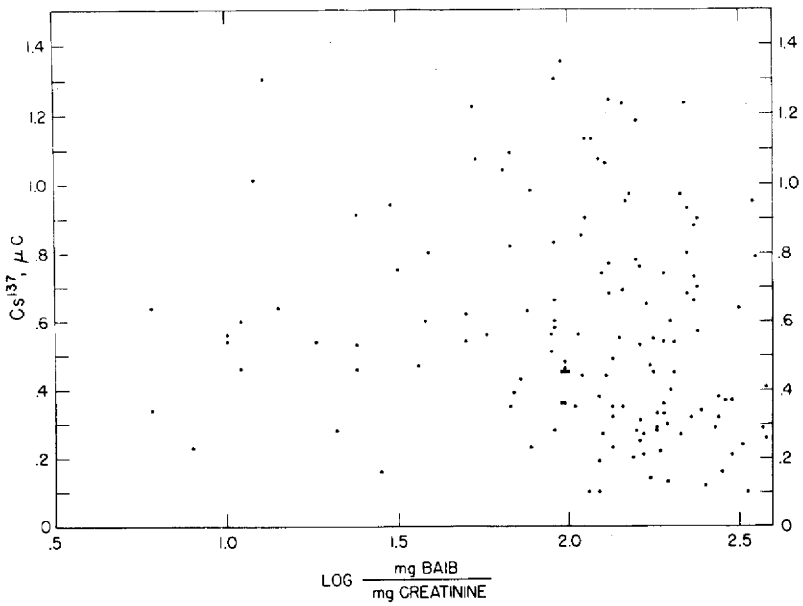

Figure 47. Urinary BAIB excretion (mg-β-amino-*iso*-butyric acid per mg creatinine) versus body load of Cs^{137} (μC), Rongelap inhabitants, 1959.

0.06; unexposed, 2.10±0.04). A scatter plot of Cs^{137} levels and BAIB levels shows no correlation (Figure 47), and neither does a similar plot between Zn^{65} levels and BAIB levels. The mean BAIB excretion level for 18 Micronesians, who were on Utirik at the time of the atomic explosion and therefore not exposed to significant radiation, was 2.21±0.13, which is extremely close to the mean value for the Rongelapese. Among the Utirik people 83% were high excretors, which is not significantly different from the Rongelap group percentage. A number of complete families are included in the Rongelap sample (i.e., father, mother and at least one child); of these the high-excretor by high-excretor matings are the only ones providing significant genetic information. There were 11 such matings with a total of 31 offspring (29 high excretors and 2 low excretors). One of the low excretor offspring was illegitimate, as determined by blood group data, but the other remains as an unexplained exception to genetic hypothesis.

Rubini et al.[23] have recently demonstrated that BAIB excretion can be a sensitive indicator of radiation exposure. The mechanism involved is probably the same as that underlying the effect of nitrogen mustard on BAIB excretion.[33] Such agents markedly increase DNA breakdown; the thymine thus released is effectively converted to BAIB in man [24,33] and excreted in urine, since no appreciable tubular reabsorption of BAIB occurs.[34] It is unlikely that the high incidence of high BAIB excretors found in Rongelap is due to radiation. The identical average BAIB values in the exposed and unexposed groups preclude the possibility that the elevation is due to the fallout exposure in 1954, and it appears that radiation exposure (at the levels received at Rongelap) has no long-term effect on BAIB excretion. The lack of correlation with the Cs^{137} and Zn^{65} body burdens rules against the elevation being due to the present retained internal radiation. The nearly identical values in the Utirik group who received a very small dose of radiation, and who have much lower body burdens of radionuclides, is also consistent with the thesis that a high incidence of the gene underlying β-amino-*iso*-butyric aciduria is responsible for the observed BAIB excretion rate. The expression of the normal genetic difference is probably due to differential metabolism of BAIB by high and low excretors.[34]

The level of BAIB excretion among the Marshallese is the highest yet reported for any population. Blood group[35] and other anthropological data on these people suggest a Southeast Asiatic origin; if this is true, a high BAIB level can be predicted for the populations of that region. Studies of some Asiatic populations have given

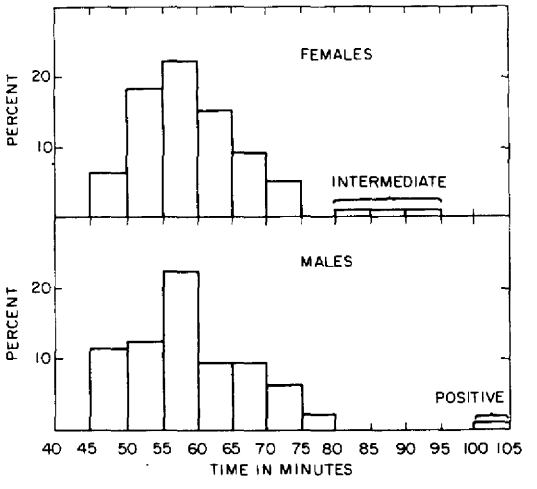

Figure 48. Percent distribution of glucose-6-phosphate dehydrogenase deficiency in males and females (percent of persons versus decolorization time).

relatively high BAIB values, which suggest that there may be an Asian focus for the high BAIB excretor gene.

Glucose-6-phosphate Dehydrogenase Determination. One male (#11) decolorized at 102 min and was classified as positive; three females (#18, 22, and 851) decolorized at 80, 85, and 93 min, respectively, and were classified as intermediates. The distributions are shown in Figure 48.

The glucose-6-phosphate dehydrogenase deficiency appears to exist in the Rongelap population, although in fairly low frequency. The number of tests done was too small for any final conclusions to be drawn, and it is important that these results be confirmed on subsequent visits, particularly with tests done in the field in order to eliminate the possibility of sample deterioration during transport.

Radionuclide Body Burden Evaluation

INTRODUCTION

In considering the evaluation of the radionuclide body burden of the Rongelap people, the following facts should be kept in mind. During the two days that the people remained on the island after the fallout occurred in 1954 (prior to their evacuation), they lived in a radioactively contaminated environment and made little or no effort to avoid inhaling the radioactive material or ingesting it in their food and water. The resulting internal radioactive contamination was reflected by significant levels of activity in their urine detected by radiochemical analysis. Following their evacuation, the people lived for 3 years on the uncontaminated islands of Kwajalein and Majuro. The people of Utirik were returned several months after the accident to their home island, since the level of contamination there was very low.

The initial body burdens of internal emitters were estimated from data obtained by radiochemical analysis of the tissues of pigs which had been simultaneously exposed, and also from a comparison of human and animal urinalysis data.[36] The mean body burden at one day was estimated (in μC) as Sr^{89}, 1.6; Ba^{140}, 2.7; I^{131}, 6.4, and the rare earth group together, 1.2. The contribution of this amount of internal contamination is small compared to the 175 r external gamma dose received. In the first few months following this acute exposure, Sr^{89} and I^{131} (plus the shorter-lived iodine isotopes) contributed the greatest internal radiation dose. Sr^{89} contributed the major portion of the beta dose to the skeleton at this early time. The highest dose to an individual tissue (100 to 150 rep) was delivered to the thyroid by I^{131} and the shorter-lived isotopes, I^{132}, I^{133}, and I^{135}.

In the spring of 1957, 3 years after the accident, four Rongelap people, two Utirik people and one unexposed Marshall Islander were taken to Argonne National Laboratory, and gamma spectrographic analyses were carried out in a whole-body counter. Distinct photopeaks indicating the presence of significant levels of Cs^{137} and Zn^{65} were detected in the spectra of exposed Rongelap people and the unexposed subject.[5,54] This experience demonstrated the feasibility of using *in vivo* whole-body counting techniques for estimating body burdens in these people. In the following year, 1958, a "portable" steel room and a whole-body gamma spectrometer were constructed at Brookhaven National Laboratory which could be transported to the Marshall Islands for use in further studies.

In July 1957, after careful radiological surveys which showed the island of Rongelap to be safe for habitation, the people were returned and settled in a completely new village which had been constructed for them. Low levels of contamination persisted on the island, which have since

been reflected in an increase in body burdens of some radionuclides. As will be shown, the increases, though manyfold, have remained far below maximum permissible levels.

ESTIMATION OF THE INTERNAL RADIATION HAZARD

The potential radiation effects that may be produced by specific quantities of internally-deposited radioisotopes can be only roughly predicted from clinically observed effects of known amounts of internally-deposited radium. These effects do not appear until a period of years (10 to 15) has elapsed. Thus, although it is possible to estimate the potential hazard in terms of the concentration of internal emitters, clinical observations made within a few years after contamination yield no data on the degree of damage that may ultimately be produced.

In evaluating the long-term effects produced by an acute internal exposure and exposure to residual contamination, Sr^{90} is clearly the critical element. Particular effort was therefore made to determine its levels in the urine of the Marshallese, and thus to estimate the body burdens.

Of the gamma-emitting fission products, Cs^{137} is of the greatest interest, even though it is of minor significance as an internal radiation hazard. Like Sr^{90}, Cs^{137} has a gaseous precursor with a half-life sufficiently long to avoid early condensation in the fireball. Cs^{137} thus follows Sr^{90} into the stratosphere. Since the fission yields and the half-lives of the two radioelements are nearly equal, they are present in the fallout in like quantities. While they have different ecological cycles because of their different chemical properties, Cs^{137} nevertheless provides a useful tracer for studying the movement of Sr^{90} through the biosphere, since its gamma-emitting properties make it readily detectable.

There is also some interest in the neutron-induced radioelement Zn^{65}, even though it, too, does not appear in levels hazardous to human beings. The interest centers chiefly around the fact that it is definitely transmitted through marine life, and thus provides a clear example of the transmission of a radioelement through the food chain to man.

Other fission products and neutron-induced activities ($Fe^{55,59}$, $Co^{57,58,60}$, Mn^{54}, Ce^{144}-Pr^{144}, Zr^{95}-Nb^{95}, and Ru^{106}-Rh^{106}) also appear in small amounts in the soil and the food chain, and thus may appear ultimately in man, but the levels are so low that little effort was made to quantify these elements in the Marshallese until whole-body counting techniques made it feasible.

The body burden of fission products can be determined in three ways. The method of choice is the direct *in vivo* measurement by whole-body spectrometry. The limitations of this method are that few whole-body counters are in existence, they require enormously bulky shielding and thus are not easily transported to various sites, and their absolute calibration is difficult. Further, this method is restricted to analysis of gamma-emitting isotopes, since, to date, a whole-body beta counter has not been developed.

A second method for calculating body burden, particularly for counting beta emitters such as Sr^{90}, is the estimation of the internal deposition from data obtained by radiochemical analysis of the urine.

Finally, it is possible to make a completely indirect estimate of the human body burden of radioisotopes by what may be called the environmental approach. In this method, the estimate of the body burden is based on the concentrations of the fission products present in the environment, chiefly the soil and the important components of the diet. In order to make this estimate, data must be obtained on the transfer of the fission products between successive elements of the ecological chain leading from soil to bone. For example, although Sr and Ca are chemically similar and thus appear together in the various components of the ecological chain, Ca is taken up preferentially by plants and animals so that it is necessary to determine the discrimination factor for each step. When these factors are known, it is possible to estimate the concentration of a radionuclide in man from its concentration in any step of the ecological chain.

All three of these approaches to the estimation of the body burdens in the Marshallese people will be considered in this report.

EXPERIMENTAL PROCEDURES

Whole-Body Counting

The gamma-ray activity from the internally-deposited fission products and the neutron-induced activities in 227 of the Marshallese people were measured with a whole-body gamma scintillation spectrometer. The technique of *in vivo* gamma-ray measurement of human beings was

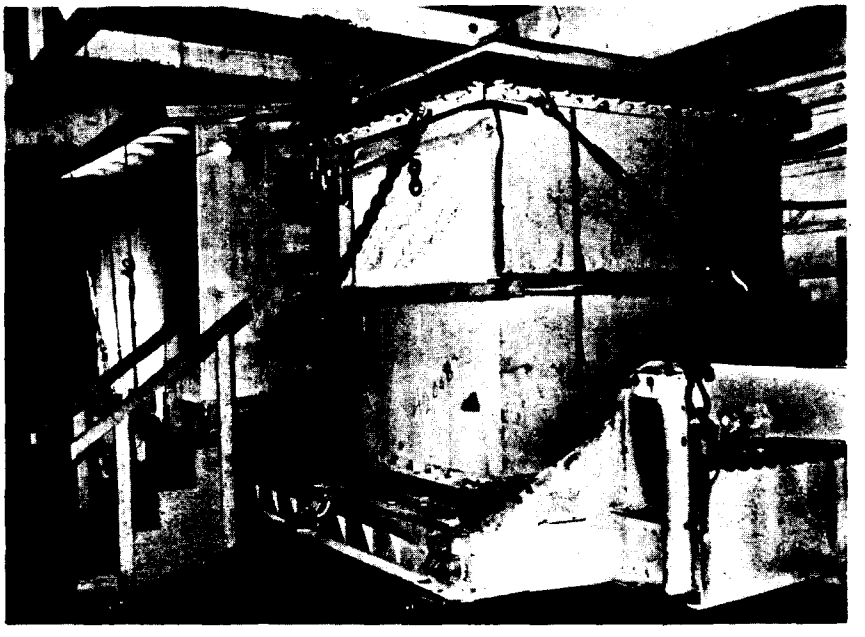

Figure 49. Whole-body counter and electronic room mounted on tracter located on tank deck of Navy LST.

Figure 50. Interior of electronic room showing 100-channel analyzer and associated electronic circuitry.

Figure 51. Marshallese subject in standard counting position inside steel room.

used to supplement quantitative radiochemical analytical procedures, and has made possible very rapid and positive identification of gamma-emitting radionuclides. The principles of *in vivo* gamma spectrometry as applied to human beings have been previously described.[37]

The steel room constructed at BNL in 1957 for the Marshallese surveys has been described previously.[6] The "portable" 21-ton steel room, 5×5×6-ft with 4-in.-thick steel walls, was used to supply the required shielding (see Figure 49). This steel room was mounted on a large trailer along with a 7×8×10-ft air-conditioned wooden room, designed to house the electronic components of the counter (see Figure 50). The trailer was mounted on the tank deck of the Navy LST, and the subjects to be counted were brought aboard up the ship's ramp from the beach on each island visited. Also available on the tank deck were shower facilities where each subject washed and then changed into an uncontaminated paper suit to avoid the possible risk of measuring any external contamination that might be present.

A 5-in. NaI (Tl) crystal (Harshaw) was placed over the subject at a distance of 19 in. above the apex of the chair (see Figure 51). The pulses from the photomultiplier were fed into a linear amplifier (Cosmic Radiation Lab. Inc., Model 101), and thence into a 100-channel analyzer (TMC-PA 100, Quartz Line Storage Pulse-Height Analyzer). Two complete detectors and analyzers with the necessary associated electronic circuitry were taken along, since considerable difficulty had been experienced during the previous year in maintaining the whole-body counting system operational under the conditions of tropical heat and humidity. Housing of the electronic equipment in an air-conditioned, dehumidified room during operation was of considerable assistance in maintaining its stability.

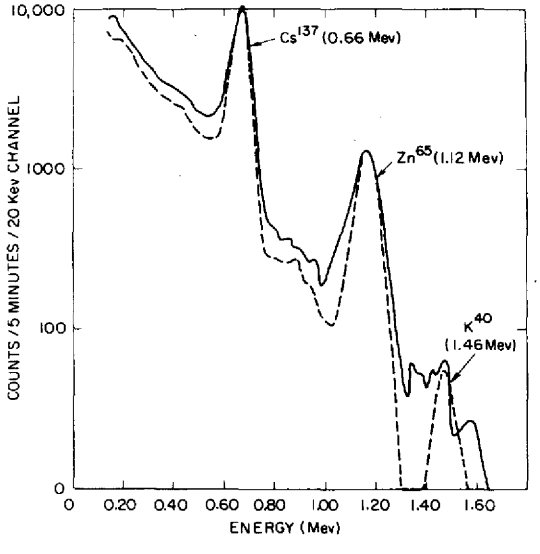

Figure 52. Gamma spectra of Marshallese male (subject #4, age 40, wt 70 kg – solid line) and normalized plastic phantom (containing 0.64 μC Zn^{65}, 1.22 μC Cs^{137}, and 140 g K – dashed line), 1959.

Since the principal photopeaks in these persons were known from the previous study to be due to Cs^{137} and Zn^{65}, and since the levels were sufficiently high, it was only necessary to count the subjects for 5 or 10 min with the particular geometrical arrangement described above. The data were printed out on a paper tape of a Victor adding machine, and the tapes were returned to BNL for analysis.

Analysis of Gamma-Ray Spectra

The quantitative interpretation of the gamma-ray pulse-height data for the discrete gamma energies involved the graphical reduction of the data. A typical spectrum of a Marshallese is shown in Figure 52. The ordinate represents counts per 20-kev pulse-height increment, and the abscissa represents gamma energy. In the graphical stripping method employed,[38,39] the activity of the highest energy gamma-ray in the mixture, K^{40} (1.46-Mev), is determined directly from the ordinate value of its total absorption peak. Then a channel-by-channel subtraction of the distribution corresponding to the abundance of this particular gamma-ray or radionuclide is made from the spectrum. When this subtraction is completed, the concentration of the gamma-ray of the next highest energy, Zn^{65} (1.14-Mev) can be estimated from the ordinate value of its peak. The distribution corresponding to the concentration of the second gamma-ray is then subtracted from the remaining distribution, and the process is continued with Cs^{137} (0.66-Mev) until all the three major gamma-ray-emitting components have been determined.

This stripping process is illustrated on a spectrum of the plastic phantom containing known amounts of K^{40}, Cs^{137}, and Zn^{65} (Figure 53). It is to be noted that at the concentrations used here (approximately the mean concentrations found in the Marshallese), the Compton contribution of

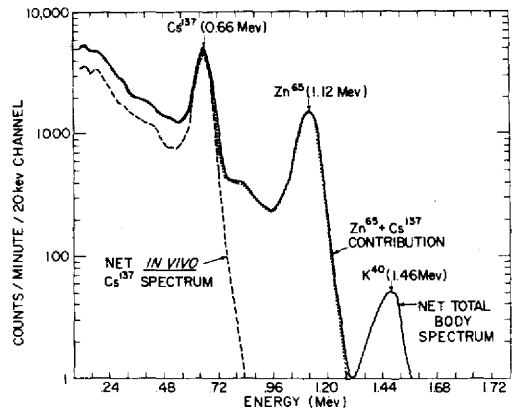

Figure 53. Gamma spectrum of phantom illustrating graphical stripping of K^{40}, Zn^{65}, and Cs^{137} from total spectrum.

one isotope to the photopeak of the other isotopes of lower energy is very small.

In order to carry out this stripping method, it is necessary to have calibrated pulse-height distribution spectra for each gamma emitter encountered. Further, these spectra must ideally be obtained from a subject of the same size and body build. To obtain these spectral data, known amounts of Cs^{137} and Zn^{65} were administered to subjects at BNL, and their spectra were obtained. Later in the study, a plastic phantom (REMAB-Alderson) was obtained and used for calibration (Figure 54). Spectra were also obtained from the phantom with known amounts of KCl, Cs^{137}, and Zn^{65}. From these spectra, an average spectrum for each isotope was obtained. The pulse-height distribution spectrum of one of the Marshallese subjects is compared with the spectrum obtained with the plastic phantom containing the same concentrations of K, Cs^{137}, and Zn^{65} in nearly identical counting geometry in Figure 52. In this way it was possible to simulate the multicomponent spectra of the Marshallese by use of the phantom.

Since it is not possible to measure a photopeak until the contributions of other peaks of higher energy and their Compton continua have been subtracted out, and since the presence of small amounts of unknown radionuclides is not always obvious in the presence of large concentrations of other radionuclides, it is possible to miss the presence of very small amounts of other fission products. However, when all the major components have been stripped out, the presence of any remaining photopeak should serve to identify the presence and concentration of other components of the spectrum.

This procedure was further complicated in this study by several factors. In the field study the subjects were measured with a 5-in. NaI (Tl) crystal. The calibration was originally carried out in the field with a Presdwood phantom, but when the Alderson plastic phantom later became available it was found to give a better approximation of the spectrum for each isotope, and therefore most of the calibration was repeated with it at BNL. However, the geometry in the field situation was rather difficult to duplicate exactly. Also, counting the subjects for 5 to 10 min was sufficient to estimate accurately the levels of Cs^{137} and Zn^{65} but not the K^{40} body concentration and trace amounts of other fission products in the presence of the relatively large amounts of Cs^{137} and Zn^{65}. The lack of a statistically significant number of counts to measure K^{40} accurately is evident from the poorly defined K^{40} photopeak of the subject as compared

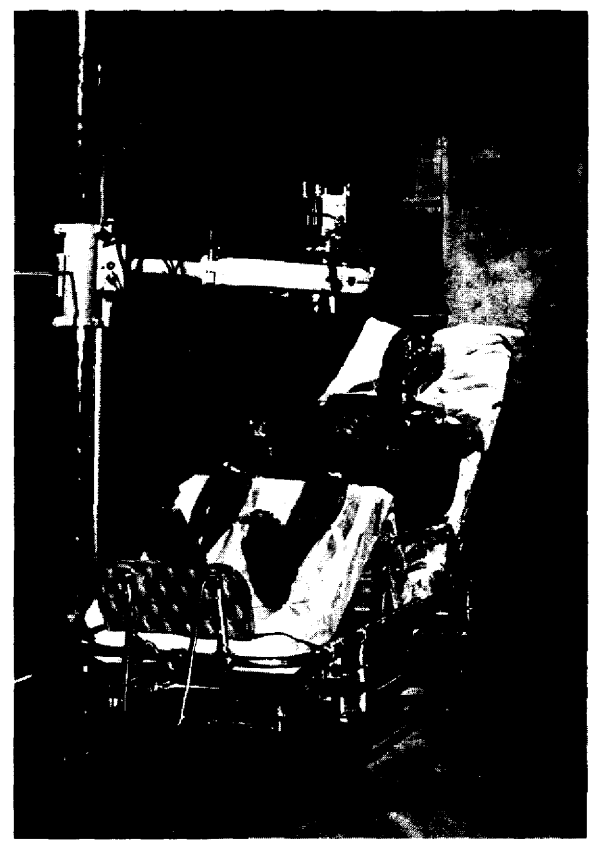

Figure 54. Calibration phantom in standard counting position in BNL whole-body counter.

to that of the phantom, which was counted for 30 min (Figure 52).

In future whole-body counting of these people, it will be possible, by counting for longer periods and using an 8-in. NaI crystal, to improve the absolute measurement of trace amounts of other radionuclides that may be present.

Since a total of 227 Marshallese persons were surveyed with the whole-body counter, in addition to numerous controls, the spectral analyses were performed with the aid of a 704 IBM computer.

Radiochemical Procedures

Twenty-four-hour urine specimens were collected in plastic bottles and sent to BNL for radiochemical analysis. A modification of the method of Farabee[40] was used for the analysis of Sr^{90}. Sr was precipitated as the alkaline phosphate, ashed with HNO_3 and H_2O_2, and dissolved in dilute HNO_3. After the solution was brought up to a volume of $\approx$800 cc, the alkaline earths were complexed with EDTA, and the *p*H was adjusted to 5.5. The solution was then passed through an ion exchange column (Dowex-50 in the Na form), and the column was rinsed with 300 cc of a solution of 1% citric acid and 0.75% EDTA at a *p*H of 5.0. The combined effluents contained >95% of the total Ca. The column was then rinsed with 6 *N* HNO_3 to remove the Sr^{90}. Carrier Sr was added to the Sr^{90} fraction and precipitated with 70% fuming HNO_3. Yttrium-90 was milked and counted by the method of the AEC Health and Safety Laboratory.[41]

The supernatant from the alkaline phosphate precipitation was measured and divided into two portions. One portion was scavanged for cesium with added carrier by means of a double precipitation of the aluminum sulfate and the chloroplatinate.[41] The second portion was analyzed for K by flame spectrophotometry.

Food samples were weighed and dry-ashed in a muffle furnace at 800°C. The ash was weighed, and a small portion was counted for gross beta activity. The ash was dissolved in dilute HNO_3 and processed by the method described above for urine analysis.

All counting was done in a low-level beta anticoincidence type of counter, designed and built at BNL. Samples were mounted on glass fiber filter discs with nylon rings and discs and Mylar film. Samples were counted against NBS standards processed and counted under identical geometry.[41]

RESULTS AND DISCUSSION

All three of the above methods were used for estimating the body burdens of gamma- and beta-emitting radionuclides in the Marshallese people. Individual values for all the people examined in 1959 may be found in Appendix 7 for gamma spectrographic analyses and in Appendix 8 for radiochemical analyses.

Environmental Estimate

One method used (the least quantitative) was the environmental estimate of body burden. The environmental estimate of internally-deposited Sr^{90} was made in two ways. In the first method, animals subsisting on diets similar to human diets were sacrificed and their tissues were analyzed radiochemically. A number of rats were collected on Rongelap Island at 2, 4, and 5 years after the 1954 accident. If the diet of these rats, primarily land plants, was sufficiently similar to the diet of human beings inhabiting this area, the rat analyses might serve as indicators of the human internal radiation contamination. The Sr^{90}/Ca ratios of various tissues of these rats were measured directly and compared to the ratios of the food and soil on Rongelap collected at the same time; that is, the environmental contamination was compared with the directly measured contamination in animal tissue. Extrapolation of the environmental data gives the equilibrium value which can be expected, whereas the direct measurement gives the value at the time of measurement (and thus the percent of the equilibrium value for the individual radionuclides).

The Sr^{90}/Ca ratios for different plant foods on Rongelap varied greatly, and the diet of the rats was too uncertain for an "average" diet to be assumed. Therefore, for a body burden estimate it was necessary to use the Sr^{90}/Ca values of the soil itself.

The "strontium-calcium observed ratio" (OR) of Comar[42] was used to denote the preferential utilization of calcium in the following manner:

$$OR_{\text{sample-precursor}} = \frac{\text{Sr/Ca of sample}}{\text{Sr/Ca of precursor}}$$

The Sr^{90} discrimination ratio in the chain from soil (*s*) to bone (*b*) via plants (*p*) can be expressed as follows:

$$OR_{b-s} = (OR_{p-s})(OR_{b-p}) = (0.7)(0.25) = 0.18 .$$

The value $OR_{b-p}=0.25$ is an approximate value obtained experimentally on rats fed a stock laboratory diet.[42] The discrimination factor of 4 for calcium against strontium from diet to bone in man has been reported by Schulert[43] and Bryant.[44] A more appropriate value for the rats in this situation might be the $OR_{bone-diet}=0.16$ obtained by a study of wild kangaroo rats living in the Nevada desert.[45]

The Sr^{90} body burden is then

$$(Sr^{90}/Ca)_b=(Sr^{90}/Ca)_s(OR_{p-s})(OR_{b-p})$$
$$=(8.4\times10^3)(0.7)(0.16)=924\ \mu\mu C\ Sr^{90}/g\ Ca\ .$$

The value obtained in this manner is approximately twice the value 470 to 545 $\mu\mu C\ Sr^{90}/g$ Ca obtained by direct radiochemical analysis of the tissues of rats living on the island during the 2-year period following detonation.[46] This difference between the indirect environmental estimate of the body burden of Sr^{90}/Ca and the results of direct analysis may reflect either errors in the discrimination ratios or perhaps lack of equilibrium between the Sr^{90}/Ca in the animals and in the soil at 2 years. The latter possibility exists, since, although the 13 adult rats analyzed by Held[47] at 4 years had values close to the 2-year level, $443\pm181\ \mu\mu C\ Sr^{90}/g$ Ca, the life span of the rat is only $\approx$2 years.

It is obvious that use of this technique to estimate the Sr^{90} body burdens of the Marshallese people is also complicated by the uncertainty of their diet. The estimates of average dietary intake of the Marshallese since their return to Rongelap are approximate, because the diet has varied during the past several years. A study made in 1958 yielded daily Sr^{90}/Ca intake levels of 67.5 Sr units ($\mu\mu C\ Sr^{90}/g$ Ca), provided that coconut crabs (see Figure 4) were excluded from their diet.[48,49] The Sr^{90}/Ca levels in the various foods are shown in Table 31. This study was based on the analysis of various food samples in what may be considered an average diet. The data were obtained from a study of the diets of 14 males on Rongelap.[47] It was assumed that half the calcium in the diet was derived from food not native to Rongelap Island. From the discrimination factor of 4 and the daily intake of 67.5 Sr units, the equilibrium Sr^{90} body burden for the Rongelap people is calculated as $\approx$17 mμC. This is very close to the equilibrium body burden (23 mμC) estimated by Woodward from the urinalysis data.[50]

Another effort was made in the 1959 survey to gather samples of meals to be assayed for Sr^{90}/Ca content. However, since the Marshallese were found to subsist to a large extent on foods not indigenous to the area, such as C rations, rice, and

Table 31

Estimate of Sr^{90} in Diet of Rongelap Adults, 1958

	A Daily intake,* g (wet wt)	*B* Ca content, mg/g	*A*×*B* Daily Ca intake, mg	*D* Fraction of total Ca intake**	*E* Sr^{90} content, $\mu\mu C/g$ Ca	*D*×*E* Contribution to total daily Sr^{90} intake, $\mu\mu C/g$ Ca
Meat from mature coconut	89	0.075	6.7	0.008	1,200	9.6
Meat from drinking coconut	75	0.14	10.5	0.013	210	2.7
Milk from green coconut	116	0.15	17.4	0.022	1,000	22.0
Pandanus, edible portion	79	0.15	11.9	0.015	930	14.0
Arrowroot	58	2.10	121.8	0.152	19	2.9
Breadfruit	45	0.60	27.0	0.034	260	8.8
Fish	139	0.13	18.1	0.023	280	6.4
Clams	45	4.00	180.0	0.225	5	1.1
Crabs, land	14	4.00	56.0	0.070	(4,000)	(280.0)
Total	660		449	0.56		67.5

The diet also included imported foods: rice, canned C rations, flour, tea, milk, salt, and sugar.

*Based on average daily diet of 14 Rongelap males.[47]
**Based on total calcium intake of 0.8 g/day.

tea, it became even more difficult to extrapolate to body burden from food.

It is obvious that further data are required on the transport of low levels of Sr^{90} and other products through the ecological cycle in this and other communities to make possible assessment from environmental data alone of the internal radiation hazard to human beings living in a fallout-contaminated area.

More reliable estimates of the Marshallese body burdens can be obtained by whole-body gamma spectrometry and by radiochemical urinalysis.

Radiochemical Analysis of Urine

Strontium-90. The urinary excretion levels of Sr^{90} for the 5 years following exposure to fallout are shown in Figure 55. The 4- and 5-year levels were much higher, after the return of the Marshallese to Rongelap in July 1957, the mean being higher by a factor of 20 in March 1958 than in March 1957.

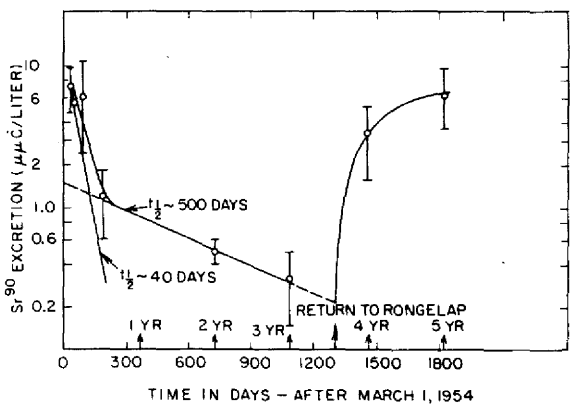

Figure 55. Urinary excretion of Sr^{90} in exposed Marshallese.

The excretion rate of Sr^{90} may be expressed as the sum of two exponential functions for the first 3 years following exposure. The major fraction of Sr^{90} is excreted early, with a biological half-life of 40 days. The smaller fraction is excreted with a half-life of 500 days. These excretion rates correspond to those reported by Cowan[51] in a case of accidental inhalation of Sr^{90}, and were used in extrapolating back to the one-day Sr^{89} body burden of the Marshallese.[36].

The 1958 Rongelap body burdens of Sr^{90}, Cs^{137}, and Zn^{65} are presented in Table 32, and also figures for percent of equilibrium and equilibrium value, estimated by Woodward[50] from urinary excretion data. These values are subject to some uncertainties, since they are based on a number of assumptions; however, they can be checked by use of other methods. For example, the estimated body burden of Sr^{90} in March 1958 was 2 $\mu\mu$C/g Ca, based on the 24-hr Sr^{90} output in urine (1 liter per 24 hr), and this appeared to be of the right order of magnitude compared with data from bone analysis. Two bone samples of vertebra and ileum from a deceased 35-year-old Rongelap male at this time indicated a level of about 3.7 $\mu\mu$C/g Ca, which gives, upon application of the normalization factor of 2 from vertebra to average skeleton,[52] an average skeletal value of 2 $\mu\mu$C/g. Thus the mean body burden of Sr^{90} for exposed Rongelap people in 1958 was estimated to be $\approx$2 mμC, or about 9% of the estimated equilibrium value of 23 mμC.[50]

The estimated Sr^{90} body burden increased from 2 mμC in 1958 to 6.0 mμC in 1959, or 26% of the estimated equilibrium value. The 1959 Sr^{90} mean urinary value in the exposed Rongelap inhabitants was 6.3 $\mu\mu$C/l or 10.5 $\mu\mu$C/24-hr urine, based

Table 32

Estimation[50] of Body Burden, in mμC, of Rongelap Population From Urinary Excretion Levels, 1958

		Cs^{137}		Zn^{65}	
	Sr^{90}, Exposed	Exposed	Control	Exposed	Control
Body burden	2*	900	1200	280	540
Equilibrated body burden	23	1300	1600	330	650
Percent of equilibrium	9	69	75	85	83
Daily intake	0.015**	2(?)		2.1–4.1	

*3.7 Strontium units (SU) determined by bone biopsy.
**15 SU assuming daily calcium intake = 1 g.

Table 33

Urinary Sr^{90} Levels, in $\mu\mu C/l$, of Marshallese, 1959

	Males		Females	
	Age 1–15	Age >15	Age 1–15	Age >15
Rongelap exposed	2.4 (2)*	7.3 ±5.1 (8)	3.9 (2)	5.4 ±2.2 (9)
Rongelap unexposed	7.1±2.0 (3)	5.6 ±2.8 (9)	–	4.2 ±2.3 (6)
Ailingnae	–	5.3 (1)	–	3.0 (1)
Utirik	–	1.5 ±0.9 (5)	3.9 (1)	2.5 (2)
Ebeye	–	0.56±0.37 (4)	–	0.70±0.70 (3)

*The numbers in parentheses are the numbers of people in the groups.

Table 34

Cs^{137} Body Burdens, in $m\mu C/kg$, of Marshallese as Measured by Whole-Body Gamma Spectroscopy, 1959

	Males		Females	
	Age 1–15	Age >15	Age 1–15	Age >15
Rongelap exposed	12.4±6.2 (9)*	14.2±5.1 (10)	11.3±2.7 (12)	11.0±3.4 (16)
Rongelap unexposed	11.8±4.8 (20)	14.0±4.7 (37)	9.9±2.1 (15)	9.8±2.8 (33)
Ailingnae	8.9 (2)	11.5±3.6 (4)	10.0±3.1 (4)	11.4±4.5 (5)
Utirik	4.6±1.2 (14)	4.5±1.6 (15)	4.5±1.9 (14)	3.6±1.3 (15)

*The numbers in parentheses are the numbers of people in the groups.

on an average 24-hr urine output of 1660 ml (see Table 33). No significant difference was found between the Rongelap exposed and unexposed groups, although the exposed adult group had a slightly higher mean value than the unexposed group. Females tended to have a lower mean Sr^{90} value than males, but the difference was not statistically significant. Relatively few children <15 years of age were tested for urinary Sr^{90}; therefore, it was not possible to compare their levels statistically with those of the adults. The mean Sr^{90} urinary value for an adult inhabitant of Utirik was 1.8 $\mu\mu C/l$, which is about 35% that of the Rongelap group. The control group on Ebeye Island (not contaminated) had still lower values, 0.62 $\mu\mu C/l$.

The lack of significant difference between the exposed and unexposed groups on Rongelap seems to indicate that essentially no residual Sr^{90} from the initial exposure is detectable in the exposed group.

Cesium-137. The Cs^{137} urinary excretion levels of the Marshallese people for the period from 50 to 180 days following exposure can be expressed as a single exponential function with a half-life of 70 days (see Figure 56). This figure is not in agreement with the value of 140 days obtained by Anderson,[53] but a biological half-time of 51 to 59 days was obtained in a clinical study made over a 4-month period following injection of $Cs^{137}Cl_2$ into two patients.[48]

The estimates of body burden of Cs^{137} in 1958, derived indirectly from urinalysis, are presented in Table 32.

The urinary excretion of Cs^{137} of 34 $\mu\mu C/l$ in 1957 indicates that the Rongelap people were exposed to a continuing low level of Cs^{137} from stratospheric fallout during 1956 while residing on Majuro. By contrast, the mean body burden of Cs^{137} in 1957 of the Utirik people (who were returned to their atoll in 1954) was 334 $m\mu C$, considerably higher than that of the Rongelap people who were residing on Majuro at that time.[50] This higher burden among the Utirik people in 1957 can be attributed to the higher level of Cs^{137} contamination on Utirik than on Majuro at that time.

Zinc-65. Zn^{65} urinary levels were not measured before 1958. With the assumptions that excretion of Zn^{65} is exponential and urinary excretion is 10% of total excretion (urinary/fecal ratio = 1/9), the March 1958 urinary excretion level of 175

$\mu\mu$C/l indicates a body burden in the exposed Rongelap group of 280 mμC ($\pm$49%) with an equilibrated body burden of 330 mμC.[50] The Zn^{65} level was therefore 85% of the estimated equilibrium level in 1958 (see Table 32).

Whole-Body Counting With the Gamma Spectrometer

Cesium-137. The body burdens of Cs^{137} of the various groups studied during the 1959 survey are presented in Table 34. The variations in levels within each group are quite large. If Cs^{137} body burden is expressed in units per unit body weight, no significant difference is found between persons older and younger than 15 years. The mean Cs^{137} level tends to be slightly lower for females than for males, but again the difference is not significant. It is to be noted that no significant difference was found between the Rongelap exposed, the Rongelap unexposed, and the Ailingnae groups. However, the mean Cs^{137} body burden of the Utirik group (4.3 mμC/kg) is (as in the case of Sr^{90}) about one-third that of the Rongelap exposed group (12.0 mμC/kg).

The mean Cs^{137} body burden of the exposed Rongelap group in 1959 was 0.57 μC (12.0 mμC/kg) compared to 0.68 μC in 1958. The level has fluctuated over the years since the original contaminating event. (See Figure 57, which shows values obtained by whole-body gamma spectrometry and by extrapolation from urinalysis data.)

Unlike Sr^{90}, which is firmly fixed in the skeletal tissue, Cs^{137} has a relatively short biological half-life, and thus readily reflects the environmental

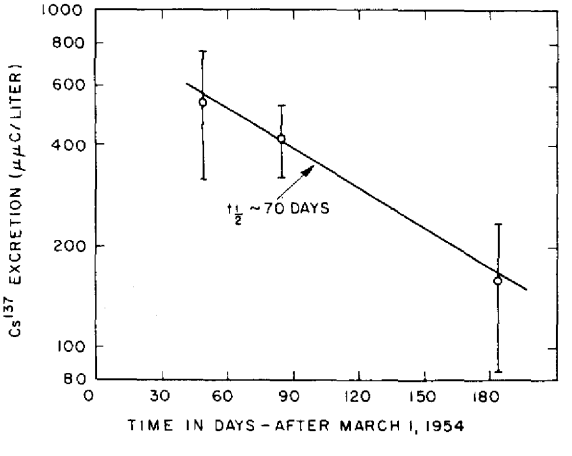

Figure 56. Urinary excretion of Cs^{137} in exposed Marshallese.

level. The slight increase in environmental level of Cs^{137} during the 1956 and 1958 periods of weapon testing was reflected in an increased body burden in the Marshallese. As pointed out, a very marked increase in Cs^{137} was also observed in the Rongelap people after they returned to their original island in 1957: the body burden in 1958 was about 0.68 μC, about 60 times as great as in 1957, and the urinary level rose by a factor of 140, because of the ingestion of Cs^{137} in food on Rongelap during the 9 months since their return. The average Cs^{137} content of 250 Americans studied in 1958 was 6.6 mμC or $\frac{1}{100}$ of the mean Rongelap body burden.[53]

The average daily intake of Cs^{137} for an inhabitant of Rongelap in 1958 (average of 13 daily rations) was estimated to be 3.9 mμC.[47] This is about 1.3% of the nonindustrial maximum permissible daily intake, which is the product of the maximum permissible concentration[57] and the daily intake of water:

$$(2\times10^{-4}\ \mu\text{C/ml})\times(1.5\times10^{3}\ \text{ml/day})$$
$$=300\ \text{m}\mu\text{C/day}.$$

Zinc-65. Zn^{65} was first detected by Miller[5,54] in 1957 in the seven Marshallese examined at Argonne National Laboratory by whole-body spectrometry, although it had been observed in high concentrations in fish as early as one year following the 1954 detonation.[55] Body burdens of Zn^{65} in 1957, measured directly, averaged 44 mμC in five Rongelap inhabitants (Figure 57) and 350 mμC in two Utirik inhabitants. Miller, in 1957, determined an effective half-life of 110 days for the elimination of Zn^{65}, which gives a biological half-life of 200 days. However, a value of 89 days was obtained for the biological half-life in two patients over a 2-month period.[48]

The mean body burden of Zn^{65} estimated from whole-body counting data was 0.36 μC in 1958 after the return of the Rongelap people to their island, or 8 times the 1957 value (Figure 57).

The estimated Zn^{65} intake in food (2 to 4 mμC/day) can be largely accounted for by the Zn^{65} levels reported for fish. In 1956, fish from Rongelap Lagoon were found to contain 0.6 mμC Zn^{65} per lb muscle, or 7.5 mμC per lb whole fish.[56]

The 1959 body burdens of Zn^{65} are presented in Table 35. As with Cs^{137}, the variation within any group is large, and no significant difference is found in Zn^{65} per unit body weight correlated

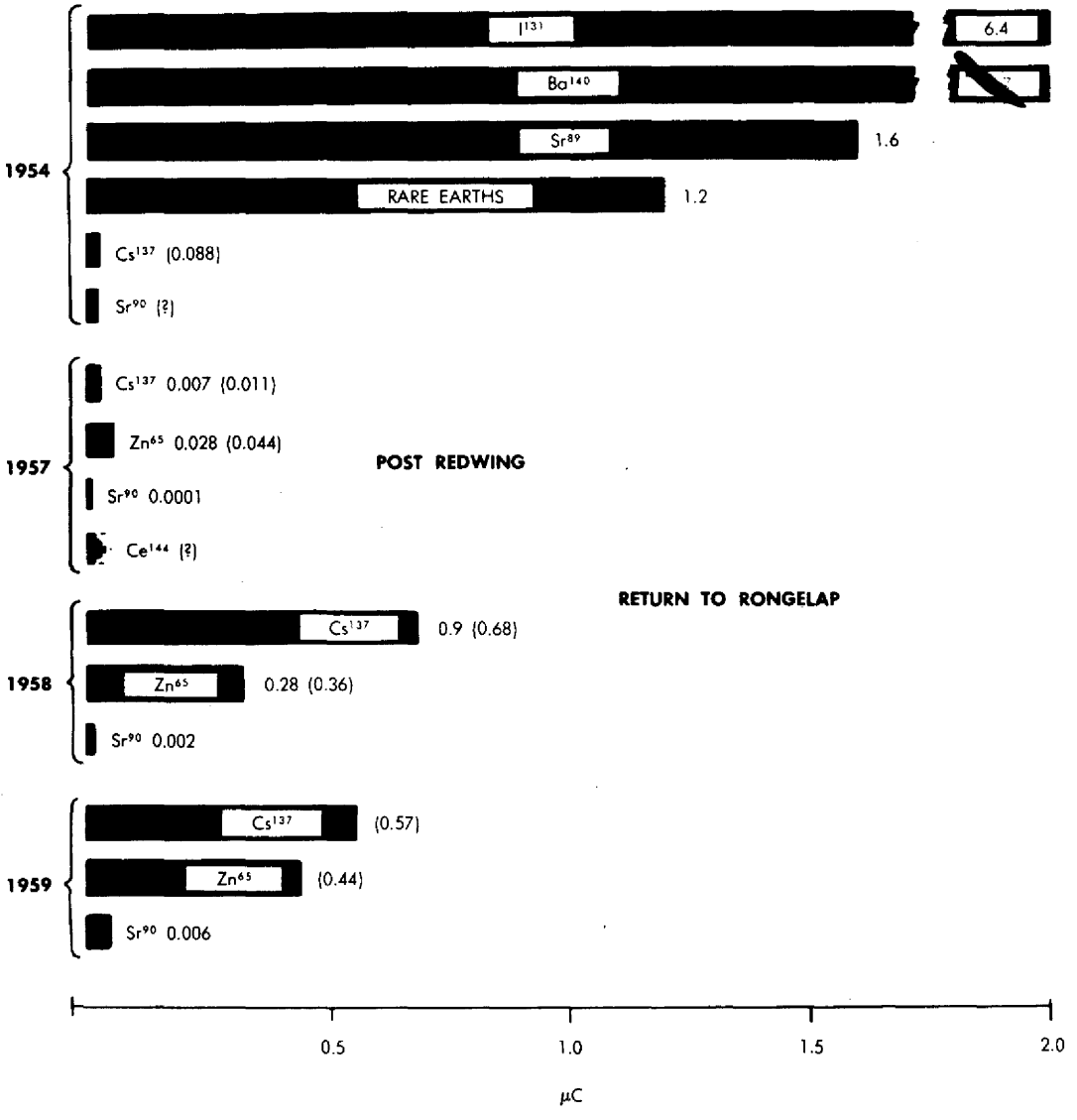

Figure 57. Estimated body burden of isotopes of Rongelap people. Values in parentheses obtained by whole-body gamma spectrometry; other values from urine analysis data.

Table 35

Zn^{65} Body Burdens, in mμC/kg, of Marshallese as Measured by Whole-Body Gamma Sepectroscopy, 1959

	Males		Females	
	Age 1–15	Age >15	Age 1–15	Age >15
Rongelap exposed	10.6±2.2 (9)*	9.9±3.1 (10)	8.6±2.6 (12)	9.4±3.3 (16)
Rongelap unexposed	7.9±2.0 (20)	9.9±2.9 (37)	9.3±2.8 (16)	8.7±2.2 (33)
Ailingnae	8.0 (2)	14.4±5.7 (4)	8.1±2.1 (4)	10.5±2.6 (5)
Utirik	2.5±0.8 (14)	4.2±1.6 (16)	2.8±0.8 (14)	2.5±0.6 (15)

*The numbers in parentheses are the numbers of people in the groups.

Table 36

Residual Gamma Activity, in counts/min/kg, in Marshallese After Subtraction of K^{40}, Zn^{65}, and Cs^{137}

	Age, yr	
	1–15	>15
Rongelap exposed	36.1	37.9
Rongelap unexposed	21.4	35.0
Ailingnae	17.2	43.0
Utirik	8.8	11.7

with age or sex. No significant difference was found between the Rongelap exposed and unexposed groups, which implies that no residual Zn^{65} activity remains in the Rongelap people from their original exposure.

The 1959 mean body burden of Zn^{65} was 0.44 μC as compared to 0.36 μC in 1958. Thus Zn^{65} body burdens do not seem to have reached a steady-state equilibrium with the environment, as is also the case with Cs^{137}. Since the source of Zn^{65} is fish, which continue to be a dietary staple, the Zn^{65} value can be expected to increase still further. The mean Utirik Zn^{65} level in 1959 was about one-third the Rongelap mean value.

Although Cs^{137} and Zn^{65} comprise the major portion of gamma-emitting radionuclides present in the Marshallese (aside from the naturally occurring K^{40}), residual gamma activity is still present after subtraction of K^{40}, Zn^{65}, and Cs^{137} contributions from the total spectrum in each subject (see Table 36). Analysis of the residual spectra did not indicate any readily identifiable photopeaks in the short counting time employed (5 or 10 min). This short counting time, along with the difficulties discussed earlier in exact calibration of absolute activities, makes the identification of minor photopeaks very difficult. Most of the difficulties can be circumvented in future field trips by the use of longer counting times, the use of an 8-in. crystal, duplication of the geometry by use of the same standard chair, and a more precise calibration of the phantom.

Summary and Conclusions

Continuing annual medical surveys of the people of Rongelap Island were carried out in March 1959 and March 1960, 5 and 6 years after their accidental exposure to fallout. During the 1959 survey 76 exposed persons, including their children, and 166 unexposed Rongelap people, who served as a comparison population, were examined. In addition, groups of children at Utirik, Majuro, and Kwajalein Atolls were examined as controls for the growth and development studies on the exposed Rongelap children. The 1960 survey was brief, only the exposed people being examined.

As a result of their exposure in 1954, many of the Rongelap people had experienced early symptoms related to the gastrointestinal tract and skin. Later they developed a significant depression of their peripheral blood elements commensurate with the calculated dose of gamma radiation (175 r to 64 people and 69 r to 18 people), and beta burns of the skin along with spotty epilation. In addition, radiochemical analyses of urine samples showed that they had acquired a low-level body burden of radionuclides. Certain other findings were possibly related to their radiation exposure, such as loss in weight of several pounds in most of the people during the first several months after exposure and suggestive evidence of slight lag in growth and development of the children based on studies of height, weight, and bone development (but inconclusive pending verification of exact ages of some of the children).

In spite of the depression of hemopoiesis, no signs of radiation illness developed in the people related to such depression, and no deaths occurred that could be related to their radiation exposure. No specific therapy was given. Recovery of the peripheral blood elements, particularly lymphocytes and platelets, was very slow over the ensuing years. The beta burns, which appeared about 2 weeks after exposure, were, for the most part, superficial in nature and healed in several weeks, with only a few lesions showing later persisting changes. Specific therapy was not necessary in most cases. The hair regrew normally, beginning at 3 months after exposure. The internally absorbed radionuclides caused no known acute effects and were excreted remarkably fast with barely detectable activity being found a year or two later. On return of the people to Rongelap Island the very low levels of radioactive contamination remaining there resulted in a rise in their body burdens of cesium-137, zinc-65, and strontium-90.

The 5- and 6-year post-exposure surveys were aimed primarily at evaluating the general medical

status of the people in relation to that of the unexposed comparison population, particularly in regard to any possible late developing effects of their exposure, their hematological status, and the influence of the slightly contaminated environment on the assimilation, excretion, and body burden of radionuclides.

Medical histories of the intervals between the examinations in 1959 and 1960 were, for the most part, uneventful and revealed no major epidemics of disease. Several deaths had occurred and new births were reported. In the exposed group, two deaths occurred and in the unexposed group, three. Two of the latter were due to influenza acquired during an epidemic on Kwajalein.

The four deaths that have occurred in the exposed people since exposure represent a *mortality rate* of 8.1 per 1000 population, compared with 8.3 for the comparison population and 6.8 for the Marshall Islands as a whole. A review of the *birth rate* of the exposed group over the past 6 years seems to indicate no noticeable effect of their exposure on fertility. The 24 births represent a rate of 48 per 1000 population, compared with 37.3 for the Marshall Islands (1957). The 20 births over a 3-year period for the comparison population represent a rate of 62 per 1000 population. A somewhat greater incidence of *miscarriages and stillbirths* has been noted in the exposed women, but because of the paucity of vital statistics in the Marshallese and the small number of people involved, the data are not readily amenable to statistical analysis.

Physical examinations showed the exposed and the unexposed people to be generally in a state of good health. No diseases were noted that could be directly related to radiation effects. The incidence of various disorders in both adults and children was about the same in the exposed and unexposed groups.

In connection with *growth and development studies*, a project on the verification of accuracy of ages of the children has not been completed and, therefore, the suggestive evidence previously presented of possible lag in statural growth in the exposed children must await confirmation. It was noted, however, that in the 6-year chronological age group, three boys and one girl out of five boys and two girls in the exposed group exhibited significantly retarded skeletal maturation as judged by x-ray examination. The birth dates of these children seemed to be fairly well established.

A *cardiovascular survey* of the adults showed no outstanding differences between the exposed and unexposed groups. The people appeared to have less hypertension on the whole than is noted in people in the continental United States.

An *arthritis survey* showed no great differences between the exposed and the unexposed people, and about the same incidence as is seen in American populations.

An *ophthalmological survey* showed no remarkable differences between the exposed and unexposed groups except possibly a slightly greater number of cases of pterygii, pingueculae, and corneal scars in the exposed group. It is not know whether this finding is of any significance in relation to their radiation exposure. Slit-lamp observations showed no opacities of the lens characteristic of radiation exposure. As a whole, visual and accomodation levels in the Marshallese appeared to be above the average in the U.S. population.

A *dental survey* showed no significant differences in either caries rate or incidence of peridontal disease between exposed and unexposed groups. The poor oral hygiene generally observed in the Marshallese had its usual results, namely, high caries rate in teen-age children, severe peridontal lesions in adults (heavy calculus and loss of alveolar bone), and edentulous mouths in the aged. Radiation exposure did not appear to have affected developing dentition in the exposed children.

Late effects of radiation. Various parameters usually associated with *aging* were measured or estimated on a 0 to 4+ scale (skin looseness, elasticity, and senile changes; greying of the hair and balding; accomodation, visual acuity, and arcus senilis; hearing; cardiovascular changes including blood pressure and degrees of peripheral and retinal arteriosclerosis; neuromuscular function; and hand strength). Comparison of these measurements in exposed and unexposed individuals of the same age groups showed no apparent differences. A biological age score was calculated for indiviuals and groups by use of an average percentage score. *Life shortening* effects of radiation have not been apparent. As noted, the mortality rate was about the same in the exposed as in the unexposed people.

The one case of *cancer* that developed in the exposed group occurred at 5 years after exposure, too soon, it is believed, to bear any particular relation to radiation exposure. *Leukemia* surveys including physical findings, studies of white cell

counts and types, alkaline phosphatase staining, and basophil counts of 4000 white cells showed no evidence of leukemia or leukemic tendency. One child in the irradiated group had 3% basophils but no other positive findings. The *cardiovascular* and *arthritis* surveys, as well as the general results of the physical examinations, have not shown any apparent increased incidence of *degenerative diseases* in the exposed people. No radiation-induced *cataracts* have been observed in any of the exposed people.

Genetic effects have not been specifically studied because of the small number of people involved. No apparent radiation-induced genetic changes have been detected on routine physical examination in the first-generation children of exposed parents.

Hematological surveys again showed considerable fluctuation in the year-to-year mean level of leukocytes in both the exposed and unexposed groups. The mean *leukocyte* level of the exposed group showed a marked decrease at the time of the 1960 survey (no unexposed people were examined). The reasons for these fluctuations are not apparent. At 5 years post exposure, exposed people still had mean *platelet* levels 10 to 15% below those of the unexposed group. However, *lymphocyte* levels appeared for the first time to equal those of the unexposed group. Mean *erythrocyte* levels were also slightly lower in the exposed people. These blood elements in the Ailingnae group also showed some slight depression below the unexposed levels but not quite so marked as seen in the Rongelap exposed group. A general anemic tendency was noted in all the Marshallese, both exposed and unexposed. *Price-Jones curves*, on the average, showed a slight microcytic tendency. *Serum iron* levels were generally normal. The fact that some of the blood elements in the exposed group have not yet returned to the levels in the unexposed group raises the possibility that a residual radiation effect on the bone marrow persists, but other, not immediately apparent, factors may be involved.

Studies of genetically inherited characteristics. Blood grouping studies in the Marshallese showed a relatively high B gene frequency, a high N gene frequency, an extremely high R^1 gene frequency, and total absence of Kell and Diego factors. These characteristics differ from those of Polynesians and suggest relationship with Southeast Asians and Indonesians. *Haptoglobin studies* showed the frequency of the Hp^1 gene to be higher than in European populations thus far tested and consistent with populations living near the equator. The distribution of haptoglobin types showed the population to be relatively homogeneous. *Transferrins* in all sera were type CC, the common European type. *β-Amino-iso-butyric acid* urinary levels showed the Marshallese to be the highest excretors of this acid of any population thus far reported. Levels in the exposed group were about the same as in the unexposed group, and no correlation was found with body burden level of radionuclides; this indicates that there is probably no correlation with radiation exposure. *Glucose-6-phosphate dehydrogenase* of the red cells appeared to be deficient in the Marshallese. Considerable caution must be exercised in evaluating the results of these studies on genetically inherited characteristics because of the small number of samples tested. The data do seem to indicate relative homogeity of the population and closest kinship with people of Southeast Asia. These data also may be useful as a base line should genetic changes appear in later generations, possibly related to radiation exposure.

Results of other laboratory studies carried out during the 5-year survey included the following: *Serum protein* levels, as has been noted before, were generally on the high side of normal; the reason for this is not apparent. *Complement fixation studies* for parainfluenza 1, 2, and 3, respiratory syncitial, psittacosis, and Q fever showed antibodies to all groups of viruses except that for Asian influenza, which probably had not yet seriously involved the people of the Marshall Islands. The antibody titers appeared to be somewhat lower in the exposed people. *Sodium levels* in the urine and food indicated about the same consumption of NaCl as in Americans. The generally lower incidence of hypertension in the Marshallese might be related to the fact that the former native diet was probably lower in salt content than the present, more westernized diet. It will be interesting to see whether the incidence of hypertension will later increase. Repeat studies of *protein-bound iodine, total iodine,* and *butanol-extractable iodine* of the sera showed levels lower than previously reported, and the previous higher readings are thought to be in error because of contaminated glassware, although some readings were still somewhat high. Four cases of *glucosuria* associated with elevated *blood sugar* were found in the unexposed population, which indicated a rather

high incidence of diabetes. Serum assay for *vitamin* B_{12} showed generally high levels; the explanation was not apparent.

Radionuclide body burden evaluation in the Marshallese people has been complicated by several things. The people were evacuated from their island soon after the accident and did not return until 3 years later. During the 5 years since the original contaminating event, additional weapons tests held in the area have contributed to the fission products in the environment. Finally, since the diet includes a variety of imported foods, the people are not living in a "closed" environment, and therefore may not be rapidly approaching equilibrium with the environmental fission products, as might be expected under other circumstances.

Body burdens of gamma-emitting fission products (such as Cs^{137} and Zn^{65}) were measured in a whole-body counter and checked by radiochemical analysis of urine specimens. Body burdens of Sr^{90} were estimated from urinary excretion as determined by radiochemical analyses. Both the external dose measurements on Rongelap Island and the levels of radioactive isotopes in the food on the island indicated that some increase in Cs^{137}, Zn^{65}, and Sr^{90} body burdens was to be expected when the people returned there in 1957. The Cs^{137} body burden in 1958 was about 0.68 μC, about 60 times as great as in 1957, and the urinary Cs^{137} level rose by a factor of 140; the mean body burden for 1959 was 0.57 μC. The mean body burden of Zn^{65} estimated from whole-body counting data was, in 1958, after the return to Rongelap, 0.36 μC, 8 times as high as in 1957, and 0.44 μC in 1959. Thus, whereas the Cs^{137} levels appear to have reached a maximum and actually to have dropped in 1959, the Zn^{65} levels have shown a continued increase which is probably related to the long biological half-life of the latter. The Sr^{90} level in 1958 estimated from excretion data was 2 mμC, about 20 times as high as in 1957 before the return to Rongelap. The estimated body burden in 1959 increased to 6.0 mμC, about 20% of the estimated ultimate equilibrium value. Little of the body burden of the exposed group is apparently due to their initial exposure, since at present there is little difference between the levels of the exposed and unexposed populations living on Rongelap Island. When these three isotopes have reached their estimated equilibrium values, the body burdens will still be of small significance in terms of radiation hazard.

This study of the internal contamination of the Marshallese has provided information (1) on the movement of Cs^{137}, Zn^{65}, and Sr^{90} from the environment to man; (2) on the rate of equilibration of these isotopes with the environment; and (3) on the discrimination factors between food and man.

IMPORTANCE OF MEDICAL SURVEYS

The Japanese populations of Hiroshima and Nagasaki being studied by the Atomic Bomb Casualty Commission and the Marshallese population of Rongelap comprise the only large groups of people exposed to acute doses of ionizing radiation. The two studies parallel each other but differ in certain important respects. The smaller number of people in the Marshallese population and the paucity of vital statistics make statistical analysis of data on this group much more difficult. However, the Marshallese studies have the advantages that the dose of radiation received by the people is better known; that the findings during the early, acute period after exposure are well documented; and that the people did not suffer from trauma, thermal burns, or marked psychic disturbance, although they did have the complications of beta burns and internal absorption of radionuclides. Study of both groups has yielded valuable information on the acute effects in human beings of radiations from atomic bomb detonations. Examinations for the more subtle late effects of radiation exposure are now receiving considerable emphasis. In the case of the Japanese, increased incidence of leukemia and possibly other malignancies as well as cataracts already has been reported. In view of this finding, the next 5 years will be the critical period for the development of leukemia in the Marshallese. Animal experimentation has indicated the possibility that still other late effects may occur in the human being such as premature aging, shortening of life span, increase in degenerative diseases, genetic changes, etc. Therefore, continued careful examination of these populations is extremely important in order that such effects may be documented, and therapeutic procedures instituted wherever possible, should such effects develop. In addition, in the case of the Marshallese, continued evaluation is indicated of the influence of persisting low levels of radioactive materials on Rongelap Atoll on the body burdens of radionuclides of the people living there.

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APPENDIX 1

Rongelap Group and Control Mean Blood Counts by Day and by Age

Postexposure day	WBC ($\times 10^{-3}$) <5	WBC ($\times 10^{-3}$) >5	Neutrophils ($\times 10^{-3}$) <5	Neutrophils ($\times 10^{-3}$) >5	Lymphocytes ($\times 10^{-3}$) <5	Lymphocytes ($\times 10^{-3}$) >5	Platelets ($\times 10^{-4}$) Male <10	Male >10	Female all ages	Total group	Monocytes ($\times 10^{-2}$) <5	Monocytes ($\times 10^{-2}$) >5	Eosinophils ($\times 10^{-2}$) <5	Eosinophils ($\times 10^{-2}$) >5
3	9.0	8.2	6.4	4.7	1.8	2.2	—	—	—	—	0.8	0.3	0.1	0.7
7	4.9	6.2	—	—	—	—	—	—	—	—	—	—	—	—
10	6.6	7.1	3.5	4.5	2.6	2.1	28.2	22.7	24.9	24.8	2.9	1.7	1.6	1.6
12	5.9	6.3	3.5	3.9	2.1	1.7	—	—	—	—	4.2	5.4	1.9	1.9
15	5.9	6.5	3.2	4.1	2.4	1.9	27.1	21.3	21.7	22.5	3.0	2.3	1.1	1.3
18	6.7	7.2	3.4	4.7	2.4	2.1	21.8	19.1	21.8	21.0	2.7	1.7	3.5	1.6
22	7.0	7.4	4.3	5.0	2.6	2.1	16.8	14.6	15.2	15.3	1.9	2.0	2.3	1.8
26	5.7	6.1	3.0	3.9	2.3	1.8	13.2	12.9	10.9	11.9	1.9	1.6	1.8	1.3
30	7.6	7.8	4.0	5.3	3.2	2.1	14.1	12.3	11.8	12.3	1.5	0.9	3.4	2.2
33	6.5	6.2	3.1	3.8	3.2	2.0	17.9	16.6	15.1	16.0	1.7	1.6	2.6	2.2
39	5.7	5.5	3.0	3.3	2.6	2.0	25.5	22.0	22.4	22.8	0.9	0.9	0.5	1.0
43	5.2	5.2	2.0	2.6	2.9	2.3	26.8	20.9	23.2	23.2	1.1	1.1	1.4	0.8
47	5.9	5.8	2.6	3.3	3.1	2.4	24.6	20.6	23.9	23.1	1.0	1.0	1.1	0.5
51	6.7	5.6	2.6	3.5	3.4	2.1	22.1	17.5	21.2	20.3	2.5	1.6	0.8	0.7
56	7.0	6.0	3.5	3.5	3.7	2.4	—	—	—	—	1.7	1.2	—	—
63	7.7	6.0	3.9	3.6	3.7	2.3	23.1	18.2	20.2	20.1	0.5	0.9	0.3	0.6
70	7.6	6.5	3.8	4.0	3.3	2.2	—	—	—	—	—	—	3.4	1.9
74	—	—	—	—	—	—	26.2	21.7	24.7	24.1	—	—	—	—
6-mo survey	8.5	6.6	4.6	4.2	3.6	2.2	24.4	20.3	23.2	22.6	1.4	1.1	2.5	1.6
1-yr survey	10.1	8.1	4.7	4.8	4.6	2.8	26.6	19.5	27.6	24.9	0.7	1.3	6.7	2.8
2-yr survey	11.8	8.6	5.9	4.8	4.7	3.1	30.0	21.4	25.5	24.7	2.7	1.5	9.6	5.3
3-yr survey	8.6	6.9	4.1	3.7	3.7	2.7	32.0	22.1	28.1	—	1.2	0.7	6.4	4.5
4-yr survey	8.9	7.5	3.3	3.4	4.6	3.6	32.5	27.1	30.8	—	1.5	1.1	7.9	4.0
5-yr survey	13.5	9.5	6.9	4.8	6.0	4.0	32.3	24.4	27.6	—	2.7	2.0	7.0	5.0
6-yr survey	—	6.5	—	3.5	—	2.7	—	—	—	—	—	0.6	—	2.7
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	2.0	2.0	9.5	4.7
Rita controls, 6 mo	10.7	7.6	5.4	5.2	4.7	3.7	35.0	27.3	30.9	30.4	1.9	1.7	4.2	4.8
Rita controls, 1 yr	—	—	—	—	—	—	37.5	24.5	29.4*	27.6	—	—	—	—
Rita controls, 2 yr	14.0	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	1.4	1.5	12.8	6.6
Rongelap controls, 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	—	1.4	0.7	6.2	4.0
Rongelap controls, 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	2.3	1.1	7.0	4.5
Rongelap controls, 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	3.7	2.4	6.2	6.0

*Excluding pregnancy.

APPENDIX 2

Ailingnae Group and Control Mean Blood Counts by Day and by Age

	WBC ($\times 10^{-3}$)		Neutrophils ($\times 10^{-3}$)		Lymphocytes ($\times 10^{-3}$)		Platelets ($\times 10^{-4}$)				Monocytes ($\times 10^{-2}$)		Eosinophils ($\times 10^{-2}$)	
Postexposure day	<5	>5	<5	>5	<5	>5	Male <10	Male >10	Female all ages	Total group	<5	>5	<5	>5
3	6.0	7.0	3.0	5.0	2.8	2.2	—	—	—	—	0.8	1.6	0.5	0.4
7	5.5	6.8	—	—	—	—	—	—	—	—	—	—	—	—
10	6.3	7.3	4.2	4.2	1.9	2.2	22.5	22.6	20.9	21.5	3.8	2.1	2.6	1.6
12	6.3	7.6	1.8	4.7	3.1	2.2	—	—	—	—	3.4	5.8	4.4	2.6
15	7.1	7.0	2.3	4.5	4.2	2.2	29.0	20.2	24.6	23.9	3.7	2.6	2.3	1.4
18	6.8	7.8	2.9	5.0	3.5	2.4	27.5	21.7	24.9	24.3	2.3	1.5	3.2	2.3
22	8.9	8.7	5.3	5.4	2.7	2.9	23.5	17.0	22.9	21.3	1.5	2.4	5.8	2.4
26	8.4	7.0	4.8	4.4	3.2	2.2	20.0	13.8	17.4	16.7	2.3	2.4	0.6	1.6
30	9.6	8.6	5.3	6.2	3.7	2.0	19.5	12.8	18.2	16.8	1.9	1.9	4.1	2.0
33	7.7	7.8	3.3	5.2	3.5	2.2	24.0	15.8	22.7	17.6	2.8	2.2	6.0	1.9
39	7.5	6.2	2.9	4.2	4.7	1.9	26.5	20.8	27.0	25.2	1.1	1.7	2.7	1.6
43	6.9	6.5	2.7	3.6	3.9	2.7	28.0	19.6	25.3	24.0	0.6	1.4	2.8	0.6
47	7.3	6.7	3.5	3.8	3.4	2.7	27.0	20.0	26.1	24.5	2.2	1.9	1.5	0.7
51	8.4	6.3	3.8	3.6	4.0	2.2	32.0	18.2	25.0	23.9	2.7	2.8	2.2	1.0
54	4.6	6.3	2.8	3.5	3.2	2.5	37.0	19.8	23.8	24.2	1.5	1.9	1.8	0.8
6-mo survey	7.7	6.5	4.8	3.9	2.7	2.2	25.2	19.2	23.9	22.7	1.1	1.4	1.5	2.2
1-yr survey	11.1	7.8	4.2	4.7	6.5	5.6	38.7	21.4	28.3	27.5	1.0	1.1	1.7	2.2
2-yr survey	11.0	9.1	4.9	5.1	4.8	3.2	51.2	17.4	26.4	26.7	3.6	1.4	9.6	6.4
3-yr survey	12.1	7.0	5.5	3.9	5.6	2.6	40.8	22.4	31.2	—	3.0	0.7	5.3	3.7
4-yr survey	11.5	7.5	2.8	3.7	7.0	3.3	33.2	24.7	33.6	—	2.2	1.1	12.6	4.2
5-yr survey	—	9.7	—	5.1	—	3.7	40.9	26.3	26.8	—	—	3.2	—	6.0
6-yr survey	—	7.3	—	3.6	—	3.0	—	—	—	—	—	0.6	—	4.0
Majuro controls	13.2	9.7	4.8	4.8	7.4	4.1	41.2	25.8	36.5	33.4	2.0	2.0	9.5	4.7
Rita controls, 2 yr	14.1	8.9	7.0	4.4	5.6	3.6	35.5	24.2	31.2	29.5	1.4	1.5	12.8	6.6
Rongelap controls, 3 yr	9.8	6.9	4.0	3.4	4.7	2.9	32.6	26.9	30.0	—	1.4	0.7	6.2	4.0
Rongelap controls, 4 yr	11.2	8.0	4.0	3.6	6.2	3.7	38.8	30.7	34.0	—	2.3	1.1	7.0	4.5
Rongelap controls, 5 yr	13.7	10.1	6.2	5.2	6.2	4.1	35.8	28.0	33.6	—	3.7	2.4	6.2	6.0

APPENDIX 3

Hematological Findings, 1959

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Plate. ($\times 10^{-3}$)	RBC ($\times 10^{-6}$)	% Retic.	Hgb., g	MCH, g ($\times 10^{-12}$)
					Rongelap and Ailingnae Exposed Population							
1*	59 F	6.9	2.8	4.0	0.2	0	0	275.5	4.35	0	13.9	32.0
2	6 M	7.3	2.4	3.9	0.7	0	0.2	255.0	4.14	0.2	12.5	30.0
3	6 M	8.6	3.4	4.9	0.3	0	0	240.0	4.30	0.1	11.7	27.0
4	43 M	8.4	3.6	4.6	0.2	0.05	0.1	277.5	5.08	0.8	15.0	30.0
5	6 M	10.4	5.7	3.9	0.5	0.1	0.1	307.5	4.37	0	11.6	26.5
6*	6 M	9.0	2.5	5.1	1.1	0.2	0.1	397.5	4.11	0.1	12.5	30.5
7	41 M	6.0	1.3	4.3	0.2	0.1	0.1	207.5	4.40	0	14.6	33.0
9	27 M	9.6	5.5	3.7	0.15	0.15	0.1	192.5	4.74	0	15.1	32.0
10	29 M	8.4	4.8	3.1	0.5	0	0	200.0	5.47	1.0	15.0	27.0
11	55 M	7.5	3.6	3.6	0.2	0.05	0.1	257.5	4.48	0.4	14.1	31.0
12	23 F	9.2	4.3	4.4	0.2	0	0.3	260.0	3.89	0	12.7	32.5
13	63 F	8.6	4.1	3.9	0.5	0	0.1	310.0	3.95	0.4	12.9	32.5
14	30 F	6.0	4.2	1.6	0.2	0	0	200.0	3.11	0	12.9	41.5
15	11 F	8.3	3.2	4.1	0.6	0	0.4	410.0	4.48	0.2	11.9	26.5
16*	44 M	7.1	3.0	3.5	0.4	0.1	0.2	235.0	5.96	0	15.4	26.0
17	8 F	8.5	3.1	4.5	0.8	0	0.1	205.0	4.44	0	12.4	28.0
18	26 F	10.3	7.2	2.4	0.3	0.05	0.4	280.0	4.43	0.3	12.0	27.0
19	10 M	8.8	5.5	2.5	0.8	0.2	0.1	317.5	5.51	0.1	12.5	22.5
20	12 M	8.4	4.5	3.2	0.6	0.1	0.05	335.0	4.79	0	12.8	27.0
21	8 F	10.3	2.8	6.6	0.7	0.05	0.2	315.0	4.55	0	12.7	28.0
22	22 F	8.9	4.3	3.5	0.3	0.1	0.5	292.5	4.04	0	12.2	30.0
23	9 M	11.2	4.4	5.8	0.8	0	0.1	312.5	4.74	0.3	12.2	26.0
24	18 F	11.9	6.8	4.4	0.4	0	0.15	235.0	4.67	0	13.9	30.0
26	17 M	11.3	5.6	4.7	0.7	0.05	0.15	522.5	5.02	0	12.8	25.5
27	31 M	15.7	8.2	6.3	0.8	0.1	0.25	245.0	4.18	0.2	14.7	35.0
28*	73 F	15.0	9.9	4.4	0.6	0	0.05	250.0	4.60	0.4	14.3	29.0
29*	70 M	9.0	5.6	2.5	0.55	0	0.3	337.5	4.53	0.4	14.1	31.0
30	64 F	7.7	5.1	2.4	0.2	0	0	245.0	4.06	0	12.4	30.5
32	8 M	8.8	3.6	4.3	0.8	0	0.1	540.0	3.70	0	12.9	35.0
33	7 F	13.2	4.5	7.2	1.2	0	0.3	375.0	4.18	0.1	12.4	30.0
34	50 F	5.6	2.2	3.2	0.1	0.1	0.15	—	4.00	0	10.0	25.0
35	18 M	11.1	7.6	3.0	0.4	0	0.1	207.0	4.76	0	15.4	33.0
36	12 M	7.0	3.4	3.6	0.3	0	0.1	307.5	4.62	0.2	12.3	27.0
37	25 M	10.7	4.4	6.1	0.2	0	0	211.0	5.50	0	17.0	31.0
39	20 F	12.4	8.3	2.4	0.7	0.1	1.0	285.0	3.88	0	12.2	31.0
40	34 M	12.4	6.3	5.0	0.5	0	0.6	317.5	4.72	0.4	14.2	30.0
41*	49 M	7.5	4.8	2.8	0.8	0	0.05	197.5	4.91	0.3	15.6	32.0
42	8 F	14.0	5.4	6.6	1.8	0.05	0.2	325.0	4.15	0	13.2	32.0
43*	71 F	11.1	4.7	3.1	0	0	0.7	230.0	3.98	0.4	11.6	29.0
44*	9 M	12.0	6.1	4.9	1.3	0	0.3	420.0	4.81	0	12.0	25.0
45*	37 F	8.5	4.0	2.7	0.8	0.15	0.4	230.0	3.86	0	12.7	33.0
46	81 M	7.8	4.8	2.0	0.5	0.1	0.5	210.0	3.94	0.6	13.0	33.0
47	13 M	10.8	5.8	4.8	0.05	0	0.2	325.0	4.06	0.1	12.7	31.0
48*	11 F	13.1	8.1	4.4	0.2	0.1	0.3	312.5	4.19	0	12.7	30.0
49	20 F	16.7	11.9	3.8	0.9	0	0.2	285.0	3.45	0.4	11.0	32.0
50*	39 M	9.2	5.4	2.9	0.3	0	0.6	280.0	5.21	0.5	16.6	32.0
52	61 F	10.3	5.5	4.3	0.9	0.05	0.05	340.0	4.50	0	13.7	30.0
53*	13 F	10.4	6.0	4.1	0.3	0.05	0.05	330.0	4.48	0.1	13.0	29.0

*Ailingnae exposed.

Hematological Findings, 1959

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Plate. ($\times 10^{-3}$)	RBC ($\times 10^{-6}$)	% Retic.	Hgb., g	MCH, g ($\times 10^{-12}$)
					Rongelap and Ailingnae Exposed Population (continued)							
54	6 M	9.6	3.1	5.8	0.7	0	0.1	290.0	4.30	0	11.8	27.5
55	80 M	9.3	4.7	3.9	0.6	0	0.1	150.0	3.56	0	11.6	32.5
56	76 F	7.7	4.4	2.7	0.3	0.1	0.2	415.0	4.41	0.6	11.6	26.0
57	105 F	5.4	2.3	2.9	0	0	0.2	170.0	4.26	0.4	10.7	25.0
58	64 F	7.6	4.1	3.3	0.2	0	0.2	162.5	3.89	0.6	13.8	35.5
59*	39 F	12.4	4.0	6.0	1.4	0.05	0.5	255.0	3.88	0	13.0	33.5
60	61 F	15.0	7.5	5.7	1.3	0	0.5	270.0	4.08	0	12.5	30.5
61	13 F	9.0	4.4	4.5	0.1	0	0	255.0	4.98	1.0	12.5	25.0
62	62 F	10.3	9.2	0.5	0.5	0	0.1	536.0	4.60	0	11.3	27.0
63	41 F	10.0	5.6	3.4	0.8	0.1	0.1	180.0	4.19	0	12.9	31.0
64	35 F	8.8	2.3	6.1	0.1	0	0.3	220.0	4.00	0	12.9	32.0
65	6 F	10.4	4.5	4.9	0.4	0.1	0.5	305.0	4.16	0.4	12.3	29.5
66	35 F	8.9	4.6	4.0	0.25	0.05	0.25	280.0	4.18	0.2	13.1	31.0
68	50 M	5.9	1.9	3.8	0.1	0	0.1	—	5.10	0	14.5	28.5
69	9 F	11.0	4.3	5.8	0.8	0	0.1	362.0	4.06	0.2	12.0	29.0
70*	22 F	7.5	4.9	1.9	0.15	0.05	0.1	242.5	4.66	0	13.5	29.0
71	33 F	10.3	4.9	4.7	0.5	0	0	260.0	4.76	0	15.3	32.0
72	11 F	10.4	5.6	4.3	0.5	0	0	337.5	4.58	0.2	12.2	27.0
73	23 M	8.7	4.9	2.4	0.3	0.2	0.2	—	4.50	0	12.5	28.0
75	17 F	8.7	4.6	3.4	0.5	0	0.2	—	4.41	0	12.0	27.0
76	16 M	6.4	1.8	4.3	0.15	0	0.1	215.0	5.03	0	14.1	28.0
77	31 M	7.8	4.4	2.2	0.5	0.1	0.6	205.0	5.29	0.4	16.7	31.0
78	42 F	8.6	4.5	3.2	0.3	0	0.5	295.0	4.29	0	13.5	31.0
79	44 M	13.7	9.2	4.1	0.1	0	0.3	147.5	4.57	0	15.3	33.5
80	51 M	10.6	6.0	3.9	0.5	0	0.1	338.0	5.22	0.6	15.4	29.5
81*	13 F	6.3	1.9	3.5	0.55	0.1	0.3	302.5	4.86	0.3	14.2	29.0
82	55 M	8.7	4.0	3.9	0.75	0.1	0.1	237.5	4.01	0.2	13.7	34.0
					Nonexposed Children of Exposed Parents							
83	5 M	10.8	4.9	4.9	0.7	0	0.3	355.0	4.49	0.2	13.2	29.2
84*	5 M	12.2	6.0	4.8	1.1	0	0.2	400.0	4.22	0.4	11.8	28.0
85	4 M	14.0	6.6	6.0	1.1	0.1	0.1	430.0	4.75	0	13.2	28.0
86	4 F	17.6	12.1	3.2	1.4	0.2	0.7	490.0	4.69	0.2	12.5	26.5
87	4 F	11.1	5.9	4.3	0.7	0	0.2	452.0	4.60	0.9	14.0	29.0
88	3 M	12.6	4.4	7.1	0.9	0	0.2	475.0	4.46	0	12.8	28.5
89	3 M	11.9	3.6	6.7	1.2	0	0.5	315.0	4.49	0	12.5	28.0
90	3 M	18.4	13.0	4.8	0.6	0	0	—	3.92	0	10.5	27.0
91	4 M	14.8	9.0	4.7	1.0	0	0	320.0	4.02	0.4	11.7	29.0
92	3 F	14.9	10.3	4.6	0	0	0	—	4.80	0	10.5	22.0
93	2 M	18.0	13.5	4.3	0.2	0	0	330.0	4.18	0.2	10.6	25.5
94	2 F	8.9	4.4	3.6	0.3	0.1	0.5	—	4.53	0.4	13.0	28.5
95	3 F	14.4	4.6	9.6	0.1	0	0	690.0	3.96	0	11.7	29.5
96	1 F	12.4	3.9	8.0	0	0	0.6	470.0	4.54	0	10.9	24.0
97	1 M	11.8	5.3	5.2	0.5	0.1	0.5	550.0	5.04	0	10.5	21.0
98	1 M	13.5	5.1	5.9	1.2	0.3	1.1	370.0	4.80	0.2	10.9	22.5
100	1 F	14.7	7.9	5.0	1.3	0.1	0.4	460.0	4.17	0	10.8	26.0
108	1 F	13.2	1.7	11.3	0.1	0	0	—	3.99	0	9.5	24.0
					Rongelap Control Population							
801	3 M	14.4	5.3	8.8	0.4	0.1	0	144.0	4.49	0.2	12.5	28.0
802	4 M	11.0	4.7	5.3	0.3	0	0.7	110.0	4.91	0	12.2	25.0

*Ailingnae exposed

Hematological Findings, 1959

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-4}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Plate. ($\times 10^{-3}$)	RBC ($\times 10^{-6}$)	% Retic.	Hgb., g	MCH, g ($\times 10^{-12}$)
					Rongelap Control Population (continued)							
803	3 M	13.9	8.5	5.0	0.1	0	0.3	139.0	4.61	0.1	13.6	29.5
805	5 F	15.5	7.9	4.8	2.8	0	0	397.0	5.52	0.8	12.0	21.5
806	4 M	11.1	4.3	5.4	0.8	0.2	0.3	450.0	4.55	0.2	11.8	26.0
807	5 M	20.2	11.3	7.3	1.0	0	0.6	—	3.64	0	10.0	27.5
808	4 F	12.7	6.6	5.0	0.9	0	0.3	535.0	4.84	0.1	13.2	27.0
809	3 M	11.8	5.1	5.3	1.2	0	0.2	600.0	3.95	0	12.5	31.5
810	4 F	9.0	3.5	4.9	0.3	0.1	0.8	340.0	4.44	0.6	12.2	27.5
811	5 F	12.6	6.8	4.3	1.3	0.2	0.05	412.0	4.11	0.4	11.9	29.0
812	5 F	14.7	4.6	6.2	3.1	0	0.9	295.0	4.53	0	12.7	28.0
813	5 M	10.6	4.1	5.8	0.7	0.05	0.2	395.0	4.45	0	12.0	27.0
814	7 M	11.1	6.3	4.4	0.15	0.05	0.05	350.0	4.25	0	13.0	30.5
815	8 M	8.7	4.2	3.7	0.7	0.1	0.1	205.0	4.05	0.2	11.4	28.0
816	9 F	11.8	5.2	5.7	0.6	0	0.1	358.0	4.24	0.1	11.4	27.0
818	8 M	12.3	7.6	3.7	0.6	0	0.15	575.0	4.79	0.4	13.2	27.5
819	10 M	6.4	2.5	3.7	0.6	0.1	0.1	195.0	4.11	0.6	12.1	29.5
820	10 M	12.2	5.6	4.0	2.3	0	0.2	412.0	4.63	0	12.4	27.0
821	10 F	9.1	2.5	6.0	0.2	0	0.3	255.0	4.47	0.1	11.9	26.5
822	13 M	9.6	4.1	4.9	0.5	0	0.05	220.0	4.55	0	14.8	32.5
823	15 M	10.4	5.7	4.1	0.6	0	0	272.0	4.51	0	12.5	27.5
825	17 F	10.8	6.0	4.6	0	0	0.1	410.0	4.76	0.5	13.2	27.5
826	22 F	10.5	7.4	2.6	0.3	0	0.1	—	4.10	0	10.0	24.5
827	19 M	4.5	2.5	1.6	0.2	0.04	0.1	—	4.55	0	12.5	27.5
828	19 M	11.5	2.3	9.0	0.1	0.1	0	289.0	5.10	0.2	15.8	31.0
829	20 F	7.3	4.7	2.6	0	0	0	205.0	4.00	0	11.9	30.0
830	20 M	7.4	3.6	3.2	0.4	0	0.1	283.0	4.93	0.2	15.8	32.0
831	19 M	8.0	3.6	3.4	0.7	0.1	0.4	252.0	5.10	0.3	16.8	33.0
832	21 F	10.8	7.7	2.6	0.3	0	0.15	270.0	4.03	0.2	10.8	27.0
833	26 M	7.9	3.2	4.0	0.3	0.1	0	325.0	5.61	0.8	15.4	27.5
834	25 M	10.1	5.9	2.7	1.0	0	0.4	303.0	5.06	0.1	14.8	29.0
835	25 F	10.1	5.7	3.2	0.7	0.1	0.3	337.0	4.29	0	13.6	31.5
836	26 M	9.5	4.9	4.5	0.2	0	0	280.0	5.40	0.1	16.2	30.0
838	27 M	10.2	3.4	6.0	0.2	0	0.5	225.0	4.75	0.2	16.8	35.5
839	31 F	11.7	7.3	3.9	0.2	0	0.2	392.0	4.24	0.6	11.7	27.5
840	29 M	9.6	3.9	4.7	0.3	0.05	0.5	253.0	5.76	1.4	15.2	26.5
841	26 F	11.2	6.2	3.6	0	0.1	0.5	230.0	4.40	0	12.6	28.5
842	35 M	10.6	5.9	4.0	0.1	0.3	0.3	272.0	5.47	0.5	18.2	33.0
843	30 F	10.2	6.8	2.7	0.6	0.1	0.05	280.0	3.60	0.2	11.9	33.0
844	40 F	9.7	5.7	3.5	0.1	0	0.3	250.0	4.51	0.5	12.5	28.0
845	29 M	8.4	3.3	4.4	0.5	0.1	0.2	257.0	5.00	0	14.1	28.0
846	36 F	12.1	8.5	2.9	0.6	0	0.1	265.0	3.78	0	11.6	30.5
849	40 M	11.9	4.9	6.0	0.7	0.1	0.3	310.0	5.42	1.0	16.8	31.0
850	48 M	11.8	7.3	4.4	0	0	0.1	255.0	4.87	0	15.4	31.5
851	50 F	8.8	4.6	3.5	0.4	0.1	0.2	280.0	4.07	0.2	13.1	32.0
852	55 F	10.9	4.9	5.3	0.5	0	0.3	278.0	4.38	0.2	12.1	27.5
853	54 M	7.6	3.4	3.2	0.5	0	0.4	205.0	4.73	0	15.2	32.0
854	54 F	10.1	6.8	2.9	0.3	0	0.1	372.0	4.73	0.1	13.6	29.0
855	54 M	8.9	5.5	2.4	0.5	0.1	0.4	230.0	4.25	0	14.2	33.5
856	60 M	6.8	3.5	2.7	0.3	0.1	0.2	255.0	4.48	0.4	13.8	31.0
858	64 F	6.6	3.6	3.0	0.1	0	0	295.0	4.30	0.8	12.9	30.0
859	66 F	9.3	4.4	4.5	0.3	0	0.2	395.0	4.12	0.2	12.6	30.5
860	69 M	8.5	3.5	4.1	0.3	0.2	0.4	262.0	3.89	0.1	11.9	30.5
861	69 F	8.3	4.7	3.1	0.2	0	0.4	205.0	4.41	0	13.0	29.5
862	85 M	8.0	4.0	3.5	0.6	0	0	260.0	4.32	1.0	14.2	33.0
863	9 M	10.3	4.0	5.3	0.7	0	0	345.0	4.88	0.6	13.7	27.0
864	33 M	9.7	4.6	4.5	0.7	0.1	0.2	170.0	5.05	0.6	16.0	31.0

Hematological Findings, 1959

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)	Plate. ($\times 10^{-3}$)	RBC ($\times 10^{-6}$)	% Retic.	Hgb., g	MCH, g ($\times 10^{-12}$)
					Rongelap Control Population (continued)							
865	26 F	8.3	4.1	3.5	0.7	0.1	0	465.0	4.53	0	15.1	33.0
866	4 F	10.7	3.9	6.0	0.5	0	0.3	250.0	5.16	0.4	11.7	22.5
867	31 F	10.7	4.1	5.7	0.8	0.1	0.1	340.0	5.07	0.1	15.0	29.5
868	36 M	6.9	3.1	3.0	0.7	0	0.1	263.0	5.19	0	15.8	30.5
869	13 M	11.1	4.9	4.3	1.0	0	0.9	435.0	4.86	0.6	14.2	29.0
870	3 M	16.2	5.2	8.3	2.7	0	0	375.0	4.84	0.6	13.2	27.0
871	46 F	10.0	6.2	3.3	0.2	0	0.3	305.0	4.29	1.0	12.7	29.5
872	16 M	9.1	4.4	3.9	0.3	0	0.5	190.0	4.44	0.8	13.6	30.5
873	40 M	9.4	6.4	2.6	0	0	0.4	285.0	4.45	0.1	13.4	30.0
874	13 M	9.5	5.8	3.2	0.3	0	0.2	300.0	4.54	0	13.0	28.5
875	42 M	7.0	3.5	3.0	0.1	0	0.3	240.0	5.42	0.6	17.7	32.5
876	20 F	7.9	4.3	3.5	0	0	0.2	360.0	4.20	0.1	13.6	32.5
878	59 M	7.9	3.8	3.1	0.5	0	0.5	200.0	4.76	1.0	12.6	26.5
880	27 M	11.6	7.1	4.2	0.2	0	0.1	220.0	5.25	0.4	16.0	30.5
881	26 M	11.7	6.0	4.5	1.2	0	0.1	205.0	4.96	0.8	16.7	33.5
882	47 M	9.0	4.3	4.3	0.3	0.1	0.2	245.0	4.87	0.3	14.8	30.5
884	64 M	9.3	5.4	3.3	0.6	0	0.1	353.0	3.82	0.2	15.4	40.5
886	50 M	12.3	6.3	4.9	1.1	0	0	309.0	4.46	1.4	15.0	33.5
887	14 M	10.5	6.9	3.4	0.1	0.1	0.1	590.0	4.46	0.2	12.9	29.0
889	34 F	7.3	3.9	2.2	0.8	0	0.4	225.0	4.40	0	13.8	31.5
891	11 F	9.5	4.5	4.1	0.9	0	0	380.0	4.62	0.6	14.9	32.0
892	11 M	9.2	4.5	4.2	0.4	0	0.2	310.0	4.45	0	14.0	31.5
893	41 F	6.3	2.9	2.9	0.3	0.1	0.1	228.0	4.16	0.6	12.8	31.0
894	62 F	10.8	8.7	1.5	0.4	0.1	0.1	285.0	4.35	0	14.8	34.0
895	29 F	14.7	7.9	5.3	1.0	0	0.4	485.0	5.07	0	15.4	30.5
896	18 F	12.8	7.9	5.7	0	0	0.3	320.0	4.06	0	13.6	33.5
898	61 F	8.9	2.2	5.4	1.1	0.1	0.2	236.0	3.96	0.3	14.2	36.0
899	65 M	6.5	3.3	2.9	0.3	0	0.1	470.0	5.20	0.4	13.8	26.5
900	2 F	18.4	9.1	6.0	2.0	0.4	0.5	470.0	4.60	0	10.6	23.0
901	2 F	16.7	7.0	9.1	0.3	0	0.3	470.0	5.07	0	11.2	22.0
902	2 F	18.5	7.4	10.1	0.4	0	0.6	292.0	4.48	0.3	9.8	22.0
903	1 F	11.7	5.4	5.5	0.5	0.2	0.1	420.0	4.66	0	7.7	16.5
904	1 M	12.5	6.0	5.2	0.5	0.1	0.7	380.0	4.53	0.3	12.2	27.0
905	2 M	15.3	10.3	4.1	0.3	0.5	0.2	615.0	6.13	0.1	12.9	21.0
906	2 F	13.3	5.2	7.6	0.4	0	0.1	473.0	4.65	0.1	12.2	26.0
908	69 F	8.0	4.2	3.5	0.1	0.1	0.1	290.0	4.03	0.6	13.4	33.0
911	6 F	8.0	3.8	3.5	0.1	0.1	0.6	325.0	3.92	0.1	11.4	29.0
912	6 M	13.5	7.3	5.6	0.5	0	0.1	410.0	4.62	0.6	11.3	24.5
913	8 M	14.3	9.9	2.3	0.6	0.1	1.4	325.0	5.12	0.1	12.7	25.0
914	24 F	6.8	3.8	2.4	0.4	0	0.3	305.0	4.77	0	13.4	28.0
916	35 F	11.4	6.9	3.9	0.1	0.1	0.3	220.0	4.01	0.8	12.2	30.5
921	5 M	38.5	19.2	8.5	10.0	0.4	0.4	207.0	4.68	0.2	13.5	29.0
922	35 F	8.8	6.5	1.9	0.1	0	0.4	335.0	4.27	0.4	11.9	28.0
923	4 F	13.9	8.0	4.3	1.0	0	0.6	462.0	4.69	0.4	11.5	24.5
925	7 F	12.4	6.7	5.0	0.5	0.1	0.1	340.0	4.92	0.4	11.6	23.5
926	8 F	9.9	3.3	6.0	0.4	0.1	0.1	304.0	4.22	0.8	13.0	31.0
927	64 M	8.4	4.7	3.0	0.5	0.1	0.1	210.0	4.28	0.4	15.4	36.0
928	46 F	7.7	3.0	4.1	0.2	0.1	0.3	375.0	3.66	0.2	12.3	33.5
930	3 F	14.2	6.7	7.1	0.4	0	0	442.0	4.65	0.2	11.3	24.5
932	24 F	9.8	6.0	3.7	0.1	0	0	300.0	4.27	0.2	11.1	26.0
933	55 M	8.6	4.6	3.0	0.9	0	0.1	205.0	4.83	0	16.2	33.5
934	24 F	12.8	9.2	2.9	0.1	0	0.5	210.0	4.56	1.2	13.1	28.5
935	61 M	8.7	5.1	2.5	0.5	0	0.7	250.0	442	1.0	15.0	34.0
939	11 M	7.9	4.0	3.3	0.6	0	0	390.0	4.12	0.1	13.6	33.0
940	9 M	16.2	6.3	8.3	0.2	0.3	1.1	390.0	5.02	0.3	13.4	26.5

Hematological Findings, 1959

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-4}$)	Mono. ($\times 10^{-1}$)	Plate. ($\times 10^{-3}$)	RBC ($\times 10^{-6}$)	% Retic.	Hgb., g	MCH, g ($\times 10^{-12}$)
					Rongelap Control Population (continued)							
944	34 M	10.0	4.4	4.5	0.7	0	0.4	175.0	5.87	0	15.0	25.5
946	9 F	10.3	2.1	7.6	0.3	0.2	0.1	290.0	4.84	0.6	12.6	26.0
947	51 M	12.0	7.3	4.2	0.1	0	0	300.0	4.40	0.1	13.6	31.0
952	4 M	13.9	5.9	6.7	0.7	0.1	0.6	540.0	4.51	0	12.1	27.0
953	44 M	9.7	4.5	4.8	0.3	0	0.2	305.0	5.15	0.6	16.0	31.0
955	6 F	7.8	4.1	3.2	0.1	0	0.4	370.0	4.24	0	12.2	29.0
956	50 F	9.1	4.9	3.8	0	0	0.3	305.0	4.34	0.8	13.4	31.0
958	27 M	12.4	7.5	3.7	1.0	0.1	0.1	235.0	4.66	0.2	13.4	29.5
959	11 F	11.5	5.8	4.0	0.5	0	0.1	340.0	4.16	0	12.2	29.5
960	9 F	10.3	4.8	4.3	0.5	0.3	0.3	350.0	4.38	0.2	12.3	28.0
963	41 M	9.6	5.2	2.3	0.4	0.2	0.9	225.0	5.03	0.1	15.4	30.5
966	27 M	7.2	3.8	3.3	0	0.1	0.1	170.0	4.81	0	15.4	32.0
967	15 M	13.6	7.2	6.3	0.1	0	0	330.0	5.03	0.1	14.5	29.0
968	57 F	7.5	2.3	5.0	0.2	0	0.1	163.0	4.23	1.2	—	—
969	41 M	11.2	5.3	5.2	0.3	0.1	0.3	390.0	4.74	0.4	10.1	21.5
971	15 M	8.2	3.9	4.1	0.2	0	0.1	385.0	4.75	0.2	13.6	28.5
973	50 M	6.4	3.4	2.6	0.2	0.1	0.1	310.0	4.99	0.3	16.7	33.5
976	12 M	11.0	6.0	4.6	0.2	0	0.2	400.0	4.61	0.1	12.8	28.0
977	13 F	9.4	3.7	5.5	0.1	0	0.2	325.0	4.76	0.7	14.2	30.0
978	8 F	7.3	2.0	3.5	0.8	0.3	0.6	380.0	4.62	0.1	13.1	28.5
979	3 F	9.8	3.5	5.4	0.7	0	0.2	295.0	4.62	0.6	12.0	26.0
980	6 F	13.0	7.4	4.8	0.5	0	0.4	315.0	4.70	0.1	13.1	28.0
989	11 M	10.6	5.1	4.5	0.9	0	0.1	345.0	5.17	0.6	14.0	27.0
991	51 F	7.6	5.0	2.7	0	0	0.1	178.0	5.08	0.2	15.7	31.0
992	2 F	9.7	3.2	5.7	0.6	0.1	0.2	630.0	4.97	0	8.6	17.5
993	12 F	10.6	5.6	4.4	0.4	0	0.2	245.0	5.25	1.2	13.8	26.0
996	7 F	11.3	3.9	5.7	1.2	0.2	0.2	410.0	4.16	0.1	11.3	27.0
1005	26 M	8.5	4.6	3.2	0.3	0.2	0.3	200.0	5.84	1.0	17.7	30.5
1007	48 M	8.8	5.9	2.7	0.1	0	0.1	190.0	4.87	0.8	12.2	25.0

APPENDIX 4

Hematological Findings, 1960, on Rongelap and Ailingnae Exposed Population

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)
1*	60 F	6.0	2.0	3.5	0.2	0.1	0.2
2	7 M	6.3	2.4	3.5	0.3	0.06	0.06
3	7 M	5.1	2.9	1.8	0.05	0.15	0.15
4	44 M	6.1	1.2	4.5	0.24	0.06	0.18
5	7 M	6.1	2.7	2.8	0.37	0.06	0.12
6*	7 M	9.6	3.6	5.1	0.77	0.1	0.1
7	42 M	5.5	2.6	2.5	0.17	0.11	0.06
8*	6 F	8.7	2.2	6.0	0.35	0.09	0.09
10	30 M	5.6	3.5	1.9	0.17	0	0.06
11	56 M	5.0	2.3	2.6	0.10	0.05	0
12	24 F	7.3	5.2	1.9	0.07	0	0.15
13	64 F	4.6	2.1	2.2	0.28	0	0.05
14	31 F	6.2	4.3	1.5	0.37	0	0
15	12 F	7.4	2.8	4.2	0.22	0.07	0.07
16*	45 M	9.1	5.8	2.6	0.55	0	0.09
17	9 F	6.7	4.0	2.6	0	0	0.07
18	27 F	5.9	3.7	1.8	0.30	0.12	0
19	11 M	4.5	1.9	2.4	0.09	0.05	0.05
20	13 M	6.4	2.9	3.1	0.32	0.06	0
21	9 F	6.0	3.7	2.1	0.24	0	0
22	23 F	7.5	4.7	2.7	0.15	0	0
23	10 M	7.2	2.8	4.1	0.22	0	0.07
24	19 F	4.6	2.1	2.1	0.41	0	0
26	18 M	11.7	6.1	3.9	1.4	0.12	0.23
27	32 M	6.7	3.1	3.4	0.13	0.07	0.07
28*	74 F	10.0					
30	65 F	5.5	3.4	1.3	0.55	0.11	0.16
32	9 M	5.7	2.5	2.6	0.40	0.11	0.06
33	8 F	6.5	2.2	3.6	0.52	0	0.19
35	19 M	5.1	2.8	2.2	0.10	0	0.06
36	13 M	6.8	3.3	2.8	0.34	0.07	0.07
39	21 F	4.9	2.6	2.2	0.10	0	0
40	35 M	7.9	5.1	2.2	0.40	0.16	0.08
41*	50 M	9.4	5.9	3.0	0.47	0	0
42	9 F	8.1	4.8	2.7	0.32	0.08	0.24
43*	72 F	5.6	3.2	2.0	0.28	0.06	0
44*	10 M	5.5	1.9	3.2	0.39	0.05	0
46	82 M	4.5	1.8	1.6	0.77	0.09	0.22
47	14 M	5.2	2.8	2.0	0.36	0.05	0.05
48*	12 F	5.3	2.6	2.6	0	0.05	0
49	21 F	12.9	7.6	4.8	0.52	0	0
51*	30 F	6.2	4.3	1.6	0.31	0	0
52	62 F	5.2	2.2	2.5	0.52	0	0
53*	14 F	7.1	4.0	2.8	0.21	0	0.07
54	7 M	6.1	3.2	2.4	0.43	0	0
55	81 M	4.8	1.7	2.9	0.19	0	0
56	77 F	7.6	5.2	2.0	0.30	0.08	0.08
57	106 F	4.8	2.2	2.4	0.10	0.05	0.05
58	65 F	6.0	3.0	2.8	0.06	0.12	0
59*	40 F	10.0	6.9	2.1	0.80	0.10	0.10
60	62 F	10.1	5.6	4.2	0.30	0	0

*Ailingnae exposed.

Hematological Findings, 1960, on Rongelap and Ailingnae Exposed Population

Subject No.	Age and Sex	WBC ($\times 10^{-3}$)	Neut. ($\times 10^{-3}$)	Lymph. ($\times 10^{-3}$)	Eosin. ($\times 10^{-3}$)	Baso. ($\times 10^{-3}$)	Mono. ($\times 10^{-3}$)
61	14 F	7.5	3.6	3.7	0.15	0	0.08
63	42 F	5.0	2.5	2.3	0.20	0	0.05
64	36 F	6.6	4.6	1.8	0.20	0	0
65	7 F	6.3	2.5	3.5	0.25	0.06	0.06
66	36 F	6.0	3.5	2.3	0.12	0	0.06
67	20 F	7.8	4.4	3.0	0.39	0	0.08
68	51 M	4.9	2.9	1.6	0.34	0	0.05
69	10 F	7.4	2.9	3.8	0.59	0.07	0
70*	23 F	4.8	2.3	2.4	0.19	0	0
71	34 F	10.8	8.3	2.3	0.11	0	0.11
72	12 F	8.4	3.3	4.9	0.16	0.08	0
73	24 M	5.3	3.6	1.5	0.10	0.05	0.05
75	18 F	7.8	5.5	1.8	0.39	0	0.08
76	17 M	4.7	1.6	2.8	0.38	0	0
78	43 F	5.6	3.5	2.1	0.06	0	0
80	52 M	10.5	6.8	3.2	0.20	0	0.20
81*	14 F	5.3	2.6	2.3	0.27	0	0.10
83**	6 M	6.2	3.1	3.0	0.06	0.06	0
84**	6 M	6.8	2.7	3.3	0.34	0	0.48
85**	5 M	8.2	3.7	4.1	0.33	0	0.08
86**	5 F	7.0	4.3	2.1	0.42	0	0.14
87**	5 F	9.1	2.9	4.3	1.7	0.09	0.09
88**	4 M	7.3	3.2	3.5	0.51	0	0.07
89**	4 M	7.5	2.7	3.9	0.53	0.15	0.22
90**	4 M	6.9	3.9	2.3	0.62	0	0
92**	4 F	11.1	4.9	4.4	1.6	0	0.22
96**	2 F	11.9	3.1	6.3	2.4	0	0.12
98**	2 M	8.8	3.5	4.6	0.70	0	0
100**	2 F	9.2	4.0	4.5	0.46	0	0.18
103**	2 F	11.6	4.8	5.6	1.2	0	0.11
104**	2 M	8.7	4.0	4.2	0.43	0	0.09
108**	2 F	9.8	3.5	5.8	0.30	0	0.20

*Ailingnae exposed.
**Nonexposed children of exposed parent or parents.

APPENDIX 5

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1959-1960

Subject No.	1959 WBC ($\times 10^{-3}$)	1959 A.P., % Neg.	1959 % Baso. per 4000-cell count	1960 WBC ($\times 10^{-3}$)	1960 A.P., % Neg.	1960 % Baso. per 4000-cell count
			Rongelap and Ailingnae Exposed Population			
1*	6.9	59	0.35	6.0	72	2.10
2	7.3	52	0.43	6.3	81	0.30
3	8.6	79	2.1	5.1	92	3.30
4	8.4	82	0.55	6.1	82	1.30
5	10.4	70	0.38	6.1	70	1.00
6*	9.0	90	0.35	9.6	88	1.10
7	6.6	52	0.33	5.5	84	1.70
8*				8.7	80	0.60
9	9.6	94	0.45			
10	8.4	86	0.33	5.6	59	0.50
11	7.5	82	0.40	5.0	87	0.75
12	8.9	63	0.30	7.3	96	0.35
13	7.9	40	0.55	4.6	82	0.48
14	6.0	37	0.20	6.2	54	0.65
15	8.6	54	0.38	7.4	81	1.25
16*	7.1	95	0.25	9.1	72	0.80
17	9.1	74	0.28	6.7	92	0.65
18	10.3	88	0.85	5.9	90	2.23
19	8.0	95	0.55	4.5	90	0.70
20	8.4	71	0.75	6.4	80	1.00
21	10.3	78	0.30	6.0	85	0.85
22	8.9	93	0.25	7.5	49	0.83
23	11.2	82		7.2	67	0.55
24	11.9	74	0.15	4.6	89	0.48
26	11.3	84	0.38	11.7	70	0.90
27	15.7	7	0.50	6.7	75	1.80
28*	15.0	91	0.23	10.0		
29*	9.0	75	0.18			
30	7.7	82	0.43	5.5	77	1.60
32	8.8	73	0.60	5.7	82	1.68
33	13.2	90	0.35	6.5	93	0.50
34	5.6	80	0.5		87	1.88
35	11.1	98	0.23	5.1	70	0.45
36	7.0	77		6.8		1.23
37	10.7	84	0.15			
38					82	
39	12.4	5	0.05	4.9	77	0.95
40	10.5	40	0.23	7.9	55	1.50
41*	7.5	84	0.25	9.4	82	0.75
42	14.0	87	0.55	8.1	83	1.28
43*	11.1	34	0.43	5.6	71	0.73
44*	12.0	55	0.23	5.5	78	1.18
45*	8.5	66	0.23		83	0.30
46	7.8	99	0.30	4.5		0.83
47	10.8	64	0.40	5.2	80	0.78
48*	13.1	35	0.30	5.3	89	0.88
49	16.7	4	0.45	12.9	83	0.68
50*	9.2	58	0.28			

*Ailingnae exposed population.

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1959-1960

Subject No.	1959 WBC ($\times 10^{-3}$)	1959 A.P., % Neg.	1959 % Baso. per 4000-cell count	1960 WBC ($\times 10^{-3}$)	1960 A.P., % Neg.	1960 % Baso. per 4000-cell count
			Rongelap and Ailingnae Exposed Population (continued)			
51*				6.2	29	0.50
52	10.3	44	0.50	5.2	58	0.60
53*	10.4	62	0.30	7.1	90	0.45
54	9.6	69		6.1	62	0.68
55	9.3	94	0.68	4.8		0.78
56	7.7	88	0.38	7.6		1.08
57	5.4	95	0.40	4.8		0.95
58	7.6	84	0.25	6.0	91	1.28
59*	12.4	66		10.0	46	0.75
60	15.0	95	0.25	10.1	82	0.78
61	8.7	65	0.18	7.5	6	0.55
62	10.3	65	0.10			
63	8.5	56	0.35	5.0	76	0.55
64	8.0	89	0.45	6.6	5	0.88
65	11.5	18	0.40	6.3	74	0.85
66	8.9	80	0.30	6.0	55	0.63
67				7.8	90	0.50
68	5.9	86	0.48	4.9	86	0.73
69	8.8	71	0.45	7.4	74	1.08
70*	7.5	80	0.35	4.8	82	0.53
71	10.3	78	0.38	10.8	69	0.43
72	10.0	79	0.28	8.4	81	0.83
73	8.7	87	0.25	5.3	63	1.13
75	8.7	83	0.18	7.8	87	0.38
76	6.4	87		4.7	69	0.73
77	7.8	68	0.40			
78	8.6	66	0.18	5.6	60	0.63
79	12.4	57	0.75			
80	10.6	72	0.35	10.5	64	0.78
81*	6.3	95	0.13	5.3	88	0.58
82	8.7	72	0.68		56	0.85
83**	10.8	74	0.10	6.2	83	0.90
84**	12.2	54	0.07	6.8	54	0.48
85**	14.0	54	0.38	8.2	85	0.65
86**	17.6	16	0.18	7.0	36	0.75
87**	11.1	41		9.1	61	1.28
88**	12.6	52	0.18	7.3	82	
89**	11.9	80	0.55	7.5	87	2.00
90**	18.4	70	0.15	6.9	83	0.50
91**	14.8	60	0.35			
92**				11.1	9	0.78
93**	18.0	85	0.15			
94**	8.9	74				
95**	14.4	96	0.30			
96**	12.4	75	0.18	11.9	90	0.75
97**	11.8	79	0.18			
98**	13.5	13	0.28	8.8	38	
100**	12.5	43	0.55	9.2	74	
103**				11.6	85	
104**				8.7	65	
108**	13.2	76		9.8	72	0.85

*Ailingnae exposed population.
**Nonexposed children of exposed parent or parents.

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1959-1960

Subject No.	WBC ($\times 10^{-3}$)	A.P., % Neg.	% Baso. per 4000-cell count	Subject No.	WBC ($\times 10^{-3}$)	A.P., % Neg.	% Baso. per 4000-cell count
			Rongelap Unexposed Population, 1959				
801	14.4	16	0.52	862	8.0	83	0.43
802	11.0	65	0.60	863	10.3	78	0.35
803	13.9	49	0.48	864	9.7	66	0.48
805	15.2	65	0.30	865	8.3	82	0.68
806	11.1	59	0.38	866	10.7	43	0.45
807	20.2	52	0.33	867	10.7	83	0.38
808	12.7	76	0.25	868	6.9	48	0.35
809	11.8	71	0.50	869	11.1	90	0.63
810	9.0	86	0.45	870	16.2	59	0.60
811	12.6	66	0.33	871	10.0	64	0.55
812	14.7	76	1.08	872	9.1	80	0.72
813	10.6	70	0.78	873	9.4	11	0.50
814	11.1	94	0.50	874	9.5	80	0.35
815	8.7	83	0.80	875	7.0	21	0.70
816	11.8	91	0.38	876	7.9	55	0.43
818	12.3	94	0.30	878	7.9	66	0.30
819	6.4	95		880	11.6	70	0.38
820	12.2	56	0.57	881	11.7	50	0.48
821	9.1	89	0.35	882	9.0	19	0.25
822	9.6	87	0.25	884	9.3	100	0.30
823	10.4	82	0.48	886	12.3	8	0.17
825	10.8	78	0.27	887	10.5	84	0.56
826	10.5	88	0.35	889	7.3	30	0.48
827	4.5	89	0.43	891	9.5	83	0.33
828	11.5	75	0.45	892		38	
829	7.3	69	0.55	893	7.5	86	0.50
830	7.4	72	0.23	894	10.8	56	0.22
831	8.0	58	0.38	895	14.7	67	0.30
832	10.8	12	0.27	896	12.8	96	0.52
833	7.9	89	0.43	898	8.9	84	0.35
834	10.1	13	0.65	899	6.5	86	0.40
835	10.1	90	0.78	900	18.4	24	0.43
836	9.5	57	0.52	901	15.4	30	0.27
838	10.2	70	0.40	902	22.0	48	0.70
839	11.7	80	0.60	903	10.8	75	0.48
840	9.6	76	0.85	904	12.7	36	0.28
841	11.2	79	0.72	905	15.3	47	0.25
842	10.6	97	0.57	906	12.7	37	0.50
843	10.2	20	0.50	908	8.0	87	0.38
844	9.7	90	0.72	911	8.0	93	0.27
845	8.4	91	0.23	912	13.5	84	0.27
846	12.1	17	0.52	913	14.3	90	0.35
849	11.9	59	0.38	914	6.8	97	0.20
850	11.8	93	0.45	916	11.4	80	0.38
851	8.8	64	0.65	921	32.4	69	0.43
852	10.9	94	0.27	922	8.8	82	0.25
853	7.6	88	0.48	923	13.6	76	0.33
854	9.6	74	0.55	925	12.4	79	0.50
855	8.9	79	0.65	926	9.9	71	0.43
856	6.8	41	0.50	927	8.4	87	0.30
858	6.6	71	0.40	928	7.7	80	0.27
859	9.3	86	0.75	930	16.6	87	0.23
860	8.5	96	0.30	932	9.8	68	0.35
861	8.3	85	0.43	933	8.6	31	0.45

Individual WBC, Basophil, and Alkaline Phosphatase Determinations, 1959-1960

Subject No.	WBC ($\times 10^{-3}$)	A.P., % Neg.	% Baso. per 4000-cell count	Subject No.	WBC ($\times 10^{-3}$)	A.P., % Neg.	% Baso. per 4000-cell count
			Rongelap Unexposed Population, 1959 (Continued)				
934	12.8	35	0.55	968	7.5	82	0.27
935	8.7	46	0.38	969	11.2	89	0.38
939	7.9	89	0.48	971	8.2	60	0.25
940	16.2	53	0.38	973	6.4	75	0.35
944	10.0	78	0.40	976	11.0	72	0.25
946	10.3	85	0.23	977	9.4	79	0.43
947	12.0	61	0.33	978	7.3	67	0.35
952	14.7	48	0.23	979	9.8	1	0.38
953	9.7	65	0.30	980	13.0	20	0.33
955	7.8	81	0.30	989	10.6	73	0.40
956	9.1	57	0.43	991	7.6	57	0.27
958	12.4	90	0.20	992	9.7	35	0.50
959	11.5	66	0.30	993	10.6	62	0.30
960	10.3	66	0.38	996	11.4	73	0.23
963	9.0	69	0.25	1003	9.2	66	0.33
966	7.2	77	0.40	1005	8.5	77	0.38
967	13.6	85	0.40	1007	8.8	82	0.38

APPENDIX 6

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT*	Cholesterol, mg %
				Rongelap Exposed					
1	O	N	Rh_1Rh_1	kk	+	Neg	1–1	0	204
2	O	N	"		+	"	2–1	1/8	148
4	A_1	N	"		+	"	2–1	0	184
5	B	N	"		Neg	"	2–1	1/4	
6	O	N	"		"	"	2–2	0	
7	A_1B	N	"	kk	+	"	1–1	0	148
9	O	M	"	kk	+	"	2–2	0	
10	O	N	"	kk	+	"	2–1	0	
11	O	N	"	kk	+	"	2–2	0	155
12	A_1B	N	"	kk	+	"	1–1	1/8	
13	A_1B	N	"	kk	+	"	2–1	0	140
14	O	N	"	kk	+	"	2–1	0	207
15	B	N	"	kk	+	"	2–2	0	135
16	B	M	"	kk	+	"	2–2	0	162
17	O	N	"		Neg	"	2–1	1/8	
18	A_1	N	"	kk	+	"	2–1	0	149
19	O	N	"	kk	Neg	"	2–1	0	
20	A_1	N	"	kk	+	"	2–1	0	
21	O	N	"	kk	+	"	2–2	0	
22	A_1	N	"	kk	+	"	1–1	0	
23	O	N	"	kk	+	"	2–2	0	141
24	A_1B	N	"	kk	+	"	1–1		
26	O	N	"	kk	+	"	2–1	1/4	
27	O	MN	"	kk	+	"	1–1	0	
28	O	N	"		+	"	2–1	1/8	
29	O	N	"		+	"	2–1	1/4	
30	O	N	"	kk	+	"	1–1	0	
31	O	;N	"	kk	+	"	1–1		
32	O	N	"	kk	+	"	2–2	1/4	
33	O	N	"		+	"	2–1	1/4	
34	A_1	N	"	kk	+	"	2–1	0	
35	O	MN	"	kk	+	"	2–1	0	
36	O	N	"	kk	+	"	1–1	0	149
37	O	N	"	kk	+	"	2–1		
39	B	MN	"	kk	+	"	2–1	1/8	
40	O	N	"	kk	+	"	2–1	0	
41	B	MN	"	kk	+	"	2–1	0	
42	O	N	"		+	"	2–1	0	
43	A_1	N	"	kk	+	"	1–1	1/16	
44	O	M	"	kk	+	"	2–2	0	
45	O	MN	"	kk	Neg	"	2–2	0	
46	B	N	"		+	"	2–1	0	
47	O	N	"	kk	Neg	"	2–1	1/4	
48	B	N	"	kk	+	"	1–1	0	121
49	O	MN	"	kk	+	"	2–1	0	
50	O	MN	"	kk	+	"	2–1	0	
51	A_1B	M	"	kk	+	"	2–1		
52	B	N	"	kk	Neg	"	2–2	0	182
53	O	N	Rh_1rh	kk	+	"	2–2	0	132

*Bentonite flocculation test; positive = 1/32 or higher dilution.

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Rongelap Exposed (continued)					
55	B	MN	Rh_1Rh_1		+	Neg	2–1	0	143
56	O	MN	"		Neg	"	1–1	1/4	
57	A_1	N	"		"	"	2–2	0	
58	A_1	N	"	kk	+	"	1–1	0	
59	O	M	"	kk	+	"	1–1	0	
60	A_1	N	"	kk	+	"	1–1	0	
61	O	MN	"	kk	+	"	2–1	1/8	
62	B	N	"	kk	+	"	2–1	0	
63	O	N	"	kk	+	"	1–1	0	150
64	O	MN	"	kk	+	"	2–1		
65							1–1	0	
66	O	MN	"	kk	+	"	2–1	0	
68	O	MN	"	kk	+	"	2–1	1/8	
69	B	N	"	kk	+	"	2–1	1/16	
70	O	N	"	kk	Neg	"	1–1	0	132
71	A_1	N	"	kk	+	"	1–1	1/4	
72	O	N	"	kk	Neg	"	2–2	1/16	137
73	O	N	"	kk	+	"	2–1	0	
74	A_2B	N	"	kk	+	"	2–1	0	
76	B	N	"	kk	Neg	"	1–1	1/4	134
77							2–1	0	
78	A_1B	N	"	kk	+	"	1–1	0	165
79	O	N	"	kk	+	"	1–1	0	127
80							2–2	0	
81	O	MN	"	kk	Neg	"	2–2	0	152
82	O	N	"	kk	+	"	1–1	0	
				Rongelap Unexposed					
815	A_1	N	Rh_1Rh_1	kk	+	Neg	2–2	0	
816	O	N	"	kk	+	"	1–1	0	
817	B	N	"	kk	+	"	2–1	0	
818	A_1	N	"	kk	+	"	2–2	1/8	
819	O	N	"	kk	Neg	"	2–1	0	
820	A_1	N	"	kk	"	"	1–1	0	
821	A_1	N	"	kk	+	"	1–1		
822	O	N	"	kk	+	"	2–2		
823	O	N	"	kk	Neg	"	2–1	0	
824								0	
825	O	MN	"	kk	"	"	2–1	1/8	163
826							2–2	0	
827							2–1	0	
828	O	N	"	kk	+	"	2–1	0	
829	O	M	"	kk	+	"	2–1	1/8	
830	A_1	N	"	kk	+	"	0	0	
831	A_1	N	"	kk	+	"	2–1	0	
832	O	N	"	kk	+	"	1–1	0	
833	O	MN	"	kk	+	"	1–1	0	
834	O	N	"	kk	+	"	1–1	0	
835	O	N	"	kk	+	"	2–1	0	
836	O	N	"	kk	+	"	1–1	0	155
838	O	N	"	kk	+	"	2–2	1/8	
839	O	N	"	kk	+	"	2–1	0	
840	A_1	N	"	kk	+	"	1–1	0	
841	O	N	"	kk	+	"	2–1	1/256	213
842	O	N	"	kk	+	"	2–2	0	

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Rongelap Unexposed (continued)					
843	O	N	Rh_1Rh_1	kk	+	Neg	1–1	0	181
844	A_1	MN	Rh_1rh	kk	+	"	2–1	.0	149
845	O	N	Rh_1Rh_2		Neg	"	2–1	1/4	
846	O	N	"	kk	+	"	2–2	0	
849	A_1	N	"	kk	+	"	1–1	0	
850	O	N	"	kk	+	"	2–1	0	164
851	A_1	N	"	kk	+	"	2–1	1/4	237
852	O	N	"	kk	+	"	2–1	0	162
853	O	N	"	kk	+	"	2–2	0	
954	B	N	"	kk	+	"	2–1	0	182
855	O	N	"	kk	+	"	2–1	0	208
856	O	N	"	kk	+	"	2–1	0	172
858	A_1B	N	Rh_1rh	kk	+	"	1–1	0	180
859	A_1	N	Rh_1Rh_1	kk	+	"	1–1	0	202
860	B	M	"		Neg	"	2–1	0	187
861	O	MN	"	kk	+	"	1–1	0	173
862	O	N	"	kk	+	"	2–2	0	
863	A_1	N	"	kk	+	"	1–1	0	
864	O	MN	"	kk	+	"	2–2	0	170
865	A_1	N	"	kk	Neg	"	1–1	0	
866							1–1		
867	O	N	"	kk	+	"	1–1		
868	O	MN	"	kk	+	"	2–1	0	207
869	O	N	"	kk	+	"	2–2	0	
871	O	MN	"	kk	+	"	2–1	0	157
872	B	MN	"	kk	+	"	1–1	0	
873	B	M	"	kk	+	"	1–1	0	187
874	B	N	"	kk	+	"	1–1	0	
875	O	N	"	kk	+	"	2–1	0	181
876	B	N	"	kk	+	"	2–1	0	154
877	O	M	"	kk	+	"	2–1	0	
878	O	MN	"	kk	+	"	2–1	0	
880	B	N	"	kk	+	"	1–1	0	
881	O	M	"	kk	+	"	2–1	0	
882	O	N	"	kk	+	"	1–1	0	
883	O	N	"	kk	+	"	2–1	0	
884							2–1	1/4	
885	B	N	"	kk	+	"	1–1	0	
886							2–2	1/64	
887	A_1B	N	"	kk	+	"	1–1	0	
888	B	MN	"	kk	+	"	2–1	0	
889	A_1	MN	"	kk	+	"	1–1	0	150
891	A_1B	N	"	kk	+	"	1–1	0	
892	A_1B	N	"	kk	+	"	1–1		
893	O	MN	"	kk	+	"	2–1	1/16	
894	O	MN	"	kk	+	"	1–1	0	152
895	B	MN	"	kk	+	"	1–1	0	146
896	O	MN	"	kk	+	"	1–1	0	165
898	O	N	"	kk	+	"	2–1	1/4	
899							2–2	0	
908	A_1	N	"		+	"	1–1	0	
911	O	MN	"		+	"	2–1		
912	A_1	N	"		+	"	2–2	1/4	
913	O	N	"		Neg	"	2–2	0	136
914	O	N	"		"	"	1–1	0	
916	A_1	N	"		+	"	2–1	0	135

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Rongelap Unexposed (continued)					
921	O	MN	Rh_1Rh_1		+	Neg	2–1	0	
922	O	M	"		Neg	"	2–2	0	
925	O	MN	"		"	"	2–1	0	129
928	O	N	"		+	"	1–1	0	239
932	O	M	"		+	"	2–1	0	
933							0	0	
934	O	N	"		Neg	"	2–1	0	
935	O	N	Rh_2Rh_0		+	"	2–1	0	
939	O	MN	Rh_1Rh_1		+	"	2–2	0	
940	A_1	N	"		+	"	2–1	0	
944	B	M	"		Neg	"	2–1	0	
947	O	N	"		+	"	2–1	0	
953	O	N	Rh_2		Neg	"	1–1	1/64	
955	O	M	Rh_1Rh_1		"	"	2–1	0	
956	O	MN	"		"	"	2–1	0	
958	O	N	"		+	"	2–1	0	
959	O	MN	"		Neg	"	2–1	0	
960	O	MN	"		+	"	2–2	0	
963	A_1	N	"		Neg	"	2–1	0	
966	A_1	N	"		"	"	2–1		
967	O	N	Rh_2Rh_0		+	"	2–2	0	
968							1–1	0	
969	O	N	Rh_1Rh_1		+	"	1–1	0	
971	O	N	"		Neg	"	2–2	0	
973	O	M	"		+	"	2–2	0	
976	O	N	"		+	"	2–1	0	
977	O	N	"		+	"	2–1	0	
978	A_1	N	"		+	"	2–1	0	
980	B	MN	"		+	"	2–1	0	
989							2–1	0	
991	A_1	N	"		Neg	"	1–1	0	
993	O	N	"		+	"	2–1	0	
996	O	MN	"		+	"	2–1	1/4	
1003	A_1B	N	"		Neg	"	1–1	0	
1005	A_1	N	"		"	"	1–1	0	
				Majuro Controls					
4000	O	N	Rh_2Rh_0			Neg	2–2	0	
4002	B	N	Rh_1Rh_1		+	"	2–1	0	
4003	O	N	"			"	0		
4004	O	N	"		+	"	2–1	0	
4005	B	M	"			"	1–1	0	
4006	O	MN	"		+	"	2–2		
4007	A_1	N	"		Neg	"	2–2	0	
4009	O	M	"		+	"	2–1	0	
4010	A_1	N	"		Neg	"	0		
4012	A_1	M	Rh_2Rh_0		+	"	2–2		
4013	O	N	Rh_1Rh_1			"	1–1		
4015	B	MN	"			"	2–2	0	
4016	O	MN	Rh_2Rh_0				2–1		
4017	A_1	N	Rh_1Rh_1		+	"	2–1	0	
4018	A_1	N	"		Neg	"	1–1		
4020	O	N	"		+	"	2–1		
4022	A_1	N	"		+	"	2–1		
4023	O	N	"		+	"	2–2	0	

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Majuro Controls (continued)					
4024	A_1	N	Rh_1Rh_2		Neg	Neg	2–2	0	
4025	O	N	"		+	"	2–1	0	
4026	A_1	N	Rh_zRh_0		+	"	2–1	0	
4028	A_1	N	Rh_1Rh_1		Neg	"	1–1	0	
4030	O	MN	"		+	"	2–1	0	
4031	O	MN	"		+	"	2–1	0	
4032	O	N	"			"	2–2	0	
4033	B	MN	"		+	"	2–1		
4034	A_1	MN	"		+	"	2–1		
4035	A_1	N	"			"	2–1		
4036	O	M	"		+	"	2–2		
4037	O	N	"			"	2–1		
4039	B	MN	"		+	"	2–2	0	
4040	O	MN	Rh_1rh			"	1–1	0	
4041	O	M	Rh_zRh_0			"	1–1	0	
4042	O	N	Rh_2			"	2–1		
4043	O	M	Rh_1Rh_1		+	"	1–1		
4046	O	N	"		+	"	1–1		
4048	B	M	Rh_zRh_0		+	"	1–1		
4049	B	N	"			"	2–2		
4050	O	MN	Rh_1Rh_1		+	"	2–2		
4055	O	N	Rh_zRh_0			"	2–1		
4056	B	N	Rh_1Rh_1			"	1–1		
4057	O	N	"			"	2–2	0	
4058	O	M	"			"	2–2		
4059	B	MN	"			"	2–2	0	
4063	A_1	N	Rh_1rh			"	2–1	0	
4064	O	N	Rh_1Rh_1			"	2–2		
4066	O	N	"		+	"	1–1	0	
4067	B	N	"		+	"	2–2		
4068	A_1	N	"			"	1–1	0	
4069	A_1	MN	Rh_zRh_0		+	"			
4070	O	N	Rh_1Rh_1			"	2–1		
4072	B	N	"			"	2–1	0	
4073	A_1	N	"			"	2–1		
4075	O	MN	Rh_1rh			"	2–2		
4078	O	N	Rh_1Rh_1			"	2–1	0	
4081	O	M	"		Neg	"	2–2	0	
4082	A_1	M	"			"	2–1	0	
4083	O	MN	Rh_zRh_0		"	"	2–1		
4084	A_1B	N	Rh_1Rh_1		+	"	2–1		
4085	B	N	"			"	1–1		
4086	B	N	"			"	2–1		
4087	B	N	"			"	2–2		
4088	O	N	"			"	2–1		
				Utirik Population					
2101	A_1B	N	Rh_1Rh_1		+	Neg	1–1	0	181
2104							2–1	0	202
2105	O	N	"		+	"	2–2		215
2107	B	N	"		+	"	1–1	0	241
2108	O	MN	"		Neg	"	2–1		133
2110	O	N	Rh_1rh		+	"	2–1	0	
2112	B	N	Rh_1Rh_1		Neg	"	2–2	1/8	
2114							2–1	0	

Blood Groupings and Haptoglobins, 1958–1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Utirik Population (continued)					
2116	O	MN	Rh_1Rh_1		+	Neg	2–2	0	
2118							1–1	1/128	
2120	O	MN	"		+	"	2–2		
2121	O	MN	"		+	"	2–1		269
2123	O	MN	"		+	"	2–2		
2125	O	MN	"		+		2–1	1/4	225
2126	A_1	N	"		+	"	1–1		187
2128	O	N	"		+	"	2–1	0	
2129	A_1	MN	"		+	"	2–1	0	237
2132	O	N	"		+	"	2–2		147
2136	O	N	Rh_1rh		+	"		1/4	176
2139	O	N	Rh_1Rh_1		+	"	2–1	0	
2140	A_1	MN	"		+	"	2–2	1/4	216
2141	B	N	"		+	"	1–1	0	172
2142							2–2		
2144	O	N	"		+	"	2–2		
2145	O	N	Rh_1rh		+	"	2–1		220
2146	O	N	Rh_1Rh_1		+	"	2–1	0	187
2148	A_1	MN	"		+	"	2–2		
2149	O	MN	"		+	"	2–1		200
2150	O	N	"		Neg	"	2–1	0	171
2152	O	MN	"		+	"	2–1		212
2154	O	MN	"		+	"	2–2		
2156	A_1	MN	"		+	"	2–2		
2157	O	N	Rh_0		+	"	1–1		195
2158	A_1	MN	Rh_1Rh_1		+	"	1–1	0	175
2162	B	N	Rh_1rh		+	"	2–1		263
2164	O	MN	Rh_1Rh_1		+	"	2–2		179
2168	O	M	Rh_0		+	"	2–2		200
2169	O	MN	Rh_1Rh_1		+	"	2–1	1/256	
2172	O	N	"		+	"	2–2		
2175	O	N	"		+	"	2–2		252
2176	A_1	N	"		+	"	2–2	0	161
2182	O	N	"		+	"	2–2	0	220
2183							2–1		217
2186	B	N	"		+	"	2–2	0	202
2188	O	N	"		+	"	2–1		166
2190	B	MN	"		+	"		0	271
2193	O	N	"		+	"	1–1	1/4	220
2198	O	N	"		+	"	2–2		251
2199	A_1B	N	"		+	"	1–1		
2202	B	N	"		+	"	2–1		
2203	O	N	"		+	"	2–1		
2204	O	MN	"		+	"	2–1		
2206	O	MN	"		+	"	2–2		
2214							2–1		
2216	O	N	"		+	"	2–2		
2223	A_1	MN	"		+	"	2–2		
2225	O	N	"		+	"	1–1		140
2229	O	N	"		+	"	1–1		167
2230	O	N	"		+	"	1–1		245
2238	O	N	Rh_1rh		+	"	2–1		256
2244							2–2		
2246	O	N	Rh_1Rh_1		+	"	2–1		197
2247	O	N	Rh_1rh		+	"	2–1		191
2248							2–1		157

Blood Groupings and Haptoglobins, 1958-1959, and BFT and Cholesterol Levels, 1959

Subject No.	ABO group	MN type	Rh-Hr	Kell	Duffy	Diego	Haptoglobin type	BFT	Cholesterol, mg %
				Utirik Population (continued)					
2252	O	MN	Rh_1Rh_1		Neg	Neg	2–1		195
2253	O	M	"		+	"	2–1		148
2256	O	N	"		+	"	2–1		179
2257	O	MN	Rh_1rh		+	"	2–1		
2279	O	MN	Rh_1Rh_1		+	"	2–2		150
2280	O	N	"		+	"	2–2		237

APPENDIX 7

Cs^{137} and Zn^{65} Body Burdens, in $m\mu C/kg$, of Marshall Island Inhabitants

Males						Females					
Age 1–15			Age >15			Age 1–15			Age >15		
Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}
Rongelap Exposed											
3	5.65	7.34	4	17.4	9.14	15	9.62	6.53	12	14.5	8.44
5	14.0	14.0	7	16.7	13.8	17	15.7	11.0	13	7.23	5.31
23	10.3	12.5	9	3.67	4.78	19	12.9	10.8	14	11.9	14.8
32	23.9	10.0	10	16.0	8.09	20	8.55	7.24	18	8.01	10.0
47	17.3	7.80	11	15.2	6.50	21	14.4	8.56	22	5.71	6.53
54	7.51	8.92	26	11.5	13.1	33	12.5	6.00	24	13.0	7.19
76	11.3	9.16	27	16.0	6.05	36	13.4	9.19	30	12.1	9.44
83	10.7	13.0	40	9.64	10.7	42	10.8	14.0	39	14.4	6.45
85	10.7	12.7	79	16.4	15.5	61	9.38	5.63	49	7.95	9.62
			82	19.4	11.4	65	6.67	10.0	58	11.2	8.73
						69	11.0	5.71	60	9.25	6.84
						72	11.1	8.20	63	17.8	6.26
									64	11.2	11.9
									66	10.5	15.6
									17	7.57	10.2
									78	14.1	11.5
Av	12.4 ±6.15	10.6 ±2.24		14.2 ±5.12	9.91±3.07		11.3±2.74	8.57±2.64		11.0±3.35	9.36±3.33
Ailingnae Exposed											
6	7.53	5.38	16	11.2	11.8	8	6.67	9.74	1	8.70	8.29
44	10.2	10.6	29	8.35	9.11	48	13.1	7.67	43	13.3	11.0
			41	15.7	15.9	53	10.5	5.38	45	11.7	7.54
			50	10.6	20.9	81	9.68	9.41	59	16.9	13.6
									70	6.50	12.0
Av	8.87	7.99		11.5±3.57	14.4±5.72		9.99±3.12	8.05±2.13		11.4±4.48	10.5±2.6
Rongelap Unexposed											
813	8.18	8.18	823	8.89	11.1	805	10.4	6.04	825	10.7	5.56
814	14.9	9.23	830	10.4	10.5	810	6.49	13.0	829	6.11	8.15
815	9.25	7.93	831	15.0	9.45	812		10.1	832	6.24	8.58
818	24.1	7.32	833	9.92	8.74	816	11.4	11.8	835	6.54	9.93
819	14.3	9.63	834	21.3	10.6	821	7.92	8.50	839	7.92	11.6
820	9.03	6.14	836	11.4	10.3	891	12.4	7.95	841	6.88	4.59
822	8.08	7.80	838	5.13	9.95	911	6.45	6.45	843	6.53	11.4
863	12.4	8.80	840	18.8	13.0	955	9.86	17.4	844	9.39	7.35
869	10.6	6.04	845	9.84	6.09	959	6.65	10.3	846	11.8	10.8
870	6.30	3.94	849	12.1	9.92	960	9.91	8.62	851	10.4	11.3
874	21.5	8.70	850	18.8	13.0	977	12.2	7.19	852	9.59	11.0
887	11.0	5.90	853	15.5	5.01	978	10.8	10.8	854	11.5	10.9
892	7.76	8.70	855	19.3	8.93	980	12.5	8.00	858	6.76	7.34
912	12.7	7.51	856	21.8	10.5	993	10.5	6.58	859	12.9	8.77
921	9.04	6.21	860	9.49	4.75	996	10.1	8.18	861	6.06	6.06
939	8.18	10.0	862	12.0	2.80	998	11.3	8.26	865	10.8	7.71

Cs^{137} and Zn^{65} Body Burdens, in mμC/kg, of Marshall Island Inhabitants

Males						Females					
Age 1–15			Age >15			Age 1–15			Age >15		
Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}	Subject No.	Cs^{137}	Zn^{65}
				Rongelap Unexposed (continued)							
940	9.27	6.56	864	13.6	17.3				867	9.36	6.24
967	14.7	11.9	868	9.72	6.61				871	17.2	5.96
971	15.4	12.1	872	19.0	13.1				876	10.0	6.10
976	8.31	4.89	873	19.2	7.23				889	20.5	12.7
			875	16.3	11.4				893	9.29	7.75
			878	13.0	8.49				894	5.61	10.6
			880	16.3	13.7				895	8.81	5.87
			881	16.7	12.9				896	8.81	11.5
			882	9.12	12.0				908	11.2	7.60
			910	24.3	14.8				914	11.4	9.94
			935	11.2	15.9				916	7.91	12.1
			944	11.4	8.64				922	14.2	9.32
			945	9.49	9.49				934	8.54	8.92
			947	18.7	9.27				942	11.4	10.9
			953	8.68	10.3				957	8.03	5.40
			958	14.8	9.57				970	9.42	6.01
			963	20.9	8.99				982	9.48	7.65
			966	9.05	6.79						
			969	19.0	12.3						
			973	12.3	6.29						
			1005	4.66	4.96						
Av	11.8±4.78	7.87±1.98		14.0±4.72	9.86±2.86		9.93±2.11	9.32±2.77		9.77±2.82	8.65±2.24
				Utirik							
2102	2.65	2.35	2101	4.03	4.81	2113	2.78	3.21	2104	4.75	3.22
2106	3.58	3.02	2103	7.82	4.26	2126	3.03	2.75	2107	3.02	1.63
2108	4.83	1.35	2105	4.61	4.02	2128	2.83	1.49	2116	4.46	1.96
2115	5.76	1.52	2110	4.16	6.63	2149	4.02	1.92	2129	2.13	3.02
2142	5.51	3.11	2112	2.79	3.23	2159	3.10	2.24	2139	7.07	3.53
2144	5.43	1.94	2114	1.67	1.39	2160	4.40	2.55	2140	4.17	2.53
2151	3.52	2.14	2120	4.05	3.40	2197	5.60	2.03	2141	2.67	2.17
2155	7.14	4.40	2121	4.26	2.28	2210	4.84	4.03	2154	2.81	2.49
2174	5.12	1.85	2123		6.01	2213	9.04	3.11	2158	3.23	3.08
2179	4.29	2.46	2125	3.77	4.56	2218	5.20	2.88	2164	3.70	1.77
2207	4.35	2.58	2145	5.25	4.07	2225	3.23	2.63	2172	2.08	2.23
2232	4.51	2.36	2152	5.39	3.49	2227	8.09	5.15	2224	4.73	2.51
2233	2.75	2.05	2157	3.81	5.88	2228	5.43	2.63	2230	2.95	2.77
2257	4.24	4.03	2166	6.39	6.22	2256	1.73	2.58	2246	2.54	2.03
			2178	4.43	3.15				2247	3.17	2.50
			2185	5.06	3.43						
Av	4.55±1.23	2.51±0.80		4.50±1.60	4.18±1.55		4.52±1.87	2.80±0.80		3.57±1.26	2.50±0.58

APPENDIX 8

Urinary Sr^{90} Values in Marshallese, 1959

Males				Females			
Subject No.	Sr^{90}, $\mu\mu C$	Volume, ml	Sr^{90}, $\mu\mu C/l$	Subject No.	Sr^{90}, $\mu\mu C$	Volume, ml	Sr^{90}, $\mu\mu C/l$
			Rongelap Exposed, Age 1–15				
76	5.2	2160	2.4	15	9.0	2160	4.1
76	4.4	2100	2.1	36	8.8	2400	3.6
Av			2.3				3.9
			Age >15				
7	10.5	1500	7.0	12	3.8	2160	1.8
9	8.3	1700	4.8	12	4.9	2200	2.2
26	15.3	2110	7.2	14	13.0	1400	9.2
26	11.4	2160	5.2	22	5.8	1400	4.1
27	4.5	1150	3.9	22	12.5	1710	7.3
27	4.5	1900	2.3	24	3.9	1500	2.6
40	18.6	1700	10.9	39	12.8	1450	8.8
79	21.7	1290	16.8	66	14.2	1510	9.4
				66	7.3	2100	3.4
Av		1690	7.3±5.1			1715	5.4±2.2
			Ailingnae Exposed, Age >15				
41	11.8	2200	5.3	45	4.0	1300	3.0
			Kwajalein – Ebeye, Age >15				
2404	0.4	940	0.4	2412	0.3	1010	0.3
2405	0.8	1920	0.4	2413	0.6	1610	0.4
2419	—	270	—	11*	0.5	320	1.5
2416	0.4	340	1.1				
2414	1.7	465	—				
2410	0.13	385	0.33				
Av			0.56±0.37				0.7±0.7
			Rongelap Unexposed, Age 1–15				
818	6.2	1100	5.6				
887	9.0	1000	9.0				
874	10.1	1500	6.7				
Av			7.1±2.0				
			Age >15				
830	12.0	2200	5.4	843	3.5	2160	1.6
830	8.0	1110	7.2	843	5.7	1710	3.3
831	5.5	1400	3.9	865	7.6	2150	3.5
838	24.1	2025	11.8	876	7.4	1000	7.4
838	5.5	1490	3.6	893	11.5	2300	5.0
849	13.4	1500	8.9	895	4.7	1050	4.4
963	6.4	1700	3.7				
963	7.8	1300	6.0				
882	3.4	850	4.0				
Av		1510	5.6±2.8			1730	4.2±2.3

*Member of original exposed Rongelap group, living on Ebeye since 1958.

Urinary Sr^{90} Values in Marshallese, 1959

Males				Females			
Subject No.	Sr^{90}, $\mu\mu C$	Volume, ml	Sr^{90}, $\mu\mu C/l$	Subject No.	Sr^{90}, $\mu\mu C$	Volume, ml	Sr^{90}, $\mu\mu C/l$
			Utirik, Age 1–15				
				2128	1.7	430	3.9
			Age >15				
2110	2.3	1750	1.3	2236	2.1	660	3.1
2123	1.7	1060	1.6	2246	1.0	510	1.9
2125	2.6	1380	1.8				
2145	1.9	680	2.7				
2152	0.14	600	0.23				
Av			1.5±0.9				2.5